Caring for patients in the intensive care setting is a challenging but potentially rewarding experience. As we enter the intensive care environment each one of us brings a unique mix of skills and knowledge. Inevitably though we must find a common ground on which to base our management, without which optimal patient care and safety cannot be achieved.

The purpose of this manual is not to provide definitive answers for each problem, nor is it meant to be prescriptive in nature, but rather it describes a number of standardised approaches and helpful guidelines to facilitate good patient care.

While you are working in this unit, no matter what your level of experience, you will encounter situations where you feel uncomfortable, confused or even scared. While this manual is intended to assist you in caring for your patients, you should not be embarrassed to seek help from those around you, including the staff specialist on duty and senior nursing staff.

Colleagues outside of the Waikato Hospital ICU that make use of these guidelines do so at their own risk. Furthermore, editions older than 12 months may vary significantly from current practice.

I would welcome your suggestions for future versions. You will find under reference sections in each chapter, references to articles which are useful further reading.

Many Intensive Care Units have developed their own sets of guidelines and medical officer handbooks, particularly the Royal Adelaide Hospital ICU handbook which for years was a useful companion. It is not surprising therefore that these guidelines draw on that document and similar documents for structure and content. The guidelines represented herein are a result of the contributions of a number of the staff of the unit, subjected to peer and colleague review. Dr Grant Howard was the driver and effector of the first several editions of this handbook at Waikato Hospital. A large debt of gratitude is owed him for this.

Dr Nicholas Barnes, Editor 2007.

The discipline of intensive care revolves around;
- the reconstruction of homeostasis when patients homeostatic control mechanisms fail.
- the withdrawal of this support in a manner which enables bodily homeostatic mechanisms to resume control.

It is acknowledged that the reflexual feedback control systems of the body form a complex topographical problem space over which it is the skill and vision of Intensive Care Specialist that enables navigation. It is noted there are many different paths to similar conclusions and there are many different styles of problem resolution. This manuscript serves to document useful guidelines and illustrate in part the styles of the involved Intensivists. It is in no way to be used as a directive for a definitive course of action without first taking into consideration each individual patient and their personal situation.

Dr Edward Coxon MBChB BMedSci (dist)

Waikato Hospital is the 600 bed tertiary referral hospital and trauma centre of the midland region of New Zealand and serves a population area of 800 000. Waikato Hospital has specialist services in most fields apart from paediatric cardiac surgery. The Waikato Intensive Care Unit has 15 beds and admits over 1100 patients a year.

The Waikato Intensive care unit is a mixed unit with 15% paediatric and 85% adult admissions. Approximately 30% of the admissions are post cardiac surgical. The remainder are a mixture of trauma, medical and surgical patients. 76% of admissions are ventilated. Our average APACHE II score is 16 and we have a mortality rate of about 9%.

The intensive care staff also assist in the management of patients in the High Dependency Unit which has 12 beds and admits over 1600 cases per year. The intensive care provides medical and nursing transport teams for inter-hospital transfers and oversees parenteral nutrition within the hospital and home parenteral nutrition for the Waikato and Midland communities. Staff also participate in trauma and cardiac arrest rosters.

The Waikato Intensive Unit has C24 classification from the Faculty of Intensive Care of the Australian and New Zealand College of Anaesthetists and as such is recognised for unlimited core intensive care specialist training.

The Intensive Care unit medical staff consists of 5 consultants, one senior registrar and ten registrars. We have a nursing staff of about 65 full time equivalents for ICU, 25 for HDU and 3 full time respiratory technicians.

The unit has an active research and teaching programs. We have a number of safety and quality initiatives in place.
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### Administration

#### Staffing

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<th>Name</th>
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</thead>
<tbody>
<tr>
<td><strong>Director</strong></td>
<td>Dr Nicholas Barnes</td>
</tr>
<tr>
<td><strong>Consultant Medical Staff</strong></td>
<td>Dr Frank Van Haren</td>
</tr>
<tr>
<td></td>
<td>Dr Nicholas Barnes</td>
</tr>
<tr>
<td></td>
<td>Dr Grant Howard (SOT)</td>
</tr>
<tr>
<td></td>
<td>Dr Rob Frengley</td>
</tr>
<tr>
<td></td>
<td>Dr John Torrance</td>
</tr>
<tr>
<td></td>
<td>Dr Peter Marko</td>
</tr>
<tr>
<td><strong>Clinical Nurse Leaders</strong></td>
<td>Mathew Hughes</td>
</tr>
<tr>
<td></td>
<td>Simon Mehari</td>
</tr>
<tr>
<td></td>
<td>Christine Craig</td>
</tr>
<tr>
<td></td>
<td>Diana Mallett</td>
</tr>
<tr>
<td></td>
<td>Alison McAlley</td>
</tr>
<tr>
<td><strong>Senior Typist / Unit Secretary</strong></td>
<td>Dianne Takiai-Dawson</td>
</tr>
<tr>
<td><strong>Administrative Assistant</strong></td>
<td>Jill Brough</td>
</tr>
<tr>
<td><strong>Research Nurse</strong></td>
<td>Mary La Pine</td>
</tr>
<tr>
<td><strong>Respiratory Technician-Charge Technical Advisor</strong></td>
<td>Paul Goble</td>
</tr>
<tr>
<td><strong>Nurse Educator</strong></td>
<td>Sarah Walker</td>
</tr>
<tr>
<td></td>
<td>Mark Reynolds</td>
</tr>
<tr>
<td><strong>Organ Donor Co-ordinators</strong></td>
<td>Alison McAlley</td>
</tr>
<tr>
<td></td>
<td>Sue MaCaskill</td>
</tr>
</tbody>
</table>

#### Registrars

There are two levels of Registrar in the unit.

- **Senior Registrar**
  - Advanced vocational trainees, rostered according to seniority and experience.

#### Registrars: vocational trainees (×2-4) or Registrars (×6-8)

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 Joint Faculty of Intensive Care Medicine, as a representative of both The Australian and New Zealand College of Anaesthetists and The Royal Australasian College of Physicians, 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

### Daily Program

<table>
<thead>
<tr>
<th>Time</th>
<th>Activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>08h00</td>
<td>Morning handover</td>
</tr>
<tr>
<td>09h30</td>
<td>Consultant led bedside ward round, followed by HDU administrative review</td>
</tr>
<tr>
<td>11h30</td>
<td>X-ray meeting (10h30 at the weekend)</td>
</tr>
<tr>
<td>16h00</td>
<td>Afternoon ward round and HDU review.</td>
</tr>
<tr>
<td>21h00</td>
<td>Evening hand over and HDU review</td>
</tr>
</tbody>
</table>

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

#### Monday Business Meeting

Alternating

- ICU business Meeting
- ICU Specialists meeting
- Incident Meeting

#### ICU Microbiology meeting

Day: Thursday

Version 3.4
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
- Orientation to the helicopter and safety issues
- Data collection and computer programs
- Transport
- Invasive and non-invasive ventilation
- Organ donation
- Intubation including difficult and failed intubation
- Cardiac Surgery
- Neurological/neurosurgical emergencies

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.

A simulator based teaching program is being developed and should form part of the orientation in the near future.

Patient admission policy

No patient may be accepted into the Intensive Care Unit without the knowledge and consent of the duty ICU specialist. Resuscitation or admission must however not be delayed where the presenting condition is imminently life threatening unless specific advance directives exist.

In general patients should be admitted to the Intensive Care where it is perceived they would benefit in some way as a result. Usually this means patients with actual or potential organ system failure, which appears reversible with the provision of intensive support measures.

Patient Triage:

ICU admission criteria should select patients who are likely to benefit from ICU care. Patients not admitted should fall into two categories, “too well to benefit” and to “sick to benefit”. Defining substantial benefit is difficult, and no pre-admission model exists to predict outcome in a given patient. Rather than listing arbitrary objective parameters, patients should be assigned to a prioritization model to determine appropriateness of admission.

Priority 1: Critically ill patients in need of intensive treatment and monitoring that is not available outside of the ICU. Generally these patients would have no limits placed on their care.

Priority 2: Patients that require intensive monitoring, and may need immediate intervention. No therapeutic limits are generally stipulated for these patients.

Priority 3: Unstable patients that are critically ill but have a reduced likelihood of recovery because of underlying disease or the nature of their acute illness. If these patients are to be treated in ICU, limits on therapeutic efforts may be set (such as not for intubation). Examples include patients with metastatic malignancy complicated by infection.

Priority 4: These patients are generally not appropriate for ICU admission as their disease is terminal or irreversible with imminent death. Included in this group would be those patients not expected to benefit from ICU based on the low risk of the intervention that could not be administered in a non-ICU setting (eg: haemodynamically stable DKA, or an “awake patient following an overdose).

Elective admissions

Where possible, elective surgical admissions should be booked at least 48hrs in advance. A book exists into which the names of prospective patients must be entered, following discussion with the surgical team (or anaesthetist) responsible for that patient and the ICU Consultant on duty for Unit 2 on the day the discussion is initiated. Bed availability must be confirmed by the anaesthetic team prior to commencing the anaesthetic on the morning of surgery.

Management of patients in ICU

Patients in The Waikato Hospital Intensive Care Unit are managed primarily by the ICU staff. Visiting Teams should be discouraged from charting drugs, fluids or other treatment directly. The exception is for elective paediatric ENT patients, where the team is permitted to prescribe medicines to be given in ICU on a ward chart, but only prior to admission to ICU.

The opinion of all Specialists involved in the case is however valued. The Duty ICU Consultant must be kept fully informed of their opinion.
All discharges must be approved by the Duty ICU Specialist.

The parent team must accept care of the patient, this acceptance must be recognised at Specialist level, either through their Registrar, or in some cases to the Specialist directly.

Other teams involved may need to be advised, e.g. the pain team, Renal Service

A careful plan for the immediate discharge period must be discussed with the accepting team, and be clearly documented in the notes including:

- Limitation of treatment where appropriate
- Non-return orders
- Clear medical management plan, including charting of the following for the next 24hrs:
  - Fluids
  - Feeding
  - Analgesia

Documentation to be completed prior to discharge:
- database form
- Discharge sheet/notes

Nurses will not send patients to the ward without first checking with the on duty Registrar

Reference:

Deaths in the ICU

Withdrawal of therapy is a Consultant-only decision.

The duty consultant must be notified as soon as the patient has been examined and certified dead, unless other specific arrangements exist (eg. where death is the expected outcome and the issue of a death certificate issue has been discussed).

The ICU Registrar must ensure:
- A death certificate has been completed if applicable
- The parent team is notified
- Referring colleagues (including GP’s) are notified
- Post-mortem consent has been acquired from the family (if indicated).

The Coroner must be notified as below:
- Every death that appears to have been without known cause, as a result of suicide, or unnatural or violent death.
- Every death in respect of which no doctor has given (or is prepared to give) a death certificate.
- Every death that occurs while the person concerned was undergoing a medical, surgical or dental procedure, or some similar operation or procedure.
- Death that appears to have been a result of any such operation or procedure.
- Death that occurred while the person was affected by an anaesthetic or that appears to have been a result of the administration to the person of an anaesthetic.
- Death of any patient detained in an institution pursuant to an order under section 9 of the Alcoholism and Drug Addiction Act of 1966
- Any death of a child or young person in the care or oversight of an Iwi Social Service, a Cultural Social Service or The Child and Family Support Service.
- Death of any patient committed in a hospital under the Mental Health Act of 1969.
- The death of any inmate within the meaning of the Penal Institutions Act of 1954
- The death of any person in police custody, or in the custody of a security officer.

Where a death is referred to the coroner, no death certificate may be issued by the ICU Registrar.

Reference: 
Taken from The Coroners Act, 2007.
### Clinical duties in the Intensive Care

#### General comments

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.

There are guidelines covering the medical procedures and the administration of most of the drugs used in the ICU. These guidelines are under constant review. The resident staff are required to be familiar with these guidelines and to consult them when required. In addition, any inconsistencies or discrepancies within them should be brought to the attention of the consultant staff.

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, consultations 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  
Obtain hand over information from the referring doctor

##### Secondary survey

Examine patient thoroughly

- Notify Duty Specialist 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

Registrars are responsible for documenting an admission note for all patients and a daily entry into the clinical notes as well as;

- Discharge summary  
- Death certificate  
- Database form collection  

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 senior.

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

#### Daily entry in clinical notes

Ensure each page is dated and labelled with the patients name and hospital number.

- Date / time / name of ICU Staff Specialist conducting the round  
  A: Mental state, GCS, airway  
  B: Ventilation, saturation (or $P_{O_2}$), chest findings  
  C: Pulse / BP / peripheral perfusion / Precordial exam.
Abdominal examination and description of feeding mode.

Peripheries

Assessment or Impression

Plan

Immediately after completion, notes must be filed in the clinical record

**Additional notation must be made in the notes when** 47

- invasive procedures are undertaken
- important management decisions are made
- significant interaction is made with patient family.

**ICU Problem List Formulation** 48

Previously we have attempted to run a computerised problem list database. This has been inconsistently used and is in abeyance. This does not however remove the obligation on the night registrar to have an awareness of active and resolved problems that an individual patient has.

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.

**Daily management issues** 49

The daily handover ward round at 08:00 is attended by the night Registrars, the incoming day staff, the duty consultant and representatives of the nursing staff (CNL 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”.

**Clinical Duties outside the Intensive Care Unit** 50

**Request for insertion of central venous access** 51

Intensive Care Staff may be approached to facilitate central venous access in a patient not residing in intensive care

- 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
- For those familiar with the equipment, the old Sonosite may be borrowed from the Department of Anaesthesia

**Cardiac Arrest Calls** 52

**Indications** 53

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** 54

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

**CPR (Cardio Pulmonary Resuscitation)** 55

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** 56

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 777 call is placed on the emergency pager, and you are required to confirm by calling the operator back on 777.

During the day, one registrar (nominated by Co-ordinating registrar if rostered) will attend trauma calls. Ensure that the ICU nursing co-ordinator for the day are aware of where you are going, and communicate with the ICU nursing team once the patient has been assessed and the likely admission destination known. There is a registrar cellphone available to facilitate ongoing communication, and you should carry this with you on leaving the unit. Remember to keep this phone charged when the opportunity arises.
Trauma team member:
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.

**Trauma call procedure**
The trauma call usually precedes the patient arrival in ED. It is worthwhile to prepare everything that might be needed (ie 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 airways.

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

**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: Chest X-ray, Pelvic X-ray and cervical spine

**Secondary Survey**
Cover in the following order:
- Head and scalp/ maxillofacial
- Cervical Spine and Neck
- Chest
- Abdomen and Pelvis
- Back and Perineum
- Extremities
- Neurology

**Role of the ICU Registrar at the trauma call:**
- 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

Do not leave ICU unattended for more than 30 minutes. Once the patient is stable and sufficient trauma team members present, you may seek permission from the trauma team leader to return to the ICU. If this is not possible within 30 minutes the on-call registrar should be called in so that the ICU is not left without medical staff.

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, particularly if ICU admission is likely.

**Intra-hospital patient transport**
- No patient may be transported from the unit without the direction of the Duty ICU Specialist.

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

Registrars should ask the Senior registrar to accompany them on their first in-hospital transport if (s)he is available. It may not be appropriate for all registrars to undertake prolonged transport, or transport to unfamiliar areas (eg MRI). Always ask the duty ICU specialist 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 written in the notes, failing which they should request formal review and notification to the unit as soon as possible.

Reference:

Version 3.4
Patient Retrieval

Introduction

The Waikato ICU is frequently involved in interhospital 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 interhospital 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 interhospital patient transfer and every attempt must be made to foster this by team members.

Specific Situations

Balloon pump--see transport compendium
Children--see table below and transport compendium. Between 5-10 kg, a Servo 300 is the most suitable ventilator. Beyond 10kg, either Parapac or Pulmonetics are suitable. Transport of critically ill children by our team is a rare event indeed, and yet is required from time to time. Whenever possible, children leaving our ICU bound for an Auckland hospital should be transported by Starship Hospital Retrieval Services or Lifeflight if not destined for PICU at Starship.

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 referred to St John’s ambulance and advised to dial 111.

Rapid response turnout--use whenever extreme emergency--notify all concerned to facilitate rapid transport. If sole registrar on duty, give pager to ED registrar prior to leaving hospital

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.

SUMMARY OF TRANSPORTING SERVICES

<table>
<thead>
<tr>
<th>Referral Source</th>
<th>Trauma In ED</th>
<th>All admitted and non-trauma Patients</th>
<th>All Interhospital Transfers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Taupo, Tokoroa, Taumarunui, Thames</td>
<td>ED A Zero</td>
<td>ICU Transport Team</td>
<td></td>
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<tr>
<td>Te Kuiti</td>
<td></td>
<td></td>
<td>ED A Zero</td>
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<tr>
<td>All other Midland Hospitals (Tauranga, Rotorua, Gisborne, New Plymouth)</td>
<td></td>
<td>ICU Transport Team</td>
<td></td>
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<tr>
<td>Neonates and children &lt;5kg</td>
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<td></td>
<td>Neonatal Transport Team</td>
</tr>
<tr>
<td>Obstetric Referrals from hospital</td>
<td></td>
<td></td>
<td>ICU Transport Team / obstetrics</td>
</tr>
<tr>
<td>Interhospital transfers but not to Waikato</td>
<td></td>
<td></td>
<td>ICU Transport Team</td>
</tr>
<tr>
<td>Roadside Retrievals</td>
<td>ED A Zero</td>
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<td></td>
</tr>
</tbody>
</table>

Version 3.4
Summary of the Transport Process

Initial call to ICU by referring hospital

Clinical details obtained by Registrar/Specialist

Acceptance of transport by ICU Specialist

ICU coordinator notified

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.
Infection Control

Introduction
Patients requiring intensive care are highly susceptible to infection due to immunosuppressive effects of drugs and disease, the use of invasive monitoring techniques and the severity of the underlying illness requiring admission. The use of broad-spectrum antibiotics may predispose to infection with resistant organisms.

Nosocomial infection delays patient discharge from the intensive care unit (ICU) and contributes significantly to morbidity. The prevalence of hospital-acquired (nosocomial) infection in the ICU can be considerably higher than other clinical areas of the hospital.

Significant risk factors for infection include:
- mechanical ventilation
- prolonged length of stay
- trauma or burns
- intravascular catheterisation
- urinary catheterisation

The four most common nosocomial infections seen in ICU are:
- Pneumonia
- urinary tract
- intra-vascular catheter-related bacteraemia
- surgical wound infection

All ICU staff are responsible for ensuring good infection control policies are adhered to, in particular good hand hygiene practice.

Hand Hygiene and Standard Precautions
Hand washing and hand disinfection remain the most important measures in the prevention of cross infection. Hands should be cleaned before and after contact with every patient and after manipulation of the patient environment. Either a 15-second handwash with soap and water, or alternatively the waterless hand gel may be used if hands are not visibly soiled.

A longer handwash with antibacterial soap is required prior to any major invasive procedures such as insertion of central venous catheter.

In addition to hand hygiene standard precautions are used for all patients:
- Wear gloves for all contact with blood and body fluids including dressings and wounds. Gloves must be changed between patients.
- Hands must be decontaminated after the removal of gloves.
- Wear a disposable plastic apron or fluid-resistant gown to protect the skin and clothing for procedures likely to generate splash or cause soiling.
- Wear a mask and eye protection to protect mucous membranes of the eyes, nose and mouth during procedures likely to generate splash or cause soiling.
- Ensure patient-care equipment is cleaned and disinfected appropriately between patient use.
- Staff who generate a sharp product (eg: needle or blade) are responsible for its safe disposal into an approved puncture resistant sharps container.

Isolation and transmission-based precautions
In addition to standard precautions, isolation and appropriate transmission-based precautions are to be used with the following:

Multi-resistant organisms (MRO)
Patients infected or colonised with the following MRO’s require isolation and contact precautions (gloves and gown/apron for direct patient care):
- Methicillin Resistant Staph. Aureus (MRSA)
- Vancomycin Resistant Enterococcus (VRE)
- Multi-resistant gram negative organisms

Meningococcal disease - proven or suspected
Patients require isolation and droplet precautions (surgical mask within 1 metre of the patient) until 24hrs of completed antibiotic treatment.

Miscellaneous
- Burns patients require isolation and contact precautions
- Febrile neutropaenic patients require isolation and contact precautions
- High risk immunosuppressed patients require isolation and contact precautions
- Respiratory syncytial virus require contact precautions

General Measures
The ICU should be kept tidy and uncluttered. Equipment not in use should be stored in a clean area.

Move the development of the unit should be kept to a minimum. This applies equally to colleagues and relatives.

All visitors are to be encouraged to wash their hands before and after visiting the patient.

Staff with communicable diseases should take sick leave.

Further reading:
Please refer to the HW Infection Control Policy and Procedures Manual for further information.

Research in the Intensive Care Unit

Introduction
Research in The Waikato Hospital ICU is strongly encouraged. Applications for research, or ideas which may lead to an application should be discussed with Dr Van Haren.
Personnel

Coordinator of Research: Dr Frank Van Haren
Nursing Research: Mary La Pine
Research Committee: Interested parties and medical/nursing staff involved in trials.

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

Presentation

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.

Assistance with the formal project may be requested from Dr van Haren

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. Further medical and nursing protocols for critical care will be added in the near future.

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.

The ICU has its own web site which is held on a server outside the hospital. This can be found at http://www.anaes-icu-waikato.org.nz. This contains basic information on the unit and has a collection of previous registrar topic presentations that can be accessed. Any suggestions and all electronic presentations should be forwarded to John Torrance.

Consent in the Intensive Care Setting

Introduction

A competent patient may give or withhold consent for any medical treatment (Code of Health and Disability Consumer’s Rights).

Unfortunately, patients in ICU often cannot have their competency established with certainty. 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 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.

Consent by relatives

Relatives or friends cannot give or withhold consent for the performance of a medical treatment.
New Zealand statute does however mandate that the treating doctor takes the family views into account in deciding whether to perform a particular treatment.

**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 many 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 here.

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

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

**Reference:**

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

**Medico-Legal Assistance**

In those situations where you may require medico-legal advice, a legal advisor may be contacted through the switchboard.

**Hospital Emergencies**

There is a Waikato Hospital Emergency Response Wall Chart in both ICU units which will guide you in any of the following situations:

- Mass casualty
- Communications or utility failure
- Cardiac Arrest
- Earthquake
- Fire (or smoke smell)
- Hazardous substance spill
- Personal safety threat
- Threat from telephone, letter or suspicious object
- Bomb or arson
- Radiation spill

Dialling “777” and thereby contacting the switchboard will in most circumstances allow you to initiate an emergency response that is appropriate to the threat.

**Fire and building emergencies**

Attend formal fire training sessions

Become familiar with location of fire exits

Assess medical condition of persons in an emergency area, and the likely effects of evacuation on them.

Advise the Floor warden of any special requirements

Follow instructions of trained accredited staff.
Clinical Procedures

Introduction

It is inevitable that during your stay in the Intensive Care Unit you will be exposed to a number of procedures with which you are not familiar. All staff are encouraged to become proficient with routine procedures:

ICU Procedures

- Endotracheal intubation
- Peripheral venous catheterisation
- Central venous catheterisation
- Arterial cannulation / PiCCO insertion
- Pulmonary artery catheterisation
- Urinary catheterisation
- Lumbar puncture
- Intercostal drain insertion or pleurocentesis
- Naso-gastric / jejunal tube insertion

Patient consent should be obtained as outlined elsewhere in these guidelines.

No member of staff is permitted to attempt a procedure without adequate training. No matter how experienced you are, repeated unsuccessful vascular punctures are unacceptable and a more experienced member of staff should be asked to help.

All procedures must be annotated in the case notes, including the indication / complications for the procedures.

Restricted procedures

Specialised procedures are generally performed by the Senior Registrar or Duty Specialist. They may not be attempted without prior discussion with the Duty Specialist.

- Percutaneous tracheostomy
- Fibreoptic bronchoscopy
- Transvenous pacing
- Pericardiocentesis
- Oesophageal tamponade tube insertion

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

Arterial Cannulae

Indications

- Invasive measurement of systemic blood pressure
- Multiple blood gas sampling and laboratory analysis

Site and catheter choice

1st choice: Radial artery

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).

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 (eg. sutured)
- The insertion site and all connectors must be visible through the applied dressing.

Complications

- Infection
- Thrombosis
- Digital Ischaemia
- Vessel trauma and fistula formation.
Central Venous Cannulae

Training

Central venous catheterisation may not be attempted by any member of staff without adequate training or supervision.

Indications

Reliable IVI 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 conditions of strict “asepsis”.
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 ICU specialist 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.

Subclavian

Avoid in situations where pneumothorax would be fatal. (i.e. severe respiratory failure, lung hyperinflation).

**Avoid in patients therapeutically anticoagulated or coagulopathic**

- Platelet count < 50 000
- INR > 2.0
- APTT > 50 sec

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 Specialist.

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

Internal Jugular

Avoid in situations where pneumothorax would be fatal. (i.e. severe respiratory failure, lung hyperinflation).

**Avoid in patients therapeutically anticoagulated or coagulopathic**

- Platelet count < 50 000
- INR > 2.0
- APTT > 50 sec

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 Specialist.

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

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).

Reference:

Line management

Routine line replacement is not required.

Lines should be removed as soon as:
- They are not required any longer.
- The patient has evidence of unexplained systemic infection (pyrexia, ↑WCC)
- Insertion site infection or positive blood culture with likely organism (S epidermidis)

Guide-wire exchanges should not be performed unless discussed with the Duty Specialist.

**Antibiotic coated CV lines should be used when access required beyond 2-3 days.**

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)

Pulmonary artery catheterisation

The PA Catheter is not a resuscitation tool and should only be inserted in a controlled environment after discussion with the Duty Specialist. Dwindling use of the PA catheter has resulted in a loss of familiarity with its use. Junior medical staff and nursing staff not familiar with this instrument should not manipulate / advance / inflate the PA catheter balloon.

Indications

Haemodynamic measurement: (cardiac output, stroke volume, systemic vascular resistance)
Measurement of right heart pressures (pulmonary hypertension, pulmonary embolus)
Estimation of preload to the left ventricle-controversial.

Insertion

PA Catheter insertion is technically difficult and requires a working knowledge of right heart pressures and waveforms. See appendix on pulmonary artery catheterisation

Monitoring PA trace

An adequate tracing should be visible on the monitor at all times. A damped tracing may represent a wedged catheter, clot at the catheter tip or inappropriate equipment set-up (wrong monitor calibration, faulty pressure transducer).

- Flush the distal lumen generously (using closed mechanism)
- Withdraw catheter until a trace is present.

NB Never withdraw the catheter with an inflated balloon.

Measurement of pressures

Pressure should be referenced to the mid-axillary line
The true wedge pressure is measured at end-expiration
PEEP may influence wedge pressures, however this is not a factor at PEEP < 10 mmHg, and patients should not be disconnected from the ventilator to measure PAC pressures.

Measurement of haemodynamics

Cardiac output measurement should only be attempted by staff familiar with the use of PA Catheters.

10 ml 5% dextrose at room temperature is rapidly injected into the appropriate lumen.
This is usually repeated three times, with results varying > 10% from average discarded.

Cardiac Pacing

Introduction

Most commonly, temporary cardiac pacing is encountered post cardiac surgery with epicardial leads. Transvenous pacing is most often accomplished using a modified pulmonary artery catheter or by a cardiologist using a suitable lead.

Indications

In circumstances where “medical” pacing with a chronodoino 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.

Flotation Catheter insertion

Flotation placed transvenous pacing wires should be placed using ECG guidance

Use aseptic technique and local anaesthesia where appropriate

- Insert pacing wire using a peel away sheath, or if necessary a PA Catheter introducer (although this may leak).
- Connect pulse generator but do not activate
- Attach V5 lead of an ECG to the distal electrode of the catheter and advance noting QRS wave form change as the catheter advances to the RV. Advance the catheter 2 cm, deflate the bulb and advance a further 1-2 cm.
- Set output and sense of pulse generator to minimum value, and rate 20 bpm > than the patients own.
- Turn generator on and gradually increase output while observing the ECG for capture

If there is no capture, or a high output is required then:

- Place generator on demand mode.
- Turn output down, advance or reposition the wire slightly
- Attempt re-capture by increasing output. An ideal capture setting would be approx 2 mA, with a routine setting placed 2-3 mA higher than capture threshold.
- Secure wire in place
A control CXR must be performed post insertion.

All staff should familiarise themselves with the code of pacing and the pacing box.

References:

Intra-aortic balloon counterpulsation (IABP)

Introduction
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 2 area.

Usually IABP’s are sited by the Cardiothoracic Team or a Cardiologist. If they are to be inserted by ICU staff, it is only to be performed by consultant staff or advanced ICU vocational trainees.

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

Procedure preparation
- IABP insertion should only be performed by staff familiar with the equipment and personnel requirements.
- Strict aseptic technique should be maintained
- Check IABP function prior to insertion
- Confirm that the helium cylinder volume is adequate
- Reference the arterial pressure manifold to the mid axillary line and calibrated.
- Connect a dedicated 5 lead ECG to the IABP
- Machine in standby mode at the following settings:
  - ECG sense trigger
  - 1:2 ratio augmentation
  - max. augmentation selected
  - inflate and deflate time at zero.

Insertion procedure
- Use local anaesthesia in awake patients
- An assistant who is scrubbed should be available
- Select a balloon size: 34 ml balloon for patients under 165cm tall, 40 ml balloon if taller.
- Cannulation the femoral artery using the Seldinger technique
- Insert IABP using sheathless technique (catheter over wire) or large bore cannula supplied.
- Insert to point approximating T4 (ie distal to origin of left subclavian artery) as an anatomical marker (2nd black marker on the IABP itself), with a subsequent control X-ray once procedure complete
- Aspirate blood from arterial port to confirm position
- Connect to pressure transducer and pump, then turn on.

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 ration 1:1 or 1:2

Maintenance
Some surgeons wish the patient to be heparinised while the IABP is in situ
The surgeon’s preference should be confirmed, and clearly documented in the patients notes. Control CXR: the tip of the IABP is radiologically opaque and should be sited at the level of T4 (= carina on centred film)

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 ICU Specialist and Cardiothoracic Surgeon / Cardiologist agree.

Generally the augmentation rate is sequentially decreased to 1:4 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.**

- If open arteriotomy has been used, surgical closure will be necessary
- Disconnect IABP tubing but do not manually aspirate the balloon before withdrawal.
- 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.
- Thrombocytopaenia
- Balloon rupture with gas embolism

**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
- 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 ICU Specialist.

**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 ICU blood gas analyser (pH < 7.20 = empyema, 7.20-7.25 = equivocal)

**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, 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

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 technique 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.

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 specialist 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 (eg. in the setting of traumatic brain injury)
Patient transportation
To protect an airway
Patients at risk of aspiration
Altered conscious state
Tracheal toilet

Techniques
Orotracheal intubation is the rule.
Blind nasal awake intubation, or fibreoptic awake intubation, may be indicated in selected patients with cervical spine injury, limited mouth opening or oro-facial surgery / trauma. These techniques should only be undertaken by staff with current experience of these techniques, and only after discussion with the duty ICU specialist.

Standard endotracheal tube choice
All patients in the Waikato Hospital Intensive Care Unit should be intubated with a low pressure high volume PVC tube (eg Portex blue line oral nasal tube)

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 hrs, and would not be exposed to significant risk during the ETT change.

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 traction 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 its removal.

Preparation
- Secure adequate IVI access
- Check all equipment prior to intubation:
  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 × ETT: estimated patient size and one smaller size. (Female = 7-8 mm, Male = 8-9 mm)
  A selection of laryngeal masks
Emergency cricothyrotomy 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

### Drugs

**Induction agent**

- eg. 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

### Miscellaneous

- Atropine 0.6-1.2 mg
- Adrenaline 10 ml of 1:10 000 solution.
- Phenylephrine 0.5 mg/ml (usually in 10 ml)

### 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-/orogastric tube if not already present.

A follow-up CAR should be performed as soon as convenient.

### 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

- Actively pursue opportunities to deliver supplemental oxygen throughout the process of difficult airway management

- 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 spont vent.

- Develop primary and alternative strategies

### Awake Intubation

Awake intubation in the ICU setting occurs less commonly than in the operating theatre. An awake intubation in the ICU should be directed by the duty ICU specialist, 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

Approach to failed intubation
Maintenance of endotracheal tubes

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 overinflation 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 every 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

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

Intubation attempts after induction of General anaesthesia

Success

Initial attempts unsuccessful, consider
- Calling for help
- Return to spontaneous ventilation
- Awakening the patient

Face-mask ventilation adequate

Non-emergency pathway
- Ventilation adequate
- Intubation unsuccessful

Alternative approaches to intubation:
- Different laryngoscope blade
- LMA as intubating conduit
- Fibre-optic intubation
- Stylette of ETT exchanger
- Light wand
- Blind oral or nasal

Face-mask ventilation not adequate

LMA adequate

LMA Not adequate or feasible

LMA adequate

Successful ventilation

Invasive airway access

Awaken patient

Emergency Pathway
- Ventilation not adequate
- Intubation unsuccessful

Emergency non-invasive vent:
- Combitube
- Transtracheal jet vent.
- Rigid bronchoscope

Fail after multiple attempts

Emergency invasive airway access:

Call for help

Version 3.4
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 deg 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.

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 specialist.

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:

Fibre-optic Bronchoscopy

Policy

Only to be performed by adequately trained staff, after authorisation by the duty ICU consultant.

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

The recommended procedure for urgent surgical airway access (not percutaneous tracheostomy)

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

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. In the event of these not being available, the simplest technique is described below.

- # 15 scalpel and handle
- Size 6.0 cuffed ETT
- Oxygen delivery circuit and ventilation device (eg. Laerdal bag)

Procedure

- Palpate cricothyroid membrane
- Perform 2cm horizontal incision through skin and cricothyroid membrane
- Insert blade handle into wound and turn vertically to enlarge wound (do not use blade or sharp instrument such as a pair of scissors)
- Insert ETT into trachea
- 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.
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 ICU consultant.

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:
- Prolonged intubation (> 10 days) or anticipation thereof.
- Prolonged upper airway obstruction
- Laryngeal pathology
- Subglottic stenosis

Airway protection
- Delayed return of glottic reflexes
- Tracheal toilet / ineffective cough mechanism

Relative Contraindications

Elevated or unstable measured intra-cranial pressure
Coagulopathic state
Platelets < 100 000 (or abnormal function eg. following aspirin)
APTT > 40 sec
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.

Equipment

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.

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 deg 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 trachy cuff, lubricate and insert dilator into trachy 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.

**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**
- Position the patient Rt side down to at least 45°, Remove any gastric tube.
- Use a Corpak 10 Fr tube 140 cm long with stylet.
- Close tube sideport and attach 3 way tap to flow through stylet hub.
- Measure out Xiphisternum to ear plus ear to nose distance from the tip of the tube.
- Insert tube to this distance, confirm intragastric placement by auscultation during air injection.
- Administer 200 mg of erythromycin or 10-20 mg of metoclopramide as slow IVI “push”.
- Inflate the stomach with 500 ml of air while the prokinetic is being administered.
- Gently advance the tube. You should feel a steady resistance. If there is increasing then sudden loss of resistance this means you have thrown the tube into a loop in the stomach. Withdraw the tube until resistance is felt and start slowly advancing the tube again. Insert the tube to the 120-125 cm mark if possible.
- Try to aspirate fluid from the tube. If fluid can be aspirated check the pH with a urine dipstick. An alkaline pH suggests duodenal or further insertion.
- Position the patient flat again and order an upper abdomen-lower chest x-ray.
- Just before x-ray exposure inject 10 mls of contrast (Gastroview) down the tube.

**Check tube position on x-ray.**

If the tube is in a satisfactory position a gastric tube can now be inserted to decompress the stomach if this is desired.

Remember to leave the stylet in any fine bore tube during manipulation of the nasogastric tube. Keep the stylet in a plastic bag at the head of the bed so it can be reinserted into the oesophageal and gastric segment of the fine bore tube during procedures such as NG tube or ET tube removal to stiffen the fine tube and prevent its accidental partial removal.

**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 (eg: 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.

Reference


Intra-Cranial Pressure Monitoring

Introduction

For expanded discussion on the role of ICP monitoring in the management of head injury and raised intra-cranial pressure refer Neurosurgical guidelines (Chapter 6, section 6.5).

Insertion of an ICP monitor is generally performed by neurosurgical staff.

Procedure

- Confirm valid indications for monitoring
- With the head stabilised in a neutral position, select insertion site: usually mid pupillary line (commonly right side) and 3cm anterior to coronal suture.
- Shave the scalp, infiltrate with local anaesthetic (5 – 10 ml lignocaine with adrenaline).
- Incise scalp sufficient to insert mini retractor.
- Use drill to create a burr hole, using spacer guard to guide depth.
- Remove retractor and using a Touhy needle burrow 2 – 3 cm medially under the galea.
- Thread the micro-sensor from the tip of the Touhy towards the burr hole, remove the Touhy needle, leaving the sensor under the skin.
- Insert the retractor again.
- Puncture the dura
- The microsensor should have been zeroed before procedure, or do it now!
- Kink the micro-sensor at the require depth (generally 2 – 2.5 cm) so that the microsensor tip is within the cranial vault, and preferably within brain parenchyma.
- Insert sensor so that kink is flush with outer skull table. Retract excess cable subgaleally.
- Bone wax may be useful in sealing the burr hole if desired.
- Secure sensor to scalp. Additional strain relief can be provided by coiling excess line and suturing to scalp.

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’ or100’ 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.

Jugular Bulb Oximetry

Introduction

Whilst not proven to affect outcome following severe head injury, jugular bulb oximetry may be useful in the monitoring and manipulation of cerebral perfusion. See Chapter 6, Neurosurgical guidelines for further information.

Indications

Use is controversial.
Closed head injury, with:
- GCS < 8
- Injury less than 24 hrs old (relative)
- ICP monitoring established

Procedure

- Prepare using aseptic technique as for central line insertion.
- Connect connect 3 way tap to catheter and flush with heparinised saline.
- Cannulate internal jugular vein on side of maximal injury (or on left in case of diffuse injury).
- Insert guidewire in retrograde fashion (i.e. towards cranial vault).
- Using Seldinger technique insert introducer and “peel away” sheath.
- Remove wire and introducer.
- Insert catheter until resistance is felt
- Aspirate to confirm vascular placement and then flush catheter.
- Secure with a tegaderm dressing without coiling catheter.
- Confirm catheter position with a C-spine X-ray (tip opposite C1).
- Continuous flush with hep saline at 3ml/hr.

**Calibration**

Continuous oximetric measurement of jugular bulb blood is not currently available in our ICU. Intermittent sampling may still be used to measure saturation and lactate extraction.

**Complications**

As for central venous cannulation.

Thrombosis: approximately 40% association within 3 days.

Poor reliability: readings from contralateral jugular vein may differ by as much as 15%.
Most patients admitted to the ICU will have had medications prescribed for concurrent or pre-morbid conditions. A new chart for each drug must be started on each patient’s arrival in ICU. Re-charting of all drugs implies an active review of the appropriateness of drug administration and dosage, in changing clinical conditions.

It is important that drug charts are accurate, legal and legible. Use of drug trade names is not acceptable practice. Similarly only drugs which are approved by the ICU medical staff may be given to ICU patients. For this reason only ICU medical staff may write (prescribe) in the patient’s drug chart while the patient is in the ICU.

Charting of drugs by outside teams must be discouraged.

On discharge to the ward it is the responsibility of the discharging medical person to review patient drug and fluid orders.

Prescription practice

In general the following principles should be considered when prescribing any drugs for ICU patients.

A drug should only be instituted where the potential benefit is well described, or the risk for adverse effects low when benefit is unproven.
Unit protocols and guidelines should be used where these exist for a given drug.
ICU patients often have vastly altered drug pharmacokinetics (what the body does to the drug) and pharmacodynamics (what the drug does to the body).
The role of therapeutic drug monitoring should be understood
Good drug prescribing practice is mandatory, including: Legible hand writing, use of generic (not trade) names for drugs, clear delineation of dose, frequency and duration of treatment.

Prescribing in Pregnancy

Prescribing in pregnancy requires knowledge of teratogenicity and suitability for the fetus.
A website which can provide this information is www.tga.gov.au/docs/html/medpreg.htm

Cardiovascular Drugs

Inotropes

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

This simple concept is in practice very difficult to perform accurately. In the ICU there are a number of ways to assess intravascular volume status although each has shortcomings.

Clinical assessment of fluid status including JVP
Variation of arterial waveform characteristics with mechanical ventilation.
Measurement of dynamic and static measurements CVP.
Blood pressure response to passive leg elevation
Right heart catheterisation.
Calculation of intrathoracic blood volume and extrapolation to extravascular lung water using PiCCO.
Echocardiographic techniques.

Except in very clear-cut cases (ie gross fluid overload or cardiac failure), in a situation where there is doubt as to the patients fluid status a trial of fluid administration should be considered.Blood pressure response to passive leg elevation may be a useful predictor of fluid loading without the risk of fluid overload.

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.
Please consult appendix on haemodynamic principles.

Catecholamines
Clinical Applications 234
Short notes on using common agents 235

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

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

Adrenaline is a useful α / β-agonist, however it does have significant β2-effect which may result in unwanted metabolic effects (hyperglycaemia, lactic acidosis).

Noradrenaline is generally held to have a predominant α-effect and is therefore useful as an inotrope-vasopressor, particularly in septic shock.

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

Adrenaline and noradrenaline, when mixed in ratio’s of 3 mg : 50 ml 5% dextrose will administer a dose in ml / hour equivalent to a dose of micrograms per min. Individualising patient doses per kg is not a useful practice, but is traditionally used to quantify dopamine administration.

Adrenaline and noradrenaline infusions should be started at 3-5 µg / 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 236

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 237

These drugs usually require a loading dose on commencement which may predispose to additional hypotension by virtue of vasodilatation.

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.

Isotropes commonly used in ICU 238

<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, Severe sepsis syndrome / shock, Cardiogenic shock, Acute severe asthma (adjunct), Anaphylaxis (correct hypovolaemia!!), Medical Pacing (1st line drug)</td>
</tr>
<tr>
<td>Noradrenaline</td>
<td>40 µg / 50 ml D5W ratio (µg / min)</td>
<td>Conditions where mixed α / β-effect is required with a predominant α-effect, ie. septic shock</td>
</tr>
<tr>
<td>Dopamine</td>
<td>200 µg / 50 ml D5W (µg / kg / min in 70kg person)</td>
<td>No advantage over adrenaline or noradrenaline, May induce more tachycardia than adrenaline (through stimulation of D-receptors), Not reno-protective, but may have direct diuretic effect.</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>250 µg / 50 ml D5W (µg / min)</td>
<td>Pure β adrenergic agent used in low cardiac output / high vascular resistance states. 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. Run at 0.05 to 0.2 µg/kg/min until bag empty (approx 24 hrs).</td>
<td>Inotrope, predominantly by Ca2+ sensitisation of mycardium. 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: 12.5 - 50µg / kg over 20 min Infusion: 10 µg / 50 ml D5W (200µg / ml) in fuse at 0.375 – 0.75 µg / kg / min. (8-15 µl / hr in 70 kg pt)</td>
<td>Cardiogenic shock due to diastolic failure, Pulmonary hypertension following cardiac valve replacement, Rescue following catecholamine induced down regulation of receptors in patients requiring ongoing chronio-inotropy. These agents may accumulate in renal failure</td>
</tr>
</tbody>
</table>

Vasopressor agents 239

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)

General principles 240

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.

Complications
Rebound hypertension
Vagal response (with possible bradycardia)
Tachyphylaxis

Vasopressor agents commonly encountered in ICU

<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 anesthetics for short periods of predictable hypotension associated with epidural or spinal anaesthesia.</td>
</tr>
<tr>
<td>Ephedrine</td>
<td>IVI:30 mg / 10 ml D5W titrated to effect IVI (1-3 mg). Oral: 25-50 mg 8hrly po.</td>
<td>No longer commonly used outside operating theatres.</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</td>
</tr>
</tbody>
</table>

Steroid use in patients requiring vasopressors:
Following the Corticus study, it is generally accepted that steroids have a limited role in septic shock.

Anti-hypertensive Agents

General Principles
Elevated blood pressure should be viewed in the context of each patient, and should include an appraisal of pre-morbid blood pressure.

Acute 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 α2-agonist (clonidine or dexmedetomidine) may be useful.

Hypertension in the setting of intra-cranial pathology may be self limiting. 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.

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 ICU Specialist.
- 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 ΔP / Δt in patients with aortic dissection

Complications
Hypotension
Tachyphylaxis
Cyanide Toxicity-Sodium Nitroprusside
Pulmonary vasodilatation causing increased pulmonary shunting and hypoxia
### Vasodilators and Other Anti-hypertensive Agents in the ICU

<table>
<thead>
<tr>
<th>Agent</th>
<th>Dose</th>
<th>Uses</th>
</tr>
</thead>
<tbody>
<tr>
<td>GTN (glyceryl trinitrate)</td>
<td>50 mg in 50 ml. Use non-PVC giving set. Range 1-20 ml / hr (0-20 mg / hr)</td>
<td>Venodilation &gt; arterial. Unpredictable hypotensive effects. Useful in cardiac ischaemia (no proven effect on outcome) Tachyphylaxis when used &gt; 24hrs</td>
</tr>
<tr>
<td>Sodium Nitroprusside</td>
<td>50 mg / 50 ml D5W. Range 0- 20 ml / hr IVI</td>
<td>Rapid control hypertension by direct arteriolar action. Cyanide toxicity may be seen at total dose &gt; 1.5 mg/kg/24hrs (tachyphylaxis and metabolic acidosis)</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>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 D5W, 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>Atenolol</td>
<td>25-100 mg bd po</td>
<td>Useful in blunting sympathetic overdrive (Head injury, agitation, thyroid crisis) Renally cleared, reduce dose in renal failure. Use all β-blockers with caution in patients with asthma or peripheral vascular disease Avoid concomitant use with Calcium channel blocking agents</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 Infusion: 50 – 200 mg / kg / min. (2.5 g in 50 ml D5W = 50 mg / ml)</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>Clonidine</td>
<td>Bolus: 150µg diluted in 10 ml NS, administer 1-5 ml IVI pm</td>
<td>Centrally acting α2-agonist. Useful in peri-operative centrally mediated hypertension, with anti-agitation benefits.</td>
</tr>
<tr>
<td>Hydralazine</td>
<td>10-20 mg as a bolus IVI 20-40 mg 6-8 hrly Very rarely administered as an infusion 1.5-5.0 µg / kg / min in cardiac surgery. (not recommended by manufacturer)</td>
<td>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 &gt; 200 mg / day.</td>
</tr>
<tr>
<td>Metyldopa</td>
<td>250 mg - 2 g / day bd po</td>
<td>Traditional agent still useful in gestational proteinuric hypertension</td>
</tr>
<tr>
<td>MgSO4</td>
<td>Bolus: 10-20 mmol IVI slowly (may cause sensation of flushing, and hypotension) Infusion: 40 mmol in 500 ml of D5W 6 hrly IVI</td>
<td>Gestational proteinuric hypertensive states (“eclampsia”) Torresades 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.</td>
</tr>
</tbody>
</table>

### Antiarrhythmic Drugs in Critical Care

#### General Principles of Treatment

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

- Hypoxemia
- High, occasionally low, pH
- Hypokalemia, hypomagnesemia
- Pre-existing drug effects or toxicity (including bronchodilators and inotropes)
- Presence of right heart lines (including pacemakers)
- Myocardial or coronary compromise, especially pulmonary edema

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 β-/α- agonist, adrenaline has declined in popularity mainly due to its metabolic side effects mediated by β-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; later stages of AV block are more likely to respond to aminophylline. 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.

### Isoproterenol

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:

- 3° or advanced 2° 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.

### Aminophylline

It may be indicated for symptomatic 2° or 3° AV block in later stages of inferior myocardial infarction, as mentioned above.

### Supraventricular Arrhythmias

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

### Adenosine

Endogenous adenosine production is enhanced by ischaemia 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, dyspnoea and cough. Sinus bradycardia or arrest and ventricular arrhythmias are frequent, but almost never actionable. 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 VT 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 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:

### Verapamil

Verapamil inhibits the slow inward Calcium channel and blocks the slow antegrade pathway in AVNRT; it stops AVRT by AV block. Its effect on SVT should be apparent within three minutes. If a 1-5 mg bolus (tailored to age and co-morbidity) is ineffective, a bigger (5-10 mg) dose 10 minutes later is recommended. Before adenosine, verapamil was the drug of choice in treatment of SVT.

It can be used to slow the ventricular response in AF, but this is rarely done now. It remains quite useful for the same purpose in multifocal atrial tachycardias (where atrial rate may also be slowed), usually as an infusion.

Hypotension may be a problem. It is obviated by preceding the bolus by 5 mmol of Calcium; there is no loss of anti-arrhythmic activity. Verapamil should be given slowly in patients with known myocardial disease.

Other side effects are similar to those of adenosine-sinus pauses, bradycardia and occasionally AF. Unlike adenosine, it should never be given to diagnose a broad-complex tachycardia: cardiogenic shock or cardiac arrest may result.
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 great moment in the ICU.

Flecainide

It slows the phase zero of the action potential, interfering with the fast inward Sodium current; it depresses the diastolic repolarisation. The action potential is not prolonged-hence its IC classification. PR, QRS and QT are prolonged.

It is a potent “PVC killer” but has suffered greatly in the CAST trial: its pro-arrhythmic propensity precludes its long-term use in post-MI patients. The bad reputation has spread. This has little to do with its benefits in the ICU, where it remains the drug of choice for both SVT and AF & flutter in patients with WPW syndrome. It slows conduction in the atrium. It is also a good drug for pharmacological cardioversion of AF and flutter in patients with normal conduction. Its pro-arrhythmic effects are of less moment in continuously monitored ICU patients.

Preferred dose in the ICU = 150 mg in 5% dextrose IVI over 30 minutes.

One important side effect is the elevation of the pacing threshold; the patient may become un-paceable. This limits its use in the pacemaker-dependent post-CABG patients. Flecainide is a “consultant only” restricted drug in New Zealand.

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 has for a long time been the drug of choice for the emergency treatment of ventricular arrhythmias.

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% D.

Amiodarone

The drug 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% D 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 IHDI.

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.

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

Procainamide

Its effects are very similar to those of quinidine; it is a Class 1A Sodium channel blocker, prolonging the QRS complex (25% “effect”, 50% “toxicity”) and the QT interval. It is less vagolytic than quinidine and has little, if any, ganglion-blocking properties.

Dose:

loading dose: 1 g in 50 cc 5% D (20 mg / cc) at 10-20 mg / min.

Alternatively, 100 mg boluses over 1 minute can be repeated at 5 min intervals, watching the BP.

It is quite effective drug for VT and probably most effective of all drugs to slow down the conduction by an anomalous pathway in WPW, AF or flutter. It is rarely used; part of the problem is the relatively long time (20 min) to load an effective dose.

Phenytoin

Beside KCl, 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, like procainamide, as 100 mg boluses every 5 min; the usual antiarrhythmic dose is approximately 700 mg (beyond 1000 mg it is unlikely to succeed).
<table>
<thead>
<tr>
<th><strong>COMMONLY ENCOUNTERED ANTIARRHYTHMIC AGENTS</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Agent</strong></td>
</tr>
<tr>
<td>----------</td>
</tr>
<tr>
<td>Amiodarone</td>
</tr>
<tr>
<td>Magnesium</td>
</tr>
<tr>
<td>Adenosine</td>
</tr>
<tr>
<td>Verapamil</td>
</tr>
<tr>
<td>Digoxin</td>
</tr>
<tr>
<td><strong>Respiratory Drugs</strong></td>
</tr>
<tr>
<td><strong>Nebulised bronchodilators</strong></td>
</tr>
<tr>
<td><strong>General principles</strong></td>
</tr>
<tr>
<td>These agents are used in the treatment of bronchospasm in Intensive Care (including acute severe asthma). These agents do not necessarily need to be delivered by nebuliser, but can be administered as a metered dose inhaler into the appropriate port on the inspiratory limb of the ventilator circuit. Once these agents have been commenced they should be reviewed daily or more often, as is the case with all prescribed drugs. This is usually assessed by improvements in audible wheeze, lung compliance, respiratory rate and blood gases.</td>
</tr>
<tr>
<td><strong>Indications</strong></td>
</tr>
<tr>
<td>Pre-existing obstructive airways disease where reversibility is suspected. Acute severe asthma. Acute exacerbation of obstructive airways disease.</td>
</tr>
<tr>
<td><strong>Parenteral Therapy in treatment of reversible obstructive airways disease</strong></td>
</tr>
<tr>
<td><strong>Indications</strong></td>
</tr>
<tr>
<td>Adjunctive therapy for acute severe asthma in patients not responding to nebulised agents.</td>
</tr>
<tr>
<td><strong>Complications</strong></td>
</tr>
<tr>
<td>Hypokalaemia, metabolic alkalosis. Arrhythmias (theophylline) Intercurrent infection Polynuropathy Lactic acidosis (β2-stimulants)</td>
</tr>
</tbody>
</table>

<p>| <strong>DRUGS COMMONLY USED TO TREAT ASTHMA AND AIRWAY OBSTRUCTION</strong> |
|-------------------------|-----------------|-----------------|
| <strong>Drug</strong> | <strong>Infusion / dose</strong> | <strong>Clinical uses</strong> |
| Salbutamol | Nebulised in N Saline (5 mg:1ml) Continuously, 2 or 4 hourly | First line bronchodilator Acute or chronic airway obstruction Asthma Adjunct in severe hyperkalaemia |
| Ipratroprium | Nebulised (500ug) alone or in addition to Salbutamol Administered qid or rarely q6h | Chronic airflow obstruction (CAO) Bronchorrhoea |</p>
<table>
<thead>
<tr>
<th>Drug</th>
<th>Infusion / Dose</th>
<th>Clinical uses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Budesonide (nebulised steroid)</td>
<td>Nebulised 1 mg bd</td>
<td>Steroid dependent obstructive airways disease</td>
</tr>
<tr>
<td>Adrenaline</td>
<td>1 mg / 50 ml 5% dextrose (= 20 µg / ml)</td>
<td>Acute severe asthma&lt;br&gt;Rapid onset and offset of action&lt;br&gt;Titrated until demonstrate pressor response (may require up to 100 µg / min)</td>
</tr>
<tr>
<td>Salbutamol IV infusion</td>
<td>3-20 µg / kg / hr in 5% dextrose (usual adult dose 0.5 – 1.0 mg / hr)</td>
<td>Acute severe asthma&lt;br&gt;Longer duration of action</td>
</tr>
<tr>
<td>Hydrocortisone</td>
<td>100 mg IV 4-8 hourly</td>
<td>All patients with acute severe asthma; wean off over 48-72 hrs once broken 2. Steroid dependent COPD</td>
</tr>
<tr>
<td>Theophylline Intra-venous infusion</td>
<td>1000 mg / 100 ml 5% dextrose&lt;br&gt;Loading: 5-7 mg/kg over 30 minutes to an hour. <em>Omit if patient normally on theophylline.</em>&lt;br&gt;Maintenance: infusion 2-4 ml/hr (0.6-0.9 mg/kg/hour or max 1 gm / day)&lt;br&gt;Levels: 55-110 µmol/L</td>
<td>No longer 1st line drug.&lt;br&gt;May improve respiratory drive in COPD&lt;br&gt;Narrow therapeutic index: proarrhythmic&lt;br&gt;Levels no longer able to be measured at Waikato Hospital.</td>
</tr>
</tbody>
</table>

### Sedation

#### Introduction
Histologically patients in intensive care were heavily sedated and often paralysed. As modes of ventilation have evolved, it has become desirable for patients to be more neurologically accessible. Anxiolysis and analgesia, not sedation, are the primary goals in the management of the critically ill patient.

Pain and anxiety are associated with significant adverse physiological responses:
- Sympathetic activity
- Intracranial hypertension
- Gastritis and gastric erosion
- Excessively catabolic state.

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.

#### Approach to 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.

#### Approach to Sedation
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 dysharmony.
- 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 8hrly (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)

**All sedation in patients with a sedation score of – 2 or – 3 should be stopped at 07h30 everyday to allow neurological assessment unless doing so would expose the patient to excessive risk as determined by the Medical Staff (eg: sedation for ICP control)**

In the Waikato Hospital ICU, propofol ± bolus opioid or infusion of short acting opioid, is the first line sedative agent where an infusion is required.
<table>
<thead>
<tr>
<th>Drug</th>
<th>Initial Dose</th>
<th>Administration</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Propofol</td>
<td>10 mg/ml (neat solution), Start at 3 ml/hr and titrate against effect Max dose &lt; 5mg/kg/hr</td>
<td>Short term sedation of intubated and ventilated patients. Anaeesthesia for minor procedures where prompt return of consciousness is required (eg tracheostomy, CVC) Potential myocardial depressant and vasodilator No analgesic effect.</td>
<td></td>
</tr>
<tr>
<td>Midazolam</td>
<td>Bolus: 1-5 mg IVI Infusion: Midazolam 60 mg in 60 ml 0.9% saline: 1-15 ml/hr</td>
<td>First dose daily or more often Effects prolonged in renal failure Anticonvulsant</td>
<td></td>
</tr>
<tr>
<td>Morphine</td>
<td>Bolus: 0.5-5 mg IVI prn, or PCA. Infusion: Morphine 60 mg in 60 ml 0.9% saline at 1-10 mg/hr</td>
<td>First line analgesic</td>
<td></td>
</tr>
<tr>
<td>Fentanyl</td>
<td>Bolus 12.5-200 μg IVI Infusion: 500 μg in 50 ml At 10 – 200 μg / hr (1-20ml)</td>
<td>Potentially potent analgesic for ventilated patients Used for procedures in ventilated patients Useful in patients with renal failure where the active metabolites of Morphine may pose a sedative risk</td>
<td></td>
</tr>
<tr>
<td>Diazepam</td>
<td>2.5-20 mg IVI / po prn</td>
<td>Anticonvulsant</td>
<td></td>
</tr>
<tr>
<td>Dexamethasone</td>
<td>Loading dose: 1 μ g / kg over 10 min. Infusion: 0.2 – 0.7 μ g / kg / hr (make up 4 μ g / ml sol in 50 ml syringe)</td>
<td>Selective α-2 agonist causing sedation and analgesia. Dose related hypotension limits effective loading dose (often omitted) Useful in agitation states where some analgesia is required but airway reflexes relatively maintained</td>
<td></td>
</tr>
<tr>
<td>Haloperidol</td>
<td>2.5-10 mg IV Iprn</td>
<td>First line major tranquiliser, use for delirium, agitation esp. in opioid / benzodiazepine withdrawal. α blocker: hypotension</td>
<td></td>
</tr>
<tr>
<td>Chlorpro-mazine</td>
<td>5-10 mg IV Iprn, or 50 mg / 50 ml D5W @ 1-10 ml/hr</td>
<td>As for haloperidol; 2nd line tranquiliser More sedating, unpredictable &amp; longer acting agent Potential for marked hypotension esp given IV</td>
<td></td>
</tr>
</tbody>
</table>

### Approach to muscle relaxants in the Intensive Care Unit

#### Introduction

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
- Facilitate acute procedures: tracheostomy, bronchoscopy.
- In selected patients with severe head injury and uncontrolled intra-cranial pressure.

### Complications

- Hyperkalaemia (suxamethonium)
- Polynervopathy and myopathy

### Commonly used muscle relaxants in the Intensive Care

<table>
<thead>
<tr>
<th>Relaxant</th>
<th>Dose</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suxamethonium</td>
<td>100-200 mg or 1-2 mg/kg</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, hyperkaemic states (K+ &gt; 5.5)</td>
</tr>
<tr>
<td>Vecuronium</td>
<td>2-8 mg IVI</td>
<td>Firstline non-depolarising agent No real advantage over pancuronium, although less tendency to cause tachycardia. Repeated dosing leads to accumulation and increased risk of myo- 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 secs) non depolariser Duration of action: 30-40 minutes Used as alternative to suxamethonium for emergency intubation</td>
</tr>
</tbody>
</table>

### Anticoagulation

#### General Principles

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 Counterpulsation: (See guidelines on IABP. Heparin use not routine).
- Continuous Renal replacement therapy (See below)

**Prophylactic use of heparin**

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).

Non-pharmaceutical 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**

- Clinical coagulopathy or thrombocytopaenia
- Therapeutic anticoagulation (eg Warfarin, heparin)
- Significant intra-cerebral haemorrhage
- Heparin Induced Thrombocytopaenia.

**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, anerysm 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:
Sixth ACCP Consensus Conference on Anti-thrombotic Therapy. Chest 2001:119; 132-175 S

**Systemic anticoagulation using unfractionated heparin**

Weight based nomograms are more effective in achieving therapeutic anti-coagulation in a shorter period of time.

- Proceed with loading dose if safe: 70 units / kg.
- Continue with 20 units / hr / kg as continuous infusion (10 000 units heparin in 100 ml Normal Saline = 100 iu / ml.
- Check aPTT 4-6 hrly and adjust infusion rate according to chart:

<table>
<thead>
<tr>
<th>APTT</th>
<th>Change in Heparin Infusion Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 150</td>
<td>Reduce by 500 iu/hr (5mls/hr)</td>
</tr>
<tr>
<td>121–150</td>
<td>Reduce by 300 iu/hr (3mls/hr)</td>
</tr>
<tr>
<td>101–120</td>
<td>Reduce by 100 iu/hr (1ml/hr)</td>
</tr>
<tr>
<td>60–100</td>
<td>No change – Therapeutic</td>
</tr>
<tr>
<td>45–60</td>
<td>Increase by 100 iu/hr (1ml/hr)</td>
</tr>
<tr>
<td>36–44</td>
<td>Increase by 200 iu/hr (2mls/hr)</td>
</tr>
<tr>
<td>&lt; 36</td>
<td>Increase by 400 iu/hr (4 ml)</td>
</tr>
</tbody>
</table>

N.B. For APPT’s within 12 hours of starting thrombolytic therapy do not discontinue or decrease the infusion unless:
- significant bleeding occurs
- APPT is > 150 secs
- Adjust the infusion as per nomogram if APPT < 50 secs.
- Heparin toxicity (prolonged APTT) in a patient that is actively bleeding should be reversed with Protamine Sulphate 50 mg aliquots IVI (usually diluted). Administration of protamine may aggravate peripheral vasodilatation and hypotension in susceptible patients.
Recombinant Factor VIIa

Introduction

The intrinsic and extrinsic pathways of coagulation serve as useful laboratory models for in-vitro coagulation tests. In vivo, the critical pathway involves exposure of tissue factor (TF) which then binds with factor VIIa (endogenous activated VII usually only 1% of total fVII). Exogenous VIIa therefore initiates vigorous coagulation in patients with endothelial injury and exposed TF, but only where sufficient fibrinogen exists in serum.

Pre-conditions for use

Intractable bleeding in the setting of:
- Surgery
- Trauma
- Medical indications (oesophageal varices, pulmonary haemorrhage, selected haematological disorders, possibly warfarin related intracranial haemorrhage)

Every effort has been made to address a surgical bleeding point (i.e. surgical exploration or angiography-embolisation). Clotting factor replacement is optimal including platelets (> 50 × 10^9/L): fVIIa will only enhance in-vivo clotting, not replace it! If enhanced fibrinolysis is suspected, then anti-fibrinolytic agents should be given before fVIIa.

Duty ICU Specialist approval required for use in ICU. All “off-label” use must be recorded for audit purposes.

Contra-indications

fVIIa use in non-haemophiliac populations, particularly the critically ill, is relatively new. Its safety has not been established in:
- DIC
- Sepsis
- Recent unstable angina
- Pregnancy

Its pro-thrombotic effect clearly requires caution where further thrombosis may lead to mortality/morbidity.

Dose

20 – 100 µg/kg as an IVI bolus. A second dose 2 hours later may be of some benefit, but further doses thereafter are unlikely to help.

Reference:
Novoseven online. Available at http://www.novoseven.com

Heparin Induced Thrombocytopenia

Introduction

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.

HITS may appear more common in the setting of continuous renal replacement therapy. This might reflect patient condition and platelet adsorption to dialysis filter.

Diagnosis

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 = 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)

Danaparoid Sodium Dosing

<table>
<thead>
<tr>
<th>IVI loading dose: (body weight adjusted)</th>
<th>Table 9</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 60 kg</td>
<td>1500 U</td>
</tr>
<tr>
<td>60-75 kg</td>
<td>2250 U</td>
</tr>
<tr>
<td>75-90 kg</td>
<td>3000 U</td>
</tr>
<tr>
<td>&gt; 90 kg</td>
<td>3750 U</td>
</tr>
</tbody>
</table>

Infusion: Danaparoid in 250 ml 5% dextrose Infuse decrementally
- 400 U/hr (44 ml/hr) for 4 hours
- 300 U/hr (33 ml/hr) for 4 hours
- 200 U/hr (22 ml/hr) titrated to a anti-Xa level target 0.5-0.8

Subcutaneous: anti-Xa U/ml
- 750 U 8-12 hrly

NB: Danaparoid Sodium is renally cleared and dose adjustment may be necessary in patients with renal dysfunction.

Reference:
Glycaemic control in the critically ill has become one of the most debated aspects of care. There is some evidence that tight control of blood sugar (4.4 – 6.0 mmol /L) is associated with improved outcome. This effect may be preserved at levels less than 8 mmol/L. This area of ICU practice is evolving and requires regular review.

**Target blood sugar level = 4.4 – 6.0 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 ketogenic 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.

Administer in a 50 ml syringe via syringe driver.

Maximum infusion rate never to exceed 25 IU/hr.

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.

**TIGHT GLYCAEMIC CONTROL IN WAIKATO ICU**

<table>
<thead>
<tr>
<th>Blood sugar on first assessment</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 3.3 mmol/L</td>
<td>Administer 20 ml 50 % dextrose, stop insulin administration if any. Ensure adequate glucose intake ( 1 ml/kg 5 dextrose or equivalent enteral feed) Repeat BSL in 30 min</td>
</tr>
<tr>
<td>3.4 – 4.3 mmol/L</td>
<td>Ensure adequate glucose intake. Repeat BSL in 30 min</td>
</tr>
<tr>
<td>4.4 – 6.0 mmol/L</td>
<td>Repeat blood sugar in 1 hr</td>
</tr>
<tr>
<td>6.1 – 12.2 mmol/L</td>
<td>Start actrapid infusion at 1 IU/hr. Repeat BSL in 1 hr</td>
</tr>
<tr>
<td>&gt; 12.2 mmol/L</td>
<td>Start actrapid infusion at 2 IU/hr. Repeat BSL in 1 hr</td>
</tr>
</tbody>
</table>

**Ongoing sugar control management**

<table>
<thead>
<tr>
<th>Blood sugar</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 3.3 mmol/L</td>
<td>Stop insulin and administer 20 ml 50 % dextrose Ensure adequate glucose intake ( 1 ml/kg 5 dextrose or equivalent enteral feed) Repeat BSL in 30 min</td>
</tr>
<tr>
<td>3.4 – 4.3 mmol/L</td>
<td>Decrease insulin infusion by half Ensure adequate glucose intake Repeat BSL in 30 min</td>
</tr>
<tr>
<td>4.4 – 6.0 mmol/L</td>
<td>Insulin infusion unchanged Repeat blood sugar in 1 hr Once stable in range repeat 2 hrly (or 4 hrly if &gt; 48 hrs post admission)</td>
</tr>
<tr>
<td>6.1 – 7.8 mmol/L</td>
<td>Increase infusion rate by 0.5 IU/hr. Repeat BSL in 1 hr</td>
</tr>
<tr>
<td>7.9 – 12 mmol/L</td>
<td>Increase infusion rate by 1 IU/hr. Repeat BSL in 1 hr</td>
</tr>
<tr>
<td>&gt; 12 mmol/L</td>
<td>Increase infusion rate by 2 IU/hr</td>
</tr>
</tbody>
</table>

**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.

**Tight glycaemic control is not practised in children or in neurosurgical patients requiring ventriculostomy or ICP monitoring (the latter at neurosurgical request because of concerns over the effect of low normal glucose levels on the injured brain)**

**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:**


**DDAVP**

**For Diabetes Insipidus**

**General**
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 µg 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)

**For Platelet dysfunction**

**Indications**

Adjunctive treatment in bleeding patients with platelet dysfunction as a result of

- Uraemia
- Cirrhosis
- Von-Willebrand’s Disease
- Drug (NSAIDs 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 µg / kg IVI over 30 minutes or 300 µg 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. The CORTICUS study results would suggest that steroids do not reduce mortality in septic shock.

**Proven Indications**

- Hypoadrenalism (Addison’s disease or crisis)
- Acute severe asthma
- Panhypopituitarism
- Haemophilus meningitis in children (discuss with paediatric team first)
- Pneumocystis Carinii pneumonia (PaO2 < 60 mmHg)
- 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></td>
</tr>
<tr>
<td>Fludrocortisone</td>
<td>0.1</td>
<td>10</td>
<td>250</td>
</tr>
</tbody>
</table>
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 $K^+ , Mg^{2+} \text{ and } PO_4^{3-}$.

Commonly encountered Renal Drugs

<table>
<thead>
<tr>
<th>Drug</th>
<th>Administration</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frusemide</td>
<td>Bolus: 20-100 mg PO / IVI prn. Infusion: 2.5-10 mg/hr (larger doses have also been in used, however this practice is not well documented in standard literature)</td>
<td>Potent loop diuretic. More effective administered as infusion. Toxicity (deafness and interstitial nephritis) increased with co-administration of aminoglycosides. $\downarrow K^+, Mg^{2+} \text{ PO}_4^{3-}$ common.</td>
</tr>
<tr>
<td>Spironolactone</td>
<td>25-100 mg bd</td>
<td>Potassium sparing diuretic. May prevent pathological cardiac modelling in low dose. 2nd line agent, diuretic resistant heart failure.</td>
</tr>
<tr>
<td>Mannitol</td>
<td>20”solution (200 mg/ml) Bolus: 100 ml pm IVI. Traditional doses of 0.5-1.0 g/kg body weight are probably excessive. Max dose 3 g/kg/day or see opposite.</td>
<td>Potent osmotic diuretic. May cause hyper-osmolar state, with increased osmolar gap (measured-calculated osm). Dose limited by ceiling of 300 mosm/L. Limited role: acute head injury with raised ICPn. Has been used but has no proven role in treatment of myoglobinemic /-uric states.</td>
</tr>
</tbody>
</table>

Gastro-intestinal drugs

Prophylaxis of gastric “stress ulceration”

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 (ranitidine 50 mg IVI 8 hrly) if patient is not enterally fed and:

- Pre-existing or intercurrent coagulopathy
- Mechanical ventilation > 48hrs

Active GI Bleeding

Diagnosis

- Revealed blood: Nasogastric blood, haematemesis, malaena
- A fall in systolic blood pressure $> 20 \text{ mmHg}$
- Drop in Hb $> 20 \text{ g / l}$ 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.
- Endoscopy ± sclerotherapy / colonoscopy / angiography and attempted vessel embolism if clinically appropriate.
Use of gastro-intestinal pro-kinetic agents

General

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).

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.

GI drugs commonly used in the Waikato ICU

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metoclopramide (maxalon)</td>
<td>10 mg 6 hrly prn</td>
<td>May be a useful first line agent in mild-moderate nausea and vomiting, may reduce gastric stasis (not first line)</td>
</tr>
<tr>
<td>Ondansetron</td>
<td>4-8 mg IVI 8hrly prn</td>
<td>Potent alternative anti-emetic (expensive)</td>
</tr>
<tr>
<td>Cyclizine</td>
<td>25-50 mg IVI 8hrly prn</td>
<td>Useful second line anti-emetic, histamine-1 receptor antagonist, possible anticholinergic side effects, including tachycardia</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>100 mg IVI Q.I.D</td>
<td>Proven pro-kinetic agent, limited by drug interactions (eg. fluconazole, aminophylline)</td>
</tr>
<tr>
<td>Ranitidine</td>
<td>50 mg 8hrly IVI 150 mg bd po</td>
<td>H2-receptor blocker, less effective acid suppression during feeding, ill defined link with increased rates of nosocomial infection, useful prophylactic agent</td>
</tr>
<tr>
<td>Omeprazole</td>
<td>Active Bleeding: 80 mg IVI bolus (own diluent) followed by 8 mg / hr of infusion (40 mg in 100 ml NS) Acute Rx: 40 mg bd IVI Established: 40 mg dly po</td>
<td>Proton pump inhibitor, effective acid suppression for 24hrs, proven efficacy in Rx of actively bleeding ulceration</td>
</tr>
<tr>
<td>Octreotide</td>
<td>Bolus: 50µg IVI Varices: 50µg / hr Fistulae: 100-200µg IVI or SC 8 hrly</td>
<td>Somatostatin analogue, proven as effective as sclerotherapy in ameliorating variceal bleeding (decreases portal pressure), decreases GI secretion volume in treatment of enteric and pancreatic fistulae (attenuated after approx. 1 week)</td>
</tr>
</tbody>
</table>
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.

The Waikato Hospital Intensive Care Unit supports in general the Waikato Hospital Antimicrobial Guide. Another useful reference is the Australian Therapeutic Guidelines: Antibiotic Version 11, 2000, Chairman, Writing Group: Associate Professor J Spicer. These guidelines and updated versions are available at www.tg.au.

This section cannot be a comprehensive guide, but should aid staff as to the unit preferences in antibiotic prescribing practice.

Resident staff may not change antibiotics without prior discussion with the duty ICU specialist.

All antibiotic charting must be reviewed daily. The indication should be recorded on the drug chart.

The unit microbiological results must be reviewed daily and recorded in the patient notes.

| Pharmacological Prophylaxis | 340 |

Prophylactic antimicrobial therapy should be restricted to situations in which it has been shown to be effective, or where the consequences of infection are disastrous.

Antimicrobials should be directed against likely causative organisms, however it is not rational to attempt to cover all possible microbes.

Antibiotics for the purpose of prophylaxis should be administered at the time of anaesthetic induction, and to cover the period of surgery and/or microbe implantation.

A second dose of antibiotic may be warranted if the operation continues beyond one half of the normal dosing interval for the agent being used.

There may be some evidence in specific types of surgery (e.g., vascular surgery) for extending prophylactic cover beyond the immediate operative period, however in general there is little evidence to support such a practice.

Please confirm antibiotic choice and duration with each individual surgeon at the time of admission of the patient to the Intensive Care Unit. The antibiotic choices given below would constitute a rational approach; however it is generally not the role of the ICU staff to direct surgical choice of agent or duration of prophylaxis.

**Pharmacological Prophylaxis by Surgical Specialty**

<table>
<thead>
<tr>
<th>Specialty</th>
<th>Procedure</th>
<th>Antibiotic Choice</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac Surgery</td>
<td></td>
<td>Cephalozin 1g q8h until chest drains removed</td>
</tr>
<tr>
<td>Orthopaedics</td>
<td>Prosthetic large joint replacement</td>
<td>Refer surgeon</td>
</tr>
<tr>
<td></td>
<td>Insertion of prosthetic or transplant material</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Internal reduction and fixation of bones</td>
<td></td>
</tr>
<tr>
<td>Neurosurgery</td>
<td>Antimicrobial prophylaxis is not indicated for CSF leakage following trauma unless it results from an open cranial vault fracture. The value of routine prophylaxis for the insertion of shunts, ventricular drains or pressure monitors remains uncertain, but is widely requested.</td>
<td>Single dose 3rd generation cephalosporin prior to placement of shunt, EVD or ICP monitor or craniotomy. 3rd generation cephalosporin on induction and for 5 days post-cranioplasty.</td>
</tr>
<tr>
<td>Abdominal Surgery</td>
<td>Single dose antibiotic is usually sufficient. If the duration of surgery is &gt;3hrs consider a second dose. If abdominal cavity soiled then a full course of therapy is warranted (see empiric treatment)</td>
<td>Metronidazole 500 mg IVI timed to end at induction + one of the following: Cephalozin 1 g IVI or Gentamicin 3-5 mg/kg IVI Or single agent Cefotetan 1-2 g IVI</td>
</tr>
<tr>
<td>Vascular Surgery</td>
<td>Elective cases involving head and neck reconstruction of the aorta and/or lower limb vasculature, particularly where groin incision involved. Surgical preference is usually for antibiotics to continue until central access or other portals for infection removed.</td>
<td>Antibiotic prophylaxis is not supported in the literature One of the following: Cephalozin 1 g at induction and 8hrly IVI Note: Vascular surgery may be one of the few disciplines where some evidence exists for “prophylactic” antibiotic administration for up to 48 hrs post-op.</td>
</tr>
</tbody>
</table>
The institution of antibiotic therapy prior to definitive bacteriological diagnosis should be based on local epidemiological data, potential pathogens and their known antimicrobial sensitivity patterns.

Whenever possible material (blood, sputum, urine or directly infected tissue) should be obtained before institution of antibiotics, however in life-threatening situations (eg. meningitis) it may be appropriate to administer antibiotics immediately rather than delay pending culture.

With the progression of diagnostic techniques such as PCR and other DNA probe techniques it may be possible to identify organisms despite prior antibiotic administration.

Where there is doubt as to the likely source of infection, or the appropriate choice of empiric antimicrobial, it would be prudent to consult the Department of Infectious Diseases.

Well chosen anti-microbials 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 appropriately.

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Once gram stain or culture results become known, the choice of agent should be rationalised appropriately.

NB: No ICU antibiotic regimen may be changed or altered without prior knowledge, and consent, of the duty ICU specialist.

### Scenario’s Requiring Empirical Use of Antibiotics in the Intensive Care

<table>
<thead>
<tr>
<th>Infection</th>
<th>Comment</th>
<th>Antibiotics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumonia Community Acquired</td>
<td>Community acquired severe pneumonia: 2 or more of RR &gt; 30 / min, PaO2 &lt; 60 mmHg on ABG, P.C02 &gt; 50 mmHg, Diffuse or multi-lobar infiltrate on CXR, Haemodynamic compromise, Mechanical ventilation</td>
<td>Erythromycin (or equivalent) 500 mg to 1 g 6 hrly IVI + Augmentin 1.2 g 8 hrly</td>
</tr>
<tr>
<td>Pneumonia Ventilator Acquired Pneumonia Or Hospital Aquired pneumonia giving rise to ICU admission</td>
<td>Pneumonia developing in a ventilated patient, that was not evident on initiating respiratory support, in a patient ventilated for &gt; 48 hrs.</td>
<td>Tobramycin 5 mg/kg daily adjusted for age and renal function + Piperacillin + tazobactam (Tazocin) 4.5 g IVI 8 hrly + Gentamicin 4-6 mg/kg daily adjusted for age and renal function</td>
</tr>
<tr>
<td>Aspiration Pneumonia</td>
<td>Antibiotics are not indicated for cases of mild to moderate aspiration lung injury. Where severe aspiration has occurred or abscess cavity suspected treat as opposite.</td>
<td>Augmentin 1.2 g 8 hrly IVI</td>
</tr>
<tr>
<td>Exacerbation COAD</td>
<td>Routine use of antimicrobial agents not proven beneficial. Consider treatment if increased cough and dyspnoea associated with increased sputum volume and purulence. Positive sputum culture (H.Influenza, S.Pneumoniae, M. Catarrhalis) may indicate colonisation only.</td>
<td>Amoxicillin 500 mg 8 hrly IVI for 5 days</td>
</tr>
<tr>
<td>Intra-abdominal Sepsis</td>
<td>Faecal peritonitis or perforated viscus</td>
<td>Amoxicillin 2 g IVI 6 hrly + Gentamicin 5 mg/kg IVI daily + Metronidazole 500 mg 12 hrly IVI + Or if patient penicillin sensitive / aminoglycoside contra-indicated Metronidazole 500 mg 12 hrly IVI + Ceftriaxone 1-2g 12hrly IVI</td>
</tr>
<tr>
<td>Pancreatitis (resulting in ICU admission)</td>
<td>Without evidence of extensive necrosis. Where enhanced CT evidence suggests infected necrosis, surgical drainage is indicated</td>
<td>Meropenem 500 mg IVI 8 hrly</td>
</tr>
<tr>
<td>Biliary sepsis</td>
<td>Acute Cholecystitis Ascending Cholangitis Antibiotic therapy should be seen as an adjunct to drainage</td>
<td>Amoxicillin 1 g IVI 6 hrly + Gentamicin 4-6 mg/kg daily IVI + Metronidazole 500 mg 8 hrly IVI</td>
</tr>
<tr>
<td>Line Sepsis complicated</td>
<td>If patient clinically septic from invasive catheter, or at risk of devastating infection (ie. prosthetic valve or graft) then remove catheter and commence treatment accordingly. Removal of infected catheter is often sufficient to “treat” localised access infection.</td>
<td>Vancomycin 1g IVI 12 hrly × 2 doses Consider adding Gentamicin 4-6 mg/kg daily IVI if patient is not already receiving gram negative cover. Review ongoing treatment with cultures according to patient progress.</td>
</tr>
<tr>
<td>Viral (HSV) encephalitis or meningo-encephalitis</td>
<td>Empirical cover viral meningitis / encephalitis where there is a clinical index of suspicion.</td>
<td>Acyclovir 10 mg/kg IVI 8 hrly Review treatment at earliest possible time when gram stain and CSF (+PCR) examination complete.</td>
</tr>
<tr>
<td>Urinary infection precipitating ICU admission</td>
<td>Treat as for severe pyelonephritis</td>
<td>Gentamicin 4-6 mg/kg IVI daily according to age and renal function + Ampicillin 2 g 6 hrly IVI. Or if aminoglycoside undesirable Ceftriaxone 1-2 g IVI daily</td>
</tr>
<tr>
<td>Bacteruria in Intensive Care Patients</td>
<td>Treatment should only be considered if patient shows signs of systemic illness / sepsis, supported by significant pyuria</td>
<td>Remove urinary catheter if possible</td>
</tr>
<tr>
<td>Hospital Aquired or complicated UTI</td>
<td>Particularly associated with instrumentation</td>
<td>Ceftriaxone 1-2g 12 hrly Or Meropenem 1g 12 hourly</td>
</tr>
</tbody>
</table>
Suspected Bacterial Endocarditis

All patients with suspected endocarditis should have at least three sets of blood cultures taken, each from a separate venepuncture site prior to starting treatment. Consult Infectious Diseases team

Cellulitis or soft tissue infection

Simple cellulitis

Cellulitis complicating pre-existing ulcer

Suspected necrotising fasciitis

Burns

No antibiotics indicated unless infection proven

Suspected Systemic Fungal Disease

Suspected Candidiasis

Suspected Aspergillosis

Microbe specific antibiotic use in the ICU setting

Where a specific microbe (pathogen) has been isolated, therapy should be tailored as outlined below.

### Microbe Specific Antimicrobial Choices in the ICU

<table>
<thead>
<tr>
<th>Organism</th>
<th>1st choice</th>
<th>2nd choice</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumococcus</td>
<td>Benzyl penicillin 1.2 g IV 4-6 hrly</td>
<td>Ceftriaxone 1 g IVI daily</td>
</tr>
<tr>
<td>Staphylococcus aureus</td>
<td>Flucloxacillin 2 g IV 6 hrly</td>
<td>Cephalothin 1 g 6 hrly IVI</td>
</tr>
<tr>
<td>MRSA (alert ID team and infection control)</td>
<td>Vancomycin 1 g IVI 12 hrly</td>
<td>Teicoplanin 400-600 mg loading dose IVI, 200-600 mg daily IVI maintenance.</td>
</tr>
<tr>
<td>Meningococcus Household contacts</td>
<td>Benzyl penicillin 1.2 g IV 4-6 hrly</td>
<td>Ceftriaxone 2 g IV daily</td>
</tr>
<tr>
<td>Enterococcus (bacteria or severe infection)</td>
<td>Amoxicillin 1-2 g IV 6 hrly + Gentamicin 5 mg/kg IVI dly</td>
<td>Vancomycin 1 g IVI daily + Gentamicin if SBE</td>
</tr>
<tr>
<td>Gp A Strep. Infection</td>
<td>Benzylpenicillin 1.8 g 4 hrly IVI</td>
<td></td>
</tr>
<tr>
<td></td>
<td>If severe infection consult ID And consider adding:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Clindamycin 600 mg IVI 8 hrly + Intragam 150 mg/kg/day x 5 days</td>
<td>Cease IG when pt. Improves</td>
</tr>
<tr>
<td>Haemophilus influenzae</td>
<td>Ceftriaxone 1g IVI dly</td>
<td>Amoxicillin 1-2 g IVI 6 hrly</td>
</tr>
<tr>
<td>E. Coli</td>
<td>Gentamicin 5 mg/kg IVI dly</td>
<td>Ceftriaxone 1 g IVI dly</td>
</tr>
<tr>
<td>Enterobacter</td>
<td>Gentamicin 5 mg/kg IVI dly</td>
<td>Ciprofloxacin 200-400 mg 12 hrly IVI</td>
</tr>
<tr>
<td>Klebsiella</td>
<td>Gentamicin 5 mg/kg IVI dly</td>
<td>Ceftriaxone 1 g IVI dly</td>
</tr>
<tr>
<td>Pseudomonas aeruginosa</td>
<td>Piperacillin 3 g IVI 6 h + Gentamicin 5 mg/kg IVI dly</td>
<td>Cefipime 1.0-2.0 g 12 hrly IVI &amp; either Tobramycin 5mg/kg IVI dly or Ciprofloxacin 200-400 mg 12 hrly</td>
</tr>
<tr>
<td>Legionella spp</td>
<td>Erythromycin 500 mg -1 g IVI 6 hrly</td>
<td>Consider newer fluoroquinolone when available in IVI formulation</td>
</tr>
<tr>
<td>Pneumocystis Carinii Infection complicating or causing ICU admission qualifies as severe, requiring steroid therapy.</td>
<td>Co-trimoxazole 320 / 1600 mg IVI 6 hrly + methylpred 40 mg bd x 5 days, methylpred 40 mg daily x 5 days, methylpred 20 mg daily x 11 days</td>
<td>Pentamidine 4 mg/kg/day IVI + Steroid therapy as noted opposite.</td>
</tr>
<tr>
<td>Clostridium difficile: 1.Mild / moderate 2.Severe, or relapse after Rx</td>
<td>Cease other antibiotics if possible 1.Metronidazole 400 mg po bd (or 500 mg IVI 12 hrly if nil by mouth) x 7-14 days 2.Repeat above</td>
<td>Vancomycin 125 mg 6 hrly given enterally</td>
</tr>
<tr>
<td>Clostridial infection (Polymicrobial Infection)</td>
<td>Surgical debridement Benzylpenicillin 2.4 g IVI 4 hrly + Gentamicin 5 mg/kg IVI daily + Metronidazole 500 mg IV bd</td>
<td>Meropenem 1g b.d.</td>
</tr>
</tbody>
</table>

Complications of antibiotic use

Drug hypersensitivity: Dermal eruptions, anaphylactoid / anaphylactic reactions

Drug toxicity: Idiosyncratic (non-dose related) or dose related.

Flucloxacillin-hepatotoxicity

Aminoglycoside-renal toxicity

Emergence of bacterial resistance

Selection of nosocomial colonising organisms (and potential pathogens)

Pseudomembranous colitis
Principles of Fluid Management in Intensive Care

Fluid charting

Prologue

All fluid prescriptions must be reviewed daily. Non-standard / bolus fluid orders must be charted individually. Fluid orders should be considered in two components:

Maintenance or replacement fluids

Daily total fluid administration including enteral feeding = 30-40 ml/kg / day or 80-120 ml/hr, selected according to patient serum Sodium and / or glucose tolerance + additional fluid tailored to excessive losses where appropriate.

- 4% dextrose and one 5th normal saline
- normal saline
- 5% dextrose

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

Resuscitation fluids

The intensive care community is divided on the relative suitability of each fluid in the resuscitation of a critically ill patient. In general if crystalloid (normal saline or Hartmann’s solution) is chosen in the first instance, no more than 2000 ml should be administered, followed by colloid.

Fluid boluses should optimally be titrated against a measurable end-point, although most in current use are at best imperfect (see appendix on pre-load).

**Composition of commonly used fluids**

<table>
<thead>
<tr>
<th>Solution</th>
<th>Concentration of solute in mmol / L</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Na</td>
</tr>
<tr>
<td>Normal Saline 0.9%</td>
<td>150</td>
</tr>
<tr>
<td>Half-Normal Saline</td>
<td>150</td>
</tr>
<tr>
<td>1/5 Normal Saline + 4% Dextrose</td>
<td>30</td>
</tr>
<tr>
<td>Hartmann's (lactated ringsers solution)</td>
<td>129</td>
</tr>
<tr>
<td>Gelofusine (500 ml)</td>
<td>144</td>
</tr>
<tr>
<td>Albunex</td>
<td>70</td>
</tr>
<tr>
<td>Hemohes (500ml)</td>
<td>150</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
  - Urea / creatinine
  - Bicarbonate
- Haematocrit
- Charted fluid balance - at best a rough guide
  - Charted intake – (Charted losses of all types + Insensible losses)

Predictors of increased cardiac output in response to administration of fluid

JVP / CVP: Useful in patients with “normal” lungs and right heart function. In other patients a trend in pressures may be useful.
- Pulmonary artery pressures (particularly diastolic), and pulmonary capillary pressure. At best these are poorly related to a response to further fluid (pre-load), but are still widely employed.
- PiCCO derived estimates of intra-thoracic blood volume and extra vascular lung water.
- Variation in arterial wave form peak with positive pressure ventilatory cycle

This is a notoriously difficult aspect of critical care practice. Please consult the appendices on haemodynamic principles. Often the decision to administer fluid is governed by a conglomeration of each of the following. If you are in any doubt consult the duty ICU specialist, but do not delay the administration of fluid 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**

<table>
<thead>
<tr>
<th>Fluid Compartment</th>
<th>Volume (ml/kg)</th>
<th>Volume (l / 70 kg) male</th>
<th>% total body weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma</td>
<td>45</td>
<td>3.150</td>
<td>4.5</td>
</tr>
<tr>
<td>Blood</td>
<td>75</td>
<td>5.250</td>
<td>7.5</td>
</tr>
<tr>
<td>Interstitial</td>
<td>200</td>
<td>14.000</td>
<td>20</td>
</tr>
<tr>
<td>Extracellular</td>
<td>250</td>
<td>17.500</td>
<td>25</td>
</tr>
<tr>
<td>Intracellular</td>
<td>350</td>
<td>24.500</td>
<td>35</td>
</tr>
<tr>
<td>Total body fluid volume</td>
<td>600</td>
<td>42.000</td>
<td>60</td>
</tr>
</tbody>
</table>

NB: These figures apply to a normal person, and may be significantly altered in a critically patient

**Electrolyte distribution**

**ELECTROLYTE DISTRIBUTION IN THE NORMAL PERSON**

<table>
<thead>
<tr>
<th>Electrolyte</th>
<th>Extracellular Fluid Compartments</th>
<th>Intracellular Fluid</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Plasma 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 (ionised)</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>

**Determinants of solute movement and concentration**

**Passive transport mechanisms**

Diffusion: movement of a solute from an area of higher concentration to one of lower
Non-ionic diffusion
Gibbs-Donnan effect: The unequal distribution of diffusible ions on either side of a membrane can be explained if one side contains a poorly diffusible ion (eg. albumin-anion), since at equilibrium;

The product of the diffusible ions in one compartment will equal the product of the same ions in the other compartment.
Within each compartment the total cationic charges equal the total anionic charges-electrical neutrality must be maintained in passive systems.

**Active transport mechanisms**

Energy requiring mechanisms which distribute a substance across a membrane, in a manner not achievable by physical forces alone. These are essential for establishing electrical and ionic differences across membranes, the basis for tissue excitability and other fundamental functions of the body.

**Electrolyte Abnormalities**

**Approach**

Electrolyte derangement should be viewed as resulting from one of the following.

Erroneous results:
Lab error
Haemolysed specimen
Factitious results: eg hyperglycaemia and hyponatraemia; lipoaemic 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.

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**

**Factitious**

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.
Alcohol (including methanol)

Measured plasma osmolarity 270-290 mmol/L
Hyperlipidaemia
Hyperproteinaemia

Neither of the above should be a problem with current ion-specific electrodes.

**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
- Burns

### Hypervolaemia (water excess)
- Renal failure: acute or chronic
- Extra-renal
- Excessive intake (IVI 5% dextrose)
- Oedematous states: CCF, cirrhosis, nephrotic syndrome, hypoalbuminaemia.

### Normovolaemia
- Psychogenic polydipsia
- SIADH
- Hypothyroidism
- Acute adrenal insufficiency

### Management of Severe Hyponatraemia with fitting or decreased LOC

Resuscitative measures and ABC principles should not be delayed.

Hypertonic saline (3, 20%) may be indicated but should not be used without prior discussion with the ICU duty consultant, unless the patient is actively fitting.

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 20 mmol/L in the first 24 hours, and certainly should not be overcorrected (serum Sodium > 130 mmol/L).

In very rare circumstances where fitting or encephalopathy are life threatening, 500 ml of 20% mannitol has been used.

### Hypovolaemic states

Restore volume with normal saline or colloid according to clinical estimate (fluid balance, weight, JVP, CVP).

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 (often ADH ↑ post surgery)
- Address underlying cause (cardiac failure etc)

### SIADH-often misdiagnosed

#### Diagnosis:
- Low serum osmolarity
- Urine osmolarity > plasma osmolarity
- Urine Sodium > 40 mmol/L with normal renal, hepatic and cardiac function, and no diuretic use.

#### Management:
- Fluid restriction (< 1000 ml / day)

### 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 (elderly, post operative nil by mouth)

#### Salt gain
- Iatrogenic administration of Sodium containing feed or IVI fluids.
- Mineralocorticoid excess

#### Management

- 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:

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

e.g; 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 / hr.

Consider DDAVP if central diabetes insipidus has been confirmed

#### Excess salt intake
- Address cause

### Reference:

### Hypovolaemic states

- Fluid restriction if safe to do so (< 15 ml/kg / day)
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Do not correct Sodium by more than 2 mmol / hr.

Consider DDAVP if central diabetes insipidus has been confirmed

#### Excess salt intake
- Address cause
Hypokalaemia: K⁺ < 3.5 mmol/L

Aetiology / classification

- Increased loss
  - Renal
  - Diuretics
  - ↓ serum Magnesium, ↓ 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:
  - β-stimulants (catecholamines, salbutamol)
  - Insulin (endogenous or exogenous)
  - Familial periodic paralysis and related syndromes (consider thyrotoxic states).
  - ↑ pH

Management

Potassium replacement intravenously or orally.

- 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⁺ > 5.0 mmol/L

Aetiology / classification

- Factitious
  - Sampling in proximity to venous infusion
  - Haemolysis: ie collection using vacuum tube systems (venous sampling via vacutainer with narrow gauge needle)
  - Extremes of thrombocytosis and leukocytosis.
- Release from intra-cellular compartments:
  - Acidosis: ↓ pH by 0.1 ≅ serum K⁺ ↑ by 0.5 mmol/L
  - Tissue disruption: tumour lysis syndromes, rhabdomyolysis, intravascular haemolysis, burns
  - Suxamethonium (note: see section on drugs for intubation 3.9.3.1.2)
  - “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)

Management

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:

**EMERGENCY TREATMENT OF HYPERKALAEMIA**

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Mode of administration</th>
<th>Mechanism of action</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium Chloride</td>
<td>10 ml IV stat, repeated in 20 minutes if appropriate</td>
<td>Membrane stabilisation</td>
<td>First line action.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Has no effect on serum potassium concentration</td>
</tr>
<tr>
<td>Insulin</td>
<td>Bolus: 10 units actrapid equivalent insulin with 50 ml 50% dextrose</td>
<td>Intracellular transfer of potassium</td>
<td>Temporising measure.</td>
</tr>
<tr>
<td></td>
<td>Infusion: 20 units of actrapid insulin in 500 ml 10% dextrose over 30-60 min.</td>
<td></td>
<td>Probable decrease in serum potassium concentration of 1 mmol/L for 30-60 minutes with some effect up to 3 hours (either method)</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>Bolus equivalent to 50-100 mmol.</td>
<td>Promotes cellular uptake of potassium by reducing hydrogen-potassium exchange</td>
<td>Temporising measure only.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Do not administer with Calcium salts</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Not appropriate in hypovolaemic acidic patients</td>
</tr>
<tr>
<td>Exchange resins: resonium</td>
<td>Sodium (or Calcium) resonium 30-60g orally or rectally 8 hourly Sometimes given with lactulose 20ml</td>
<td>Exchanges K⁺ for alternative cation in gut, therefore action delayed for &gt; 120 minutes</td>
<td>Definitive but delayed treatment.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>May be difficult to administer in ICU patients with abnormal gut motility.</td>
</tr>
<tr>
<td>β2 stimulants</td>
<td>Nebuliser: 10-20 mg by nebuliser over 10 minutes IVI: 0.5 mg IVI over 10-15 minutes</td>
<td>Intracellular transfer of potassium</td>
<td>Temporising agent although concern exists over the use of an arrhythmogenic agent the critically ill.</td>
</tr>
</tbody>
</table>

Hypophosphataemia: Serum Phosphate < 0.7 mmol/L

Low serum phosphate is associated with serious clinical consequences, and is probably under-appreciated in critically ill patients.
Some studies suggest an incidence of up to a third of all ICU patients may be phosphate deficient.

### Aetiology 386

**Inadequate input**
- GIT phosphate binders (eg. laxatives, antacids)
- Starvation
- Vomiting or nasogastric suctioning
- Relative or absolute Vit D deficiency

**Transcellular shifts**
- Carbohydrate loading, re-feeding phenomenon.
- Drugs: Insulin, catecholamines, steroids, β2-agonists

**Excessive losses**
- Massive diuresis
- Dialysis, including continuous replacement modalities.

### Clinical effects 387

All energy requiring processes may be involved.
- Cardiac: Decreased contractility.
- Respiratory: Failure to wean.
- Muscle / bone: Myopathy, Rhabdomyolysis, Osteomalacia.
- Haematological: Dysfunction of all formed elements of blood.
- Renal: Acute tubular necrosis.

### Phosphate replacement 388

**PHOSPHATE REPLACEMENT IN THE ICU**

<table>
<thead>
<tr>
<th>Serum phosphate:</th>
<th>Dose (IV):</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 4-6hrs</td>
</tr>
<tr>
<td>&lt; 0.5 mmol/L</td>
<td>0.64 mmol/kg over 8-12hrs</td>
</tr>
</tbody>
</table>

### Acid-Base Disturbances in the ICU 389

**Introduction 390**

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 391**

### The concept of pH:

\[ \text{pH} = \text{negative log of the hydrogen ion concentration. Normal range = 7.36 - 7.40} \]

#### Regulation of pH 392

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.

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.

\[
[H^+] = \frac{K \times PCO_2}{[HCO_3^-]} \\
\text{Henderson Equation}
\]

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

From both the above it is clear that any mechanism responsible for regulating or affecting pH does so by changing the relative concentrations of \( HCO_3^- \), \( P_{CO_2} \) or \( H^+ \) directly.

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

- **Adjusting minute ventilation** (increasing respiratory rate or tidal volume) to manipulate \( P_{CO_2} \)
- **Buffering systems**:
  - Bicarbonate ion
  - Haemoglobin
  - Protein substrates
  - Phosphate
- **Renal compensation**: delayed > 6-12hrs

#### Primary and secondary acid-base derangements 393

End point: “constant” \( PCO_2 : HCO_3^- \) ratio

<table>
<thead>
<tr>
<th>Characterising Acid -Base disturbances</th>
<th>Primary Change</th>
<th>Compensation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acid-Disorder</td>
<td>( \uparrow PCO_2 )</td>
<td>( \uparrow HCO_3^- )</td>
</tr>
<tr>
<td>Respiratory acidosis</td>
<td>( \downarrow PCO_2 )</td>
<td>( \downarrow HCO_3^- )</td>
</tr>
<tr>
<td>Respiratory alkalosis</td>
<td>( \downarrow HCO_3^- )</td>
<td>( \uparrow PCO_2 )</td>
</tr>
<tr>
<td>Metabolic acidosis</td>
<td>( \downarrow PCO_2 )</td>
<td>( \downarrow HCO_3^- )</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>( \uparrow HCO_3^- )</td>
<td>( \uparrow PCO_2 )</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>( PCO_2 \text{ in mmHg} = (1.5 \times HCO_3^- \text{ in mmol/L}) + 8 )</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>( PCO_2 = (0.7 \times HCO_3^-) + 21 )</td>
</tr>
<tr>
<td>Acute respiratory acidosis</td>
<td>( \Delta pH = 0.008 \times (PCO_2-40) ) or approx 1 mmol/L ( \uparrow ) in HCO_3^- per 10 mmHg increase in ( P_{CO_2} )</td>
</tr>
<tr>
<td>Chronic respiratory acidosis</td>
<td>( \Delta pH = 0.003 \times (PCO_2-40) ) or approx 4 mmol/L ( \uparrow ) in HCO_3^- per 10 mmHg increase in ( P_{CO_2} )</td>
</tr>
<tr>
<td>Acute respiratory alkalosis</td>
<td>( \Delta pH = 0.008 \times (40-PCO_2) )</td>
</tr>
<tr>
<td>Chronic respiratory alkalosis</td>
<td>( \Delta pH = 0.017 \times (40-PCO_2) )</td>
</tr>
</tbody>
</table>

### 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.

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

<table>
<thead>
<tr>
<th>Determinants of the anion gap</th>
<th>Table 24</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unmeasured anions</td>
<td>Unmeasured cations</td>
</tr>
<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

- **Lactic acidosis**
- **Ketocidosis**
- **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.
  - Form deficit = \( (24-\{\text{HCO}_3^-\}) \times (\text{body weight} \times 0.6) \) in mmol.
  - Generally one third to one half of the estimated deficit should be replaced and then acid-base status reviewed.

### Metabolic Alkalosis

#### Aetiology

- **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^+ resorption
  - 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.

**Nutrition**

**Enteral Nutrition**
The prevalence of malnutrition is increasing in hospitalised patients due to the aging process of the general population and the development of aggressive medical and surgical treatments for chronic debilitating diseases.
The positive consequences of enteral feeding however may go beyond nutrition and extend to immune modulation, and possibly bacterial translocation through the gut.
Enteric feeding is the preferred mode of nutritional support and should be considered in all patients admitted to the ICU.

**Advantages**
In some patient subgroups (trauma) early enteral feeding improves patient outcome.
Enteral feeding helps retain gut integrity and reduce atrophic changes.
May reduce the incidence of gastric erosions and stress ulceration
Cost effective: Cheaper than TPN!
Complications of central access for TPN are reduced (invasive procedures, infective risk)

**Disadvantages**
Regurgitation / aspiration (no difference gastric versus distal feeding)
Diarrhoea: diarrhoea may be a result of osmotic load to the gut, however it is not the most likely reason for diarrhoea in critically ill patients, and other causes should be sought and excluded.

**Indications**
All ICU patients with a secure airway and functioning gut may receive enteral feeding.
Patients admitted post surgical intervention should have the intention to feed cleared with the surgeon in charge.
Patients with operatively placed jejunostomy may commence feeding within 6 hours of placement (again, confer with the surgeon)
Where gastric feeding has not been established by day 5 of ICU admission (or earlier if undernourished), a post-pyloric (duodenal / jejunal) tube should be considered for distal feeding. Use of hypercaloric feeds may be considered to ensure reasonable intake.
Consider placing a fine bore feeding tube, to reduce irritation and ulceration, once feeding has been established for a reasonable length of time (5-7 days).

**Contra-indications**

**Absolute:**
Non-functional gut: anatomical disruption, obstruction, gut ischaemia
Generalised peritonitis
Severe shock states

**Relative:**
Expected short period of fasting (except trauma patients)
Abdominal distension while feeding enterically
Localised peritonitis, intra-abdominal abscess, severe pancreatitis
Comatose patients at risk of aspiration
Extremely short bowel (< 30 cm)

**Feeding Guideline**
(see algorithm below)
- Place a 12F or larger nasogastric tube to allow reliable aspiration (orogastric tube should be considered in patients with anterior and middle cranial fossa trauma).
- Check position of feeding tube with abdo X-ray prior to feeding. It may not be obvious from standard CXR or AXR that the tube is adequately placed, requiring a modified film or both views.
- Nurse the patient at 30-45 degrees head up.
- Commence feeds at 30 ml/hr and feed continuously according to the attached protocol.
- Aspirate the tube 4 hrly (do not attempt routine aspiration of jejunostomies, naso-duodenal or naso-jejunal tubes.
- Flush jejunostomy or gastrostomy tubes with 10-20 ml of saline 6 hourly if not being used.

**The Waikato Hospital Enteral Feeding Algorithm**

**Prokinetics:**
- If feeding is persistently not tolerated > 48hrs then consider
  - Reduction in narcotic dosage
  - Use of a prokinetic agent: metoclopramide 10 mg IVI 6 hrly, then if necessary erythromycin 100 mg IVI 4hrly.
  - Post-pyloric feeding

**Choice of enteral feed**
- Most patients should be commenced on a standard isocaloric feed such as Nutrison standard multifibre.
- Nutritional supplementation should be adjusted to provide approximately 25-35 kCal / kg weight / day of non protein energy, and 1.0-1.5 g / kg body weight of protein per day.
- Immuno-fortified feeds (with glutamine, arginine, nucleotides, omega-3-fatty acids) have shown some benefit in small studies to date. Their use is accepted to be of benefit in polytrauma patients. Despite this there is as yet no defined place for these feed types in the ICU setting.

**Parenteral Nutrition**
- **General**

  Historical attempts at hyper-alimentation may have resulted in the role of PN in the ICU diminishing over the last 10 – 20 years. Concerns still exist over potential immuno-suppression, hyperglycaemia and the infection risk coupled with central venous access.

**PN should not be ordered unless requested by the duty ICU specialist.**

**Indications**
- Parenteral nutrition should only be considered in patients who are not suitable for adequate enteral feeding. Indications for specialised nutritional support apply; route selection is based on specific pathology present which might contraindicate enteral feeding.

**Short term:**
- No enteral intake likely >7-10 days in previously well or mildly malnourished
- No enteral intake likely >5-7 days if previously malnourished or currently catabolic
- Weight loss > 10 % starting body weight

**Long term:**
Structural or functional short bowel syndrome

**Vascular access**
TPN may be administered by central or peripheral access if an appropriate formula is used.

**Complications**
- Depression of immune function
- Gut villous atrophy
- Metabolic imbalance:
  - Electrolyte disturbance
  - Glucose intolerance
  - Hyperosmolar dehydration syndrome
  - Rebound hypoglycaemia on ceasing TPN
  - Hyperbilirubinaemia
- Fluid imbalance
- Trace element and vitamin deficiency

Complications of central venous access.

**Charting TPN**
**Choice of formula**
Waikato Hospital commonly uses 2 pre-mixed ‘3-in-1’ formulae.

“Parenteral nutrition -glutamine supplemented” (should be charted in this way). Must be given centrally. Usually commenced at 60ml/hour. Final hourly infusion rate approximates patient weight if non-catabolic or add 20ml/hour if catabolic.

Peripheral parenteral nutrition- 0.6 kCal/ml. Start at 80 ml/hr and increase to 120 ml/hr as tolerated. Beware extra volume.

Points to note:
- Referral is made on a customised referral form
- If patients blood sugar estimation exceeds 10 mmol/L, the managing team should be encouraged to commence an insulin sliding scale
- A sticker in the TPN folder outlines the expectation for blood sampling and approach to possible line infection. This should be stuck in the patient notes on commencement
- A note should be made in the patient notes if TPN is refused or discontinued
- A daily round is generally appropriate, but for longer courses or where cessation has been recommended by ICU staff, the patient may not be rounded on
- During the week the receptionist will fill out the lab results sheet; at the weekend this should be done by the short day registrar

**Suspected vascular access infection in TPN patient**
See TPN folder

**Blood and Blood Products**

**Introduction**
The decision to transfuse a patient, or administer other blood products, outside of these guidelines is the prerogative of the duty ICU specialist.

Whenever reasonable, the patient’s informed consent to proceed with transfusion should be obtained.

**Blood transfusion**

**Acute resuscitation**
Excessive ongoing haemorrhage is usually surgical in origin. In these circumstances transfusing blood products should be viewed as a bridging procedure until definitive treatment is undertaken.

Platelet count and coagulations studies should be performed, and if abnormal addressed as required.

Blood replacement in an otherwise fit patient should be considered once blood loss is anticipated to exceed 25% of total blood volume (or 1000-1500ml).

A full cross match may take up to 20 minutes, if blood is required faster than this consider one of the following:
- Group specific (ABO, Rh+) blood without full compatibility testing may be available faster (5-10 minutes).
- “O negative” blood can be issued immediately in a true emergency, while similarly “O positive” blood can be used for men, or women past child bearing age.

**Elective transfusion**
Traditionally a haematocrit of 30% or absolute haemoglobin of 10 g/L have been used as a trigger to transfuse a patient. In stable patients with adequate oxygenation there is no need to transfuse until Hb ≤ 70.0 g/L. Critically ill patients with poor oxygenation, myocardial ischaemia, acute head injury or ongoing risk of blood loss may require earlier transfusion.

Elective transfusion in the Waikato Hospital ICU should be conducted according to the algorithm below:

**Elective transfusion of ICU patients**
Platelet transfusion

Indications

Permission may have to be sought from a Haematologist prior to platelets being issued.

Spontaneous haemorrhage is rare at platelet counts of > 10^9/µl (or > 20^9/µl in febrile patients)

Prophylactic transfusion before surgery or invasive procedure:
- Platelet count < 50^9/µl
- Platelet count > 50^9/µl where there is evidence of abnormal platelet function (eg. uraemia, aspirin therapy)

Uncontrolled haemorrhage:
- Transfuse platelets at platelet count < 100^9/µl
- Consider transfusing platelets at any threshold if there is reason to suspect platelet dysfunction.

Bone Marrow failure, TTP, ITP, or H.I.T.S.
Seek advice from haematology team.

Dosing of platelets

One dose of platelets usually means pooled donor platelets from 4 or more donors.

One dose approximates 3-3.5 × 10^11 platelets, or enough to increase the platelet count by 20-25 × 10^9/µl at 24 hours, in the absence of further problems.

Risk of transfusion

In general the risk is similar to that for blood transfusion with the following addition:
- There is a higher risk of bacterial contamination than whole blood (0.6 / 1000 cases per dose)
- HLA allo-immunisation may occur in 45-62% of long term recipients, resulting in transfusion resistant thrombocytopenia.
- Platelet specific antibodies may develop (4% of patients)

Adjunctive treatment

Administration of DDAVP 0.3-0.4 µg/kg over 30 minutes may increase levels of factor VIII:C and VIII:vWF with increased platelet adhesion.

Indication

- Haemophilia A, type I von Willebrand’s Disease.
- Bleeding post cardio-pulmonary bypass
- Uraemia
- Platelet dysfunction secondary to aspirin

Other scenarios where platelet dysfunction is suspected and platelet transfusion might be delayed, or stock exhausted.
**Fresh Frozen Plasma**

### Indications

- Prophylactic transfusion prior to surgery or other invasive procedure
  - Patients on warfarin or VIT K deficiency: consider partial reversal with 1 mg VIT K or full reversal with 10 mg VIT K if time allows (24-36hrs).
  - Prolonged INR or APTT in patients with liver disease
  - Inherited coagulation factor deficiency when factor concentrates not available.

- Haemorrhage
  - Warfarin or VIT K deficiency
  - Prolonged INR or APTT in patients with liver disease
  - Inherited coagulation factor deficiency when factor concentrates not available

- Massive transfusion:
  - Consider administering calcium as citrated stored blood is calcium deficient, retarding the clotting cascade.
  - Whole stored blood does not contain clotting factors in any appreciable number
  - Consider transfusion when INR > 1.5 or APTT > 40 seconds.

- Plasma exchange in TTP and related syndromes.

### Dosing of FFP

10-15 ml/kg (average 2-4 units) according to clotting profile.

**Cryoprecipitate**

### Indications

- Diffuse microvascular bleeding and fibrinogen < 1.0 g/L
- DIC
- Massive transfusion
- Hereditary hypofibrinogenaemia

### Dose

Generally 2-4 units.

### DIC

#### Definition:

A process representing disordered balance of the haemostatic and fibrinolytic systems, usually in response to severe pathophysiological stimuli as part of multisystem organ dysfunction. Characterised by:

- Microthrombi formation causing microvascular obstruction
- Consumption of platelets and clotting factors
- Thrombocytopaenia

#### Diagnosis-DIC screen

- Blood smear examination for evidence of red cell fragmentation, haemolysis, thrombocytopaenia.
- Extended coagulation screen:
  - Prolongation of thrombin clotting time, APTT, Prothrombin time.
  - Hypofibrinogenaemia
  - Low factor VIII
  - Elevated fibrin breakdown products (FDP’s).
- Liver function tests and renal function review.

#### Treatment

- Treat the underlying cause
- Replace blood components as assessed by above DIC screen if patient bleeding or at risk of bleeding.

**Controversial therapies**

- Heparin, fibrinolytics, antifibrinolytics (aminocaproic acid) and other agents have been described in the literature. They do not form part of standard therapy and should not be attempted without ICU consultant approval, and not before exhausting other therapies at the advice of the haematology specialty service.

**Blood transfusion reaction guidelines**

#### Introduction

A wide range of reactions can be manifest upon infusion of blood products.

The response to a suspected reaction depends on the urgency of the transfusion and the magnitude of the adverse reaction.

#### Suspected transfusion reaction

- Stop the infusion and check the patient details against that of the blood product. If there is any discrepancy then discontinue the transfusion.
- If patient and product details are correct then proceed as follows.

#### Mild reactions

- Temperature rise < 1.5 °C without hives, rash, bronchospasm or cardiovascular compromise: restart transfusion at slower rate.

#### Moderate reactions

- If temperature rise > 1.5 °C other manifestations, administer antipyretic (paracetamol 1g) and restart transfusion of the same unit after 20 minutes.

#### Severe reactions

- If any signs or symptoms in addition to temperature rise, discontinue the transfusion and return the blood product to blood bank for re-crossmatch and culture.
- Treat as for anaphylaxis:
  - Volume resuscitation
Adrenaline or vasopressor as necessary.
Bronchodilators if significant airway outflow obstruction.
Adjuncts: anti-histamine, theophylline, corticosteroids may not be appropriate in ICU population and should be discussed with duty ICU specialist.
## Clinical Management

### Introduction

The purpose of this chapter is not to dictate rigid policies on the most appropriate way to manage every patient. Rather it is to provide guidelines on reasonable clinical practice based on the available evidence and where that is lacking, based on consensus practice.

As you will come to realise during your stay in the ICU very few patients read the appropriate textbooks prior to becoming ill. Patients therefore may not behave in a “classical” or expected manner. It is in these patients that these guidelines may help you to adopt a reasonable and standardised approach.

### Cardio-Pulmonary Resuscitation

#### Introduction

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 15:2 in all instances except when an ETT is in place, when it is 5:1 with no compression pause. The rate of compression is 100 / min.
- 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, up to 3 times (200J, 200J to 300J, 360J or equivalent biphasic) if necessary.

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

- secure airway
- ventilate with 100% oxygen
- IV administration.

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 first three defibrillations fail. 1 mg of adrenaline every 3 minutes is an acceptable minimum. Vasopressin 40.i.u IV is a suitable alternative in VF / VT arrests.

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.

If VT / VF persists after 9 defibrillating shocks, give amiodarone 150 mg IVI or lignocaine 1-1.5 mg/kg. Give earlier if a defibrillating shock seems transiently successful.

### Reference:

**Basic and Advanced CPR: Management Plan for Adult Collapse**
Induced hypothermia following cardiac arrest

Introduction

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.

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 proprofol, with intermittent muscle relaxant to ablate shivering if prominent
- Actively cool, using water cooled blanket to temperature target of 33 (32.5-33.5) deg centigrade for 12 hours, as measured with rectal temp probe.
- After 12 hours actively re-warm to 37 degrees.
- 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

Reference:


Respiratory Therapy

Introduction

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 ICU specialist.
## Respiratory Failure

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

### Failure to Oxygenate Adequately

\( P_{\text{a}O_2} < 60 \text{ mmHg} \) under the following conditions:

- \( F_{\text{i}O_2} \) 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 and arterial blood gas, but rather inferring the need for “assistance” as stated below.

### Failure to Ventilate Adequately

\( P_{\text{a}CO_2} > 50 \text{ mmHg} \), unless in the presence of a primary metabolic alkalosis (pH normal or elevated)

## Aetiology

| Lung insult | Pulmonary oedema (hydrostatic-cardiogenic, or leaky capillary-ARDS) |
| Airway pathology | Proximal: COAD, asthma, bronchiectasis, sputum retention |
| Neuroromuscular | Depressant drugs |
| 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

### 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:

- \( P_{\text{a}O_2} < 75 \text{ mmHg} \) on an \( F_{\text{i}O_2} > 0.4 \) (40\% \( O_2 \))
- \( P_{\text{a}O_2} \) to \( F_{\text{i}O_2} \) ratio < 150

Abnormal ventilation as indicated by:

- \( P_{\text{a}CO_2} > 60 \text{ mmHg} \)
**Oxygen Delivery Systems**

<table>
<thead>
<tr>
<th>Classification</th>
<th>Device</th>
<th>Oxygen Flow (l/min)</th>
<th>Approx F\textsubscript{2}O\textsubscript{2} as percent</th>
<th>Comment / Indication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Variable delivery</td>
<td>Nasal Catheters</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></td>
<td>(eg Hudson)</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</td>
<td>6-15</td>
<td>F\textsubscript{2}O\textsubscript{2} = 21 + 4 per l/min</td>
<td>Fixed delivery device at all but extreme of respiratory effort.</td>
</tr>
<tr>
<td></td>
<td>masks</td>
<td></td>
<td></td>
<td>More expensive than hudson type mask, but preferable in unstable patients</td>
</tr>
<tr>
<td>Fixed Delivery Devices</td>
<td>Venturi type masks</td>
<td>2-8</td>
<td>24-50% according to manufacturer instructions</td>
<td></td>
</tr>
<tr>
<td></td>
<td>CPAP devices</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>BiPAP system</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ventilators</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**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.

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 patients 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**

**Introduction**

Mechanical ventilation is one of the mainstays 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 ICU specialist.

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.*

**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
- Relaxant anaesthesia

**Objectives of mechanical ventilation**

Improve patient oxygenation and improve ventilation perfusion mismatch.

To improve alveolar ventilation and reduce P\textsubscript{a}CO\textsubscript{2}.

To increase end expiratory lung volume, preventing or treating lobar or pulmonary collapse and atelectasis.

To increase functional residual capacity: PEEP may help improve oxygenation through lung recruitment, and reduce lung injury with the prevention of repeated opening and closing of alveoli.

To unload the respiratory muscles when there is 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:

<table>
<thead>
<tr>
<th>Complication</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemodynamic</td>
<td>Increased intrathoracic pressure-unmasking of hypovolaemia (although there is significant benefit to LV performance with application of PEEP).</td>
</tr>
</tbody>
</table>
| Respiratory  | Nosocomial pneumonia  
Volutrauma  
Barotrauma  
Ventilator dependency |
| Metabolic    | Post-hypercapnic metabolic alkalosis  
SIADH |
| Local        | Pressure effects from ETT, tracheostomy or face masks. |

**Ventilator settings**

Default ventilator settings, and principles in optimizing ventilation in ICU patients (see appendix for explanation of ventilation modes and terminology)

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

<table>
<thead>
<tr>
<th>Mode</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>SIMV, volume control.</td>
<td></td>
</tr>
<tr>
<td>Oxygen</td>
<td>Titrated delivered oxygen to provide an oxygen saturation &gt; 90% (an equivalent arterial PO$_2$ of &gt; 60 mmHg), unless otherwise specified by the duty ICU specialist.</td>
</tr>
<tr>
<td>Tidal Volume</td>
<td>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). Generally in the Waikato Hospital ICU 8-10 ml / kg is the preferred volume. Peak and plateau airway pressures resulting from instilling the chosen tidal volume should not exceed 40 cmH$_2$O and 32 cmH$_2$O respectively in the short term, and preferably not more than 35 cmH$_2$O and 28 cmH$_2$O respectively for more than a few hours.</td>
</tr>
</tbody>
</table>

Reference:


Respiratory rate

10 to 25 breaths per minute adjusted to an arterial blood gas P$_{aCO_2}$ 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).

In some patients attempting to normalize arterial carbon dioxide content may expose the patient to the risk of barotrauma and / or volutrauma. It is important that the duty ICU specialist is notified if this is suspected.

PEEP

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

<table>
<thead>
<tr>
<th>F$_{iO_2}$</th>
<th>0.3</th>
<th>0.4</th>
<th>0.4</th>
<th>0.5</th>
<th>0.5</th>
<th>0.6</th>
<th>0.7</th>
<th>0.7</th>
<th>0.8</th>
<th>0.9</th>
</tr>
</thead>
<tbody>
<tr>
<td>PEEP</td>
<td>5</td>
<td>5</td>
<td>6</td>
<td>6</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td>12</td>
<td>12</td>
<td>14</td>
</tr>
</tbody>
</table>

Spontaneous mode of ventilation

Indications

Consider allowing the patient to breathe spontaneously if:

F$_{iO_2}$ ≤ 0.4 and PEEP ≤12 cmH$_2$O and

PEEP and F$_{iO_2}$ values are trending downwards.

The patient has spontaneous breathing efforts

Haemodynamically stable

Newer generation ventilators may facilitate the use of spontaneous modes in patients who are sicker, generally however this should be discussed with the duty ICU specialist.

Mode

“Pressure support”

Support level

Titrated 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$_{iO_2}$, 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% per each day of MV, being 6.5% at 10 days and 19% 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.

Minimum requirements for extubation

Improving clinical condition.
Patient stable on F_iO_2 < 0.4 with a P_aO_2 > 60 mmHg
PEEP < 5-8 cmH_2O
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

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%.

Spontaneous breathing trial

- The patient should receive no more than a PEEP of 5 cmH_2O 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).
- Allow 120 minute trial (some suggestion 30 minutes may predict adequately)
- If trial successful extubate

Markers of successful Spontaneous Breathing Trial

Objective
Gas exchange acceptable (Oxygen sat > 90%, P_aO_2 > 50 – 60 mmHg, Increase in P_aCO_2 < 10 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:

Chosen 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, and desaturation.

Factors that influence success of weaning

Increase in work of breathing
Increase metabolism (increasing CO_2 production)
Fever, sepsis, carbohydrate excess
Reduction in pulmonary or chest wall compliance
Pulmonary oedema, acute respiratory distress syndrome, atelectasis, pneumonia
Bronchospasm, retention of secretions
Obesity, abdominal distension
Unfavourable respiratory circuit characteristics
Delayed response and high negative pressure to ‘trigger’ high resistance circuitry
Inspiratory flow rate unable to match peak inspiratory flow
Reduction in respiratory muscle power
Electrolyte abnormalities (Hypokalaemia, hypomagnesaemia, hypophosphataemia, metabolic alkalosis)
Cardiovascular failure (Left ventricular failure, shock, anaemia)
Polyneuropathy of the critically ill
Myopathy (eg endocrine or carcinomatous)

Depression in respiratory centre
Excess respiratory depressant drugs
Hypothyroidism
Pain
Brain Injury
Ventilation in the prone position

Introduction:

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. 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 suggests > 12hrs is recommended. 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: PaO₂/FiO₂ 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 counterpulsation.
- 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:


Non-invasive ventilation (NIPPV)

Introduction

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 hypercarbic states

Indications

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

Accepted indications

- Acute exacerbations COAD: Good evidence that NIPPV useful in hypercapnoeic patients.
- 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.

Less accepted indications

- NIPPV 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).
  - 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 NIPPV

- Patient with impaired level of consciousness, including those that are in-extremis.
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 528
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 529
- Aerophagia or gastric distension - Aspiration lung injury
- Mask intolerance and heightened anxiety
- Pressure necrosis of the face

Reference:

Extra-corporeal Lung Support 530

Introduction 531
ECLS is a system of veno-venous extracorporeal gas exchange treatment. 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 for ECLS 532
ECLS has not been proven beneficial in any randomised controlled trial.
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).
- Tidal volumes of 5 ml / kg produce plateau pressures above 35cm H2O.
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 533
Treatment failure is associated with
- Age > 50 years
- Mechanical ventilation > 1 week
- Presence and severity of other organ dysfunction

Actioning ECLS 534
ECLS is no longer performed at Waikato Hospital. Referral to the Intensivist on call for the CVICU is required for consideration of a patient for ECLS.

Reference:
Alpard K A, ZwischenbergerJB. Perfusion 1998; 13: 3-15

Hyperbaric Oxygen Therapy (HBO) in Critically Ill Patients 535
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 is accessed through HMS Philomel at the Devonport Naval Base. 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 North Shore Hospital or Auckland Hospital for subsequent management.

Reference:
Weaver Lk et al. NEJM 2002; 347:1057

Aspects of Renal Failure in Intensive Care 536

Introduction 537
Renal failure in ICU almost invariably involves at least one other organ dysfunction or failure, and carries a mortality of up to 70% in this setting.
The advent of continuous renal replacement therapies and the low efficiency dialytic modalities (SLEDD) have revolutionised this aspect of care.
Conversely “renal protective” strategies have not been proven beneficial in large multi-centre trials.
The decision to commence a patient on continuous renal replacement therapy is often not based on clear indicators (see below) and it should always be discussed with the duty ICU specialist.
The renal unit should be alerted as early as possible to review any patient that might need ongoing renal support beyond the acute critical care period.
Indications for dialysis

The classical compartmentalisation of pre-renal, intra-renal and post-renal factors hold true in ICU with the following considerations:

A missed, but reversible, cause of renal failure has dire consequences—“time is kidney”.

Renal perfusion (blood flow and GFR) in critically ill patients may become directly related to systemic blood pressure as local autoregulation fails. Hypotension, even a marginal decrease, will not be well tolerated.

The renal interstitium is relatively hypoxic even under optimal conditions. When subjected to a multilevel endothelial insult as a result of sepsis or SIRS there is a ready predisposition to a vasomotor nephropathy and progression to overt ATN.

The pharmacokinetics of many drugs in ICU are severely deranged, exposing the patient to a much greater risk of nephrotoxic effects.

Drugs and toxins (including radio-contrast) should not be administered without consideration of their toxicity.

Occult or overt increases in intra-abdominal pressure should always be considered in patients with abdominal distension with or without previous surgery. When considered it should be measured and if necessary addressed (see Intra-abdominal pressure measurement, section 3.1.4).

Assessment of renal function in a given patient

Biochemical markers

Serum creatinine is a poor marker of renal reserve. A rise in serum creatinine > 120umol / L may not occur until more than 75% of renal function is lost.

All patients should have their renal function calculated, and drugs tailored according to the Cockroft and Gault equation:

\[
\text{Creatinine Clearance (ml/sec)} = \frac{(140-\text{patient age}) \times \text{weight in kg} \times 0.8 \text{ for females}}{5000 \times \text{Serum creat. conc in mmolL}^{-1}}
\]

From this equation it can be appreciated that a 20 year old 100kg male will have 6 times the creatinine clearance of an 80 year old 50kg woman, even though they have the same serum creatinine concentration.

Urine

The minimum urine output required to excrete obligatory daily solute load is 0.5 ml/kg / hr

Urine electrolyte analysis is of little use in ICU to diagnose aetiology of renal failure but maybe useful in specific electrolyte abnormalities.

Urine sediment: Unhelpful unless a specific reason exists (true vasculitis, nephritis)

Renal imaging

Ultrasound or nuclear imaging techniques may be useful where pre-existing pathology is suspected, or the renal vasculature has been compromised by surgery or trauma.

Thoracic aortic dissection may extend distally and compromise renal blood flow, particularly where a significant false lumen exists. As soon as is practical following acute repair, investigation of aorto-renal vascular anatomy should be performed to facilitate a fenestration procedure if necessary.

Post-renal pathology, whilst uncommon in ICU, is embarrassing to miss and should be excluded where there is any doubt about the cause of renal failure.

Renal protective strategies

Good practice

The cornerstones of good renal protection are not the administration of various drugs, but critical care practice ie.

Adequate fluid resuscitation (sometimes a difficult concept)

Haemodynamic support to maintain both a good renal perfusion pressure and adequate blood flow. Where necessary this may involve inotrope and / or vasopressor support. Despite historical anxiety about the use of vasopressors (noradrenaline), it is not harmful to the kidneys and may in fact increase renal blood flow in animal studies.

Avoid nephrotoxic drugs where necessary

Treat intercurrent infection

Active surveillance for abdominal compartment syndrome, where this is appropriate.

Drug therapy

The following drugs have been used to promote urine output, but have not been found to impact in a positive way on progression to dialysis or mortality.

Low dose dopamine (increase in urine output secondary to direct tubular effect and minor increase in β-effect)

Furosemide (must be delivered to lumen of tubule to be effective)

Mannitol

Aminophylline

The routine use of these drugs is not advocated.

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 a 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 acidemia
- 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 administration

The attempted removal of cytokines and inflammatory mediators is not yet proven to reduce mortality in humans.

Modes of dialytic therapy in the ICU

<table>
<thead>
<tr>
<th>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</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sustained low efficiency dialysis: essentially slow intermittent dialysis</td>
</tr>
</tbody>
</table>

Continuous veno-venous renal replacement therapy: 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

<table>
<thead>
<tr>
<th>Mode</th>
</tr>
</thead>
<tbody>
<tr>
<td>CVVHD</td>
</tr>
<tr>
<td>Blood flow rate</td>
</tr>
<tr>
<td>150 ml/min</td>
</tr>
<tr>
<td>Dialysate flow rate</td>
</tr>
<tr>
<td>2000 ml/hr</td>
</tr>
</tbody>
</table>

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 prolong filter life and prevents clotting in the filter. Anticoagulation is however widely practiced, the 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”.
- Protamine 100 mg made up to 50 ml with Normal Saline, starting at 5 ml / hr is infused via separate syringe pump into the patient.

The aim of the titration between heparin and protamine infusions is to achieve an APTT across the filter of > 100 sec, while maintaining a patient APTT < 50 sec.

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 lactate buffered and contains \( [K^+] = 1 \text{ mmol/L} \). Add \( K^+ \) according to the table below

<table>
<thead>
<tr>
<th>Plasma potassium conc</th>
<th>Add to 5l standard dialysate solution</th>
<th>Final dialysate ( K^+ ) conc.</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.

Buffering solution

The most dialysate solutions contain lactate as a buffer, as this results in a stable solution. Lactate is then metabolised to bicarbonate in the liver. Commonly therefore patients on CVVHDF will have moderately elevated lactate levels. In the context of a stable patient (unchanged or improving whole blood pH) this should not cause alarm.

Solutions using bicarbonate as a buffer need to be prepared just prior to use. They are more expensive, and more prone to contamination. Bicarbonate buffer fluid is not indicated unless there is significant hepatic impairment causing or contributing to a lactate of over 7.0mmol/L.

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 ICU specialist, 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, sequestering of platelets)
- Air embolism

Increased requirement for experienced staff, and increased nursing workload.

The CVVHDF circuit: A simplified representation of CVVHD
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.
End-tidal CO$_2$ monitoring (calibrate and establish arterial-end tidal difference).
ICP monitoring
Jugular Bulb oximetry
Brain tissue oxygen measurements

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 > 70 mmHg). The addition of jugular venous oximetry has introduced a cerebral blood flow aspect, however added benefit has not been clearly shown. A SjVO$_2$ < 55 % may be suggestive of a low perfusion state, and is associated with poorer prognosis. A high SjVO$_2$ however may equally be evidence of hyperemia or decreased tissue extraction, although the significance of this observation is less clear in a therapeutic sense.

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)
Consider in patients where cerebral status cannot be determined for other reasons (ie sedation for ventilation in polytrauma).
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).

Measurement of Jugular venous saturation may be of additional use in titrating cerebral perfusion pressure where:
A sustained rise in ICP cannot be reduced with maximum medical therapy and cranial decompression may not be possible or appropriate.
Doses of vasopressor required to sustain a CPP > 70 mmHg become non-sustainable or dangerous.

Ventilation of the head injured patient

Maintain P$_{a}$O$_2$ > 80 mmHg
Maintain normocapnia: P$_{a}$CO$_2$ between 35-40 mmHg. Hyperventilation to P$_{a}$CO$_2$ as low as 25 mmHg 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 (ie. impending surgery), or unless guided by jugular bulb oximetry.

Low level PEEP has not been proven to affect outcome of head injury adversely, and is beneficial in the prevention of secondary pulmonary pathology.

Haemodynamic priorities

Maintain perfusion pressure:
Mean arterial pressure (MAP) > 90 mmHg in the absence of an ICP monitor,
Cerebral perfusion pressure (see algorithm below) > 70 mmHg where ICP is being monitored (CPP = MAP-ICP).

Avoid inotrope or vasopressor use until patient adequately fluid resuscitated.

**Fluid maintenance**

- Aim for euvoeiaemisa
- Use crystalloid (usually normal saline) unless a specific contra-indication exists

**Osmotherapy**

<table>
<thead>
<tr>
<th>Indications:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Signs of trans-tentorial herniation</td>
</tr>
<tr>
<td>Progressive neurological deterioration not attributable to systemic pathology.</td>
</tr>
</tbody>
</table>

The patient should be euvoeiaemic prior to initiating osmotherapy.

**Bolus therapy** may be better than infusions: consider-

- Mannitol 0.25 g / kg or (100 ml of 20% )
- Hypertonic saline: 10-20 ml of 4M saline to a serum Sodium concentration of 150-155 mmol/L

**Measured osmolality should generally not exceed 320 mmol/L.**

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.

**Sedation**

- First 24-48hrs: It may be appropriate to use a short acting agent such as propofol to facilitate repeated neurological assessment, particularly where no ICP measurement exists.
- Labile neurogenic hypertension, sympathetic storming or emergence agitation: Consider β-blockade or clonidine.
- Barbiturate use/thiopentone may be used, however levels are no longer measured at Waikato Hospital. Phenobarbitone is an alternative.

**Other management issues relating to neurotrauma**

**Steroids**

- not proven useful and likely harmful in the trauma setting.

**Antibiotics**

- A single dose of antibiotic is sufficient to cover insertion of monitoring catheters.
- A fracture base of skull is not an indication for antibiotic prophylaxis in the absence of a CSF leak.
- CSF should be sent for MC&S every second day. All sampling of CSF, or other disruption of the drainage system should be performed using aseptic technique and according to EVD guidelines.
- 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, 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 SCD when available

**Stress ulcer prophylaxis**

- consider if patient likely to be ventilated for > 48hrs, and not tolerating enteral feeding

**Avoid hyperthermia**

- Hyperthermia should be avoided and treated as possible

**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 evidece of cellular distress at low normal glucose levels.

**Reference:**


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**Cerebral Perfusion Pressure Algorithm**
**Status Epilepticus**

**Definition**

Prolonged or repetitive seizures that occur without a period of recovery between attacks. Refractory status epilepticus refers to ongoing seizures for more than 20-30 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
Acquire IV1 access.
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: Ca²⁺, Mg²⁺, K⁺, PO₄⁻³
- 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.
Further investigations:
- 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, or where muscle relaxants have been administered to the patient.
- Lumbar Puncture: LP’s are generally not indicated.

**Useful Drugs in the Treatment of Status Epilepticus**

<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 IV1</td>
</tr>
<tr>
<td>Midazolam</td>
<td>Consider only in patients with protected airway in ICU setting</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 IV1 as bolus followed by 0.5-1.0 mg/hr IV1 infusion</td>
</tr>
<tr>
<td>Phenytoin</td>
<td>First line treatment along with Diazepam (or other benzodiazepine)</td>
<td>Loading dose 15-20 mg/kg IV1 over 30 minutes with telemetry. Maintenance 300 mg/24hrs titrated to therapeutic range</td>
</tr>
<tr>
<td>Pheno-</td>
<td>Consider if seizure uncontrolled within 20 minutes. Consider only if benzodiazepine and phenytoin therapy have failed.</td>
<td>Loading dose 20 mg / kg over 30 min 1.5 mg/kg/min as infusion</td>
</tr>
<tr>
<td>barbitone</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Ketamine | NMDA receptor antagonist, may be helpful when GABA receptor response to other drugs less effective | Loading dose: 1 - 5 mg / kg
Infusion: 1 - 5 mg / kg / hr
Propofol | Anaesthetic agent used to control refractory status in the intubated patient | 1-2 mg/kg followed by 2-10 mg/kg/hour.
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.

**Reference:**

**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):**
  - 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 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.
- Anticonvulsant prophylaxis (generally with phenytoin) is given for the first 21 days after rupture.

**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 used in the management of vasospasm.

Aim to minimise secondary damage due to cerebral vasospasm, using Nimodipine preferably given orally or gastrically or if necessary IV (Calcium channel antagonist) 10 mg/hr IVI (preferably via CVC)

Nimodipine administration may precipitate hypotension, in general noradrenaline is used to maintain the desired mean arterial pressure (MAP)

**Fluid administration:**

- Maintain at least normovolaemia using 0.9% saline IVI (generally minimum 2.5l/day)
- Hypervolaemia has been advocated as a means of maintaining cerebral flow. Measures of adequate volume status should include:
  - Warm and well perfused patient
  - Urine output at least 0.5 ml/kg / hr, preferably > 1.0 ml/kg / hr
  - CVP > 12 mmHg
  - Maintain normal serum electrolytes and total osmolality

**Adjuncts to treatment**

Direct and chemical (papaverine) angioplasty may play a role in refractory cerebral vasospasm, and should be discussed with Neurosurgeon and Neuroradiologist.

**Approach to diagnosis and management of cerebral vasospasm**
Cardiothoracic Guidelines

Introduction

The Registrar on duty is expected to accept the patient on return from theatre.

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 Cockcroft 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 (eg 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 (ie 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.

Respiratory Care

Default ventilator settings on return to ICU

The nursing staff will prepare a Siemens Servo 900C (in consultation with the Cardiac Anaesthetic Staff delivering the patient) generally consistent with the following:

- F\textsubscript{O\textsubscript{2}} as decided by Cardiac Anaesthetic Staff
- Mode: SIMV/IPS, at rate 10 breaths / min, 10ml/kg tidal volume, PEEP 5
- Nursing Staff change to IPS 10/5 after initial observation period and ascertain whether respiration adequate

Exubation

Once the patient is awake and comfortable they may be converted to spontaneous ventilatory modes.

Exubation 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: P\textsubscript{O\textsubscript{2}} > 70 mmHg on F\textsubscript{O\textsubscript{2}} ≤ 0.4, with normal P\textsubscript{C\textsubscript{O\textsubscript{2}}}
- Normal acid-base status: pH 7.35-7.45, HCO\textsubscript{3}⁻ > 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.

Reference:

Management of Bleeding

Introduction

A set of baseline bloods including clotting profile are performed on accepting the patient from theatre. A TEG 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.

Therapeutic interventions

In the presence of bleeding, treatment should be aimed at correcting the deficit ie.

- TCT high, consider heparin reversal with protamine (TCT may be elevated in other conditions)
- INR > 1.5 or APTT > 1.5 times normal: Consider FFP as first line treatment.

Platelets should be transfused at a low threshold following surgery with CPB even if quantitatively normal.

The role of DDAVP, postoperative aprotonin and other antifibrinolytics is not yet clear. Ask the Duty ICU Specialist prior to administering these products.

Avoid hypertension, as it may exacerbate bleeding.

Using PEEP to reduce mediastinal bleeding is not an evidence based practice. It is likely to exacerbate hypotension due to hypovolaemia, and should not be applied in a level above routine without discussion with the Duty ICU Specialist.

Reference:

Hypotension

Introduction

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 resternotomy is made. Resternotomy 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 ICU Specialist 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 ICU specialist without delay.

Approach to Hypotension (systolic BP < 90 mmHg) 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).

Patients may receive up to 2000 ml of colloid IV1 according to the parameters defined above. No further fluid should be given beyond that limit without informing the duty ICU specialist 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 hypoperfusion (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 of

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.

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 β-blocker with which you are familiar).
- Clonidine 15-45 µg prn IVI.

**Antibiotic administration**

Routine prophylaxis for the non-allergic patient is cephazolin 1g q8h. This is discontinued after chest drain removal.

**Special requests from Surgeons**

In general, particular standing requests from individual surgeons are undesirable. Requests should preferably be entered in the operation note and remain negotiable. Nevertheless, the following standing requests are noted as a general guide to practice:

- Mr Parkinson: GTN at 1 mg/hour for CABG patients unless hypotensive, frusemide 40mg and oral postassium supplements from first postoperative day unless contraindicated, atrial pacing unless “contraindicated” for 24 h
- Mr Lin: GTN at 1-3 mg/hour for radial graft patients unless hypotensive. Will discuss request for blood pressure targets with aortic surgery
- Mr Kerjival: “routine” atrial pacing and Hb target of 70g/l

At no time should these requests stand in the way of proper care of the patient as defined by the Intensivist on duty.

**Microbiology Guidelines**

**Introduction**

Sepsis is a common cause of death in critically ill patients. The detection of active infection, as opposed to colonisation with ICU flora, is difficult but important.

Regular routine microbiological examination is not cost effective in the ICU, and infective screens should only be ordered for specific indications, using the guidelines listed below.

Simple preventative measures are extremely important in the containment of infection and the prevention of bacterial resistance. i.e. Compulsory hand washing by all staff who come into contact with patients. Hands should be washed both before and after patient contact.

Strict aseptic technique for all procedures

Rational prescription of antibiotics.

**Glossary:**

**Systemic inflammatory response syndrome: “SIRS”**

Describes clinical picture following any insult (trauma/major surgery, burns, pancreatitis, hypersensitivity reactions) activating a significant inflammatory reaction.

Defined by the presence of at least 2 of the following:
- Temp > 38 °C or < 36 °C
- Heart rate > 90 bpm
- Respiratory rate > 20 bpm (or P\_{aCO_2} < 32 mmHg spontaneously breathing)
- Plasma WCC > 12000 /mm\(^3\) or < 4000 /mm\(^3\) or > 10\(^\%\) immature neutrophils (band cells)

**Sepsis**

The presence of SIRS as defined above in the presence of a proven microbiological pathogen

**Septic Shock**

Sepsis with hypotension, despite adequate fluid resuscitation, along with the presence of perfusion abnormalities that may include but are not limited to
- Lactic acidosis
- Oliguria
- Acute alteration in mental status

**Nosocomial infection**

Clinically evident infection that was neither present nor incubating at the time of admission to hospital (generally held to appear > 48hrs after admission)

**Colonisation**

The detectable presence of micro-organisms on / in a patient that are not pathogenic or elicit an inflammatory response.

**Screening for sepsis**

**Components of Sepsis Screen—Standard**

<table>
<thead>
<tr>
<th>Site of specimen</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urine microscopy and culture</td>
<td>Pyuria is common in the ICU and may not be indicative of active infection. Routine examination of urine is not indicated as asymptomatic bacteruria is common. Sequential courses of antibiotics will not eradicate infection while the catheter remains in situ. Local instillation of antibiotics into the bladder is similarly ineffective.</td>
</tr>
</tbody>
</table>
Candiduria alone is not significant but must be interpreted with fungal cultures of other parts of the patient. Tracheal aspirates may yield false positives (30%), and false negatives. BAL should be undertaken to confirm suspicion of ventilator associated pneumonia (VAP). Specimens which yield > 5 % intracellular organisms, and confirm presence of white cells without epithelial contamination indicate VAP. Treatment should only be initiated if the patient is systemically unwell. Antibiotics will not eradicate colonising organisms, but may well promote bacterial resistance. Tracheal aspirates may yield false positives (30%), and false negatives. BAL should be undertaken to confirm suspicion of ventilator associated pneumonia (VAP). Specimens which yield > 5 % intracellular organisms, and confirm presence of white cells without epithelial contamination indicate VAP. Treatment should only be initiated if the patient is systemically unwell. Antibiotics will not eradicate colonising organisms, but may well promote bacterial resistance. 

Blood cultures must be taken by fresh venous or arterial puncture. Skin organisms grown from a single bottle are usually considered a contaminant, but should not be dismissed lightly in the presence of a deteriorating patient.

### Components of sepsis screen-directed

<table>
<thead>
<tr>
<th>Fungal cultures</th>
</tr>
</thead>
<tbody>
<tr>
<td>CSF</td>
</tr>
<tr>
<td>Pleural Tap</td>
</tr>
<tr>
<td>X-ray sinuses</td>
</tr>
</tbody>
</table>

Directed or bronchoscopic examination of chest flora.

### Investigation of Pneumonia

#### Community acquired pneumonia

#### Microbiology

- **Common organisms:**
  - Streptococcus pneumoniae, Haemophilus influenzae, Influenza A
- **Other organisms:**
  - Bacteria: Legionella sp, Gram-bacilli, S. Aureus
  - Viral: Influenza B, Paramyxovirus, Adenovirus, RSV
  - Other: Mycoplasma, Chlamydia Psittaci (birds), TB, Chlamydia pneumoniae

### Investigations

- Full blood count and differential
- Biochem: ICU profile
- ABG
- CXR

### Microbiology

- Blood cultures ×2
- Endotracheal aspirate or sputum for microscopy and culture (urgent gram stain)
- Respiratory viral antigen and culture (if not intubated then use nasopharyngeal swab)
- Serology: for atypical bacteria
- Direct antigen detection: Urine (pneumococcal Ag), serum (Legionella Ag) by PCR.
- Pleural fluid: where significant effusion present

### In immuno-compromised host

- Extend spectrum of detection on sputum: fungal stain and culture, Pneumocystis Carinii stain and acid fast bacilli.
- Viral studies for CMV, HSV, EBV
- HIV serology if appropriate
- Consider broncho-alveolar lavage or lung biopsy if initial cultures negative.

### Nosocomial pneumonia in the ICU

#### Introduction

- Incidence: Up to 20% of all ICU patients, 70% of patients meeting criteria for ARDS.

#### Clinical diagnosis, including use of tracheal aspirates, has poor sensitivity and specificity.

### Diagnosis

A diagnosis of nosocomial infection (including ventilator associated pneumonia) should be considered if:

- New and persistent CXR changes
- Tachycardia and tachypnoea
- Fever or hypothermia
- Leucocytosis or leucopaenia
- Purulent sputum
- Deteriorating lung function or increasing ventilatory requirement.

#### Confirmation of diagnosis:

- Broncho-alveolar lavage: Only specimens with Epithelial cell count < 1 % considered
  - > 5 % intracellular organisms considered diagnostic
  - < 5 % intracellular organisms, treat with antibiotics only if patient unstable and subsequent culture reveals > 10⁴ CFU / ml.

### Treatment

Empiric treatment should be guided by the initial gram stain. See antibiotic guideline (ch 4.11).

### Vascular Catheter Sepsis

#### Introduction

It is no longer common practice to remove or replace central access routinely, but only when infected or no longer required.

Suspect line sepsis in the presence of:

- New or unexplained fever
- Or ↑ in WCC
- Deterioration in organ function
- Positive blood culture with likely organism in a patient with sepsis.
- Evidence of local infection (inflammation or pus at the insertion site)
### Guidelines

**623**

Attempt to confirm bacteraemia by taking blood cultures from a peripheral vein (cultures from the line may only indicate colonisation).

Remove line on suspicion of infection. Intra-vascular catheters are not routinely submitted for culture.

### Treatment

**624**

Removal of the infected line will usually result in resolution of clinical sepsis.

Antibiotics are indicated only if sepsis is severe, progressive following removal of the line, or if the patient is high risk (eg. prosthetic implants).

Refer to antibiotic guidelines for selection of antibiotics.

### Subsequent venous access

**625**

In ICU central access may be necessary for ongoing antibiotics or inotropes, so that a new line may have to be inserted immediately.

Where possible wait 24 hours before re-inserting a new line at a new site.

Guidewire exchanges may only be performed where mechanical problems complicate a new catheter site.

### Antibiotic Locking of long term vascular access

**626**

Long stay, surgically implanted catheters may be precious, and should not be removed without consulting the duty ICU specialist.

### Removing long term vascular access

**627**

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.

### Antibiotic locking

**628**

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 antibiotic line locking may be trialled to save the intravascular catheter.

Generally Gentamycin 5 mg / ml or Vancomycin 1mg / ml is used depending on the microbe grown. Amikacin (1.5 mg / ml), ciprofloxacin, flucloxacillin, linezolid and teicoplanin have all been described. Where groad spectrum is required, a combination of ciprofloxacin and teicoplanin has been shown to be stable.

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.

### Fungal infections

**629**

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.
- Prolonged use of intravascular catheters
- Co-existent use of immunosuppressive therapy.

The Department of Infectious Diseases should be consulted where any doubt exists with regard initiation of antifungal therapy.

### Indications for antifungal therapy

**631**

- Prophylaxis in patients following bone marrow transplant or neutropaenic patients.
- Single positive blood culture in a high risk patient
- 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

**632**

See antimicrobial guidelines.

### Drug / Toxin Overdose

**633**

The majority of overdoses are polypharmacological and respond to general supportive measures. Overall mortality is low and usually relates to cardio-respiratory arrest and/or uncontrolled siezures 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

**635**

- Intubated patients
- Uncontrolled siezures
- 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

<table>
<thead>
<tr>
<th>Gastric lavage</th>
</tr>
</thead>
<tbody>
<tr>
<td>The place of gastric lavage in acute poisoning is debatable, and is only of benefit in the hyperacute phase of poisoning (&lt; 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 patient need to be evaluated.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Procedure</th>
</tr>
</thead>
</table>
| 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. |

<table>
<thead>
<tr>
<th>Charcoal</th>
</tr>
</thead>
</table>
| 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. |

<table>
<thead>
<tr>
<th>Indications for administering activated charcoal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Virtually all patients presenting with a drug overdose.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Contra-indications</th>
</tr>
</thead>
</table>
| Elemental metals (lithium, iron)  
| Pesticides  
| Strong acids or alkalis  
| Cyanide  
| Late presentations > 4-6 hrs post ingestion. |

<table>
<thead>
<tr>
<th>Specific Overdoses</th>
</tr>
</thead>
</table>
| 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 ICU specialist prior to commencing therapy not considered part of basic resuscitation measures. |

Management of the unconscious, undetermined overdose
Withdrawal of Treatment in the Intensive Care

Introduction

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

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 unprecedented, and will certainly lead to death.

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.

Severe irreversible condition: A patient has a severe and irreversible condition impairing cognition or consciousness, but where death may not occur for many months. Life sustaining treatment should not be instituted for the underlying condition, but again may be used to achieve a specific goal (eg. waiting for arrival of a family member).
The decision making process

Generally, there should be inter-professional team consensus to withdraw therapy.

The duty ICU specialist 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 not 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.

Reference:

“ANZICS” guidelines on withdrawal and limitation of intensive care therapy, draft format-unpublished

Brain death and organ donation

Declaration of brain death

This procedure is an absolute requirement prior to organ donation.

Where clinical examination is to be used alone, this must be performed by 2 doctors of the status prescribed by local jurisdiction.

The two doctors may choose to 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 Working Party on Brain death and Organ Donation 1998
“ Recommendations concerning Brain Death and Organ Donation”)

Pre-conditions

A cause of coma must be identified and documented.

Reversible causes of coma must be excluded:
- coma caused by drugs / poisons-Morphine, Midazolam, barbiturates etc
- unresponsive state caused by neuromuscular blocking agents-vecuronium, pancuronium etc
- coma caused by hypothermia-core temperature must be ≥ 35° C
- coma caused by metabolic or endocrine disturbance-the patients should have:
  - normal renal function
  - normal hepatic function
  - normoglycaemia
  - normal electrolyte profile

Clinical assessment of brain stem function

It is recommended that this procedure is performed separately by 2 doctors in separate examinations (current guidelines recommend at least two hours apart though this is likely to change) to ensure that death is not confirmed until a minimum of 6 hours after onset of coma (or 24 hours if due to hypoxic-ischaemic brain insult).

A minimum of four hours observation with confirmation of absence of pupilllary, corneal, gag/cough and breathing reflexes must occur, 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.

In some cases this period may be longer (eg. in primary hypoxaemic injuries).

Testing brain stem function

A response at any stage deems the patient is not brain dead and further testing does not proceed.

- absent pupillary responses to light (directional and consensual)
- tests cranial nerve III
- absent corneal reflexes (avoid unnecessary repetition so as not to injure the cornea)
- tests cranial nerve V + VII
- 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 nerve VII + VIII
- absent gag reflex tests cranial nerve IX + X
- absent cough reflex tests cranial nerve IX + X
- absent response to painful stimuli within the cranial nerve distribution
- 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 by placing oxygen tubing into the ET tube and insufflate with 100% oxygen at 2 l/min
  - look for apnoea clinically
  - sample ABG 10-15 minutes following disconnection from the ventilator.
  - the P,CO2 should be > 60 mmHg
Time of death 653
The legal time of death is at the time of the completion of the second test of brain death studies / or whatever time the doctor performing the second set of brain death studies documents on the appropriate form and / or in the patient notes.

Non-clinical certification of brain death 654
Objective demonstration of the absence of cerebral blood flow is required if brain death is suspected and the preconditions (2b) 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 will not allow assessment of all the brain stem reflexes
- Where the effects of sedation agents cannot be excluded
A 6 hour period of observation of absent brain function is preferred prior to radiological examination when the absence of cerebral blood flow may be established by either:
- radionuclide cerebral perfusion scan
- 4 vessel angiography
Certification of brain death is then undertaken after the respective scan has been verified by a practitioner certified to do so.
The legal time of death is at the time of radiological testing.

Frequently asked questions: Exclusions to the diagnosis of brain death. 655
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.

Admission to ICU of Potential Donors 6.10.3
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 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 Intensivist's assessment/family discussion.

It is not intended at this stage to intervene in a different way for patients presenting to the ED in collapse in terms of the decision whether to intubate or not, i.e. there is no plan to intubate/ventilate patients in the hope that their families/society at large may benefit in the instance that the patient becomes a potential donor.

Sound clinical decision making as prevails currently must continue to apply.
**Paediatrics 6.11**

### Introduction 6.11.1

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 6.11.2

#### Policy 6.11.2.1

- **Definition of paediatric patient 6.11.2.1.1**

  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 (eg: 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.

  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.

  What follows is an ABCD approach to the physiological and anatomical factors that affect assessment and management of the paediatric patient.

#### General Paediatric Care in ICU 6.11.2.1

**Airway 6.11.2.1.1**

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 4.0, so 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.

**Breathing 6.11.2.1.2**

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 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>
Don't get caught out:  

**6.11.2.1.2**  

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 6.11.2.1.2**

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.

**Circulation 6.11.2.1.3**

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. 

### Normal age related hear rate

<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)  

Normal blood volume is estimated at 80mls/kg (neonate 90ml/kg, adult 70ml/kg) 

Initial fluid challenge in shocked child should be 20mls/kg of warmed 0.9% saline. Repeat as necessary. 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. No one you know has done this frequently, but many should have done it more often. The technique is an old one that was reintroduced because of practical difficulties and frequent failures involved in venous access in collapsed paediatric patients. 

4ml/kg packed cells can be expected to raise Hb by 10g/l. 

Don't get caught out:  

- Hypotension is a late sign and thus not required for a diagnosis of shock.

**Disability 6.11.2.1.4**

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>
### Motor Score

<table>
<thead>
<tr>
<th>Score</th>
<th>&lt; 12 months</th>
<th>&gt; 12 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>Normal movement</td>
<td>Obey 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>
</tbody>
</table>

### Verbal Score

<table>
<thead>
<tr>
<th>Score</th>
<th>&lt; 2 years</th>
<th>2-5 years</th>
<th>&gt; 5 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>Babbles, words or sentences as normal</td>
<td>Appropriate</td>
<td>Orientated</td>
</tr>
<tr>
<td>4</td>
<td>Less than usual ability</td>
<td>Inappropriate</td>
<td>Confused</td>
</tr>
<tr>
<td>3</td>
<td>Cries to pain</td>
<td>Cries/screams</td>
<td>Inappropriate</td>
</tr>
<tr>
<td>2</td>
<td>Moans to pain</td>
<td>Moans to pain</td>
<td>Moans to pain</td>
</tr>
<tr>
<td>1</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
</tbody>
</table>

### Drugs

**6.11.2.1.5**

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. Use your common sense—concentrated potassium infusions are never given via peripheral vein in any patient irrespective of age.

### Investigations

**6.11.2.1.6**

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

**6.11.2.1.7**

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. A copy of Nelsons is locked in the library cupboard, but is largely supplanted by Internet resources available now.


### Bronchiolitis

**6.11.3**

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 are 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

**6.11.3.1**

- Profuse coryza, congestion and a low-grade fever
- Signs of lower respiratory tract involvement include cough, dyspnoea, wheezing and 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%

Simpler systems like the AVPU system may be preferred.

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.
otitis media, retractions, fine rales and diffuse, fine wheezing.
Rarely - myocarditis, supra-ventricular and ventricular dysrhythmias and inappropriate secretion of antidiuretic hormone
Chest radiographs - usually reveal hyperinflation and 20-30% will show lobar infiltrates and/or atelectasis. May be normal.

### Laboratory

<table>
<thead>
<tr>
<th>6.11.3.2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood</td>
</tr>
<tr>
<td>Microbiology</td>
</tr>
</tbody>
</table>

### Treatment

<table>
<thead>
<tr>
<th>6.11.3.3</th>
</tr>
</thead>
<tbody>
<tr>
<td>If severe enough for ICU admission, the child will require:</td>
</tr>
<tr>
<td>Oxygen to maintain saturation 92-94%</td>
</tr>
<tr>
<td>Barrier nursing</td>
</tr>
<tr>
<td>Disturbance of the child is minimized</td>
</tr>
<tr>
<td>Secure IVI access and appropriate dextrose containing fluid infusion-paediatric/other staff may need to assist with this before or after ICU admission</td>
</tr>
<tr>
<td>Keep NPO until confident intubation will not be required</td>
</tr>
<tr>
<td>Bronchodilators – see below</td>
</tr>
<tr>
<td>CPAP is used as a trial of treatment for children with moderate to severe respiratory distress or frequent relatively brief apnoeas.</td>
</tr>
<tr>
<td>Prolonged apnoeas do not usually respond satisfactorily to CPAP. 5cm H₂O usually prescribed and adjusted upwards if felt necessary.</td>
</tr>
<tr>
<td>CPAP has not been proven to have a role in bronchiolitis in any clinical trial, but at the least seems to provide symptomatic benefit.</td>
</tr>
<tr>
<td>Caffeine or aminophylline are prescribed if requested by Intensivist or Paediatrician</td>
</tr>
<tr>
<td>Appropriate monitoring - If on CPAP or unstable, monitor PaCO₂ transcutaneously continuously if available</td>
</tr>
<tr>
<td>If ventilated, an arterial line should be inserted if possible. Papaverine added as per guideline.</td>
</tr>
<tr>
<td>Ventilatory support – these patients should be nasally intubated with the appropriate size tube</td>
</tr>
<tr>
<td>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)</td>
</tr>
<tr>
<td>Look for chest movement and EtCO₂ trace – adjust the ventilator as appropriate to ensure PaO₂&gt;60mmHg and the desired PaCO₂. Higher PaCO₂ may be tolerated. End-tidal CO₂ monitoring must be used (see separate guideline).</td>
</tr>
<tr>
<td>Insert a nasogastric tube and feed enterally</td>
</tr>
<tr>
<td>Treatment is largely symptomatic</td>
</tr>
<tr>
<td>Sedation - would involve the use of morphine 0.5mg/kg diluted in 50ml 5% dextrose plus/minus midazolam 0.5mg/kg diluted in 50 ml dextrose at 0 – 10ml per hour. Phenobarbitone and chloral hydrate may be considered. Muscle relaxants are often used for the first several hours.</td>
</tr>
</tbody>
</table>

### Emergencies in Ventilated Children with Bronchiolitis

<table>
<thead>
<tr>
<th>6.11.3.4</th>
</tr>
</thead>
<tbody>
<tr>
<td>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:</td>
</tr>
</tbody>
</table>

**“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.

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

<table>
<thead>
<tr>
<th>6.11.3.5</th>
</tr>
</thead>
<tbody>
<tr>
<td>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.</td>
</tr>
<tr>
<td>Antibiotics - should be strongly considered in a sick young infant, especially presenting with apnoeas</td>
</tr>
<tr>
<td>- Use in other admissions to ICU with bronchiolitis is controversial, but the incidence of bacteraemia in this group is said to be about 9% which may justify relatively liberal initial empirical use until blood cultures are proven negative. Antibiotics are given if requested by Intensivist or Paediatrician.</td>
</tr>
</tbody>
</table>
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:**

### Croup 6.11.4

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 6.11.4.1

- 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: 6.11.4.1.1

- 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 6.11.4.1.2

- 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 X-ray department (e.g. for lateral neck Xray).

### Management of severe croup 6.11.4.2

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
  - 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**

<table>
<thead>
<tr>
<th>Drug Dose</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dexamethasone 0.3 mg/kg PO/IV then 0.15mg/kg/12hourly for maintenance</td>
<td>Duncan A in Oh’s Intensive care manual 5e, 2003; 1007-1014.</td>
</tr>
<tr>
<td>Prednisone 2mg/kg PO then 1mg/kg/12hourly for maintenance</td>
<td>Fitzgerald D and Kilham H.Croup: assessment and evidence-based management. MJA: 2003 Oct 6; 179(7): 372-7.</td>
</tr>
<tr>
<td>Budesonide 2-4mg Neb then 2mg/12 hourly for maintenance</td>
<td></td>
</tr>
<tr>
<td>Adrenaline (1:1000 ie 1mg/ml)-1ml 1:1000 starting minimum dose up to 0.5ml/kg (max 5mls) Neb</td>
<td></td>
</tr>
</tbody>
</table>

**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:

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.

<table>
<thead>
<tr>
<th>Age</th>
<th>Common organisms</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 1 month</td>
<td>Group B Streptococci, Enterococci, Gram -ve bacilli, 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, N. meningitidis, 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%, septicicaemia 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** 6.11.6.2

Causes can be subdivided into infectious and non-infectious causes.

**Clinical features** 6.11.6.3

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** 6.11.6.4

- **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.

**Investigations** 6.11.6.5

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>Blood &amp; urine</td>
<td>Influenza A, CMV, mycoplasma, chlamydia serology</td>
</tr>
<tr>
<td>Urine</td>
<td>Pneumococcal antigen</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>

<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) complex seizures 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** 6.11.6.6

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** 6.11.6.7

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
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 (e.g., resistant pneumococci).
- 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 e.g., encephalitis

Beware:

It seems to have become common practice to perform a CT scan prior to LP over concerns regarding intracranial hypertension and the risk of herniation, but:

- A "normal" CT scan does not exclude raised ICP especially in children.
- Death from herniation following LP can occur despite a normal CT scan.
- So the decision to perform a lumbar puncture should be based purely on clinical findings.

An LP is performed at the discretion of Paediatric staff prior to ICU admission (and generally performed by those staff if felt indicated) or at the direction of Intensivist and Paediatrician after ICU admission.

If the ICU resident staff lack the requisite experience to perform this (e.g., in a small infant), Paediatric staff will usually be asked to perform the LP.

### Management

<table>
<thead>
<tr>
<th>Airway</th>
<th>If the child is obtunded and unable to protect their own airway then intubate the trachea.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breathing</td>
<td>If spontaneous respiratory effort is inadequate then support with positive pressure ventilation.</td>
</tr>
<tr>
<td>Circulation</td>
<td>If there is evidence of poor peripheral perfusion or overt shock give a 20mls/kg bolus of warmed 0.9% saline and reassess. This may need to be repeated. Shock resistant to fluid therapy will necessitate inotropic support. (See section on shock).</td>
</tr>
</tbody>
</table>

**Observe for seizures and evidence of raised ICP. Treat these aggressively (see separate sections on non-traumatic coma & status epilepticus).**

### Age Appropriate Antibiotic Treatment for CNS Infections

<table>
<thead>
<tr>
<th>Age</th>
<th>Empiric treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 1 month</td>
<td>Cefotaxime &amp; Amoxicillin /penicillin G</td>
</tr>
<tr>
<td>1 to 3 months</td>
<td>Ceftriaxone &amp; Amoxicillin /penicillin G</td>
</tr>
<tr>
<td>&gt; 3 months</td>
<td>Ceftriaxone</td>
</tr>
</tbody>
</table>

**Additional feature**

- Consider adding to above guideline

**Complicated case**

- Recent head trauma or neurosurgery
- Indwelling CSF shunt
- Immunosuppression

- Gram +ve cocci or diplococci seen in CSF
- HSV Encephalitis not excluded

**Vancomycin**

**Acyclovir**

Once sensitivities are known, broad-spectrum antibiotic cover can be changed to narrow-spectrum monotherapy.

Duration of treatment is at discretion of Paediatrician or Intensivist.

Don't forget to inform public health if meningococcal disease is strongly suspected or proven or if other notifiable diseases are implicated.
Guidelines for the Management of a child with DKA 6.12

Clinical Findings 6.12.1
dehydration
hyperglycaemia
ketacidosis
electrolyte derangement
hyperosmolar state

Treatment Rationale 6.12.2
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 dysbalance and can result in cerebral oedema. This occurs in approximately 7 per 1000 paediatric DKA episodes. It is more common in newly diabetics.

In view of this, there has been a recent trend to replacing volume deficit slowly, as well as slowly correcting hyperosmolality.

Initial management 6.12.3
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.
Assess conscious level.

Investigations 6.12.4
Electrolytes, urea, creatinine, serum osmolality, glucose, CBC, blood gas (venous if not arterial), blood cultures, plasma betahydroxybutyrate, urinalysis and CXR.

Lines & tubes 6.12.5
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 6.12.6
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 6.12.7
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

Choice of fluid 6.12.8
Use 0.9% Saline for replacement and maintenance while glucose > 15mmol/l
Use 0.45%Saline/ 5%Dextrose (equal volumes 0.9%Saline & 10%Dextrose) when glucose < 15mmol

Correcting hyperglycaemia 6.12.9
Should be done slowly. Aim for 2-4mmol/l/hour.
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.
Start insulin (Actrapid) infusion at 0.05units/kg/hour (titrate +/- 0.025units/kg/hour)
No bolus of insulin, unless life threatening hyperkalaemia.
Aim to keep glucose 10-20mmol/l until acidosis resolving (pH>7.3, HCO3 > 15)
Note that prescription of normal sliding scale insulin is inappropriate.

Correcting osmolality 6.12.10
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.
Calculated Osmolality = (\([\text{Na}^+] \times 2\) + [urea] + [glucose])

From a practical view, ensure \(\text{Na}^+\) is not falling.

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.

**Correcting acidosis**  
6.12.11
Bicarbonate is rarely indicated. Consult Intensivist.

**Potassium replacement**  
6.12.12
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.

If K+ > 5mmol/l then do not give potassium
If K+ 4-5mmol/l give 0.1mmol/kg/hour (approximately equivalent to 20mmol/l KCl)
If K+ 3-4mmol/l give 0.2mmol/kg/hour 40
If K+ < 3mmol/l give 0.3 mmol/kg/hour 60
If patient anuric give half the amount of potassium suggested above.

**Phosphate**  
6.12.13
Hypophosphataemia is common and can be corrected if desired with potassium dihydrogen.

**Observe for signs of raised ICP**  
6.12.14
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.

**Pitfalls**  
6.12.15
In severe ketoacidosis beta hydroxybutyrate predominates over acetoacetate. Ketostix react with acetoacetate NOT beta hydroxybutyrate. So may underestimate severity of ketonuria.
Don't forget to correct sodium for presence of hyperglycaemia
\(\text{Corrected sodium} = \text{measured sodium} + \frac{\text{Glucose}-5}{3}\)
Osmolality and glucose often drop rapidly on initiation of treatment, but thereafter decrease insulin infusion rate if falling faster than 2-4mmol/l/hour.
**Spinal Injuries**

**ICU Admission Policy**

<table>
<thead>
<tr>
<th><strong>Assessment and admission to ICU</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with clinically complete or near complete cord injury at or above C5 in the cervical region near-complete signalled by motor power less than 3 in majority of muscle groups below level of injury are assessed with a view to routine ICU admission.</td>
</tr>
<tr>
<td>In thoracic cord injuries, injuries above T6 producing clinically complete or near complete lesion as above are assessed with a view to routine ICU admission.</td>
</tr>
<tr>
<td>Exceptions in both cases are patients with such severe associated morbidity or comorbidity that the Intensivist on duty explicitly advises against ICU admission.</td>
</tr>
<tr>
<td>In every case of spinal cord or significant spinal column, an Orthopaedic team’s advice will have been sought if the referral did not come from that Orthopaedic team directly.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Radiological Assessment of the Spine</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>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 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.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Critical Care Management of Spinal Cord Injury</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>As in other injuries and critical illnesses, priorities revolve around airway, breathing and circulation as the starting point of treatment.</td>
</tr>
<tr>
<td>In an emergency or with an unco-operative patient a rapid sequence induction with MILS and avoiding suxamethonium beyond the first 48 or so 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.</td>
</tr>
<tr>
<td>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 and use of chronotropes, inotropes and vasopressors.</td>
</tr>
</tbody>
</table>
Appendices

Haemodynamic Principles

Introduction

When faced with a patient who may have some haemodynamic impairment you should have a systematic approach to assessing and managing this important issue. The following is one way:

Ask four questions:
- Is the blood pressure actually low?
- Is there any evidence of shock (or poor tissue perfusion)?
- Does this patient require more fluid?
- Do I need to introduce an inotrope, a chronotrope or a vasopressor?

Diagnosing hypotension

Absolutely low blood pressure

Low blood pressure is variously defined as

- Systolic BP < 90 mmHg or Mean arterial pressure < 60 mmHg.
- These are implied limits at which vital organs continue to autoregulate blood flow.

Relatively low blood pressure:

Individual patients may “normally” have elevated or indeed low blood pressure. Hypotension would then be considered as:

- Systolic BP 30% lower than known values for that patient (or an absolute drop of 20 mmHg).

Is there any evidence of shock?

Bedside indicators:

- Cerebral perfusion: restlessness or confusion
- Renal blood flow: oliguria ( < 0.5 ml/kg / hr)
- Cool peripheries (unreliable)

Simple investigations

- ECG: evidence of regional ischaemic changes
- ↓pH on arterial blood gas
- ↑ Serum lactate
- Central venous oxygen saturation.

Surrogate end-points

Generally unwieldy eg. gastric tonometry and measurement of gastric mucosal pH.

Does this patient require more fluid resuscitation?

Introduction-Assessment of Cardiac Preload

This is the hardest question to answer.

All texts and wise men will urge you to adequately volume resuscitate the patient, while simple to say, it is difficult to fulfil.

Defining preload

Pre-load refers to the degree of ventricular filling which infers the degree of stretch of myocytes during diastole.

Starling Curve

The more fluid returned to the heart (venous return), the greater the contractile force of the heart and the greater the volume of blood expelled.

At some point the distension of the heart exceeds its ability to contract and the ventricle will fail.

The Frank Starling Curve

![Figure 1](image_url)

Should I give more fluid?

Patients on the volume responsive part of the Starling curve should increase their cardiac output in response to further intravenous fluid.

How do I know where the patient is on the curve?

In a number of patients this is not problematic as they have haemorrhagic shock, vomiting, diarrhoea or some other reason for absolute or relative (eg. epidural, anaphylaxis) intra-vascular volume contraction

There are a number of techniques for assessing or inferring left ventricular end-diastolic volume as the determinant of pre-load in patients where this is not clear-cut. Two of these, the pulmonary artery catheter and the PiCCO are described separately in this appendix.

Clinical estimates of hydration (moist mucous membranes, skin turgor…) are almost useless in ICU.
Estimation of JVP, or indeed invasive measures of right heart (CVP) or left heart (pulmonary capillary wedge pressure) filling pressures have some relevance in patients with hearts that have normal structure and function. Often this is not the case in ICU.

**So what should I do?**

Often clinical practice relies on your impression and an assessment of the risk of giving more fluid than not doing so. ie. It would be “easier” to give fluid to a hypotensive person with a clear chest who is ventilated, than one with chest crackles who is developing respiratory failure, even if this is due to some other pathology.

The decision to implement a fluid challenge is inextricably linked with a duty to closely observe the results and act accordingly.

**Do I need to introduce an inotrope, a chronotrope or a vasopressor?**

Once the three questions above have been addressed adequately it may become necessary to use an agent to bring about an increase in blood pressure and therefore organ perfusion. ie.

Organ perfusion: \[ Q = (P_i - P_o) \times C \]
- \( Q \) = organ perfusion
- \( C \) = regional conductance (a function of vascular radius, and blood rheology)
- \( P_i - P_o \) = pressure gradient across the tissue bed or organ (generally blood pressure, BP)

BP = Systemic vascular resistance (SVR) × cardiac output, (ie = HR × Stroke volume × SVR).

Manipulation of blood pressure and therefore organ perfusion relies on changing one of the following three parameters therefore: heart rate, stroke volume and vascular resistance.

While the response to a given agent is not always totally predictable, the commonly used drugs would be used with the following expectation:

### INOTROPIC SUPPORT

<table>
<thead>
<tr>
<th>Drug</th>
<th>Action</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenaline</td>
<td>Mixed alpha and beta receptor activity</td>
<td>Will usually result in increased cardiac output.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Vasopressor effect unreliable, but dominant beta effect not seen as for dobutamine.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Associated with beta-2 activity and unwanted metabolic effects (hyoperglycaemia, lactate production)</td>
</tr>
<tr>
<td>Noradrenaline</td>
<td>Predominant alpha activity</td>
<td>Used as a vasopressor with some increase in cardiac performance.</td>
</tr>
<tr>
<td>Milrinone</td>
<td>Methylxanthine. Non-adrenergic receptor activity. Increases cAMP intracellularly by modulating phosphodiesterase activity. May also facilitate diastolic relaxation</td>
<td>Cardiogenic shock due to diastolic failure.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pulmonary hypertension following cardiac valve replacement.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rescue following catecholamine induced down regulation of receptors in patients requiring ongoing chrono-inotropy.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>These agents may accumulate in renal failure.</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>Synthetic beta agonist</td>
<td>Chronotrope and inotrope</td>
</tr>
<tr>
<td>Dopamine 1</td>
<td>&lt; 2.5 µg / kg / min (D-receptor stimulus)</td>
<td>Diuretic</td>
</tr>
<tr>
<td>Dopamine 2</td>
<td>2.5-10 µg / kg / min</td>
<td>Add beta stimulus as for dobutamine</td>
</tr>
<tr>
<td>Dopamine 3</td>
<td>&gt; 10 µg / kg / min</td>
<td>Increasing alpha stimulus.</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>Vasopressor</td>
<td>Predominantly used in anaesthetics for short periods of predictable hypotension associated with epidural or spinal anaesthesia.</td>
</tr>
</tbody>
</table>

The “PAC” was designed in an effort to quantify and therefore manipulate haemodynamic parameters ie.

1. Estimation of cardiac pre-load by measuring pulmonary artery occlusion pressure
2. Estimation of cardiac output by thermodilution

**Pulmonary artery occlusion or wedge (PCWP) pressure**

Under conditions described in the clinical procedures section of this guideline the PA catheter is inserted into a central vein. The PA catheter is introduced into a segmental pulmonary artery using a flow directed technique described below.

**Swan-Ganz-Haemodynamic Waveforms**

A typical Pressure trace during Pulmonary Artery Catheterisation (from left to right) and corresponding diagrammatic representations of compartments; RA Right Atrium, RV Right Ventricle, PA Pulmonary Artery and PAOP Pulmonary Artery Occlusion Pressure.
The premise behind the use of this catheter is that PAOP is determined by left atrial pressure which bears a relationship to left ventricular end diastolic pressure and this in turn relates to left ventricular end-diastolic volume as the final arbiter of pre-load.

This relationship does not hold true if:
- There is not a continuous column of fluid between sensor and left atrium.
- There is mitral regurgitation.
- The compliance characteristics of the left ventricle are abnormal.

Given the above it is not surprising that the PAOP has proven to be an unreliable predictor of preload in clinical practice.

### Complications of Pulmonary Artery Catheterisation 7.3.1.2

- Time spent inserting the catheter may distract from resuscitating the patient.
- Insertion and mechanical problems, thrombosis and infection are similar to those observed with central access cannulation.
- Balloon induced problems:
  - Balloon rupture
  - Catheter knotting
  - Pulmonary infarction
  - Pulmonary artery rupture
  - Pulmonary and tricuspid valve damage
  - Endocarditis
  - Arrhythmia’s

### Place in therapy 7.3.1.4

The impact of a PA catheter, and the haemodynamic variables obtained with it on management, and outcome, are not well defined. Particularly disappointing has been the inability of pulmonary wedge (or occlusion) pressure to reflect in any useful way the pre-load status of a given patient.

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.

Proponents of its use argue that failure of clinical judgement in diagnosing type of shock, or instituting successful treatment is an indication for catheterisation of the right heart, to assess haemodynamic and metabolic variables reflecting type, severity and course of circulatory compromise.

Those who do not favour use of the PA catheter argue that clinical judgement (or less invasive monitoring) is not inferior to catheterisation of the right heart.

The lack of direct evidence to support the use of this type of catheter, and the increasing body of evidence documenting its inability to predict response to fluid loading have resulted in declining use of the PA catheter in clinical practice.

A PA catheter is infrequently inserted within the ICU at Waikato Hospital. Other methods of monitoring are generally preferred.

### “The PiCCO-catheter / monitor” 7.4

#### Introduction 7.4.1

This method evolved as an alternative to catheterisation of the right heart in an attempt to elucidate further the concept of cardiac pre-load, and patient fluid status.

#### Aims 7.4.1.1

- Measurement of cardiac output by less invasive means than catheterisation of the right heart.
- Estimation of pre-load, intra-thoracic blood volume and by inference “extra-vascular lung water”.

#### Technique: 7.4.1.2

Trans-pulmonary thermodilution measurement and analysis of pulse contour.

#### Estimation of cardiac output 7.4.1.3

A bolus of injectate, usually cold fluid, given into a large central vessel (eg. superior vena cava) can be detected in a large artery (eg. femoral or axillary) as a temperature change, giving rise to a pulse contour.

This is similar in theory to the thermodilution principle used in the PA catheter where cold fluid of a known volume and temperature is injected proximally into the catheter and a temperature change detected more distally by a temperature sensor as the colder fluid mixes with blood. The magnitude of the temperature change detected at the thermistor can be used to estimate the volume of blood into which the cold fluid was diluted, and hence the cardiac output.
When this is done across the entire pulmonary circulation however, and is detected in an artery the “pulse contour” generated by the temperature change is flatter and longer, but nevertheless still gives reliable results when extrapolated to predict cardiac output.

### Estimation of pre-load, intra-thoracic blood volume and extra-vascular lung water 7.4.1.4

The derivation of these parameters using this technique is not simple, nor intuitive, and requires extensive extrapolation of data.

### Inferences inherent in estimation of cardiac pre-load using the PiCCO technique 7.4.1.5

By examining the contour of the temperature “pulse wave” generated in the systemic arterial tree by injecting cold fluid centrally, it is possible to make some inferences relating to given blood volumes.

#### Inference 1 7.4.1.6

An injected indicator (cold fluid) always mixes with largest volume accessible. Cold water injected into a central vein will mix into fluid in the following spaces, which together comprise the intra thoracic thermal volume or ITTV.

---

### PICCO Dilution and Redistribution Of Temperature

![Diagram of PICCO Dilution and Redistribution Of Temperature](image)

Where:

- **GEDV** = The volumes of each of the heart chambers are visualised in diastole as representing the state of largest volume. The sum of all fluid in all the chambers is called the global end diastolic volume.
- **PBV** = Pulmonary blood volume, or that volume of blood in the lung vasculature.
- **EVLW** = Extra vascular lung water. The volume of fluid in the lung, that is not in the lung vasculature.
- **ITBV** = intra thoracic blood volume is the sum of all blood in the heart chambers and that in the pulmonary vasculature (PBV)

#### Inference 2 7.4.1.7

By analysing the shape and time characteristics of the temperature wave form (or pulse) it is possible to make certain assumptions with regard to the volumes of mixing as given above.

### PICCO The Fudge Factor

![Diagram of PICCO The Fudge Factor](image)

Where:

- **At** = Arrival time. Time taken for cold injectate given centrally to be first detected in the systemic arterial tree.
- **MTt** = Mean transit time. Time taken for a defined amount of the cold injectate to have passed the thermistor.
- **DSt** = Down Slope time. Exponential down slope time, equivalent to the rate of decline of the temperature pulse form as extrapolated by the PiCCO software.

Intra thoracic thermal volume (ITTV) as defined above can be calculated by the derivation:

\[
\text{ITTV} = \text{MTt} \times \text{the cardiac output.}
\]

Pulmonary Thermal volume (PTV) can be calculated by the derivation

\[
\text{PTV} = \text{DSt} \times \text{the cardiac output.}
\]

By subtracting PTV from ITTV the global end diastolic volume (GEDV) can be calculated. GEDV has been proven to correlate with intra thoracic blood volume (ITBV) in a linear fashion.

The leap of faith is that wave form analysis produced by a transpulmonary thermodilution technique, can be used to calculate cardiac output. Derived fluid volumes may allow estimation of both preload (using global end diastolic volumes or intra thoracic blood volumes), and the amount of extravasated fluid into the lungs (given by extra vascular lung water).

Once you accept this principle you can the use these numbers to guide both further fluid therapy and administration of inotropes or vasopressors.

<table>
<thead>
<tr>
<th>Normal Ranges for PiCCO Evaluation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Variable</strong></td>
</tr>
<tr>
<td>Cardiac Index (Cl)</td>
</tr>
<tr>
<td>ITBI</td>
</tr>
<tr>
<td>EVLWI</td>
</tr>
<tr>
<td>-------</td>
</tr>
<tr>
<td>SVRI</td>
</tr>
</tbody>
</table>
Principles of ventilation

Introduction

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 delivering gas in a manner that mimics the normal tidal volume and breathing frequency pattern.

In contrast oxygenation support is designed to supplement the F\textsubscript{O\textsubscript{2}} 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.

Pressure Generators

These deliver gas at a preset 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 preset volume, independent of compliance, or preset 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 preset 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 discoordination, allowing more rapid weaning.

Synchronised intermittent mandatory ventilation (SIMV)

Is designed to avoid “breath stacking” by partitioning the inspiratory time into patient initiated or spontaneous breaths.

Neither IMV nor SIMV has been clearly shown to allow easier weaning than T-piece trials.

Pressure support ventilation (PSV)

During this type of ventilation the patient breaths are supported to a preset pressure using additional gas flow. Inspiration is usually terminated when the inspiratory gas flow falls to about 25% of the initial flow rate.

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.

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 preset tidal volume.

There are many other forms of ventilation which at the moment are still being investigated. These include airway pressure release ventilation, bilevel ventilation and proportional assist ventilation.
Objectives of mechanical ventilation 7.6

To improve alveolar ventilation and reduce P,CO₂.
To improve oxygenation and ventilation perfusion mismatch.
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.

Other Ventilatory strategies 7.7

Reverse I:E Ratio 7.7.1

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 P,O₂.

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).

Ventilation Mechanics 7.8

Tidal volumes: 7.8.1

Traditionally an average tidal volumes of around 10-15 ml/kg were used, and 10 ml/kg can be considered a reasonably safe tidal volume in most patients including children. In patients with poorly compliant lungs or acute respiratory distress syndrome lower tidal volumes should be used (< 8 ml/kg).

Respiratory frequency 7.8.2

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 7.8.3

Generally this should be set on the ventilator to 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.

Positive end expiratory pressure 7.8.4

Introduction 7.8.4.1

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 F,O₂ and maintain alveolar / small airways recruitment.

Using PEEP 7.8.4.2

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 7.8.4.3

PEEP is generally contraindicatd 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, which is due in part to a decrease in venous return and an increase in alveolar and therefore pulmonary blood pressure, ie an increase in RV afterload, and alteration of left ventricular geometry (intraventricular septum shifted towards the left).

Other complications may include a decrease in renal blood flow and possibly a reduction in portal blood flow.

Note: PSV and VSV are spontaneous modes. They cannot be used in paralysed patients.
Weaning from PEEP

In general to wean from PEEP we usually reduce the fractional inspired oxygen concentration first until it is down to approximately an FiO2 of 0.5.

The PEEP is then slowly removed 3-5 cmH2O at a time, providing the Pao2 is > 60 mmHg.

Reference:
Synopsis of Intensive Care Medicine, L J G Worthley, Churchill Livingston, Ch 41, Mechanical Ventilation 1994.

The Sedation - Agitation Score

One of many sedation scores developed to assess and target depth of sedation. This score is not interchangeable with Glasgow Coma Score, the purpose of which is to assess depth of coma following an insult to the brain.

Classification of anti-arrhythmic drugs

The standard classification of antiarrhythmic drugs was developed by Singh and Vaughan Williams based upon the drug's electrophysiological mechanisms of action:

- Class I drugs act by blocking the Sodium channel, and are divided into 4 groups, IA, IB, IC, and IV based on their effects on repolarization and potency towards blocking the Sodium channel
- Subclass IA drugs are potent Sodium channel blockers (prolong QRS interval), and also usually prolong repolarization (prolong QT interval) through blockade of potassium channels
- Subclass IB drugs have the lowest potency as Sodium channel blockers, produce little if any change in action potential duration (no effect on QRS interval) in normal tissue, and shorten repolarization (decrease QT interval)
- Subclass IC drugs are the most potent Sodium channel blocking agents (prolong QRS interval), and have little effect on repolarization (no effect on QT interval)
- Class II drugs act indirectly on electrophysiological parameters by blocking beta-adrenergic receptors (slow sinus rhythm, prolong PR interval, no effect on QRS or QT intervals)
- Class III drugs prolong repolarization (increase refactoriness) by blocking outward potassium conductance (prolong QT interval), with typically little effect on the rate of depolarization (no effect on QRS interval)
- Class IV drugs are relatively selective AV nodal L-type Calcium-channel blockers (slow sinus rhythm, prolong PR interval, no effect on QRS interval)

Miscellaneous In addition to the standard classes, IA-C, II, III, and IV, there is also a miscellaneous group of drugs that includes digoxin, adenosine and Magnesium with actions that don't fit the standard four classes.

**VAUGHAN WILLIAMS CLASSIFICATION OF ANTIARRHYTHMIC DRUGS**

<table>
<thead>
<tr>
<th>Class</th>
<th>Action</th>
<th>Drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Sodium Channel Blockade</td>
<td>Quinidine, procainamide, disopyramide</td>
</tr>
<tr>
<td>IA</td>
<td>Prolong repolarization</td>
<td>Lignocaine, mexiletine, tocainide, phenytoin</td>
</tr>
<tr>
<td>IB</td>
<td>Shorten repolarization</td>
<td>Encainide, flecainide, propafenone</td>
</tr>
<tr>
<td>IC</td>
<td>Little effect on repolarization</td>
<td>Propranolol, esmolol, acebutolol, l-sotalol</td>
</tr>
<tr>
<td>II</td>
<td>Beta-Adrenergic Blockade</td>
<td>Ibutilide, dofetilide, sotalol (d/l), amiodarone, bretylium</td>
</tr>
<tr>
<td>III</td>
<td>Prolong Repolarization (Potassium Channel Blockade; Other)</td>
<td>Verapamil, diltiazem, bepridil</td>
</tr>
<tr>
<td>IV</td>
<td>Calcium Channel Blockade</td>
<td>Adenosine, digitalis, Magnesium</td>
</tr>
<tr>
<td>Misc</td>
<td>Miscellaneous Actions</td>
<td></td>
</tr>
</tbody>
</table>

The Vaughan Williams Classification –an explanation

The Vaughan Williams classification is relatively simple and is useful as a conversational shorthand based on mechanism of action, for its ability to predict adverse effects, and for preliminary decisions regarding drug therapy, but it has a number of important drawbacks:

Drugs within a class are not necessarily clinically similar; a patient may respond well to one drug in a given class, but not another

Almost all of the currently available drugs have multiple actions; it is rarely apparent which of these actions is responsible for suppression of an arrhythmia in a given patient

The metabolites of many of the drugs contribute to or are primarily responsible for their antiarrhythmic actions (e.g.-procainamide and its metabolite, N-acetylprocainamide; encainide and its metabolite, 3-methoxy-O-desmethylencainide)

The stereoisomers of several drugs can have different actions:

* The stereo isomers of disopyramide (Class IA) have opposite effects on repolarization; the predominant effect in a given patient depends on the degree of stereospecificity exhibited in elimination of the drug by that patient
* Only the l-isomer of sotalol has beta-adrenergic blocking activity
Some of the most widely used drugs (procainamide, disopyramide, amiodarone, sotalol) have multiple actions which might explain their utility in treating a broad range of arrhythmias.

### Class Toxicities

<table>
<thead>
<tr>
<th>Class I</th>
<th>Class II</th>
<th>Class III</th>
<th>Class IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proarrhythmic effects: IA-Torsades de pointes</td>
<td>Sinus bradycardia AV block</td>
<td>Sinus bradycardia Torsades de pointes</td>
<td>Sinus bradycardia AV block Negative inotropic effect</td>
</tr>
<tr>
<td>Class Toxicities of Antiarrhythmic Drugs</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

#### Class I

- Proarrhythmic effects: IA-Torsades de pointes
- IC-CAST proarrhythmia
- Negative inotropic effect
- Infranodal conduction block

#### Class II

- Sinus bradycardia
- AV block
- Depression of LV function (adrenergic-dependent)

#### Class III

- Sinus bradycardia
- Torsades de pointes

#### Class IV

- Sinus bradycardia
- AV block Negative inotropic effect

### Antiarrhythmic Actions of Antiarrhythmic Drugs

<table>
<thead>
<tr>
<th>Drug (Class)</th>
<th>Class I Actions</th>
<th>Class II Actions</th>
<th>Class III Actions</th>
<th>Class IV Actions</th>
<th>Other Actions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quinidine (IA)</td>
<td>++</td>
<td>+</td>
<td>++</td>
<td>+</td>
<td>Alpha-adrenergic blockade</td>
</tr>
<tr>
<td>Procainamide (IA)</td>
<td>+++</td>
<td>+</td>
<td>+</td>
<td></td>
<td>Ganglionic blockade</td>
</tr>
<tr>
<td>Disopyramide (IA)</td>
<td>+++</td>
<td>+</td>
<td>+</td>
<td></td>
<td>Anticholinergic</td>
</tr>
<tr>
<td>Lignocaine (IB)</td>
<td>+++</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mexiletine (IB)</td>
<td>+++</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phenytoin (IB)</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Encaaine (IC)</td>
<td>+++</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flecainide (IC)</td>
<td>+++</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Propafenone (IC)</td>
<td>++</td>
<td>+</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moricizine (I)</td>
<td>++</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Propranolol (II)</td>
<td>+</td>
<td>+++</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Esmolol (II)</td>
<td>+</td>
<td>+++</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sotalol (II / III)</td>
<td>++</td>
<td>+++</td>
<td></td>
<td></td>
<td>Class II actions stereo specific</td>
</tr>
<tr>
<td>Amiodarone (III)</td>
<td>+++</td>
<td>+</td>
<td>++</td>
<td>+</td>
<td>Alpha-adrenergic blockade</td>
</tr>
<tr>
<td>Ibutilide (III)</td>
<td>+++</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Verapamil (IV)</td>
<td>+</td>
<td>+</td>
<td>+++</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diltiazem (IV)</td>
<td></td>
<td></td>
<td>+++</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adenosine (Misc.)</td>
<td>+</td>
<td>++</td>
<td></td>
<td>Enhances potassium conductance</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Inhibits cAMP-induced Ca(^{2+}) influx</td>
<td></td>
</tr>
</tbody>
</table>
### Useful equations in Intensive Care

#### Standard Haemodynamic Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Formula</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cardiac Index</strong></td>
<td>$CI = \frac{CO}{BSA}$</td>
<td>2.5 – 5 l/min/m²</td>
</tr>
<tr>
<td><strong>Systemic Vascular Resistance</strong></td>
<td>$SVR = \frac{MAP - RAP}{CO} \times 79.9$</td>
<td>750 – 1500 dyn.sec/cm²/m²</td>
</tr>
<tr>
<td><strong>Systemic Vascular Resistance Index</strong></td>
<td>$SVRI = \frac{MAP - RAP}{CI} \times 79.9 \times BSA$</td>
<td>1400 – 2400 dyn.sec/cm²/m²</td>
</tr>
<tr>
<td><strong>Pulmonary Vascular</strong></td>
<td>$PRV = \frac{MAP - RAP}{CI} \times 79.9$</td>
<td>150 – 250 dyn.sec/cm²/m²</td>
</tr>
<tr>
<td><strong>Stroke Volume Index</strong></td>
<td>$SVI = \frac{CI}{HR}$</td>
<td>33 – 47 ml/beat/m²</td>
</tr>
<tr>
<td><strong>Left Ventricular Stroke Work Index</strong></td>
<td>$LVSWI = (MAP - PAOP) \times SVI \times 0.0136$</td>
<td>50 – 120 g/m²/beat</td>
</tr>
<tr>
<td><strong>Right Ventricular Stroke Work Index</strong></td>
<td>$RVSWI = (MAP - RAP) \times SVI \times 0.0136$</td>
<td>25 – 55 g/m²/beat</td>
</tr>
<tr>
<td><strong>Arterial Oxygen Content</strong></td>
<td>$CaO_2 = (Hb \times 1.34 \times SaO_2) + (PaO_2 \times 0.0003)$</td>
<td>17 – 20 ml/100ml</td>
</tr>
<tr>
<td><strong>Venous Oxygen Content</strong></td>
<td>$C_vO_2 = (Hb \times 1.34 \times S_cO_2) + (P_cO_2 \times 0.003)$</td>
<td>12 – 15 ml/100ml</td>
</tr>
<tr>
<td><strong>Oxygen Delivery Index</strong></td>
<td>$DO_2I = CI \times C_aO_2 \times 10$</td>
<td>550 – 750 ml/min/m²</td>
</tr>
<tr>
<td><strong>Oxygen Consumption Index</strong></td>
<td>$VO_2I = CI \times (C_aO_2 - C_vO_2) \times 10$</td>
<td>115 – 160 ml/min/m²</td>
</tr>
<tr>
<td><strong>Oxygen Extraction Ratio</strong></td>
<td>$O_2ER = \frac{VO_2I}{DO_2I}$</td>
<td>0.24 – 0.4</td>
</tr>
<tr>
<td><strong>Shunt Equation</strong></td>
<td>$\frac{Q_s}{Q_t} = \frac{(C_aO_2 - C_cO_2)}{(C_cO_2 - C_vO_2)} \times 100$</td>
<td>5 – 15 %</td>
</tr>
<tr>
<td><strong>End Capillary Oxygen Content</strong></td>
<td>$C_cO_2 = (Hb \times 1.34 \times 1.0) + (PAO_2 \times 0.003)$</td>
<td>80 – 100 ml/100ml</td>
</tr>
<tr>
<td><strong>Aveolar Gas Equation</strong></td>
<td>$P_{a}O_2 = F_{i}O_2 (760 - 47) - (P_{a}CO_2 \times 1.25)$</td>
<td>100 – 650 mmHg</td>
</tr>
</tbody>
</table>
### Content of commonly used enteral feeds

#### NUTRITIONAL COMPARISON OF ENTERAL FORMULAS (PER LITRE)

<table>
<thead>
<tr>
<th>Per Litre</th>
<th>Nutrison Protein Plus Multifibre</th>
<th>Nutrison Standard Multifibre</th>
<th>N-source Renal</th>
<th>Pulmocare</th>
<th>Resource For kids</th>
<th>Nova Source 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kcal</td>
<td>1250</td>
<td>1000</td>
<td>2000</td>
<td>1500</td>
<td>1000</td>
<td>2000</td>
</tr>
<tr>
<td>Protein (g)</td>
<td>63</td>
<td>40</td>
<td>74</td>
<td>62.4</td>
<td>30</td>
<td>90</td>
</tr>
<tr>
<td>Fat (g)</td>
<td>49</td>
<td>39</td>
<td>100</td>
<td>92.0</td>
<td>50</td>
<td>88</td>
</tr>
<tr>
<td>CHO (g)</td>
<td>141</td>
<td>123</td>
<td>200</td>
<td>106</td>
<td>110</td>
<td>214</td>
</tr>
<tr>
<td>Na mmol</td>
<td>48</td>
<td>43</td>
<td>43.5</td>
<td>57</td>
<td>17</td>
<td>35</td>
</tr>
<tr>
<td>K mmol</td>
<td>43</td>
<td>38</td>
<td>20</td>
<td>48.6</td>
<td>33</td>
<td>43</td>
</tr>
<tr>
<td>Phos (mg)</td>
<td>29</td>
<td>720</td>
<td>650</td>
<td>1055</td>
<td>800</td>
<td>1050</td>
</tr>
<tr>
<td>Osmolarity</td>
<td>280</td>
<td>330</td>
<td>700</td>
<td></td>
<td>390</td>
<td>790</td>
</tr>
<tr>
<td>Fibre (g)</td>
<td>15</td>
<td>15</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

#### Guidelines for the use of patient controlled anaesthesia (PCA)

### Introduction

PCA is a useful analgesic technique:

- Small and frequent intravenous bolus doses of opioid can be given whenever the patient becomes uncomfortable, or when a painful stimulus is anticipated, enabling individual titration of pain relief. This flexibility helps to overcome the wide interpatient variation in opioid requirements.
- The intensity of acute pain is rarely constant, and PCA means that the amount of opioid delivered can be rapidly titrated.
- Patients can also titrate the amount of opioid delivered against dose-related side effects.

The inherent safety of the technique lies in the fact that, as long as the machine is in PCA mode only (i.e. there is no continuous infusion), further doses of opioid will not be delivered should the patient become excessively sedated.

### Contraindications

- Patient confusion
- Inability to understand the technique.
- Untrained staff (medical and nursing)

### Standard PCA use in ICU

If patients commence a PCA within ICU from an ICU Medical Staff members prescription, either the APS acute pain nurse may be told or the pink form returned to Level 3 recovery so appropriate follow-up may take place.

Oxygen is generally administered, but is not required if oximetry is satisfactory.

Caution with other opioids and sedatives.

It must be clear that if others press the button for the patient, some advantages are lost.

The IV line must contain a non-return valve.

Change syringe every 24 hours and line every 48 hours.

It is customary to chart oxygen, naloxone and an antiemetic on discharge to the ward. Acceptable physiological parameters must also be entered on the PCA chart for ward use.

### Observations

- Pain score, sedation score and respiratory rate need continuous measurement appropriate to the overall condition of the patient, but with a minimum of q 2-4 hourly recordings.
- Data on total demands, “good” demands, amount in syringe, and cumulative dose hourly should be able to be accessed by the bedside nurse.

### Programable Variables

#### Loading Dose

Patient controlled analgesia may not be effective for some time if moderate to severe pain is present from onset.

During the recovery period, to make the patient comfortable before PCA is commenced, a loading dose of opioid may be required.

#### Bolus Dose

The optimum bolus dose is that which results in appreciable analgesia without significant side effects. A dose of 1mg is the usual initial dose.

The dose may be increased or decreased by 0.5mg if effect is insufficient or excessive respectively.

#### Lock-out interval

This is the time from the end of delivery of one dose until the machine will respond to another demand. This allows the patient to feel the effect of one dose before receiving a subsequent dose. Most patients have an inherent maximum frequency of demand-the average rate is 3-5 doses per hour.

#### Continuous (background) infusion

A continuous infusion decreases the inherent safety of PCA, as opioid will be delivered regardless of how sedated the patient may be.

#### Drug concentration

Standard prescriptions are employed (see PCA chart).

#### Total Dose Limit / 4 hourly Limit

The use of dose limits is not usual practice. The inherent safety mechanism of PCA lies in the belief that somnolence or even drowsiness should supervene before overt narcosis. This may not be true where drug clearance is altered (morphine -6 – glucuronide in renal failure), but the clinical significance is unclear.
### Standard Prescriptions for PCA

<table>
<thead>
<tr>
<th><strong>Morphine</strong></th>
<th>7.13.4.1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Usually the opioid of first choice.</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Fentanyl</strong></th>
<th>7.13.4.2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Indications:</strong></td>
<td>7.13.4.2.1</td>
</tr>
<tr>
<td>The use of Fentanyl in PCA's is generally reserved for:</td>
<td></td>
</tr>
<tr>
<td>Patients who are unable to tolerate Morphine</td>
<td></td>
</tr>
<tr>
<td>Patients with renal impairment, in whom accumulation of Morphine 6 glucuronide can lead to over sedation, although the rationale for this is questionable</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Tramadol</strong></th>
<th>7.13.4.3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Indications</strong></td>
<td>7.13.4.3.1</td>
</tr>
<tr>
<td>Elderly</td>
<td></td>
</tr>
<tr>
<td>Patients with respiratory depression or compromise</td>
<td></td>
</tr>
<tr>
<td>Excessive drowsiness with narcotic PCA</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Miscellaneous Agents</strong></th>
<th>7.13.4.4</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pethidine</strong></td>
<td>7.13.4.4.1</td>
</tr>
<tr>
<td>Is not used in PCA's in The Waikato Hospital Intensive Care Unit. This is because norpethidine toxicity is a very real possibility with the doses which could be used in a PCA.</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Methadone</strong></th>
<th>7.13.4.5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Has an extremely long half life compared to other opioids (15 to 40 hours). This makes fast titration with methadone more difficult, and may lead to significant accumulation of the drug.</td>
<td></td>
</tr>
<tr>
<td>Methadone is not used in PCA's in The Waikato Hospital ICU for the above reasons.</td>
<td></td>
</tr>
<tr>
<td>Methadone is most commonly used for the management of chronic pain, or in suppression withdrawal symptoms in individuals addicted to opioids.</td>
<td></td>
</tr>
</tbody>
</table>

### Special Situations

<table>
<thead>
<tr>
<th><strong>PCA and the opioid tolerant patient</strong></th>
<th>7.13.4.6.1</th>
</tr>
</thead>
<tbody>
<tr>
<td>These patients are likely to require much larger doses of opioids and a background infusion benefits many. To calculate an appropriate background infusion, base it upon 50-100% of the patients usual opioid requirements.</td>
<td></td>
</tr>
<tr>
<td>The bolus dose which is ordered (in mg.) is normally the same as the background infusion in mg/hr.</td>
<td></td>
</tr>
</tbody>
</table>

### Incomplete Cross Tolerance

<table>
<thead>
<tr>
<th><strong>Illicit Opioids</strong></th>
<th>7.13.4.6.2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Where a patient has been using illicit opioids the amount and purity of the drug they have been using is difficult to ascertain. As a rough guide, one should assume purity to be no more than 50%. It is safer to be conservative.</td>
<td></td>
</tr>
</tbody>
</table>

### Problem Solving and PCA's

<table>
<thead>
<tr>
<th><strong>Patient Confusion</strong></th>
<th>7.13.4.7.1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient confusion and inability to understand the use of a PCA is an absolute contraindication and other means of analgesia should be used.</td>
<td></td>
</tr>
</tbody>
</table>

### Nausea and Vomiting

<table>
<thead>
<tr>
<th><strong>Pruritus</strong></th>
<th>7.13.4.7.3</th>
</tr>
</thead>
<tbody>
<tr>
<td>An appropriate antiemetic should be given.</td>
<td></td>
</tr>
<tr>
<td>If the patient has low analgesic requirements, then a decrease in the size of the bolus dose should be tried.</td>
<td></td>
</tr>
<tr>
<td>Non-opioid analgesia such as regular paracetamol and NSAID suppositories (if no contraindications such as lower GI surgery) should be added.</td>
<td></td>
</tr>
<tr>
<td>Individual patients may be more sensitive to a particular opioid. If other measures have failed, then it is worth considering a change to a different opioid. However, remember that opioids are not the sole cause of post-operative vomiting.</td>
<td></td>
</tr>
</tbody>
</table>

### Pruritus

<table>
<thead>
<tr>
<th><strong>Pruritus</strong></th>
<th>7.13.4.7.4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pruritus may be caused through both histamine release and u-receptor activation. It is more commonly seen with Morphine that Fentanyl. In the hospital setting, pruritus is most often caused by lying on a plastic covered mattress! (pruritus is present on the back only).</td>
<td></td>
</tr>
<tr>
<td>If the distribution of the pruritus is on the face and trunk, then it is likely to be due to the opioid.</td>
<td></td>
</tr>
<tr>
<td>Treatment options are:</td>
<td></td>
</tr>
</tbody>
</table>
Change the opioid. E.g. Morphine to Fentanyl.
Titrate small (50-100 mcg) doses of Naloxone.
Antihistamines—but these may lead to sedation and respiratory depression.

**Sedation and Respiratory Depression** 7.13.4.7.5

These are unusual with a “pure” PCA-i.e. with no background infusion, with an appropriate size of bolus dose and no additional sedative agents.

If a patient does have a sedation score of -1 (constantly or frequently drowsy-falls asleep during conversation but easy to rouse), then a reduction in bolus dose (usually by half) and ceasing any background infusion is indicated.

If the patient has a respiratory rate of 8 or less, in addition to sedation score of -1, then titration of Naloxone in 100mcg doses should be considered, in addition to the above.

If the patient has a sedation score of -2 or less (somnolent, difficult to rouse) then Naloxone should be administered regardless of the respiratory rate.

Problems not infrequently arise in the opioid dependent patient, who becomes sedated, yet complains of high pain scores. It should be explained to these patients that they cannot be safely given more opioid, and that complete pain relief may be an unrealistic aim for them.

Addition of non-opioid pain relief may be of value.

**Urinary Retention** 7.13.4.7.6

Urinary retention can occur, and should be treated with catheterisation. Be wary of the possibility of sedation / respiratory depression in the patient who has been treating the pain of a distended bladder with their PCA, who then has the pain relieved by a catheter.

**Inhibition of Bowel Motility** 7.13.4.7.7

To some extent, inhibition of bowel function is inevitable. Patients should be discouraged from using their PCA to treat “wind” pains.

**Hypotension** 7.13.4.7.8

Opioids will not in themselves cause hypotension, but may unmask hypovolaemia (through a reduction in sympathetic tone).

**Guidelines for Performance of Sensory Evoked Potentials** 7.14

**Introduction** 7.14.1

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** 7.14.2

Test 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** 7.14.3

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** 7.14.4

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 dural 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** 7.14.5

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.

**References**


[Precautions

[Recording

[Preparations

[Interpretation

[References]
**Introduction 7.15.1**

Referral and transport of a patient to the MRI scanner should be initiated by the duty ICU specialist, 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 7.15.1.1**

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 to that required in a scrapyard 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 7.15.1.2**

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 Codman ICP monitors and PiCCO catheters are considered unsafe in the MRI and may have to be replaced or removed.

**Procedure 7.15.2.1**

Complete patient consent issues and MRI risk screening document.

**In ICU 7.15.2.1.1**

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 3 × 2 m rigid extension sets, which are coiled with micropore (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 active 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 7.152.1.2**

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

**In the MRI 7.15.2.1.3**

- 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.
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