The Critical CARE Course©

Core Assessment Revision & Education

We realise these are unusual times & are happy to share our course manual to try to help you understand some more of the basics of intensive care. Please do let us know if you've used it - there's a really quick link to do that here via the QR code or the link.

https://forms.gle/z2jYh8mMzDcCc6im8

Endorsed by the Intensive Care Society

Table of contents

FOREWORD

My first experience of intensive care was in 1976 as a newly qualified house officer, delivering to the ICU a patient we had resuscitated from a cardiac arrest. As an aspiring general practitioner, it seemed to me to be a forbidding place: a physiology laboratory from which bedside medicine had been banished. Later on, as a cardiology registrar, I found my interest in intensive care stimulated by the uncomfortable fact that I lacked the skills required to care for my sickest patients: I was obliged to surrender them to the anaesthetists – who were clearly far more competent than I. So, I decided to change direction, to become 'an intensivist', and discovered that I could indeed become a general practitioner after all, but with added skills to provide quality holistic care to the most severely ill patients in the hospital, in the safest place in the hospital.

Critical illness challenges our existence as patients, our courage as relatives, and our capacities as clinicians. From a professional perspective we must integrate the multidisciplinary care of patients with complex diseases and comorbidities. We must master practical technical skills, and temper these with non-technical skills to minimise distress to our patients and provide compassionate support to their families. We must support each other in the ICU and our colleagues outside, so that patients get the best care. And we must recognise our limitations, and the fact that it is easy to make things worse, not so easy to make them better.

This course provides an excellent foundation for acquiring these skills and then mastering them through a lifetime of practice in our new speciality of intensive care medicine. The authors and tutors are all front-line, high-quality clinicians. From them you will gain scientific knowledge, learn team-based decision-making, and become high-reliability practitioners. And above all, you will appreciate the true meaning of placing the patient at the centre of care.

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Foundation Dean of the Faculty of Intensive Care Medicine 2010-13.

INTRODUCTION

Welcome to Critical Care Course© manual.

Stepping into a critical care unit for the first time as a healthcare provider in unlike any other experience in medicine. Surrounded by banks of hi-tech equipment, the very sickest patients in the hospital are tended to by scores of people from a wide multidisciplinary team, whilst the emotions of often desperate friends and families are only too evident. This heady mix of technology, teamwork and tension makes for an exciting, hopefully inspiring, but always daunting environment when it is first witnessed. Yet those who are drawn into critical care soon find themselves learning the trade and find themselves taking on the roles that seemed such a long way off at first sight.

This course intends to supplement existing educational opportunities and help contextualise that learnt in clinical practice by providing a solid knowledge base. This in turn will help critical care practitioners to progress from those earlier stages through to confident, key decision-makers within the team. Whilst those completely new to critical care may find the course quite detailed, and those approaching the FICM exam would find it lacks sufficient detail, it is aimed at helping everyone in between to gain knowledge and confidence in their work.

The course is intended to be equally suitable for nursing staff, medical staff, critical care practitioners and other allied healthcare specialists engaging in the care of the critically ill, such as physiotherapy and OT, and the workshop sessions in particular benefit from the collaborative approach that exists when all these groups working together. On the medical front, it is intended to meet the needs of any core trainee with an interest in critical care, whilst also offering the opportunity for early specialist trainees to consolidate their knowledge or for keen foundation doctors to stretch themselves. It is hoped nursing staff will gain the vital knowledge and understanding required for taking on higher levels of responsibility within critical care, and critical care practitioners will find it useful in promoting high standards in the care they deliver and for their own career paths.

Throughout the course our contributors have kept in mind that they are trying to help you with the very practical business of delivering great care, rather than getting bogged down in theory, and with making sure this is relevant to the way critical care is delivered in the UK.

Study time and budgets in the NHS are not only small, but come under pressure from other course requirements, so it is with this in mind that the course is limited to just one day. Feedback from previous courses has consistently indicated that workshops are a far better learning medium than lectures, so the course is dominated by interactive workshops led by experienced critical care practitioners and supplemented by this pre-course manual. In effect this manual takes the place of a series of lectures and is it essential that candidates take enough time to read and understand this manual fully before the course. The discussions that arise in the workshops will then help to put what has been read into practice and will have the benefit of all group members reaching roughly the same starting point so that those discussions meet the needs of as many candidates as possible.

Although we have worked hard to assure the accuracy of this manual and it includes everything we feel is needed to manage an on-call in the ICU as well as possible, it will inevitably not be comprehensive in every detail, and local practice may differ, or indeed it may contain inaccuracies we have not spotted! The information in this manual is intended as a knowledge springboard to facilitate learning in clinical practice and on the workshop section of the course, and the course cannot take responsibility for decisions made by candidates in clinical practice. Reference should be made to local policies, guidelines and clinical leadership, which must always take precedence over the material in this manual.

One quick word about terminology: Confusion abounds as to how the specialty should be referred to, and what to call the environments that provide it. Historically referred to as "Intensive Care", it is now much more commonly known as "Critical Care", whilst the training programme refers to "Intensive Care Medicine", as does the title of the Faculty (The FICM). Meanwhile a "Critical Care Unit" runs the risk of being confused with the other "CCU", the "Coronary Care Unit", and is therefore variably referred to by alternative terms such as the Intensive Care Unit ("ICU") or Intensive Therapy Unit ("ITU"). You can see why this is not straightforward!

For the purposes of a unified approach in this manual we propose to adopt the following: The provision of care will be referred to as "critical care" or "critical care support", irrespective of where this care is provided (including in the ED, in ward areas prior to transfer to a more appropriate area or in high care areas), whilst a specific unit designed for the provision of critical care will be referred to as the "Intensive Care Unit" or "ICU".

In distributing the manual in electronic rather than printed form the intention is not only to be more environmentally friendly, but also to facilitate a more responsive approach to corrections, improvements or changes in practice, which can be integrated into regular updates with ease. In this respect the course would be very grateful indeed for your feedback and suggestions either face to face on the workshop day, or via email: thecriticalcarecourse@gmail.com.

Finally, this course is the result of a great deal of hard work provided by many contributors, listed in the following section. The editorial group would like to express their gratitude to all those who have provided their time and expertise in completing this project.

We hope you find this manual useful and that you enjoy the course.

Laura Kocierz, Andy Burtenshaw & Jonathan Hulme

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CHAPTER 1: DECISION MAKING IN CRITICAL CARE

Dr Andy Burtenshaw & Dr Chris Bassford

LEARNING OBJECTIVES

- To describe how the principles of medical ethics apply to intensive care unit admission decisions
- To give examples of the factors which frequently complicate decision making around intensive care unit admission
- To give examples of how the views of the patient may exert strong influence on admission decisions
- To explain why communication (including subsequent documentation) is so important in intensive care decision making

An enormous number of decisions are made on intensive care units every day, but often the most important decisions are to be made at the start and end of a patient's involvement with intensive care. Of these, the decision to admit a patient for intensive care support can be one of the most challenging. It frequently needs to be made under time-critical conditions, with limited available information, and in a significant proportion of these scenarios the patient lacks the capacity to participate in the decision process.

The decision to offer intensive care support is frequently akin to offering a means of sustaining life, or to look at it another way, denying a patient intensive care support will often result in the inevitable death of the patient. However, exposing a patient and their loved ones to the burden associated with intensive care without an acceptable possibility of a healthy outcome is usually viewed as inappropriate. Furthermore, critical care resources within each hospital are limited and need to be managed carefully to ensure maximal benefit is obtained from them.

Whilst some admissions are clear-cut both in their likelihood of achieving benefit and the probable quality of that outcome, a very large number are much less clear. Patients often present with multiple co-morbidities, impaired functional capacity and serious presenting pathologies which carry with them low chances of successful treatment, and in these cases, decisions can be very challenging. There is often a lack of detailed information about the patient's co-morbidities and functional capacity, or about the presenting pathology (which has frequently not yet been fully

investigated). When these factors are added to a deficit in high quality literature upon which to form evidence-based assessments of outcome potential, they conspire to make predictions inaccurate and highly subjective.

The concept of an acceptable outcome is also very complicated and will mean something very different to each individual. For some people the prospect of surviving an intensive care unit stay but being left with significant disabilities is unacceptable, whilst others would choose to live regardless of their ongoing functional incapacity and care needs. There is clearly no one correct position on this subject, and where possible the views of the patient must be sought.

To further complicate the issue, there has been a gradual change in the purpose of admission to the intensive care unit over the last decade or two. Traditionally the bar for consideration for intensive care therapy was the requirement for organ support that could not be provided elsewhere in the hospital. However, there is increasing recognition that admission for closer observation and management (facilitated primarily by staffing levels, staff experience and available monitoring equipment) may also offer significant benefit, and initiatives such as NELA (The National Emergency Laparotomy Audit) have promoted intensive care unit admission in high-risk cases even when they do not require advanced organ support.

The decision to admit to ICU is therefore often challenging, and a decision to deny intensive care support can often not be reversed as the opportunity has passed. Making good decisions remains very much a challenge, and there is significant research being undertaken with the aim of identifying the most relevant admission decision factors and providing a framework with which to apply them to real life scenarios.

Whilst we await more sophisticated approaches to decision making, the four basic principles of medical ethics represent a useful framework upon which to base one's decision.

In the context of intensive care unit admissions these might be summarised as follows:

THE 4 PRINCIPLES OF MEDICAL ETHICS

BENEFICENCE

Admitting the patient to the intensive care unit should be for the express purpose of offering benefit to the patient. From a medical perspective, this usually refers to the treatment of a reversible pathology, but each patient will have an individual view on whether the likely outcome is acceptable to him or her, or indeed whether the offer of treatment is likely to condemn him or her to an outcome they would find unacceptable.

NON-MALEFICENCE

Admissions to ICU are often accompanied by a burden to the patient, often in the form of distress and discomfort. This should not be undertaken without the realistic potential of offering benefit to the patient.

JUSTICE

The provision of critical care should be equitable between patients. Therefore, it should not be limited by resources, but rather by effective, individualised, and where possible patient involved decision-making. It should not be inappropriately biased by other factors (e.g. age or learning disabilities) but should also not result in the consumption of resources that are then unavailable to subsequent patients who need them.

AUTONOMY

Wherever possible the patient should be involved in the decision-making process, or as a substitute where this is not possible, their likely views should be ascertained from their family or friends. Of these four ethical pillars, it is autonomy that usually requires the most attention, with effective decision-making most commonly being facilitated by honest discussions with patients and their families to arrive at informed decisions based on both the likelihood of survival and the nature of that outcome. Everyone's definition of a "good outcome" is different, with the most important view being that of the patient.

Effective communication is a vital pre-requisite for good decision-making. It is often the cornerstone to understanding the nature of the pathology and likely prognosis following communication with parent teams and is essential in obtaining the views of the patient. Similarly, communication which takes place with patients and their families at this stage is of vital importance in helping them understand how decisions

have been arrived at, what will happen next, and potential outcomes. It is essential to ensure the notes contain good documentation of the discussions that have taken place and the rationale for all decision-making, including the decision whether to admit for intensive care support and whether any limitations on treatment or decisions not to resuscitate in the event of cardiorespiratory arrest have been made.

Finally, decisions to withdraw life-sustaining treatment are often contentious. However, they are usually characterised either by deterioration refractory to escalating treatment (in which case the result is inevitable) or by a failure to respond to treatment over a period of time. In the latter case, in addition to there usually being a great deal more information available than at the time of admission, there is also usually time to explore other options, facilitate a multidisciplinary team approach to the decision to withdraw, or seek second opinions. The time available also usually enables much less time-pressured discussions with family members (and sometimes the patient themselves) than in the case of admission decisions.

SUMMARY

- The decision to admit a patient for intensive care support can be challenging
- Time-pressure, a relative lack of information, and a lack of patient involvement frequently complicate this process
- Decisions should adhere to the four basic principles of medical ethics
- Effective communication, both with other hospital clinicians and with patients, or their families or friends, is critical to arriving at what will be perceived as correct, and mutually acceptable decisions
- Good documentation of the decision process is vital

CHAPTER 2: ASSESSMENT AND INITIAL CRITICAL CARE MANAGEMENT OF A PATIENT

Dr Abby Ford

LEARNING OBJECTIVES

- Understand what constitutes critical illness and the criteria for admission to an intensive care unit
- Understand scoring systems and 'red flags' that indicate a patient is deteriorating
- Understand levels of care available to support the critically ill patient
- Understand how to assess the critically ill patient whilst initiating treatment where appropriate

INTRODUCTION

Critical illness is any life-threatening condition whether acute, acute-on-chronic or chronic in nature.

Intensive care offers an enhanced level of physiological support to patients to enable time and treatment of potentially reversible aspects to take effect. There are no magic bullets in intensive care!

Patients considered for ICU admission should generally have an acute, potentially reversible condition with an underlying level of health that is compatible with both longer-term survival and benefit from the intensive care treatment. Thus, admission might be considered for a patient with pneumonia and multiple sclerosis who is still independently mobile but be declined for a patient who is bedbound and PEG fed as a result of their MS with pneumonia which is likely to be a marker of progressive deterioration and a terminal event. As described in chapter 1 these decisions are often complex and in so far as possible should also take into account the views of the patient.

PATHWAYS OF REFERRAL INTO THE INTENSIVE CARE UNIT

Intensive care unit admission may be planned or an emergency.

Planned admissions usually support surgical intervention where the patient requires enhanced nursing care and monitoring or perioperative organ support.

The criteria for perioperative intensive care admission will vary between hospitals depending on the skill mix available on the surgical wards. For example, some hospitals admit every patient with an epidural to an intensive care unit whilst others offer advanced nursing care in the ward environment.

Absolute indications for post-surgical admission are where the patient requires ventilation in the immediate post-operative period e.g. cardiac surgery, liver transplantation and major maxillofacial reconstructions.

The intensive care unit senior team should be consulted about any planned ICU admission in advance: surgery should not proceed until the bed has been confirmed. Not communicating effectively risks an inability to provide the anticipated patient care or may adversely influence the hospital's capacity to offer critical care for co-existent or subsequent emergency admissions.

Emergency admissions may come from anywhere in the hospital including the emergency department, wards, coronary care, interventional radiology, cardiac catheterisation laboratories or operating theatres. Tertiary centres also accept transfer of patients from peripheral hospitals for specialist treatment e.g. for neurosurgery. 'Non-clinical transfers', i.e. those where the patient is moved between intensive care units simply to free up capacity in the originating hospital are not considered acceptable except in the most extreme circumstances.

Whilst there is a preference that patient referrals to intensive care should come from senior clinicians as this generally facilitates a comprehensive discussion of the factors influencing admission and any ethical considerations or treatment limitation

decisions, it is nonetheless a central tenet of intensive care that early interventions improve outcomes and that referrals are accepted from a wide range of clinical staff.

Diagnosis of critical illness in a ward patient is usually based on deteriorating physiology that may be identified using early warning scores based on vital signs observations. These scores enable nursing staff to identify patients who are deteriorating and escalate care through the patient's own medical team, critical care outreach or the medical emergency or cardiac arrest teams. All hospitals should use NEWS (National Early Warning Score II) or an equivalent validated "track and trigger" system. These scores are validated for adults over 16 years old except pregnant women. Children and pregnant women should be assessed using specific observation charts appropriate for their physiology.

Six simple physiological parameters form the basis of the scoring systems:

1. Respiratory rate
2. Oxygen saturations
3. Temperature
4. Systolic blood pressure
5. Pulse rate
6. Level of consciousness

Of these physiological parameters, respiratory rate is the most sensitive, but unfortunately is often the least accurately recorded. The scoring systems are not infallible, common reasons for failure to identify high-risk patients include:

- Insufficient frequency of observations or failure to perform observations
- Incorrect calculation of total score
- Failure to escalate to medical teams
- Patients with abnormal or blunted physiological responses e.g. beta-blockers, pacemakers, immunocompromised patients.

Other disease specific scores may also initiate a referral to intensive care by identifying patients at risk of deterioration. Examples of this include the Glasgow score ≥ 3 for pancreatitis or either a P-POSSUM or NELA risk score predicted mortality greater than 10% for emergency laparotomy.

It is important to note that whilst scoring systems represent an objective means of screening for high-risk patients who may benefit from intensive care, they lack the

fidelity to reliably inform decision-making. They will not always pick up all suitable patients, and similarly those with high scores will not always require or be appropriate for intensive care support. They should be used in conjunction with clinical judgement, multidisciplinary discussions and patient (or family) consultation.

LEVELS OF SUPPORT IN INTENSIVE CARE

- Level 0: normal ward-based care
- Level 1: ward-based care with enhanced monitoring or ICU input
- Level 2: 'HDU' level care, support for one failing organ system other than advanced respiratory support, 1:2 nurse to patient ratio
- Level 3: 'ICU' level care, support for at least 2 organ systems, or advanced respiratory support, 1:1 nurse to patient ratio
- Level 4: technically not an official classification: generally refers to patients on extra-corporeal support e.g. ECMO with a 2:1 nurse to patient ratio. Occasionally used by units to flag additional staff requirements.

ASSESSMENT OF THE CRITICALLY ILL PATIENT

Assessment of the critically ill patient should follow a structured approach allowing identification and simultaneous treatment of life-threatening conditions as the assessment proceeds. Any change in the patient's condition should prompt re-assessment.

Whilst a traditional history taking approach may be appropriate for many situations this may lead to delayed recognition and management of life-threatening issues in the acutely unwell patient. For this reason, we advocate the use of a 'CABCDE' approach, where much of the detail is gained after immediate safety is assessed and assured:

C Catastrophic haemorrhage control

A Airway (plus c-spine)

- Is the patient talking? Is the airway maintained and what sound is the patient making? Clinical examination
- Consider manual manoeuvres, airway adjuncts and definitive management (i.e. does the patient require tracheal intubation?)
- Call for help early if there are airway concerns

B Breathing

- Respiratory rate, pattern, depth and saturations?
- Oxygen administration if indicated
- Chest examination
- Consider arterial blood gas analysis

C Circulation

- Heart rate, blood pressure, central capillary refill time, urine output?
- Consider obtaining large bore venous access
- Consider need for blood tests including group and screen or cross match
- Remember 5 sites of significant blood loss: chest, abdomen, pelvis, long bones, floor
- Volume resuscitation and which fluids are indicated

D Disability

- GCS, pupil size, focal neurological signs, confusion / delirium
- Check blood sugar
- Immediate sedation and analgesia requirements?

E Examination and extra tests

- If the patient is stable then a more thorough history, review of the notes and examination may be appropriate
- Look at other observations including core temperature
- Review other investigations e.g. CXR, CT, ECG, ECHO
- Review or obtain multidisciplinary team input as appropriate

Remember the history may not be available from the patient and you will often have to use secondary sources such as notes and collateral history from relatives, paramedics, police etc.

Never assume the diagnosis the patient has been given is correct, always make your own assessment of the clinical signs.

At all times ASK FOR HELP EARLY IF THE PATIENT IS UNSTABLE.

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CHAPTER 3: AIRWAY MANAGEMENT

Dr Laura Beard and Dr Phil El-Dalil

LEARNING OBJECTIVES

- Describe basic airway manoeuvres and how to perform an airway assessment
- Be aware of advanced airway manoeuvres
- Comprehend the NAP 4 recommendations pertinent to critical care
- Understand the difference between the emergency management of tracheostomy displacement with patent upper airway versus emergency laryngectomy management

INTRODUCTION

Basic airway skills are a necessity for any health care professional involved in the care of unwell patients, whether that is in the intensive care unit, emergency department or on the wards. It is therefore important that health care professionals are able to recognise the signs and symptoms of a compromised airway and intervene appropriately in order to prevent hypoxaemia.

RECOGNISING AN OBSTRUCTED AIRWAY

Speak to the patient

- If the patient is able to talk normally you can usually be reassured that their airway is patent

General Features

- Distress
- Agitation (could be secondary to hypoxia)
- Reduced conscious level
    - May be the cause of airway compromise i.e. drugs, intracranial event
    - May be a consequence of an obstructed airway (hypercarbia/severe hypoxaemia)

Airway Features

- Facial trauma, facial swelling
- Excessive secretions, drooling (due to inability to swallow secretions)
- Added sounds: snoring, hoarse voice, inspiratory stridor
- Look inside the mouth for a cause of obstruction i.e. vomit/food/swelling

Respiratory Features

- Paradoxical chest movement (see-saw movement)
- Hypercarbia (rising P_aCO_2 due to inadequate ventilation)
- Loss or reduction in non-invasive end-tidal CO_2 (airway obstruction or apnoea)
- Hypoxaemia (late sign)
- Apnoea

Causes of airway compromise include

- Foreign bodies i.e. food bolus, vomit, secretions
- Reduced conscious level causing collapse of the soft tissues of the pharynx and posterior displacement of the tongue
- Laryngospasm
- Infection: e.g. epiglottitis, laryngo-tracheitis
- Altered airway anatomy e.g. obesity, trauma, haematoma, swelling or tumour
- Thermal inhalational injury which can lead to delayed swelling of the airway

MANAGING A COMPROMISED AIRWAY

If the airway is compromised call for help early

Use basic airway manoeuvres and adjuncts until help arrives

BASIC AIRWAY MANOEUVRES

Look inside the mouth

- Clear any visible obstruction under direct vision using a suction catheter or Magill forceps

If signs of compromise are still present, attempt to open the airway with basic manoeuvres such as the head tilt/chin lift and/or jaw thrust. These manoeuvres aim to open the airway by lifting the tongue and pharyngeal tissue anteriorly.

Head tilt, chin lift

- Apply pressure to the forehead, tilting the head back on the atlanto-occiptal joint
- Lift the mandible forward with two fingers applied below the chin (Figure 3.1)
- This should not be used in those with suspected or known cervical spine injuries

From https://en.wikipedia.org/wiki/Head_tilt/Chin_lift#/media/File:Tongue_blocking_airway.svg

Figure 3.1: Head tilt, chin lift

Jaw thrust

- Place your fingers behind the angle of the mandible and lift upwards
- The thumb or index fingers can then be used to open the mouth (Figure 3.2) or alternatively to hold an oxygen mask to the face simultaneously
- This is safer in patients with c-spine injuries if in-line stabilisation is maintained & care taken to restrict movement to the mandible & soft tissues alone

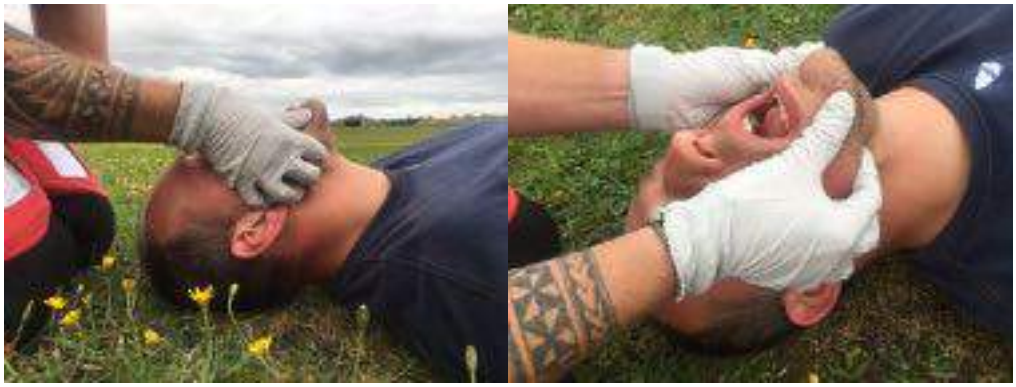


Figure 3.2: Jaw thrust

If the patient is vomiting, they should be placed in the lateral or recovery position to reduce the risk of airway compromise and aspiration. The patient may require definitive airway management if unable to protect their own airway from the risk of aspiration in this position; there may be other indications for tracheal intubation. Following any intervention, the patency of the airway and the adequacy of breathing should be reassessed i.e. has the snoring noise improved, is the chest rising and falling, is the face mask misting, if capnography is being used is there an end tidal CO_2 trace?

AIRWAY ADJUNCTS

Airway adjuncts are useful when simple manoeuvres alone are not fully effective

Oropharyngeal (OP) airway or Guedel airway

Use: To prevent the tongue partially or completely obstructing the airway. It can be used alone or in combination with a jaw thrust and head tilt/chin lift. An oropharyngeal airway (OPA) can also be used to assist oropharyngeal suctioning and should be considered as a means of improving expiratory gas flow as much as inspiratory gas flow because even low levels of expiratory gas flow restriction may lead to gastric inflation, progressive ventilatory difficulty and increased aspiration risk. The use of OP airways as a bite block has been advised against.

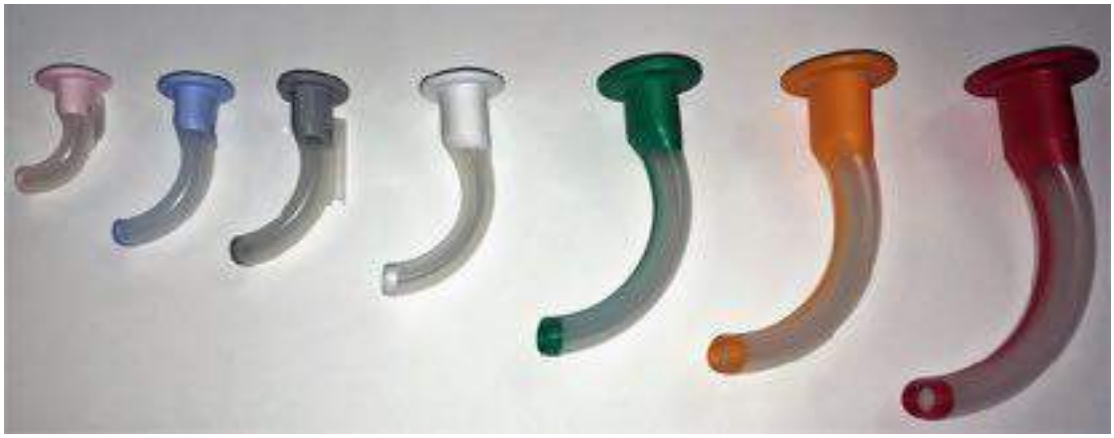


Figure 3.3: Oropharyngeal airways

Sizing: OP airways come in a variety of colour coded paediatric and adult sizes (Figure 3.3). Adult sizes range from 3 to 5: measuring from the centre of the first incisors to the angle of the mandible can identify the correct airway. Measurement from the tragus of the ear to the corner of the mouth is also used.

Insertion: In an adult they are inserted 'upside down' with the concavity facing upwards until the back of the soft palate is reached. The OPA is then rotated through 180 degrees and advanced; in some cases, a simultaneous jaw thrust may assist with the insertion bringing the tongue into a more anterior position within the OPA concavity. In children, the OPA is inserted with the concavity facing down to prevent damaging the child's vulnerable soft palate; a tongue depressor is often helpful.

Complications: Insertion of an OPA can stimulate the gag reflex causing vomiting and the potential for aspiration. Consequently, their use is avoided in the conscious or semi-conscious patient. They can also cause trauma and bleeding.

Nasopharyngeal (NP) airway

Use: To relieve partial or complete airway obstruction by allowing a conduit through the soft tissues of the posterior oropharynx. They are less likely to stimulate the gag reflex than an OPA and better tolerated in semi-conscious and conscious patients. They can also be used in patients with limited mouth opening and to assist in the passage of suction catheters.

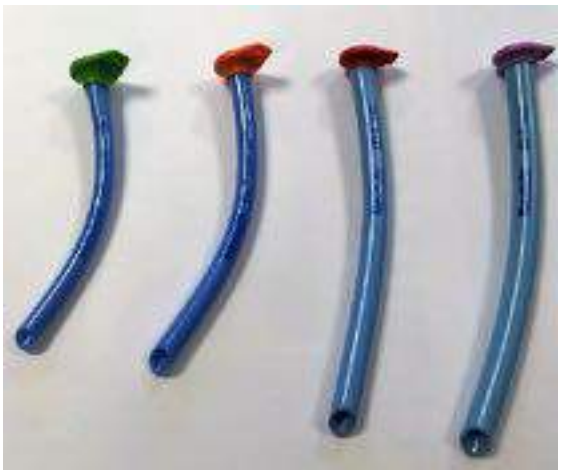


Figure 3.4: Nasopharyngeal Airways

Sizing: In adult women, a size 6 (internal diameter in mm) is recommended and size 7 in adult men.

Insertion: A well-lubricated nasopharyngeal airway is inserted horizontally along the floor of the nasopharynx following the curvature of the nasopharyngeal airway. The tip of the correctly sited NP airway should sit just above the epiglottis. The right nostril is most commonly selected as it allows the bevel to face the nasal septum, reducing trauma.

Complications: Excessive force should be avoided during insertion as this can lead to trauma and epistaxis (use with caution in coagulopathic patients). Use with caution

in patients with a possible base of skull fracture due to the rare possibility that the nasopharyngeal airway could pierce the cribriform plate. Emergency airway management of the patient with a traumatic brain injury who has trismus will often include the insertion of NP airways, promoting airway management with correct insertion techniques over low incidence complications.

Supraglottic Airway Devices (SAD)

The most commonly used SAD in the emergency setting is the i-gel®. Some hospitals may also have standard 1st generation SADs available, commonly referred to as a Laryngeal Mask Airway (LMA) (Figure 3.5).

Figure 3.5: Supraglottic Airway Devices (left: LMA Supreme ® ; right: i-gel ®)

Use: A SAD can be used as a primary device through which to oxygenate and ventilate the unconscious (or anaesthetised) patient in elective or emergency settings, or rescue device where simple manoeuvres and adjuncts have failed or following unsuccessful intubation. As a rescue device, a SAD is only be used in the short–medium term after which a definitive airway (i.e. endotracheal tube) is inserted. It is possible to intubate through an i-gel® using a bronchoscope but only those with the pre-requisite skill set should attempt this.

Sizing: They are sized according to patient weight: size 3 (30-50kg), size 4 (50-70kg), size 5 (70-100Kg).

Complications: Insertion of a SAD can cause dental damage, trauma of the soft tissues and laryngospasm. An initially well-seated SAD can become dislodged on moving the patient, leading to an inadequate seal. This may also occur as a consequence of patient anatomy and results in hypoventilation and gastric insufflation, increasing the risk of regurgitation and aspiration.

If an i-gel® is unavailable or does not allow adequate ventilation due to a leak, then an alternative SAD such as the LMA can be tried as an alternative. A standard LMA lacks a gastric channel or bite block but has an inflating cuff, which may provide a better seal in some patients.

The i-gel® and LMA are not definitive airways, meaning there is still a risk of airway soiling. The only definitive airways are those in which a tube with an inflatable cuff sits below the level of the cords.

DEFINITIVE AIRWAYS:

- Endotracheal tube (oral and nasal)
- Tracheostomy tube

Advanced airway management and intubation are fundamental skills for anaesthetic and intensive care doctors. The 4th National Audit Project (NAP4) undertaken by the Royal College of Anaesthetists (RCOA) and The Difficult Airway Society (DAS) found that a disproportionate number of airway complications occur within ICU and Emergency Departments. A major cause of these events was poor airway assessment and poor planning combined with the complexity and urgency of the cases. It is essential that before any patient is intubated, the correct preparations, equipment and skilled personnel are present and that an airway strategy has been made and verbalised within the team.

Endotracheal Tubes (ETT)

Typical indications for Endotracheal Intubation in Intensive Care

- Inability to maintain a patent airway with basic manoeuvres and adjuncts
- Airway protection e.g. reduced conscious level, prevention of aspiration of stomach contents or blood, or preventative airway protection in the event of impending airway obstruction e.g. swelling secondary to anaphylaxis or inhalational injury
- Requirement for invasive ventilation to manage respiratory failure, including apnoea, or to facilitate further clinical investigations or therapies

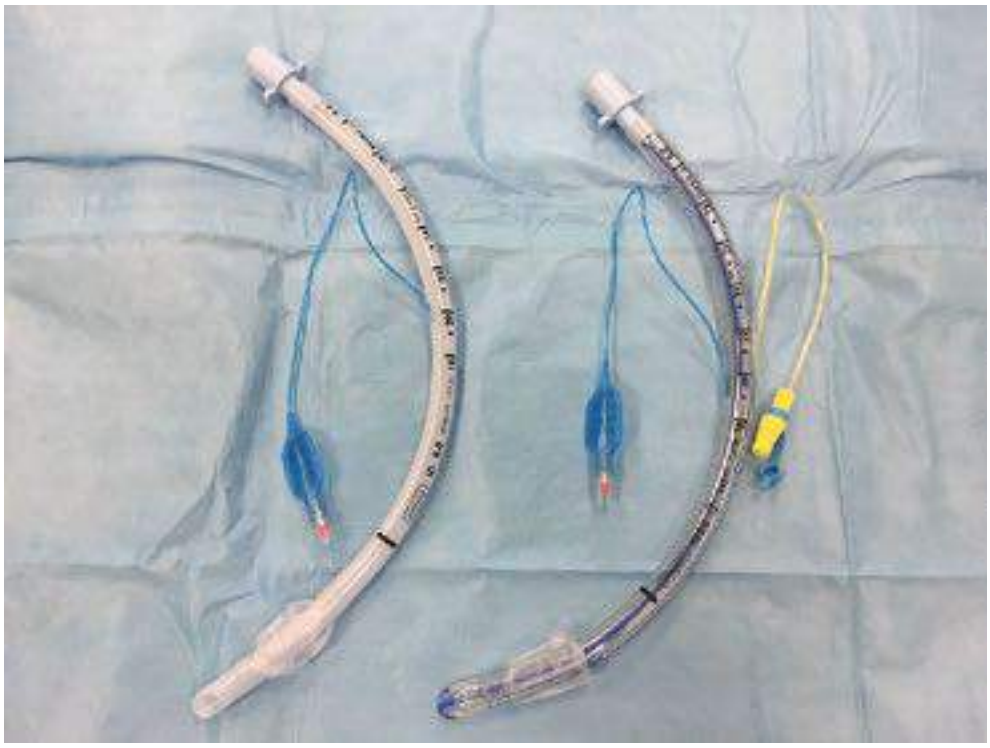


Figure 3.6: Endotracheal Tubes

Parts (Figure 3.6)

- Inflatable cuff: located at the distal end of the tube. It is inflated to a pressure of 20-30 cmH_2O once below the cords and helps prevent airway soiling
- Pilot balloon: Used to provide visual or tactile confirmation of cuff inflation and incorporates a port through with to inflate the cuff and to monitor cuff pressure
- Murphy's eye: Second opening at the tip of the tube that allows ventilation through this side opening should the distal opening of the ETT become occluded
- Numerical markings: represent the distance (cm) from the tip of the tube
- A radio-opaque line runs along the tube to allow identification on a plain radiograph

- A universal 15mm connector allows the ET tube to be connected to a bag valve mask or breathing circuit
- Bevelled tip to reduce trauma
- Subglottic suction port: available on some ETTs. Permits low-pressure suction to be applied to aspirate pooled secretions from above the cuff, with the aim of reducing micro-aspiration and ventilator associated pneumonia rates.

Sizing: Depends on the size of the individual. Adult women commonly require a 7.0-8.0mm, and adult men an 8.0-9.0 mm, internal diameter ETT. There are formulae for the calculation of ETT diameter and length in children.

Insertion: See "Performance of an RSI" below.

Complications:

- Failed intubation (including oesophageal intubation)
- Endobronchial intubation (usually into right main bronchus)
- Local trauma (including dental damage, mucosal damage, bleeding)
- Pulmonary injury (including pneumothorax)

PREDICTING DIFFICULT INTUBATION

Intubation using direct laryngoscopy requires a clear line of view from the upper teeth to the glottis. Difficulty in intubation can be due to either difficult laryngoscopy (i.e. inability to view the vocal cords) or difficulties advancing the endotracheal tube into the trachea.

History

- Does the patient describe previous difficulties with anaesthesia or have an airway alert form or other documentation in their possession or notes?
- Does the patient have any difficulties with neck movement or mouth opening secondary to medical conditions (e.g. rheumatoid arthritis, ankylosing spondylitis) or previous medical/surgical interventions, (i.e. radiotherapy, cervical fusion)?
- Is poor dentition likely to complicate airway management or is there any dental work such as bridges, caps, crowns or loose teeth that may be at risk of damage?
- Is the patient at risk of aspiration? (see Table 3.1)
- When did they last eat and drink?

Table 3.1: Risk factors for aspiration

Upper Airway	Oesophagus	Full Stomach	Lower Oesophageal Sphincter (LOS) Incompetence	Raised Intra-Abdominal Pressure
Loose teeth	Achalasia	Recent oral intake	Hiatus hernia	Abdominal distension
Bleeding eg epistaxis	Strictures	Delayed gastric emptying eg pain, trauma	Pregnancy	Pregnancy
Pharyngeal pouch		Drugs eg opioids	History of LOS incompetence	Obesity
		Bowel obstruction	Drugs eg atropine, opioids	

(Table information from Training in Anaesthesia the essential curriculum, Pg4-5.)

Examination

The mnemonic LEMON is a useful structure for the different features of an airway assessment that can help to predict difficult laryngoscopy

LOOK EXTERNALLY (for cosmetic features that may predict a difficult airway)

- Facial: Small mouth, macroglossia, facial deformity, facial hair, trauma
- Dentition: Protruding teeth, loose teeth, anterior gaps
- Body habitus: Obesity, short neck, large breasts
- Neck: Large goitre, infection/abscess, malignancy, trauma
- Previous medical/surgical interventions: Scars (thyroidectomy, radical neck dissection)

EVALUATE

- Mouth Opening: A patient with normal mouth opening should be able to insert their first three fingers between their incisors. Reduced mouth opening increases the risk of difficult intubation and airway management

- Thyromental distance: The distance from the upper border of the thyroid cartilage to the tip of the mandible when the head is fully extended. A distance of less than 6cm increases the risk of difficult laryngoscopy
- Sternomental distance: The distance from the top of the manubrium to the tip of the mandible (chin) with the neck fully extended and mouth closed. A distance of less than 12.5cm is associated with an increased risk of difficult laryngoscopy.

MALLAMPATI SCORE (Figure 3.7)

- Whilst seated the patient is asked to open their mouth fully and extend their tongue without phonating. The different classes are based on how much of the pharyngeal anatomy is visible.
    - Class 1: Visualisation of the soft palate, fauces, uvula, anterior and posterior pillars
    - Class 2: Visualisation of the soft palate, fauces and uvula
    - Class 3: Visualisation of the soft palate, and base of uvula
    - Class 4: Soft palate not visible

(http://www.frca.co.uk/article.aspx?articleid=257)

Figure 3.7: Mallampati Classification

OBSTRUCTION

- Features suggestive of airway obstruction (i.e. stridor, inhalational injury)

NECK MOBILITY

- Assess ability to flex / extend the neck (a reduced range of movement increases the likelihood of a difficult airway)
- Any patient with c-spine immobilisation will have a reduced range of movement making airway management more challenging

It is important to remember that no predictive test will ever be 100% sensitive and unexpected difficult airways will be encountered.

Laryngoscopy

Cormack and Lehane devised a classification of glottic visualisation during laryngoscopy (Figure 3.8). The purpose of this grading is to facilitate objective grading and documentation of the direct laryngoscopy view so clinicians undertaking subsequent airway management have a guide as to the likely difficulty of the procedure. It should be noted this technically refers to direct laryngoscopy (DL) rather than the view at videolaryngoscopy (VL) (alternative grading systems have been suggested for VL), but it is good practice for documentation to clarify how this view was achieved so future airway management is fully informed.

Grade I: complete glottis visible (easy intubation)

Grade II: anterior glottis not seen (use of adjuncts (i.e. a bougie) may be necessary)

Grade III: epiglottis seen, but not glottis **(difficult airway)**

Grade IV: epiglottis not seen **(difficult airway)**

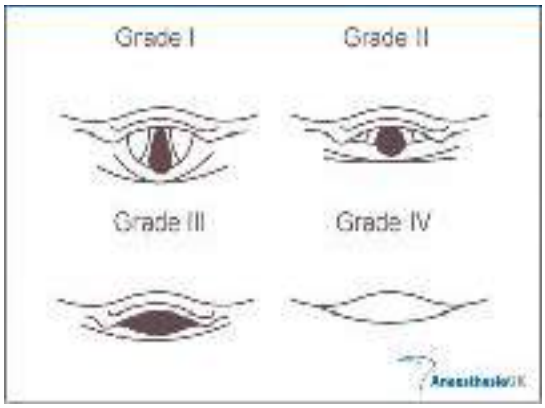


Figure 3.8

Larngoscopy view: Cormack and Lehane

(http://www.frca.co.uk/article.aspx?articleid=257)

RAPID SEQUENCE INDUCTION

A rapid sequence induction (RSI) is an intravenous induction technique for patients at high risk of vomiting and aspiration (see Table 3.1). RSI aims to minimise the time taken to achieve intubating conditions and securing of the airway with an endotracheal tube. Once anaesthetic agents have been given ventilation should either be avoided or delivered using low pressures only until the airway is secure. This measure in addition to the use of cricoid pressure aims to further reduce the risk of aspiration/regurgitation.

CRICOID PRESSURE

The cricoid cartilage is found at the level of C6 and is the only complete cartilaginous ring of the upper airway. Backward pressure applied to the cricoid is transmitted to the oesophagus causing it to be compressed between the cricoid and posterior vertebrae and is intended to prevent or minimise passive oesophageal flow of gastric contents. The manoeuvre is performed by identifying the cricoid cartilage below the cricothyroid membrane and applying posterior pressure to the cartilage using the thumb and index finger. Only trained personnel should undertake the manoeuvre as incorrectly applied cricoid pressure can distort the airway anatomy hindering laryngoscopy attempts. Cricoid pressure can be uncomfortable for patients, so at the start of induction a pressure of 10N should be applied, subsequently increasing to 30N when the patient is unconscious.

The use of cricoid pressure is controversial as, in practice, it may not effectively do what it is intended to do whilst making airway management more challenging. It continues to be recommended in current UK practice, but it is not used in many other countries. Although it is applied with the aim of reducing aspiration risk, if a patient vomits it should be removed immediately to avoid oesophageal rupture.

PERFORMANCE OF RSI

PREPARATION / EQUIPMENT

Personnel

- Health care professional with advanced airway training e.g. intensive care doctor
- Senior skilled supervision should be immediately available if a difficult airway is predicted
- Skilled assistant (competent to perform cricoid pressure)
- Assistant to call for help if required

Equipment

- Oxygen and consideration of high flow nasal oxygenation (apnoeic oxygenation)
- Suction with Yankauer sucker attached, turned on and within reach
- Airway equipment
    - Self-inflating bag and mask
    - 2 Laryngoscopes (check that they are both functioning)
    - ET tube of predicted size & one smaller size (check integrity of pilot balloon)
    - Bougie

  - Airway adjuncts (OP & NP airways)
  - Rescue devices (i-gel, or other 2nd generation SAD)
  - Specialist equipment if required (video-laryngoscope / fibreoptic scope)
  - Surgical airway kit available
- Drugs
  - Induction agent (appropriate for physiological state of patient)
  - Muscle relaxant e.g. rocuronium (1.2mg/kg) or suxamethonium (1 - 1.5mg/kg)
  - Drugs for maintenance of anaesthesia e.g. propofol and fentanyl
  - Emergency drugs e.g. positive inotropes, vasopressors and vagolytic agents
- Monitoring equipment
  - Oxygen saturation probe
  - E_TCO_2 monitor (capnography - ideally waveform)
  - Cardiac monitoring (three lead)
  - Blood pressure monitoring (non-invasive / invasive)
- Ventilator
- Patient trolley that should be able to tilt head down in case of regurgitation

Plan

- If difficult intubation is anticipated consider what could be done to optimise intubation conditions, e.g. use of additional equipment such as a video-laryngoscope
- Additional specialists required e.g. consultant in anaesthesia / ICU / ENT
- Failed intubation plan (see DAS guidelines below)

PROCEDURE

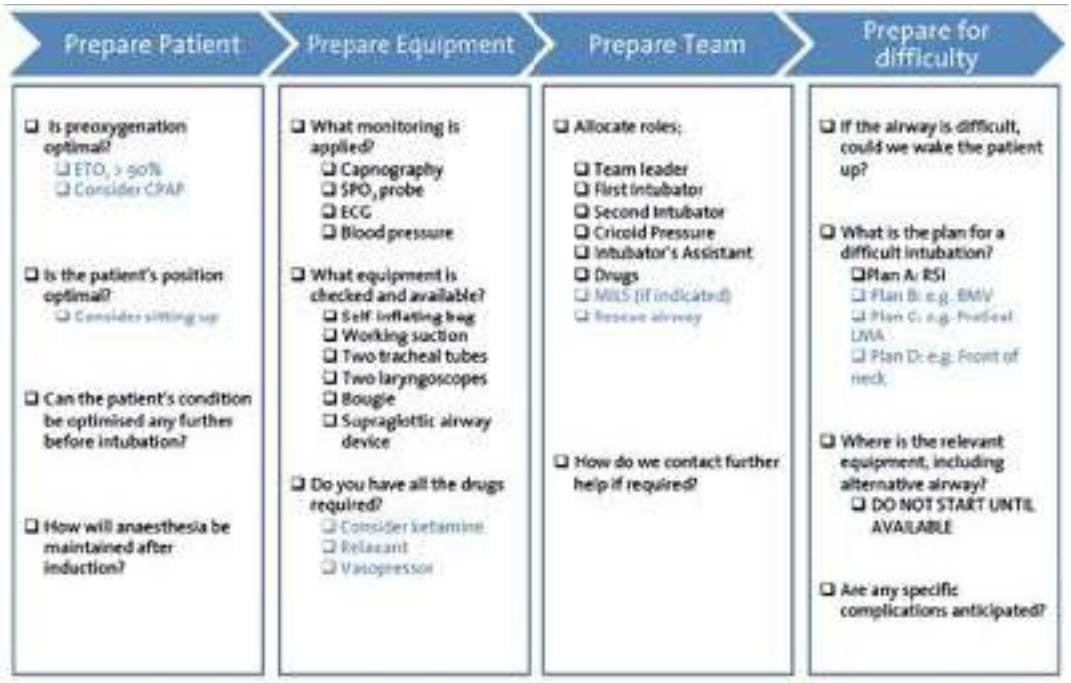
Patient

- Attach monitoring
- Insert large bore cannula and run IV fluids to ensure patency and as a flush to carry drugs
- Ensure that the patient position is optimised
  - 'Sniffing the morning air'
  - If obese consider using the Oxford Pillow
- Pre-oxygenate for 3-5 minutes on F_iO_2 1.0
  - Ensure tight seal with mask and maintain capnography trace
  - In patients not achieving SpO_2 > 93% despite pre-oxygenation augment with supplemental low-pressure ventilation ± CPAP
  - Aim for an E_TO_2 of 0.85

- Pre-oxygenation / denitrogenation significantly reduces the time to desaturation
- Deliver 15L/min oxygen via nasal cannulae to facilitate apnoeic oxygenation until the airway is secured (this is well tolerated in the short term by most awake patients; if not, turn up flow rate at induction. Easily forgotten!)

- Ensure suction available
- Perform checklist prior to induction (See Figure 3.9)

Figure 3.9: Checklist: (Example from NAP4)



RSI

- Skilled assistant to locate cricoid cartilage and apply pressure
- Give chosen induction agents and start fast running infusion of fluids
- Avoid routinely ventilating the patient (to reduce risk of aspiration) but if hypoxaemia inevitable (patient remains hypoxaemic despite pre-oxygenation) or likely, continue supplemental low-pressure ventilation until ready to intubate
- Intubate the trachea as soon as conditions allow
- The laryngoscope is held in the left hand and the tongue is displaced to left. The laryngoscope is then positioned in the vallecular space and the epiglottis lifted
- The vocal cords and tracheal inlet should be visible. If the view is obstructed a bougie can be used. Release of cricoid pressure will improve the view in some

circumstances: remove it early if struggling. Call for help early if intubation is found to be challenging and consider the use of an alternative laryngoscope
- Insert the endotracheal tube so that the cuff is below the level of the cords and inflate the cuff
- Connect to a self-inflating bag or Mapleson C circuit to confirm ventilation and confirm end tidal CO_2 trace. A minimum of 3 capnograph waveforms should be seen in order to exclude an oesophageal intubation
- Tell assistant to release cricoid pressure (they should not do this until told to do so by the person intubating the patient)
- Secure the endotracheal tube with ties or tape
- Maintain anaesthesia
- Continuous E_TCO_2 monitoring should be used throughout to allow early recognition of oesophageal intubation, tube displacement or accidental extubation.

Post intubation checks

- Make a note of tube length at the teeth
- Auscultate the chest to ensure equal air entry with the aim of excluding an endobronchial intubation
- Ensure adequate chest expansion and monitor tidal volumes (target 6ml/kg ideal body weight)
- Monitor E_TCO_2
- Check ETT cuff pressure (should be between 20-30cmH_20)
- Check ETT position (chest radiograph)

Complications

- Laryngospasm
- Haemodynamic instability (including cardiac arrest and death)
- Adverse drug reactions
- Regurgitation ± aspiration
- Failed intubation (including oesophageal intubation)
- Hypoxaemia (including “Can’t Intubate, Can’t Oxygenate” (CICO) scenarios)
- Endobronchial Intubation (usually right main bronchus leading to reduced breath sounds on the left)
- Local trauma (e.g. dental damage, mucosal trauma, bleeding)

DIFFICULT AIRWAY SOCIETY (DAS) GUIDELINES

DAS have produced guidelines for the management of the unanticipated difficult intubation and the "Can't Intubate Can't Oxygenate" (CICO) scenario. There are unfortunately multiple, well-documented airway related critical incidents, where poor management has resulted in serious morbidity or mortality. Management of an unanticipated difficult airway requires maintenance of situational awareness to prevent task fixation. These guidelines provide a stepwise, structured response to a potentially life-threatening scenario. It is vital that anyone involved in advanced airway management is familiar with these algorithms.

It is vital to remember that oxygenation is more important than intubation

DIFFICULT INTUBATION GUIDELINES

Plan A: Tracheal Intubation

- Maintain oxygenation throughout (pre-oxygenation, apnoeic oxygenation, return to face mask ventilation with airway adjuncts if necessary)
- Maximise likelihood of successful intubation at the first attempt (head up / ramped positioning and adequate neuromuscular blockade)
- Maximum of 3 intubation attempts (1 further attempt by a more experienced colleague is permitted) with the aim of limiting hypoxaemia inducing attempts before going on to the next part of the rescue algorithm and reducing airway trauma that can make intubation more difficult
- Do not repeat the same action and expect a different result. If the initial attempt is unsuccessful, do something different i.e. optimise position, remove cricoid pressure, perform external laryngeal manipulation (Backward, Upward, Rightward Pressure: BURP,) use a bougie or video-laryngoscope
- If the above attempts are unsuccessful, declare the 'failed intubation' clearly

Plan B: SAD insertion

- Maintain oxygenation with a SAD
- Cricoid pressure should be relaxed during insertion
- Maximum of 3 attempts at SAD insertion permitted

- Following successful insertion of a SAD, consider waking the patient up; this is rarely an option in the critically ill patient and a definitive airway will need to be inserted instead. This is usually achieved by fibre-optic guided tracheal intubation via the SAD or by insertion of a surgical airway

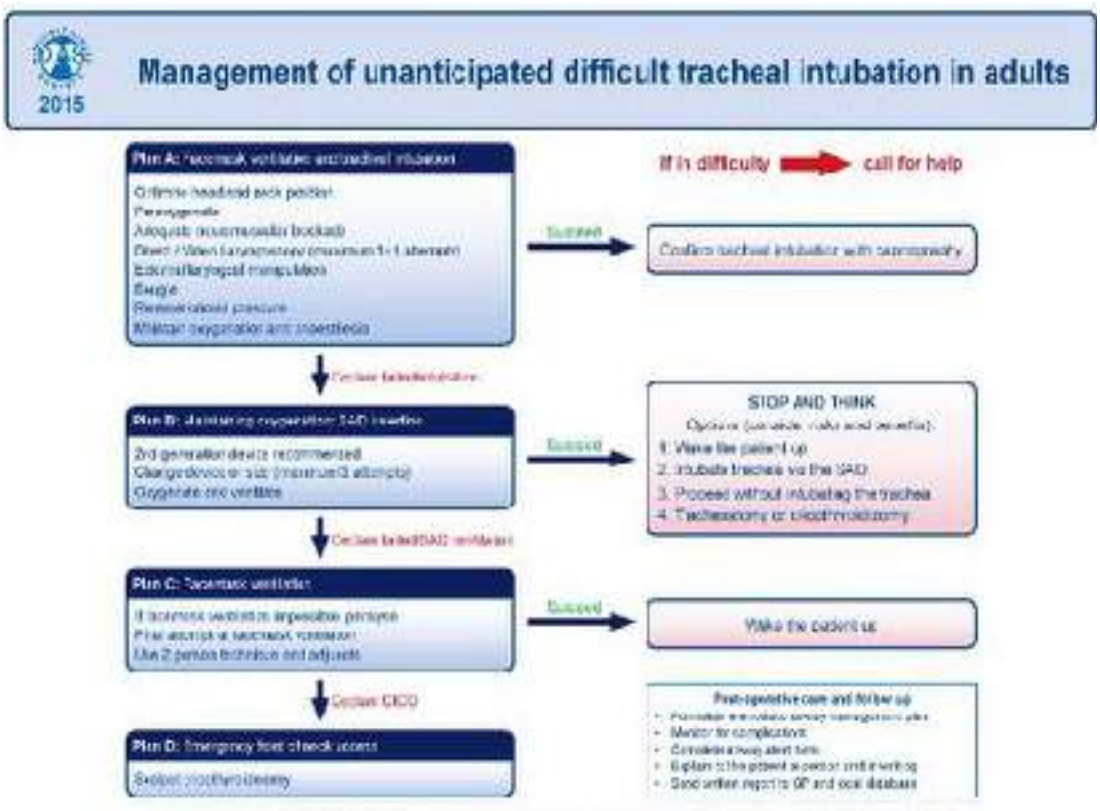
Plan C: Facemask Ventilation

- If initial attempts are unsuccessful, use a combination of airway adjuncts, 2-handed technique and adequate muscle relaxant to maintain oxygenation

Plan D: Emergency front of neck access

- Declare a CICO scenario
- Call for help (again), give 100% oxygen, ensure adequate muscle relaxation
- Perform scalpel cricothyroidotomy

DAS GUIDELINES



TRACHEOSTOMY

A tracheostomy is a definitive airway, which can be inserted using a percutaneous or surgical technique. Simply put it consists an opening (stoma) in the trachea in connection with the skin surface. Depending on the reason for insertion, tracheostomies may be a temporary or a permanent measure.

Indications

- Weaning following a period of prolonged ventilation
    - Benefits:
        - Reduces dead space

- Reduces need for sedation (allowing improved patient interaction, facilitating neurological assessment, reduces cardiorespiratory depression)
- Permits intermittent periods on and off a mechanical ventilator without having to remove or insert an airway to facilitate each change
- May allow oral nutritional intake

- Long term mechanical ventilation
- Component part of a surgical procedure e.g. post extensive maxillofacial surgery
- Inability to maintain upper airway patency e.g. due to neurological impairment or primary airway problem
- Facilitate pulmonary toilet where there is an inability to clear respiratory secretions e.g. due to increased production or a compromised cough
- Protect the airway in patients at high risk of aspiration e.g. neuromuscular disorders

Tracheostomy vs. Laryngectomy (Figure 3.10):

Following the creation of a tracheostomy the pharynx remains in continuation with the trachea via the larynx, meaning the patient can still be intubated if necessary. However, following a laryngectomy, the larynx is removed, and the proximal end of the trachea is connected to the anterior neck. Consequently, there is no connection between the upper airway and the trachea. Therefore, it is physically impossible to intubate, oxygenate, or ventilate via the upper airway.

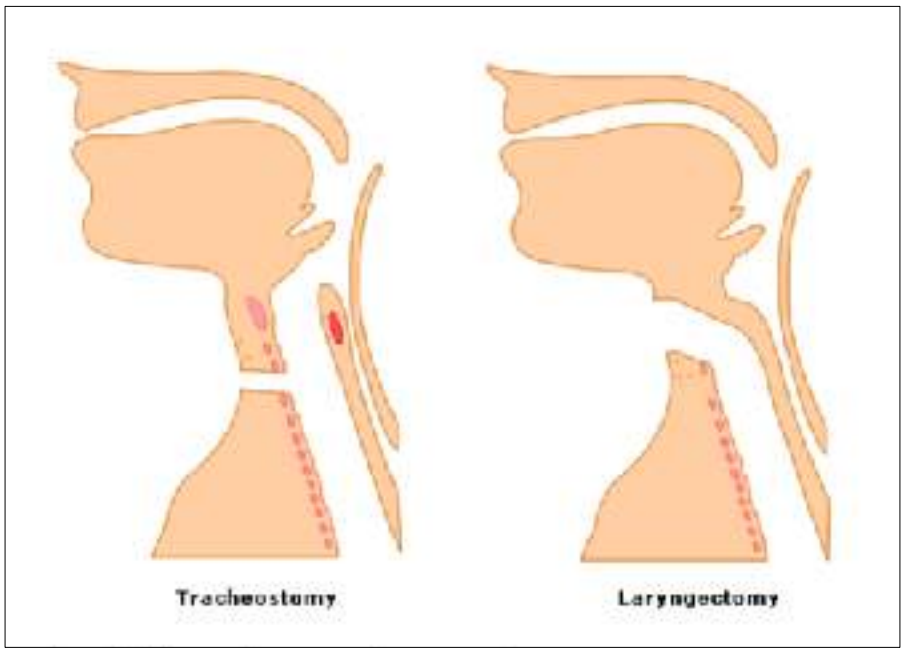


Figure 3.10: Tracheostomy versus laryngectomy

It is often very difficult to tell the difference by only externally examining the stoma. In recognition of this the National Tracheostomy Safety Project (NTSP) have designed 'bed-head signs' to be displayed in the patient's bed space which clearly describe what procedure the patient underwent (percutaneous / surgical tracheostomy or laryngectomy), when it was performed and what size tube is in place.

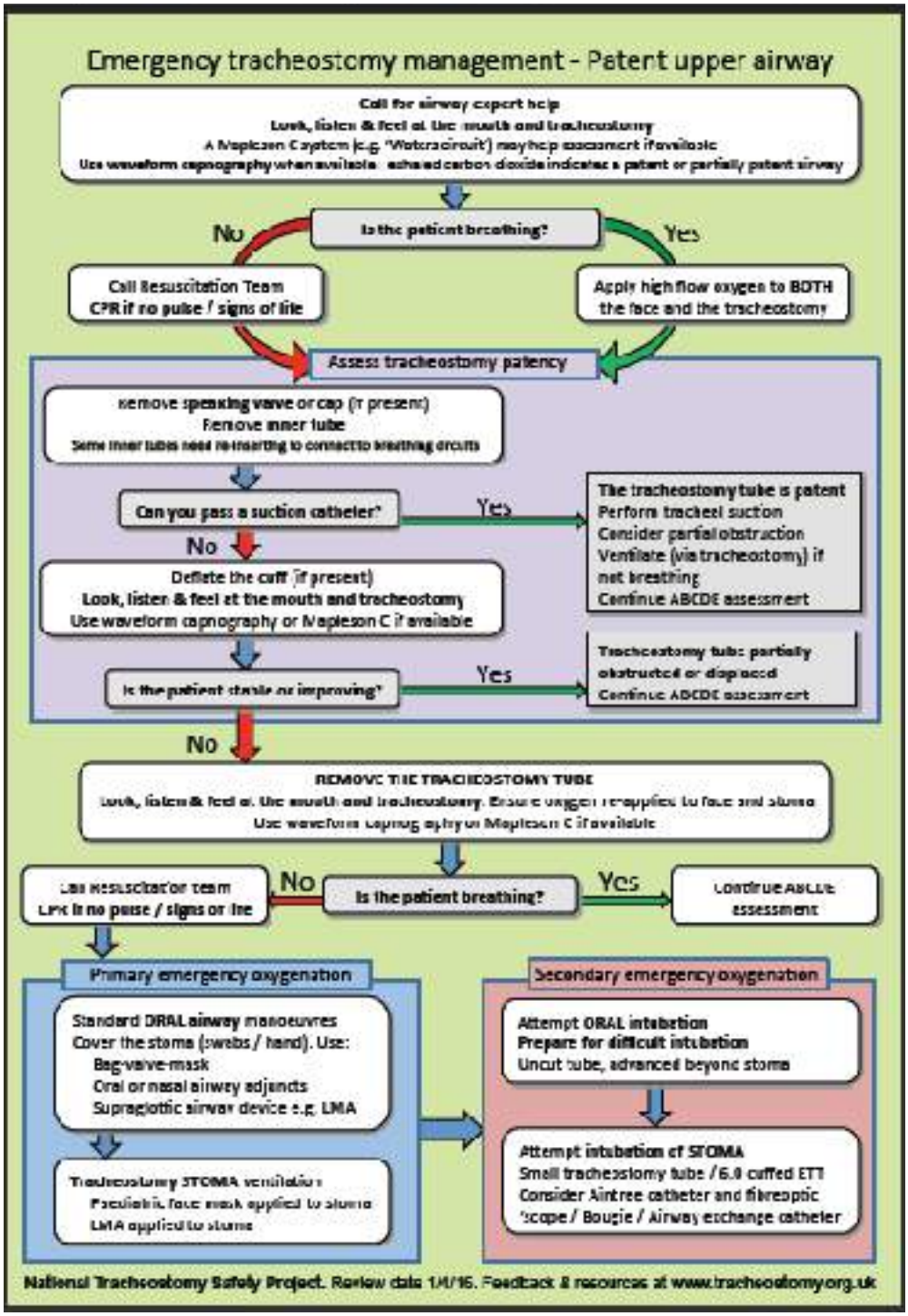
Complications

NHS Improvement receives approximately 600 tracheostomy related incidents per year with 60-70% of cases resulting in harm. The following are some of the potential complications.

- **Immediate:** Bleeding, tube misplacement, loss of upper airway, tube occlusion, pneumothorax, surgical emphysema
- **Delayed**: Tube displacement, tube blockage (usually due to secretions: can occur at any time), infection of stoma site or bronchial tree (pneumonia), tracheal ulceration or necrosis which can lead to stenosis or haemorrhage
- **Late**: Tracheal stenosis, tracheomalacia (collapse of the trachea), granulomata of the trachea

The NTSP have devised concise stepwise algorithms for the emergency management of a compromised airway in the tracheostomy and laryngectomy patient. (Figure 3.11).

The course of action to take should a tracheostomy tube become displaced will also depend on whether there is an established stoma i.e. will the passage remain patent if the tube falls out so that a tube can be easily re-inserted. The time taken to establish a reliable tract depends on the technique used; a surgical tracheostomy is usually established within 2 days, whilst a percutaneous stoma takes 7-10 days. Extreme care should be taken when re-inserting a displaced tracheal tube as it has the potential to cause bleeding, a false passage, tracheal injury and surgical emphysema all of which will further compromise an emergency situation.



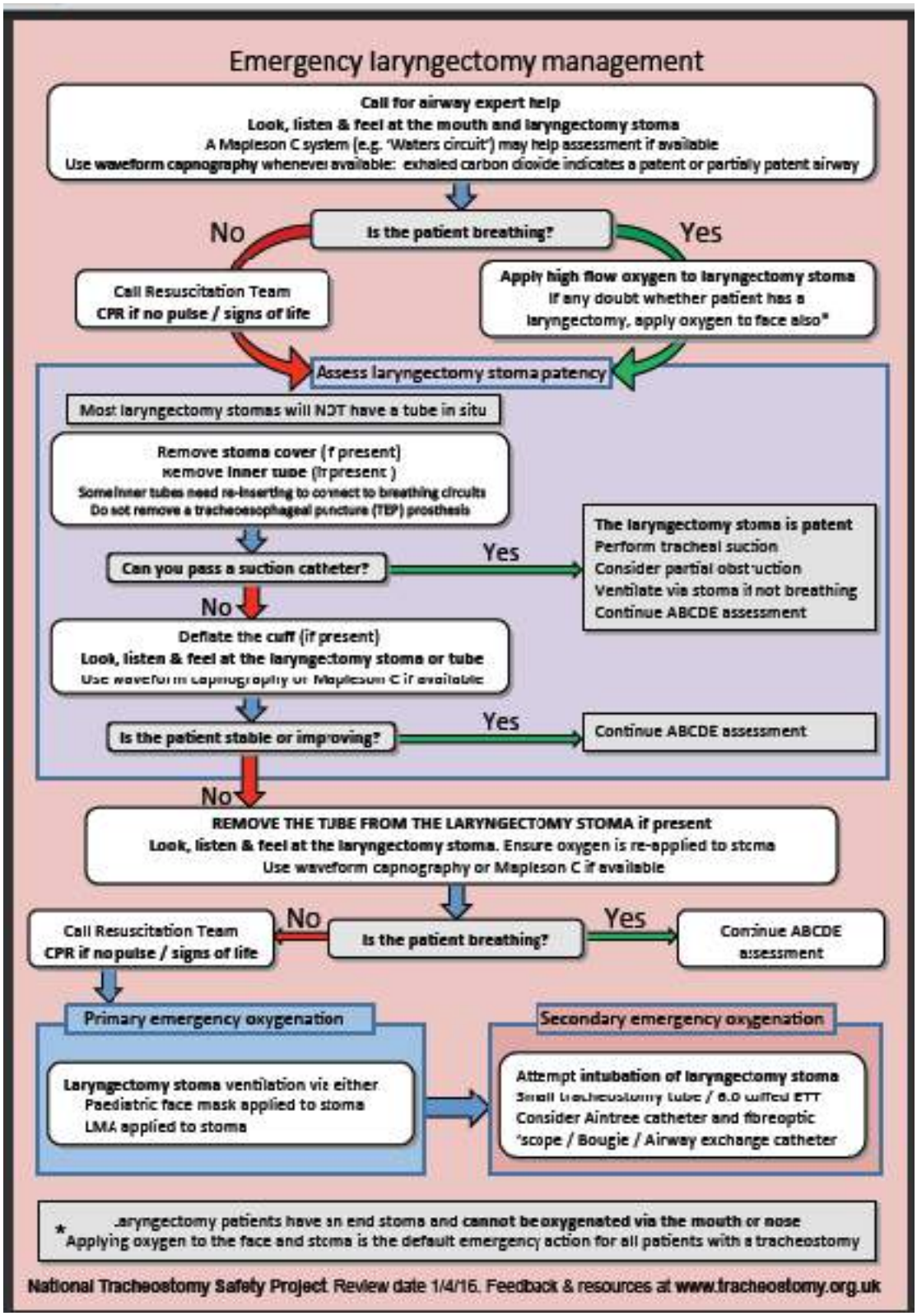


Figure 3.11: NTSP Emergency Tracheostomy and Laryngectomy management

http://www.tracheostomy.org.uk/Resources/Printed%20Resources/Patent%20Airway%20Algorithm.pdf

http://www.tracheostomy.org.uk/Resources/Printed%20Resources/Laryngectomy%20Algorithm.pdf

SUMMARY

- A patient who is able to speak indicates a patent airway (but does not exclude partial obstruction)
- Call for help early in the event of a compromised or difficult airway management
- Use of basic airway manoeuvres, adjuncts or a supraglottic airway device can be a life-saving intervention
- The mnemonic LEMON is a useful structure for a thorough airway assessment
- An intubating checklist is a useful tool to ensure all necessary equipment, drugs and personnel are available prior to intubation and that everyone knows what the plan is and who will perform what action in an emergency
- An airway strategy should be formulated and verbalised amongst the team prior to intubation
- Oxygenation is vital; intubation is secondary
- Familiarity with DAS and NTSP guidelines is essential
- Laryngectomy patients cannot be intubated, oxygenated or ventilated via the upper airway

FURTHER READING

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- Emergency Airway Management. Burtenshaw A, Benger J, Nolan J. Cambridge University Press. 2015
- Guidelines for the management of tracheal intubation in critically ill patients. Higgs et al. British Journal of Anaesthesia, 120 (2): 323e352 (2018)
- The TEAM Course (Training in Emergency Airway Management) is a high-fidelity simulation based course run by the Royal College of Anaesthetists and aimed at trainees within anaesthesia, intensive care and emergency medicine.
- Difficult Airway Society
- National Tracheostomy Safety Project

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CHAPTER 4: OXYGEN DELIVERY AND RESPIRATORY PATHOPHYSIOLOGY

Dr Ryan O'Leary and Dr Laura Troth

LEARNING OBJECTIVES

- To be able to describe the physiology of oxygen carriage and delivery, and to define and classify hypoxaemia
- To be able to describe lung capacities, their clinical relevance and the concepts of alveolar ventilation and dead space
- To define ARDS and describe its diagnosis, aetiology and management including rescue techniques for patients failing conventional ventilation

OXYGENATION

Lack of oxygen supply to body tissues, particularly the brain and heart, will have catastrophic effects in a very short time unless promptly recognised and addressed. In order to develop confidence with the management of hypoxaemia, one must first have a sound grasp of the underlying fundamental principles of how oxygen is delivered to the body tissues for utilisation in respiration, from the lungs down to the capillary beds. This pathway is better known as the **oxygen cascade,** summarised below in Figure 4.1.

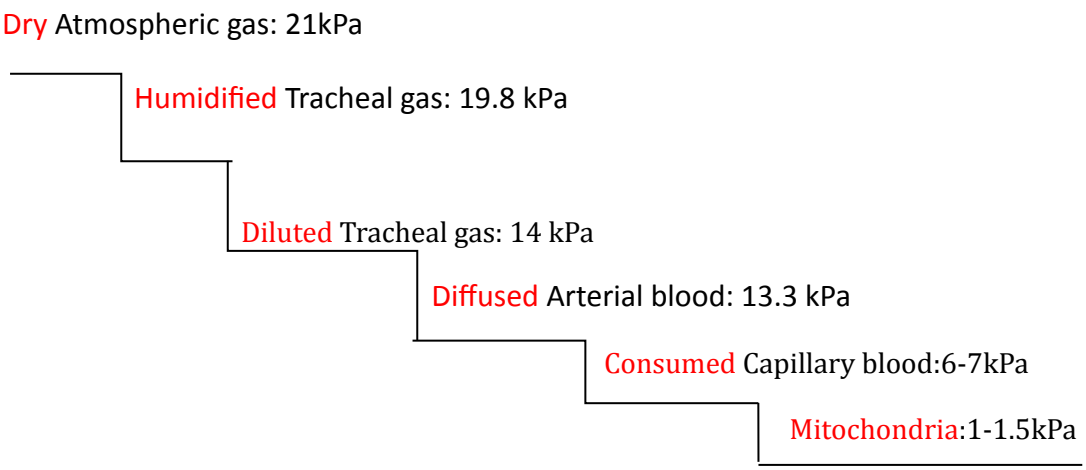

FIGURE 4.1: THE OXYGEN CASCADE

Oxygen in the air forms 21% of its total composition and at sea level, atmospheric pressure is 101.3 kPa. Therefore, the partial pressure of inhaled oxygen ($p0_2$) is approximately 21 kPa.

As oxygen enters the upper airways, it is warmed and humidified, an essential process to prevent the drying out of the tracheobronchial tree and ciliary dysfunction. Water vapour is now present in the inspired gas mixture, which drops the $p0_2$ slightly to 19.8 kPa.

As the inhaled gas mixture enters the alveolus, it is mixed with expired gases. The existing alveolar gas mixture that is rich in carbon dioxide and deficient in oxygen therefore dilutes the inhaled oxygen. From the alveolus, oxygen diffuses across the alveolar membrane and into the pulmonary capillary bed; the pulmonary capillaries will then coalesce to form the pulmonary veins that drain into the left atrium and form the basis of the oxygenated blood supply for the body. The resultant arterial $p0_2$ is usually between 10 and 13 kPa.

This oxygenated blood travels around the body through the arterial system. Throughout this journey, oxygen is extracted from the blood to meet the metabolic demands of the various tissues and therefore its oxygen content gradually falls. By the time it reaches the mitochondria of tissues to take part in aerobic respiration, the $p0_2$ will have fallen to between 1 and 1.5 kPa.

Now that the oxygen cascade has been explored, it is possible to look at the various mechanisms by which hypoxaemia can result, and strategies to correct these various deficits.

MECHANISMS OF HYPOXAEMIA

There are four different mechanisms of hypoxaemia and these can be diagnostically useful at the bedside:

- Low inspired oxygen concentration
- Alveolar hypoventilation
- Diffusion defects
- Ventilation/perfusion (V/Q) mismatch and shunt

It is helpful to consider the first two in tandem, as the mechanism by which hypoxaemia is produced in both cases is broadly similar. The **alveolar gas equation** helps to understand why both processes can ultimately lead to reduced oxygen content in the blood, and can be used to calculate the partial pressure of oxygen within the alveolus:

$$P_AO_2 = [F_iO_2 \times (P_{atm} - P_{H2O})] - (P_aCO_2 / RQ)$$

P_AO_2 = Arterial partial pressure of oxygen
P_aO_2 = Alveolar partial pressure of oxygen
F_iO_2 = Fraction of inspired gas that is oxygen
P_{atm} = Atmospheric pressure
P_{H20} = Partial pressure of water
RQ = Respiratory Quotient

The respiratory quotient (RQ) is the ratio of carbon dioxide production to oxygen consumption, typically 0.8. Therefore, the alveolar oxygen content is dependent on both the fraction of inspired oxygen, and the partial pressure of carbon dioxide within the alveolus.

Low inspired oxygen concentration, in practice and at sea level, is rare. It occurs when the gas mixture being delivered contains a lower percentage of oxygen than the usual 21% (and therefore a lower partial pressure as described above). As can be seen in the equation above, reducing the inspired oxygen content will therefore reduce the partial pressure of oxygen in the alveolus available for diffusion into the capillary bed. Here, assuming adequate ventilation, this can be corrected by increasing the inspired oxygen concentration.

Alveolar hypoventilation is significantly more common and can be encountered in many different scenarios. Neurological dysfunction, the effects of sedative drugs, and pre-existing respiratory diseases are a few possible causes, however any scenario which leads to a depression of normal respiratory function and hypoventilation will have the same effect. As ventilation decreases, the rate of clearance of carbon dioxide from the alveoli will fall, leading to a build-up of carbon dioxide, which in turn leads to a reduction in alveolar $p0_2$ and ultimately a reduced amount of oxygen available for diffusion into the pulmonary capillaries. In this case, the goal will be to aid carbon dioxide clearance from the alveoli and will require manual assistance with ventilation (usually through bag-mask ventilation in the absence of an advanced airway) and management of the precipitating cause, if possible.

Diffusion defects are another important cause of hypoxaemia, and in severe cases may be refractory to increasing the inspired oxygen concentration. Oxygen is less soluble than carbon dioxide, and therefore at the alveolar unit, it is harder to get oxygen into the blood than it is to get carbon dioxide out. Oxygen will travel down its concentration gradient from the oxygen-rich environment of the alveolus to the relatively oxygen-poor capillary blood and bind with haemoglobin, until equilibrium is reached between the two. This equilibrium will generally occur within approximately 0.25 seconds. Blood typically takes about 0.75 seconds to pass through the pulmonary capillary, allowing a degree of reserve. During times of

increased cardiac output this transit time is reduced and additionally, the oxygen demand of the body will increase. In health, this can generally be compensated for, as the diffusion time of oxygen will remain static at 0.25 seconds. However, if there is a defect seen at the alveolar-capillary interface restricting diffusion, such as pulmonary fibrosis, then the diffusion time of oxygen will be increased, and in severe disease processes the time taken for oxygen to diffuse may exceed the capillary transit time, even at rest, leading to hypoxaemia. In less severe cases, desaturation will be seen only on exercise, where cardiac output is increased and therefore the time for capillary transit is shortened. With severe diffusion defects, hypoxaemia may be only partially reversible. Avoidance of situations that lead to an increase in cardiac output is important in advanced disease states. Increasing inspired oxygen can be partially effective as it increases the diffusion gradient but will not completely resolve the hypoxaemia.

V/Q mismatching is a common form of hypoxaemia. It occurs where there is a failure of either alveolar ventilation such that blood passes through the pulmonary capillary system without picking up oxygen (**shunt**), or a failure of alveolar perfusion where oxygen in the alveoli cannot be passed into the blood (**increased physiological dead space**) as illustrated in Figure 4.2.

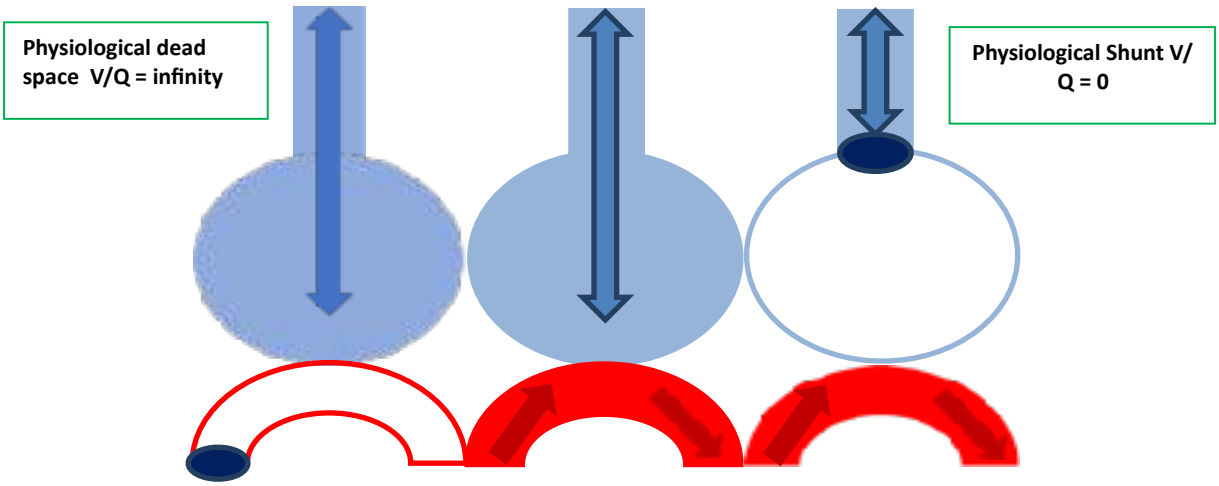

Figure 4.2: V/Q Mismatch

There is always a small amount of shunt present even within healthy lungs due to the drainage of venous blood from the bronchial circulation, as well as the lower parts of the lung receiving proportionally higher perfusion (due to the effect of gravity) than ventilation. Any process where there is impairment of normal alveolar ventilation can lead to an increase in shunting. Pulmonary consolidation is a good example: increased mucus and pus production by alveolar macrophages leads to physical blockage within the alveolar units and an inability for inspired gases to reach the alveoli. Therefore, the blood leaves the pulmonary capillaries still de-oxygenated, and mixes with blood returning from other, well-ventilated lung units in the pulmonary veins, leading to an overall reduction in oxygen content in the arterial

blood and resultant hypoxaemia. The body will attempt to compensate by a process known as **hypoxic pulmonary vasoconstriction**, which aims to divert blood away from under-ventilated lung units, towards those units with better V/Q ratios. In pure shunt, increasing the inspired oxygen content is unlikely to be enough to overcome this shunt, as the under-ventilated units will stay under-ventilated regardless of inspired oxygen and haemoglobin is already becoming fully saturated as it leaves the normal parts of the lung. Therefore, the mixture between well-oxygenated and poorly oxygenated blood will still occur, and hypoxaemia persist in spite of increasing the F_iO_2. Shunt reduction can be achieved in different ways depending on the precise underlying process. An increase in mean airway pressure can aid in recruitment of previously collapsed alveoli, therefore improving ventilation in these areas. This can be through the application of continuous positive airway pressure (CPAP) in the spontaneously breathing patient or the application of positive end-expiratory pressure (PEEP) in those with assisted ventilation. The same can also be beneficial in pulmonary oedema where the alveoli are filled with a transudate fluid, by creating a pressure gradient to promote the removal of this extra-vascular fluid. In the case of severe consolidation, where the fluid contained within the alveoli is exudative, simply increasing mean airway pressure will have some effect, but it is likely further manoeuvres will be required to further reduce shunting in the affected areas. Endotracheal intubation, suctioning, chest physiotherapy, bronchoscopy and postural drainage may all be required to promote mucociliary clearance, allowing the re-expansion of affected alveoli. Other causes of shunt can include extra-pulmonary compression, which promotes alveolar collapse and atelectasis, such as pneumothorax, pleural effusion, or abdominal distension (including obesity).

Increased physiological dead space occurs where the lung is being ventilated normally, but there is an impairment of pulmonary perfusion. With small defects such as sub-segmental pulmonary embolism, this may not manifest as hypoxaemia clinically, however with bigger defects, such as pulmonary emboli affecting a main pulmonary arterial branch, or saddle embolus, hypoxaemia may be profound. In the case of massive pulmonary embolism thrombolysis, interventional radiological intervention or even surgical intervention may be indicated. In the most severe cases, extra-corporeal membrane oxygenation (ECMO) may be required.

OXYGEN CARRIAGE AND DELIVERY

Hypoxic insult to the body tissues can be caused by other extra-pulmonary factors affecting the blood's ability to carry oxygen, or the body's ability to utilise it effectively. Before this can be fully understood however, knowledge of how the body carries oxygen is required.

Most of the oxygen carrying capacity of the blood is formed via haemoglobin. The actual contribution that dissolved, unbound oxygen makes to the oxygen content of the blood is low because it is such a small amount. Each haemoglobin molecule is capable of binding 4 molecules of oxygen in its fully saturated state. Once one molecule of oxygen has bound to haemoglobin it is easier for subsequent oxygen molecules to bind, giving rise to the sigmoid shape of the oxygen dissociation curve seen in Figure 4.3 below.

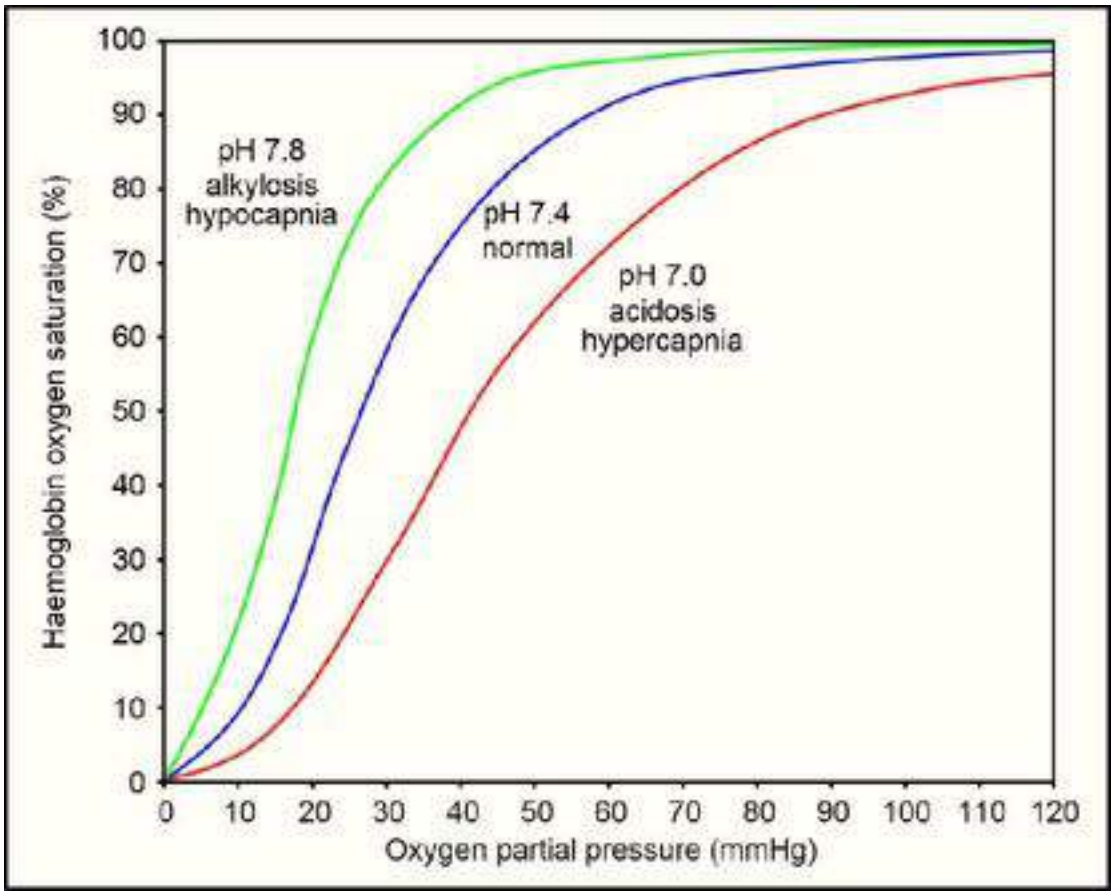

FIGURE 4.3: OXYGEN DISSOCIATION CURVE

Oxygen delivery is heavily dependent on the presence of functional haemoglobin - without this, tissue hypoxia is inevitable under normal atmospheric conditions, regardless of the inspired oxygen concentration or the presence of normal lungs.

TISSUE HYPOXIA

There are four main types of hypoxia that will be discussed:

- Hypoxaemic hypoxia
- Stagnant hypoxia
- Anaemic hypoxia
- Cytotoxic hypoxia

HYPOXAEMIC HYPOXIA refers to the arterial oxygen content of the blood being lower than normal, which will ultimately lead to a lower oxygen delivery to the mitochondria for respiration.

STAGNANT HYPOXIA occurs where there is a lack of adequate blood flow to the tissues - the oxygen content of the blood is normal, but the rate of delivery is such that the body is unable to get the well-oxygenated blood to the tissues for use by mitochondria, for example in cardiogenic shock. The key to treatment is restoring adequate blood flow.

ANAEMIC HYPOXIA relates to a lack of FUNCTIONING haemoglobin in either quantity or quality, therefore hugely reducing the blood's ability to carry oxygen effectively. Haemoglobin may be present, but unable to carry sufficient oxygen such as in carbon monoxide poisoning, where CO binds to haemoglobin with greater affinity than oxygen and forms carboxyhaemoglobin, rendering the haemoglobin unable to carry oxygen. As haemoglobin is unable to carry oxygen, the delivery of oxygen to the tissues falls significantly, leading to tissue hypoxia. Anaemic hypoxia also occurs in anaemia, where the haemoglobin present is functional, but in a vastly reduced quantity. The solution to this problem will depend on the process; in the case of a true anaemia, blood transfusion will raise the haemoglobin and therefore the oxygen-carrying capacity of the blood. In the case of CO poisoning, however, measures must be employed to reduce the amount of carboxyhaemoglobin present. The administration of 100% oxygen or early use of hyperbaric oxygen will significantly reduce the half-life of carboxyhaemoglobin.

CYTOTOXIC HYPOXIA relates to the situation where oxygenation in the lungs is adequate and oxygen carrying capacity of the blood is unaffected, but the mitochondria are effectively poisoned, and therefore unable to utilise the oxygen delivered to them. For example, in cyanide or carbon monoxide poisoning even if the oxygen delivery to the mitochondria is increased exponentially it will have no effect, as the mitochondria cannot utilise the oxygen. The only remedy for this situation is an antidote for the underlying poison, if one exists (e.g. hydroxycobalamin and sodium thiosulphate for cyanide).

VENTILATION

Now the process of tissue oxygenation has been covered, along with causes of hypoxia and potential solutions, it is prudent to look at the process of ventilation, specifically:

- Lung volumes and their clinical relevance
- Differences between spontaneous and mechanical ventilation
- Anatomical and physiological dead space

LUNG VOLUMES

As a point of basic revision, a volume refers to one measurement only, whereas a capacity refers to two or more lung volumes added together. The relevant lung volumes and capacities are detailed in Figure 4.4.

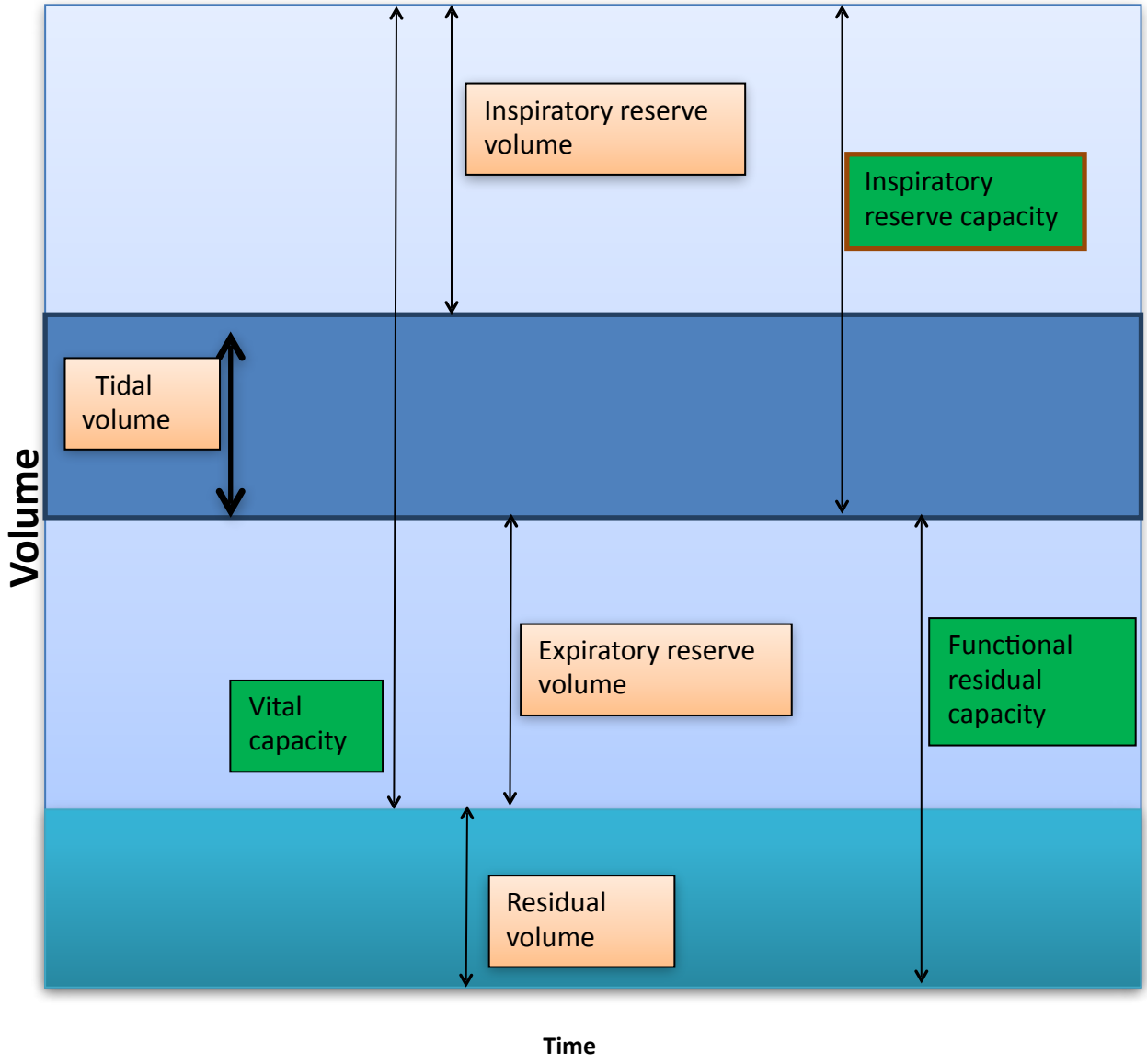

Figure 4.4: Lung Volumes

Tidal volume is the volume taken with each breath during normal ventilation. It is usually 6-8 ml/kg - approximately 500ml in a healthy adult subject. Tidal volume should be carefully considered when making adjustments on a mechanically

ventilated patient. Minute volume is the tidal volume multiplied by the respiratory rate i.e. the total amount of gas shifted by the patient per minute.

Expiratory reserve volume is the volume exhaled during a forced expiration (excluding the tidal volume). This has clinical relevance as it forms part of the **functional residual capacity** (which is the expiratory reserve volume and the residual volume added together). The residual volume is the volume left in the lungs after a full forced expiration that is permanently left in the lungs and does not take part in gas exchange. The functional residual capacity, therefore, is the amount of air left in the lungs at the end of a normal tidal volume. This has two important clinical applications. The first is the potential to provide a well-oxygenated reservoir prior to the induction of anaesthesia. This can be achieved by pre-oxygenating the patient with 100% oxygen. This high concentration of oxygen will wash out the nitrogen-rich gas mixture found in the lungs, replacing it with an oxygen-rich environment. Thus, when the patient is rendered apnoeic through the use of anaesthetic agents, the time taken for the patient to desaturate is significantly longer. This is particularly important in the intensive care environment, where patients are already physiologically less stable with higher oxygen consumptions and therefore more risk of desaturation. The second important concept is that reduced functional residual capacity (FRC) is present in many disease states that will be encountered. Many patients presenting to intensive care are likely to have a reduced functional residual capacity. This can be due to primary pulmonary pathology causing lung base collapse, intra-abdominal pathology causing diaphragmatic splinting, cardiac dysfunction, or the presence of pre-existing conditions such as obesity. As with reduced FRC, the time to desaturation is reduced.

ANATOMICAL AND PHYSIOLOGICAL DEAD SPACE

Dead space, within the lungs, is defined as the volume of gas within the lungs that does not take part in gas exchange. This can be either because the gas is located in an area incapable of gas exchange, or it is located within an alveolus that is not being perfused.

Anatomical dead space is the volume of gas contained within the conducting airways. Because it is found within the trachea, bronchi, and bronchial tree, which do not have alveolar tissue within them, it is incapable of taking part in gas exchange. This volume generally equates to 150ml of each breath in an average adult.

Physiological dead space is a combination of both alveolar dead space and anatomical dead space. Alveolar dead space is the amount of gas contained within alveoli that are ventilated but are not perfused. In healthy subjects, this occurs at the apices of the lung, which are under-perfused in the upright position due to the

effects of gravity (the lungs are preferentially better perfused at the bases in this position). This alveolar dead space is typically 20ml in a healthy adult, leading to a physiological dead space of 170ml, which needs to be subtracted from each tidal volume to understand what volume is taking part in gas exchange for each breath.

ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

ARDS is a well-recognised clinical entity that can complicate, or be the primary cause for, intensive care referral and admission. Its prevalence in UK intensive care units varies widely, with reports from 2.5% to 19% of ICU admissions being either caused by or complicated by it. It is characterised by widespread inflammation of lung parenchyma, with accompanying increased capillary permeability.

Diagnosis

The diagnostic criteria used are the Berlin criteria: all must be present for ARDS to be diagnosed:

- Onset within 7 days of a recognised clinical insult
- Bilateral opacities present on chest CT or chest radiograph not fully explained by pleural effusions, lobar or lung collapse or nodules
- Decreased P_aO_2/F_iO_2 ratio (<300mmHg or <40kPa) in presence of at least 5cmH_2O CPAP or PEEP
- Respiratory failure not attributable solely to cardiac failure or fluid overload

ARDS can be further sub-divided into mild, moderate and severe ARDS on the basis of the P_aO_2/F_iO_2 ratio:

• Mild:	26.7kPa - <40kPa	(200 - <300mmHg)
• Moderate:	13.3 - 26.6kPa	(100 - <200mmHg)
• Severe:	<13.3kPa	(<100mmHg)

Aetiology

ARDS can result either from a direct pulmonary insult, such as severe community-acquired pneumonia, or from an extra-pulmonary insult. Common extra-pulmonary causes of ARDS include severe sepsis, pancreatitis, burns, and severe trauma. Whatever the underlying cause, the pathophysiology is the same - high circulating levels of inflammatory mediators cause increased pulmonary capillary permeability, with a resultant widespread increase in extra-vascular lung water, inflammation of alveolar tissue, increased V/Q mismatching and resultant impairment of gas

exchange. This process is diffuse, occurring throughout the lung. Once significant lung inflammation has occurred, the injured lung tissue releases further inflammatory mediators, leading to further progression of the pulmonary injury and potential development of a systemic inflammatory response, leading to cardiovascular instability.

The 'baby lung' concept states that in a patient with ARDS, the proportion of lung tissue unaffected by the disease process and therefore containing normally aerated tissue is similar to that found in a 5-year-old child - approximately 300-500ml. This small amount of normally aerated tissue will behave in much the same way as normal lung tissue - which has important implications for the development of optimal ventilator management strategies for ARDS patients. Previous studies exploring high tidal volumes versus low tidal volumes have been stopped early due to clear evidence of higher mortality in the high tidal volume groups, thought to relate to the baby lung concept, and the predilection to barotrauma and volutrauma affecting the normally aerated areas of lung when higher tidal volumes are used.

Management

There are currently no drug treatments with any evidence of benefit in ARDS. Therefore, management of ARDS is directed towards providing optimal supportive care. The ARDSnet group has collated many studies and trials looking at various aspects of ARDS management to make best-practice recommendations and guidelines. These have been adopted by most UK intensive care units although in practice many units are still demonstrated to not adhere well to them.

Perhaps the most important concept is lung-protective ventilation. A large study published in the New England Journal of Medicine by ARDSnet in 2000 showed a clear mortality benefit when lower tidal volumes were used compared to higher tidal volumes (6 ml/kg versus 12 ml/kg of predicted body weight). Therefore, it is now best practice to use lower tidal volumes per kilogram of predicted body weight when ventilating patients with ARDS.

Sedation is required for almost all patients undergoing invasive mechanical ventilation to facilitate effective ventilation and reduce ventilator dyssynchrony. The use of neuromuscular blocking agents has been found to increase the later risk of development of intensive care unit acquired weakness. However, a trial in 2010 (ACURASYS, published in New England Journal of Medicine) showed a reduction in 90-day mortality when patients received an infusion of cisatracurium (a neuromuscular blocking agent) for the first 48 hours of their ARDS ventilation without higher rates of prolonged muscle weakness. The use of neuromuscular blocking agents is recommended when managing the patient with moderate-severe ARDS.

Fluid balance is important in the management of ARDS. One of the primary causes of pathology in ARDS is the presence of non-cardiogenic pulmonary oedema and large amounts of extra-vascular lung water - by this reasoning it would appear logical that avoiding largely positive fluid balances would be beneficial for recovery. Indeed, this is the practice in many intensive care units, despite a lack of clear evidence for reduction of mortality with conservative fluid strategies. A paper published by the ARDSnet group in 2010 showed a reduction in time that patients received mechanical ventilation, but no impact on mortality. A conservative fluid strategy is recommended for patients with ARDS, including the avoidance of large volumes of IV fluids, and indeed the promotion of diuresis and negative fluid balance.

Ventilator Strategies will be covered in chapter 5.

FURTHER READING

- Thomas the Tank Engine and Friends improve the understanding of oxygen delivery and the pathophysiology of hypoxaemia. Cosgrove JF, Fordy K, Hunter I, Nesbitt ID. Anaesthesia. 2006 Nov; 61(11): 1069-74
- Guidelines on The Management of Acute Respiratory Distress Syndrome. ICS & FICM ARDS Guideline - July 2018

CHAPTER 5: RESPIRATORY SUPPORT

Dr Gareth Sellors

LEARNING OBJECTIVES

- Understand different types of non-invasive respiratory support and indications for use
- Understand principles of mechanical ventilation & initial ventilator settings
- Describe different modes of mechanical ventilation and their clinical application

INTRODUCTION

Breathing is the co-ordinated contraction and relaxation of the muscles of respiration, causing cyclical pressure change within the respiratory tract, resulting in cyclical gas flow.

Spontaneous ventilation is the cyclical increase and decrease in volume of alveoli caused by the cyclical gas flow generated by breathing.

Mechanical ventilation uses a machine either to assist, or to replace spontaneous ventilation with the aims of:

- Achieving adequate alveolar ventilation
- Ensuring alveoli continue to perform exchange of oxygen and carbon dioxide
- Reducing the work of breathing
- Optimising the expansion of alveoli and preventing collapse
- Controlling the expansion of alveoli and preventing over-expansion
- Preventing excessive volume change in alveoli (volutrauma)
- Preventing excessive pressure from being applied to the respiratory system (barotrauma)

Mechanical ventilation is classified as non-invasive or invasive.

NON-INVASIVE VENTILATION (NIV)

This is a commonly used mode of respiratory support that has application for a wide range of causes of respiratory failure in many clinical areas. No sedation is required to apply NIV and the patient may eat, drink and communicate. NIV is easier to trial than invasive ventilation and may be an appropriate ceiling of escalation. NIV may be offered in a ward setting and within the intensive care unit.

Table 5.1: Classification of Non-invasive ventilation

Non-cyclical positive pressure NIV	Continuous Positive Airway Pressure	CPAP
	High Flow Nasal Cannulae	HFNC
Cyclical positive pressure NIV	Non-invasive positive pressure ventilation	NIPPV

NIV does not use an invasive airway device such as an endotracheal tube or a tracheostomy. The successful use of NIV is dependent upon the patient-machine interface. These interfaces commonly include hoods, facemasks and flexible, nasal cannulae (Figures 5.1-5.3).

Figure 5.1: Hood https://uk.intersurgical.com/info/literature

Figure 5.2: Mask

https://uk.intersurgical.com/info/literature

Figure 5.3: High Flow Nasal Cannulae
Images © 2019 Fisher & Paykel Healthcare Limited

Table 5.2: Indications for NIV

Hypercapnic acute respiratory failure in COPD with acidaemia (pH 7.25 to 7.35)	NIPPV
Hypercapnic respiratory failure secondary to chest wall deformity	NIPPV
Hypercapnic respiratory failure secondary to neuromuscular disease	NIPPV
Cardiogenic pulmonary oedema	HFNC CPAP NIPPV
Acute hypoxaemic respiratory failure without hypercarbia	HFNC CPAP
Obstructive sleep apnoea (OSA) and obesity hypoventilation syndrome	CPAP
Trans-nasal humidified rapid insufflations ventilatory exchange (THRIVE)	HFNC

CONTRA-INDICATIONS TO NIV

- Patient distress or refusal
- Respiratory arrest
- Impaired consciousness
- Upper airway obstruction
- Copious secretions
- Inability to clear secretions
- Pneumothorax or significant bullous lung disease
- Major facial trauma
- Presence of high upper GI anastomosis
- Vomiting and bowel obstruction
- Significant airway or upper GI haemorrhage
- Haemodynamic instability

Safe and successful use of NIV requires skilful application and vigilance for the complications.

COMPLICATIONS OF NIV

- Patient distress
- Poorly-fitting interface leading to leaks and ineffective support of ventilation
- Facial abrasions
- Ocular abrasions
- Drying of mucous membranes
- Nasal trauma and epistaxis
- Increased intraocular pressure
- Increased intracranial pressure

- Aerophagia and gastric distension
- Hypotension
- Delay in recognising need for endotracheal intubation may lead to increase in mortality

CPAP AND HFNC

Confusion has arisen because CPAP is both a physiological concept and a technique of respiratory support.

As a physiological concept, the term CPAP should not be used interchangeably with PEEP. CPAP is the application of positive airway pressure throughout the respiratory cycle. PEEP refers to a specific point-of-time and is the presence of positive airway pressure at end-expiration.

As techniques of respiratory support, CPAP and HFNC both allow the application of PEEP to spontaneously ventilating patients. In contrast to NIPPV, CPAP and HFNC do not provide a driving pressure above end-expiratory pressure.

CPAP

A common form of CPAP respiratory support uses high-pressure oxygen through a precision Venturi device to generate a high flow (20-155 litres per minute) of oxygen and air mixture. This flow reaches a spontaneously breathing patient via a flexible hose and a mask or hood. The circuit is pressurised using a CPAP valve on the outflow tubing. Commonly used valves may generate 2.5 to 10 cmH_20 of circuit pressure and the system usually incorporates a second 20 cmH_20 valve that acts as a safety valve should the main CPAP valve fail. To achieve adequate circuit gas supply to match required peak inspiratory flow, circuit gas flow is titrated so that there is a flow of gas through the CPAP valve even during inspiration.

Three key safety feature points must be considered. The pressure within the CPAP circuit needs to be monitored to ensure accuracy of pressure delivery. A safety CPAP valve or equivalent must be incorporated to ensure no overpressure in the event of primary valve malfunction. There should be a means of entraining room air should the patient's peak inspiratory flow exceed that supplied by the circuit.

CPAP provides a more precisely controlled level of CPAP than HFNC. CPAP respiratory support uses large volumes of medical oxygen and is therefore difficult in transit.

Humidification is less reliable than with HFNC and is not possible using a hood interface.

HIGH FLOW NASAL CANNULAE (HFNC)

HFNC provide high flows of heated, humidified air and oxygen mixtures to spontaneously ventilating patients. An electric motor draws oxygen and filtered room air over a humidifier and into a heated breathing tube. The patient interface is a pair of soft, flexible nasal prongs with a head strap for secure attachment. HFNC may deliver a high FiO_2. Heating and humidification preserve mucosa and mucociliary function. HFNC causes a variable, uncontrolled and unmeasured CPAP effect depending upon the flow rate selected, the patient interface and patient factors.

Emergency intubation of hypoxaemic patients and intubation of patients with difficult airways can risk profound hypoxaemia. THRIVE is trans-nasal rapid insufflations ventilatory exchange. There is increasing evidence that use of HFNC for pre-oxygenation and continuing HFNC after the administration of induction and neuromuscular blocking agents leads to aventilatory mass flow (AVMF) and successful apnoeic oxygenation. In the near future, THRIVE might form part of routine process during intubation of the critically ill.

NIPPV

Cyclical, positive pressure above end-expiratory pressure is synchronised with the patient's respiratory effort resulting in increasing tidal volumes and alveolar ventilation. A commonly used mode is pressure support ventilation (PSV) with CPAP. The most commonly used patient-machine interface is the facemask, although hoods and visors are available. NIPPV is an intermittent technique. When the need for NIPPV support becomes permanent, then interface tolerance of the mask limits success. Failed NIPPV may require invasive ventilation, although frequently NIPPV represents the limit of escalation for clinical reasons. Randomised trial evidence shows there are significant benefits in the prompt application of NIPPV in patients with acute exacerbation of COPD. NIPPV reduces endotracheal intubation, length-of-stay, complications and mortality. NIPPV patients are commonly managed within a respiratory ward setting.

INVASIVE VENTILATION

Invasive ventilation requires an invasive airway; examples include endotracheal tubes, nasotracheal tubes and tracheostomies.

During invasive Intermittent Positive Pressure Ventilation (IPPV), a cyclical ventilator pressure through an invasive airway causes bulk flow of gas to the alveoli.

Primary ventilator controls are given in Table 5.3.

Table 5.3: Primary ventilator controls

Positive End Expiratory Pressure	PEEP	
Breath control variable	Tidal volume	VCV
	Inspiratory pressure	PCV
Respiratory rate	RR	
Oxygen concentration	FiO_2	
Timing	Inspiratory time +/- pause	
	Expiratory time	
	I:E ratio	
Mode of ventilation	See classification given in **TABLE 5.5**	

POSITIVE END EXPIRATORY PRESSURE (PEEP)

Retention of alveolar volume plays a key role in preservation of oxygenation and the prevention of alveolar collapse. Optimal PEEP is challenging to decide and is achieved when there is maximal oxygenation, with minimal end-expiratory atelectasis, minimal alveolar over-expansion and preserved oxygen delivery. A typical initial PEEP is 5cm H_20, however this may need to be increased significantly in certain conditions.

POTENTIAL BENEFITS OF PEEP

- Promotion of "open lung" by recruiting collapsed alveoli
- Increase in functional residual capacity
- Reduction in intra-pulmonary shunt
- Redistribution of lung water from alveoli
- Reduced lung injury by preventing cyclical closure of alveoli
- Improvement of left ventricular function in heart failure due to reduction in wall tension

POTENTIAL DOWNSIDES OF PEEP

- Alveolar over-distension
- Decreased venous return and cardiac output with possible reduction in oxygen delivery

POSSIBLE STRATEGIES FOR "OPTIMAL" PEEP

1. Refer to the ARDSNet PEEP/ FiO_2 tables given in table 5.4
2. Set an arbitrarily high PEEP in the range 15 to 20cmH_20
3. Titrate PEEP to maximum compliance
4. Set PEEP slightly above lower inflection point of pressure/volume loop
5. Perform a Stairways Recruitment Manoeuvre (SRM)
6. Target a reduction in intrapulmonary shunt using PAFC and SvO_2 monitoring

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It should be noted that the available evidence for some of these strategies that are frequently described is conflicting; not all may be shown to be safe and efficacious although used in current clinical practice.

Table 5.4: ARDSNET PEEP Tables

Strategy 1		**Strategy 2**	
Lower PEEP and higher FiO_2		**Higher PEEP and lower FiO_2**	
FiO_2	PEEP	FiO_2	PEEP
0.3	5	0.3	5
0.4	5	0.3	8
0.4	8	0.3	10
0.5	8	0.3	12
0.5	10	0.3	14
0.6	10	0.4	14
0.7	10	0.4	16
0.7	12	0.5	16
0.7	14	0.5	18
0.8	14	0.5-0.8	20
0.9	16	0.8	22
0.9	18	0.9	22
1.0	18-24	1.0	22
		1.0	24

BREATH CONTROL VARIABLE

VOLUME-CONTROL VENTILATION (VCV) delivers a specified inspiratory tidal volume (V_T). With a set V_T, inspiratory pressure will vary with compliance and resistance of the respiratory system.

PRESSURE-CONTROL VENTILATION (PCV) delivers a specific inspiratory pressure. With a set inspiratory pressure, V_T will vary with compliance and resistance of the respiratory system. Minute ventilation is less stable in PCV compared to VCV.

Desired tidal volume will depend upon presenting pathologies and subsequent pathophysiology.

VENTILATOR-ASSOCIATED LUNG INJURY (VALI) has several components: hyperinflation and shearing (volutrauma), alveolar rupture and pneumothorax (barotrauma) and the release of inflammatory mediators (biotrauma). Low tidal volume ventilation reduces VALI, and therefore low tidal volumes are preferred in the range 4-8mlkg^{-1} predicted body weight.

LUNG PROTECTIVE VENTILATION (LPV) is the current standard of care for mechanical ventilation. LPV uses low tidal volumes of 6mlkg^{-1} predicted body weight. This figure was selected from the intervention arm of the influential ARDSNet clinical trial.

RESPIRATORY RATE

An appropriate starting range is 12 to 16 breaths per minute in an adult patient. Patients with a very high metabolic rate, a severe metabolic acidosis or severe ARDS ventilated with low tidal volumes may require a considerably higher respiratory rate.

$$Minute\ ventilation = tidal\ volume \times respiratory\ rate$$

Minute ventilation affects P_aCO_2 and therefore appropriate respiratory rate is a key control in achieving CO_2 targets.

FRACTIONAL INSPIRED OXYGEN CONCENTRATION FIO_2

Select the lowest possible FiO_2 to meet the oxygenation goals avoiding complications such as absorption atelectasis and oxygen toxicity. Oxygenation goals vary depending on clinical context e.g. SpO_2 88-95% for ARDS; SpO_2 post cardiac arrest management 94-98%.

TIMING

INSPIRATORY FLOW TIME is the period of inspiratory flow.

INSPIRATORY PAUSE TIME is the period during which the lungs are held inflated but there is neither inspiratory nor expiratory flow. The aim of the inspiratory pause is to allow better gas distribution.

INSPIRATORY TIME

$$Inspiratory\ time = inspiratory\ flow\ time + inspiratory\ pause\ time$$

EXPIRATORY TIME is not set and is the time between the end of one inspiration and the beginning of the next.

I:E RATIO is the ratio of inspiratory time to expiratory time. I:E ratio is usually set at 1:2. A higher ratio, for example 1:1 may result in an increase in mean airway pressure and better oxygenation, but at the expense of a shorter expiratory time. If expiration is too short, expiration may be incomplete leading to breath stacking and an increase in end-expiratory pressure.

INVERSE RATIO VENTILATION (IRV) describes the use of an inspiratory time greater than expiratory time (I:E ratio > 1:1). IRV leads to an increase in mean airway pressure and may lead to improved V/Q matching and, in turn, oxygenation. Risks include dynamic hyperinflation, barotrauma and cardiovascular instability. To tolerate IRV, the patient may require sedation and neuromuscular paralysis.

Table 5.5: Classification of ventilator modes

Breath initiation	Machine-initiated	
	Patient-initiated	Pressure-triggered
		Flow-triggered
Cycling to expiration	Machine-controlled	Time-cycled
	Patient-controlled	Flow-cycled
Breath sequence	Continuous Mandatory Ventilation CMV	
	Intermittent Mandatory Ventilation IMV	SIMV
		APRV
	Continuous Spontaneous Ventilation CSV	ASB or PSV
Additional controls	Adaptive control	PRVC Autoflow
	Servo control	Automatic Tube Compensation

BREATH INITIATION

MACHINE-INITIATED BREATHS are initiated at a set time determined by the respiratory rate.

PATIENT-INITIATED BREATHS are initiated by the patient's inspiratory effort. Patient-initiation may be either triggered by pressure or triggered by flow.

PRESSURE TRIGGERING: The ventilator monitors the circuit pressure in expiration. A reduction in pressure to a pre-set threshold caused by the patient breathing in triggers inspiration.

FLOW TRIGGERING**:** The ventilator maintains a base flow of gas through the ventilator circuit. When measured inspiratory flow due to patient inspiratory effort reaches a pre-set threshold inspiration is triggered. This is generally more sensitive and more specific than pressure triggering, resulting in earlier support of patient-initiated breaths and fewer false triggers.

CYCLING TO EXPIRATION

In time cycling, an inspiratory time is specified. When this time has elapsed, the ventilator switches to expiration.

In flow cycling, the expiratory trigger is based upon measurement of inspiratory flow. An example would be when inspiratory flow falls to 25% of peak inspiratory flow, the ventilator switches to exhalation.

BREATH SEQUENCE

MANDATORY BREATH: a breath in which the timing and/or the size of the breath is controlled by the ventilator.

CONTINUOUS MANDATORY VENTILATION (CMV): Every inspiration is a mandatory breath. Spontaneous breaths are not permitted between mandatory breaths. The respiratory rate is chosen and determines the frequency of mandatory breaths.

CMV may be either volume-controlled (VC-CMV) or pressure-controlled (PC-CMV).

Depending upon manufacturer, CMV may also be called Assist-Control, Volume Control or IPPV.

SYNCHRONISED INTERMITTENT MANDATORY VENTILATION (SIMV): Respiratory support is partitioned between spontaneous and mandatory breaths. SIMV permits spontaneous breaths between mandatory breaths and with an active exhalation even during mandatory breaths. The respiratory rate chosen directly affects the frequency of mandatory breaths. The actual frequency of mandatory breaths will not exceed this setting.

SIMV may be either volume-controlled (VC-SIMV) or pressure-controlled (PC-SIMV).

PRESSURE SUPPORT VENTILATION (PSV): Pressure-controlled mode in which each breath is patient-initiated and patient-terminated (flow-cycled). PSV does not provide mandatory breaths (although modern ventilators have an apnoea alarm such that

the ventilator will switch to a mandatory backup mode if parameters fall below a set threshold). The patient determines the respiratory rate.

SIMV WITH ASB/PSV: These modes are frequently used in combination allowing support of spontaneous breaths occurring between mandatory breaths.

AIRWAY PRESSURE RELEASE VENTILATION (APRV): A pressure-controlled, time-cycled mode of ventilation used in spontaneously breathing patients. This mode is characterised by relatively long periods of positive airway pressure interrupted by regular, short pressure releases. Unrestricted spontaneous breathing may occur throughout any part of the respiratory cycle.

ADDITIONAL CONTROLS

PRESSURE REGULATED VOLUME CONTROL (PRVC) AND AUTOFLOW™

These controls are used in volume control modes. Rather than use a constant flow, adaptive control algorithms automatically adjust the inspiratory flow to deliver the chosen tidal volume at the lowest pressure.

AUTOMATIC TUBE COMPENSATION (ATC)

ATC uses servo control algorithms that calculates the airway resistance of the tracheal tube or tracheostomy and then uses this output to generate enough pressure (in proportion to the inspiratory or expiratory flow) to overcome the additional resistance load of the airway.

THE EQUATION OF MOTION

If the respiratory system is modelled as a single resistance connected in series with a single capacitor, ventilatory pressure applied at the airway opening (P_{ao}) has three components which are related by the equation of motion: elastic load, resistance load and PEEP (represented here as P_o) (see Table 5.6).

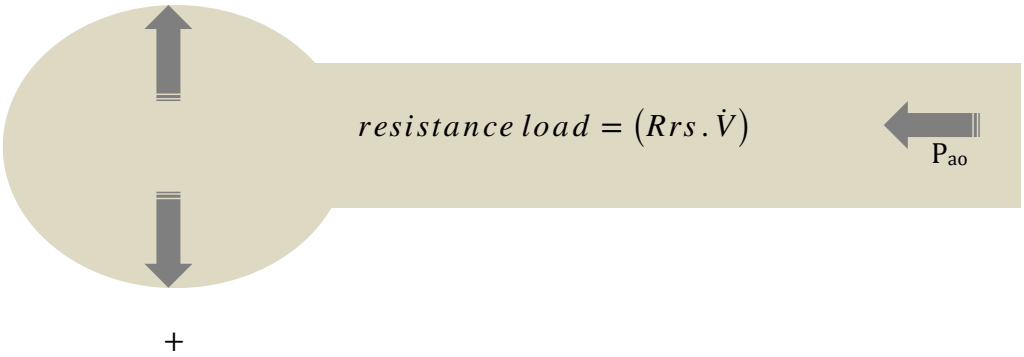

Figure 5.4: Equation of Motion

$$\underline{Pao = \text{elastic load} + \text{resistance load} + PEEP}$$

$$Pao = \left(\frac{V}{C}\right) + \left(Rrs \,.\, \dot{V}\right) + Po$$

Pao = ventilatory pressure applied at airway
V = volume
$\dot{V}$ = flow
Po = total PEEP
C = compliance
Rrs = resistance of respiratory system

The equation of motion (Figure 5.4) demonstrates that within this model only one variable can be controlled at a time either pressure, volume or flow.

Table 5.6: Components of the Equation of Motion

Elastic load	Resistance load	PEEP
Pressure to expand lungs and chest wall	Pressure to deliver gas at required flow	Positive end-expiratory pressure
$\text{elastic load} = \left(\frac{V}{C}\right)$ $V = \text{volume}$ $C = \text{compliance}$	$\text{Resistance load} = Rrs \,.\, \dot{V}$ $Rrs = \text{resistance of respiratory sys}$ $\dot{V} = \text{flow}$	Po

PEAK PRESSURE

Peak airway pressure is measured when there is inspiratory airflow in the circuit during the inspiratory flow time. The resistance load predominantly influences peak airway pressure. High peak pressure indicates a high imposed resistance load by factors such as bronchospasm, retained secretions and ETT obstruction.

PLATEAU PRESSURE

Plateau pressure is measured when there is not inspiratory airflow in the circuit at the end of the inspiratory pause. The elastic load predominantly influences plateau pressure. High plateau pressure indicates reduced compliance of the respiratory system. Lung compliance is the key concern and plateau pressure may be elevated in pathologies such as pulmonary oedema, ARDS and pneumonia. The ARDSNet trial targeted a plateau pressure of less than 30cmH_20, although more recent publications suggest that the plateau pressure is less important than the driving pressure, which is essentially the difference between the plateau pressure and the PEEP. In practice it is calculated by dividing tidal volume by measured compliance, with evidence suggesting harm at driving pressures > 14kPa.

MEAN AIRWAY PRESSURE

Mean airway pressure is the average pressure during a complete respiratory cycle. Mean airway pressure is not set but depends on factors including: peak airway pressure, plateau pressure, PEEP, respiratory rate, I:E ratio and contour of the pressure waveform. The mean airway pressure is calculated by the ventilator using the area under the pressure-time curve. Mean airway pressure is used as a surrogate marker of mean alveolar pressure. Increasing mean alveolar pressure may improve V/Q matching and can be related to improved oxygenation. A mean airway pressure of more than 20 cmH_20 may indicate significant lung pathology and indicate the need for advanced respiratory management.

FURTHER VENTILATORY STRATEGIES

PRONE VENTILATION

Prone ventilation improves oxygenation in the ventilated patient by promoting pulmonary blood flow through well-expanded, anterior lung regions that are now in the dependent position. Patients with severe ARDS have been shown to have lower mortality with early initiation of 12-18-hour proning sessions. (click here) As a procedure there is a risk of line or tube dislodgement, damage to pressure areas, promotion of cardiovascular instability, or limb damage, but recent evidence suggests that it remains an underutilised strategy. It requires careful co-ordination with an experienced team, with advanced planning should an adverse event occur.

HIGH FREQUENCY OSCILLATORY VENTILATION (HFOV)

HFOV is an unconventional mode of ventilation that does not rely on bulk transfer of gas. HFOV requires specialised ventilators and applies controlled positive pressure and adds an oscillatory pump to cause cyclical pressure variation within the airway. HFOV may be described as a “vibrating CPAP”. HFOV is characterised by low V_T (less than dead space) and high frequency (3-15Hz). Its use has diminished following two large publications that concluded it was either no better than more conventional ventilation, or worse, increased mortality.

CHAPTER 6: CARDIOVASCULAR PATHOPHYSIOLOGY

Dr Irmeet Banga and Dr Niro Karunasekara

LEARNING OBJECTIVES

- Understand basic concepts of cardiovascular physiology
- Understand principles of how the cardiovascular system can be compromised by illness
- Be aware of treatment modalities in the shocked patient

INTRODUCTION

Knowledge of the determinants of tissue perfusion is essential in understanding the pathophysiology of shock and how to manipulate dysfunctional physiology in the shocked patient. In this section there are basic concepts governing tissue perfusion, different shock states encountered in critical illness and monitoring of shock from clinical signs through to advanced techniques on the ICU.

BASIC CONCEPTS

CARDIAC OUTPUT

Cardiac output (CO) is the product of Stroke Volume (SV) and Heart Rate (HR) as expressed in the equation:

CO = HR x SV

Changes in either parameter alter CO.

STROKE VOLUME AND ITS DETERMINANTS

Stroke volume (ml) is the volume of blood ejected by each ventricular contraction and is determined by **afterload**, **pre-load** and **contractility**. These can all be manipulated in the ICU.

PRELOAD refers to the length of cardiac muscle fibre immediately prior to cardiac contraction; it is analogous to the End Diastolic Volume (EDV). Surrogate pressure

markers such as Central Venous Pressure (CVP) or Pulmonary Artery Occlusion Pressure (PCWP/PAOP) correspond poorly to actual EDV and their use for this purpose continues to decline.

Increasing EDV, and so cardiac output, with fluid therapy as described by the **Frank-Starling law** of the heart is described later.

AFTERLOAD refers to the tension needed in cardiac muscle fibres before shortening (contraction) happens so is a measure of the resistance fibres have to overcome to contract. The surrogate clinical marker is the Systemic Vascular Resistance (SVR).

CONTRACTILITY refers to the ability of cardiac muscle fibres to contract, taking into account the preload supplied and afterload to overcome. It is also affected by the autonomic nervous system and catecholamines.

FRANK STARLING LAW OF THE HEART

The strength of myocardial contractility is proportional to the initial length of cardiac muscle fibre. Using the analogy of end diastolic volume to preload and fibre length, the greater the EDV, the greater the stroke volume and so the greater the cardiac output (Figure 6.1). This mechanism is important in maintaining equity in right and left ventricular output but is also an important principle in terms of understanding the role of preload in manipulating cardiac output including using changes in cardiac output as a one element in the assessment of fluid management.

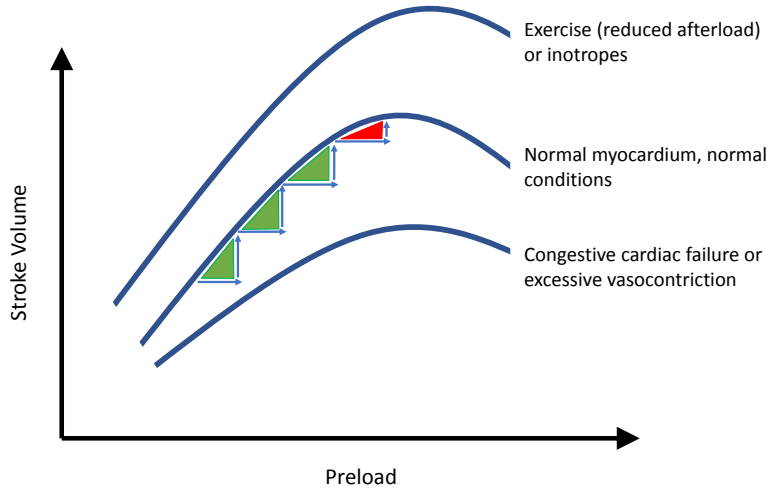

Figure 6.1: Frank-Starling Law of the Heart

There is a finite SV a ventricle can achieve even in the non-diseased heart. Positive inotropy in addition to fluid loading can achieve even higher cardiac outputs, effectively moving the curve higher in this diagram. Over-zealous fluid administration in patients with impaired ventricular function can accelerate cardiac failure. The graph illustrates how fluid responsive patients demonstrate significant increases in cardiac output following fluid challenge, how this incremental increase diminishes as optimal fluid status is approached, and the potential for reducing cardiac output in the context of excessive fluid administration. Just as under resuscitation harms patients, over administration of fluid can cause damage. The different curves demonstrate how different physiological conditions, such as the normal heart under exercise conditions, the effect of exogenous inotropes or the presence of congestive heart failure influence the response to preload.

Determinants of Mean Arterial Pressure

Mean Arterial Pressure (MAP) is the product of CO and SVR (a surrogate marker of afterload). This can be expressed in the following equations:

MAP (mmHg) = CO x SVR = HR x SV x SVR

SVR = [(MAP-CVP)/CO] x 80

The SVR is a derived value available from cardiac output monitoring that can assist with titration of vasopressor therapy.

MAP can also be approximated as Diastolic Blood Pressure (DBP) in mmHg plus 1/3 of the difference between Systolic Blood Pressure (SBP) and DBP:

MAP = DBP + 1/3(SBP-DBP)

This mathematical calculation is less helpful in understanding cardiovascular physiology than the first equation. The former describes parameters that become deranged and can be manipulated by our treatment.

OXYGEN DELIVERY

(Please see respiratory physiology section for oxygen cascade (Figure 4.1) and oxygen-haemoglobin dissociation curve (Figure 4.3))

The ultimate aim of cardiac output monitoring is to enable adequate oxygenation of end-organ tissues.

Oxygen Delivery to the tissues is expressed as $\boldsymbol{DO_2}$. This is a product of cardiac output and arterial oxygen content of blood (C_aO_2). C_aO_2 reflects oxygen dissolved in blood as well as oxygen bound to haemoglobin.

DO_2 (ml/min) = CO x C_aO_2 x 10

VO2 reflects **oxygen consumption** at the tissues; it is normally 200ml/min. In normal circumstances DO_2 vastly outstrips VO_2. However, in critically ill patients VO2 can be markedly increased and the DO_2 can fall below VO2 resulting in tissue hypoxia with anaerobic respiration and consequent lactic acidosis.

ADRENOCEPTORS

It is essential to understand the main receptor types that modulate the effects of catecholamines, as these are the commonest therapeutic targets in the critically ill patient.

Adrenoceptors are G-protein coupled cell membrane receptors activated via catecholamines. Endogenous catecholamines include: adrenaline, noradrenaline and dopamine. They exert their biological effect via intracellular 2nd messenger systems. They are broadly divided into α and β receptors, each with sub-divisions:

Table 6.2: Adrenoceptor Classification and Action

Receptor	Distribution	Effect of Stimulation (Agonism)
α1	Vascular smooth muscle	Vasoconstriction
α2	Central nervous system	Sedation and analgesia
β1	Platelets, Heart	Platelet aggregation, inotropy and chronotropy
β2	Bronchial and vascular smooth muscle	Smooth muscle relaxation
β3	Fatty tissues	Lipolysis

Shock is a pathological problem: it is a state of cellular and tissue hypoxia due to reduced oxygen delivery ± increased oxygen consumption or inadequate utilisation. Some of the measures used in intensive care to assess end-organ perfusion and aerobic metabolism include the following:

$S_{CV}O_2$

This is a measure of venous saturation in a jugular central venous sample ($S_{cv}O_2$) as a surrogate marker of the balance/flux of DO_2 and VO_2 and adequacy of tissue oxygenation.

Normal oxygen extraction at the tissues is 25-30% resulting in a $S_{cv}O_2$ of 65%.

Sc*V*O$_2$ <65% implies inadequate tissue oxygen delivery
Sc*V*O$_2$ >80% implies inadequate tissue oxygen extraction

LACTATE

Inadequate DO_2 leads to anaerobic tissue respiration and increased lactic acid production. Lactate (as measured by arterial blood gas samples) is utilised as a marker of adequacy of tissue perfusion. Important to remember high lactate can arise due to other reasons as well (e.g. β-adrenoreceptor stimulation, reduced clearance).

High lactate is a marker of illness severity and associated with worse outcomes. Effective lactate clearance has been associated with better prognosis. It has been proposed as a measurement of adequacy of resuscitation and included in sepsis management bundles for this purpose, but this link is controversial.

THE GLYCOCALYX AND MICROVASCULAR CHANGES IN SHOCK

Vascular endothelium has a proteinaceous covering known as the glycocalyx. It has a wide variety of postulated functions including a role in endothelial integrity. The glycocalyx is sheared off the endothelium as a consequence of insults such as trauma, septic shock and hypervolaemia, and this leads to a loss of vessel integrity, capillary leak and consequent oedema. Steroids have a postulated role in avoidance of damage to the glycocalyx, as does avoidance of hypervolaemia.

There is a strong association between reduction of microcirculatory blood flow and development of multi-organ failure in shock and particularly sepsis.

CIRCULATORY SHOCK

Circulatory shock is a syndrome where **perfusion to the tissues is insufficient to meet its metabolic requirements**. The causes can be grouped into 4 main aetiologies. It is important to note the following aetiological classifications are not mutually exclusive, with shock often manifesting with a mixed clinical presentation. Each cause will be illustrated with a typical example and its clinical signs.

HYPOVOLAEMIC SHOCK

This is characterised by hypoperfusion caused by reduction in circulating volume such as occurs with haemorrhage, large gastrointestinal losses, and other losses of intravascular fluid. The loss can be **absolute or relative**. In patients with pancreatitis or an acute surgical abdomen, fluid is sequestered from the intravascular compartment into diseased tissues leading to relative intravascular hypovolaemia. The loss of preload has a deleterious effect on SV and therefore CO and tissue perfusion.

Diarrhoea and Vomiting as an Example

Case Summary:
76-year-old female. Nursing home resident. History of chronic leg ulceration. Febrile. Commenced on flucloxacillin for cellulitis in the community. Subsequent diarrhoea for 5 days.
Clinical Presentation:
SpO2: Poor trace, RR: 30, Central Capillary Refill Time (CRT) >5 secs. BP: 75/40, HR: 130 Sinus Tachycardia, U/O: 10ml/hr, pallor, diaphoretic and drowsy.

Take home message:
The patient is likely to have antibiotic-associated diarrhoea with possible *C. difficile* infection. Hypotension distinguishes this as a **late presentation** of hypovolaemic shock. The 4 stages of shock described by ATLS are often not seen in reality and unlikely to be a helpful categorisation. Other factors affect physiological observations including the heterogeneous nature of patient presentation and the effect of medications such as β-blockers. The patient needs emergency fluid resuscitation with frequent re-assessment and antibiotics.

VASODILATORY SHOCK

Numerous conditions lead to a loss of peripheral vascular tone. The resulting drop in SVR reduces MAP and tissue perfusion. The leading cause is septic shock. Other causes include spinal cord injury and loss of sympathetic vascular tone. Histamine release associated with anaphylaxis causes a precipitous drop in SVR. Profoundly low SVR is also seen in liver failure.

Pneumonia and Septic Shock as an Example

Case Summary:
49-year-old male. 30 pack year history of smoking and alcohol excess. Presenting to ED Resus with 4-day history of cough productive of yellow sputum.

Clinical Presentation:
Looks unwell. SpO2 79% in air. RR 40. Coarse crepitations in left lower zones. CRT >4 secs. BP 60/50. Atrial fibrillation with fast ventricular response rate 130. Anuric. Obtunded. Temperature 38.9°C. Lactate 8.

Take home message:
The patient is in septic shock. They require immediate treatment as per the sepsis six guidelines. Early presentation sepsis may present differently to the scenario

described above. Peripheral pulses may be bounding as CO is increased in response to low SVR to maintain MAP and tissue perfusion.

CARDIOGENIC SHOCK

The reduction in cardiac output in cardiogenic shock can be due to:

- Reduction in myocardial contractility (commonest cause being ischaemia)
- Dysrhythmias
- Valvular pathology (causing a drop in SV)

Myocardial Infarction as an Example

Case Summary:
65-year-old female. 40 pack year history of smoking. Presenting with a 4-hour history of central crushing chest pain. ECG: Acute ST segment elevation in chest leads V1-V4.

Clinical Presentation:
Patient diaphoretic and agitated. SpO2 80% on high flow oxygen. Frothy pink sputum. Coarse crepitations globally on auscultation of chest. HS I + II + added III. Pan systolic murmur. JVP not seen. CRT 6 secs. BP 90/40 HR 100 with frequent ventricular ectopic beats. Lactate 15 mmol/l.

Take home message:
This patient is in acute left ventricular failure secondary to a massive antero-septal MI with secondary pulmonary oedema and mitral regurgitation. Her presentation is ominous, and she has a high risk of death. She requires prompt revascularisation, positive pressure ventilation and is likely to need inotropic and mechanical left ventricular support.

Table 6.3 (below) shows classical features of left and right ventricular infarction. Presentation is often mixed.

Table 6.3	Left Ventricular Infarction	Right Ventricular Infarction
ECG	Anterior, septal, lateral changes	Inferior changes Posterior changes (if left circumflex coronary artery dominant supply to RV)
Physiological changes	Acute LV dysfunction Pulmonary venous return impaired and pulmonary oedema Reduced Cardiac Output	Acute RV dysfunction Classic triad: -Raised JVP -Clear lungs -Hypotension Due to loss of RV preload
Arrhythmias	Ventricular tachyarrhythmias and BBB	Bradyarrhythmias due to SAN/AVN ischaemia
JVP	Not elevated	Elevated

OBSTRUCTIVE SHOCK

Obstruction to forward flow from the heart can be due to reasons that are **intrinsic or extrinsic to the circulation**:

Causes **extrinsic** to the circulation:

- Cardiac tamponade
- Caval compression (pregnancy, malignancy)
- Tension pneumothorax
- Abdominal compartment syndrome
- Excessive positive intra-thoracic pressure (e.g. life-threatening asthma)

Causes **intrinsic** to the circulation:

- Embolus (Massive pulmonary, air or amniotic emboli, or vascular occlusion)

Pulmonary Embolus as an Example

Case Summary:
75-year-old male. 6 weeks post total hip replacement complaining of acute dyspnoea.

Clinical Presentation:
Agitated, dyspnoeic with pleuritic chest pain. SpO2 84% on high flow oxygen. Chest x-ray clear. Raised JVP. BP 65/40. HR 120 sinus tachycardia. S-waves in lead I, Q waves and T-wave inversion in lead III. Swollen right calf.

Take home message:
The patient is likely to have suffered a massive PE, resulting in acute right ventricular failure due to outflow obstruction of the right ventricle. The hypotension and raised JVP reflect this. They require thrombolysis or thrombectomy.

THE OUTCOME OF SHOCK

Many patients in shock develop Multiple Organ Failure (MOF) as the result of poor tissue perfusion and hypoxia. Much of the research on the outcome of MOF centres on sepsis-related MOF. There is clear correlation between the number of organs affected and risk of mortality. This correlation has resulted in the development of scoring systems that aim to predict the risk of death in sepsis-related MOF. An example is the Sequential Organ Failure Assessment Score (SOFA).

Introduction to Cardiovascular Monitoring

Ultimately the purpose of CVS monitoring is to differentiate between patients requiring/responsive to volume resuscitation and patients requiring inotropes/ vasopressors to maintain tissue oxygenation.

Static Markers of Volume Response

There is limited evidence for the clinical signs we are traditionally taught about as indicators of shock and their effectiveness in determining which patients are volume responders/responsive to fluid resuscitation:

- Intermittent Blood Pressure Monitoring: May improve with fluid resuscitation. Non-invasive measurement can be inaccurate if the cuff size is inadequate. Hypotension is invariably a **late** sign of shock
- HR: rate-limiting medications may confound this clinical sign. Typically, high HR in septic shock

- CRT: Prolonged in shock
- Urine Output: Will be reduced in shock but is a very poor marker of adequacy of resuscitation
- CVP/PCWP: Very poor predictor of volume status and volume responsiveness
- $S_{cv}O_2$: A measure of the balance of DO_2 and VO_2. Abnormalities are suggestive of shock but are a poor gauge for adequacy of resuscitation
- Lactate: High lactate is an indicator of illness severity but not a reliable marker of volume state or responsiveness

Most intensivists do not advocate 'Aggressive' goal-directed resuscitation in shock to meet specific end-points. Prior recommendations included targeting a specific CVP, MAP and $ScVo_2$ but this approach can worsen outcome through over-zealous fluid and excess pharmacological treatment.

CARDIAC OUTPUT MONITORING

There are propriety technologies (e.g. PiCCO, LiDCO) that calculate the area under the curve during systole in an arterial line trace to calibrate and measure CO. Analysis of the arterial waveform (**pulse-contour analysis**) subsequent to calibration allows for continuous CO monitoring. Other values such as SVR are derived.

PULMONARY ARTERY FLOATATION CATHETERS (PAFC) calculate CO by similar principles but are mainly used in a cardiac ICU setting; it is a much more invasive technique.

OESOPHAGEAL DOPPLER is another minimally invasive means by which to measure SV by measuring the blood flow in the descending aorta, using an oesophageal probe.

Dynamic Markers of Volume Response

PULSE PRESSURE VARIATION (PPV) AND STROKE VOLUME VARIANCE (SVV) can be used to gauge responsiveness to fluid resuscitation. The pulse pressure is the difference between the systolic and diastolic BP. There is a variation in PP and SV between inspiration and expiration; this variation is exaggerated in hypovolaemic patients. A reduction in the PPV/SVV (typically to <10%) in response to fluid bolus therapy is indicative of a patient who is no longer volume responsive. This tool is validated only in patients who are sedated and mechanically ventilated, in sinus rhythm, and where tidal volumes are both greater than 7ml/kg and consistent and

therefore less likely to confound the measurement of PPV/SVV. These constraints frequently limit the applicability of this measure.

Along similar principles, respiratory variation in Inferior Vena Cava (IVC) calibre measured by **Trans-Thoracic Echocardiography** (TTE) and **Thoracic Bio-impedance** can be evaluated. Reduction in variability implies loss of volume responsiveness but its evaluation must also take into account whether the patient is self-ventilating or being passively ventilated, and the magnitude of breaths being taken. The impact of AF is, however, less significant.

PASSIVE LEG RAISE**:** In patients who are spontaneously breathing or in situations where PPV/SVV is likely to be non-meaningful this manoeuvre can be utilised to assess fluid responsiveness. With the patient sat at 45° the bed or trolley is tipped into a Trendelenburg position so that the patient's torso is level and the legs raised without flexing at the hips. Auto-transfusion of lower limb blood will result in a sustained rise in mean arterial pressure if fluid responsive. The pressure transducer must remain level with the heart to give a valid reading.

INTRODUCTION TO CVS MANAGEMENT

Determining the cause of cardiovascular failure is paramount to implementing an effective treatment plan. A hypovolaemic patient will require fluid administration in order to optimise preload; a patient with pathological vasodilatation may require vasopressors to maintain their perfusion pressure; and a patient with poor cardiac contractility may benefit from inotropic support.

FLUIDS

Fluid boluses are routinely administered to haemodynamically unstable patients. This first line method of management is reasonable in a patient with evidence of end organ hypoperfusion likely to be improved with volume loading. Causes of hypotension should be sought and regular assessment conducted.

Fluid boluses (125-250ml aliquots) as part of a fluid challenge are aimed at increasing blood volume, preload and cardiac output (although no evidence confirms this in septic shock). This should subsequently increase organ perfusion but is not always the case and is dependent on the cause of the hypotension.

Current evidence supports only crystalloid boluses, rather than colloids, in the resuscitation of critically ill patients. Patients in haemorrhagic shock should be treated with the early replacement of blood products.

The physiological benefits of a bolus may only be transient, lasting only for a few hours, or less, in a critically ill patient with leaky capillaries, and so regular reassessment is vital.

Crystalloids are not innocuous substances and overuse can lead to tissue oedema, poor gas exchange in the lungs, ARDS and glycocalyceal injury.

VASOPRESSORS

Table 6.4 lists vasopressor actions

Table 6.4	Mechanism of Action	Effect	Other
Metaraminol	**α1**, some β	↑SVR	↓ HR and CO
Noradrenaline	**α1**, β	↑ SVR	↓ HR and CO Venoconstriction ↑myocardial O_2 demand ↑PVR
Vasopressin	**V1**, V2, V3	↑ SVR	↑PAP

Vasopressors must be used with an awareness of possible deleterious effects. For example:

- An increase in BP may be noted, but vasoconstriction can be detrimental in a failing heart through increased afterload reducing overall cardiac output

- In haemorrhagic shock, first line management should always be volume replacement with blood products. Vasopressors may be needed early whilst volume administered still lacks behind the deficit but can impair perfusion further

INOTROPES

Table 6.5 lists inotrope actions

Table 6.5	Mechanism of Action	Effect	Other
Adrenaline	**α1**, α2 at high dose β at low dose	↑SVR ↑CO	↑ myocardial O_2 consumption Coronary artery dilatation
Dobutamine	**β1**, β2	+ve inotropy	↑myocardial O_2 demand, ↑BP, ↑HR
Enoximone	↑cAMP (phophodiesterase inhibition)	+ve inotropy ↓SVR	Possible ↑ HR

Cardiovascular agents should not be used in place of adequate fluid resuscitation.

There are other agents that can be used in specialist practice; however, this is beyond the scope of this manual.

MECHANICAL/INTERVENTIONAL

An INTRA-AORTIC BALLOON PUMP (IABP) is a large bore catheter inserted into the femoral artery so that the balloon tip lies in proximal descending aorta. The IABP aims to improve cardiac performance by reducing afterload and myocardial oxygen demand. It is used only as temporary supportive measure.

EXTRACORPOREAL MEMBRANE OXYGENATION **(ECMO)** enables the direct oxygenation of blood and the removal of carbon dioxide via an external circuit. Again, this is only used for temporary cardiorespiratory support.

A VENTRICULAR ASSIST DEVICE **(VAD)** is temporary artificial cardiac pump to support the ventricles.

OTHER INTERVENTIONS

Other interventions include measures to improve the environment in which the heart and cardiovascular system function:

Mechanical ventilation

- Improving oxygenation will increase oxygen delivery
- Improving CO_2 clearance will aid acid-base balance
- Beware that excessive airway pressures and PEEP may lead to reduced venous return to the heart (a drop in preload)

Renal replacement therapy

- Adjusts electrolyte levels, fluid status, and acid-base balance

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CHAPTER 7: NEUROPATHOPHYSIOLOGY

Dr Sian Bhardwaj

LEARNING OBJECTIVES

- To explain the importance of cerebral blood flow (CBF), cerebral perfusion pressure (CPP) and intra-cerebral pressure (ICP), and the factors that influence them
- To describe the initial management of common neurological emergencies
- How to interpret neurological investigations and imaging
- An approach to prognostication in patients with brain injury

INTRODUCTION

According to ICNARC case mix data, 1 in 10 patients admitted to intensive care in 2014-2015 required neurological support, either as a result of an isolated pathology or part of a wider constellation of critical illness. The majority of patients present with time critical conditions in which immediate recognition and prompt treatment can bear significant influence on outcomes. The primary aim in the management of these patients is to treat the underlying condition where possible, to minimise secondary brain injury and to optimise the chance of a good neurological outcome. Therefore, we need to consider some basic physiological principles.

CEREBRAL PHYSIOLOGY

There are some unique physiological principles that determine the delivery of oxygen to the brain.

CEREBRAL VASCULAR SUPPLY

Blood flow to the brain occurs via four vessels: the right and left internal carotid and vertebral arteries. These all join a circuit within the brain called the Circle of Willis (Figure 7.1) from which vessels originate to supply intracranial structures, and which

provides a system which maintains a degree of resilience in the face of a diminished or failed supply from one or more vessels.

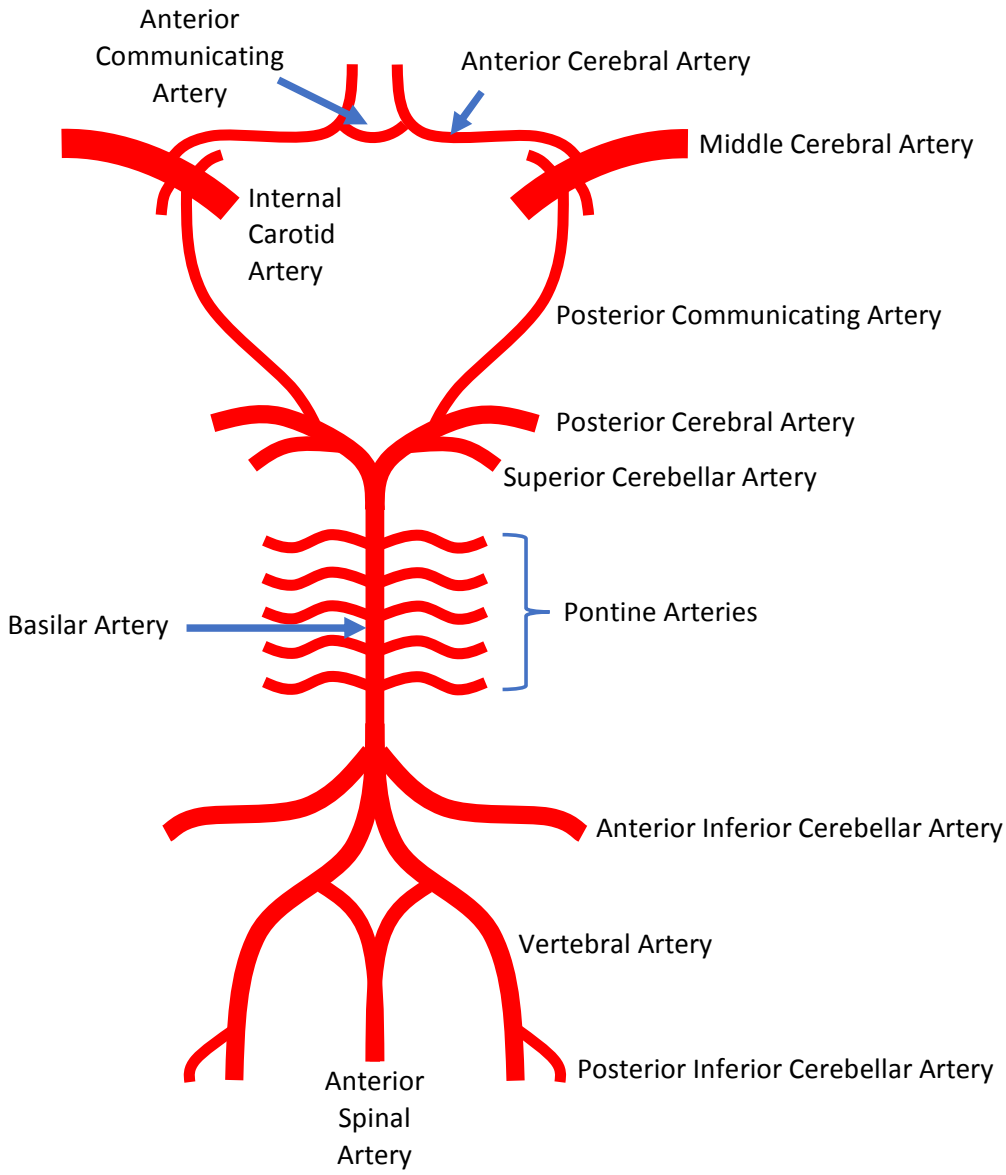


Figure 7.1: Circle of Willis

Venous blood drains via sinuses into the jugular veins.

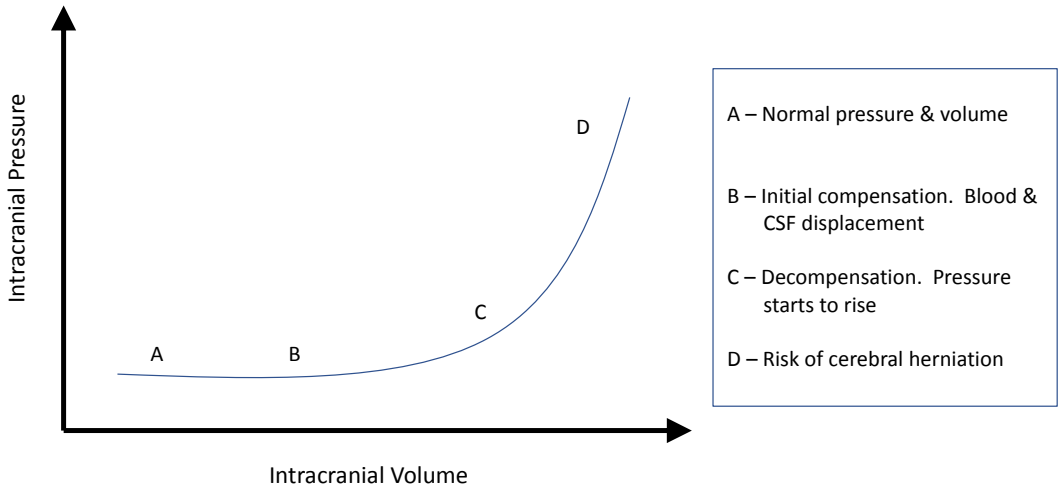
The brain is unique in that it has very limited capacity for anaerobic metabolism, which means irreversible neuronal damage occurs within minutes of interruption of cerebral blood supply.

INTRA-CRANIAL PRESSURE

The skull is a rigid, non-compliant 'box' that contains the brain, cerebral blood and cerebrospinous fluid (CSF). The pressure within this box is known as the intracranial pressure (ICP). Normal ICP is 7-15 mmHg.

It is possible to compensate for a small increase in volume by reducing the volume of an alternative component – usually the venous blood and CSF. However, this buffer is rapidly exhausted, and any further increases in volume lead to decompensation and dramatic increases in ICP (Figure 7.2).

Figure 7.2: Cerebral Compliance



As the ICP rises, there is a progressive risk that vital cerebral structures will herniate laterally through the falx, inferiorly through the tentorium or externally through skull openings, particularly the foramen magnum, all with associated further reductions in the blood supply resulting in ischaemia and cellular death. This is often referred to as 'coning' and is almost always a terminal event mediated by secondary brainstem compression.

CEREBRAL BLOOD FLOW

Factors affecting Cerebral Blood Flow (CBF) can be determined by considering the Hagen-Poiseuille equation, and the CPP as a function of mean arterial pressure and ICP:

$$CBF = \frac{\Delta P \prod r^4}{8\eta l}$$

P = CPP
r = Radius of cerebral vessel
η = Viscosity of fluid
l = Length of vessel

$$CPP = MAP - ICP \text{ (or CVP)}$$

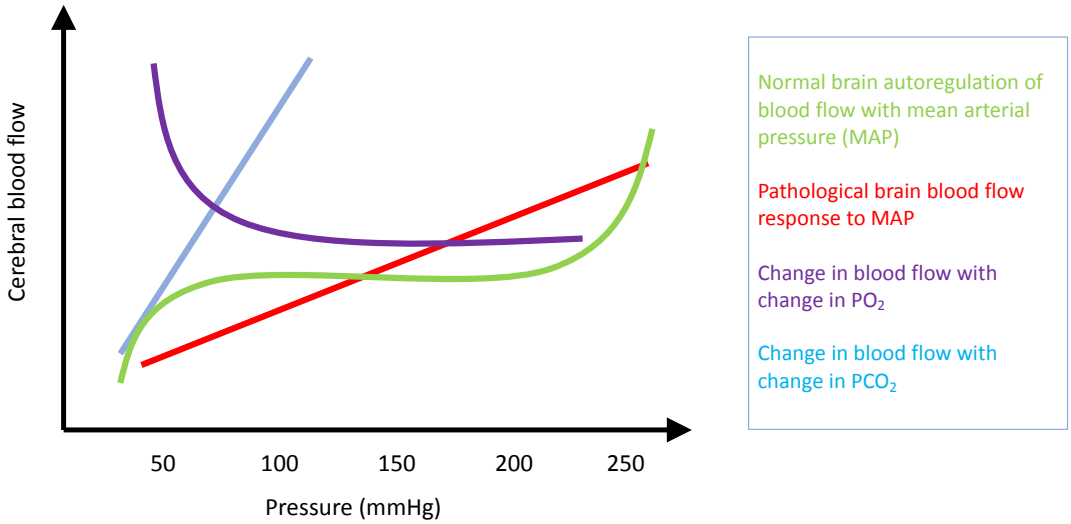
From the equations above, one can see that in order to maintain cerebral perfusion, an adequate mean arterial pressure and a low ICP are required. If ICP rises, a higher MAP is required to maintain cerebral perfusion. If CPP is inadequate, then cerebral ischaemia and reduced consciousness will result. Persistently low CPP can contribute to secondary neuronal injury and through the development of cerebral oedema as a consequence of ongoing cellular damage, can in turn lead to further increases in ICP and a progressive decline in CPP making the situation worse. Normal CPP is approximately 60-70 mmHg in a supine patient.

Cerebral blood flow is autoregulated in the healthy brain providing there is a CPP roughly between 60-150 mmHg. This autoregulation is disrupted in the diseased brain and the relationship between perfusion and flow becomes linear. This is one of the reasons it is important to ensure that MAP is maintained in patients with cerebral pathology.

Other factors that affect CBF include arterial oxygen and carbon dioxide tension (Figure 7.3).

CBF increases markedly when P_aO_2 drops below 7kPa (50mmHg). CO_2 is a potent cerebrovasodilator and there is a linear relationship between P_aCo_2 and CBF. Studies have shown that it is optimal to maintain a normal P_aCO_2 (4.5-5kPa) in patients with brain injury in order to provide sufficient CBF whilst also balancing the risk that excessive vasodilation will further increase ICP.

Figure 7.3: Autoregulation of Cerebral Blood Flow



Secondary brain injury is a complex process driven by activation of many molecular and cellular pathways. This can lead to disruption of autoregulatory processes and results in cerebral oedema. There are both systemic factors and intracranial factors that can contribute to secondary brain injury. The main management aims are to normalise the factors we know can influence cerebral oxygen delivery, reduce the cerebral metabolic demands and to ensure timely surgical intervention occurs if indicated.

Table 7.1: Clinical approach to neurological emergencies

Common neurological emergencies	Presenting features	Specific management considerations
Traumatic Brain Injury	• History of trauma • Blood from ear/nose • Agitation • Reduced GCS	• Protect C spine • Secure airway • Early imaging
Cerebral Haemorrhage or Ischaemic stroke	• Focal neurology • Headache • Reduced GCS	• Thrombolysis / thrombectomy
Status Epilepticus	• Persistent seizures • Dilated pupils • High lactate • Consider sub-clinical status	• Anti-convulsants • EEG • Normoglycaemia
Meningitis or Encephalitis	• Headache • Photophobia • Neck stiffness • Rash • Pyrexia, agitation • Low GCS	• Early antimicrobials • Consider dexamethasone for pneumococcus meningitis
Hypoxic Encephalopathy	• History of anoxic event e.g. Post resuscitation	• Delayed prognostication

CLINICAL APPROACH TO NEUROLOGICAL EMERGENCIES

Patients presenting with neurological emergencies will often require intensive care interventions to facilitate diagnosis and ongoing management. Many patients will require early cerebral CT scans and if presenting in a state of agitation or reduced GCS, will need emergency airway management with intubation and sedation to enable this to occur safely.

The approach to RSI should consider minimisation of further increases in ICP and optimisation of CPP. The choice of induction agents in the context of head injury and other intracranial pathology is often the source of discussion. Commonly used induction agents include ketamine, thiopentone (particularly useful for its anti-convulsant properties) and propofol, although the latter two agents must be used with caution to avoid significant drops in systemic blood pressure and compromised CPP. Dose adjustment and vasoconstrictors may be needed. Ketamine is favoured by many practitioners in view of its more favourable cardiovascular stability profile and is frequently used in various critical care settings such as prehospital, emergency department and intensive care unit. Concerns over the potential increase in ICP with the use of ketamine seem unsubstantiated although some would advocate co-induction with an opioid (e.g. fentanyl) as a means of blunting the sympathetic

A	Airway patency & protection more likely to be compromised if patient not responding to voice
	C-spine protection if trauma
	Avoid tight ETT tie around neck
B	Ensure adequate ventilation
	Aim for $PaCO_2$ 4.5 – 5 kPa
	Aim for normal PaO_2
	Avoid high PEEP
C	Treat underlying hypovolaemia
	Avoid hypotonic fluids
	If hypotensive then maintain MAP to preserve CPP. Use vasopressors if required
D	Assess and document GCS prior to sedation
	Assess and monitor pupillary response
	Treat seizure activity
	If severe agitation likely to need RSI
	Consider temporary therapy management of raised ICP (eg mannitol / hypertonic saline)
	Avoid head down position
	Normoglycaemia
E	Assess for other injuries / rash
	Normothermia

response to laryngoscopy although the most appropriate dose is not known. Etomidate use has declined significantly as a result of its association with increased mortality in the context of sepsis but may nonetheless be appropriate in non-infective intracranial pathology.

When anaesthetising a patient with an acutely compromising severe neurological injury (e.g. infection, stroke, trauma) the option to wake them up and utilise an alternative strategy later is not usually practicable. Problems with orotracheal intubation will be dealt with through other means including videolaryngoscopic or fibreoptic intubation or surgical cricothyroidotomy during which continued paralysis is highly desirable. Consequently, the use of rocuronium is the first choice for many practitioners as it provides good intubating conditions whilst avoiding the potential increase in ICP associated with suxamethonium use, and that associated with any coughing or straining as the suxamethonium wears off before a non-depolarising muscle relaxant is administered.

The widespread availability of sugammadex to reverse rocuronium if felt necessary has further diminished the use of suxamethonium.

Ventilation should be targeted to $PaCo_2$ 4.5 - 5 kPa and both hypo- and hyperoxaemia avoided as these have been shown to contribute to secondary brain injury.

Many patients present with a high blood pressure. Cardiovascular aims should target BP in the high – normal range. Hypotension should be avoided and if present should result in prompt management and investigation for alternative causes e.g. haemorrhage, sepsis.

Glucose and temperature should be normalised. Ongoing sedation will reduce the cerebral metabolic demand and help prevent secondary injury.

CT IMAGING

Most patients who present with a suspected neurological emergency will require a CT scan of their head to look for underlying pathology. It is important to ensure the patient is stable enough prior to transfer to CT scan. For patients with a low GCS or severe agitation, RSI and airway protection are vital prior to transfer. A balance between initial resuscitation and stabilisation vs. rapid imaging needs to be struck to minimise delay in definitive treatment for time critical neurological injuries.

Most patients will require a non-contrasted CT scan of their brain as a minimum, with contrast commonly indicated for those with suspected haemorrhage.

CT angiography is indicated in those patients with suspected ischaemic strokes.

CT Images of common neurological emergencies (Figures 7.4-7.6):

(Images from Worcestershire Acute Hospitals radiology dept)

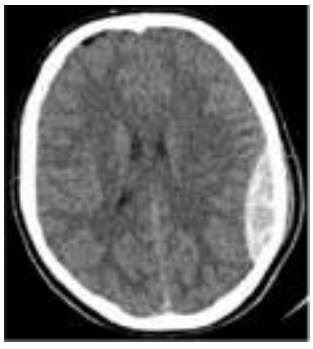


Figure 7.4:
Extra Dural Haematoma

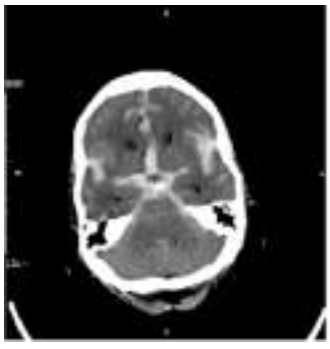


Figure 7.5:
Subarachnoid Haematoma

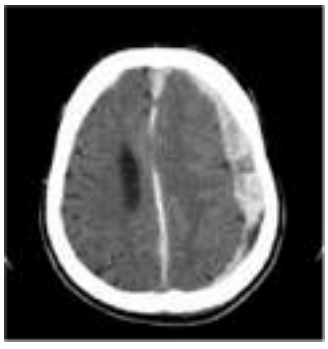


Figure 7.6:
Acute Subdural Haematoma

ONGOING ICU CARE

Some patients presenting with neurological emergencies will require immediate transfer to tertiary neurosurgical care for intervention to minimise the risk of secondary brain injury. These patients commonly include those with severe traumatic brain injury, traumatic haematomas, acute bleeds and acute hydrocephalus. Images will need to be linked to the receiving tertiary centre, as a priority and prompt referral is required to ensure timely intervention can be delivered. Common interventions include evacuation of expanding haematomas, shunt procedures for hydrocephalus, coiling or clipping of aneurysms, decompressive craniotomy and direct monitoring of ICP.

Other patients will not require transfer and can be managed in a general Intensive Care Unit.

It is important with all patients the systemic factors that can contribute to secondary brain injury are optimised: Normal oxygenation, normocapnia, maintain MAP, adequate sedation to reduce cerebral metabolic demands, normoglycaemia and prevention of pyrexia and hyponatraemia.

NEUROPROGNOSTICATION

The intensive care management of these patients aims to optimise cerebral recovery and to obtain the best neurological outcome possible. However, it remains very difficult to predict those patients who will make a good recovery, and those who are likely to be left with profound disabilities.

Available evidence and associated consensus guidance have indicated that decisions about neurological outcome should not be made be at presentation based on history and scans alone: a period of observation whilst aiming to maintain cardiorespiratory stability and minimising potential secondary insults is advised. This allows multimodal assessment and communication to understand better the likely views of the patient. In patients with perceived devastating brain injury, this period is at least 24 hours and depending on the clinical context may be much longer: after cardiac arrest, neuroprognostication in the unconscious patient is not advised until 72 hours. In many cases it is appropriate to continue support for several days longer before making an assessment.

There are some early signs that if found once the patient is no longer under the influence of sedative or paralysing drugs mean that outcome is likely to be poor. These include absent pupillary and corneal reflexes, bilateral absence of somatosensory evoked potentials (SSEP), and an unreactive EEG in combination with CT evidence of cerebral oedema. Patients who display several of these signs are likely to have poor outcome and withdrawal of life sustaining treatment can be considered.

CONCLUSION

Neurological emergencies account for many admissions to the intensive care unit. Management should focus on rapid stabilisation, imaging and interventions to prevent secondary brain injury. Predicting outcome in these patients can be difficult and invariably requires a delayed assessment over a period of days.

CHAPTER 8: ACUTE KIDNEY INJURY AND RENAL REPLACEMENT THERAPY IN INTENSIVE CARE

Dr Phillip Howells

LEARNING OBJECTIVES

- Basic anatomy and function of the kidneys
- Key metabolic functions
- The kidney is vulnerable in critical illness
- Renal function can be supported artificially in the intensive care setting

INTRODUCTION

Acute kidney injury (AKI) is common and may be the main reason for a patient's referral or occur as a complication of critical illness. AKI is associated with increased risk of death in the critically unwell. AKI can be life threatening but is treatable with medical and renal replacement therapy whilst kidney recovery is awaited. Renal replacement techniques also have a role in the management of certain poisonings; this will be covered elsewhere.

DEFINITION OF AKI

AKI is defined as a rapid (over hours or days) reduction in the glomerular filtration rate. Various classifications have been proposed, but none are universally accepted. The Kidney Disease Improving Global Outcomes (KDIGO) criteria are one widely used set of criteria and may be met by either the creatinine or urine output criteria (Table 8.1). Importantly, mortality is higher with higher-staged AKI.

Table 8.1: KDIGO Criteria

Stage	Creatinine criterion	Urine output criterion
1	26.5umol/l (0.3mg/dl) or 1.5-2x increase	<0.5ml/kg/h >6h
2	2-3x increases	<0.5ml/kg/h >12h
3	>=353.6umol/l (4mg/dl) or >3x increase or RRT	<0.3ml/kg/h for >24h or anuria >12h

Creatinine is the main biomarker of renal function. It is produced as a waste product from muscle tissue at a relatively constant rate and is primarily cleared by filtration by the kidney (a small amount is actively excreted by the proximal convoluted tubule). Creatinine will rise in AKI or chronic kidney disease (CKD). Creatinine is non-linearly related to glomerular filtration rate and therefore a large proportion of renal function must be lost before a rise in creatinine occurs (Table 8.1). Creatinine is lower in pregnancy and the healthy elderly – high-normal creatinine in these groups can represent serious renal disease. Estimated glomerular filtration rate (eGFR) is based on a formula of age and creatinine, designed for assessing stable CKD, not AKI. In AKI, a laboratory may report a moderately reduced eGFR despite having a total loss of renal function and therefore a true GFR of zero. Therefore, eGFR should not be used to assess a critically ill patient. There are various equations for estimating a true creatinine clearance, such as Cockcroft-Gault, which should be used when this is needed, for example for drug dosing (Figure 8.1).

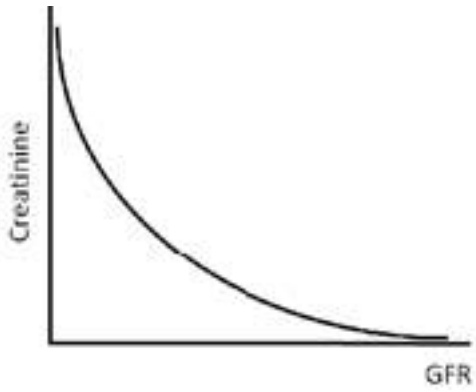


Figure 8.1: GFR and Creatinine Inverse Relationship

Novel biomarkers are currently under investigation for AKI diagnosis and to detect renal recovery following AKI, but none are yet in widespread use. Molecules under investigation include cystatin-C, neutrophil gelatinase-associated lipocalin, kidney injury molecule 1, interleukins 6 and 18 and cycle arrest biomarkers IGFBP7 and TIMP-2.

CAUSE OF AKI

The cause of AKI is usually classified into pre-renal, intra-renal and post-renal causes (Table 8.2).

Table 8.2: Causes of AKI

	Common in intensive care	Less common and rare
Pre-renal	• Shock	• Renal artery stenosis
Intra-renal	• Rhabdomyolysis • Nephrotoxic drugs • Contrast nephropathy • SIRS/sepsis • Abdominal compartment syndrome	• Interstitial nephritides • Glomerulonephritides • Haemolytic uraemic syndrome • Crystal nephropathies
Post-renal	• Prostatic hypertrophy • Drugs • Surgical injury to ureter • Acute urinary retention	• Ureteric/bladder clot • Nephrolithiasis • Urinary tract pathogens • Renal tract strictures • Calyceal shedding • Ureteric or bladder malignancy • Congenital abnormalities • Prostate cancer • Retroperitoneal fibrosis

ASSESSMENT AND MANAGEMENT

The priorities in the assessment and management of the patient with AKI should be to provide resuscitation, determine the cause of the AKI, address precipitant factors and manage life-threatening complications. This should be done by following the ABCDE process, with focussed history, examination and investigations that will determine immediate management as discussed in the assessment and initial management of the critically ill patient and cardiovascular pathophysiology chapters.

The potentially life-threatening features are:

- Pulmonary oedema (detected by cardiorespiratory examination, oxygenation assessment, chest x-ray and/or lung ultrasound)
- Hyperkalaemia (there are no reliable clinical features before cardiac arrest, so requires blood gas measurement (arterial or venous), as well as a U&E and ECG)

- Acidaemia (tachypnoea and blood gas analysis)
- Uraemia (azotaemic) complications (pericarditis – may present as cardiac tamponade and shock or ECG changes, or delirium)

Therefore, alongside a focussed physical examination, blood gas analysis, chest x-ray and ECG (or point of care ultrasound) are crucial. In patients with or at risk of AKI, modifiable risk factors should be addressed. Withdrawal of nephrotoxic drugs will require the balancing of risks and benefits in the critically unwell. This is of particular importance for nephrotoxic antibiotics and transplant rejection agents.

Adequate renal perfusion pressure is the cornerstone of managing pre-renal renal failure, achieved by fluid resuscitation and vasopressor/inotropic support as needed. Post-renal failure requires drainage of the obstructed system, with urinary catheterisation or nephrostomies for upper tract obstruction. Intra-renal failure is managed by removal of precipitating agents and treatment of the causal disease. Often this will require liaison with a nephrologist, especially if immunosuppression is required.

FLUIDS AND URINE OUTPUT

Traditional teaching has suggested a target urine output of 0.5 ml/kg/hour for most intensive care patients, sometimes with higher targets. However, aggressive fluid loading independent of raised blood pressure has only been shown to be effective in contrast induced nephropathy, although it may also be useful in rhabdomyolysis and rare crystal-forming disorders. High venous pressure (CVP >12 mmHg) may cause venous congestion and worsen renal dysfunction. Therefore, whilst plentiful fluid resuscitation is very commonly indicated in AKI, particularly as pre-renal failure is by far the most common cause, careful and frequent reassessment of fluid status is required to avoid the deleterious effects of over-resuscitation. Oliguria is a physiological response and giving fluid solely to drive urine output can lead to fluid overload.

Historically, loop diuretics such as furosemide were given with the rationale that inhibiting the sodium-potassium-chloride pumps in the loop of Henle would reduce renal metabolic load and promote urine flow. This has been discredited and risks hypovolaemia and the false reassurance of an artificially raised urine output (which may be concomitant with vasodilatation and circulating volume loss). The main role of diuretics is to treat hypervolaemia where there is still urine output. Low (so-called renal) dose dopamine (which was also thought to improve renal blood supply) has been shown to have no effect on renal outcome.

INVESTIGATIONS

Investigations should:

- Identify AKI (based on creatinine and urine output measurement)
- Identify metabolic complications, especially those that are life-threatening (as discussed above)
- Identify causal factors

Diagnostic tests may therefore include:

- Clinical assessment for hypoperfusion/shock (see cardiovascular pathophysiology chapter), identification of sepsis and/or cardiogenic shock
- Urine dip for protein and blood (this helps define syndromes of renal injury)
- Urine microscopy for neutrophils or eosinophils, Bence-Jones protein, protein, urinary electrolytes
- Renal tract ultrasound to identify upper or lower tract obstruction.
- Intra-abdominal pressure measurement
- Blood film, haptoglobins, bilirubin
- CK, ANA (including anti-dsDNA), ANCA, anti-GBM, ENA, serum electrophoresis, immunoglobulins, cryoglobulins, hepatitis, CMV and HIV serology

Occasionally, additional investigations are needed for rarer diagnoses:

- MRA/renal artery ultrasound/renal artery angiography
- Renal biopsy

Consultation with a nephrologist is important, especially in intra-renal acute kidney injury where specific therapies (such as immunosuppression or plasma exchange) may be indicated.

SHORT AND LONG-TERM IMPLICATIONS

AKI is associated with higher mortality, with odds ratios for death of 2.1 for stage 1, 2.9 for stage 2 and 6.9 for stage 3 AKI. ICU and hospital stay are increased with increasing severity, and the most damaged kidneys are least likely to recover. In the longer-term, patients with

an episode of AKI have an incidence of developing chronic kidney disease of as much as 8% per year. Therefore, patients who are discharged home from ICU with an episode of AKI should receive regular renal function surveillance by their general practitioner or a nephrologist.

RENAL REPLACEMENT THERAPY

As discussed above, the major acute complications of renal dysfunction are:

- Hyperkalaemia
- Metabolic acidosis
- Fluid overload
- Complications of uraemia (usually encephalopathy or pericarditis)

Renal replacement therapy (RRT) is required where these cannot be managed (or are anticipated to become unmanageable) by more conservative means. RRT can also be used for the removal of some poisons. Where patients have chronic ESRF (end stage renal failure) and are RRT-dependent anyway, concomitant critical illness may require an alternative method of RRT until they are sufficiently recovered for their normal therapy. Sites for long-term renal access (i.e. arteriovenous fistulae) must be protected whilst patients are on the ICU, avoiding devices in the same limb.

RRT can be provided by:

- Peritoneal dialysis
- Intermittent haemodialysis
- Continuous RRT techniques (CRRT)

In the UK, continuous RRT techniques are most commonly used on the ICU, as the systems are simpler and require less training and staff costs, although the disposable components are more expensive. Haemodynamic stability is also better than with IHD and results in less significant swings in drug clearance rates on and off RRT. Elsewhere in Europe, IHD is much more frequently used and the benefit of one technique over the other has not been demonstrated by randomised trials.

Peritoneal dialysis involves the infusion of dialysate into the peritoneal cavity for prolonged periods, which causes diaphragmatic splinting, raised intra-abdominal pressure and relatively inefficient clearance and therefore is rarely used in ICU except in the paediatric ICU setting. Even those patients on regular PD are usually supported by continuous haemofiltration techniques when critically unwell.

Renal replacement therapy requires:

- Large-bore dual lumen venous access
- CRRT machine
- An appropriate method of circuit anticoagulation
- Consumables including circuit piping and fluids
- For diafiltration techniques, an external water supply may be needed
- Adequate nursing support (RRT requires additional nurse training and is very labour-intensive for nurses)

Adequate vascular access is essential and is provided by a large two-lumen cannula placed in a central vein (sometimes with a third narrow lumen for additional access). Poor flows limit the time the filter is actually running for and, therefore, the effectiveness of filtration. In general, right internal jugular access is preferred, followed by femoral, then left IJV and lastly subclavian (due to risk of subclavian stenosis). However, there may need to be compromise with other considerations around central access, such as avoiding using too many sites at once.

The modern renal replacement therapy filter consists of synthetic filaments with blood flowing on one side and filter fluid on the other. Substrate can be removed from the fluid by convection (the primary mode of action in haemofiltration), in which substrate is carried by the ultrafiltrate along a pressure gradient (also known as solvent drag), or by diffusion, where molecules diffuse preferentially along a concentration gradient across a membrane (dialysis).

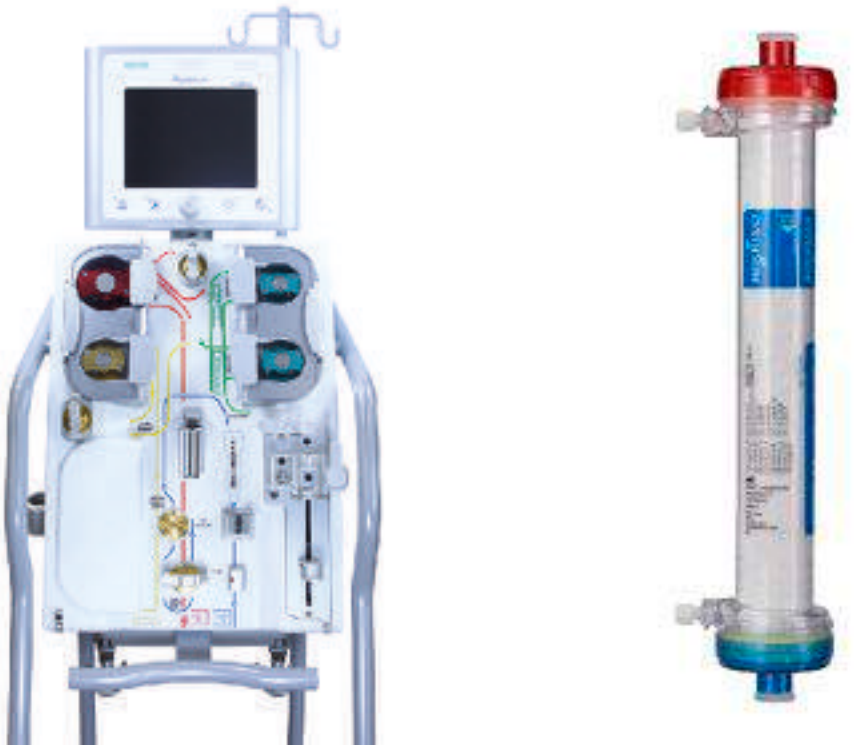


Figure 8.2: Renal replacement machine (left) and close up of filter (right)
All photographs by kind permission of Nikkiso UK

Different types of filtration circuit are shown in Figures 8.3-8.5. Continuous Veno-Venous Haemofiltration (CVVH) is the most common in use in the UK; Continuous Veno-Venous Haemodialysis (CVVHD) is rarely used, but more often the two are combined to produce Continuous Veno-Venous Haemodiafiltration (CVVHDF). Blood is removed via one lumen of the vascular catheter and pumped under pressure through microfilaments constructed with a semi-permeable membrane. In CVVH, ultrafiltrate passes across the pores and is removed, taking waste products with it and replacement fluid is added before or after the filter (see below). In CVVHD, dialysate flows through the filter in the opposite direction to blood, removing waste by diffusion. In CVVHDF, both processes run concurrently. Blood is then returned to the patient via the other lumen. CVVHDF optimises solute removal but may slow maximal filter flow rates compared to CVVH.

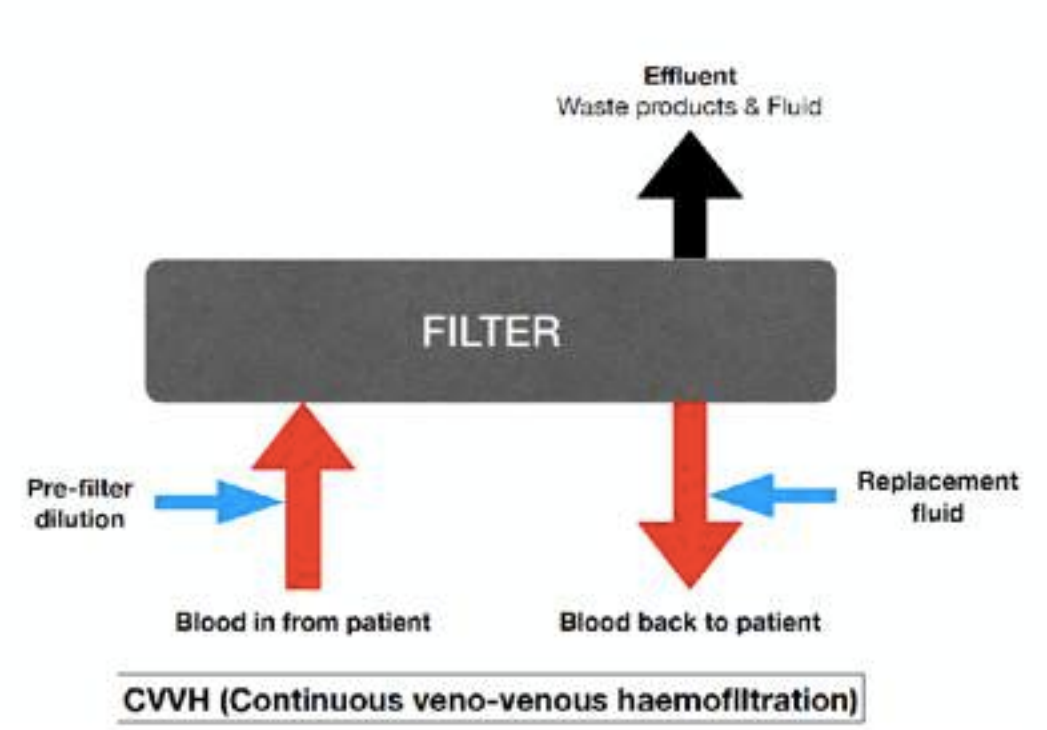


Figure 8.3: CVVH

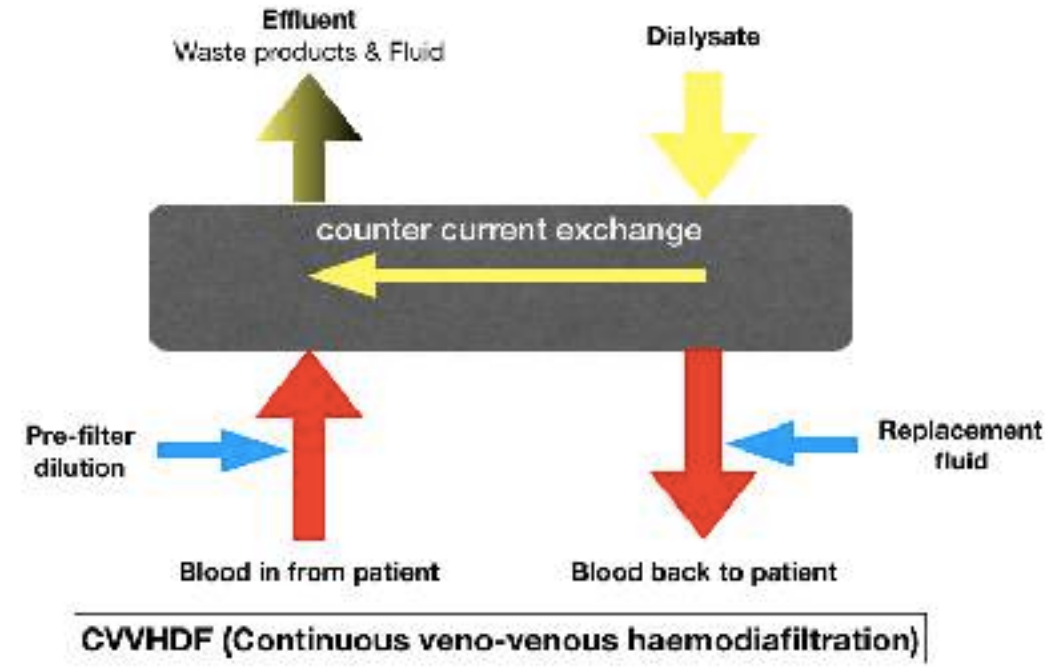


Figure 8.4: CVVHDF

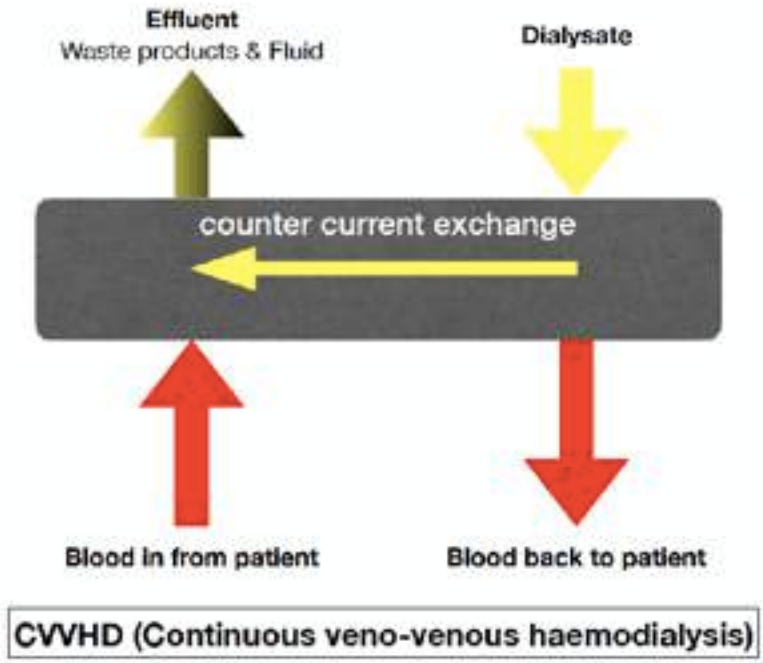


Figure 8.5: CVVHD

RRT is dosed according to effluent flow rate per kilogram bodyweight. Low dose regimes (20-25 ml/kg/24 hours) are as effective as higher doses but it is often necessary for filters to run at higher rates as there will be prolonged periods during the day when there is no filtration (for example, to allow for fluid or circuit changes or for patients to undergo other therapies or investigations such as intra-hospital transfer to the CT suite).

TROUBLESHOOTING

The effective delivery of renal replacement therapy requires a skilled healthcare team: the nurse caring usually leads ongoing management of a RRT machine for the patient. Many sessions of therapy are without issue but it is common to need to address aspects that interfere with effective filtration. These are related to patient, vascular access or machine problems.

Recommended approaches to troubleshooting the more common problems are considered here.

Low "arterial" pressure alarm (suction problem on inflow of blood into machine)

PROBLEM	ACTION
Kinked / clamped line	Unkink / unclamp
Clot in line / inflow circuit	Replace clotted device Modify anticoagulation regime
Access device against wall	Rotate access device (ie Vascath) Consider alternative site
Hypovolaemia	Switch to neutral fluid balance Consider fluid administration

High venous pressure alarm (blood return)

PROBLEM	ACTION
Kinked / clamped line	Unkink / unclamp
Clot in line / inflow circuit	Replace clotted device Modify anticoagulation regime
Position of line causing obstruction	Rotate access device (ie Vascath) Consider alternative site

Disconnection alarm

PROBLEM	ACTION
True disconnection	Clamp lines Consider gas embolus Fluid resuscitate Consider line sterility
Kinked / clamped line	Unkink / unclamp
Clot blocking pressure sensor	Circuit change Evaluate anticoagulation regime
Pump flow speed to low for a well functioning Vascath	Increase pump speed

Increasing Transmembrane Pressure

PROBLEM	ACTION
Haemofilter clotted	Circuit change Evaluate clotting regime
Kinked / clamped line	Unkink / unclamp
Blood flow too low for ultrafiltration	Increase pump speed

Air in circuit
A true gas embolus is a major emergency and should be managed as such

PROBLEM	ACTION
Small gas bubbles (eg from CO2 from bicarbonate based replacement fluid)	Follow machine degassing protocol
Inflow line disconnection	Clamp line Consider gas embolus Fluid resuscitate Consider line sterility
Turbulence close to air sensor	Consider override (DANGEROUS)

ANTICOAGULATION

RRT circuits require anticoagulation for most patients. An inadequately anti-coagulated circuit will rapidly form clots, requiring it to be changed. This leads to time off the filter, loss of blood and higher costs. Agents for anticoagulation include:

- Heparin (most common)
- Citrate (binds calcium in the circuit)
- Epoprostenol (prostacyclin)

Typically, heparin is started as a small bolus, then as an infusion and added to the circuit pre-filter. This may be titrated to the APTT ratio. If circuit life is short, further anticoagulation may be needed. Care should be taken to look for complications of bleeding. The risk versus benefit should be considered if there are contraindications to anticoagulation, such as immediately following surgery. Other agents may occasionally be used when heparin is contraindicated, such as bilvalirudin or argatroban. Patients with pre-existing coagulopathies may not require anticoagulation at all, and in those with an absolute

contraindication to anticoagulation it is possible to adjust the filter settings to maximise filter life, albeit at the expense of filter efficiency.

RRT replacement fluid is usually a balanced crystalloid solution, with weak anions provided by either lactate or bicarbonate. Bicarbonate solutions have a shorter shelf-life (because carbon dioxide leaches through plastic containers) and are therefore more expensive, but lactate clearance is better than would be achieved with a lactate-based fluid. Lactate based solutions require adequate liver function to convert lactate and should not be used for patients with lactate over 4mmol/l whether as a result of liver impairment or excessive lactate production. Most ICUs are now using only bicarbonate solutions and consequently manufacturers are now reducing or ceasing lactate solution production.

Fluid can be added pre-filter (which increases filter lifetime but slows solute clearance) or post filter (which improves solute clearance but reduces filter life). Fluid replacement may be less than that removed, which leaves the patient in "negative fluid balance", allowing net fluid removal, or as much as is removed is replaced ("in balance", "neutral", or "equal balance"). In theory, a patient could receive fluid directly via the filter, but in practice fluid boluses are given intravenously when needed. In practice it is the overall fluid balance of the patient which is important, and this will need to take into account all fluid inputs (enteral feed, nutrition, sedation, other drugs and antibiotics etc.) as well as all losses. When managing renal replacement, the nurse will need to take all these factors into account when attempting to achieve the fluid balance "prescribed" by the intensive care physician.

Cessation of renal replacement therapy occurs when (or indeed if) there is renal recovery. This is often heralded by the return of urine output and successful liberation from CVVH is more likely with urine output of over 400ml/day. Patients require monitoring to ensure renal action is adequate. In patients without renal recovery, consultation with nephrology about establishing long-term RRT will be needed.

Trials starting RRT early in the course of AKI rather than later have not shown definitive benefit of this strategy. Suggestions that moving small to middle-sized molecules in sepsis (such as TNF alpha and IL-6) have also not been shown to have mortality benefits in clinical trials, so RRT should not be used solely for this purpose, but to compensate for organ failure.

SUMMARY

AKI is a common clinical finding in the critically ill patient and is associated with significant morbidity and mortality. Careful assessment and investigation are essential in the effective management of AKI and may require collaboration with a nephrologist to optimise the

clinical outcome. An understanding of the mechanism of action of common renal replacement techniques in the ITU is vital in providing and maintaining effective therapy.

CHAPTER 9: NUTRITION AND GASTROINTESTINAL TRACT ISSUES

Dr Laura Tulloch, Dr Kieran Donnelly and Dr Jon Hulme

LEARNING OBJECTIVES

- Understand common GI related pathology and management including specific GI monitoring
- To understand considerations & techniques for nutritional support during critical illness and associated complications

THE GASTROINTESTINAL (GI) TRACT AND CRITICAL ILLNESS

The gastrointestinal (GI) tract has a role in digestion and essential endocrine, immune and barrier functions.

It is also implicated in many common pathological conditions in the critically ill; bacterial translocation of gut organisms and related toxins is postulated to be a significant driver of critical illness multi-organ dysfunction.

NUTRITION

Malnutrition occurs in up to half of critically ill patients leading to an increased risk of infection, loss of lean muscle mass and poor wound healing so it is important to assess nutritional requirements and provide support as necessary.

NUTRITIONAL REQUIREMENTS

Average nutritional requirements (adult) are shown in Table 9.1:

Average Adult Requirements	Amount (unit/kg/day)
Energy	25-30 kcal/kg/day
Protein	1-1.25 g/kg/day (up to 2g/kg/day if severely catabolic)
Water	30 ml/kg/day
Sodium	1-2 mmol/kg/day
Potassium	0.7-1 mmol/kg/day
Magnesium	0.1 mmol/kg/day
Calcium	0.1 mmol/kg/day
Phosphate	0.4 mmol/kg/day

MICRONUTRIENTS

Most enteral nutrition (EN) and parenteral nutrition (PN) feeding regimens have vitamin and trace element supplementation. Critical illness increases requirements for vitamins A, E, K, thiamine, vitamin C and pantothenic and folic acids.

Some specialist centres measure vitamin and trace elements levels in patients with specific pathologies (burns, short bowel syndrome, chronic malabsorption); most intensive care units do not measure these routinely.

ADJUNCTIVE NUTRITION

There is interest in immune-nutrition and probiotic use as a modulator of pro-inflammatory GI dysfunction, but current evidence does not support routine micronutrients replacement (e.g. glutamine, arginine, fish oils and antioxidants).

ROUTE OF NUTRITION

Whenever possible patients are fed enterally which enables a more complete diet, maintains gut structural integrity, improves bowel adaptation after surgery, reduces infection, is more cost effective and can protect the stomach from stress ulceration than parenteral nutrition.

WHEN TO INITIATE FEEDING

The 2016 Society for Critical Care Medicine and American Society for Parenteral and Enteral Nutrition guidelines make the following recommendations for initiating nutritional support:

- Start Enteral Nutrition (EN) within 24–48h following onset of critical illness & ICU admission
- Withhold EN until patient fully resuscitated and/or haemodynamically stable due to rare risk of precipitating GI ischaemia
- Start PN early when enteral route is not feasible or sufficient in high-risk or poorly nourished patients
- In previously well-nourished patients, it is acceptable to wait 1 week before starting PN if using the enteral route is still not feasible

ENTERAL FEEDING

Types of tube

Nasogastric (NG) feeding tubes are preferred to oral where possible. All feeding tubes should be radio-opaque to help identify their position on x-ray.

Use large bore ("Ryles") tubes (12-14 F) if gastric drainage is required; otherwise use fine bore feeding tubes (<12F), as they are more comfortable and better tolerated. If long-term EN is needed, consider a percutaneous enterogastrostomy (PEG) tube.

Post pyloric feeding with a nasojejunal tube or jejunostomy is indicated if there is persistent gastroparesis, in acute severe pancreatitis and for post-anastomotic feeding following upper GI surgery.

Safe NG tube placement

Feeding via a misplaced NG tube into the lungs can be fatal and is classed as a Never Event. National Patient Safety Agency, now NHS Improvement guidance, regarding confirmatory preconditions prior to NG feeding must be followed.

Initiating NG feeding

Enteral feed often started at a low rate (e.g. 30ml/hr) and if tolerated, built up to the target intake. Intolerance may present as high gastric residual volumes, diarrhoea or abdominal distension; residual volumes are measured by aspirating via the NG tube every 4 hours.

If aspirates are consistently greater than 200-300ml, consider prokinetic agents (erythromycin 125mg QDS NG and metoclopramide 10mg TDS IV); give both for 48hrs (tachyphylaxis occurs within a few days). There is an inconsistent relationship between the volume aspirated and actual residual volume; this is a pragmatic approach.

Types of NG feed

Standard NG feeds contain 1 kcal/ml and daily requirement of electrolytes, trace elements and vitamins. Specialised feeds are available: high fibre, high protein, low sodium and concentrated (1.5kcal/ml concentrated feed may be useful if fluid restriction is required). Consult with the nutrition team about their use.

Complications

- Misplacement of NG tube: Aspiration pneumonitis
- GI problems: Diarrhoea (common), reflux, abdominal pain, nausea and vomiting
- Metabolic: Hyperglycaemia (common), re-feeding syndrome
- Infective: Sinusitis, aspiration pneumonitis

PARENTERAL FEEDING

A combination of carbohydrates and lipids (30-40%) provide for the energy requirement. Current preparations consist of soya bean oil emulsified with glycerol with added amino acids, electrolytes and micronutrients.

Parenteral Nutrition (PN) should be used when adequate enteral intake cannot be established e.g. bowel obstruction, ileus, high small bowel fistula or malabsorption. PN can also supplement inadequate enteral nutrition (i.e. <60% of full feed requirement).

Due to the risk of infection and sepsis it is important to use a dedicated intravenous line for PN such as central venous catheters (CVC), peripherally inserted central catheters (PICC) or in the case of dedicated peripheral PN solutions, peripheral cannulae. CVC can stay in longer than peripheral cannulae and more hyperosmolar solutions (irritant to peripheral veins) can be infused which deliver more adequate nutritional support at reduced volumes.

Initiating PN

PN is infused continuously through a dedicated line. On commencement, it is important to monitor for re-feeding syndrome, hyperglycaemia and fluid overload.

Complications

- Catheter related: Infection, thromboembolism and misplacement
- Metabolic: Refeeding syndrome, hyperglycaemia, liver dysfunction (hepatic steatosis, intrahepatic cholestasis)
- Fluids and electrolytes: Re-feeding syndrome, vitamin deficiency (folate, thiamine, vitamin K), fluid overload

MONITORING THE GUT

Assessment of bowel frequency and character, tolerance of enteral feed and clinical examination are all important in successfully establishing gut function. Impairment of gut function is associated with worse outcomes. Biochemical investigations can also be used to assess nutrition, specifically electrolytes and albumin.

Further information can be found here.

INTRA-ABDOMINAL PRESSURE (IAP)

The abdomen is a compartment, albeit a relatively compliant one. With high compartment pressure, local organ perfusion and function risks compromise and deleterious extra-abdominal effects can occur. Intra-abdominal hypertension is a sustained or pathological IAP ≥ 12 mmHg; intra-abdominal compartment syndrome (IACS) is an IAP > 20 mmHg with new or worsening organ dysfunction.

Risk factors

Risk factors are any causes of increased rigidity of the compartment wall (e.g. abdominal surgery, obesity), increased intraluminal contents (e.g. gut obstruction), increased abdominal contents (e.g. pneumoperitoneum, gut oedema) and capillary leak (e.g. massive fluid resuscitation, pancreatitis).

Measurement

This is performed using a pressure transducer to measure bladder pressure that approximately equals IAP at the end of expiration in the supine patient in the absence of active abdominal muscle contraction.

To do this, instil 25ml of saline via a urinary catheter and clamp it distal to the aspiration port to keep the fluid in the bladder; wait 60 seconds to allow detrusor muscle relaxation. Insert a 16G needle that is connected to a pressure transducer zeroed at the iliac crest in the mid-axillary line through the aspiration port to measure the pressure. Repeat 4-6 hourly; the trend and clinical status may be more significant than absolute values.

Management

Target suspected cause and limit contributing factors: abdominal wall compliance improves with sedation, analgesia ± neuromuscular blockade; NG free drainage, prokinetics and enemas reduce intraluminal contents; abdominal contents (e.g. ascites) can be drained; capillary leak can be limited by judicious fluids administration.

Other management options include paying attention to protective lung ventilation strategies, consideration of renal replacement therapy to remove fluid and the use of vasopressors to manipulate blood pressure (Aim MAP ≥ IAP + 50). Laparostomy may be needed if other methods fail and can achieve rapid resolution of cardiovascular and respiratory compromise. General surgeons will need to be involved early in this eventuality.

Further information and treatment algorithms may be accessed from the World Society of Abdominal Compartment Syndrome.

RE-FEEDING SYNDROME

Electrolyte disturbances occur when malnourished patients start any form of artificial nutrition. Profound electrolyte disturbance (hypophosphataemia, hypokalaemia and hypomagnesaemia) can cause serious complications such as muscle weakness, respiratory distress, rhabdomyolysis and arrhythmias.

Pathogenesis

Rapid carbohydrate administration after prolonged fasting causes hyperglycaemia with metabolic and hormonal changes. There is increased insulin secretion plus promotion of glycogen, fat and protein synthesis and increased basal metabolic rate. Transcellular shifts in phosphate, potassium and magnesium, and others, lower serum levels.

Prevention

High-risk patients need to be identified and receive dietician input. Start vitamin supplementation (oral thiamine or intravenous Pabrinex®) prior to feeding and continue for 10 days. Start feed slowly; monitor plasma electrolytes daily and correct electrolyte disturbances.

GLUCOSE MANAGEMENT

Hyperglycaemia and insulin resistance in the critically ill are common. Plasma glucose variability is associated with worse morbidity and mortality; both low and high levels are injurious. For most, a target range of 6 – 10 mmol/L is reasonable with or without a variable rate insulin infusion. Tighter control (e.g. 4.5 – 6 mmol/L) has been studied and no longer practiced due to worse outcomes, usually due to unintended hypoglycaemia.

GASTRO-PROTECTIVE MEASURES

Critically ill patients are at increased risk of stress ulceration and bleeding.

Risk factors include mechanical ventilation, coagulopathy, GI ulceration or having had a bleed in the past year, trauma and severe burns.

Pharmacological stress ulcer prophylaxis reduces GI bleeding and there is no superiority of one pharmacological agent over another. Commonly used drugs include H_2 antagonists (ranitidine 50mg TDS IV / 150mg BD PO) and proton pump inhibitors (omeprazole 40mg OD IV / 20-40mg OD PO). Use is associated with increased risk of nosocomial pneumonia and Clostridium difficile infection: it is therefore important to review the need daily.

Enteral feeding reduces ulceration risk. In lower risk patients, most clinicians stop pharmacological prophylaxis when enteral intake is achieved (i.e. NG feed tolerated).

WATER AND ELECTROLYTES

Critically ill patients can have significant fluid and electrolyte shifts. Careful attention to fluid balance is imperative to reduce major electrolyte disturbances and hypovolaemia.

Daily fluid requirement in health is 30 – 35 ml/kg/day that can be given via an enteral or intravenous route. Additional needs due to bleeding, capillary leak, drains, NG aspirates, high stoma outputs, fistulae, diarrhoea and polyuria should be addressed with replacement of fluid appropriate to what is being lost (i.e. is it water or an electrolyte rich fluid that is being lost?).
Electrolyte disturbances in the critically ill patient are common. Artificial nutrition regimens will provide the average daily requirements of the key electrolytes, but requirements vary depending on underlying illness. Most intensive care units regularly measure key electrolytes and correct them to the normal range (see Table 9.2).

Managing sodium and fluid balance issues on ICU can be complex and are beyond the scope of this chapter: simplistically, water excess results in hyponatraemia and water deficit results in hypernatraemia.

Hyponatraemia (Na < 135 mmol/L) is a common problem in hospitalised patients and the management will depend on the cause. Important iatrogenic causes include excess hypotonic fluid administration (5% dextrose IV) and drugs such as diuretics and PPI. Fluid overload is compounded by the requirement of multiple infusions, IV drugs and PN. Reviewing and rationalising drug volumes and where possible giving more concentrated versions can help to reduce the total volume of fluids being infused. Sodium loss (e.g. from GI or renal tract) and excess water retention (e.g. SIADH) is often found in the critically ill patient.

Hypernatraemia (Na > 145 mmol/L) has multiple causes, but on ICU it is often secondary to water deficit or excess administration of sodium salts. Many drugs have very high sodium contents (saline solutions, sodium bicarbonate, antibiotics) so it is prudent to review the drug chart and discuss any concerns with the intensive care pharmacist. Other methods of reducing sodium intake on ICU include changing to low sodium feed, or in the setting of water deficit, to give additional water via the NG tube.

GI PATHOLOGY

There are numerous problems, medical and surgical, that affect the gut leading to critical illness or occur as a complication of it. The range of all of these pathologies and the specific treatments is beyond the scope of this introduction but are well covered in other intensive care texts.

Electrolyte supplementation (Table 9.3)

Electrolyte abnormality	Common causes	Clinical symptoms	Treatment	Comments
Hypokalaemia Mild: 3.0-3.4 mmol/L Moderate: 2.5-2.9 mmol/L Severe: <2.5mmol/L	Increased losses: Renal GI-vomiting, diarrhoea, ileostomy, fistulae Drugs- diuretics, steroids Trans-cellular shifts: Insulin, salbutamol metabolic alkalosis	Often asymptomatic Symptoms most commonly occur K <2.9 mmol/L Weakness Constipation Ileus Respiratory failure Arrhythmias	Oral: Mild/ moderate hypokalaemia with no symptoms Sando K 2 tablets tds/QDS Intravenous: Severe hypokalaemia or symptomatic Peripheral: 40mmol KCl/L, Max 10mmol/hour Central: 1mmol KCl/ml Max 20-40mmol/hr	In hypokalaemia refractory to supplementation check for hypomagnesaemia Replacing potassium peripherally can result in fluid overload. Where continuous replacement is likely to be required this is best done with an infusion and given centrally.
Hypomagnesaemia Mild: 0.5- 0.69 mmol/L Moderate/severe: <0.5 mmol/L	Diarrhoea Vomiting Malabsorption Alcoholism Chronic renal failure Re-feeding syndrome Drugs: PPIs, diuretics	Often asymptomatic Symptoms most commonly occur Mg <0.5mmol/L Confusion Seizures Muscle weakness Tremor Arrhythmias	Intravenous: Mild/ asymptomatic: 20 mmol (5g) $MgSO_4$ in 100ml infusion fluid over 2-4 hours Moderate/Severe or symptomatic 40 mmol (10g) $MgSO_4$ in 250 ml infusion fluid over 2-4 hours	Often associated with resistant hypokalaemia and hypocalcaemia Use lower doses in renal failure Can be given peripherally and centrally Rapid administration may cause hypotension and flushing Oral supplementation is rarely indicated on ICU
Hypophosphataemia Mild: 0.6-0.79 mmol/L Moderate: 0.32-0.59 mmol/L Severe: <0.32 mmol/L	Malnutrition Sepsis Alcoholism Re-feeding syndrome	Often asymptomatic Symptoms usually occur PO_4 <0.32 mmol/l Muscle weakness Decreased cardiac contractility Convulsions Respiratory failure	Oral: Phosphate-Sandoz 1-2 tablets tds Intravenous: Phosphate polyfusor Give over 6-12 hours Mild- 1-2ml/kg Moderate-severe 2-5ml/kg	Phosphate polyfusor contains 0.1mmol/ml Excessive doses of phosphate may cause hypocalcaemia and hyperkalaemia Use lower doses in renal failure Can be given peripherally and centrally

Hypocalcaemia Mild: 1.08-1.17 mmol/L Moderate 1.0- 1.07 mmol/L Severe <1.00 mmol/L	Renal failure Pancreatitis Hypoparathyroidism (post thyroidectomy or parathyroidectomy) Tumour lysis synd.	Often asymptomatic Symptoms usually occur Ca2+ <1.00 mmol/L Neuromuscular excitability e.g. cramps, twitching, numbness Tetany Seizures Prolong Q-T interval and ventricular fibrillation	Oral: Adcal D3 2 tablets bd/tds Intravenous: Moderate hypocalcaemia with symptoms or asymptomatic severe hypocalcaemia 10ml of 10% calcium gluconate over 5mins Severe hypocalcaemia with symptoms 10ml 14.7% calcium chloride	40% of plasma calcium is bound to albumin, but it the ionized/unbound (Ca2+) fraction of calcium that is important physiologically. Ionized calcium is calculated by most blood gas analysers Calcium gluconate contains 2.2 mmol calcium and may be given peripherally Calcium chloride contains 10 mmol calcium, is very irritant and should be diluted in 50ml 0.9% sodium chloride if given peripherally Extravasation may cause tissue damage

CHAPTER 10: TRANSFER

Dr Adam Low and Dr Hannah Bawdon

LEARNING OBJECTIVES

- To be able to articulate the importance of thorough preparation prior to transfer
- To describe a methodology for making comprehensive preparations for the transfer of a critically ill patient
- To describe the utility of a checklist in this context

INTRODUCTION

Transfer and retrieval medicine is regarded as a medical sub-specialty in its own right. The ethos of conducting safe stabilisation and transfer of a critically ill patient depends upon "the rule of *right*":

"The *right* patient is moved at the *right* time by the *right* people, with the *right* equipment having undertaken the *right* training"

Therefore transfers, even in the hyperacute setting, need appropriate consideration, planning and co-ordination.

DEFINITIONS

The following simple definitions are useful when considering transfers:

Primary transfer: From scene of illness or injury to emergency department (pre-hospital)

Secondary transfer: Hospital to hospital transfer or department to department

Tertiary transfer: For specialist care to a tertiary centre

Quaternary transfer: International repatriation or for specialist care

Transfer team dynamic

Typically, the team will consist of:

- Doctor or ACCP trained in trasnfer: normally with advanced airway skills/intensive care experience
- Nurse: typically, with intensive care/ED experience
- Paramedic: medical knowledge and transportation modality knowledge (road ambulance or air ambulance)
- Additional member: e.g. perfusionist /trainee / observer
- Co-ordinator: may be remote and includes ambulance control

Training

To undertake a safe transfer the team must have core practical skills to allow interventions to be undertaken to stabilise the patient before or during the transfer. All team members should have a good working knowledge of the transfer equipment.

Transport options

Critically ill patients can be moved by road or air. Remember the time advantage of air transfers is not always realised due to need for loading, unloading and moving to and from a landing site. Factors determining mode of transport include medical considerations, stability of the patient, likely requirement for interventions en route and any time critical conditions, as well as weather, geography of the sector and the distance between centres.

Preparation

The team should have a shared mental model of the steps required to move the patient safely from one location to the next. The team should not be suffering from fatigue, distraction or illness themselves that may impair their performance. A team that works regularly together will assess and stabilise a patient rapidly and efficiently, reinforcing the importance of training.

Initial assessment and stabilisation

The transfer team should undertake a full systematic review (ABCDE or systems based approach) of the patient and their medical history. Early establishment on a portable ventilator will allow accurate equilibration with ABG analysis before departure. All lines should be secured and checked for patency. Endotracheal tube depth should be checked and chest x-ray reviewed for position relative to the carina, as should central lines. Do not delay a time critical transfer to site a central line. Depending on transport modality new lines may by inserted, for example UK land ambulances have limited access to the left side so it may be preferable for cannulae and arterial lines to be on the right side. Careful consideration should be made to pressure areas and position of infusion pumps.

Packaging

Extra time spent ensuring the patient is well packaged is time well spent. Some transfer teams will have specialist trolleys with charging points for syringe drivers and monitors that facilitate safe packaging. The key underlying principle is to ensure monitoring cables are not tangled and likely to dislodge, infusion lines are not kinked or under tension that risk dislodgement on patient movement and the patient is not at risk of pressure sores or neuropraxia. Temperature loss is a real risk and so hypothermia prevention is key. Consider placing a continuous temperature monitoring probe for the transfer.

Equipment

The team should be familiar with all the equipment used and be able to troubleshoot simple problems. This equipment should be regularly checked, and safety tested. Battery life should be assured with charge maintained when power sources are available.

PREPARATION

OXYGEN

Oxygen supply should be ample for the duration of the transfer. The following calculation is made to estimate oxygen requirements for the transfer of a ventilated patient:

Litres of Oxygen Required =
2 x ((minute volume + bias flow) x estimated duration of transfer (mins))

Equally ensure you have adequate supplies of drugs required for infusions and any emergency drugs required. For international repatriation, consideration of the legal aspect of controlled drug movement is essential, alongside custom clearance forms for medical equipment.

CHECKLISTS

Transfer medicine is the ideal setting for challenge response checklists. This ensures all steps have been adequately taken to ensure patient safety during a transfer, and things are not overlooked or forgotten. One team member will read out each item on the list and the second team member responds. If an item has not been addressed the checklist is paused until the necessary intervention completed. Some teams may also utilise “Standardised Operating Procedures” which will ensure all teams working for the organisation do things in a standardised way, thus assuring quality. A good checklist should be succinct but cover all important points in a systematic way. Anything too lengthy risks becoming an unhelpful distraction. This example from the KIDS (Kids Intensive Care and Decision Support) retrieval

team highlights the key elements of a safe paediatric transfer: (link from kids.bch.nhs.uk below)

COMMUNICATION

Good communication is essential throughout, both between the transfer team, but also between referring and receiving clinicians. Conference calls may be helpful, facilitated by the transfer team to ensure everyone shares the same plan and ideas. Informing receiving unit of time of departure and estimated arrival time is essential, as well as any delays that occur along the way. Make sure you know where you are going, especially if travelling to an unfamiliar institution, and ask for staff with security passes to meet you at a designated point to minimise delays on arrival. You should keep a thorough record of the transfer, interventions undertaken and physiological parameters that will facilitate audit, quality improvement initiatives and critical event reviews.

ADDITIONAL CONSIDERATIONS

Always ensure personal safety throughout. Thorough assessment and stabilisation of the patient should minimise any interventions required en-route. Ensure adequate clothing, personal protective equipment, warmth and comfort are maintained throughout. If motion sickness is a possibility, consider seating position in transfer and prophylactic anti-emetics. Monitors should be visible at all times with appropriate alarm settings. Ensure you have money/bank cards with you and a means of returning to your place of work in case the ambulance is re-tasked. Personal insurance should be considered and is offered by some organisations (e.g. Intensive Care Society).

SPECIAL CIRCUMSTANCES

Children: A number of specialist paediatric and neonatal retrieval teams exist in the UK; for example, the KIDS team in the West Midlands. These generally consist of a designated doctor, nurse and ambulance technician/response driver or pilot, who are tasked to go out to critically ill children at base hospitals and assist with stabilisation before retrieving them to an appropriate paediatric intensive care facility. One tasking can take several hours; it is therefore important intensive care teams remain up to date with relevant protocols and training, in case they need to undertake a paediatric transfer themselves. Telephone advice is always available from specialists at the receiving centre or retrieval service; this advice can be supplemented by online resources, for example clinical guidelines available on the KIDS website (http://kids.bwc.nhs.uk/healthcare-professionals-2/clinical-guidelines/)

Major trauma: Since the introduction of major trauma networks nationwide in 2012, significant changes have occurred to the way trauma care is organised in the UK. The most severely injured patients are either taken directly to a regional major trauma centre or transferred on following stabilisation in a trauma unit.

SUMMARY

- Remember the 'rule of *right*': "The *right* patient is moved at the *right* time by the *right* people, with the *right* equipment having undertaken the *right* training"
- Preparation is essential to a safe transfer; the patient, drugs, equipment and transfer team all need consideration
- Correct use of a checklist ensures vital steps are not missed, even in pressurised situations
- Specialised transfer teams exist for children and major trauma; however, they are not guaranteed to be available - train for all situations!

FURTHER READING

- http://www.ics.ac.uk/ICS/guidelines-and-standards.aspx (Guidelines for the transport of the critically ill adult, 3rd Edition 2011) e.g. checklists in pages 43-45
- https://www.aagbi.org/sites/default/files/braininjury.pdf (Recommendations for the Safe Transfer of Patients with Brain Injury, May 2006)
- https://www.aagbi.org/sites/default/files/interhospital09.pdf (AAGBI Safety Guideline: Interhospital Transfer, February 2009) e.g. checklist on page 14
- https://nswhems.files.wordpress.com/2015/12/predeparture-checks.pdf (Sydney HEMS Prehospital Predeparture Checks)
- http://kids.bch.nhs.uk/wp-content/uploads/2015/12/KIDS-pre-transfer-checklist-Dec-2015.pdf
- http://kids.bwc.nhs.uk/healthcare-professionals-2/clinical-guidelines/ (KIDS Clinical Guidelines)
- ABC of Transfer and Retrieval Medicine. Dec 2014, BMJ Books. ISBN: 978-1-118-71975-6A

CHAPTER 11: HUMAN FACTORS

Dr Kathryn Grange and Dr Simon Richards

LEARNING OBJECTIVES

- Explain the importance of Human Factors within the healthcare setting
- Describe current key theories in cognitive processing
- Identify examples of the application of human factors in the clinical setting (e.g. Checklists, Communication strategies)

KEY POINTS

- Human error is common in healthcare
- Automated thinking processes are prone to error and bias
- An awareness of whether system 1 or system 2 thinking processes are being predominantly engaged (and when to change) promotes a balance of efficiency and effectiveness
- Non-technical skills are techniques and strategies designed to mitigate the effects of "Human Factors"

INTRODUCTION

The study of human factors is a rapidly expanding scientific discipline that seeks to identify how the human condition influences our work and contributes to clinical errors. The study of why errors occur goes on to explore how they might be mitigated. No one is immune to error; irrespective of knowledge or experience; we are all vulnerable, and it is this pervasive sense of vulnerability that is the cornerstone to individuals recognising their own fallibility.

Research indicates 10% of patients admitted to hospital suffer some form of adverse event, with human error implicated in up to 80% of these cases. Despite this, healthcare lags behind other industries such as the military or aviation in terms of human factors awareness. There are many reasons for this, and undoubtedly the need to tailor care to the individual needs of each different patient, together with the sheer breadth of care needs which are provided for are large contributors. Nevertheless, it is also postulated healthcare practitioners often dismiss human factors principles as 'psychobabble', lacking an evidence base for improving patient outcomes, yet the evidence gathered from adverse events tells a

different story. The concept of human factors has gained considerable traction in recent years, highlighted by adverse events such as the crash of United Airlines flight 1549 in 2009 that landed in the Hudson River, and the death of Elaine Bromiley in 2005, which involved a failed intubation and led to tragic consequences through a failure to provide oxygenation by an alternative means. Cases such as these have driven the inclusion of human factors training into medical education, establishing systems to support reliable performance. Engagement in these systems (e.g. checklists) involves adjustments to clinical practices for the benefit of patients.

Human factors awareness and training aims to mitigate the risks of human error through adaptations to the working environment or behaviours (often referred to as non-technical skills) and contributes to a safer environment for staff and patients alike. Numerous models exist that attempt to explain the motivations behind certain human behaviours, and through extensive research these have led to the recognition and development of non-technical skills. These are increasingly considered to be just as important as clinical skills and technical knowledge, have the advantage of being transferrable between different areas of a clinician's own practice just as much as they are often applicable between entire specialty areas, and are equally important at every stage throughout clinicians' careers.

In this chapter, we will first consider the theoretical underpinnings of human factors before considering how these are applied in practice, specifically looking at Crew Resource Management, Communication strategies and finally the utilisation of checklists.

PRINCIPLES

There are three key principles in human factors; an appreciation that error is normal, human performance is variable, and any outcome is the result of a complex interaction between systems, processes, people and culture. The 'Swiss Cheese' model of accident causation, the paradigm first proposed by James Reason in the early 1990's, which highlights how the breach of multiple layers of protection usually precedes a mistake, exemplifies this. Some of these are human errors, some process or system errors.

If we are to understand human error, we must first attempt to understand why we think the way we do before we can begin to understand how errors might arise, and how we can reduce risk.

Cognitive processing is the 'process of thinking'. It involves the handling of both conscious and unconscious thoughts. The most prominent model of conscious thought is the 'Dual Process Model' discussed in Daniel Kahneman's "Thinking, Fast and Slow". This states there are two broad categories of human cognition: automatic (system 1; unconscious; fast) and analytical (system 2; conscious; slow). The key features of each are shown in Table 11.1. It is

likely humans initially evolved using predominantly automatic processing in a semi-conscious mode, with analytical thought (and awareness) developing later.

Table 11.1: Categories of Human Cognition

Table 11.1	System 1 (Automatic)	System 2 (Analytical)
Characteristics	Fast. Reflexive, skilled. Effortless. Looks for patterns. Creates narrative to explain events. Triggers emotions.	Slow. Deliberate, rule-based. Effortful. Can handle abstract concepts.
Reliability	Low, prone to error and bias.	High, few errors.
Advantages	Speed of response in a crisis. Easy completion of routine or repetitive tasks. Creativity through associations, so good for expansive thinking.	Good for reductive thinking. Can handle logic, maths, and statistics. Allows reflection and consideration of the "bigger picture", options, pros and cons, consequences.
Disadvantages	Jumps to conclusions. Unhelpful emotional responses. Can make errors that are not detected and corrected, such as wrong assumptions, poor judgements, and false causal links.	Slow, so requires time. Requires effort and energy, which can lead to decision fatigue. Prone to distraction and loss of thought place. Limited capacity therefore reduced effectiveness if preoccupied.

The advantage of analytical thinking over automated thinking is flexibility. It doesn't rely on pattern recognition (see cognitive biases below), so it is not thrown when no pattern is recognised and is less prone to diversion by small errors or incorrect pattern recognition.

(e.g. abdominal pain + tachycardia + fever = appendicitis, in contrast to abdominal pain + tachycardia + positive pregnancy test = ruptured ectopic pregnancy).

This model is useful in practice. When reflecting on our own performance, it is easy to evaluate which mode of thinking we were following and therefore identify where a change could potentially have made a difference. Equally, you may recognise a tendency to automated thinking in others, and this may prompt a conversation to take the approach back to a more analytical one.

This is not to say automated thinking is all bad. Consider the ABCDE approach to an emergency scenario. This simply provides a structure to a series of automated thoughts prompting the identification of abnormalities and corrections as you proceed. The best approaches balance engagement of both systems, but the key lies in recognising when to transition between them. Common examples of prompts to transition include failure of a patient to respond as expected, results of tests contradicting the working diagnosis, or senior review (e.g. a ward round).

It is important to recognise you are less likely to respond to these cues if you are distracted, for example if you are **H**ungry, **A**ngry, **L**ate or **T**ired (HALT). This is because automated cognition is fast and requires little energy, but you have to actively engage in analytical thought, which is less easy to do as the confounding factors build up and this is why there is often a natural tendency to drift towards automated, type 1 cognition.

However, analytical thought also has its limitations. It is a single channel thought process that is prone to reduced effectiveness if you are preoccupied, and it is often difficult to continue from where you finished if you find yourself distracted. More often you must backtrack on the preceding steps, which can make for a slow and inefficient process in a busy environment. It can also be easy to become fixated; so engrossed in one thought process that you lose track of time and context (the bigger picture) – known as 'paralysis by analysis' and represented under the umbrella term as a loss of "situational awareness".

Cognitive biases or heuristics are a valuable set of information processing shortcuts and are specific examples of automated thoughts (see Table 10.2). They are often learnt from personal experience and are most useful in situations when exhaustive searching would be too demanding or slow. They are based on conscious and/or unconscious patterns of recognition and are used to quickly determine what the next step or action ought to be. They are therefore fundamental to efficient working, but they have a major flaw, in that they are frequently inherently biased due to their tendency to draw us to one conclusion over another based on psychological factors rather than objective reasoning. The first step to avoiding these traps is to be aware of some common heuristics.

Table 11.2: Heuristics

Heuristic	Characteristic
Availability	The tendency to judge an event as more likely if it readily comes to mind (e.g. recent diseases encountered, or complications seen in personal experience as opposed to those listed in textbooks)
Anchoring	The disposition to persist with an initial judgement regardless of new information to the contrary (e.g. the A&E diagnosis that is not challenged). Also referred to as fixation bias.
Confirmation	The tendency to actively seek evidence to support a given view, rather than evidence that might refute it. (e.g. looking for evidence that an endotracheal tube is in the trachea immediately following intubation, such as auscultating over the chest, rather than actively excluding evidence that it is not, such as ensuring that any breath sounds heard over the epigastrium are not louder than those in the chest.)
Representative	Decisions based on recognising a typical example of a disease without considering base rates or the possibility of an atypical case (e.g. a rare presentation of a common disease is more common than a common presentation of a rare disease)
Premature Closing	Prematurely ending the decision-making process before it has been fully verified (e.g. satisfaction a diagnosis is correct when the first symptom is explained rather than ensuring the whole clinical picture fits.)
Sutton's law	The diagnostic approach of going for the obvious. Based on the story of Willie Sutton, a bank robber who when asked by the judge why he robbed banks responded, "Because that's where the money is!"

Following these principles, let's consider how we can use them to influence our clinical practice.

PRACTICE

Non-technical skills have been developed from years of study in Human Factors. They are commonly described using four key domains; see Figure 11.1 (taken from the Anaesthetists Non-Technical Skills (ANTS) system):

Figure 11.1: ANTS System

The key factor linking all four domains is **Communication**.

TEAM WORKING

Healthcare teams are diverse: from consultant surgeons and radiographers to paramedics and hospital porters. In critical situations, optimal communication is essential, requiring each team member to be fully engaged in the process. This requires skilled team leadership to drive and focus actions, directing team members with individual skills and responsibilities while encouraging them to voice and escalate concerns.

SITUATIONAL AWARENESS

'The ***perception*** of elements in an environment within time and space, ***comprehension*** of their meaning and ***projection*** of their status into the near future.'

This involves gathering information, recognising and understanding its importance and using this to anticipate future events. This is often done well initially, but as time progresses situational awareness can lapse. It is best maintained by actively monitoring the situation, keeping team members updated and engaged, and minimising distractions, but we all know this is more easily said than done during a busy on-call in a noisy clinical environment!

There are many barriers to good situational awareness. These commonly include fatigue, work overload, poor communication, staff complacency, environmental distractions and incorrect information processing (starting off on the wrong foot).

There may be many clues to a loss of situational awareness, for example:

- Recognising task fixation/preoccupation
- Confusion
- Lack of communication (everyone is quiet)
- Improper procedures or protocols
- Failure to meet targets (e.g. time to CT head)

DECISION MAKING

Good decisions rely on adequate situational awareness. Planned decisions should take into account all relevant information to formulate plans, whilst considering alternative options, including their risks and benefits, to come to a considered plan of action (e.g. the decision to take a high-risk patient to theatre for a laparotomy for a bowel obstruction vs. 'drip and suck' and reassess). Decisions must be clearly communicated to the rest of the team, including the rationale behind them, then executed and evaluated, with amendments made as necessary. Common mistakes include jumping to early decisions without first exploring alternative options, failure to consult others or challenge expert opinion, or failure to review the outcomes of those decisions.

TASK MANAGEMENT

'Organising resources and activities required to achieve goals.'

This has four steps:

Planning and preparing

The need to have a clear plan that is clearly communicated to the team
Make a plan B in case plan A becomes non-viable

Prioritising

Decide on task urgency and prioritise tasks
Avoid overloading one team member with multiple, time-critical tasks

Identifying and utilising resources

Consider the expertise of your team and allocate tasks accordingly

Providing and maintaining standards

Follow appropriate guidelines and protocols where applicable
Ensure adequate documentation takes place and consider allocating the role of a scribe

We will now look at specific techniques used within healthcare to address common

CREW RESOURCE MANAGEMENT (CRM)

This is a team management tool initially developed and used routinely within the aviation industry and translated into healthcare, often substituting the word "crisis" in place of "crew" in the CRM acronym. It utilises a systematic approach that can be applied during critical events or high stress situations and seeks to avoid errors due to missed information and distraction. It focuses on optimising communication by prioritising clear, calm and concise dissemination of information, thus ensuring team members are kept briefed.

It incorporates:

- A clear leadership structure.
- A defined process for raising questions or concerns.
- Established routes for escalation of those concerns.
- Policies to minimise distractions.
- Utilisation of challenge checklists (see below).
- Standardised training

The potential uses of CRM in healthcare are evident. Consider the well-run trauma call: All examination findings and communication run through the team leader. Each team member is allocated a clear role and list of responsibilities and is individually empowered to raise concerns via the team leader. Standardised trauma course teaching emphasises the importance of using names and feedback loops – e.g. 'Sarah, please put out a major transfusion call on 2222, and come back and tell me when you've done it'. Concerns are further escalated using formalised speech, an increase in pitch and volume of voice, and via direct eye contact and verbal acknowledgement with the team leader.

COMMUNICATION STRATEGIES

Critical or untoward incidents virtually always involve some form of communication failure. Several techniques have been introduced to healthcare in recent years to improve multidisciplinary communication and facilitate the rapid escalation of concerns.

Structured communication tools are being used increasingly to reduce communication barriers across disciplines, providing a shared mental model for the handover of information and to act as a memory prompt to reduce omissions. They encourage participants to prepare for communication, keeping the conversation brief and limited to relevant information. Research has shown when receiving complex information concentration begins to wane at around 20 seconds. Use of a clear and familiar structure can extend this to two minutes. Therefore, emphasis should be on including key facts only; further details can always be sought later.

SBAR

SBAR is a commonly used and useful tool for framing critical conversations requiring immediate attention and action.

→ Briefly describe the current situation

Clear, succinct overview of pertinent issues

→ State the pertinent history

What got us to this point?

Summarize the facts

PACE

Where an individual team member identifies a concern an effective means of escalating and resolving concerns is required. One method is represented by progression through the PACE mnemonic and facilitates escalation from raising awareness all the way to taking control in the very rare situation this might be required.

A patient is having an OGD under sedation. Oxygen saturations are falling.

"John, is everything OK? Are you happy with sats of 85%?"

"Dr Smith, this patient's saturations are falling. They are 80% and should be higher." -

"Doctor you need to stop what you're doing. The patient is unstable. His saturations are only 75%!"

"This is an emergency. We must stop the procedure!" – Intervenes and commences airway management. – or **STOP, STOP, STOP.**

BLUF

Originally a military strategy, BLUF stands for **B**ottom **L**ine **U**p **F**ront, and is particularly useful in emergencies when you need to summon help quickly. A good example is the early-hours phone call to the ITU consultant:

Option 1

'Dr Smith, it's David here. I'm calling about an unwell child in resus.

'He's a four year old, 16kg child with a three day history of sore throat and difficulty swallowing. Mum states that he's been worse today, and is now having difficulty breathing.

'His obs are stable, with a HR 140, BP 90/40, SpO2 94% on air, GCS 15. He is stridulous, and his work of breathing is increasing. I think he's got acute epiglottitis and is going to require intubation.

Option 2

'Dr Smith, it's David here. I need you to come in immediately for a paediatric airway emergency in resus.'

'I've got a 16kg four-year old with likely epiglottitis. Obs are currently stable, but he is becoming more distressed and stridulous and is going to shortly require intubation.'

'The paeds and ENT consultants are en route and theatres are preparing for our arrival. Please meet me in resus ASAP.'

Option 1 uses a more structured approach and gives all relevant information, but option 2 means the consultant is already putting their shoes on to reach you as soon as possible.

CHECKLISTS

Checklists recognise many tasks in healthcare have now become too complex for an individual to reliably perform in every instance. Even for simple tasks, such as inserting a central line, checklists have improved adherence to infection control procedures with dramatic reductions in line infection rates.

Checklists are, therefore, being increasingly implemented in healthcare. They, like most other strategies in human factors training, are already widely used in other industries to facilitate the rapid, structured handover of information, improving safety and situational awareness. A 'challenge checklist' requires a response from questioning – most commonly this is 'CHECK', meaning the information has been received, understood and agreed. To be effective, checklists must be unambiguous and quick to perform, focusing on essential points (those that could swiftly prove fatal, common omissions or elements to improve team working), rather than exhaustive.

The most well known is the WHO Surgical Safety Checklist. This should be completed for every patient that enters the operating theatre and is designed to ensure the whole theatre team is informed about the patient, intended procedure, potential or anticipated adverse events (e.g. allergies) and actions should they occur.

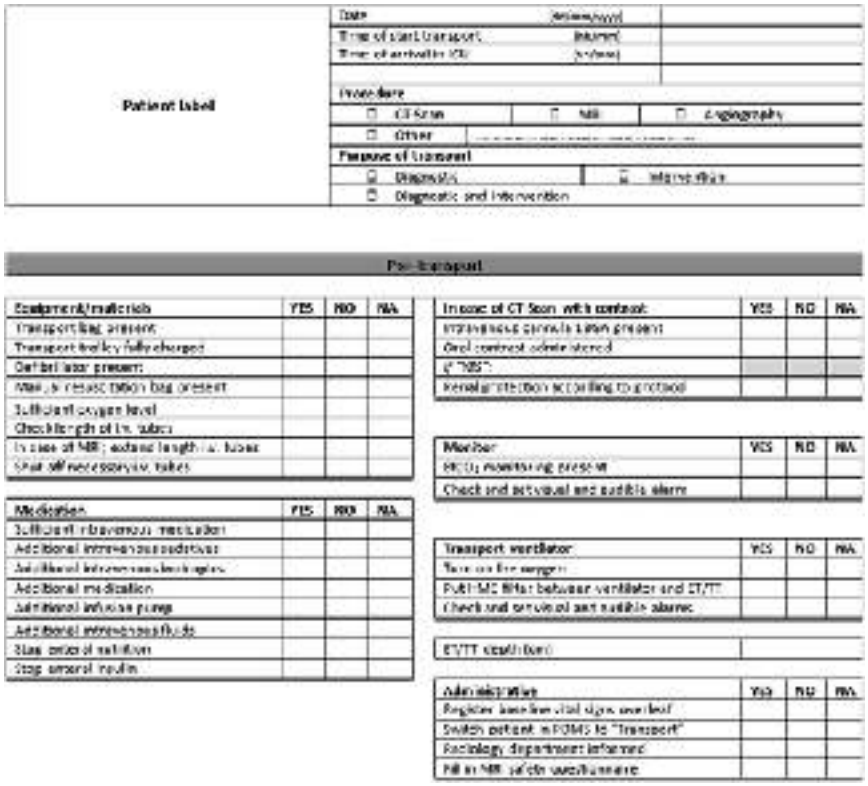


Figure 11.2: Hospital Transfer Checklist Example

The WHO checklist has been modified for specific situations for use outside the operating theatre, e.g. for RSI in Intensive Care (Figure 3.9). Further amendments are made in life-

threatening emergencies to eliminate delays to treatment whilst maintaining safety. These use the same checklists with shaded areas that can be left out. Examples include the checklists used for emergency intubations or category 1 caesarean sections, where delays may have unacceptably deleterious consequences.

Alternative indications for checklists include hospital transfers (Figure 11.2), where unfamiliar environments, teams and clinical situations make omissions and human errors more likely. Checklists are useful to ensure all equipment is available and the transferring team is adequately trained, briefed and prepared for adverse events that may arise.

SIMULATION TRAINING

Most human factors training will utilise simulation training. Simulation training has once again been pioneered by the aviation industry to analyse human behaviour under stress, and to identify key steps in the management of critical incidents. Identification of these points then allows pre-planning for critical eventualities, aiming to reduce panic and irrational responses in the face of adversity and introduce a systematic approach to problem solving, thereby reducing the risk of potential error.

Simulation training is becoming commonplace in medical education, with increasingly high-fidelity situations being replicated and is increasingly being recognised as a highly effective training modality. In addition to allowing us to explore our vulnerabilities and formulate mitigating action plans, processes reinforced through active simulation are much more effectively learnt than through more traditional, passive learning experiences such as reading or lectures.

While it is not the same as ‘real-life’, simulation nevertheless provides excellent opportunities to use and practice the non-technical skills outlined in this section.

SUMMARY

Human factors are a developing area of modern medicine and underpin all clinical behaviours. Their importance becomes particularly clear in emergency scenarios, but is equally important in promoting safe, comprehensive care in less urgent scenarios. They encompass a wide range of behaviours, including how we think, how we act and how we communicate, and are equally important to every member of the team irrespective of role or seniority.

FURTHER READING

- http://www.rcoa.ac.uk/system/files/AaE-ANTS-HANDBOOK.pdf
- https://learningcentral.cf.ac.uk/bbcswebdav/institution/Medic/Undergraduate/Human%20Factors%20%26%20Non-Technical%20Skills%20(SBAR)/src/ppts/Human%20Factors%20and%20NOTECHS.pdf
- http://icmwk.com/wp-content/uploads/2016/02/Trauma-ATACC-Manual-2014.pdf
- http://patientsafety.health.org.uk/sites/default/files/resources/human_factors_in_healthcare_course_handbook_en_march_2013.pdf
- http://www.who.int/patientsafety/research/methods_measures/human_factors/human_factors_review.pdf
- Gawande, A (2010). *The checklist manifesto: how to get things right.* New York, Metropolitan Books.
- Kahneman, D (2011). *Thinking, Fast and Slow*. New York, Farrar, Straus and Giroux

CHAPTER 12: END OF LIFE MANAGEMENT IN THE INTENSIVE CARE ENVIRONMENT

Dr Hafiz Aladin & Dr John Bleasdale

LEARNING OBJECTIVES

- Recognise the importance of effective end of life management in the intensive care environment
- Describe the difference and example rationales for decisions to
  - Withhold treatment options from the outset
  - Limit escalation of treatment either at the time of admission to intensive care or during the provision of intensive care support
  - Withdraw treatment from a patient currently receiving intensive care support
- Understand the current position on the use of DNAR orders and the requirements for communication with patients and relatives.
- Understand the basics of an advanced directive (or living will)
- Describe the role of organ donation in epidemiological terms
- Explain the difference between DBD and DCD
- Describe the fundamentals of BSDT as a pre-requisite for DBD

INTRODUCTION

Effective and compassionate end of life care is vital within Intensive care due to the frequency with which deaths occur (as a consequence of the severity of illness) and the fact this deterioration can often be very rapid. Most patients and families hope for a recovery following deterioration in health, especially if their care has been “escalated”, but where this recovery does not occur it is vital to manage the end of life phase in such a way that maintains patient comfort and limits distress for their loved ones. Increasingly marginal cases are admitted to the intensive care unit because of a lack of certainty or effective predictive tools to establish whether, on an individual basis, treatment may be effective. This is increasing the frequency with which effective palliation must be delivered in the intensive care environment.

TREATMENT AND RESUSCITATIONS DECISIONS

Society has developed an unrealistic belief we can prolong life indefinitely and death is no longer inevitable but a failure of medicine. Though we may be able to prolong life the primary goal of medicine is the reinstatement of a patient's health to a quality of life they find acceptable. For the majority the prolongation of life, sometimes without a return to prior levels of health, is an appropriate goal. However, the prolongation of life at all costs, without regard to the quality of that life or the burdens imposed by treatment, may not always be desirable.

In intensive care the hardest decisions are those concerning when to withhold or withdraw treatment. Despite this the majority of patients who die in intensive care departments do so after a decision to withhold or withdraw life-prolonging care.

It may seem withholding a treatment or withdrawing a treatment that has already started are very different decisions. This is not the case, for just as a doctor cannot offer a treatment that is non-beneficial neither can a treatment continue if it is no longer providing any benefit. The decision relies on a very clear understanding of benefit and the ultimate aim of treatment. If we accept "benefit" is the restoration of the patient's health to a quality of life they find acceptable and if the potential success of a treatment carries a "cost" that the patient would find unacceptable then the treatment should not be commenced in the first place, or should be withdrawn if already started.

It is imperative for every patient who is critically ill, limits for active treatment and the role of cardiopulmonary resuscitation are considered. All attempts should be made to have a discussion with patients who have capacity regarding DNACPR decisions. No assumptions should be made about the patient's wishes including whether they would wish to know about a DNACPR order.

ASSESSING CAPACITY

It is essential we ensure a patient is capable of making any decision regarding their treatment. The Mental Capacity Act 2005 states it must be assumed all persons have capacity. It also makes clear all practicable steps have to be taken to help them make a decision and that merely because a decision is unwise does not indicate incapacity.

The Act provides guidance on assessing a person's ability to make decisions; a person is unable to make a decision if unable:

- To understand the information relevant to the decision
- To retain the information
- To use or weigh that information as part of the process of making the decision, or
- To communicate his decision (whether by talking, using sign language or any other means)

Because of their medical condition, many intensive care patients could be considered to lack capacity. The Act makes it clear that the extent of the patient's condition and complexity of the treatment do not automatically preclude the patient from providing a valid consent. Though incompetent to consent on one treatment a patient may be more than capable of consenting, or refusing their consent, for another. As each consent issue has to be viewed as an independent event, "at the material time", we must make an assessment of a patient's capacity for each decision.

If a patient, who has capacity, does not wish to know about a DNACPR order, attempts should be made to seek agreement to share such information with those close to them.

ALTERNATE DECISION MAKERS

For patients who lack capacity the treating team must make treatment decisions based on the best interests of the patient. Very occasionally a patient may have, prior to their loss of capacity, appointed a Welfare Attorney; this is a person who, if clearly specified, is responsible for ensuring decisions are made in accordance with best interests of the patient. More frequently there is no such provision, and, in these circumstances, there is a hierarchy of people who can act as advocates for the patient to assist the treating team in determining a patient's best interests. If none of these can be found, then an Independent Mental Capacity Advocate (IMCA) must be consulted.

Even when there are relatives, friends or an IMCA to help the treatment decision rests with the treating team and if, after prolonged discussions with the advocates, an agreement regarding the patient's best interests cannot be reached then the Court of Protection will need to be approached for a declaration.

You should make it very clear to them the role of the advocate is an advisory one only and not to give them the impression that they are responsible for any decisions.

ADVANCE DECISIONS

An advance decision can cover a wide range of circumstances, it is a statement, expressed when a patient has capacity, of their wish to refuse a particular type of medical treatment or care if they become unable to make or communicate decisions for themselves at some point in the future. They are called advance decisions in England and Wales, and advance directives in Scotland.

If a patient lacks capacity and information about a written or verbal advance refusal of treatment is recorded in their notes or is otherwise brought to your attention, you must bear in mind that valid and applicable advance refusals must be respected.

DIAGNOSING DEATH

"*Death entails the irreversible loss of those essential characteristics which are necessary to the existence of a living human person and, thus, the definition of death should be regarded as the irreversible loss of the capacity for consciousness, combined with irreversible loss of the capacity to breathe. The irreversible cessation of brain-stem function whether induced by intra-cranial events or the result of extra-cranial phenomena, such as hypoxia, will produce this clinical state and therefore irreversible cessation of the integrative function of the brain-stem equates with death of the individual and allows the medical practitioner to diagnose death*"

Association of Medical Royal Colleges 2008

Hence death can be defined as either the irreversible cessation of circulatory function, or the irreversible loss of function of the brain and brain stem.

DIAGNOSING AND CONFIRMING DEATH FOLLOWING A CARDIORESPIRATORY ARREST

This is how the vast majority of deaths in the UK are diagnosed. Following a cardiorespiratory arrest death can be diagnosed by any appropriately trained individual by confirming the irreversible cessation of neurologic, cardiac and respiratory activity.

The deceased should be observed for a minimum of five minutes to confirm that irreversible cardiorespiratory arrest has occurred (lack of pulse and heart sounds). After five minutes of continued cardiorespiratory arrest the absence of the pupillary, corneal and motor response to supra-orbital pressure need to be confirmed.

DIAGNOSING AND CONFIRMING DEATH IN A PATIENT IN COMA

Approximately 4% of all deaths on Intensive care Units (that is 1 in 500 UK deaths) are diagnosed this way. For many of us (except those working in neurosurgical units) this will be a relatively infrequent occurrence so a standardised approach for undertaking and recording the necessary clinical tests has been produced by the Faculty of Intensive Medicine ("*Form for the Diagnosis of Death using Neurological Criteria*")[1]. It is recommended that this form is used whenever diagnosing death using neurological criteria. The diagnosis is based on demonstrating the absence of response to stimuli in the distribution of the cranial nerves and apnoea for five minutes.

- The tests should be completed by at least two medical practitioners, who should have been registered with the General Medical Council (or equivalent professional body) for more than five years and have been trained to complete the tests. At least one must be a consultant
- They must not have (or be perceived to have) any conflict of interest and they should not be a member of the transplant team
- The entire set of tests must be completed on two occasions

Those undertaking the tests must be satisfied that they know the cause of the irreversible brain injury being tested and that they have excluded any potentially reversible causes of coma and apnoea.

A period of observation may be needed to be confident that the brain injury is irreversible. This is particularly the case following a cardiac arrest, when 24 hrs of observation after re-warming is recommended.

Before undertaking the tests, several preconditions must be met:

- The patient must have a GCS of 3/15 and be apnoeic
- Stabilisation of the patients is essential
- Consistent MAP >60mmHg
- Biochemical haemostasis
    - Core temperature >34°C
    - Na^+ 115 – 160 mmol/L
    - K^+ > 2 mmol/L
    - PO_4^{2-} 0.5 – 3.0 mmol/L

- Mg^{2+} 0.5 – 3.0 mmol/L
- Blood glucose 3.0 – 20.0 mmol/L

- Other causes must be excluded
  - Residual sedative drug effects
  - Endocrine abnormality
  - Residual neuromuscular blockade

Only once both clinicians are satisfied all these preconditions have been met can they progress to test for the absence of brain-stem function and the apnoea test.

The tests themselves require very basic clinical skills. The tests apply a noxious stimulus in the distribution of the cranial nerves and assess for a response. If a response were to be forthcoming it would demonstrate that there must be functioning synapses within the brain stem and the patient would not be dead by neurological criteria, hence the tests are for the absence of a response.

The tests (and cranial nerve being tested), repeated on each side, are:

- Do the pupils react to light (II, III)
- Do the eyelids react to corneal stimulation (V, VII)
- Is there a response to the injection of 50 mls of ice-cold water over 1 minute into the ear (III, VI, VIII)
- Is there a gag reflex (IX, X)
- Is there a motor response to supraorbital nerve compression (V, VII)

Once the absence of cranial nerve responses has been demonstrated the persistence of apnoea can be tested.

The test is the absence of spontaneous breaths during 5 minutes of continuous observation.

For the test to be effective the $PaCO_2$ must be high enough to provide a stimulus to breathe at the start of the test and rise by at least 0.5 kPa during the test (the recommended starting $PaCO_2$ is >6.0 kPa with a pH <7.4). Also, as cardiac pulsation may trigger supported breaths if the patient is connected to the ventilator, it is necessary to disconnect the patient from the ventilator to test for apnoea. Therefore, to maintain adequate oxygenation, either a catheter needs to be inserted into the trachea with an oxygen flow of >6 L/min or a Mapleson B circuit may be required to provide some CPAP if oxygenation is a problem.

Cardiovascular stability must also be maintained during the apnoea test.

The complete set brain-stem function and apnoea tests must be repeated a second time. There is no fixed time period to wait between the two sets of tests, as long as all the preconditions are met for the start of the second set of tests. The doctors completing the second set of tests do not need to be the same individuals who completed the first set of tests, as long as they fulfil the requirements set out above.

The time of death is to be recorded as the time of completion of the first set of tests

It is good practice to give any family the opportunity to witness the tests. They should be prepared beforehand for the nature of the tests and the possibility of spinal reflexes and their relevance. It should be explained to the family the purpose of the tests is to confirm death has occurred.

When death is diagnosed by neurological criteria, the patient is dead even though ventilation and circulation can be maintained in the short term and the patient doesn't look dead (in the classical cardiorespiratory arrest sense). The next step is to consider withdrawal of support, the ethical justification for which (necessity and the preservation of life) has passed.

ORGAN DONATION

Organ donation for transplantation offers those with end stage organ failure improved survival and quality of life and has become an increasingly important part of the responsibilities of the intensive care team. Consideration of a patient's wishes regarding organ donation is an essential component of our care for patients at the end of life in the intensive care unit and though occurs at a time of great emotional distress for a patient's family and friends it has been shown to provide them some comfort knowing some good has resulted from the death of a loved one.

In spite of significant investment in organ donation and transplantation, rates of organ donation in the UK remain significantly lower than other western countries and the supply of organs for transplantation consistently falls short of demand.

In 2016/17 only 1413 people donated organs after their death that resulted in 3710 deceased donor transplants. 457 people died whilst waiting for a transplant and a further 875 were removed from the waiting list because they had become too ill to receive one in the time they had been waiting. The current active transplant waiting list numbers approximately 6,400 but this number underestimates the true need as more and more individuals with diabetes and hypertension survive to develop end-stage organ failure.

For transplanted organs to function well in their new host they need to be relatively undamaged prior to donation and be donated in a manner that prevents significant deterioration in their performance during the process. Ischaemic time of organs prior to

retrieval is the single biggest contributor to loss of function during the process. Warm ischaemia is most damaging to retrieved organs and is when there is inadequate oxygenation of tissues (systolic arterial pressure < 50mmHg, SpO2 <70%, or both) at normothermia.

There are three circumstances whereby someone can donate organs to others

1. **Living donation**: A healthy person, usually but not exclusively related to the recipient, chooses to undergo an elective procedure to remove an organ, or part of an organ, which is then transplanted into the recipient. Not considered further in this chapter.

2. **Deceased donation**: Organ donation proceeds after the diagnosis of death.

- **Donation after circulatory death (DCD):** Occurs following cardiorespiratory arrest, usually following the withdrawal of organ support in an intensive care unit (controlled DCD). The decision to withdraw organ support needs to be independent of the decision to donate organs and in the patient's best interests. In some centres, with resident retrieval teams, organ donation can occur following unexpected cardiac arrest (uncontrolled DCD). Organs will have an inevitable and unpredictable period of warm ischaemia before the circulation stops and retrieval can begin

- **Donation after brain death (DBD):** The donation of organs after the confirmation of death by neurological criteria. The heart continues to beat and in situ cooling before the circulation is arrested just before organ removal can prevent warm ischaemia.

Between 2001/2 and 2016/17 the number of organ donors after cardiac death (DCD) has risen from 37 to 584, in the same time period the number of donors after brain death (DBD) has only risen from 736 to 829.

The only absolute contraindications for organ donation are:

- Active invasive cancer in the last 3 years (excluding non-melanoma skin cancer and primary brain tumour)
- Haematological malignancy
- Untreated systemic infection
- Variant CJD
- HIV disease (but not HIV infection)

The process of organ donation

- All Intensive care Units should have a comprehensive organ donation policy based on national guidance
- Decisions regarding the withdrawal of treatment must be consistent and transparent and must be made before the potential for organ donation is considered
- No treatment specifically aimed at organ donation should be instituted before the decision to withdraw treatment has been made. UK law permits life-sustaining treatment as being in the best interests of the individual if they have expressed an interest in being a donor

In the UK, NHS Blood and Transplant (NHSBT) is the authority that oversees donation and transplantation. Screening of potential donors, family discussion and consenting and placing of the donated organs are undertaken by specifically trained Specialist Nurses for Organ Donation (SNODs) and they should be contacted as soon as possible Referrals to the SNOD team should be made as soon as death by neurological criteria is suspected or confirmed, or once the decision to withdraw treatment has been made in patients whose best interests are no longer served by continued treatment.

The SNOD will approach the family about organ donation and manage the whole process, which can sometimes take over 24 hours due to delays occurring at any of the stages in the lengthy and complex pathway. They will

- Check to see whether the potential donor is on the organ donation register (ODR)
- Clarify the clinical situation, understand the family's ideas, concerns and expectations and meet any family needs (e.g. faith leaders)
- Explain the processes surrounding organ donation and obtain the family's agreement for donation to take place (There is very good evidence that the SNODs are much better at these conversations than clinicians)

- Tissues type the donor, check their medical history and organise any necessary screening tests
- Liaise with transplant teams across the UK so potential recipients can be identified.
- Identify and organise the retrieval team(s)- there may be different abdominal and thoracic teams
- Book theatre time for the donation and conduct any discussions with HM Coroner (if needed)
- Arrange transport for the donated organs to the transplantation centres
- Remain with the donor and support their family throughout the process

NHSBT operates an integrated UK-wide service (Figure 12.1). Though SNODs are locally based, embedded in hospitals with regional on call rotas, the retrieval team may have to travel some distance to the donation site and the organs can then be distributed to many different transplant centres throughout the UK.

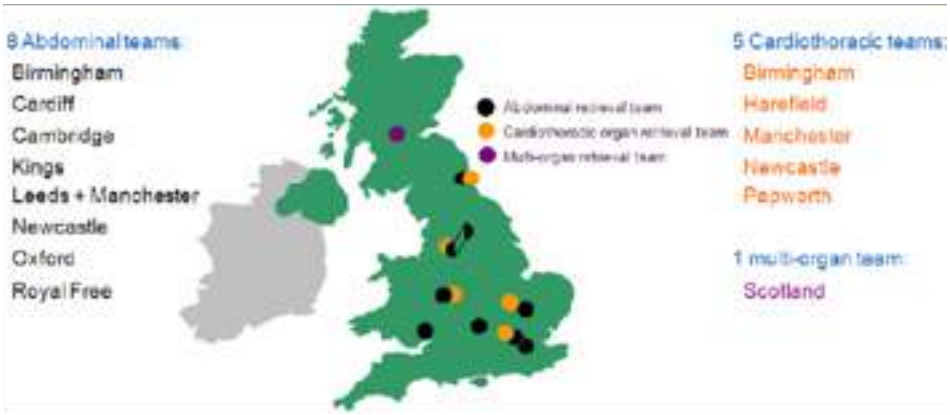


Figure 12.1. Distrubition of team providing the National Organ Retrieval Service (NORS)(2)

Once we reach the stage of organ donation the retrieval team will be in an operating theatre at the donor's hospital, the transport teams will be ready and for some organs the recipients will have been called in to their local transplant centre. The donation can then begin.

DBD: The donor is transferred to theatre, an anaesthetic administered (to prevent spinal reflexes) and the retrieval teams inspect the organs in situ before making the final decision to proceed with the donation. They then dissect the organs but do not remove them from their blood supply. They pack the thorax and abdomen with ice to cool down the organs and at the last moment stop the heart to arrest circulation before ligating the last few vessels and removing the organs. The retrieval team close the thorax and abdomen.

DCD: The process is quite different. The donor may be transferred to theatre or remain in the Intensive care for the withdrawal of care. The family will often want to stay with their loved one until death is confirmed so allowances for their needs are essential. Once the heart has stopped and there has been asystole for 5 minutes death can be confirmed, and the donation begin. Depending on the warm ischaemic time the retrieval team can then begin the surgical procedure. Maximum warm ischaemic times are organ specific. The surgical procedure for the retrieval or organs for DCD is a modified, and more rapid, version of that for DBD.

SUMMARY

Discussions about "ending life" are frequently difficult. The complexity of critical illness, including the interplay of multiple co-morbidities with the current illness, means effective communication is vital in order reach a decision about withdrawal of treatment. More often than not patients lack the capacity to take part in this decision and their views may not be known. Rapid deterioration may also make the situation hard for family members to accept. In addition, differences in understanding between family members and conflicts of opinion between family members and clinical staff are common and must be managed with care.

Death can be diagnosed in terms of cardiorespiratory death, or the irreversible cessation of brainstem function. Intensive care specialists need to be familiar with the procedures for diagnosing death in both circumstances

Organ donation remains a relatively infrequent event. The legal and ethical frameworks that underpin organ donation can appear complex and concerns are often raised within the clinical community about conflicts of interest and the lawfulness of interventions before death. There is a robust process for identifying and managing potential donors and without effective mechanisms in place potential donors can be missed.

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