



CRITICAL AND ACUTE CARE MODULE

Intensive Care

Making the most of your time in ICU
as a trainee intern

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Welcome to ICU!

By the end of your run my only goal is to make you think that ICU is really cool, or at least understand what we do.

Hopefully you also get a bit more of an idea about what we do, and how ICU fits into the journey of a critically unwell patient.

Nobody is expecting you to become expert in the management of multi-organ failure, but seeing these patients at their most unwell will give you context for assessing them elsewhere in the medical world.

Four days is a very short time to do this, so this document aims to give you a brief overview of what you can expect to see, as well as some ideas of how you might use your time productively.

Any suggestions for how we can improve the run are very welcome and I am happy to be contacted -
samuel.talbot@ccdhb.org.nz

Hope you enjoy your time with us!

Sam Talbot

ICU education fellow



The Department

Our ICU is on level 3 of Wellington hospital, located opposite the theatre complex. The HDU is located on level 5, but you will probably not spend much time there.

We have 3 “pods”, each with an allocated long day registrar and consultant of the day:

- North: beds 8-16
- Central: beds 1- 7 and 17-18
- South: beds 19-24



The duty intensivist (DI) is responsible for all patients in the unit, accepting/declining referrals, and arranging flight transfers. We also have a number of other fellows and registrars on any given day, whose duties include flights, transfers, non-clinical work, MET calls etc.

Each patient has a primary nurse, and each pod has a senior nurse co-ordinator. The ACNM-ops is responsible for the overall running of the unit. There are also other specialty ACNMs, nurse educators, resource nurses, technical nurses, flight nurses, PAR nurses and research nurses who you will also meet.

Our allied health teams include speech and language therapists, physiotherapists, occupational therapists, social workers, dieticians, clinical support workers and more.

We have an amazing non-clinical administration team who manage the medical and nursing teams, and our joint clinical directors are Paul Young and Alex Psirides, both ICU consultants in the department.

The consultants, ACNMs and administration staff all have offices in the non-clinical corridor at the south end of the department. There is also a staff kitchen which you are welcome to use, as well as our large seminar room (Maraenui) where handover and teaching usually takes place.

Scrubs are available in the theatre changing rooms opposite the ICU. Unfortunately we do not have enough lockers to provide you storage for your belongings, but these can be left in the corridor at the back of the staff kitchen atop the registrar lockers.

The ICU routine

08:00

Morning handover (Maraenui seminar room)

~ 08:30

Ward round (ICU pods x 3)

11:00

Radiology meeting (Radiology seminar room L2)

16:00

Afternoon ward round (starts ICU bed 19)

20:00

Evening handover (Maraenui seminar room)

~ 20:30

Night ward round (starts ICU bed 19)

Tuesday and Friday 11:30

Infectious disease/micro round (ICU fishbowl)

Thursday 12:00 - 15:00

Registrar teaching (Maraenui seminar room)

Friday afternoon

ICU consultant meeting/weekend handover

Trainee intern shifts

You will be rostered to either a dayshift (08:00 - 17:00) or evening shift (14:00 - 22:00).

Your best bet is usually to find the “flight doctor” (see roster in non-clinical corridor or in the ICU fishbowl. This person is a registrar who usually carries the MET phone, and sees referrals.

During your shifts you can expect to:

- Join ward rounds, review investigations, examine patients, write notes
- Attend Medical Emergency Team (MET) calls
- Perform or observe appropriate clinical procedures
- Participate in handover of patients
- Join in with registrar teaching and ICU meetings

Given the unpredictable nature of ICU, there will be things of interest or opportunities that you may want to stay and see through to their conclusion (such as an aeromedical retrieval or trauma call). You are more than welcome to do so; these can be great learning opportunities.

You should also think about your own safety/wellbeing. We expect you to have adequate rest (minimum 8 hours) between shifts - i.e. if you stay late and finish at midnight, don't worry about coming in for 8 am handover.

If you are unwell or will be late for any reason, please let us know.

Flights

The Wellington ICU team is also responsible for providing the region's inter-hospital flight transfer and retrieval service.



The duty intensivist and the nursing flight co-ordinator work together to arrange these flights and the service is delivered by the flight nurses and the Life Flight team. Transport of ICU level patients will usually have a doctor as well, and there are dedicated flight shifts on the registrar roster.

You are absolutely welcome to join the flight team should the opportunity arise, but unfortunately there may be reasons why this is not possible (e.g. family needing to travel with the patient).

If you are keen to fly, then speak to the duty intensivist in the morning and let them know. You are most likely to be able to go on a “nurse-only” fixed wing transfer, but very occasionally TI's do manage to get out on the helicopter or more unstable patient transfers!



Do also consider your own wellbeing: flying is great fun but obviously Wellington conditions are changeable and there can be lots of turbulence.

The fixed wing cabin pressure is variable, and the helicopter is not pressurised - what this means is that if you have a sinus infection or similar, you should not fly. If you have bad travel sickness, otherwise feel unwell, or are worried in any way about your ability to fly, please think seriously about whether you really want to and how your presence could affect the patient and flight team if you became unwell.

Also, many flights return late - see previous advice about



ensuring adequate rest.

PAR team

The “patient at risk” or PAR service is not a part of ICU per-se, but many of the team are senior ICU nurses, and they serve as a valuable resource for highlighting deteriorating patients and following up on patients who have recently been stepped down from ICU.

You are not specifically allocated to work with the PAR nurses, but will meet them at MET calls and would be a good person to spend time with if the unit is quiet.

Referrals

ICU referrals will either come directly to the DI from the primary specialty registrar or consultant; to the registrar holding the MET phone; from MET or trauma calls.

These can be really good opportunities to review an unwell patient, in whom there is often still some diagnostic uncertainty, so you should try to join wherever possible.

It is good to think about what information is useful to the ICU team when receiving referrals, and you will see what goes into the decision making about admitting or not to ICU.

MET Calls

New Zealand Early Warning Score Escalation Pathway

ZONE	Indicator		Mandatory Action	SCORE
YELLOW	Total EWS 1-5	➡	Manage pain, fever or distress. Increase frequency of vital sign monitoring	1
ORANGE	Total EWS 6-7 Acute illness or unstable chronic disease	➡	Discuss with nurse in charge and inform PAR nurse. Increase frequency of vital signs monitoring.	2
RED	Any vital sign in the red zone or total EWS 8-9 Likely to deteriorate rapidly	➡	Registrar review within 20 minutes & consider ICU referral. Inform PAR nurse, house officer and nurse in charge. Increase frequency of vital signs monitoring.	3
BLUE	Any vital sign in the blue zone or total EWS 10 or more Immediately life threatening critical illness	➡	Dial 777, state 'Medical Emergency Team' & give your location. Support Airway, Breathing & Circulation	MET

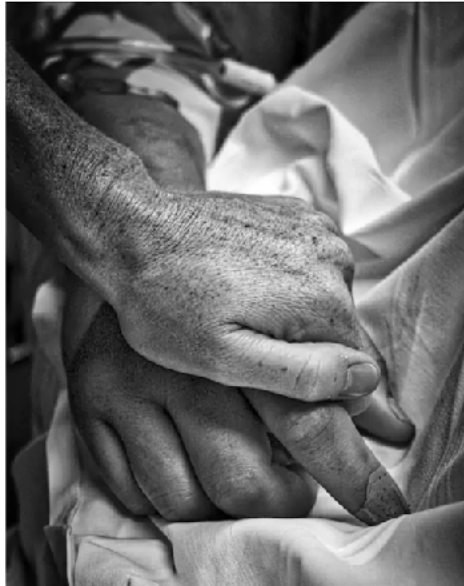
One of the ICU registrars carries the “medical emergency team” or MET phone, and will attend emergency calls throughout the hospital. These are often great learning opportunities and you should try to attend these where possible - make yourself known to whoever is carrying the phone and they will let you if the phone goes off.

Many of our emergency admissions come from MET calls, and even those patients that we do not admit provide good learning opportunities: initial assessment and stabilisation of an unwell patient; assessing and managing risk of deterioration/differentiating between the stable and unstable patient; discussing goals of care and setting treatment limitations; working with other specialties and communicating in an emergency scenario.

Death and dying in ICU

Patients in the intensive care unit are inherently at high-risk of morbidity and mortality - about 8% of patients will die during their ICU admission.

A big part of intensive care is shared decision making with patients and their relatives to determine what therapies and interventions are appropriate (i.e. likely to achieve an acceptable outcome for the patient).



Sometimes patients will die with ongoing maximal intensive care treatments, but this is not usually the case. If we do our job well, very few patients die in ICU unexpectedly; instead we try to spot deterioration early and if this is occurring despite maximal intervention

(depending on earlier shared goals of care), to recognise that the patient is dying, and usually to shift to a focus on providing a comfortable and dignified death.

You are likely to see parts of this journey during your run, including: goals of care discussions; discussions about withdrawal of life-sustaining treatment; palliative care reviews; and patients who are in the last hours to days of life.

Everybody's response is different, and all are equally valid. Some people will find a particular situation very upsetting, and others will not. It may also take a bit of time to process your response to these events and your feelings may change. This is all normal and everybody you work with is likely to have felt the same at some point.

If you are worried about yourself (or anybody else) after a challenging experience, you are very welcome to speak to anybody in the ICU or university team.

In Wellington ICU, the consultants are all very approachable and would be happy to speak in private about both the medical specifics of a case or your emotional response to it.

We also have a clinical psychology team Amie and Annabel who, though very busy with other duties as well, are an excellent team, to speak to at our weekly case discussions focusing on the non-medical aspects of challenging cases.

Organ donation

It is worth mentioning that organ donation is a unique and very important part of intensive care, and is something that you will very rarely encounter in other specialties.

If there is a patient proceeding to organ donation whilst you are with us, this can be a very interesting experience to be involved in, and you are encouraged to attend discussions and observe the process.

The ODNZ website has lots of interesting and useful information about this:



Organ Donation New Zealand
New Zealand's official donor agency

Raising concerns

You are an important part of the team during your ICU run, and we want you to feel included and welcome.

Any concerns about bullying, incivility or being made to feel unwelcome should be escalated and will be taken seriously and you should contact Amanda Collins or Tessa Ramsden, or speak to any of the ICU consultants.

Presenting a patient

We are really keen that you feel part of the team and a good way to get involved is to find a patient to present on the afternoon or evening ward round.

This is not a test! You can make notes and refer to them throughout, and you can ask for help if the patient's issues are complex.



Speak to the patient and their family/Whānau if able and ask how they are finding ICU and elicit any concerns - this can be extremely helpful and is sometimes overlooked.

Speak to the primary nurse - they will know all about the patient and will be able to help talk you through their chart/notes

As a suggestion, you might want to structure your handover something like this (but everybody is different!):

- Patient details and relevant background
- Reason for admission to ICU and how many days they've been here
- Main issues
 - Primary diagnosis
 - Complications/secondary issues
 - Depending on how sick they are, a systems based approach can be useful
- Plan from the morning ward round and how they have progressed during the day
- Expected trajectory over next shift and any anticipated problems/outstanding tasks.

For example:

"This is Brian, a 73 year old who is in the ICU after elective cardiac surgery. He has had worsening angina for 6 months and was found to have 3 vessel disease on angiography, and has had a CABG x3 2 days ago which was fairly straightforward. Yesterday he had some issues with low blood pressure, needing noradrenaline and vasopressin, but this has improved and he is not on any blood pressure support any more.

On this morning's ward round our impression was that he is making good progress and there were no new issues. Our plan was to remove his drains, which has been done, and to continue as per the elective cardiac pathway. He has sat out in a chair and his pain is well controlled with sevredol. He feels he is doing well and had no concerns.

If he continues to progress well, he should be ready to step-down to the ward tomorrow, and there are no specific concerns for overnight. Please could somebody order and review his post-drain removal chest XR?"

Often the consultant leading the ward round will ask for a bit more information or want to clarify a specific point - for a complex patient this might not be something you know the answer to, and it is absolutely OK to say so - this is much better than guessing or making up an answer if you are unsure!

Be sure to allow the bedside nurse an opportunity to contribute to your handover or raise any concerns that they might have as they will know the patient better than anyone - it can be useful to ask them about this directly at the beginning or end of your handover if you haven't already.

If you require anything to be signed off for your patient presentation, the consultants will all be happy to do this, but it is useful to let them know in advance so that they can focus on giving you helpful feedback, rather than waiting until after the ward round.

What to do when the unit is quiet

- Ask the DI if there are any flights you could join
- Check in with the MET phone registrar
- Join the PAR nurse (#6785)
- Ask the bedside nurses about what they are doing and what supports the patient is receiving
- Ask the registrar or consultant about their favourite ICU topic/why they got into ICU/anything really!
- Speak to one of the allied health team members about their role in ICU and how they help patients recover
- Speak to a patient or their family about how their ICU stay has been. What have they found hard? What has the team done well?
- Ask someone about one of the *suggested topics in red* from the rest of this document

Useful resources

Wellington ICU RMO resources

<https://wellingtonicu.com/pro/rmoresources/>

Internet book of critical care

<https://emcrit.org/ibcc/toc/>

Life in the fast lane

<https://litfl.com/>

Deranged physiology

<https://derangedphysiology.com/main/home>

The Alfred ECMO guidance

<https://ecmo.icu/#menuRoot>

National tracheostomy safety project

<https://tracheostomy.org.uk/>

Organ Donation New Zealand

[https://www.donor.co.nz/healthcare-professionals/
intensive-care-unit-guidelines/](https://www.donor.co.nz/healthcare-professionals/intensive-care-unit-guidelines/)

Basics of intensive care medicine

A whole specialty is a lot to cover in a short run.

This guide aims to give you a bit of an intro to the ICU specific interventions and common problems that you might encounter so you can hit the ground running!

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Suggested learning/discussion points are highlighted like this

Don't worry about finding out the answer to every single one! They are just there to give you some prompts about discussions you could have with the ICU team if you encounter a specific scenario and are interested to learn a bit more about ICU.

Who needs ICU?

This is a complex question and although it is one we ask multiple times per day when taking referrals, there is not really a one-size-fits-all answer.

The key difference about an intensive care unit is really how closely we can watch a patient - ICU patients will have 1:1 nursing 24 hours per day, and HDU patients usually 2:1. Additionally, there is a team of medical staff whose sole responsibility is looking after a relatively small number of patients and are generally quite skilled in emergency management of most problems. Most patients will also have enhanced physiological monitoring to allow earlier detection of deterioration, which ranges from continuous SpO₂ and ECG to invasive devices like pulmonary artery catheters and intracranial pressure monitors.

All of this observation allows us to avoid intervening longer than would be possible in a less resourced environment, and relatively often, we bring patients to the ICU and then don't do very much different to what would happen on the wards.

You will probably hear during your time with us, the secret to intensive care medicine is that most things will get better if you leave them alone, and often patients just need a bit more time: e.g. for antibiotics to work, for their kidneys to recover, or their bronchospasm to resolve.

The other main difference is in the treatments that we can offer - we can provide organ support in the form of

artificial airways, mechanical ventilation, vasoactive medications, renal replacement therapy and more. These all help to treat life threatening organ failure, and reduce the chance of things getting worse, but fundamentally they are very rarely fixing the underlying problem.

The key point here is that the underlying problem needs to be one that we expect to get better, and that most of what we are doing in ICU is buying time for that to happen. If a patient has a progressive, chronic illness for which there is no reversibility (e.g. organ transplant), then ICU care is unlikely to benefit them.

We frequently treat exacerbations of chronic conditions (COPD, heart failure, diabetes etc) but try to recognise when that person's long term health has deteriorated to a point at which they are unlikely to recover to a level acceptable to them, and avoid giving them inappropriately aggressive/invasive treatments.

The potential harms of ICU should not be underestimated: all of our procedures and medications carry risk, sedation or delirium significantly hamper patients' involvement in discussions about their care, and ICU patients are inherently less mobile due to all of the devices etc that they acquire and are at increased risk of infection, VTE and pressure sores.

To summarise then, we try to admit patients who have established, or are at risk of, organ dysfunction as a result of a reversible primary problem in whom the expected benefit of ICU treatments outweighs the potential harms associated with it.

Airways

Many ICU patients will have an “artificial” airway. This can be for a number of reasons:

- overcome a primary airway obstruction
- provide (and subsequently wean from) invasive mechanical ventilation for respiratory/neuromuscular failure
- “take control” in an unstable patient of any cause
- facilitate anaesthesia for a procedure or operation
- protect the lungs from aspiration in an unconscious patient

What are some causes of airway obstruction?

What might be some red flags for an anatomically difficult airway?

How would an airway plan change in the presence of these red flags?



Basic airway manoeuvres

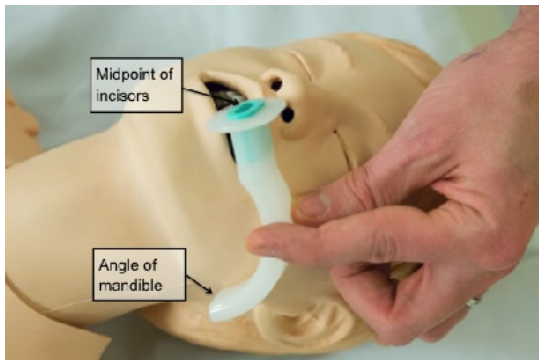
A key skill we hope you develop during your EAC module is to recognise and initially manage an obstructed airway.

Basic airway skills are the backbone of any airway plan and these are what keeps the patient alive long enough for an airway expert to secure the airway more definitively!

You will be taught about these elsewhere in the module, but the key skills that you would be expected to be comfortable with when managing an obstructed airway are:

- gentle suctioning of the oropharynx
- head tilt/chin lift
- jaw thrust
- oropharyngeal and nasopharyngeal airway insertion
- 2 person bag-valve-mask ventilation

A key
any

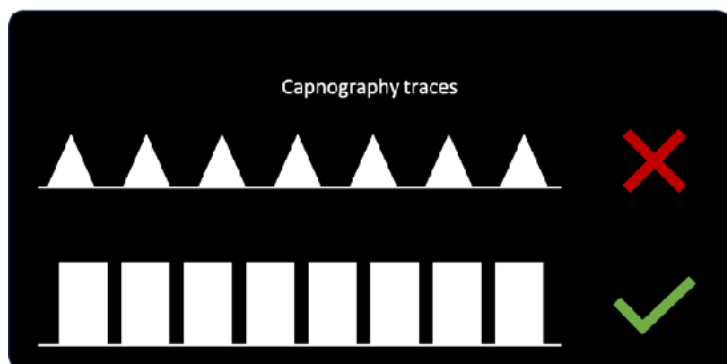


part of
airway

intervention is to re-assess whether you have achieved airway patency - a good jaw thrust is better than a badly seated LMA or misplaced endotracheal tube.

Signs of airway patency are:

- improvement of any airway noise or “see-saw” breathing in a spontaneously ventilating patient (note a completely obstructed airway will not generate noise!)
- misting of the airway device
- chest rise and fall
- “top-hat” end-tidal CO₂ trace



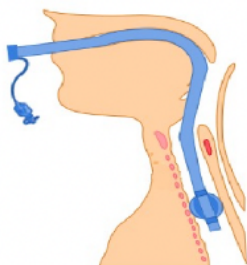
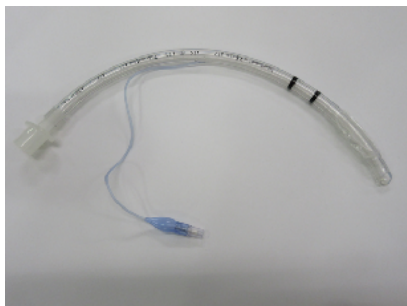
End-tidal CO₂ is an integral part of airway management and is widely available now with portable small devices like the EMMA capnograph being available.

NB: Oxygen saturations are not a marker of airway patency! It can take some time for these to change, so if you are worried about an airway, get to work before the sats drop.

What other information does the ETCO₂ trace give us?

Endotracheal tubes

Most intubated patients will have a cuffed oral endotracheal tube (COETT). The tip of the tube sits below the level of the vocal cords and above the level of the carina (endotracheal) and has a cuff a couple of centimetres from the end to form a seal with the tracheal walls. The cuff is inflated via a small tube connected to a “pilot balloon” at the proximal end. There is also a hole in the distal tube called the “Murphy’s eye” to prevent occlusion of the entire tube should the end become blocked or abut the trachea. The tube has centimetre markings to indicate the depth of insertion and a size in millimetres referring to its external diameter.



The purpose of the cuff is to allow positive pressure ventilation (preventing gas from leaking back out the upper

airway under pressure) and to (imperfectly) protect the airway from aspiration.

Occasionally patients will have another type of ETT in situ but you are unlikely to see these in ICU (e.g. nasal ETT)

Find the airway trolley in ICU

What are some causes of hypoxia in an intubated patient?

Rapid sequence intubation

Intubation in the critically ill is a high-risk event - you are taking a patient who is already unwell and giving them an anaesthetic that will further obtund their airway reflexes, stop them breathing and potentially impair their cardiac output. Furthermore you are doing this in an ICU bedspace which tend to have a lot of infusions/other equipment and with a patient who may be agitated and uncooperative.

As such, we try to make the situation as controlled as possible using a checklist so that the whole team is aware of the plan and any potential risks.

Look at the RSI checklist in ICU

What factors make ICU patients more at risk of aspiration, hypoxia and hypotension during intubation than an elective surgical patient?

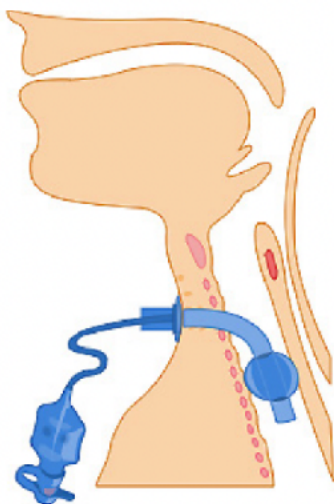
How might we overcome these risks?

Tracheostomy

In some patients, a tracheostomy will be used:

- anticipated or unanticipated airway difficulty requiring surgical tracheostomy
- weaning from invasive ventilation after a period with COETT in a patient who is too weak to be extubated
- ongoing airway protection in bulbar weakness

Tracheostomy insertion is more invasive than intubation and is performed either percutaneously in ICU using a Seldinger (needle-wire-tube) technique or in theatre by ENT surgeons using a surgical technique. In both cases a passage is created between the anterior neck and the trachea and a tube placed there to maintain its patency.



What might be some indications for a surgical tracheostomy rather than a percutaneous one?

Tracheostomy complications can rapidly develop into true emergencies, particularly if patients are in areas with limited airway experience. This has been the focus of a lot of safety and quality improvement within ICU and there are some excellent resources online:

- <https://www.globaltrach.org/>
- <https://tracheostomy.org.uk/>

What complications may arise in a patient with a tracheostomy?

How would you manage a patient whose tracheostomy tube has become dislodged?

Look at the NTSP tracheostomy emergency algorithm

How does a laryngectomy differ from a tracheostomy?

Breathing

Very simply, breathing in ICU comes down to two variables: oxygenation and ventilation; and everything we do is aimed at optimising these.

Oxygenation

Simply put, we can increase the number of oxygen molecules we breathe in; maximise the surface area over which these can diffuse; and reduce the amount of stuff in the way.

What this translates to is:

- give increased fraction of inspired oxygen/ FiO_2
- use of positive end-expiratory pressure (PEEP) or continuous positive airway pressure (CPAP) to maintain alveolar recruitment
- suctioning, position changes and chest physiotherapy to avoid mucous plugging and collapse
- proning to improve ventilation/perfusion matching
- therapies targeted at the underlying pathology (e.g. diuretics for pulmonary oedema, antibiotics for pneumonia)

How does PEEP help in cardiogenic pulmonary oedema beyond recruitment of alveoli?

FiO_2

When considering FiO_2 , the device and patient's work of breathing matters. Think about these two patients

- a patient breathing comfortably in PACU via a Hudson mask at 4L/min
- a patient with severe pneumonia has a RR of 30 and a much higher work of breathing on a Hudson mask at 4L/min

The comfortable patient has a low peak inspiratory flow rate (PIFR) and they don't entrain much room air, so the overall oxygen concentration of the gas they inspire (FiO_2) is quite high. The patient with pneumonia has a much higher PIFR (over 10x the flow via the Hudson) and will entrain lots of room air, diluting the FiO_2 .



Devices that overcome this are:

- **Non-rebreathe/reservoir systems:** the oxygen filled reservoir allows the patient to take a maximal breath and entrain almost pure oxygen, provided the bag remains full! There is no point in putting one of these on at a lower flow rate.
- **Venturi masks:** these have a set diameter of air inlets which at a set oxygen flow rate entrain air to dilute the inspired mixture to a pre-determined FiO_2 . Again these must be at the set flow rate: too low and the FiO_2 will be less; higher flow rates won't increase the FiO_2

proportionally as the device still allows a large entrainment of air.

- **High flow oxygen:** either nasal or facemask delivery systems. By providing flows close to or greater than the PIFR (e.g. 40-60L/min), the amount of entrained air is minimised. The FiO_2 can be adjusted by the machine using a mixture of oxygen and air.

PEEP and CPAP

These two concepts are the same, with PEEP referring to invasive ventilation and CPAP usually reserved for non-invasive ventilation.

The aim of both is to prevent alveolar collapse at end-expiration, and so to increase the available surface area for gas exchange. They also increase the alveolar pressure, and can help to promote fluid re-absorption in pulmonary oedema by reducing the pressure gradient into the alveolus.

CPAP also has a role in preventing upper airway collapse in obstructive sleep apnoea, and so has a role in improving ventilation as well.

What factors increase the likelihood of alveolar collapse and therefore which patients need higher PEEP when intubated?

Proning

For patients with severe respiratory failure, we can “prone” them to improve ventilation/perfusion matching.

How does proning help V/Q mismatch?

Ventilation

Alveolar minute ventilation is ultimately what matters (i.e. the volume of fresh gas mixture that reaches the alveoli over a minute), as this is the gas that takes part in gas exchange and clears CO₂.

This is dependent on the respiratory rate and tidal volume (minus the dead space gases that are not participating in gas exchange), so we can alter each of these parameters to improve ventilation.

Again, to keep it simple, we can improve someone's ventilation by:

- Reducing the resistance to their own work of breathing
- Increasing their own respiratory drive
- Providing pressure support to their breaths
- Fully taking over their breathing with mandatory ventilation
- Reducing deadspace

Airway resistance

A narrow airway has higher resistance to flow and a patient will have to work harder (to generate a greater pressure gradient) to overcome this, potentially leading to reduced ventilation if they tire.

If we can increase the diameter of the airways, this will improve ventilation. In practice we give bronchodilators to achieve this, both inhaled/nebulised and intravenous.

Which drugs can be used via the intravenous route as bronchodilators?

Increasing respiratory drive

In practice, this only really applies to patients with opioid toxicity or similar, where reversal of the drug effect increases their sensitivity to CO₂.

Positive pressure ventilation

If the patient has a patent airway and we can achieve a good seal with a breathing circuit (either via facemask or airway device), we can provide positive pressure ventilation.

Non-invasive ventilation (NIV) generally refers to bi-level support via a tightly fitted facemask, in which the ventilator is set to deliver both an inspiratory and expiratory pressure, usually synchronous to the patient's own breathing.

Invasive positive pressure ventilation (IPPV) requires a cuffed airway device (ETT or tracheostomy) and can be delivered using various modes. In Wellington ICU, we use a mode called AMV (automatic mechanical ventilation) which is a brand name for a hybrid mode that basically lets the ventilator decide how best to ventilate the patient.



In both NIV and IPPV the ventilator generates pressure which creates a gradient and drives gas into the lungs. It also measures flow in and out of the lungs and calculates the volumes delivered. We can supplement the patient's own breaths (by detecting negative or inspiratory flow and synchronising with this) or fully take over ventilation if the patient's own effort is inadequate.

What is negative pressure mechanical ventilation?

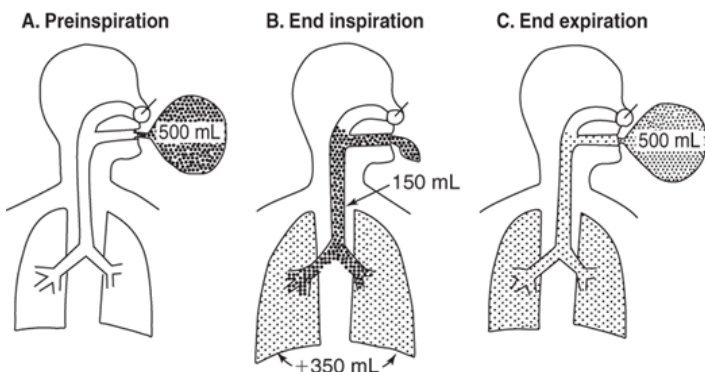
What modes of IPPV do you know about?

What are some relative contraindications to NIV?

What are some complications of positive pressure ventilation?

Deadspace

Think of taking a 500ml breath in: about 150ml of this does not participate in gas exchange as it is just going in and out of the upper or conducting airways (mouth/nose/trachea/bronchial divisions) where there are no alveoli. This is anatomical deadspace.



Think of the following two patients with the same alveolar minute ventilation:

- 10 x 600ml (minus 150ml deadspace) breaths per minute = 4500ml alveolar minute ventilation
- 30 x 300ml (minus 150ml deadspace) breaths per minute = 4500ml alveolar minute ventilation

Which of these patients is working harder and is more likely to become tired and hypoventilate?

If we connect a patient up to lots of extra breathing circuit apparatus (e.g. 200ml), this equipment deadspace exacerbates the problem: instead of needing to take a 500ml breath to achieve 350ml alveolar ventilation, they now need to take a 700ml breath, which requires more work to achieve.

This is particularly an issue in children, in whom anatomical and equipment deadspace is proportionally larger compared to their smaller tidal volumes. To overcome this, we minimise the length of ventilator tubing, extensions, number of filters and nebulisers etc. in the circuit.

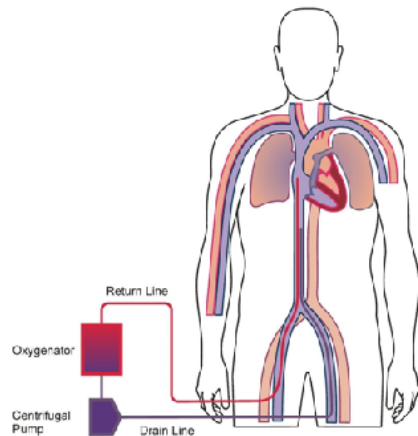
In a patient with respiratory muscle weakness, one of the benefits of a tracheostomy is that it bypasses a significant portion of the upper airways and reduces anatomical deadspace, and reducing the work needed to achieve the required alveolar ventilation.

A note on ECMO

Ultimately if someone has really severe respiratory failure and is on maximal conventional supports with refractory hypoxaemia or hypercapnoea with severe respiratory acidaemia, they may be offered V-V ECMO as a bridge to survival or occasionally lung transplant.

This is co-ordinated in NZ out of CVICU in Auckland and it is rare that patients are commenced on this treatment in Wellington. The key factor in the decision to provide ECMO is how reversible the underlying problem is, as the procedure is not without risk and does nothing to fix the underlying lung pathology, only allowing us to avoid further harming the lungs with excessive positive pressure ventilation.

The Alfred ICU in Melbourne has some excellent information about both V-V and V-A ECMO: <https://ecmo.icu/#menuRoot>



Circulation

Lots of patients in the ICU have circulatory dysfunction even without overt shock, and the cause is often multifactorial.

For example we often see hypotensive post-cardiac surgery patients who have a bit of bleeding, impaired LV function, and vasodilation who require a balanced approach to fix each problem.

Fluid therapy

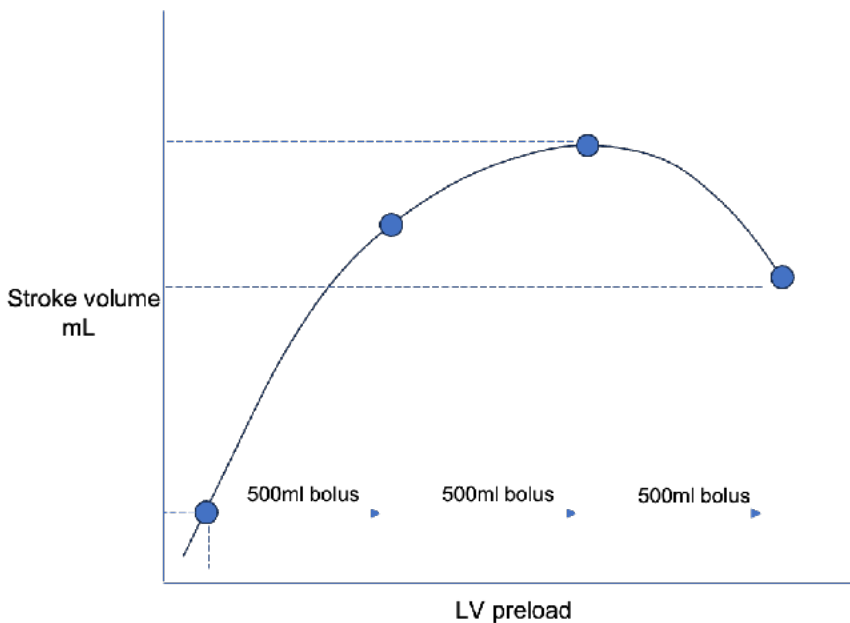
By the time most patients are admitted to the ICU, they have usually received quite a lot of fluid. With all of the infusions of drugs that they receive during their admission, it is not usually necessary to administer more fluid after the initial resuscitation phase, in fact we often need to give diuretics once their shock starts to improve to offload the extra fluid.

0.9% saline is a reasonable choice of fluid in small amounts, but when given in large volumes it increases AKI and incidence of hyperchloraemic metabolic acidosis, so why not use something that does the same job better?

In general we advocate giving a balanced crystalloid (Plasmalyte or Hartmann's/Ringer's lactate solution) for most indications, with the exception of patients with brain injury in whom 0.9% saline is the preferred crystalloid to prevent cerebral oedema.

The aim of a fluid bolus is to improve left ventricular preload and hence stroke volume and cardiac output. As such, a fluid bolus for a shocked patient should be administered rapidly and its effect on organ perfusion immediately assessed following completion.

To achieve adequate flow rates to give a decent fluid bolus (250-500ml over <15mins), the patient needs adequate IV access +/- a pressurised system (pressure bag, hand pump, 50ml syringe and 3-way tap, rapid infusion device).



Improvement in blood pressure or markers of perfusion following a fluid bolus is reassuring. Failure to improve suggests that the patient is near the apex of the Frank-Starling curve (see above) and further fluid is likely to do more harm than good.

What are some non-invasive predictors of fluid responsiveness?

What factors affect flow rate through a cannula?

Vasoactive drugs

A cornerstone of ICU is use of vasoactive drugs: technically this refers to any drug which acts in a positive or negative way to affect the cardiovascular system; in practice we usually mean drugs that act to increase blood pressure and/or cardiac output.

What are the determinants of systemic blood pressure?

How does blood pressure relate to organ perfusion?

Broadly speaking you will hear us refer to vasopressors, inotropes and chronotropes. In reality most drugs have more than one action, and even a pure vasopressor (e.g. phenylephrine) will also improve cardiac contractility through increased venous return and the Frank-Starling mechanism.

To keep things simple, however, this is a summary of vasoactive drugs and their actions (from the excellent Internet Book of Critical Care website - although note we use a lot of dopamine!):

Rough properties of various vasopressors

Drug Typical dose range	Target	Effect on - Heart rate - Inotropy - Ectopy	Effect on systemic vascular resistance	Effect on cardiac output	Effect on blood pressure	Effect on pulmonary vascular resistance	Main uses	Safe for peripheral use?
Inodilators								
Dobutamine 2-20 mcg/kg/min	$\alpha\beta\beta\beta$	$\uparrow\uparrow\uparrow$	$\downarrow$	$\uparrow\uparrow\uparrow$	Variable	$\downarrow$	Cardiogenic shock	
Milrinone 0.375-0.75 mcg/kg/min	cAMP	$\uparrow\uparrow\uparrow$	$\downarrow\downarrow$	$\uparrow\uparrow\uparrow$	Variable	$\downarrow\downarrow$	Cardiogenic shock	
Isoproterenol 2-10 mcg/min	$\beta\beta\beta\beta$	$\uparrow\uparrow\uparrow\uparrow$	$\downarrow$	$\uparrow\uparrow\uparrow$	Variable		Bradycardia	Yes
Pure Vasopressors								
Vasopressin 0.01-0.06 U/min	V1 & V2	$\downarrow$	$\uparrow\uparrow\uparrow$	$\leftrightarrow/\downarrow$	$\uparrow\uparrow\uparrow$	$\downarrow$	Distributive shock, Pulmonary HIN	No.
Phenylephrine 40-180 mcg/min	$\alpha\alpha\alpha\alpha$	$\downarrow$	$\uparrow\uparrow\uparrow$	Variable	$\uparrow\uparrow\uparrow$	$\uparrow\uparrow$	Distributive shock	Yes
InoPressors								
Norepinephrine 0-40 mcg/min* AKA noradrenaline	$\alpha\alpha\alpha\beta$	$\uparrow$	$\uparrow\uparrow\uparrow$	$\leftrightarrow/\uparrow$	$\uparrow\uparrow$	$\leftrightarrow$	Shock (most types)	Yes, for short period with monitoring
Epinephrine 0-20 mcg/min* AKA adrenaline	$\alpha\beta\beta\beta$	$\uparrow\uparrow\uparrow$	$\uparrow$	$\uparrow\uparrow\uparrow$	$\uparrow\uparrow$		Bradycardia, cardiogenic shock, sepsis, anaphylaxis	Yes
Dopamine, low 1-4 mcg/kg/min	Dopa-R	$\leftrightarrow$	$\downarrow$	$\uparrow$	$\downarrow$		Zombie apocalypse (absence of better agents).	Probably not
Dopamine, medium 4-10 mcg/kg/min	$\alpha\beta\beta\beta$	$\uparrow$	Variable	$\uparrow\uparrow$	Variable			
Dopamine, high 10-20 mcg/kg/min	$\alpha\alpha\alpha\beta$	$\uparrow\uparrow$	$\uparrow\uparrow$	$\uparrow$	$\uparrow\uparrow\uparrow$	$\uparrow$		

*Listed ranges are typically used doses in the United States, but there is no true "maximal" dose. Some countries may tend to use higher doses than others. At very high doses, pressors may lose some receptor specificity. The best dose is the dose required to keep the patient alive – in some cases very high norepinephrine or epinephrine doses may be needed.

-The Internet Book of Critical Care, by @PulmCm

Why does adrenaline increase lactate? Does this matter?

Invasive monitoring/access

Most patients who are critically unwell will have invasive arterial monitoring and probably some form of central IV access.

The role of the arterial line is primarily to give a continuous, accurate, beat-to-beat blood pressure reading to allow titration of vasoactive drugs. We can also sample from the line very frequently, such as ABG's during a major haemorrhage resuscitation. We usually insert an arterial

line in the radial artery either by palpation or using ultrasound guidance, using sterile ANTT.

Central lines are commonly inserted in critically ill patients to facilitate administration of concentrated drug infusions, as well as in situations where peripheral IV access is difficult and secure short-medium term access is required. We usually insert an internal jugular line, and use of real-time ultrasound guidance and full sterility are the accepted standard.

What are some short and longer term complications of central line insertion?

What are we looking for on a CXR after central line insertion?

We can also transduce a central line (which has 4-5 separate lumens) to measure central venous pressure. Whilst this is not a hugely reliable single parameter for haemodynamic monitoring, the trend of an increasing or decreasing CVP can give useful information.

As well as arterial and central venous access, you may come across other vascular devices in ICU:

- “Vascath” or “Quinton line” or temporary dialysis catheter: a large central line that has two large bore lumens to provide renal replacement therapy
- Pulmonary artery or “Swan-Ganz” catheter: used in high risk cardiac patients to invasively measure cardiac output and other haemodynamic parameters

- Peripherally inserted central catheter (PICC): medium term central IV access with less incidence of infection/complication than a CVC. One or two lumens.

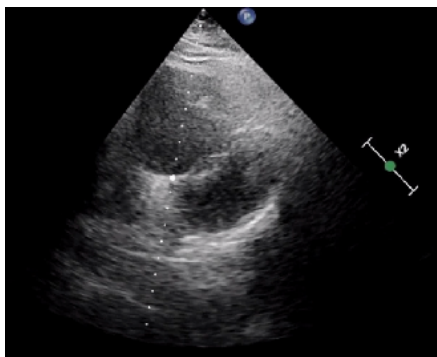
Echocardiography

Focused echocardiography (and other ultrasound protocols) are increasingly common in ICU and elsewhere in emergency and acute care - commonly referred to as point of care ultrasound (POCUS).

The benefit of POCUS is that it is a non-invasive, relatively rapid, repeatable and non-ionising examination; however, its limitations lie in its subjectivity, so adequate training and quality assurance is important.

Questions that even a very basic focused TTE can help answer include:

- is there severe LV or RV dilatation or impairment?
- are there significant pericardial or pleural effusions?
- is there evidence of significant valve or aortic pathology?



Massive RV dilation and impairment in PE



Pericardial effusion with early tamponade

Mechanical circulatory support

Patients with inadequate cardiac output despite maximal medical management may require mechanical support, which includes:

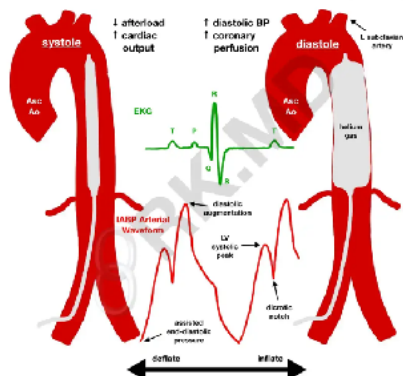
- Intra-aortic balloon pump (IABP)
- Impella devices
- Ventricular assist devices
- Veno-arterial (VA) ECMO (see previous section on VV-ECMO)

In our ICU you are most likely to see a patient with an IABP, and very occasionally may see a patient receive VA-ECMO. These are all invasive procedures that can be used for a limited duration, so they are limited to patients with relatively reversible pathologies, or who would be candidates for hyperacute heart transplant (e.g. VA-ECMO for fulminant myocarditis).

IABP is the most commonly used device and provides the least amount of support, but its primary role is to improve coronary perfusion during diastole and reduce LV afterload in systole. It is most commonly used in cardiogenic shock post MI.

A balloon catheter is placed in the proximal descending aorta where it synchronises to either the ECG or pulse waveform and inflates during diastole/deflates during systole.

INTRA-AORTIC BALLOON PUMP (IABP)



Central nervous system

No doubt you will hear about the “triangle of anaesthesia” during your EAC module - hypnosis, analgesia and muscle relaxation.

Think about a critically hypoxic patient: they will be agitated, with a high respiratory drive and likely heading towards intubation. Once they are on the ventilator, if we let the induction anaesthetic wear off, they would likely become agitated and “fight” the ventilator or potentially not tolerate their ETT.

As such, many critically ill patients receive some form of sedation and analgesia to facilitate uncomfortable and invasive interventions, and occasionally also require neuromuscular blockade .

Sedation

We try to minimise use of sedating drugs where possible, both in dose and duration of therapy, particularly for delirium where the patient is not a risk to themselves or others.

Even if a patient requires ongoing IV sedation, it is good practice to perform a sedation hold (i.e. pause sedation and allow it to wear off) if safe to do so to ensure that the drug does not accumulate and to re-assess the ongoing need for sedation.

Commonly used drugs are:

- **Propofol:**

- backbone of ICU sedation for intubated patients, can be given as a bolus and infusion.
- agonist at central GABA receptors.
- accumulates with prolonged infusions but not as much as other agents, so wears off relatively quickly.
- vasodilation and reduced myocardial contractility can lead to hypotension and suppresses airway reflexes/respiratory effort so not used by infusion for non-intubated patients

- **Dexmedetomidine and Clonidine:**

- Alpha-2 agonists, cause noradrenaline re-uptake in the CNS, reducing neurotransmission
- Provide some analgesia as well as sedation
- Useful for non-intubated patients or as a second-line agent for patients on maximal propofol.
- Main SE's are hypotension and bradycardia



- **Ketamine:**
 - NMDA antagonist
 - Used as induction agent, and by infusion for analgesia. Bolus can be useful for severely agitated patient or procedural sedation.
 - More CVS stable than propofol, main SE's are tachycardia and hallucinations

- **Antipsychotics:**
 - Dopamine antagonists: haloperidol/olanzapine/quetiapine
 - IV haloperidol for rapid sedation in severely agitated patient; oral agents for hyperactive delirium not responding to non-pharmacological methods.
 - pretty safe but need to be mindful of QT prolongation, dystonia and very rarely neuroleptic malignant syndrome

- **Midazolam**
 - Benzodiazepine, agonist at GABA receptor
 - Can be used as bolus for rapid sedation in severely agitated patient or at induction of anaesthesia, but accumulates significantly with infusion so is a third-line agent for continuous sedation

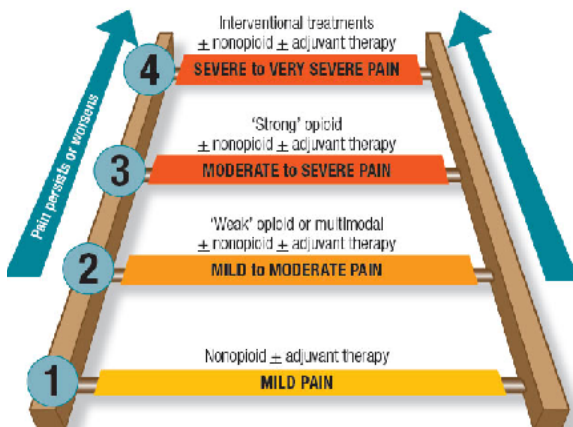
Analgesia

It is very important not to miss pain as a cause of agitation in a confused or sedated patient, and to provide analgesia rather than more sedation. Lots of ICU patients will be sore, either from their underlying problem, our interventions or simply from being in bed 24/7 and unable to easily change position.

As with any other acute pain patient, we use the WHO analgesic ladder, albeit with a few considerations due to their critical illness:

- reduced clearance/accumulation of active metabolites (e.g. avoiding morphine in AKI)
- more susceptible to nephrotoxic medications (e.g. avoiding NSAIDs in shocked patient)
- reduced absorption/no enteral route (e.g. ileus)

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receive a combination of paracetamol and strong opioid +/- adjuncts such as ketamine infusion, NSAIDs if appropriate, alpha-2 agonists or gabapentinoids.

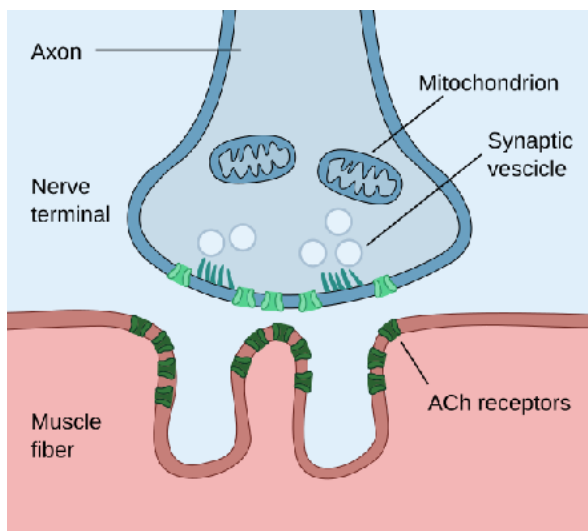
As well as systemic analgesia, we often use regional anaesthetic techniques for post-operative and trauma patients, e.g. nerve or fascial plane local anaesthetic catheters, epidurals.

Neuromuscular blockade

The most common indication for neuromuscular blocking drugs (NMBDs) in the ICU is to facilitate endotracheal intubation as part of an RSI.

Patients will also often return from theatre with residual neuromuscular blockade from their anaesthetic, and part of our admission process is to assess for this. Often it is desirable to rapidly turn off sedation and assess a patient's neurological status (e.g. post-neurosurgery) and this may necessitate administration of a reversal agent.

Our most commonly used NMBD is rocuronium, which is a non-depolarising antagonist at the post-synaptic acetylcholine receptor in the neuromuscular junction.



There are few patients in the ICU who require continuous neuromuscular paralysis, but occasionally it is necessary to do so in order to facilitate another ICU intervention:

- severe respiratory failure
- temperature management after cardiac arrest
- refractory raised intracranial pressure

In this instance we tend to use atracurium or cisatracurium infusion, as these drugs are not dependent on hepatic or renal function for metabolism/elimination.

In all instances, it is preferable to minimise the duration of neuromuscular blockade, and is mandatory to provide adequate deep sedation during this period to prevent inadvertent patient awareness.

How do non-depolarising neuromuscular blocking drugs work?

How do we assess for residual neuromuscular blockade?

What drug is given for reversal of rocuronium and how does it work?

Cerebral perfusion

Quite a few of our patients in the ICU will have some form of brain injury, be it from trauma, hypoxic-ischaemic injury, stroke etc.

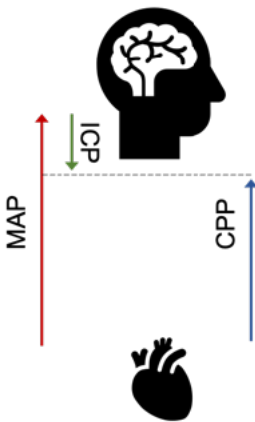
Unfortunately the brain is very susceptible to damage and the primary injury is irreversible. As such the emphasis is on preventing secondary injury to adjacent areas which can become compromised by tissue swelling and impaired blood flow.

To achieve adequate oxygenation of the surviving brain tissue, we must:

- have enough oxygen in the blood
- deliver that blood to the brain
- minimise anything that massively increases the oxygen demand of the brain

Achieving adequate oxygenation is simple enough for most neuro patients (unless they have a concomitant lung injury, in which case VV-ECMO may be an option) see earlier section.

Blood flow to the brain is usually controlled by auto regulation such that it remains constant across a range of blood pressures. In brain injury, this mechanism is disrupted and cerebral blood flow (CBF) becomes primarily dependent on the cerebral perfusion pressure (CPP), which is the mean arterial pressure (MAP) minus the intracranial pressure (ICP).

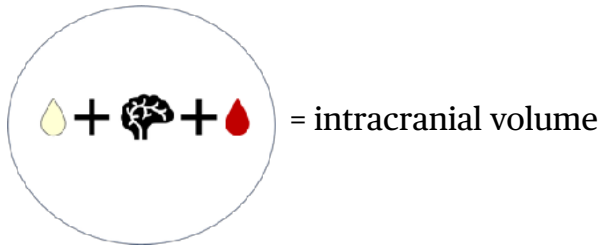


To achieve adequate CPP (>60mmHg) therefore, we want a high MAP and low ICP.

As such, we target a higher MAP than usual in brain injured patients (>90 vs >65mmHg), often using vasopressors to achieve this.

This target is gradually reduced as the patient improves, but we may need to “hyper-tense” them again if there is a deterioration in neurology.

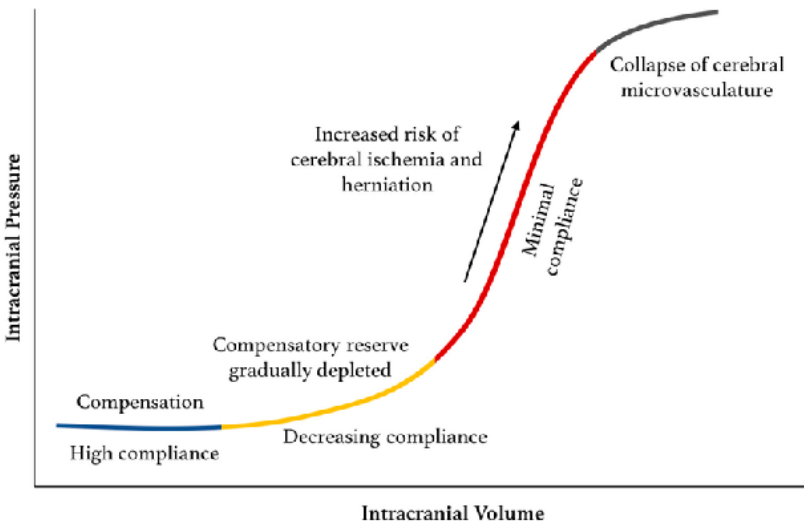
$$CPP = MAP - ICP$$



Intracranial pressure is proportional to the intracranial volume, which is comprised of CSF, brain tissue and blood.

Assuming patent CSF drainage, as the volume of blood (intracranial haemorrhage) or brain (cerebral oedema) increases, the ICP remains low as CSF is displaced and reabsorbed.

At some point, however this compensatory mechanism is overcome, or fails (e.g. obstructive hydrocephalus) leading to a rapid increase in ICP with small further changes in volume (see below graph).



ICP management is divided into “tiers”, starting with the very basics that every brain injured patient should receive:

Measure	Rationale
Head up positioning to 30 degrees with head in midline	Improved venous drainage
Avoiding extrinsic venous compression (e.g ETT ties/collars)	Improved venous drainage
Normocapnoea (may require intubation and IPPV to achieve)	CO ₂ is a potent vasodilator and small increases in a patient without intact auto regulation will dramatically increase the intracranial blood volume leading to increased ICP
Avoiding straining/coughing	Straining increases CVP and reduces venous drainage
Avoiding hypotonic fluids/hyponatraemia	Avoid osmotic gradient which will lead to cerebral oedema
Assessing for and treating seizure	Seizures massively increase cerebral metabolic rate

In the unconscious patient with a severe brain injury, we will usually request insertion of an ICP monitor which allows direct measurement, and urgent treatment of any increases.

Treatment of raised ICP includes:

- hyperventilation (reduce CO₂ induced vasodilation, but avoid for prolonged period as hypocapnoea can lead to vasospasm and reduced CBF)
- osmotic therapy with saline or mannitol (reduced cerebral oedema)
- deepening sedation +/- neuromuscular blockade
- evacuating subdural or extradural blood
- draining CSF (see EVD) if obstructive hydrocephalus
- barbiturate coma (dramatically reduces cerebral metabolic rate)
- decompressive craniectomy (last ditch measure to allow more space and treat critically raised ICP - improved survival but not good neurological outcomes)

What are some clinical signs of a severely raised ICP?

Why might somebody need to be intubated after a traumatic brain injury?

Ventricular drainage

Patients with obstructive hydrocephalus and raised ICP will be offered emergency ventricular drainage, usually with an external ventricular drain (EVD). This is usually in the context of intraventricular haemorrhage resulting in obstructed CSF outflow.

The EVD is set to a certain height (corresponding to desired ICP) and if the pressure rises above this, CSF will drain into the chamber. This can be adjusted as needed, and can be used in an ICP crisis to rapidly reduce ICP.

Occasionally patients will have a permanent shunt inserted if their hydrocephalus is the result of a chronic problem.

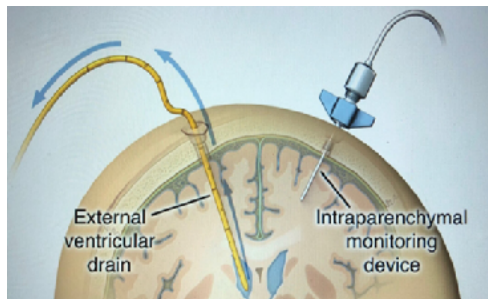
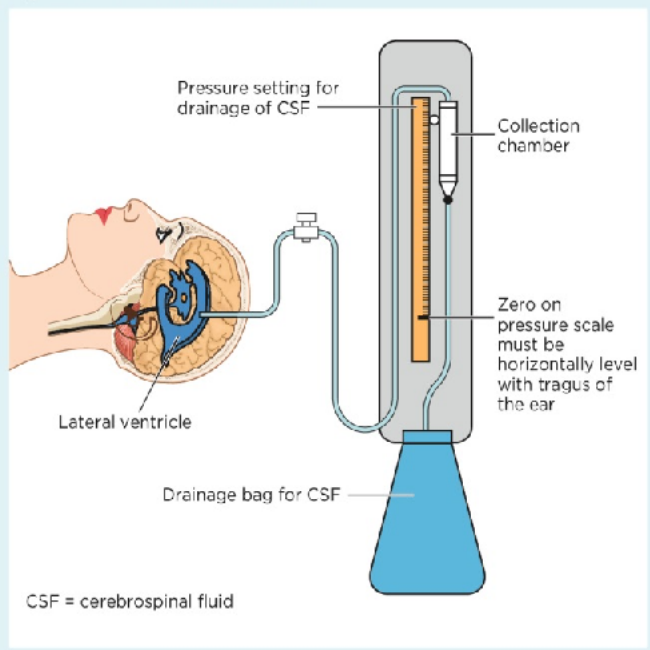


Fig 2. **External ventricular drain**

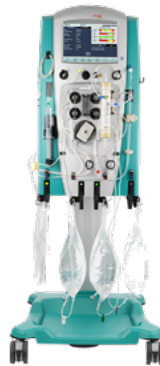


Kidneys

Acute kidney injury is very common in ICU, most often as a manifestation of another problem causing multi-organ dysfunction, but sometimes as the primary issue.

Most of the management of AKI is just good supportive care and avoiding doing things that harm the kidney further, but occasionally patients will require emergency renal replacement therapy (RRT).

In ICU this usually entails insertion of a “vascath” or temporary dialysis catheter, and use of a mode of RRT called continuous venous-venous haemodiafiltration (CVVHDF).



What are the indications for emergency RRT?

As well as emergency RRT, we sometimes have patients on long-term RRT who are admitted with other problems, and in these cases will often continue with their usual

regime (e.g. intermittent haemodialysis or peritoneal dialysis) with help from the renal team.

We also need to think about the metabolism and clearance of the medications that these patients are taking, and will often adjust the dose or interval of renally cleared drugs in patients with AKI.

Which common classes of drugs are nephrotoxic?

Take a look at a patient's drug chart - would any of these drugs need to be stopped/changed if the patient developed a severe AKI?

GI tract

As with the kidneys, many ICU patients will have issues with GI tract and hepatic function as a result of their primary diagnosis, or occasionally as the reason for their ICU admission.

Ileus and pseudo-obstruction are common in ICU, particularly amongst surgical patients. Prevention in ICU is primarily by avoiding opioids where able, encouraging mobilisation and early enteral intake.

In some cases, patients will be unable to safely swallow and it is common in the ICU for patients to have NG tubes inserted, both for enteral feeding and medication administration.

If a patient is likely to be unable to establish enteral feeding soon after admission, or has already had a prolonged period of malnutrition at the time of admission to ICU with ongoing issues with absorption, they may require total parenteral nutrition. This is organised by the dieticians and must be given via dedicated central venous access.



Liver

The liver does an amazingly good job of keeping the rest of the body in check - liver failure patients are extremely sick, and unlike respiratory or circulatory failure, they can look remarkably well right up until they are in established multi-organ failure.

What functions does the liver perform?

As with kidney injury, there are very few specific treatments to fix a liver injury, but unfortunately we do not have an effective “liver replacement therapy” to buy the patient time to recover in the same way as RRT does.

Patients with acute or acute-on-chronic/decompensated liver failure have a high mortality rate as the ongoing worsening hepatic function leads to a spiral of multiorgan failure that is hard to recover from unless transplantation is an option. As such, early involvement and transfer to a specialist transplant centre for appropriate patients is indicated.

For patients with established acute or decompensated chronic liver failure who are not suitable for transplant, ICU is unlikely to be able to alter their prognosis unless there is a very reversible cause for their deterioration (e.g. variceal bleeding).

As such, we don't see that many patients with liver failure in our ICU as they will be transferred to Auckland, or are unlikely to benefit from ICU.

What is relatively common is acute liver *injury* which is usually a bystander to another process, such as shock, trauma or occasionally drug induced. Usually this gets better with cessation of the causative process and time, but we will monitor these patients for evidence of liver failure.

What findings would distinguish acute liver injury from failure?

Final thoughts

I really hope that you enjoy your time with us - it was my time in ICU/anaesthetics as a medical student that shaped my decision to pursue it as a career.

You are not expected to learn everything in this guide, nor do we expect that everybody wants to do intensive care! What I hope is that, whatever your interest, you see some element of it in ICU, and that this guide and the team who have made ICU their career, can help you understand better what it is that we do.

As a side note, some people in ICU are very enthusiastic about specific topics like physiology and pharmacology, and their way of sharing this joy is to ask you questions about it! They are not trying to make you feel ignorant, but I know it can feel embarrassing when you are put on the spot, particularly in front of others. A good way to deal with this is to ask them lots of questions yourself - they just want you to be engaged and interested.

Look forward to working with you all and hope you enjoy your time with us!

Sam

