WARWICK MEDICAL SCHOOL

HANDBOOK OF CRITICAL CARE

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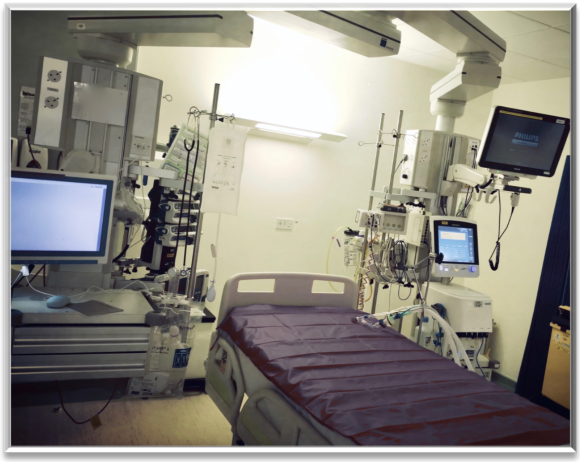

Preface

In the past, the exposure to critical care was provided as an "add-on" to surgical rotations, as such, successive interest in the specialty was highly dependent upon the experiences encountered, and the individuals involved in teaching. Over the years, the principles of critical care have been introduced during the early years of medical school curriculum. It is an important skill for a foundation year doctor to recognise a critically ill medical or surgical patient and initiate appropriate management strategy. A sound knowledge of basic sciences forms a strong foundation for managing critically ill patients.

This handbook hopes to outline some commonly encountered situations within the critical care unit and explores the basics of the physiology that underpin the principles of care.

The chapters in this book are selected from the learning outcomes of phase 3 curriculum, particularly acute medicine block. The topics on airway management and fluid therapy are included in the previously published handbook of peri-operative medicine. In order to simplify and help you learn, at your own pace, we have added links to videos and online material which should facilitate understanding of the concepts we present.

The handbook can be referred to throughout training, and some of the concepts will even hold true to trainee doctors. Despite technological advances over the years, understanding of how the human body functions have changed little, therefore appreciation of such concepts would be useful regardless of which specialty is chosen following graduation.

Alongside the previously published handbooks on Perioperative Medicine and Essence of Clinical Procedures this handbook is an ideal companion for medical students.

We wish you luck during this block and hope this handbook is a reference for further learning in the years to come.

We value your feedback, please email your comments and suggestions to
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INTRODUCTION TO CRITICAL CARE MEDICINE

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The principles behind critical care stretches back to the Crimean War in the 1850s when nurses cared for critically ill patients in a separate area near the nursing station. Beyond this, neurosurgical patients in Baltimore, were managed and monitored in a dedicated area, away from other post-surgical patients. However, the development of intensive care as a specialty is often traced back to Copenhagen in 1952, following a significant polio epidemic. Hundreds of patients suffered life threatening respiratory impairment and were artificially ventilated by hand. Bjorn Ibsen, the anaesthetist involved, has been credited as the father of intensive care as he established positive pressure ventilation to be the treatment of choice for such patients, and therefore set up the first critical care unit in Europe.

Over the years, intensive care has seen growing interest and development within hospitals, as patient's life expectancy increases, co-morbidities become more complex and medical capabilities improve. However, despite this, demand for intensive care beds often far out strips supply, and suitable patient selection is paramount to ensure efficiency and fairness of care.

Criteria for critical care admission

Surgical patients with significant co-morbidity may be admitted to critical care for postoperative care and monitoring. Patients deteriorating on the ward or post-surgical patients should be discussed with the senior clinician responsible for the intensive care department. Ultimately admission depends on:

- Reversibility of the illness and the intended benefits of intensive supportive care.
- Significant comorbidities need to be assessed and the baseline physiological performance needs to be ascertained, as ultimately success with therapy in intensive care is far more likely in those with a greater physiological reserve.
- Patient wishes should be respected if they have expressed a preference against admission to intensive care and have an valid advanced directive.

Level of critical care

There are escalating levels of care provided depending on clinical need. Ward based care is termed "Level 1" care, whereby the needs can be met on an acute ward.

Level 2 care is used to describe a High Dependency Unit (HDU) level of care. This includes a greater degree of monitoring and support than can be provided on a regular ward. Nurse to patient ratio is often 1:2 and is reserved for patients requiring organ support (such as vasopressors, invasive blood pressure monitoring or non-invasive ventilation) or following surgery, whereby a more frequent and greater degree of monitoring is required.

Level 3 indicates intensive care and such patients require significant organ support (usually two or more systems), such as invasive ventilation, inotropic support, or renal dialysis). Nurse to patient ratio is 1:1 on a Level 3 unit, such as Intensive Care.

Categories of organ support:

- Basic Respiratory Monitoring and Support – need for more than 40% inspired oxygen, intensive respiratory physiotherapy.
- Advanced Respiratory Support – Mechanical ventilatory support
- Circulatory Support – Inotropic support or vasopressor support
- Neurological monitoring and support – Invasive neurological monitoring
- Renal Support – need for Renal Replacement Therapy (RRT)

Assessment of the deteriorating patient

A clear, logical approach is required when assessing an acutely unwell patient who may in turn require critical care support. However, in most instances, early detection and simple therapies are often sufficient to improve the clinical derangement. The acronym "ABCDE" is widely employed, and establishes solid foundations in assessment and planning for further care.

Airway

Assessment

Assess the patency of a patient's airway, according to breath sounds and visual inspection. Such information can give rise to the potential causes of compromise:

- Oral
    - Relaxation of soft tissues (tongue/posterior pharyngeal wall)
    - Foreign Body
    - Blood/Vomit
- Laryngeal
    - Oedema (Burns/Inflammation/Swelling)
    - Laryngeal Spasm
- Below Larynx
    - Anaphylaxis
    - Bronchospasm
    - Secretions

Sounds such as gurgling, wheezing and stridor indicate a potential for airway obstruction. If there is an obvious cause of obstruction on assessment (such as dentures, teeth or foreign objects), an attempt should be made to remove these if safe and appropriate.

Furthermore, as a result of prolonged compromise, patients may become cyanotic, and experience cardiovascular collapse. Bradycardia develops after severe hypoxia, due to activation of peripheral chemoreceptors as a response to low PaO_2, and through alteration of cardiac resting membrane potential. As a result of the drop in the cardiac output and peripheral vasodilation, there is a fall in blood pressure and systemic vascular resistance that can ultimately progress to asystole and death.

Intervention
If there is an indication of compromise, the first technique is to deliver adequate oxygen. 15L/min oxygen via a non-rebreathe mask is first line and subsequently the concentration of delivered oxygen can be altered following clinical response.

If there is visible airway soiling or gurgling sounds, the oropharynx should be suctioned under direct vision. If there is a failure to resolve this, then simple manoeuvres such as head tilt/chin lift can improve airway patency (caution with head tilt in patients with suspected or known cervical spine injury). Airway adjuncts are next line and either an oropharyngeal or nasopharyngeal airway should be inserted after appropriate sizing.

If there is further deterioration beyond this or the above steps do not resolve the patient's compromise, alert the anaesthetist on-call and place a "peri-arrest" call via switchboard.

Breathing
Assessment
Following airway assessment and stabilisation, breathing and ventilator effort should be stabilised. A focussed respiratory examination is prudent, starting with:

- Inspection
    - Assess respiratory effort, use of accessory muscles, tracheal recession, evidence of cyanosis or chest wall trauma.
- Palpation
    - Establish degree and effectiveness of ventilation and bilateral comparison. Confirm position of trachea and clarify any deviation away from the midline.
- Percussion
    - Useful for the assessment of pneumothorax (hyper-resonance suggestive of pneumothorax). However, this is difficult to perform in a busy and noisy environment.
- Auscultation
    - Clarify equal air entry and presence of added sounds (such as wheeze or crepitations).

A chest X-Ray and arterial blood gas assessment are simple investigations that can be performed at the patient's bedside and can give an idea about causes of respiratory compromise and physiological impact.

Intervention
Similar to airway management, oxygen should be administered as per the patient's requirement. If the respiratory effort is poor, a Bag Valve Mask can be utilised to aid the respiratory rate and depth. Oxygen saturations and respiratory rate should be monitored.

If there is suggestion of a pneumothorax, a needle decompression (mid axillary line, 4th intercostal space) or finger thoracostomy (mid axillary line, 4th intercostal space), should be performed.

Audible wheeze indicates bronchoconstriction and bronchodilators and steroids are likely to be of use. Diuretics can be considered in those in whom fluid overload is the cause of symptoms. In some cases, a combination of different therapies is likely to improve the condition.

Circulation

Assessment

Visual inspection of the patient may reveal pallor, cyanosis or distended neck veins. Peripheral and central pulses should be palpated ascertaining: rate, rhythm, character and volume. Central capillary refill time (sternal) should be measured (normal refill time is <2 seconds). If the pulses are palpable, heart rate and blood pressure should be measured.

If there is evidence of haemodynamic compromise secondary to arrhythmias or a patient appears "peri-arrest", then defibrillation pads should be applied.

Intervention

Achieving venous access is vital, and following doing so, bloods should be taken for:

- Full Blood Count
- Urea & Electrolytes
- Glucose
- Clotting
- C-Reactive Protein (CRP)
- Troponin (if cardiovascular cause suspected).

If the patient appears hypovolaemic (low blood pressure, tachycardia, and cutaneous signs of fluid depletion), a bolus of 250-500ml of intravenous fluid should be administered and the response assessed. Urine output gives an idea of fluid status and a fluid balance chart should be commenced measuring hourly fluid input and output.

Disability

Assessment

Confusion and wakefulness should be assessed using:

- AVPU – Assessment of patient responsiveness and scored depending on whether they are or respond to; Alert, Voice, Pain, Unresponsive
- Glasgow Coma Scale (GCS): Assessment of patient's best Eye, Voice response and Motor activity. A maximum score of 15 is achieved, whereby the lower score is 3/15 (if only 1 point is scored for each domain)

Table 1.1. Glasgow coma scale scores

Feature	Response	Score
Eye opening	Spontaneous To speech To pain None	4 3 2 1
Verbal response	Orientated Confused Inappropriate words Incomprehensible sounds None	5 4 3 2 1
Motor response	Obeys commands Localises pain	6 5 4

	Flexion-normal Flexion-abnormal Extension None	3 2 1

A glucose reading should be taken, as hypoglycaemia can be easily missed cause of a reduced GCS.

Intervention
The cause for a low GCS should be ascertained, looking at potential causes such as:

- Hypoxia, hypercarbia
- Inadequate perfusion (shock)
- Altered metabolic states (such as hypoglycaemia or htperglycaemia)
- Intoxication (drug overdose/poisoning)
- Medical Conditions (stroke/epilepsy)
- Head Injury/Trauma

Therefore each cause should be ruled out following clinical assessment, examination and appropriate investigations. A multi-specialty approach is likely to be required to stabilise and improve the condition.

If the patient is hypoglycaemic (Blood sugar <4mmol/L), then replacement depends on:

- Awake and no IV access – Oral glucose containing agents can be given via the mucosa (Hypostop/Glucogel/Dextrogel)
- IV access - IV infusion of approximately 25g glucose (50ml of 50% glucose, or 100ml 20% glucose)
- If no IV access and low GCS – 1mg IM glucagon (mobilisation of stored glycogen in the liver).

Exposure

Assessment of acute blood loss or injuries and cutaneous changes should be assessed.

The patient's temperature should be measured and warming instigated if the patient is significantly hypothermic.

Severity of Illness scoring

A number of scoring systems exist to assess the severity of physiological insult and to predict the outcome. One of the earlier used scoring systems was the Acute Physiology, Age and Chronic Health Evaluation (APACHE) which inspired other scores such as the Simplified Acute Physiology Score (SAPS). The advantages of quantifying critical illness include:

- Provision of a common language

- Provides an estimate of mortality
- Enables a method whereby practice and processes can be examined.
- Generate risk adjusted data for use in clinical trials

However, prior to admission to the intensive care unit, the degree of deterioration is assessed by the now adopted: National Early Warning Score 2 (NEWS 2)", developed by the Royal College of Physicians in 2017. Nursing and auxiliary staff can record observations such as level of consciousness, respiratory rate, heart rate, blood pressure, oxygen saturations and temperature. The degree of variation from normal values is used to calculate a total "NEW" score. The total score determines the urgency of assessment and indicates potential deterioration.

Table 1.2. Vital parameters recorded in the NEWS chart

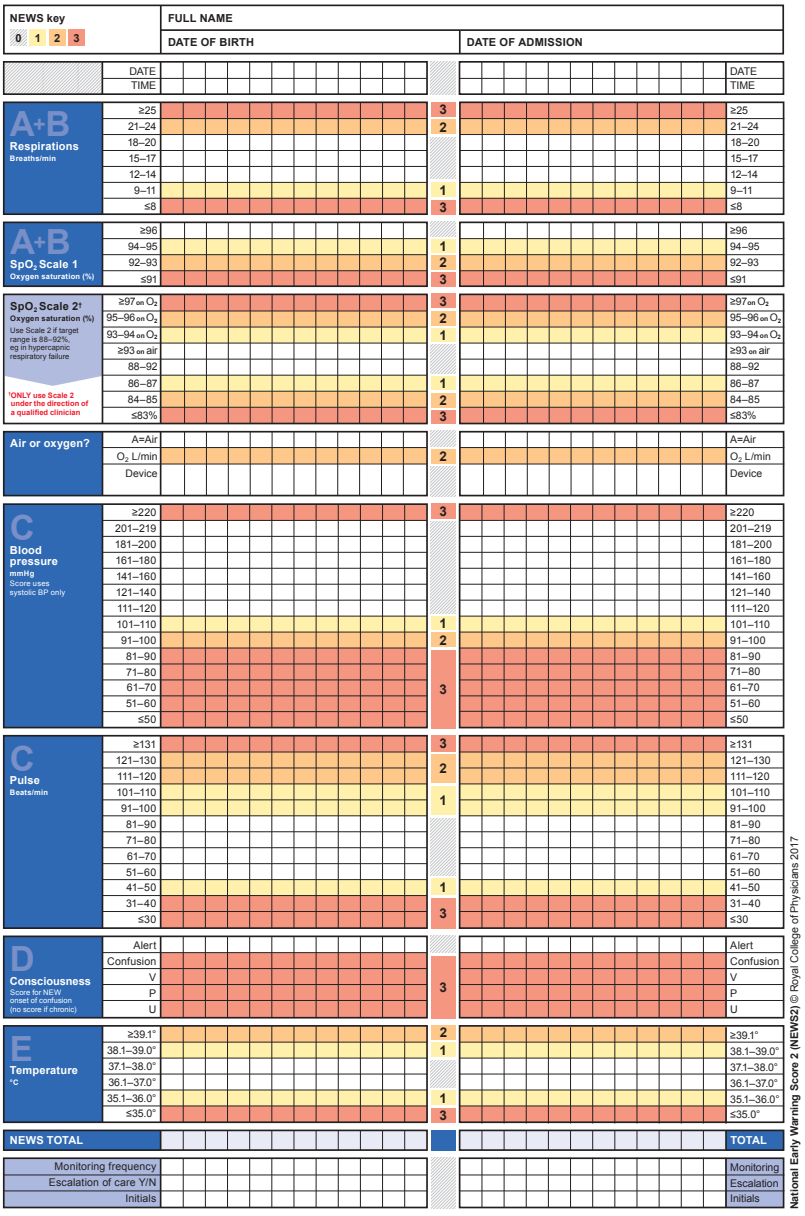

Table 1.3. Urgency of response based on the NEWS score

NEW score	Frequency of monitoring	Clinical response
0	Minimum 12 hourly	• Continue routine NEWS monitoring
Total 1–4	Minimum 4–6 hourly	• Inform registered nurse, who must assess the patient • Registered nurse decides whether increased frequency of monitoring and/or escalation of care is required
3 in single parameter	Minimum 1 hourly	• Registered nurse to inform medical team caring for the patient, who will review and decide whether escalation of care is necessary
Total 5 or more Urgent response threshold	Minimum 1 hourly	• Registered nurse to immediately inform the medical team caring for the patient • Registered nurse to request urgent assessment by a clinician or team with core competencies in the care of acutely ill patients • Provide clinical care in an environment with monitoring facilities
Total 7 or more Emergency response threshold	Continuous monitoring of vital signs	• Registered nurse to immediately inform the medical team caring for the patient – this should be at least at specialist registrar level • Emergency assessment by a team with critical care competencies, including practitioner(s) with advanced airway management skills • Consider transfer of care to a level 2 or 3 clinical care facility, ie higher-dependency unit or ICU • Clinical care in an environment with monitoring facilities

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MANAGEMENT OF SHOCK

2

Shock is defined as acute circulatory failure resulting in inadequate organ perfusion and cellular hypoxia. The ultimate result of shock due to any cause is cellular hypoxia and deranged cell function.

Cardiovascular physiology

The cardiovascular system is mediated by the autonomic nervous system. Sympathetic and parasympathetic outflow govern the response to different physiological states and its effects occur as a result of interaction with adrenoreceptors.

The Heart
Sympathetic control: leads to increased heart rate (chronotropic effect), cardiac output (inotropic effect), and speed of conduction through the AV node.
Parasympathetic control: via the vagus nerve (cranial nerve X)
Circulation
Stimulation of alpha and beta adrenoreceptors leads to an increase in tone and rise in systemic vascular resistance.

Basic cardiovascular principles help us understand the significance of shock, and what we can do as clinicians to overcome derangements to optimise physiology.

Cardiac Output

Amount of blood ejected from the left ventricle in one minute

Stroke Volume

Amount of blood ejected by the left ventricle in each contraction

Stroke volume in turn is made up of three separate factors:

Preload
This is the measure of ventricular filling during the relaxation phase of the cardiac cycle (diastole). The greater the filling, the greater the myocardium is stretched which leads to a greater output of blood from the ventricles when the heart contracts. However, there is a limit to the degree of stretch; the heart cannot keep increasing cardiac output by stretching indefinitely.

This denotes the volume of venous return to the heart which is determined by the venous capacitance, volume status and the pressure difference between mean systemic

venous pressure and right atrial pressure. Right atrial pressure is directly affected by intra-thoracic pressure. The volume of venous blood returned to the heart determines the stretch of myocardial muscle fibre.

Myocardial contractility
The Frank-Starling law states that the force of contraction of the cardiac muscle is proportional to its initial length (which depends upon the preload – the filling of the heart in diastole). Within physiological limits, an increase in end diastolic volume produces a more forceful contraction and an increase in stroke volume (SV).

Afterload
This is the resistance against which the heart pumps against, which is referred to as the Systemic Vascular Resistance (SVR). An increase in afterload, leads to a reduction in stroke volume and therefore cardiac output.

Ultimately, if there is a reduction in venous return to the heart as there would be in shock, there is impaired ventricular filling, reduced stretch and cardiac contraction – leading to a fall in cardiac output.

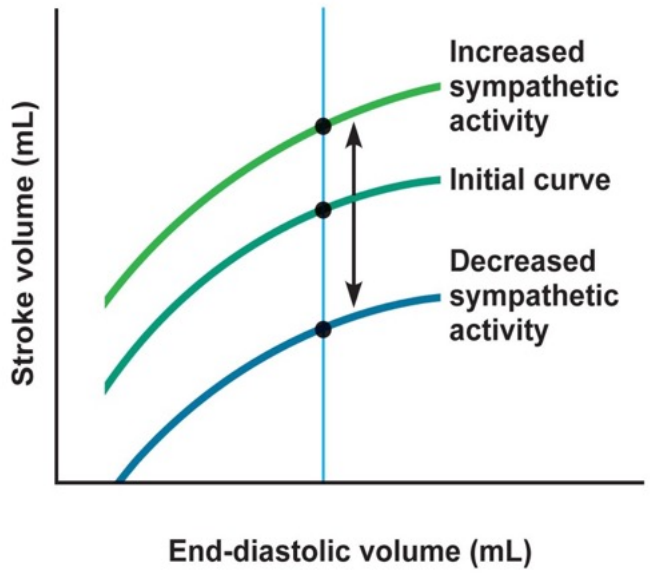


Figure 2.1: Relationship between stroke volume and end-diastolic volume

The above concepts help us to develop and understand a further equation:

Cardiac output is dependent on blood pressure and inversely related to blood pressure. We need to ensure an adequate perfusion pressure and flow to tissues, which enables Oxygen delivery.

Shock can be classified into different types depending on the cause:

- Hypovolaemic shock
- Cardiogenic shock
- Obstructive shock
- Distributive Shock
    - Anaphylactic shock
    - Neurogenic shock
    - Septic shock

Hypovolaemic shock

This is where there is an inadequate circulating volume (Normal circulating volume in adults is approx. 70-80ml/kg).

Causes

A. Fluid Loss
    - Endogenous – fluid lost into internal body cavities (intestinal obstruction or peritonitis)
    - Exogenous – burns, diarrhoea, vomiting or diuresis
B. Blood Loss
    - External
    - Internal – retroperitoneal bleeding, pelvic cavity, intrathoracic cavity
C. Iatrogenic – inappropriate diuretics, prolonged fasting, bowel preparation.

In hypovolaemic shock, circulating blood volume is reduced and venous return to the right atrium falls and therefore stroke volume and cardiac output are reduced.

The overall physiological response to hypovolaemia, detected by the carotid baroreceptors, which leads to increased sympathetic activity and a rise in the SVR, as blood is redirected towards central vessels. As this mechanism is exhausted, the heart tries to maintain cardiac output by increasing heart rate in the face of a falling stroke volume.

Diagnosis of Shock

Depending on the degree of hypovolaemia, signs of failing organs due to inadequate perfusion can be seen.

Respiratory Rate – Tissue hypoxia results in a metabolic acidosis, which in turn stimulates hyperventilation, leading to a compensatory respiratory alkalosis.

Skin Colour and Perfusion and Temperature – Pallor and mottling indicates reduced perfusion and significant vasoconstriction, which in turn leads to a capillary refill time greater than 2 seconds.

Urine Output – a fall in urine output is a late sign and indicates poor renal perfusion and oxygen delivery. NB health urine output is >0.5ml/kg/hr

Consciousness – Can be impaired with falling cerebral perfusion

Table 2.1. Classes of haemorrhagic shock

	Class I	Class II	Class III	Class IV
Blood volume lost	<15%	15–30%	30–40%	>40%
Heart rate (bpm)	<100	100–120	120–140	>140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate	14–20	20–30	30–40	>35
Urine output (mL/hr)	>30	20–30	5–15	Negligible
Mental status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic

Investigations

Blood Lactate – decreased oxygen delivery leads to the production of lactate due to anaerobic respiration.

Central Venous Oxygen Saturation – This can be used if the patient has a central line inserted. When there is a greater degree of shock, there is greater oxygen extraction taken up by the respiring tissues. Therefore, blood returning back to the heart has a lower saturation then one would expect in a healthy, well hydrated individual.

Management of hypovolaemic shock

Resuscitation (Reverse the deteriorating physiology)

- ABCDE assessment and support. Call for help
- Provision of 100% Oxygen to aid Oxygen delivery.
- Fluid therapy is likely to make the most difference and is often the critical step in terms of managing hypovolaemia.
- Consider the type of fluid replacement. Largely speaking crystalloids are sufficient; however, avoid excessive administration if haemorrhage is considered as this can further dilute the haematocrit and effective blood volume.
- A fluid bolus is likely to indicate fluid responsiveness and 250-500ml can be given and changes in the above parameters can be identified.
- Approximately 20ml/kg of fluid may need to be given to a patient over the course of their resuscitation.

Treat the cause (prevent deterioration)

- Inform the surgical team if an intra-abdominal cause is suspected
- Treat sepsis (below)
- Damage Control Resuscitation and Surgery and resuscitation with blood products

Damage control resuscitation

- Aims to treat the lethal triad – coagulopathy, acidosis and hypothermia
- Considers a balanced approach to transfusion of blood products including blood, FFP and platelets. Use of emergency O- blood.
- Assessment of other electrolytes such as Potassium (often rises following blood transfusion), and Calcium (often falls following transfusion)
- Aim to normalise Fibrinogen using Cryoprecipitate
- Use of Tranexamic Acid (anti-fibrinolytic – stabilizes a pre-existing formed clot)

Blood transfusion

The decision to transfuse blood depends on the patient's response and estimated blood loss. The main aim of blood transfusion is to restore haemoglobin and oxygen carrying capacity. The type of blood used depends on the urgency of transfusion.

In patients with exsanguinating haemorrhage, group O negative blood should be used. The blood bank can issue group specific blood in 10 to 20 minutes. Fully cross-matched blood is preferred but it can take 45 to 60 minutes to obtain. For stable patients requiring transfusion, cross-matched blood should always be used.

Cardiogenic shock

In cardiogenic shock the cardiac output falls due to pathology in the heart itself and is defined as a cardiac index of less than 1.8 L/ minute/m^2. (Cardiac index is cardiac output per meter of body surface area).

Causes of cardiogenic shock include

- Myocardial infarction, myocarditis
- Arrhythmias
- Cardiac tamponade
- Tension pneumothorax
- Acute aortic incompetence
- Left ventricular aneurysm

Management includes supportive measures, oxygen therapy, cautious use of fluids with careful monitoring of central venous pressure or pulmonary wedge pressure. Other monitoring should include continuous ECG, 12 lead ECG, urine output, urea and electrolytes and blood gases. Patient should be preferably managed in the coronary care unit. Inotropic support, vasodilators and mechanical circulatory support may be needed. Further management depends on the specific cause of cardiogenic shock.

Distributive Shock

In this type of shock, there is peripheral vasodilation and impaired distribution of blood flow.

Septic Shock

In septic shock, the presence of severe infection triggers a massive inflammatory response with systemic activation of leucocytes and release of a variety of potentially damaging mediators.

Common sources of sepsis include abdomen, chest, wounds, urinary tract and intravascular lines. These mediators result in profound vasodilatation, increased capillary permeability and myocardial depression. Several mediators such as nitric oxide, bradykinin, histamine, prostaglandins and cytokines (interleukin-1, tumour necrosis factor and interleukin-6) are involved in the initiation of sepsis.

- *Haemodynamic* changes include severe vasodilatation, myocardial depression and intravascular pooling of blood.
- *Microvascular* changes include increased capillary permeability, microembolisation and arteriovenous shunting. There may be primary disturbance of cellular metabolism and cells are unable to use oxygen and as a result oxygen extraction is impaired. The patient will be hypotensive with warm peripheries.

Management

Management of septic shock includes rapid resuscitation to restore oxygenation, appropriate supportive measures, diagnosis and eradication of source of sepsis and judicious use of antibiotics. Aggressive fluid resuscitation and inotropic support is required in the early stages to optimise oxygen delivery. The initial resuscitative treatment strategy is referred to as “Sepsis 6”.

Sepsis six

Components of sepsis six include

1. Administer oxygen (to keep oxygen saturation >94%, 88-92% if there is a risk of CO_2 retention, e.g. COPD)
2. Take blood cultures
3. Give IV antibiotics
4. Give IV Fluids
5. Check serial lactate. If lactate >4 mmol/L, re-check after each 10ml/kg of fluid challenge
6. Measure urine output

Anaphylactic shock

This is due to an anaphylactic reaction, which is mediated by immunoglobulin E (IgE) antibodies. Activation of mast cells causes release of histamine and serotonin.
Clinical features of anaphylactic shock:
Cutaneous: Flushing, erythema, urticarial rashes and swelling
Respiratory: Bronchospasm, oedema of the glottis and tongue result in airway obstruction and stridor.
GIT: Abdominal pain, diarrhoea, nausea or vomiting.
CVS: Hypotension, tachycardia and cardiovascular collapse.

Neurogenic Shock

This occurs following significant damage to the spinal cord which leads to a disruption and loss of sympathetic tone. Depending on the site of injury, cardioaccelerator fibres can be affected which alters blood pressure and heart rate. If the injury is above T4, then the patient may show signs of bradycardia and hypotension and the presence of warm peripheries (due to venous pooling and systemic vasodilation).

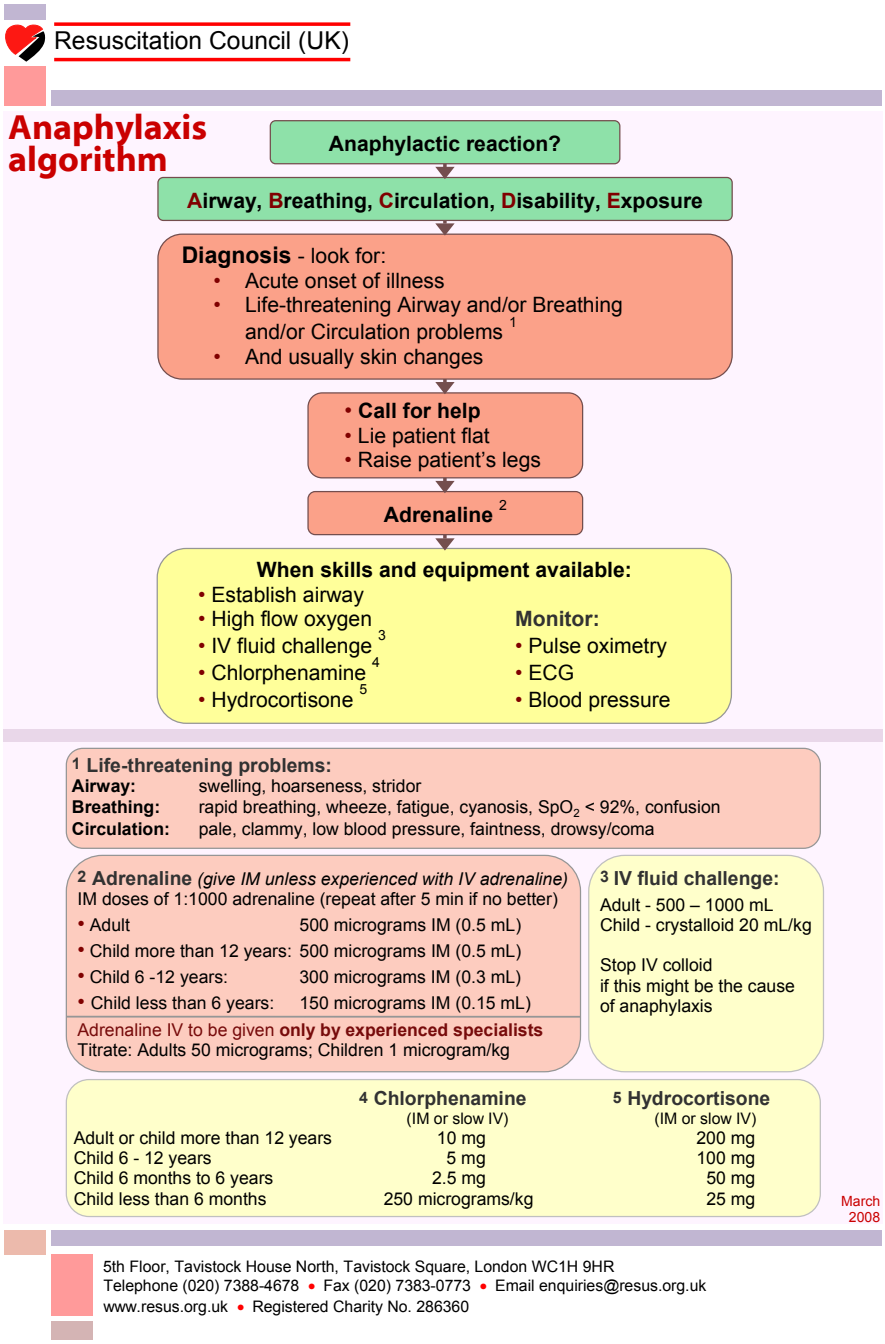
The management of hypotension differs to that from other causes of shock, as the patient is not fluid deplete (low intravascular volume). Therefore, the mainstay of management is through the use of vasopressors and inotropic drugs.

Inotropic drugs

Inotropes are drugs that increase the cardiac output by increasing the contractility or heart rate via their action on adrenoreceptors:
α - peripheral vasoconstriction (increased SVR)
β1- increased heart rate and contractility
β2 – vasodilation and bronchodilation

Table 2.2. Effect of inotropic drugs on alpha and beta effects

	α	β1	β2
Adrenaline	++	+++	++
Noradrenaline	+++	+	
Dopamine	+	++	+
Dobutamine		++	+



Ref: https://www.resus.org.uk/anaphylaxis/emergency-treatment-of-anaphylactic-reactions/ (accessed on 4.11.2019)

Obstructive Shock

This is as a result of extra cardiac obstruction to blood flow. The heart is required to pump against an increased afterload. Examples include; pulmonary embolism, aortic stenosis and tension pneumothorax.

Consequently, the patient becomes tachycardia and has an elevated SVR initially. There is venous congestion which can be seen with a raised JVP

Special considerations

Age

Elderly patients' exhibit reduced sympathetic activity; hence they cannot compensate well for hypovolaemia. Due to atherosclerosis many vital organs are very sensitive to reduced blood flow. They also have reduced respiratory reserve which limits the ability to meet the increased oxygen demand. Reduced renal function reduces the ability to preserve volume. They may also be on drugs (such as B-blockers) which mask the signs of shock.

Pregnancy

This is a hypervolaemic state; hence the clinical signs may not be manifested till a large volume of blood has been lost. Hypovolaemic shock can result in decreased fetal perfusion.

Drug history

Beta blockers and calcium channel blockers can obtund the compensatory responses to hypovolaemia. Any drug that has significant effect on myocardial contractility, heart rate and peripheral vascular tone can alter the response to shock.

Pacemaker

Patients with a pacemaker have a fixed heart rate and are unable to respond to hypovolaemia.

Further reading

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RESPIRATORY FAILURE

3

Respiratory failure occurs when the respiratory system is no longer able to meet the metabolic demands of the body. The major function of the lungs is to get oxygen into the body and drive carbon dioxide out.

Getting oxygen in

This happens in two steps:

1. Bringing oxygen from the atmosphere to alveoli
2. Transfer of the oxygen across the alveoli to the capillary blood.

The alveolar gas equation explains the partial pressure of oxygen in the alveoli.

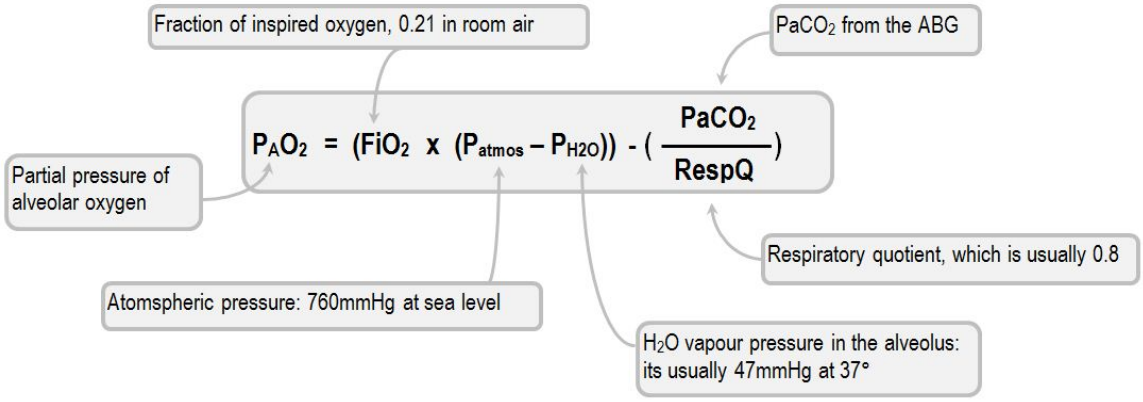


Figure 3.1. Components of the alveolar gas equation (reproduced with permission from https://derangedphysiology.com)

The above equation is important as it demonstrates the main factors impacting alveolar oxygen content: Fraction of inspired oxygen (FiO_2), atmospheric pressure and partial pressure of carbon dioxide.

Our main strategies to increase the oxygen tension in the alveoli would be to either increase the amount of oxygen supplied to the patient (FiO_2) or to increase the atmospheric pressure (typically performed in a pressure chamber).

It's important to note that conditions that increase blood carbon dioxide levels (and hence in the alveoli) are associated with a decreased alveolar oxygen tension.

The alveolar gas equation also explains why there is a "cascade" of oxygen between the atmosphere and the arterial blood supply. Atmospheric oxygen passing into the lungs is diluted through the addition of water vapour. As the oxygen reaches the alveoli from atmosphere, there is a drop in its tension. Because of a phenomenon called physiological shunting, there is a further drop in arterial oxygen tension when

compared to that in alveoli. In a healthy adult the arterial oxygen tension (PaO_2) is about 13 kPa.

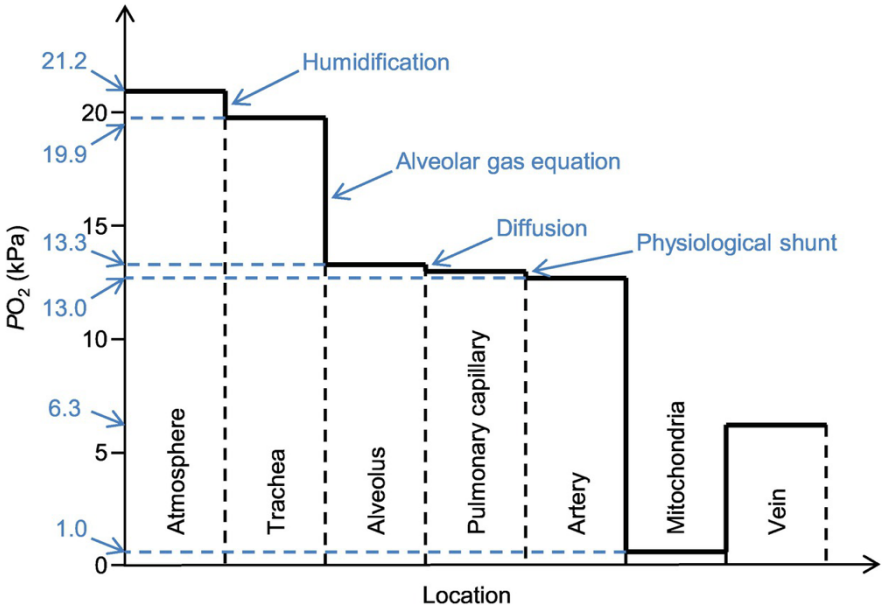


Figure 3.2. The factors affecting oxygen cascade

Driving carbon dioxide out

CO_2 elimination is largely dependent on alveolar ventilation. That is the usable gas exchange that occurs, taking into consideration the portion of gas flow during inspiration that remains within the lungs.

Alveolar ventilation = Respiratory Rate x (Tidal Volume - Dead Space)

Dead space is that portion of the tidal volume that does not take part in gas exchange. Therefore, any changes in $PaCO_2$ are dependent on:

- Respiratory rate
- Tidal volume
- Ventilation-perfusion (V/Q) matching.

CO_2 crosses the alveolar membrane very readily and so diffusion abnormalities and shunting have little effect on CO_2 elimination. Terminologies such as dead space, shunting, diffusion abnormalities are discussed in detail later.

Respiratory failure

In general, patients require respiratory assistance due to airway problems, failure to ventilate or failure to oxygenate. Often all three problems exist simultaneously. The act of respiration grossly includes two components.

A. Ventilation – a mechanical process whereby the ambient gas is taken into the alveoli.

B. Gas Exchange - takes place between alveoli and the capillary blood. Based on this, respiratory failure can be categorized into two groups depending on the cause, those due to ventilatory defects and those due to impaired gas exchange.

Conventionally respiratory failure is also classified as Type I and Type II based on the effects of the failure.

Type I is associated with hypoxaemia. The CO_2 level may be normal or even low.

Type II is associated with hypoxaemia and hypercapnia.

- Hypoxaemia: PaO_2 less than or equal to about 8 kPa when breathing room air
- Hypercapnia: $PaCO_2$ more than or equal to about 6.5 kPa

Pathophysiological mechanisms of respiratory failure
Any pathophysiological mechanism that leads to poor ventilation or impaired gas exchange can result in respiratory failure.

Ventilation defects

Hypoventilation is marked by a rise in $PaCO_2$ and a fall in PaO_2. The causes of hypoventilation can be enumerated in a systematic way starting from the central control in the brain above to the respiratory apparatus below.

- Brainstem
  - brainstem injury e.g. due to trauma, haemorrhage, hypoxia or infection
  - metabolic encephalopathy
  - depressant drugs
- Spinal cord
  - trauma, tumour, transverse myelitis
- Nerve root injury
- Nerve
  - trauma
  - neuropathy e.g. Guillain Barre
  - motor neuron disease
- Neuromuscular junction
  - myasthenia gravis
  - Eaton-Lambert syndrome
  - neuromuscular blockers
- Respiratory muscles
  - fatigue
  - disuse atrophy
  - myopathy
  - malnutrition
- Respiratory system
  - airway obstruction (upper or lower)

- decreased lung, pleural or chest wall compliance, e.g. pneumothorax and pleural effusion.

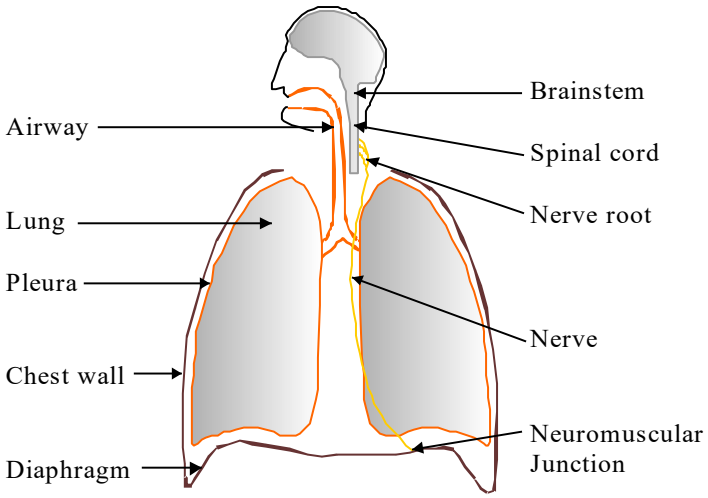


Figure. 3.3. Causes of hypoventilation

Impaired gas exchange

Gas exchange failure (often leading to hypoxemia) most often occurs at a microscopic level at the pulmonary capillary-alveolar interface. Classically injuries are divided up into:

1. Diffusion defects
2. Ventilation perfusion (V/Q) mismatch.

   V/Q mismatch can either be dead space or shunt or a mixture of both to varying extents.

   - Dead space ventilation - *alveoli are ventilated but not perfused (wasted ventilation)*
   - Shunt – *alveoli are perfused, but not ventilated (wasted perfusion)*

Often, in acute lung injury, a variety of abnormalities are present in the same lung.

Diffusion defects

This is caused by conditions such as thickening of the alveoli, as in pulmonary fibrosis or increased extracellular fluid, as in pulmonary oedema. This results in impaired gas exchange. As the passage of oxygen from alveolus to capillaries is more difficult, hypoxemia ensues. CO_2 is more freely diffusible than O_2. Therefore, hypercapnia occurs only in advanced stages.

Ventilation/perfusion mismatch

Dead Space Ventilation: Alveoli that are *ventilated but not perfused* results in wasted ventilation. An extreme example of this is a pulmonary embolus. More frequent clinical situations are hemorrhage or hypotension where perfusion pressure to apical lung units may fall, leading to alveolar dead space.

Shunt: Alveoli are *perfused but not ventilated*; well oxygenated blood becomes mixed with deoxygenated blood. This occurs in airway collapse, pneumonia, pulmonary contusion, ARDS/ALI (acute respiratory distress syndrome / acute lung injury). The intracardiac causes for shunting include right to left shunt e.g. Fallot's tetralogy, Eisenmenger's syndrome. This form of respiratory failure is relatively resistant to oxygen therapy.

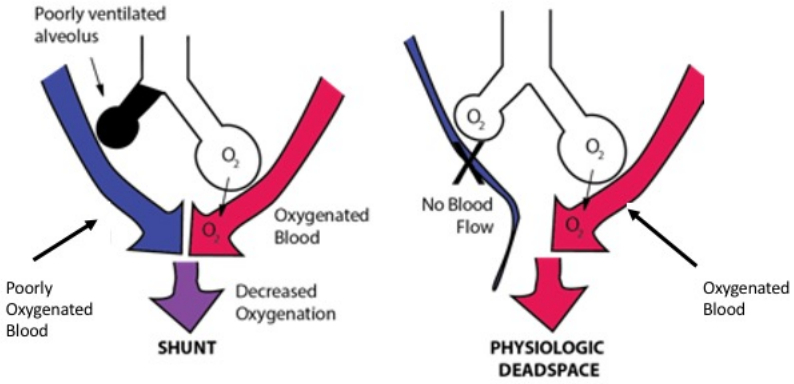


Figure 3.4 Ventilation Perfusion mismatch (reproduced with permission from Dr Dr Christine Whitten, www. airwayjedi.com)

Clinical signs

The features of respiratory failure are those due to increased work of breathing, due to hypoxia and hypercarbia and due to end-organ hypoxia. Clinically patients can be cyanotic due to an accumulation of deoxygenated haemoglobin (> 5 g/dl).
Signs of respiratory compensation

- tachypnoea is an early and sensitive indicator
- use of accessory muscles
- nasal flaring
- intercostal, suprasternal or supraclavicular recession

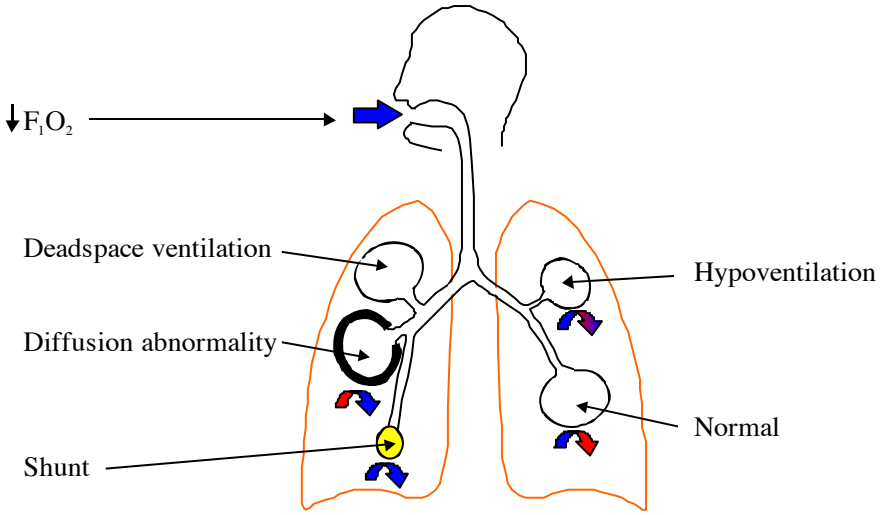


Figure 3.5. Physiological mechanisms of respiratory failure

Effects of hypoxia and hypercarbia on the autonomic nervous system

There is initial stimulation of the sympathetic nervous system causing tachycardia, hypertension and sweating. Later, bradycardia and hypotension ensues.

End-organ hypoxia

Virtually all tissues in the body are dependent on oxygen. Depending on the severity and duration of hypoxia findings may include altered mental status, loss of consciousness, convulsions, cardiovascular depression, cardiac arrest, multi organ failure.

Monitoring and investigations

Pulse oximetry

The relationship between oxygen saturation and PO_2 is described by the oxygen dissociation curve (ODC). The pulse oximeter is a very useful non-invasive means to monitor the oxygenation status of haemoglobin. The curve tends to highlight that a fall in saturation below 90%, represents a drastic fall in PaO_2.

Table 3.1. Typical arterial and venous oxygen saturations

Blood sample	Saturation %	PaO_2 (kPa)
Arterial	100	13.3
Venous	75	5.3

A saturation of 100% represents a PaO_2 more than 13 KPa and a saturation of 75% represents a PaO2 of 5.3 kPa (typical venous saturation).

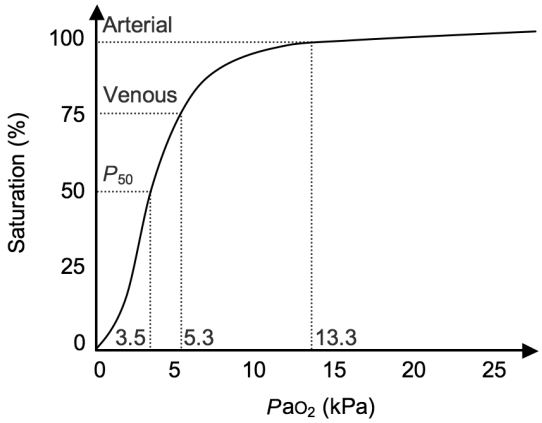


Figure 3.6. Oxygen dissociation curve

Arterial blood gas analysis

Blood gas analysis gives rapid assessment of PaO_2, $PaCO_2$, pH, bicarbonate levels and other values like hemoglobin saturation etc. It is useful in identifying the severity of respiratory impairment and subsequently to monitor the response to treatment. Imaging investigations like Chest X-Ray and CT are relatively straightforward to perform and give further assessment of underlying conditions or causes of decline. Further systems and laboratory investigations can be undertaken accordingly.

For details on blood gas interpretation, watch the video on the following link and refer to the chapter on acid-base disorders in handbook of peri-operative medicine
https://www.youtube.com/watch?v=KudrLakBgeU

Management of respiratory failure

Prompt recognition of signs of acute respiratory failure, early involvement of the critical care team and initiation of treatment is important in the management of respiratory failure. An attempt should be made to find and treat the underlying cause. Principles of management include optimising:

1. *Gas exchange* - increasing the inspired oxygen concentration and providing ventilatory support
2. *Tissue oxygenation* - increasing tissue blood flow by means of adequate cardiac output and an adequate haemoglobin concentration.

- The initial management of any type of respiratory failure is – ABC: Airway, Breathing, Circulation and the provision of 100% Oxygen until the cause is understood.
- Hydration with intravenous fluids can be considered to optimise cardiac output.
- Chest physiotherapy: Percussion and vibration along with breathing exercises and adequate coughing helps to clear secretions.
- Inotropic support may be required to optimise tissue blood flow.
- Bronchospasm should be treated using bronchodilators
- Antibiotics may be necessary to treat existing respiratory infection.

- Ventilatory support: Non-invasive positive pressure ventilation or continuous positive pressure ventilation improves the oxygenation. Tracheal intubation and invasive ventilation may be required to treat hypercapnia associated ventilatory failure.

Oxygen therapy

In acutely ill patients, oxygen delivery relies on maintaining a patent airway. Give oxygen empirically in all sick patients or when there is respiratory distress or hypotension.

Hypoxia can arise from the methods above, however they can broadly be classed into 4 types:

- Hypoxic Hypoxia – When the delivery of oxygen into the patient is impaired.
- Anaemic Hypoxia – Whereby the oxygen carrying capacity is impaired (i.e low Haemoglobin)
- Stagnant Hypoxia – Whereby the cardiac output is poor and delivery of oxygen to tissues is impaired.
- Histotoxic Hypoxia – Impaired mitochondrial function, seen in sepsis.

Therefore, correction of other factors such as anaemia, cardiac output and treatment of sepsis help to improve oxygen delivery and utilisation.

Provision of oxygen helps to saturate Haemoglobin and improve dissolved oxygen concentration. However, oxygen solubility is low and dissolved oxygen accounts for only one third of the resting tissue oxygen requirements.

When oxygen is used for long periods it should be humidified by passing through a humidifier or a simple water bath. Humidified oxygen helps to clear secretions and minimises respiratory losses of heat and moisture.

Oxygen therapy devices

Fixed performance devices: These devices are patient independent as the patient receives a constant predetermined FiO_2 regardless of changes in respiratory parameters. e.g.: face mask with reservoir bag,

- Non-rebreathe reservoir mask
  A facemask with a reservoir bag delivers a FIO_2 of 0.85 at flow rates of 10-15 L/min. Unlike a simple face mask which can only provide a set oxygen flow rate, the one with a reservoir bag has an additional reservoir for oxygen, however, care should be taken to ensure that the bag is well inflated and not obstructed.

- Venturi Mask.
  They are 'air entrainment masks' that function based on the Venturi principle. The colour coded adapter fits on the end of the mask and entrains a certain portion of air at a given flow rate. This leads to a dilution of supplied oxygen and is able to generate a fixed FiO_2.

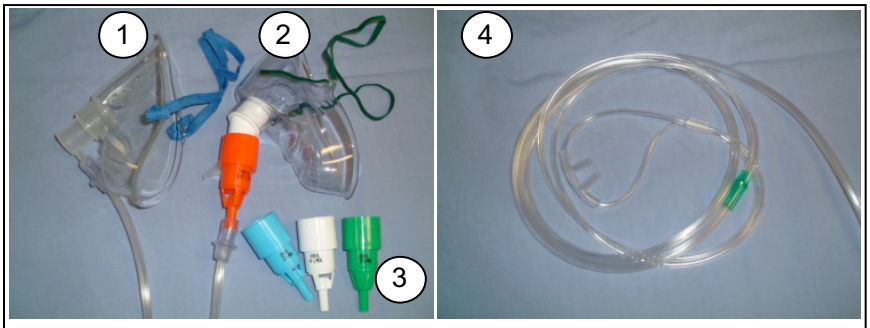
Table 3.2. FiO_2 delivered by venturi mask and required oxygen flow

FiO_2	Colour	Required Oxygen Flow Rate L/min
0.24	Blue	2
0.28	White	4
0.31	Brown	6
0.35	Yellow	8
0.40	Red	10
0.60	Green	15

Variable performance devices: The performance varies with patients' respiratory efforts. Variable performance devices use the dead-space of the nasopharynx or face masks as a reservoir of oxygen. They cannot deliver high inspired concentrations of oxygen. There are two main categories, nasal cannula and facemasks.

- Nasal cannulae
  This uses the dead space of the nasopharynx as a reservoir for oxygen. When the patient inspires, entrained air mixes with the reservoir air and the inspired gas is enriched. The FiO_2 depends on the magnitude of flow of oxygen, the patient's minute ventilation and peak flow. Typically, each additional 1l/minute of O_2 flow with nasal cannula represents an increase in the FiO_2 by 4%. So 1 litre is 24%, 2 litres is 28% etc. At 6 litres (44%), it is not possible to raise the FIO_2 further, due to turbulence, in the tubing and in the airway. Often at higher flow rates, patients experience discomfort and dryness of nasal mucosa.

- Face Masks
  e.g Hudson face mask - Typically delivers FIO_2 of 0.35 at 4 L/min of oxygen flow rate and with a normal respiratory pattern. Ambient air is entrained through the holes on both sides of the mask. Holes also allow exhaled gases to be vented out.



1. Hudson mask
2. Venturi mask
3. Venturi mask adaptors
4. Nasal cannulae

Figure 3.7. Oxygen delivery devices

Non-invasive ventilation

Non-invasive ventilation involves delivering pressure support during phases of the respiratory cycle.

CPAP (Continuous positive airway pressure)
Pressure is delivered throughout the respiratory cycle. There are multiple reasons why this might improve breathing.

1. Counteracts intrinsic positive end expiratory pressure (PEEP)
2. Decreases preload and afterload in congestive cardiac failure (CCF)
3. Improves lung compliance in CCF
4. Decreases the work of breathing

CPAP aims to recruit distal air spaces and alveoli in an effort to increase more surface area for ventilation and gas exchange. It is able to do this by generating pressure throughout the respiratory cycle to keep the alveoli patent. The physiology is similar to blowing up a balloon – it's hardest to inflate when completely empty, but inflating further once there is some air within the balloon is much easier and requires less effort.

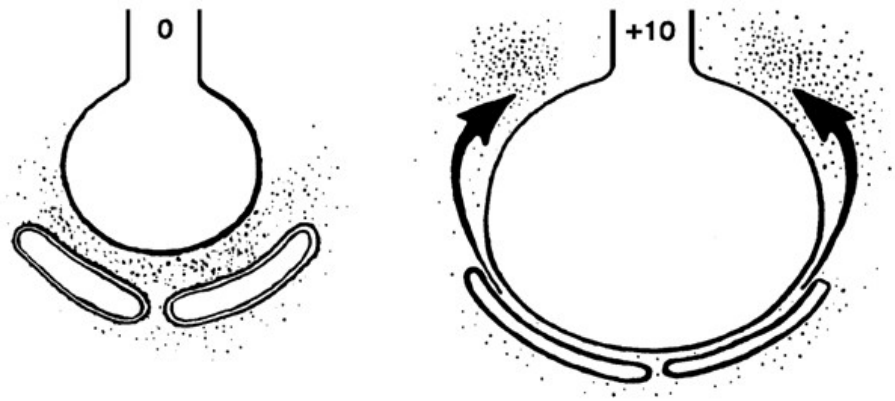


Figure 3.8. Illustration to demonstrate the effect of CPAP on alveoli.

For more details on CPAP click this link
https://emedicine.medscape.com/article/304235-overview

BiPAP (Bilevel positive airway pressure) – Often referred to as NIV

BiPAP delivers CPAP but also senses when inspiratory effort is being made and delivers a higher pressure during inspiration. When flow stops, the pressure returns to the CPAP level. This positive pressure wave during inspiration unloads the diaphragm, decreasing the work of breathing. This technique of ventilation is particularly useful for

patients with COPD who develop Type II respiratory failure. BiPAP helps to increase the patient's minute ventilation and to reduce $PaCO_2$.

Patients presenting with respiratory failure, with normal levels of consciousness, no major secretion problems and who are hemodynamically stable, a trial of BiPAP or CPAP should be attempted prior to considering intubation and a mechanical ventilator.

For basic understanding of non-invasive ventilation click this link
https://www.youtube.com/watch?v=Jms3qM8069Y

High flow nasal oxygen therapy

This is a newer method of oxygen delivery that is being utilised on critical care patients, and those with or at risk of respiratory impairment. High flow nasal oxygen therapy is able to supply warmed, humidified oxygen to patients at flow rates up to 75L/min. The system can control inspired oxygen concentration by mixing air and oxygen. This is far better tolerated by patients as it avoids the need for tight fitting masks and mucosal discomfort. Cold dry gases can exacerbate heat loss and is associated with poor compliance. For each 10L/ min of oxygen flow, it provides CPAP of 1cm H_2O. It has a number of proposed uses:

- Acute hypoxaemeic respiratory failure (able to deliver an FiO_2 close to 1.0)
- Airway management / tracheal intubation in critical care
- Apnoeic oxygenation
- Extubation and postoperative care (avoidance of atelectasis and pneumonia)
- Adjunct in treatment of CCF

Indication for tracheal intubation

The main indication for intubation is airway protection / control of the airway: Such circumstances include:

1. Loss of gag/cough reflex e.g. head injury with GCS <8 (to prevent aspiration).
2. Airway obstruction: acute laryngeal oedema – e.g. inhalation burn, Ludwig's angina, epiglottitis.
3. Anticipated loss of control of the airway: anticipated laryngeal oedema– e.g. neck trauma, acute stridor etc.

Patients are usually intubated for controlled mechanical ventilation and a tracheal tube or tracheostomy provides a good seal for controlled ventilation (definitive airway). The inspired volumes and pressures are consistent when compared with non-invasive methods. Finally, the presence of an artificial airway facilitates removal of obstructive material from the airway (airway toileting – suctioning of secretions).

Indications for mechanical ventilation can either be ventilation failure or oxygenation failure, as discussed under the topic of causes for respiratory failure. At times, even when ventilatory efforts are good and gas exchange is intact, there can still be impaired oxygen extraction at tissue level such as in low cardiac output states, sepsis, cyanide toxicity etc. warranting assisted ventilation. Modes of ventilation and

descriptions about the various ventilatory parameters are beyond the scope of this section.

For further reading

Deranged physiology In: Chambers, D., Huang, C., & Matthews, G. (2019). Oxygen Cascade. In *Basic Physiology for Anaesthetists* (pp. 80-81). Cambridge: Cambridge University Press. doi:10.1017/9781108565011.022

https://www.cambridge.org/core/books/basic-physiology-for-anaesthetists/oxygen-cascade/1D6A781C44C83938FF8DA50D4A31E77D

Singer M and Webb A. Oxford handbook of critical care, 3rd Edn, 2009. Oxford University Press

Acute kidney injury (previously described as acute renal failure) is characterised by the sudden deterioration of kidney function over a period of hours to days. This equates to a decrease in glomerular filtration rate (GFR), with subsequent accumulation of waste products, acid-base disturbance, fluid and electrolyte disturbance. This is typically detected clinically through a rise in creatinine and a fall in urine output. Although the kidneys have multiple functions (e.g. metabolism, endocrine), as well as fluid and electrolyte balance, the GFR is generally accepted as the index of renal function. In chronic renal failure there is irreversible loss of nephrons resulting in permanent impairment of solute excretion.

Normal GFR is 120ml/min and symptoms of failure occur as the GFR falls below 30ml/min. An inability to clear fluid will result in intravascular volume overload and its subsequent signs and symptoms. As the GFR is difficult to measure, estimates of renal function are based on blood urea and creatinine levels in conjunction with urine output. Normal values for urea and creatinine will vary amongst laboratories, but an upper value of 105-120 µmol/L is usually applied. As GFR falls, serum creatinine rises, but only starts to do so when about 50% of glomerular filtration has been lost.

AKI is therefore heralded by a rapid rise in urea and creatinine along with (but not necessarily) oliguria or anuria.

- Normal urine output in adults is usually 1ml/kg/h.
- Oliguria is defined as the production of urine less than 400-500 ml/day (17-20 ml/h)
- Anuria means absence of urine, in practice the term is applied to urine production less than 100ml/day in an adult.

Stages of acute kidney injury

The degree of renal impairment has been defined using the RIFLE criteria (**R**isk of acute renal failure, renal **I**njury, renal **F**ailure, **L**oss of renal function, **E**nd stage renal disease). However, the Kidney Disease Improving Global Outcomes (KDIGO) and Acute Kidney Injury Network (AKIN) criteria uses the baseline creatinine levels to classify AKI.

Table 4.1. Stages of acute kidney injury

Stage	Serum Creatinine	Urine Output
1	1.5-1.9 x baseline or ≥ 26.5 µmol/L increase within 48 hours	≤ 0.5ml/kg/hr for 6-12 hours
2	2.0-2.9 x baseline	≤ 0.5ml/kg/hr for ≥12 hours
3	3.0 x baseline or Increase in creatinine to ≥353.6 µmol/L or Initiation of renal replacement therapy Or In patients <18 years, decrease in eGFR< 35ml/min/1.73 M^2	≤0.3ml/kg/hr for ≥24 hours or Anuria for ≥12 hours

Causes of acute kidney injury

To function properly, the kidney requires: (1) normal blood flow; (2) functioning glomeruli and tubules to separate and process an ultra-filtrate containing waste products from the blood; and (3) drainage and elimination of formed urine from the body. The sudden interruption of any of these processes will lead to ARF. Disorders are therefore classified on the basis of their primary site of interference with these processes.

Prerenal: Conditions which interfere with blood delivery to the kidney and are most commonly functional (and potentially reversible) in nature.

Renal (intrinsic): Diseases which cause actual injury to the kidney (glomeruli, tubules, interstitium, small blood vessels) are grouped under intrinsic causes.

Postrenal (obstructive): Conditions which interfere with normal drainage and elimination of formed urine.

Table 4.2. Causes of acute kidney injury

Prerenal	
True intravascular depletion of volume	Haemorrhage, over diuresis, poor fluid intake, vomiting, diarrhoea
Decreased effective circulating volume	Sepsis, congestive heart failure, cirrhosis or hepatorenal syndrome, nephrotic syndrome
Impaired renal blood flow because of exogenous agents	Angiotensin-converting enzyme inhibitors, nonsteroidal anti-inflammatory drugs
Intrinsic	
Acute tubular necrosis	Ischemia Nephrotoxic drugs (aminoglycosides), radiocontrast agents, pigments (myoglobin or haemoglobin)
Glomerular disease	Acute glomerulonephritis
Microvascular disease	Atheroembolic disease (cholesterol-plaque), thrombotic thrombocytopenic purpura, haemolytic uraemic syndrome,
Macrovascular disease:	HELLP syndrome or postpartum acute renal failure.
Interstitial disease	Renal artery occlusion, severe abdominal aortic disease (aneurysm) Allergic reaction to drugs, autoimmune disease: (systemic lupus erythematosus or mixed connective tissue disease),

	pyelonephritis, infiltrative disease (lymphoma or leukaemia)
Postrenal	
Obstruction	Benign prostatic hypertrophy or prostate cancer, cervical cancer, retroperitoneal disorders, invasive pelvic malignancy, intraluminal bladder mass (clot, tumour or infective occlusion), neurogenic bladder, urethral strictures

HELLP = haemolysis, elevated liver enzymes, and low platelets.

Prerenal acute kidney injury

Since the main determinant of GFR is renal blood flow (RBF), any decrease in renal blood flow will result in a decrease in filtration. The kidney intrinsically reacts to changes in perfusion pressure over a mean arterial range of 80 – 180 mm Hg, to maintain constant flow (autoregulation). Therefore, within this range the blood flow to the kidney is independent of the actual blood pressure. However, below this pressure the blood flow tends to be pressure dependent. In hypertensive patients this may be higher, therefore emphasising the importance of "normal" blood pressure, i.e. normal for the patient. In prerenal failure it is the perfusion of the kidneys which is abnormal. The glomeruli and tubular systems function normally, though continued insult will lead to damage. However, most often, poor perfusion is due to a reduction in effective circulating volume. Such conditions would include classical hypovolaemia secondary to haemorrhage, as well as relative hypovolaemic states such as sepsis or cardiac failure.

Intrinsic acute kidney injury

This implies that the actual function of the kidneys is abnormal, either at the vascular (vasculitis, renal artery or vein occlusion), glomerular or tubular level (glomerulonephritis, interstitial nephritis). This may be due to infection or immune mediated disease but more commonly is a consequence of anoxia or toxins (90%).

Persistent ischaemic or toxic insults, eventually lead to death of tubular cells, referred to as acute tubular necrosis (ATN). It is difficult to distinguish it from other causes but it can be suspected following a lack of response to large volume intravenous fluid infusion.

Fortunately, ATN is a reversible condition, provided the initial insult is removed, as tubular cells have the ability to regenerate. ATN secondary to ischaemia tends to be more severe than that caused by nephrotoxic agents. The nephrotoxins most commonly encountered are drugs (NSAIDs, antibiotics, chemotherapeutics) and systemically released endotoxins such as myoglobin from rhabdomyolysis, immune complexes and calcium.

Often the problem is confounded by either pre-existing renal disease or untreated prerenal failure. Therefore, use of nephrotoxic agents in this at risk groups, can lead to a further deterioration of renal function. For example, the use of NSAIDs becomes hazardous during times of precarious renal blood flow i.e. during postoperative period and trauma.

Postrenal acute kidney injury

Obstruction of the ureters and urinary system will eventually result in back pressure being transmitted to the tubules. Since filtration relies on the gradient across the glomerulus, there will be resultant loss of GFR.

It's important to recognise that renal function steadily deteriorates after the age of 30, so elderly patients are particularly at risk of developing renal dysfunction in the perioperative period.

Prognosis of acute kidney injury

The commonest cause of AKI is prerenal, due to acute hypovolaemia and generally has a reversible cause, with a better outcome. The other large group is those seen in the critical care setting where it is part of multiorgan failure, with a correspondingly poorer prognosis - up to 70% mortality. Anuria (<10-15ml/hr) carries the highest risk, this probably reflects that it is seen more commonly in ischaemia then nephrotoxicity. Overall mortality is about 50%; death is usually related to the underlying disease.

Signs and symptoms of acute kidney injury

The most important step is recognition of reversible causes. Hypovolaemia often manifests itself as a decline in renal function as the body attempts to correct the deficit through its normal physiological mechanisms.

In the absence of obvious hypoperfusion more subtle signs need elucidating. This will involve a thorough history and examination of all systems, starting with the skin and working through, looking for prerenal, intrinsic and post-obstructive pathologies. The actual signs of renal dysfunction relate to volume overload and toxin accumulation.

Acute volume overload may result in reduced oxygenation secondary to pulmonary oedema. With this in mind, it becomes crucial to deliver intravenous fluids in a controlled manner.

Clinical signs

- Asterixis*and myoclonus
- Pericardial or pleural rub
- Peripheral oedema (if volume overload is present)
- Pulmonary crepitations (if volume overload is present)
- Elevated right atrial pressure (if volume overload is present)

** Asterixis describes a motor disturbance characterised by intermittent lapses of an assumed posture*

Toxaemia due to uraemia is most symptomatic: confusion, agitation, seizures and vomiting, pruritis, pericarditis, pericardial and pleural effusions. Asymptomatic and acute rise in plasma potassium may lead to fatal cardiac dysrhythmias and characteristic ECG changes.

Investigations in AKI

The following biochemical investigations are helpful:

- *Urinalysis/microscopy*: protein, glucose, myoglobin red/white cells, organisms and tubular casts
- *Urine electrolytes and osmolality*: give an indication as to whether normal renal function is preserved i.e. sodium and water conservation.
- *Serum electrolytes*. Sodium and Potassium can be markedly affected depending on whether they are poorly filtered or cleared in excess. In most instances of AKI, clearance is affected, so potassium levels rise and sodium falls (due to the dilutional effect of poor fluid removal)
- *Urea and creatinine*: usually elevated and will give some idea as to the severity and time scale. As both are by-products of metabolism, they can dramatically increase in catabolic states. Creatinine in particular can be falsely elevated by drugs or lowered by the disease process itself i.e. glomerulonephritis. However, the plasma creatinine does give an indication of creatinine clearance (almost exclusively cleared by filtration) and thus GFR.

Table 4.3. The relationship between creatinine and GFR

Creatinine (µmol/l)	Reduction in GFR (%)	GFR (ml/min)
<100	normal	120
180	50%	60
360	70-85%	18 - 36
720	90-95%	6 - 12

- *Arterial blood gases* -useful for assessment of acid/base status and potassium level.
- *Imaging studies* - focused on causes; in particular a renal ultrasound or CT KUB will allow elimination of any postrenal causes.

Management of AKI

Treatment can be sub-divided into the acute therapy aimed at:

- Restoring circulating volume which would be instigated in any hypovolaemic patient.
- Treatment of the cause (if not hypovolaemia).
- Specific therapy aimed at replacing the function of the kidneys. In patients with absolute anuria (no urine), urinary tract obstruction should be excluded.

Principles of immediate management

- Restoration of circulating volume

- Management of immediate life threatening consequences (acidosis & hyperkalaemia)
- Careful search of underlying cause
- Exclude urinary tract obstruction
- Summon help from specialist team.

Initial therapy should in all cases begin with intravenous fluid replacement, the establishment of effective renal blood flow is paramount. However, caution must be exercised as volume overload can result in fatal consequences, typically pulmonary and cerebral oedema. To aid this, close observation and monitoring must be instigated. A urinary catheter is advocated, with hourly urine output readings. This is in addition to typical observations of; heart rate, blood pressure, respiratory rate, temperature and conscious level.

If there is any doubt about the direction of therapy, early specialist referral must be sought, either from renal physicians or more appropriately in severe illness, the critical care team. The main aim of initial treatment is to restore effective circulating volume and thus renal perfusion.

The role of diuretics is controversial, they may have a role in volume reduction if the kidneys still have some function, and they may also reduce tubular oxygen consumption by inhibiting the Na^+-K^+-Cl^- pump. Furosemide can be considered in volume overloaded states, but caution must be exercised, as their use can cause further deterioration.

Should restoration of adequate circulatory volume with fluid, not provide a sufficient blood pressure. Then the use of inotropes and vasopressors may be indicated to establish sufficient perfusion pressure, since renal blood flow is pressure driven below the kidneys' autoregulatory range.

Restoring perfusion and re-establishing function is the best way of restoring fluid and electrolyte balance. However, raised potassium levels can be fatal and any value over 6mmol/l should be monitored closely and over 6.5mmol/l acted upon.

Treatment of hyperkalaemia

- Measures to protect the heart: Calcium chloride or calcium gluconate has membrane stabilising effects.
- Measures to redistribute the serum K^+
    - $ß_2$ agonists such as salbutamol
    - Insulin and Dextrose - Insulin reduces potassium levels by causing a shift to intracellular stores.
    - Sodium bicarbonate - drives the potassium inside the cells.
    - Hyperventilation in ventilated patients, they alkalinise the blood. H^+ comes out of the cells to neutralise the pH. To maintain the electrical neutrality K^+ enters inside the cells.
- Measures to eliminate K^+ from body
    - Resins: Sodium and calcium resins.
    - Haemodialysis or haemofiltration

In addition any cause should be treated such as infection, immune mediated disease or relief of obstruction.

Renal replacement therapy (RRT)

Should initial therapy (fluids) fail to restore function then the next step is RRT.

The key principles of RRT

- Ultrafiltration – solutes and plasma water are forced through a semipermeable membrane by high ultrafiltration pressures (Convection)

- Dialysis – Solutes and plasma move across a semipermeable membrane across their concentration gradients (Diffusion)

These can be used either alone or in combination, they may also be used continuously or intermittently. Each method has its advantages and disadvantages, though there is no clear evidence to support one over the other.

For general principles renal replacement therapy refer to
https://youtu.be/UWMQtgqWL2w

In clinical practice the following are used:

Peritoneal dialysis (CAPD): The simplest and most cost effective method uses the peritoneal membrane for dialysis. However poor solute clearance and risk of infection limit the role of this technique in AKI.

Intermittent haemodialysis: Large volumes of fluid and solute are removed quickly and most efficiently using both filtration and dialysis, this can cause significant hypotension. Therefore, it is not used conventionally in cases of acute deterioration in renal function.

Continuous venovenous hemofiltration (CVVH): Solutes are removed entirely by convection. The hydrostatic pressure results in filtration of plasma across the hemofilter membrane. Dialysate fluid is not used.

Continuous venovenous hemodialysis (CVVHD): As in intermittent hemodialysis, dialysate fluid is run counter current to the direction of blood flow at a rate of 1 to 2 L/hour. Solutes are removed by diffusion. In CVVHD, ultrafiltration is limited to the rate at which net fluid removal is desired, and no intravenous fluid replacement is required.

Continuous venovenous hemodiafiltration (CVVHDF): This is the standard RRT used in intensive care units, it utilises both filtration and dialysis. The continuous flow allows small amounts of fluid and solute to be removed in a controlled manner. Requires venous access via a central vein using a vascular catheter (vascath). Common insertion sites for a vascath include internal jugular vein and femoral vein, with subclavian access considered in some situations. Both IHD and CVVHDF are invasive and require specialist supervision.

For principles of CVVH refer to https://youtu.be/cBBpgoUGv_A

Indications for renal replacement therapy

- Volume overload with pulmonary oedema
- Hyperkalaemia
- Acidosis
- Symptomatic uraemia
- Specific toxin removal
- Sepsis (removal of cytokines)

The timing of RRT without specific indication remains controversial, though there is some evidence that early aggressive treatment may be beneficial. During the recovery phase of acute renal failure, care should be taken to avoid further insults to the kidney (hypotension and use of nephrotoxic drugs). By six months, the kidneys will have recovered most of their function, although some may progress to chronic renal failure.

Further reading

Renal replacement therapy in critical care. Lisa Gemmell, Robert Docking, Euan Black, *BJA Education*, Volume 17, Issue 3, March 2017, Pages 88-93, https://doi.org/10.1093/bjaed/mkw070

Perioperative acute kidney injury Gross, Jamie L et al. BJA Education, Volume 15, Issue 4, 213 - 218

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MANAGEMENT OF ACUTE POISONING AND DRUG OVERDOSE

5

Acute poisoning is a common emergency that presents to the emergency depai and to intensive care. Poisoning accounts for >100,000 hospital admissions per year in the UK. Poisoning may be accidental, iatrogenic or may be due to suicidal intention. Consequently, patient history is sparse and often unavailable. The majority of the patients are young, otherwise medically fit and they will recover with basic supportive care. Following the initial assessment and management in the emergency department, they may require admission to intensive care for the following reasons

- Altered level of consciousness
- Respiratory failure
- Cardiac arrhythmias
- Cardiac conduction disturbances resulting A-V block
- Hypotension
- Acute renal failure

Drug overdose may occur through several routes: oral, inhaled, skin contact or intravenous. The following are the most commonly consumed poisons:

- Industrial or agricultural chemicals
- Household products
- Plant and animal toxins, fumes
- Recreational drugs: Amphetamines, Ecstasy

Increasingly drug overdoses are becoming more common and include:

- Paracetamol
- Tricyclic antidepressants (amitriptyline)
- Salicylates (aspirin)
- Opioids (codeine, morphine, diamorphine, fentanyl, oxycodone)
- Amphetamines, cocaine, ecstasy
- Benzodiazepines (diazepam, temazepam)

General principles of management

Drug overdose should always be considered in an unconscious patient. General principles of management involve assessment, resuscitation and identification of the specific poison. Often drugs are taken in combination which can affect their pharmacological profile. In circumstances where the patient is unable to provide a history, a collateral history is paramount, particularly from the paramedics, accompanying friends/family or even the GP.

- Resuscitation using an ABC (airway, breathing and circulation) approach
- Identification of type of overdose, type of drug and amount
- Limiting the absorption of drug
- Enhancing the elimination of drug
- Specific treatments and antidotes
- Organ supportive care (as required)
- Psychiatric review if deliberate overdose

Supportive care and monitoring

Patients in whom an overdose is suspected should be cared for in an appropriate environment which typically tends to be resus within an emergency department. Beyond this, an acute medical ward or intensive care may be suitable depending on the sequalae of the overdose. Intubation may be necessary should the patient's GCS be inappropriate, for ventilatory support or to facilitate organ support.

If cardiovascular impairment is suspected, a cardiac monitor should be in place and possibly an arterial line for beat to beat analysis of blood pressure or blood gas analysis.

GCS and pupillary size need frequent monitoring as they fluctuate according to drug concentration and pharmacokinetics. Body temperature and glucose should be monitored regularly whilst the patient has been admitted.

Table 5.1. Specific signs in poisoning and overdose

Drug Class	Typical examples	Common Findings
Anticholinergic	Atropine	Altered mental status, dilated pupils, urinary retention, hyperthermia, dry mucous membranes
Cholinergic	Organophosphates	Salivation, lacrimation, Urination, Defecation, Nausea/Vomiting, Muscle Weakness
Opioid	Heroin, morphine	CNS & Respiratory depression, pupillary constriction
Salicylates	Aspirin	Altered mental status, respiratory alkalosis, metabolic acidosis, tinnitus, tachycardia, sweating
Serotonin Syndrome	MAOI, TCA, SSRI	Increased muscle tone, hyperthermia, hyperreflexia
Sympathomimetics	Cocaine, Amphetamine	Agitation, Dilated pupils, sweating, tachycardia, hypertension

Investigations

Blood tests are typically first line and aside from a routine panel of tests, a paracetamol level is often performed 4 hours post ingestion. A full blood count, urea & electrolytes, glucose, lactate and clotting are necessary to assess the impact of the ingested drug on the patient's physiology.

Urine and plasma toxicology can give an indication of ingestion of other common drugs.

An ECG to detect changes in PR interval, QRS length, rhythm, rate as conduction defects and electrical abnormalities can arise depending on the offending drug.

A Chest X ray can be considered if pulmonary oedema or aspiration is suspected.

TOXBASE is a widely used platform that offers evidence backed guidance and advice for investigation and management of a multitude of poisons and drug ingestions or exposure.

Limiting the absorption of drug

Activated Charcoal
Works on the principle of binding with a drug in the stomach, thereby preventing absorption, via excretion of the drug-charcoal complex in the faeces. However, it is only useful if taken within 1 hour of ingestion, with a required dose of 1g/kg.
Side effects can include – vomiting, aspiration and prevention of absorption of other drugs (e.g antidotes). Useful for drugs such as: aspirin, paracetamol, digoxin, TCA.

Induced Emesis
Syrup of Ipecac can be given to patients which results in rapid onset emesis.
However, this practice is no longer advocated as its use confers no additional benefit.

Gastric Lavage
This principle involves passing saline or water into the patients stomach via a large bore nasogastric or orogastric tube and aspirating the stomach contents. The patient is placed in left lateral with their head down and the technique is repeated until the aspirate remains clear. This is only useful if performed early whilst the contents are still within the stomach. If the patient's GCS is reduced, then the airway should be protected via intubation.
Side effects can include – vomiting, aspiration, laryngospasm, visceral damage.

Whole bowel irrigation
Performed with polyethylene glycol to reduce bowel transit time. Useful in patients in whom activated charcoal is not appropriate.

Endoscopy
Rarely used, but can be considered if drugs are actively placed into the GI tract or for toxic metals.

Enhancing the elimination of drug

- Forced diuresis - infusing fluids to increase urine output and therefore increasing elimination of the drug.

- pH manipulation-altering pH of urine to increase elimination of the drug. Alkalisation of urine enhances the excretion of acidic drugs such as salicylates and phenobarbitone. Acidification of urine enhances the excretion of alkaline drugs such as amphetamines. These drugs become

ionised in the urine and become trapped without entry back into the circulation.

- Haemodialysis is useful for drugs with low protein binding, small molecular weight and small volume of distribution.

Specific treatments and antidotes

Paracetamol (Acetaminophen)

Responsible for 30,000 UK hospital admissions per year, resulting in 345 deaths. In most cases however, there are no symptoms or signs. Ingested paracetamol at 150mg/kg can be fatal, but 200mg/kg is quoted as the threshold for potential paracetamol induced hepatic injury. This is via hepatocellular necrosis in overdose due to saturation of metabolic pathways and exhaustion of glutathione stores.

Normally, paracetamol is conjugated with glucuronide and excreted by the kidneys in urine. In normal metabolism in healthy individuals, 5% of paracetamol metabolism undergoes conversion to toxic N-acetyl-p-benzoquinone amine (NAPQI), which is conjugated to glutathione and again excreted by the kidneys (Figure 5.1, A). In overdose, both mechanisms are overwhelmed, and NAPQI builds up causing hepatocellular damage (Figure 5.1, B).

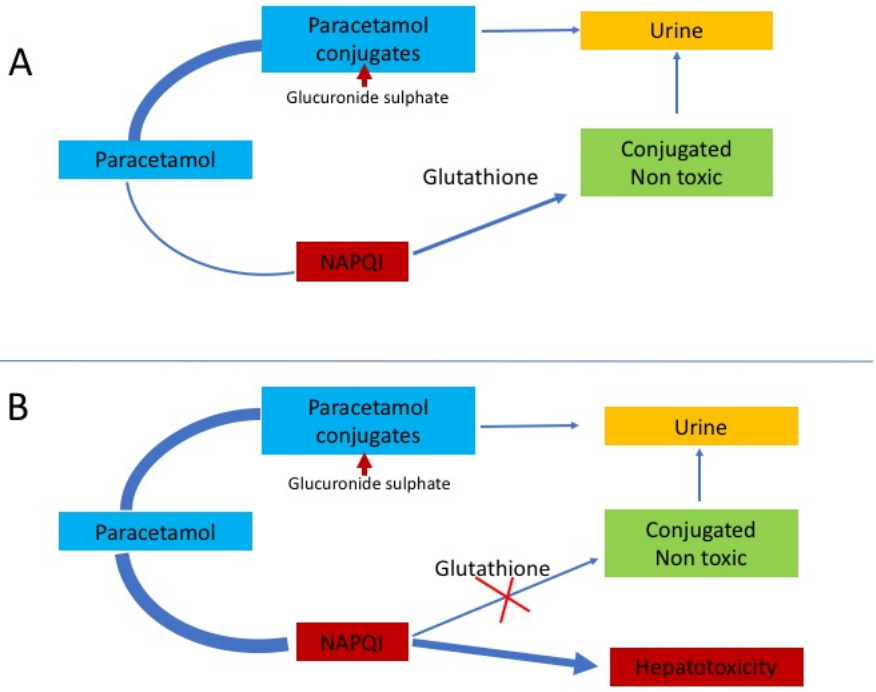


Figure 5.1 Metabolism of paracetamol. A) in normal healthy individuals and B) in paracetamol overdose.

Treatment for paracetamol toxicity involves replenishing glutathione store with either Methionine (oral) or N-acetyl cysteine (iv). N-acetyl cysteine tends to be favoured at

most units in the country and the decision to treat depends on the blood paracetamol concentration at a number of hours following ingestion. This is guided by the nomogram. Patients whose plasm concentration is on or above the treatment level should be treated with N-acetylcysteine.

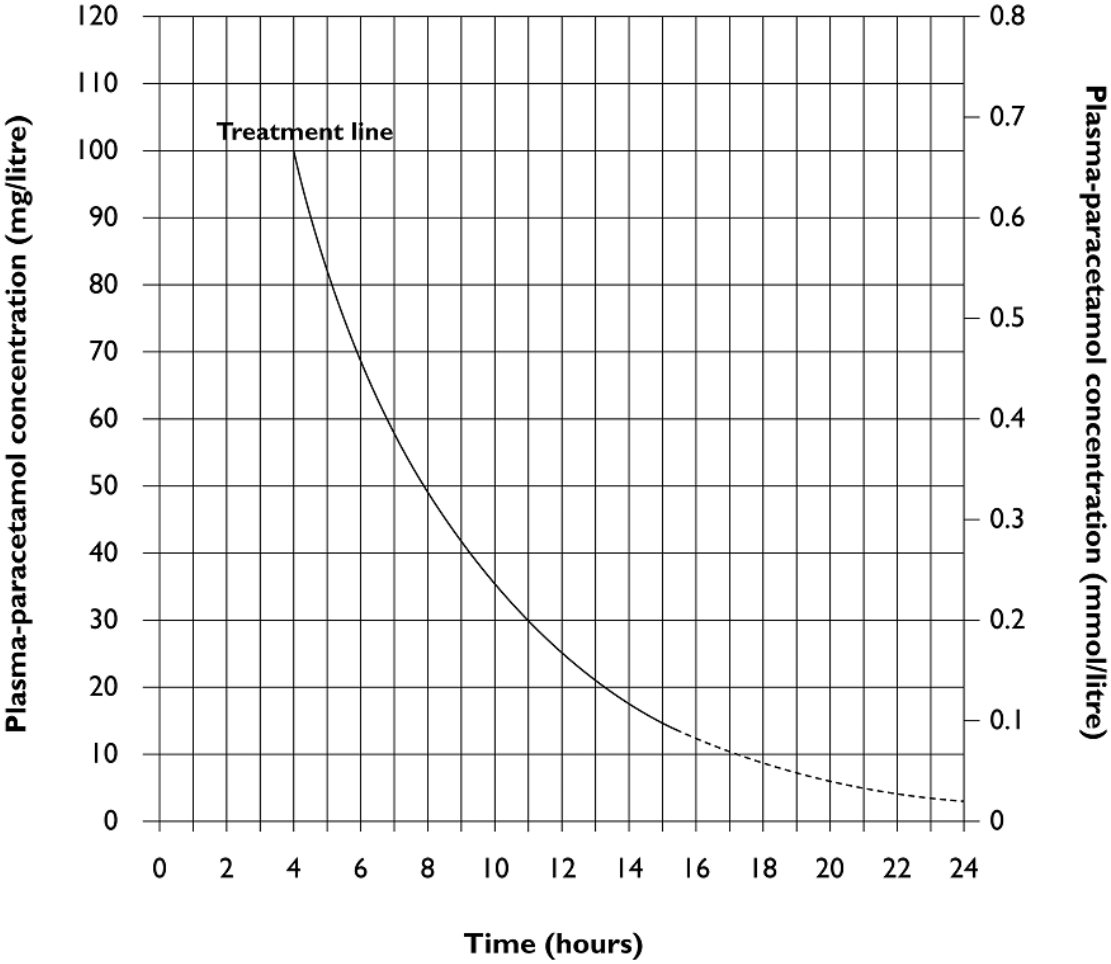


Figure 5.2 Paracetamol overdose treatment graph (reproduced from https://bnf.nice.org.uk)

A loading dose of 150mg/kg of N-acetylcysteine is given over 1 hour, this is followed by a dose of 50mg/kg over 4 hours, following which a dose of 100mg/kg over 16 hours is given. There is current discussion as to whether to maintain a continuous infusion, but this is yet to be adopted nationally.

Liver function, creatinine, INR and blood gas analysis should be performed if the presentation is delayed.

The King's College Hospital Criteria is used to determine whether a patient needs referral to a tertiary hepatic transplant unit in view of fulminant hepatic failure:

- Arterial pH < 7.30
- INR > 6.5 (PT > 100s)
- Creatinine >300µmol/L
- Hepatic Encephalopathy (Grade III or IV)

Carbon Monoxide poisoning

This is an odourless, colourless gas, that is toxic to humans as it has a greater affinity for haemoglobin molecules than oxygen. Oxygen delivery to tissues is impaired, therefore tissue hypoxia and ischaemia can ensue.

It's important to recognise carbon monoxide poisoning in addition to patients with inhalational or burns injuries.

Patient's may present with headache, tachycardia and nausea, which can progress to convulsions, coma, cardiovascular instability and failure.

Bedside investigations include measurement of oxygen saturations (typically normal), arterial blood gas to measure PaO_2 (may also be normal) and COHb. In health, COHb is approximately 0.5%, but the severity of symptoms increases alongside the concentration of carbon monoxide.

Treatment is centred around reducing the concentration of carbon monoxide, by removing the offending pathology and increasing the FiO_2. Hyperbaric oxygen therapy is another modality that is described, however the national poison information service (NPIS) does not recommend its use with co-existing smoke injury.

Tricyclic antidepressants overdose

The incidence of tricyclic antidepressants (TCA) overdose is on the rise, given renewed interest in the medication for chronic pain and anxiolysis. The predominant cause of death is through blockade of sodium channels, thereby interrupting cardiac conduction. The side effects are driven by their anti-cholinergic properties, so patient's experience; dry mouth, dry skin, dilated pupils, hyperreflexia and tachycardia.

Clinical signs include metabolic acidosis, prolonged PR interval, wide QRS complex, arrhythmias, convulsions and coma.

Activated charcoal can be administered if ingestion was within an hour of presentation, but otherwise 1-2ml/kg of 8.4% sodium bicarbonate is used to stabilise the sodium channels and prevent further deterioration.

Benzodiazepine overdose

Patients who have taken a benzodiazepine overdose are likely to present with a depression of cardio-respiratory function and a reduced level of consciousness. Deaths associated with overdose of benzodiazepines are often as a result of mixed overdoses.

Flumazenil is a selective $GABA_A$ antagonist that can be used in isolated benzodiazepine overdose. Its half life is short, so the dose will need to be repeated or given as an infusion. The side effects include ventricular tachycardia, increased

intracranial pressure and seizures if TCAs have been taken in conjunction with the benzodiazepines.

Salicylates overdose

Aspirin is a weak acid which in excess, uncouples oxidative phosphorylation. This is typically responsible for aerobic metabolism, therefore as a result of an aspirin overdose, anaerobic respiration predominates and a lactic acidosis is generated. Patients can present with vomiting, dizziness, tinnitus and hyperventilation (early). It produces mixed respiratory alkalosis (as a result of hyperventilation) and metabolic acidosis (lactic acidosis). Later and more serious features include: seizures, hypotension, heart block and coma.

Management is largely supportive and activated charcoal can be considered depending on timing of ingestion. Haemodialysis may be required depending on the dose taken and physiological impact. Alkalinisation of the urine may also offer some benefit.

Organophosphates

This category of drug is responsible for a number of deaths worldwide, most notably in developing countries. It is mainly a feature of certain pesticides, but in recent times has become an intentional "nerve agent" as part of a more sinister agenda.

Organophosphates inhibit the action of the Acetylcholine Esterase (AchE) enzyme responsible for the breakdown of Acetylcholine (Ach) in the nerve cleft. As a result, there is an excess of Ach which leads to an exaggerated cholinergic or muscarinic response.

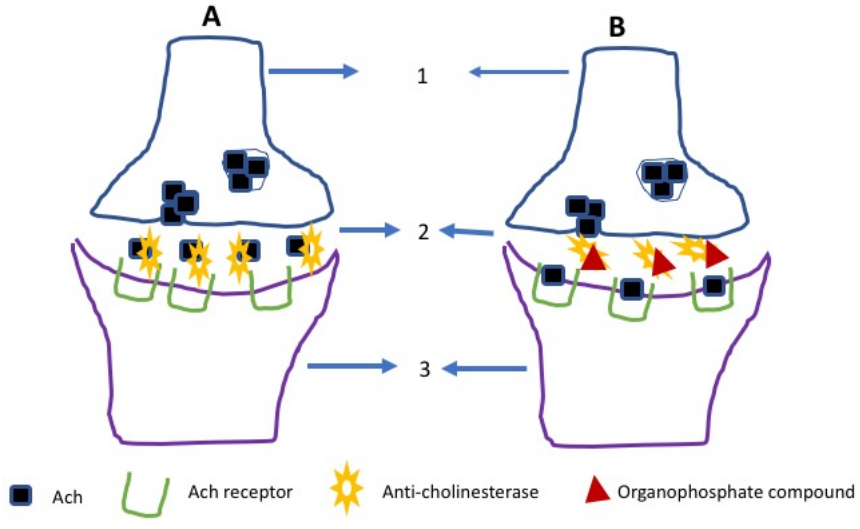


Figure 5.3. Mechanism of organophosphate effect at neuromuscular junction or nerve cleft. 1. Pre-synaptic neuron, 2. Nerve cleft/ neuromuscular junction and 3. Muscle cell or postsynaptic neuron

Patients present with symptoms such as Salivation, Lacrimation, Defecation, Urination, GI upset, Emesis, Miosis. These symptoms can be recalled using the acronym “SLUDGEM”.

Treatment is supportive depending on the clinical presentation, but antidotes such as atropine or pralidoxime can be considered due to their anti-muscarinic properties.

Psychiatric review

For patients with possible intentional overdose, a psychiatric review should be requested. This should take place before the patient is discharged from the hospital.

Further reading

Hulse, E. J. *et al.* (2019) ‘Organophosphorus nerve agent poisoning: managing the poisoned patient’, *British Journal of Anaesthesia*, 123(June), pp. 457–463.

CARDIO-PULMONARY RESUSCITATION (CPR)

5

Cardiac arrest is the absence of clinically detectable cardiac output and in prac terms it means an absence of a palpable carotid pulse.

Causes of cardiac arrest in adults

The causes of cardiac arrest can be mainly classified in to cardiac and non cardiac causes.

Cardiac causes
-Ischaemic heart disease – almost 80 % of cardiac arrests
-Valvular heart disease

Non-cardiac causes
- Overdose of drugs like sedatives, narcotics etc
- Peripheral circulatory failure due to bleeding, sepsis etc
-Respiratory failure (hypoxia, hypercapnoea)
-Hypovolaemia
- Hypothermia
- Electrocution
-Acute poisoning and drug overdose.

Cardio-pulmonary resuscitation (CPR) includes basic life support (BLS) using no equipment except for protective purposes and advanced life support (ALS) using advanced equipment like a defibrillator, tracheal intubation and drugs.

Basis for CPR

Immediate BLS is a holding measure until a definitive treatment for the underlying condition and the rhythm can be provided. By maintaining adequate ventilation and circulation, BLS delays the rate of deterioration of the brain and the heart until ALS can be provided by experienced personnel. In healthy individuals, absence of circulation for longer than 3-4 minutes will produce permanent brain damage. Rapid defibrillation/ALS has the capacity to establish the return of spontaneous circulation (ROSC).

The Resuscitation Council (UK) guidelines have been adapted from the 2015 ERC Guidelines and are tailored specifically to clinical practice in the UK.

Management of cardiac arrest

The cardiac arrest victim who is collapsed requires immediate attention. It is important to monitor and interpret the ECG, which enables providers to choose an appropriate treatment regime during ALS.

Rhythms associated with cardiac arrest

1. *Ventricular fibrillation (VF)* and *pulseless ventricular tachycardia (pVT)*: Both these rhythms should be treated with prompt defibrillation. VF is the most common rhythm at the time of cardiac arrest in adults.

2. *Asystole*: Cardiac arrest with no ECG complexes. It is important that a correct diagnosis of asystole is made and VF is not missed by checking that the electrodes are connected correctly and checking the gain on the ECG monitor.

3. *Pulseless electrical activity (PEA):* Presence of cardiac electrical activity in the patient with no signs of life. Exsanguination, pulmonary embolism or cardiac tamponade are important causes.

Adult in hospital resuscitation (algorithm 1)
The first responder to an in-hospital cardiac arrest should follow the following steps.

- Make sure the rescuer and the patient are safe – is it safe to approach?
- Check for life – Gently shake the shoulders and shout loudly to see if the patient responds.
- If there is a response, leave the patient as you find them, provided it is safe to do so and assess the patient for the reason for cardiac arrest and reassess regularly. Urgent medical assessment is required and follows an ABC approach. Give a high concentration of oxygen, attach the monitor and get venous access while waiting for the medical team.
- If unresponsive summon for help immediately.
- Open the airway – using airway manoeuvres such as a head-tilt chin-lift or jaw thrust
- Assess for signs of life
    - Whilst maintaining the airway, look for coordinated chest movement, listening and feeling for the air movement and palpate for a carotid pulse – **Maximum 10 seconds.**

Signs of life or a pulse present

- Keep the patient in the recovery position while waiting for an urgent assessment by the medical emergency team. Follow ABCDE, give oxygen, attach the monitor and start IV fluids while waiting for the team.

No signs of life or no pulse

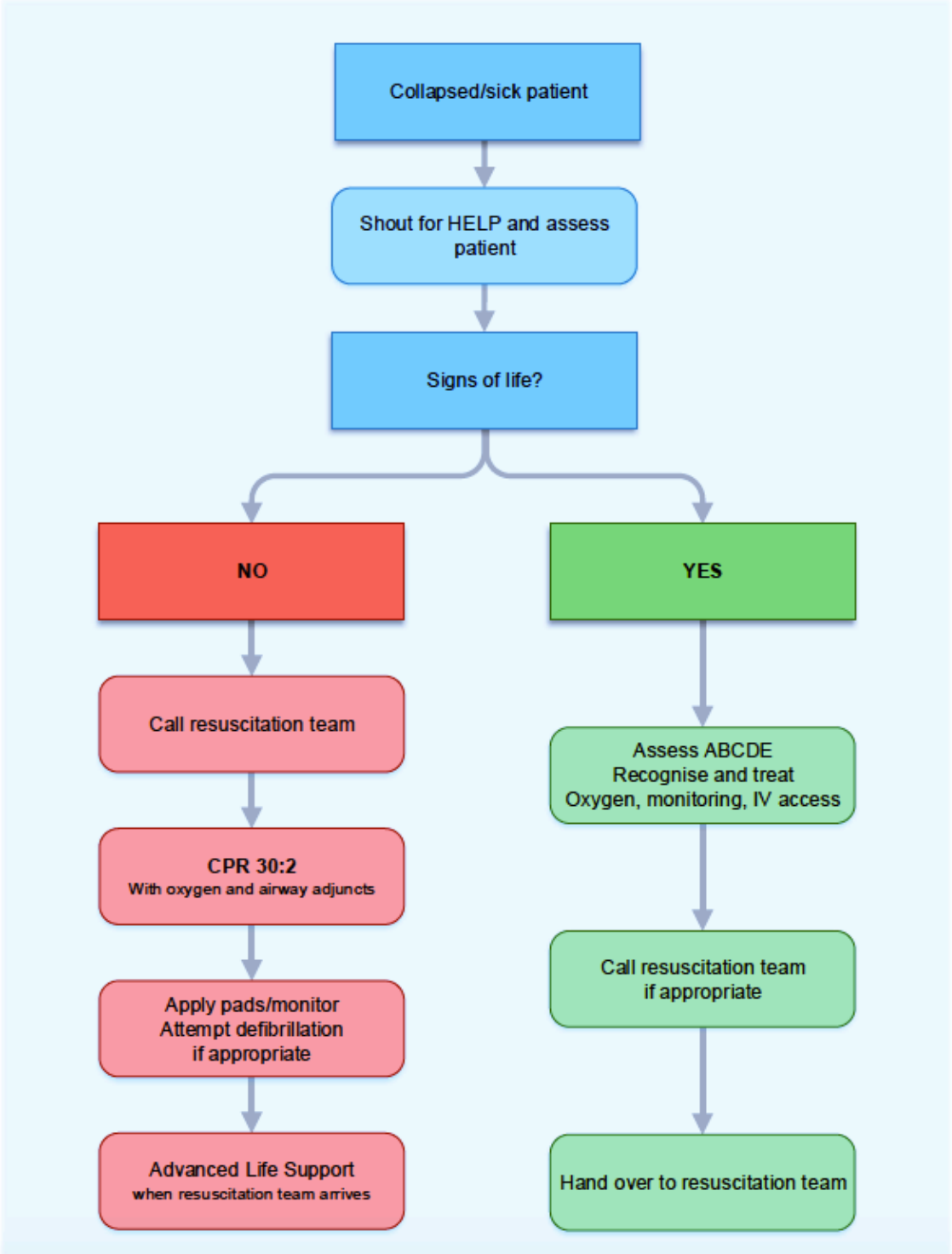
- If the patient does not have pulse or other signs of life -
    - One person starts CPR while the other goes to call the arrest team, get the arrest trolley (which contains the monitor and defibrillator, airway equipment, drugs etc).

- Perform 30 external cardiac compressions followed by 2 ventilations. (30:2)
    - External cardiac compressions are done at a rate of 100-120/min, for a depth of 5-6 cm at the middle of the lower half of the sternum.
    - Maintain airway and breathing using the appropriate equipment available immediately.

    - The tidal volume should resemble that of normal breathing, just enough to rise the chest with inspiratory time of 1 second and respiratory rate of 10/minute. Oxygen should be supplemented as early as possible.
    - Ventilation and compressions are to be done separately until the airway is secured after which they can be done asynchronously.
    - As soon as the defibrillator becomes available attach the electrodes to the patient and analyse the rhythm.
    - A feedback device can be used to assess the effectiveness of compressions.

- Minimise interruptions to external cardiac compressions. External cardiac compressions are to be interrupted only to analyse the rhythm and for defibrillation but to be recommenced once the defibrillation is attempted, not waiting to assess the rhythm or feel for the pulse.
    - CPR has to be continued until the patient shows signs of life or the arrest team arrives. *When using a manual defibrillator use the ALS algorithm*.
    - Change the person providing compressions every two minutes.

- The exact course of action following in hospital cardiac arrest depends on the following factors-
    - Site – if it is in clinical areas like CCU/ICU, where patients are already monitored the diagnosis is made almost instantaneously or non-clinical areas when there might be a delay in exact diagnosis.
    - Number and proficiency of staff members – if there is only one staff member then he/she should go and call for assistance before initiating CPR.
    - Availability of equipment and drugs in the immediate vicinity.



Algorithm 1. Adult in hospital resuscitation
(https://www.resus.org.uk/resuscitation-guidelines/)

Importance of quality chest compressions

External cardiac compressions produce cardiac output by compressing the heart against the spine with the cardiac valve acting competently (cardiac pump theory), or the heart acting as a valveless conduit by increasing the intrathoracic pressure to squeeze blood out of the heart (thoracic pump theory). The compression rate refers to the rate at which the compressions are to be done and not the number of compressions that are given, which depends upon, apart from the rate of compression, also on the number and duration of interruptions for the other steps of CPR.

Advanced life support

Current ALS guidelines recognise two different pathways during cardiac arrest:

1. Shockable rhythms:
   VF or pulseless VT, whereby the treatment strategy is centred around prompt defibrillation to initiate and reorganise myocardial contraction
2. Non-Shockable rhythms:
   Asystole or PEA, whereby chest compressions and drugs are administered whilst reversible causes are corrected.

Therefore, the aim of ALS is to ensure the defibrillator is available and the pads are placed onto the patient's chest at the earliest opportunity to work out which side of the algorithm to execute.

Shockable rhythm
(VF / pulseless VT) (algorithm 2)

- Once cardiac has been confirmed, start compressions and ventilation in a ratio of 30:2.
- As soon as the adhesive pads are attached to the patient and leads connected to the defibrillator, stop compressions to assess the rhythm.
  - If VF/VT
    - Undertake defibrillation – Delegate to the individual operating the defibrillator.
    - Chest compressions are continued whilst the defibrillator is being charged.
    - Oxygen (if open circuit) and all other staff should step away from the patient. If the oxygen is within a closed circuit, then this can remain attached.
    - Once charged, a shock is delivered of 150-200 J biphasic / 360 J monophasic.
    - Chest compressions are immediately re-started and the above sequence is continued until ROSC is achieved or the rhythm moves onto the non-shockable side of the algorithm.

  - Drugs

- Prefilled syringes are available for use in cardiac arrest within the arrest trolley.
- Adrenaline 1mg (1:10000) is given after the third shock and repeated following every other shock.
- Amiodarone 300mg is given after the third shock only.
- Administration of drugs is followed immediately by a 20ml flush of Normal Saline.

Check for a pulse if there is any organised electrical activity during brief pauses in compression (after 2 minute cycle) –

- If there is a pulse, then start post-resuscitation care.
- If there is no pulse, continue CPR and switch over to non-shockable part of algorithm.

Non-shockable rhythm (PEA and asystole) (algorithm 2)

- Commence CPR in the ratio of 30 compressions to 2 ventilations.
- If an advanced airway is secured, the ventilation and compressions can be performed asynchronously.
- Attach the defibrillator pads as above, and assess the rhythm.
- If there is a non-shockable rhythm, continue chest compressions and administer 1mg Adrenaline (1:10000). This is repeated after alternate cycles.
- Check the rhythm and pulse after 2 minutes of CPR.
- If there is no change in ECG –
    - Continue CPR and recheck the ECG in 2 minutes.
    - Give Adrenaline1 mg every 3-5 minutes (alternate loops).

Reversible causes of cardiac arrest

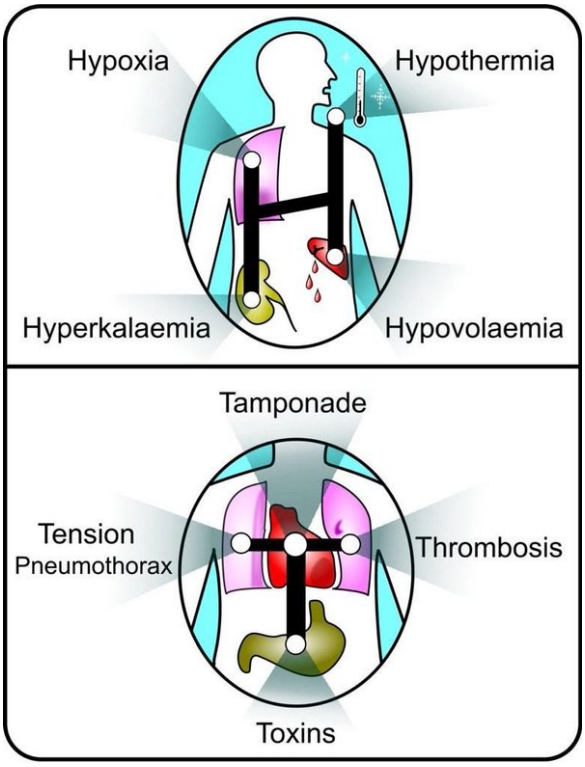
These can be broadly classified into the 4Hs and 4Ts. Throughout ALS, it is important to consider these potential causes to see if treatment can be focussed to rectify the arrest.

- Hypoxia
- Hypothermia
- Hyperkalaemia
- Hypovolaemia
- Thrombosis
- Tension Pneumothorax
- Tamponade (Cardiac)
- Toxins

Practical aspects of defibrillation

- Place pads across the patient's chest - usually over the right apical region and left mid axillary line (Bi-axillary or Anterior-posterior placement can also be considered)

- Safety is of paramount importance during defibrillation. It is important to avoid defibrillating over ECG electrodes and making any physical contact with the patient during defibrillation. Keep the oxygen at least one metre away from the patient's chest unless they are connected to a closed circuit.



(https://www.resus.org.uk/resuscitation-guidelines/)

Figure 5.1. Reversible causes of cardiac arrest

Airway Devices

i-gel

Referred to as a supraglottic device. This is inserted into the patient's mouth and sits above their glottis (vocal cords). This is the main difference between an i-gel and an endotracheal tube (for which one needs to be intubated and the tube sits within the trachea, below the vocal cords).

The cuff is made of a fixed elastomer gel and does not require inflation. It is relatively easy to insert and requires minimal training, which is why it is the first choice for advanced airway management in an arrest situation, including pre-hospital care. Capnography can be easily attached to the hub of the i-gel.

Tracheal tube

The tracheal tube has generally been considered the optimal method of managing the airway during cardiac arrest. There is evidence that, without adequate training and experience, the incidence of complications, such as unrecognised oesophageal intubation (2.4–17% in several studies involving paramedics) and dislodgement, is unacceptably high. Prolonged attempts at tracheal intubation are harmful; the cessation of chest compressions during this time will compromise coronary and cerebral

perfusion. Therefore, for the untrained operator, an i-gel should be considered as the airway of choice.

Capnography

Use waveform capnography whenever an advanced airway is inserted (iGel or tracheal intubation). Its use includes:

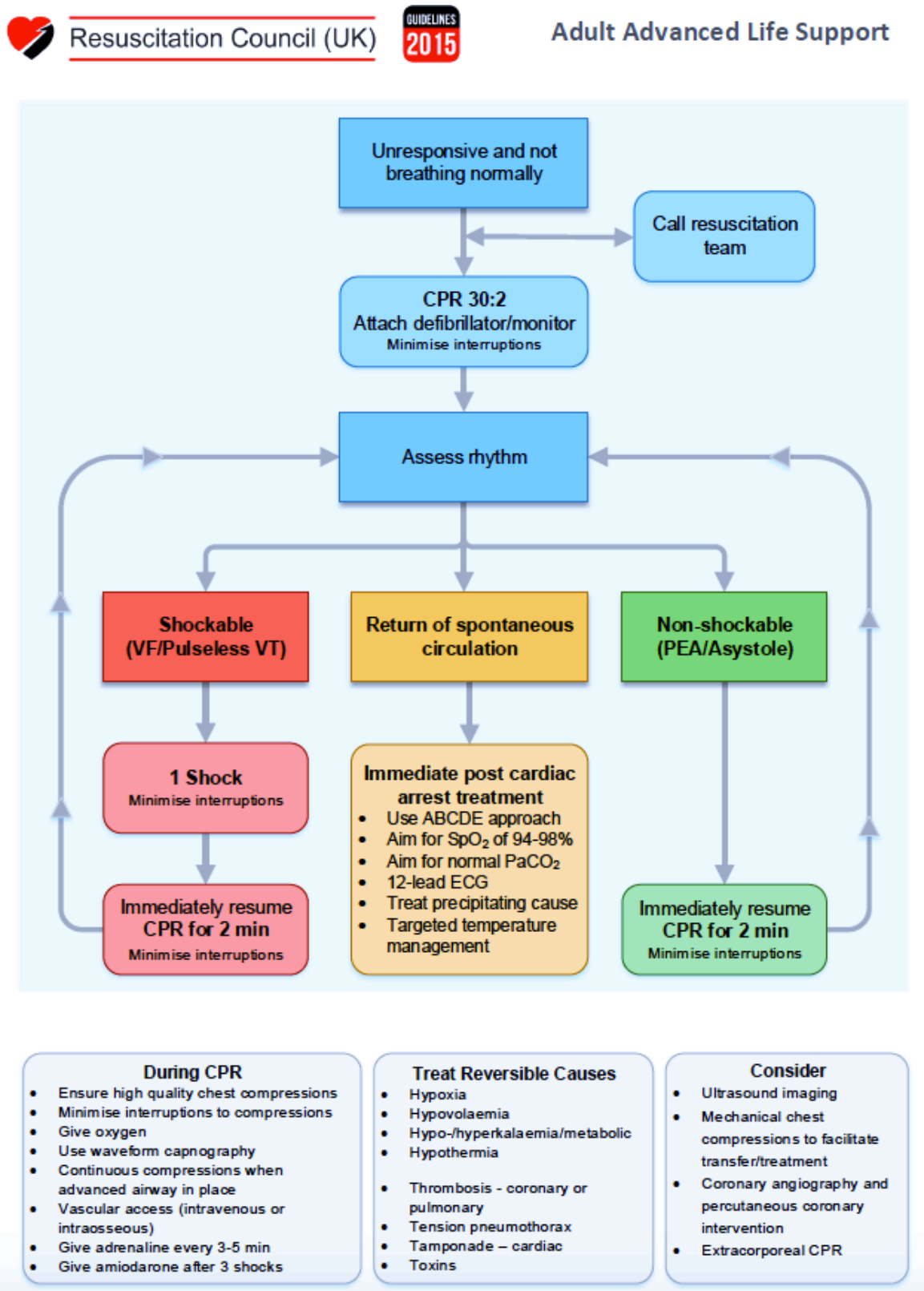
- Assessment of the quality of chest compressions and rate of ventilation. CO_2 is produced from respiring tissues, and if clinically detectable in the exhaled sample (end tidal), then the assumption is that the chest compressions are effective in delivering blood to and from respiring tissues. End-tidal CO_2 values are associated with compression depth and ventilation rate and a greater depth of chest compression will increase the value.
- Able to help confirm accidental oesophageal intubation.
- Identifying ROSC (return of spontaneous circulation) during CPR. An increase in end-tidal CO_2 during CPR can indicate ROSC and prevent unnecessary and potentially harmful dosing of adrenaline in a patient with ROSC. If ROSC is suspected during CPR withhold adrenaline. Give adrenaline if cardiac arrest is confirmed at the next rhythm check.

Administration of other drugs during CPR

- *Magnesium*: 2 g given peripherally IV (repeated every 10-15 minutes). Indicated for: VT, torsades de pointes, or digoxin toxicity associated with hypomagnesaemia.

- *Calcium*: 10 ml of 10% calcium chloride IV. This is indicated only when the PEA is due to hyperkalaemia, hypocalcaemia and overdose of calcium channel blocking drugs and magnesium.

- *Sodium Bicarbonate*: 50 mmol (50 ml 8.4% solution). Routine use is not recommended but can be considered in cases of cardiac arrest due to hyperkalaemia or tricyclic antidepressant overdose and is to be repeated only with blood gas guidance.



Algorithm 2. Adult ALS treatment (*https://www.resus.org.uk/resuscitation-guidelines/)*

Peri-arrest arrhythmias

In all patients with arrhythmias, oxygen should be administered, an intravenous line should be secured for administration of drugs and a 12 lead ECG should be recorded. Electrolyte disturbance mainly K^+, Mg^{2+}, should be corrected.

The treatment of an arrhythmia depends on the effect that it has on the patient as well as the type of arrhythmia. The arrhythmia can produce haemodynamic instability producing the following adverse features:

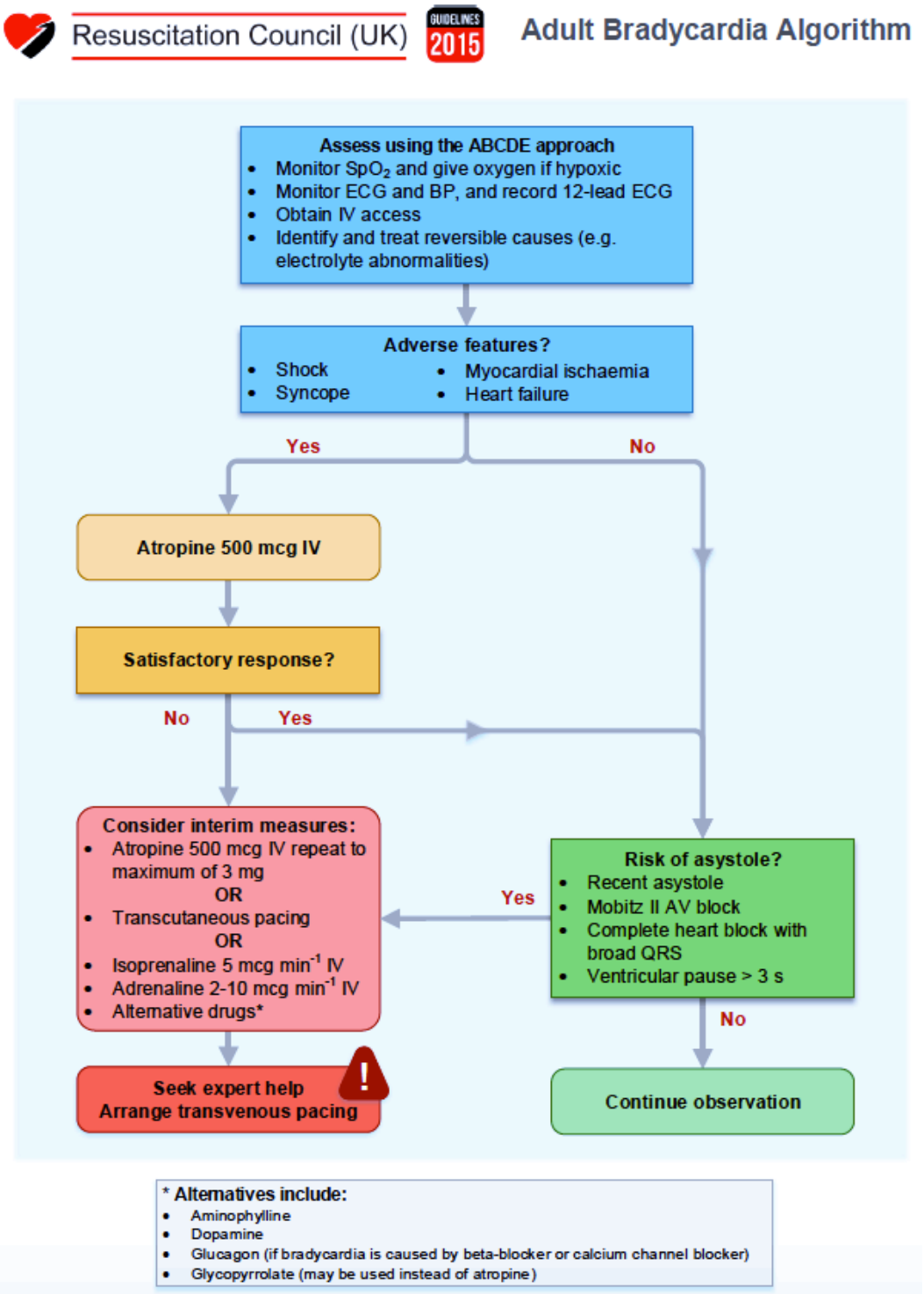
- Signs and symptoms of low cardiac output
    - Poor perfusion (peripheries – pallor, brain – impaired consciousness)
    - Reflex sympathetic activity (sweating, cold and clammy extremities)
    - Hypotension (systolic BP <90 mmHg)
- Very high heart rate – Excessive heart rate, by reducing the duration of diastole, (during which coronary perfusion occurs) reduces coronary perfusion producing signs of coronary ischaemia like chest pain. Broad complex tachycardias (ECG QRS width ≥12 milliseconds) which are of ventricular origin are more serious than narrow complex tachycardias (ECG QRS width ≤12 milliseconds) which are of supra ventricular origin.
- Excessive bradycardia – defined as heart rate <40/min. Cardiac output may decrease with such a slow heart rate and can also occur with heart rate of <60/min in a heart with very poor reserve.
- Signs and symptoms of heart failure such as raised JVP, hepatic engorgement and pulmonary oedema.

Bradycardia (algorithm 3)

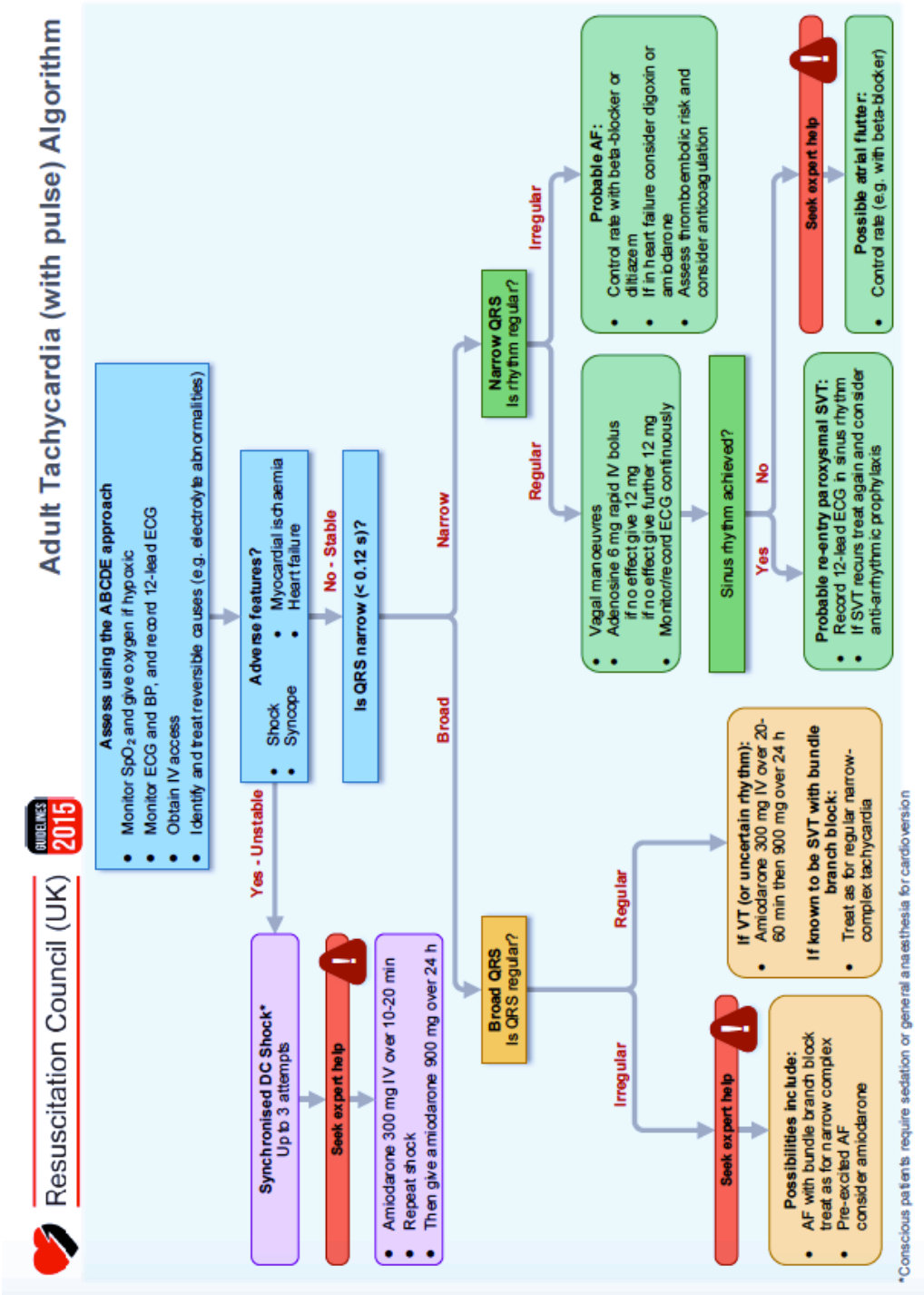
Bradycardia is heart rate <60/min. Adverse features such as shock, syncope, myocardial ischaemia and heart failure can manifest and necessitate immediate management.

Tachycardia (algorithm 4)

- Irregular narrow complex tachycardia is most likely to be atrial fibrillation or atrial flutter with variable AV block.
- A 12 lead ECG should be recorded.
- If the duration is >48 hrs (risk of thrombus increases) do not use cardioversion (electrical or chemical) until the patient is anticoagulated for at least 3 weeks or the trans-oesophageal echocardiogram shows no atrial thrombus.
- If the AF is of <48 hrs duration, the cardioversion can be achieved with amiodarone or electrical (which has got increased success rate).
- Drugs such as adenosine, digoxin, diltiazem, or verapamil should be avoided in patients with ventricular pre-excitation syndrome (WPW syndrome), this may increase the risk of pre-excitation by blocking AV node.



Algorithm 3. Bradycardia algorithm (*https://www.resus.org.uk/resuscitation-guidelines/*)



Algorithm 4. Tachycardia algorithm (*with permission from Resuscitation Council, UK. https://www.resus.org.uk/resuscitation-guidelines/)*

Post resuscitation care

The quality of management after ROSC influences the final outcome from cardiac arrest. Post resuscitation care is usually delivered in areas like ICU/HDU/CCU. If the patient is unconscious, it is better to intubate the trachea and ventilate the lungs to guarantee normocarbia and oxygenation and to prevent aspiration.

The patient may need invasive monitoring like arterial blood pressure, central venous pressure, cardiac output etc. Maintain electrolytes levels within the normal range, especially potassium and magnesium. Avoid hyperthermia and at the same time judicious use of mild hypothermia (temperature 32-36° C) for 12-24 hrs may be beneficial as it may minimise reperfusion injuries (only in VF arrests). However, hypothermia can produce detrimental effects like shivering which increases the oxygen demand and increased bleeding & wound infection in the perioperative period.

Patients with high blood sugar levels can have adverse neurological outcomes following cardiac arrest. There are no immediate neurological signs that can indicate outcome following cardiac arrest. Highly specific signs indicating poor outcome are absence of pupillary light reflex and motor response to pain by third day. Prognostication is very difficult before 72 hours post arrest. Modalities such as brain MRI are used to help aid decision making.

Ethical dilemmas surrounding resuscitation

CPR was originally conceived to save the lives of patients dying unexpectedly and efforts should be taken to minimise it to be used in patients in whom the underlying condition and general health makes the chances of success unlikely. A do not attempt resuscitation order, and more recently RESPECT form should be established as per the hospital protocol in a patient who does not want to be resuscitated or will not survive the cardiac arrest even if the CPR is attempted.

Videos

Cardiac Arrest Video
https://www.youtube.com/watch?v=jQYHQr3ebLo

ABCDE Assessment
https://www.youtube.com/watch?v=KNqoXboSVUI

Further Reading

1. Advanced Life Support Course. Provider Manual, 7th Edn. 2016. London: Resuscitation Council (UK) & ERC

2. https://www.resus.org.uk/resuscitation-guidelines/adult-advanced-life-support/