Perioperative Medicine

A Practical Handbook for Medical Students

The Royal London Anaesthesia Dept

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Published by The Royal London Anaesthesia Group

Printed by Book Printing UK www.bookprintinguk.com
Remus House, Coltsfoot Drive, Peterborough, PE2 9BF

Printed in Great Britain

Foreword

For many people anaesthesia seems very technical and mysterious. Most medical students have a limited exposure to the way we work, despite us being the biggest hospital speciality. Perioperative medicine is now growing as a field and understanding a patient's surgical journey is useful knowledge for any doctor.

We hope the information within this handbook will help you to understand some of the principles of perioperative care. We have pieced together some common themes in our speciality as well as some of the basic science underlying our work. This book may not help you pass exams, but it is a simple guide to encourage your curiosity and learning.

We wrote this book to help you understand an underrated, underestimated, and unique medical speciality, we hope you enjoy it!

Good luck!

Dr Manish Verma

Anaesthesia Registrar

North East Thames Rotation

The Pre-Operative Assessment: History

All patients undergoing anaesthesia should have a preoperative assessment. This is a focussed history and examination to plan for anaesthesia, surgery, and recovery. Patients are seen on the morning of surgery but are also often seen in a clinic prior to major surgery. A “system based” approach helps us assess the severity of co-morbidities. Here are some of the common lines of enquiry:

Airway History

-Fasting time
-Loose teeth
-Neck movement
-GORD/ OSA

Social History

- Smoking/ETOH
-Exercise tolerance
-Care at home?

Previous Anaesthetic History

-Adverse reactions
-Known ‘difficult airway’
- Previous post-operative nausea and vomiting (PONV)

Drug History

- Allergy/Anaphylaxis
-Anti-coagulants (bleeding)
-Antihypertensives (hypotension)
-Insulin dependent? (Hypo/Hyperglycaemia)

Family History

- ‘Malignant Hyperthermia’
-‘Suxamethonium Apnoea’

Medical History

- ***Respiratory:*** Recent URTI? COPD (Bronchospasm/Difficult ventilation)
-***Cardiovascular:*** Recent MI, IHD, Hypertension (Perioperative cardiac event)
-***GI/ Endocrine:*** GORD, Diabetes (Aspiration and wound healing)
--***Renal/Liver:*** Impairment? Renal Replacement? (Drug clearance)
- ***Neuro:*** Prev CVA, Myasthenia (Muscle relaxants, Stroke risk)
-***MSK:*** RA, Arthritis, Ankylosing Spondylitis (Positioning and airway)

The Pre-Operative Assessment: Fit for Anaesthesia?

Having a general anaesthetic and surgical procedure is a major physiological insult. There is a necessity to assess perioperative risk of morbidity/mortality.

Exercise Tolerance tells us about a patient's ability to tolerate anaesthesia and the physiological stress of surgery. A crude method of quantifying exercise tolerance is using metabolic equivalents (METS). This represents a multiplier of basal activity.

METs	Example activity
1 – 4 METS	Can walk indoors
4 – 7 METS	Climb a flight of stairs or walk up a hill
7 – 10 METS	Moderate activities like golf and bowling
>10 METS	Strenuous sport e.g. swimming, manual labour

The question "Can you walk up a flight of stairs?" is a simple screening tool. A tolerance of **4 METS** or greater is a predictor of better outcomes following major surgery (e.g. lower rates of cardiorespiratory events).

Sophisticated tools for exercise tolerance assessment also exist including risk scores (Dukes, RCRI, NSQuip etc) and functional assessment (6-minute walk, Shuttle test, CPEX). Patients are assessed in specialist pre-assessment clinics when planning for elective major surgery. Perioperative medicine is an anaesthetic subspeciality which focusses on assessing, optimising and advising about these risks.

The Pre-Operative Assessment: ASA Score

The ASA score is a simple rating system which categorises patients into risk groups. Using the score, we can broadly describe the anticipated risk of surgery to the patient. It is also used for patient group categorisation in clinical studies. It helps us communicate surgical risk succinctly. Every patient is designated a score for the pre-operative WHO checklist before surgery commences.

AMERICAN SOCIETY OF ANAESTHESIOLOGISTS (ASA) SCORE

ASA 1: A normal healthy patient

ASA 2: A patient with mild systemic disease (e.g. well controlled asthma)

ASA 3: A patient with severe systemic disease (e.g. angina on exercise)

ASA 4: A patient with severe systemic disease that is constant threat to life (e.g. angina at rest)

ASA 5: A moribund patient who is not expected to survive without the operation (e.g. uncontrolled haemorrhage as from a ruptured Aortic Aneurysm)

ASA 6: A declared brain-dead patient whose organs are being removed for donor purposes

E: An E is added to the status number to designate an emergency operation

The Pre-Operative Assessment: Airway assessment

If the operative plan involves general anaesthesia, sedation or the risk of conversion to a general anaesthetic an **airway assessment** must be performed. This helps the anaesthetist predict the risk of difficult **ventilation** or **intubation.**

Ventilation describes our ability to move air in and out of the lungs when a patient is asleep. We often resort to bag mask ventilation (BMV) after anaesthetic induction and before securing the airway. **Intubation** describes inserting a tube into the trachea itself.

Anaesthetists support the airway of elective and emergency patients, and this can be more difficult in some patients than others. It is import identify early the patients who might have a difficult airway.

The 'LEMON' Airway assessment method
L= Look externally **(**facial trauma, large incisors, beard or moustache, and large tongue**)** **E**= Evaluate the 3-3-2 rule **(**incisor distance <3 fingerbreadths, hyoid/mental distance <3 fingerbreadths, thyroid-to-mouth distance <2 fingerbreadths**)** **M**= Mallampati score **(**Score ≥3**)** **O**= Obstruction **(**Presence of any condition that could cause an obstructed airway, including presence of foreign bodies**)** **N**= Neck mobility **(**Limited neck mobility**)** Patients with predictors of a difficult airway **()** may be difficult to ventilate, intubate or both. Patients without any predictors can sometimes have an 'unanticipated' difficult airway

The Pre-Operative Assessment: The Mallampati Score

A simple test for the risk of a difficult **intubation** is the Mallampati score

<u>Ask the patient to "Open their mouth and stick out their tongue"</u>

Class 1- Faucial pillars, soft palate and uvula visible

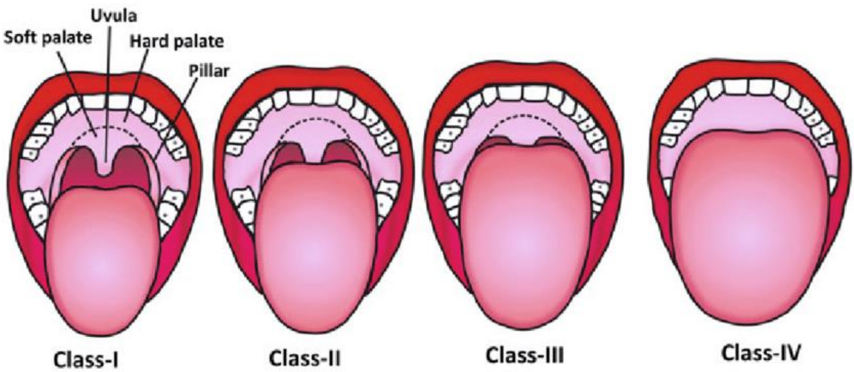
Class 2- Faucial pillars, soft palate visible

Class 3- Soft palate visible only

Class 4- Soft palate not visible

*Class 3 & 4 are associated with increased risk of difficult **laryngoscopy***

This test has a low sensitivity/specificity for predicting difficult airways

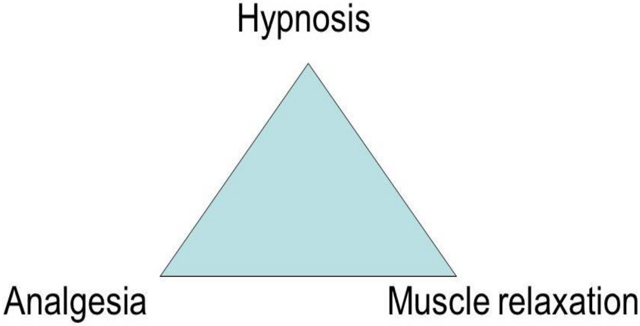


Types of Anaesthetic: General Anaesthesia

It is unclear what mechanism of action facilitates sedation in general anaesthesia. There is no single unifying theory on how general anaesthetics work, however we think that some of these drugs have selective blocking effects on several CNS proteins and ion channels.

In general, sedatives cause reversible loss of conscious awareness and some loss of response to noxious stimuli (anti-nociceptive effect). We call this loss of awareness a **"hypnotic"** effect. Sedation does not reduce response to pain effectively in isolation, so adjunctive **analgesia** is used to reduce sedation requirements and facilitate surgery. **Muscle relaxants** are used in some operations to facilitate surgical and intubation conditions. A combination of these techniques is a ***"balanced anaesthetic***". In theory, any single element could be used to facilitate surgery alone, but with dangerous consequences for the patient's physiology or wellbeing. The "art" of general anaesthesia is to titrate these elements carefully and minimise the detrimental effects.

The "triad" of anaesthesia describes the balance of producing ideal operating conditions.



General Anaesthesia: The Induction

The transition to unconsciousness in general anaesthesia is a critical time. **Induction agents** are hypnotics given to commence sedation, the point at which the patient “falls asleep”. There are several choices, with varying profiles.

DRUG	SYSTEMIC EFFECTS	OTHER POINTS
Propofol	Common induction agent. Lipid suspension with ‘milk like’ appearance. Induces apnoea & depressed laryngeal reflexes	Causes pain on injection. Causes hypotension. May have anti-emetic properties. Can be used for sedation
Thiopentone	Traditional ‘RSI’ agent. Induces anaesthesia in one ‘arm brain cycle’. Not useful for LMA insertion.	Anticonvulsant properties. Strong alkali which damages tissue
Ketamine	Causes a ‘dissociative’ anaesthetic effect where consciousness is lost by alteration in perception	Cardiostable. Can be given Intramuscularly. Hallucinations are common. Analgesic effects. Used in Trauma anaesthesia
Midazolam	Benzodiazepine used in day case procedures. Induces loss/impairment of consciousness through GABA potentiation	Anticonvulsant properties Sedative ‘hangover’ effects. Amnesic effects. Risk of airway obstruction.
Volatile Anaesthesia	The original method of general anaesthesia. Slower onset when used as an induction agent. Poorly tolerated due to pungence and ‘excitation’ effects.	Bronchodilator and vasodilator Used in Paediatrics for “gas inductions”

Rapid Sequence Induction (RSI)

This is performed when there is risk of aspiration of gastric contents (e.g. abdominal operations, un-starved patients, gastro-oesophageal reflux disease). We conduct intubation with pressure on the cricoid cartilage (to obstruct the oesophagus) without facemask ventilation. The aim is to intubate the trachea as quickly and safely as possible. The ‘classical RSI’ used cricoid pressure, Thiopentone and Suxamethonium. Evidence varies regarding its efficacy. In modern practice there are several approaches to perform a ‘modified RSI’.

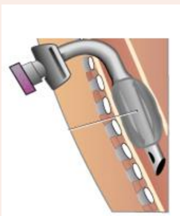
Airway Control: Manoeuvres and Adjuncts

Following anaesthetic induction, the patient's airway relaxes, and the strong opioids and muscle relaxants used often stop the patient ventilating. We then take control of the of ventilation and maintain a patent airway

Initial use of simple manoeuvres (**chin lift, head tilt, jaw thrust**) opens the airway. We can then commence 'bag and mask' ventilation. If this is difficult, we can use airway adjuncts to assist us.

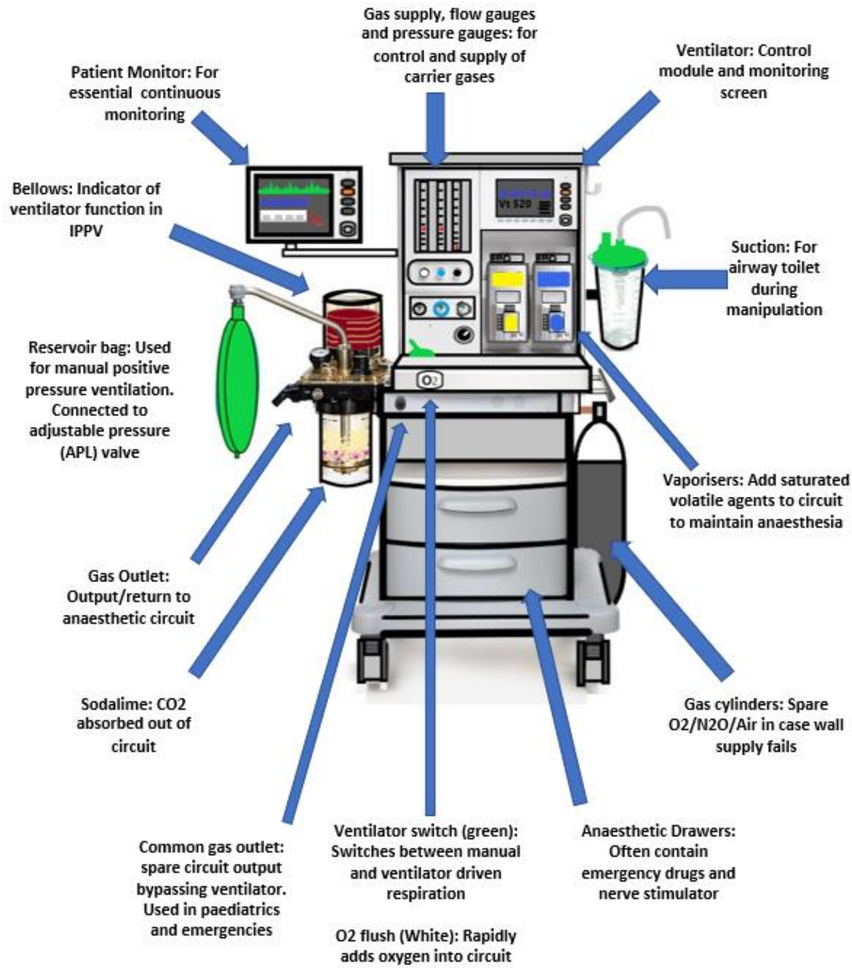
AIRWAY ADJUNCT	COMMENTS
Oropharyngeal Airway (Guedel) 	This is a rigid plastic curved device which prevents the tongue from blocking the airway. It is sized by measuring from incisors to the angle of the jaw. It is inserted upside down in adults, hitting the hard palate. This avoids catching the tongue, it is then twisted through 180 degrees before it is fully inserted. It is only tolerated if gag reflex is absent. It is used to facilitate bag and mask ventilation. It does **not** protect the airway from aspiration.
Nasopharyngeal Airway 	The NP airway is inserted through the nose to open the airway to the pharynx. It has a flanged end to prevent inhalation or swallowing of the device. Older devices had a safety pin through the flange. It is most often used in spontaneously ventilating patients with reduced GCS. It is often (not always!) avoided where there is risk of basal skull fracture.
Supraglottic Airway Devices (LMA) 	The laryngeal mask airway (LMA) has led to a revolution in airway management. These are inserted via the mouth to sit above the larynx. It has a tip which partially obstructs the oesophagus whilst the mask sits over the laryngeal inlet. Muscle relaxants are not routinely required. Multiple 'second generation' Laryngeal masks are available. These have gastric ports and integrated bite blocks (E.g. Proseal, Auragain, or I-Gel). Many operations are done using these devices alone, reducing airway manipulation and shortening recovery times.

Airway Control: The Definitive Airway

AIRWAY INTERVENTION	COMMENTS
Endotracheal Intubation 	The placement of a tube through the vocal cords to facilitate ventilation. With an inflated cuff this is the standard ‘secured airway’. Tracheal intubation most often requires muscle relaxation and direct or indirect laryngoscopy. Tubes are commonly inserted orally but can be inserted nasally when surgeons need access to the mouth. **Indications for Emergency Intubation include:** • Apnoea • Respiratory failure • Airway protection: GCS <8 • Airway obstruction • Haemodynamic instability or cardiac arrest
Front of Neck Access 	Front of neck access allows us to access the trachea whilst avoiding the naso/oropharynx. In an ‘airway emergency’ front of neck access is appropriate management of a patient we ‘Can’t Intubate, Can’t ventilate/Oxygenate’ (CICO) following the induction of anaesthesia. This is done via the cricothyroid membrane, a ‘cricothyroidotomy’. An ‘elective tracheostomy’ is used when weaning intensive care patients or for those with facial tumours. These can be for short term weaning or long-term airway maintenance.

General Anaesthesia: The Anaesthetic Machine

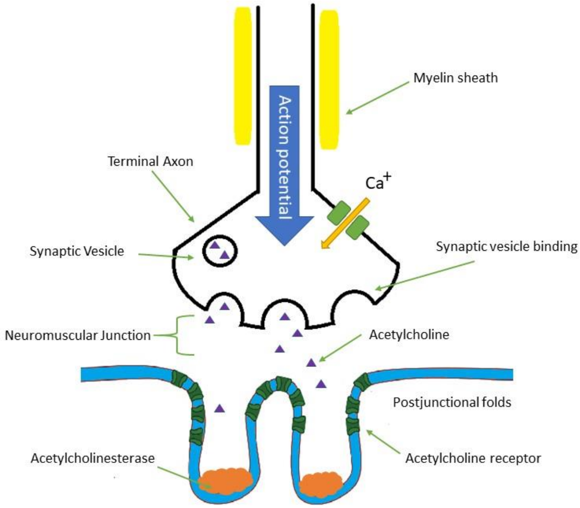
The anaesthetic machine can seem daunting at first. Here is a simple guide to a traditional machine.



General Anaesthesia: Muscle relaxants

Muscle relaxants are often used to intubate a patient's trachea. They reduce muscle tone in the head and neck and allow easy manipulation of the jaw and access to the laryngeal inlet. They are also used to relax muscles for specific operations, improving surgical access and conditions.

They interfere with the action of acetylcholine at the neuromuscular junction (NMJ). They are powerful drugs, related to the alkaloid curare.



They can be categorised into **two** groups:

DEPOLARISING muscle relaxants (e.g. Suxamethonium) These bind to the Ach receptor at the NMJ, initially triggering muscle 'fasciculations'. Suxamethonium is fast acting and used as an **emergency drug**

NON-DEPOLARISING muscle relaxants (e.g. Atracurium, Vecuronium, Pancuroniun or Rocuronium) reduce Ach activity through competitive inhibition.

General Anaesthesia: Maintenance

After an anaesthetic induction, the patient is kept asleep for the operation using volatile anaesthesia or intravenous maintenance.

Inhalational agents are the most common means of maintenance. These ‘volatiles’ are halogenated ethers; we know surprisingly little about how they work! Early inhalational agents included nitrous oxide and chloroform. They are kept in **vaporisers** which consist of a vaporising chamber containing the liquid anaesthetic. The air in this chamber becomes saturated and is combined with the bypassing flow of gas delivered to the patient.

Isoflurane, Sevoflurane and Desflurane are commonly used in modern practice. They are targeted towards a measured **Mean Alveolar concentration (MAC).** One MAC is “the concentration needed to suppress purposeful movement in response to a ‘standard surgical stimulus’ in 50% of subjects”. The measured end tidal volatile concentration reflects a MAC value corresponding to a patient’s sedation.

An alternative form of maintenance is **Total Intravenous Anaesthesia (TIVA),** this often uses Propofol for sedation and Remifentanil for analgesia. This technique uses mathematical models from population studies to calculate an appropriate infusion rate for the anaesthetic agents. As nothing is measured, supplementary modified EEG analysis (e.g. BIS) is used to gauge how asleep the patient is

Equipment: Basic Monitoring

The monitors record a vast array of patient telemetry. Basic monitoring is standardized and improves patient safety.

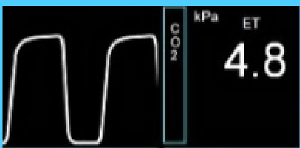
Device	Description	Problems/Pitfalls
Pulse Oximetry 	Oxygen saturation is estimated by measuring transmission of infra-red light through pulsatile vascular tissue beds (e.g. the finger or earlobe). This gives a value SpO2 in %. Normal oxygen saturations are above 95% in healthy, non-smoking, individuals.	Inaccurate/difficult readings with poor perfusion/ cold hands/nail polish/carbon monoxide poisoning & methaemoglobinaemia
Non-Invasive Blood Pressure (NIBP) 	Cuff inflates above systolic pressure and then deflates. Return of blood flow causes oscillation in the cuff which is sensed by a transducer. The resultant values are displayed as the blood pressure. This can be cycled to occur during the operation at fixed intervals.	If cuff too small: Over-reads If cuff too large: Under-reads Can damage fragile skin. Prone to artefact from external compression. Intermittent/non-continuous technique
ECG 	Used to determine heart rate, ischaemia & arrhythmias. Usually three leads are placed on the patient's chest or upper limbs/trunk.	Difficult to interpret subtle changes in waveforms. Interference occurs with movement/diathermy

Equipment: Airway Monitoring

All patients undergoing ventilatory support require airway monitoring.

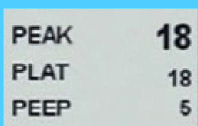
<table>
<tr><th>Parameter</th><th>Measurement tool</th><th>Considerations/Pitfalls</th></tr>
<tr><td rowspan="4">Airway Gases

(CO2, O2, Vapour)


</td><td colspan="2">A sensor continually samples and measures the quantity of certain gases in the breathing circuit. This is often via a small 'side stream' tube connected to the circuit.</td></tr>
<tr><td>End tidal CO2
(Capnography)</td><td>The gold standard marker of airway patency. Is not always representative of arterial CO2. Affected by V/Q mismatch.</td></tr>
<tr><td>Oxygen concentration

(Inspired/expired)</td><td>Important for titrating inspired O2. Affected by water vapour and requires water trap.</td></tr>
<tr><td>Anaesthetic vapour concentration

(Inspired/Expired/MAC)</td><td>Used to titrate sedation in volatile anaesthesia. Water vapour can cause interference.</td></tr>
<tr><td>Airway Pressures


</td><td colspan="2">The anaesthetic machine contains a ventilator which controls delivery of gases to and from the patient's lungs during positive pressure ventilation. Low airway pressures may indicate a leak or disconnection. High airway pressures suggest obstruction, bronchospasm or inadequate relaxation.</td></tr>
</table>

Equipment: Invasive monitoring

Patients who are physiologically unstable, critically ill or require rapid manipulation in their blood pressure often have central venous and arterial access. These devices allow careful titration of vasoactive medications.

Device	Description	Problems/pitfalls
Intravascular IBP **(Arterial line)** 	A cannula is inserted into an artery to give “beat-to-beat” blood pressure values. Blood samples and ABGs can also be taken at regular intervals. Some ‘cardiac output’ monitors can use the arterial line to estimate preload, after-load and contractility.	Placement can damage the artery causing pseudo-aneurysms or can result in a necrotic distal limb. Requires specialist nursing knowledge and transducer sets to be assembled. Medications can inadvertently be given and can damage tissue. Can be affected by over and under damping.
CVC (Central venous catheter) 	A cannula is inserted into a central vein (commonly the internal jugular, femoral or subclavian). To measure central pressure (CVP) it must reach the superior vena cava. The lines are more often used for giving vasoactive and venotoxic medication. Large lines can be inserted for dialysis (‘vascaths’) or rapid blood infusion (‘trauma lines’)	Placement can result in pneumothorax, haemothorax, hydrothorax or infection. CVP was commonly used to assess “vascular filling status” but does not give a global marker of cardiac function. It is now considered a crude measure of filling status.

Types of Anaesthetic: Local Anaesthesia

Local anaesthetic (LA) can be used to facilitate minor operations to major surgery and avoids many of the airway and physiological risks of general anaesthesia. They produce temporary blockade of neuronal transmission when applied to a nerve axon. Clinically they produce reversible loss of sensation in a circumscribed area. They are synthetic esters or amides which bind to axonal sodium channels and inhibit their function. Local anaesthesia can be directed towards any nerve tissue in the body. Detailed knowledge of nerve planes and neuro-axial techniques helps the anaesthetist direct the LA to the correct place.

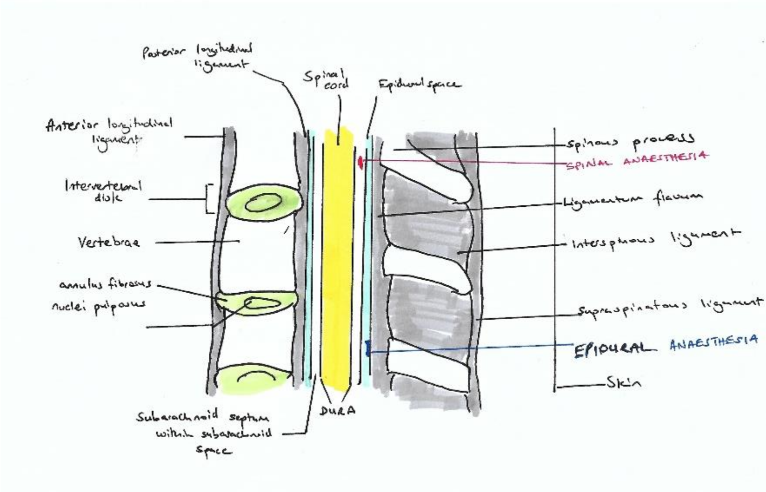
The onset, duration of action and cardiotoxicity of local anaesthesia varies dependent on the choice of drug. LA agents are weak bases, in acidic conditions and infected tissues they become trapped (ionised) away from local nerves and do not function well. This explains why local anaesthesia works poorly when incising abscesses.

Local Anaesthetic	**Max. dose without adrenaline (mg/kg)**	**Max. dose with adrenaline (mg/kg)**
Lidocaine	3	7
Bupivucaine	2	2
Ropivucaine	3	3

Local anaesthetics may be toxic if enough is absorbed systemically. Clinical toxicity appears to relate to the effects of the drug on other excitable membranes in the CNS or heart. There are protocols for managing these ‘local anaesthesia toxicity’ emergencies, they involve infusion of a lipid emulsion (intralipid) which ‘sweeps up’ the drug from the circulation.

Types of Anaesthetic: Neuroaxial Anaesthesia

If local anaesthesia is infiltrated near the cauda equina, epidural space or into the CSF analgesic and physiological effects can be elicited to facilitate surgery.



Spinal: Injection of LA into the subarachnoid (intra-thecal) space (below L2) using a fine bore pencil-point needle. Duration is approximately 2 hours. This block causes sympathetic, motor and sensory blockade. It renders the patient unable to move their lower limbs and provides analgesia. It is commonly used for lower limb surgery, hip and knee arthroplasty and caesarian section.

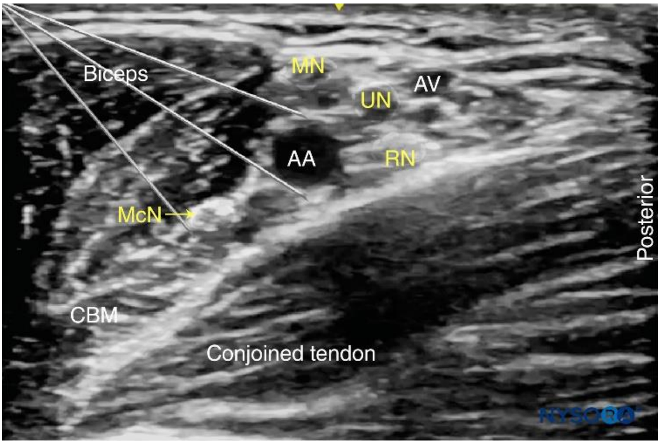
Epidural: A catheter is inserted into the epidural space, allowing either a bolus or infusion if LA for analgesia at roughly the level of insertion. This technique is continuous and can be topped up. Sympathetic and motor blockade is less profound then a spinal. Commonly used for labour analgesia, multiple rib fractures and management of open thoraco-lumbar surgery.

Caudal: Injection of LA into the caudal canal (a low epidural) to block sacral and lumbar nerve roots. Commonly used as analgesia in paediatric patients.

Types of Anaesthetic: Regional Anaesthesia

Local anaesthetic can be injected or infused around a target nerve. It can be directed towards nerve bundles in different ways to facilitate different effects. The advantage being that systemic sedatives and opiates can be avoided/reduced and therefore minimise side-effects and hasten recovery. They also provide great intra-operative analgesia.

The nerves can be targeted using surface anatomy or can be assisted with evoked potentials from a nerve stimulator. The most common way to perform regional blocks in the modern era is to use ultrasound and directly visualize the nerve. Blocks can use high volume into tissue planes or can be directed in small volumes around specific nerves/plexuses.



Ultrasound image of an axillary approach brachial plexus blockade. Median (MN), Ulnar (UN), and Radial (RN) nerves Axillary artery (AA) Musculocutaneous nerve (MCN) coracobrachialis muscle (CBM).

[Image source NYSORA https://www.nysora.com/techniques/upper-extremity/axillary/ ultrasound-guided-axillary-brachial-plexus-block/]

Anaesthetic Drugs: Emergency drugs

Anaesthetic agents can rapidly alter a patient's physiological status. Patients can become hypotensive, bradycardic and unstable. The anaesthetist's job is to maintain a physiological state that ensures adequate organ perfusion. A set of emergency drugs is prepared to assist us in maintaining cardiovascular stability.

ADRENOCEPTOR		
SUBTYPE	**LOCATION**	**EFFECT of activation**
Alpha 1	Smooth muscle	Vasoconstriction
	Heart	Increased duration of contraction
Alpha 2	Pre-synaptic	Sedation and vasodilatation
Beta 1	Heart	Increase heart rate and force of contraction
Beta 2	Smooth muscle	Vasodilation
Beta 3	Fat cells	Glycogenolysis, glycogenolysis, lipolysis

Emergency Drug	Effects
Ephedrine .... mg/ml..	Used to treat hypotension. Releases norepinephrine stores. Alpha and beta agonist. Exhibits tachyphylaxis (reduced response on repeat doses). Chronotropic.
Metaraminol .... mg/ml.	Potent vasoconstrictor used to treat hypotension. Predominantly a direct alpha 1 agonist. Bradycardia via baroreceptor reflex.
Atropine .... mg/ml.	Muscarinic antagonist used to treat bradycardia. Crosses blood brain barrier (BBB). Antimuscarinic side effects.
Glycopyrrolate .... micrograms/ml.	Quaternary ammonium compound used to treat bradycardia. Fewer central side effects than atropine. Does not cross BBB.
Adrenaline .... micrograms/ml.	Used to treat hypotension/anaphylaxis/ cardiac arrest. Potent Beta agonist

Anaesthetic Drugs: Intravenous (IV) fluids

Patients undergoing general anaesthesia are kept ‘nil by mouth’ (**NBM**) and develop a fluid deficit. The operative insult causes additional fluid loss. We try to replete and maintain intravascular volume by giving IV fluid.

We give isotonic fluid to encourage the fluid to stay in the intravascular space. Fluids can be categorised as colloids and crystalloids, however there is no evidence of colloids superiority and they are being used less often.

Crystalloids: Contain a water-soluble crystalline substance capable of diffusion through a semi-permeable membrane (e.g. Hartmanns, Plasmalyte)

Colloids: Contain high osmolality particles in a solvent (e.g. Gelofusin, Starches)

Blood products: Packed red cells, Fresh Frozen Plasma, Platelets,

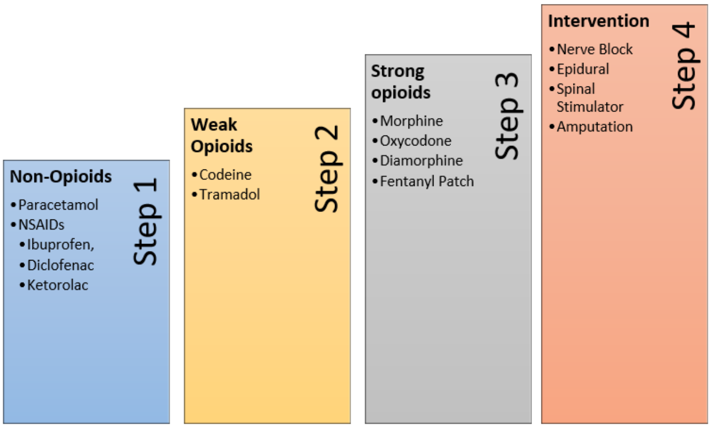
Cryoprecipitate, Human Albumin Solution etc.

In addition to fluid deficit of starvation, patients can bleed intraoperatively and develop **hypovolaemic shock.** There were traditionally four classes of haemorrhagic shock, these were derived from patient observations.

Shock Class	% Loss	Blood loss (ml)	Heart Rate	Blood Pressure	Pulse Pressure	Respiratory Rate	Mental State
I	15	<700	<100	Normal	Normal	14-20	Anxious
II	15-30	750-1500	100-120	Normal	Narrow	20-30	Anxious
III	30-40	1500-2000	120-140	Decreased	Narrow	>30	Confused
IV	>40	>2000	>140	Decreased	Narrow	>35	Lethargic

Anaesthetic Drugs: Analgesics

Intra-operative analgesia reduces anaesthesia requirements and helps with post-operative recovery. Severe post-operative pain and the stress response to surgery cause increase morbidity and mortality. Use of different drug classes is called **multimodal analgesia** and often has a **synergistic effect**. Analgesia is tailored to meet specific patient needs depending on current drug/allergy history and the type of operation. The modified WHO analgesic ladder describes a cumulative method of initiating analgesic therapy



DRUG	Mechanism of Action
Paracetamol	Antagonise COX 3
NSAIDS	Antagonise COX 1 and COX 2 in the arachadonic acid pathway and subsequently reducing prostaglandin
Ketamine	NMDA receptor antagonist
Local anaesthetics	Various methods proposed. Thought to antagonise sodium channel receptors

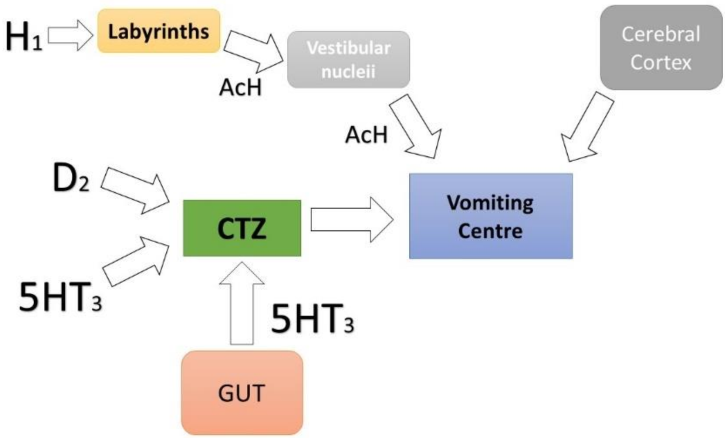
Anaesthetic Drugs: Opioid Analgesia

Opioids are frequently used by anaesthetists for induction/airway management and for intraoperative and postoperative pain. They are derived from the opium poppy. Modern agents are natural or synthetic substances which have an affinity for opioid receptors. Short acting opioids are used for intermittent periods of airway or surgical stimulation. Intermediate and longer acting opioids also reduce post-operative pain. As well as analgesic effects longer acting opiates have a list of common adverse side effects.

Body system	Adverse effects
Central Nervous System	Sedation, Euphoria, Confusion, Dysphoria, Hallucinations, Dependence, Tolerance.
Respiratory	Respiratory depression and arrest. Cough suppression.
Gastrointestinal/urinary	Nausea and vomiting, constipation, urinary retention, delayed gastric emptying.
Cardiac	Hypotension, bradycardia, vasodilation
Endocrine	Release of ACTH, prolactin and gonadotrophic hormone is inhibited
Ocular	Meiosis (pupillary constriction)
Musculoskeletal	Large doses may occasionally produce generalised muscle rigidity especially of the thoracic wall, this interferes with ventilation.
Pregnancy and Neonatal effects	Can cross the placenta and cause neonatal respiratory depression.

Anaesthetic Drugs: Anti-emetics

Anaesthetic agents and surgery can trigger post-operative nausea and vomiting. The morbidity is often underappreciated, but it is extremely unpleasant and can impair surgical results. Nausea is complex but emesis is mediated by the vomiting centre in the brainstem. Antiemetics block the stimulatory pathways involved.



Anatomical site	Receptors and Drugs
Vestibular nuclei and the labyrinth	**Histamine (H1):** Cyclizine, Promethazine **Muscarinic ACh (M3):** Hyoscine, Atropine, Glycopyrrolate
Gut wall and mesenteric plexus	**Serotonin (5-HT3):** Ondansetron
Vomiting centre	**ACh (M3) and some Histamine (H1)**
Chemoreceptor trigger zone (CTZ)	**Dopamine(D2):** Metoclopramide, Domperidone, Prochlorperazine, Haloperidol **Serotonin (5-HT3)**

Anaesthetic Drugs: Anti-microbials

Many surgical procedures require antibiotic prophylaxis. All hospitals have specific microbiology protocols. All doctors in every speciality have a duty of **antibiotic stewardship** – a co-ordinated programme of appropriate prescribing and use of antimicrobials to reduce the spread of multi-drug resistant organisms.

Asking about the allergy status is vital and antibiotics should be administered before knife to skin or application of tourniquet. Dosing and drug choice should consider renal and hepatic function, immunocompromised state, and concomitant therapies.

Cell wall synthesis inhibition	Protein synthesis inhibition	Nucleic acid synthesis inhibition	Cell membrane function inhibition	Uncertain mechanism
Beta-lactams* Penicillins, cephalosporins, carbapenems **Glycopeptides*** Vancomycin **Polypetides*** Bacotrecin	**30S** Aminoglycosides*, tetracyclines **50S** Macrolides, lincosamide, oxazolidinone	**Folic Acid synthesis inhibitors** Sulphonamides, trimethoprim* **RNA polymerase inhibition*** Rifampicin **DNA structural disruption*** Metronidazole **Topo-isomerase targeting*** Norfloxacin, ciprofloxacin	Amphotericin B*, Nystatin*	Isoniazid*, Ethambutol

Surgical Safety: The WHO checklist

We are constantly striving to have the safest operating theatres possible. Mistakes do happen in theatres, there have been times where the wrong operation was performed, surgery occurred on the wrong side or the wrong patient has been operated on. These are now called **"Never Events"** as they are preventable by good practice.

The World Health Organisation (WHO) introduced 5 steps of safer surgery. Since inception there has been a reduction in surgical and anaesthetic complications. This has several defined elements.

<u>Briefing:</u> Introduce the team, discuss the patients including any special requirements and the order of the list.

<u>Sign in:</u> A pause to identify any potential problems of the case on arrival to theatre/anaesthetic room.

<u>Time out:</u> A final check before the operation is commenced.

<u>Sign Out:</u> At the end of the list to identify any problems during the case.

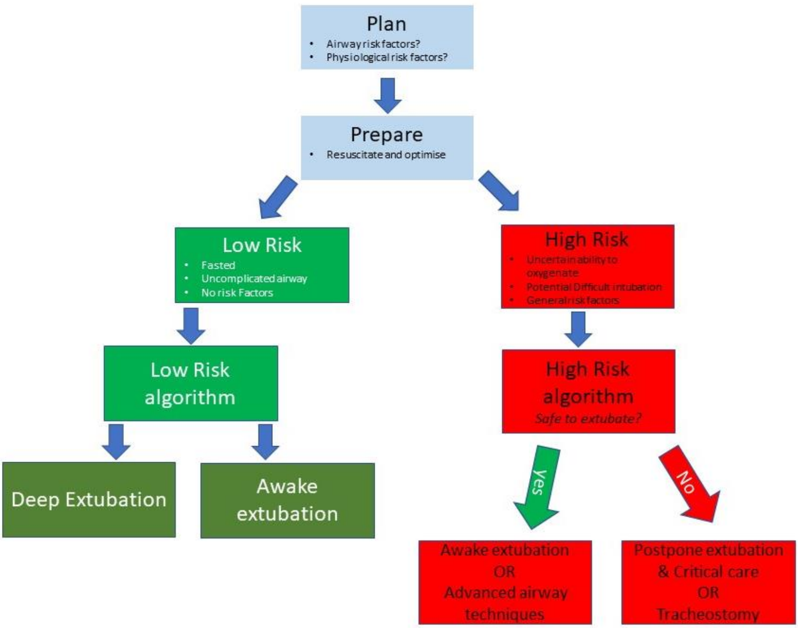
National Safety Standard for Invasive Procedures (**NatSIPPS**) have been developed to set out the key steps necessary to deliver safe care for patients undergoing invasive procedures and allows organisations to standardise the processes that underpin patient safety. These can be adapted for local practices (**LocSIPPS**) and trusts are encouraged to have these safety checklists in place.

General Anaesthesia: Emergence and Extubation

Extubation and emergence phases are critical as induction with the potential for serious complications. Each anaesthetist will have their own planning techniques however some basic universal principles apply.

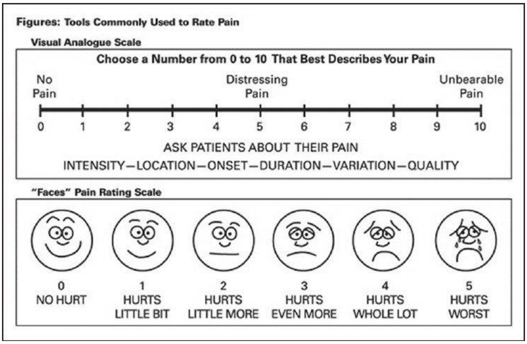
First, we should ensure the operation is finished, wounds are dressed, swab counts are performed, and we are ready to sign out. The patient should have received adequate analgesia and antiemetics. Any residual neuromuscular blockade should be tested for and reversed if necessary. The patient should be haemodynamically stable and adequately resuscitated, they should be pre-oxygenated and positioned appropriately.

Maintenance agents are metabolised or excreted. The volatile anaesthetics are exhaled, and intravenous agents are metabolically cleared. The process of emergence begins when the maintenance agent is stopped. An extubation plan can be made based on difficult airway society (DAS) guidelines (below).



Post-Operative Pain: Recognise, Assess and Treat (RAT)

How do we manage the patient with post-operative pain? Assessment of these patients can be tricky. Post-operative pain limits chest physiotherapy and mobility so can cause significant morbidity and even mortality. The **RAT** approach is a way of managing a patient’s pain.



Recognise: *Is the patient in pain?*

- Ask
- Look for features of pain (frowning, moving easily, sweating?)
- Are there any 'pain behaviours' occurring? (agitation, anger, confusion, anxiety)

Assess: *Severity and nature*

- Measure severity: Verbal rating scale/Numerical rating scale/ Visual scales
- Assess type: Acute, Chronic, Nociceptive or Neuropathic?
- Consider Other factors: Physical, psychological or social factors influencing pain

Treat (and Reassess!)

- Treat the underlying cause if possible!
- Non-drug and drug treatments are equally important
- Pain is a **bio-psycho-social** phenomenon. This means we must often take a multimodal and multifactorial approach to treat it effectively.

Critical Care: Identifying the sick patient

Unwell patients usually have deranged physiology (e.g. tachypnoea, tachycardia and hypotension) as a result of their illness. There may be a trend of worsening physiological observations. **Early Warning Scores (EWS)** can be used to quantify the extent of physiological deterioration. It is vital to recognize the deteriorating patient, to provide resuscitation and escalate concerns.

NEWS2 (national early warning system 2) is the latest EWS measuring: Respiratory rare, oxygen saturation, systolic BP, pulse rate, level of consciousness or new confusion and temperature. It has two oxygen scales to select from to accommodate for COPD patients.

Outreach teams are usually multidisciplinary, led by senior ITU nurses. They can help to improve care by:

- Identifying the deteriorating patient using EWS or clinical concern and responding to calls for help with those patients
- Facilitating timely admission to critical care
- Providing practical and educational support to ward doctors and nurses
- Providing continuity of care and follow-up for patients discharged from critical care to a general ward

Scoring systems have been developed to quantify the severity of illness of ICU patients and as a guide to the level of therapeutic intervention they may require. They are commonly derived from the degree of acute physiological derangement, comorbid status and admitting diagnosis. Examples include APACHE, SOFA and SAPS.

Critical Care: Organ failure & support

Signs of organ failure	Treatment
Respiratory failure Type I: hypoxia Type II: hypercapnia	Non-invasive or invasive ventilation to recruit under-aerated lung and Increase minute volume Secretion clearance (chest physiotherapy, suctioning). Extra-corporeal carbon dioxide removal or membrane oxygenation (ECMO).
Cardiovascular failure Dysrhythmia Hypotension due to vasoplegia (low peripheral vascular resistance) Poor organ perfusion due to poor cardiac contractility (low cardiac output)	Anti-arrhythmic drugs; cardiac pacing. Vasopressor drugs (e.g. noradrenaline). Inotropic drugs; mechanical devices (e.g. Intra-aortic balloon counter pulsation, ventricular assist devices, ECMO).
Renal failure (aka Acute Kidney Injury) Low urine output; rising uncleared metabolites	Continuous Veno-venous haemofiltraion (CVVHF).
Gastrointestinal and Hepatic failure Poor absorption of nutrition Coagulopathy	Total parenteral nutrition (TPN). Clotting products
Central Nervous System Acute confusion Peri neurosurgical intervention	Treating and monitoring intra-cranial pressure; extra-ventricular or lumbar drains

Critical care therapies exist to manage organ failure until disease control is achieved. When an organ exceeds its physiological capacity, it is deemed to be ‘in failure’ other organs are co-dependent and subsequently fail leading to multiorgan failure (MOF) and death.

Critical Care: What is Critical Care for?

Critical care offers treatments which are not always available on the wards:

- **Nursing:** The biggest benefit to patients is from the increased nursing to patient ratio (1:1 on ITU and 1:2 on HDU)
- **Organ Support:** Critical care can provide more complex modalities of organ support than a general ward.
- **Sedation/Intubation/Ventilation:** Sedation may sometimes be therapeutic but is often used to facilitate delivery of complex care in a dignified and tolerable way.
- **Complex monitoring:** This includes arterial lines, allowing us to take repeated Arterial Blood Gases easily and with minimal trauma, CVP monitoring and continuous ECG monitoring.
- **Deliver carefully monitored medication:** Some of the drugs given in ITU are difficult to deliver safely on the ward. Examples include concentrated potassium solutions and vasoactive drugs which need judicious monitoring and delivery through central access.
- **Peri-operative optimization:** Some patients with chronic disease are admitted to critical care due to high morbidity and mortality risk following major surgery. Some types of surgery require close and careful monitoring of the operative site (e.g. revascularized ischaemic limbs).

Because of critical illness, but also because of some of our interventions, critical care patients are at risk of a range of complications. These include delirium, ventilator induced lung injury, ventilator associated pneumonia, critical illness polyneuropathy and myopathy, venous thrombo-embolism, pressure ulcers and acute haemorrhagic gastritis. It is not an easy or simple decision to admit a patient to critical care and it should involve a senior multidisciplinary approach.

Critical Care: Making a referral?

If your patient is sick enough to need referral to critical care, then you should already have involved your senior. Critical care is often a traumatic and difficult place for a patient to go, carefully considered decision making prevents inappropriate admissions and unnecessary suffering. Experience and data tell us that patients need a certain level of physiological reserve to survive critical care. It is also important to make a *meaningful* recovery. Referrals are usually the remit of senior staff.

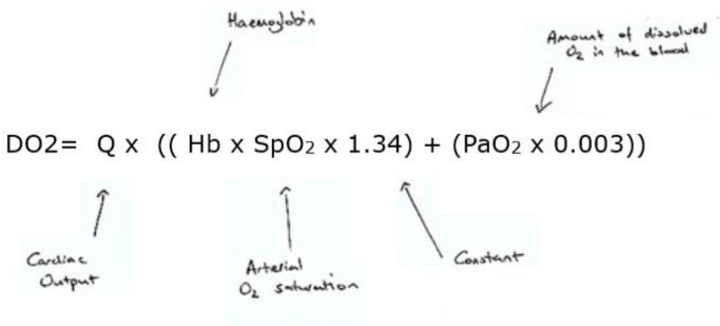
If you are communicating information about complex or unwell patients, using the **SBAR** tool can help you structure your conversation. You may find it to be a useful framework for referring between specialities.

Situation	Introduce yourself I have a sick patient! Name. Age. The main problem is… (e.g. hypoxia, hypotension despite fluid resuscitation, a high work of breathing, decreasing GCS etc.)
Background	Presenting complaint, length of time in hospital, Important PMH. Recent events that have led to this situation.
Assessment	'A to E' assessment findings Only list abnormal findings and any interventions performed
Recommendation	I would appreciate it if you could review this patient. Do you have any suggestions for anything I should do in the meantime?

A substantial proportion of patients in critical care die. Thus, a further role for the specialty is the provision of good acute palliative care and support for friends/relatives. **Organ donation** is also an important critical care responsibility. In care of the dying patient we can support and counsel families if their relative is a suitable candidate for donation and expressed pre-morbid wishes to donate.

Physiology and Anaesthesia: Oxygen Delivery

A key physiological target during anaesthesia is Oxygen delivery (DO2). Maintaining aerobic respiration in tissues prevents the onset of organ dysfunction. This can be summarised in an equation:



The equation above describes how blood oxygen delivery is dependent on perfusion and oxygen carriage.

We can optimise these factors perioperatively:

Oxygen content (CaO2)

• Maintain an adequate Hb and replace lost blood by transfusion if required**.**

• Aim for high oxygen saturations, providing supplemental oxygen when needed

Cardiac Output (Q)

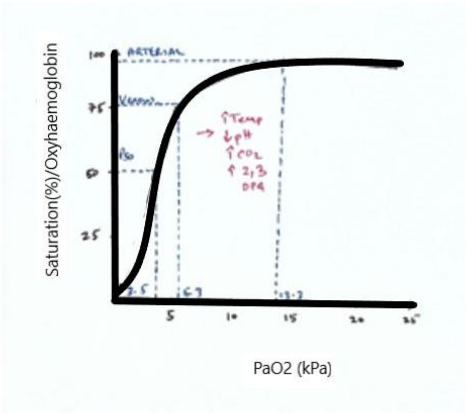
• Optimise heart rate and rhythm (treat arrhythmias).

• Optimise stroke volume (preload and contractility-fluid and inotropes).

• Maintain perfusion pressure to ensure oxygen delivery (afterload-vasopressors)

Oxygen Content (CaO_2): SaO_2 and Hypoxia

An important physiological concept is the sigmoid oxyhaemoglobin saturation curve. A 1% fall in SaO_2 at 90% is much more significant than a 5% drop at 100%. The curve illustrates this concept.



It is a sigmoid shaped curved due to allosteric modulation and co-operative binding of Hb. As Hb binds oxygen it changes structure and it becomes easier for it to bind further oxygen. When saturations drop below 90%, we enter the linear portion of the curve where oxygen partial pressures fall rapidly.

When cells do not receive adequate oxygenation, they undergo hypoxia. There are 4 types of hypoxia.

1. **Hypoxic hypoxia** PaO_2 <12kPa. Causes include low FiO_2, Hypoventilation (opiates or benzodiazepines), Increased A-a gradient or V/Q mismatch
2. **Anaemic hypoxia** Normal PaO_2 but inadequate oxygen carrying capacity in the blood
3. **Stagnant hypoxia** Normal PaO_2 and oxygen carrying capacity in the blood, but reduced tissue and organ perfusion e.g. reduced cardiac output
4. **Histotoxic hypoxia** Inability of the tissues to utilise oxygen at a cellular mitochondrial level. Occurs in sepsis and with toxins e.g. cyanide

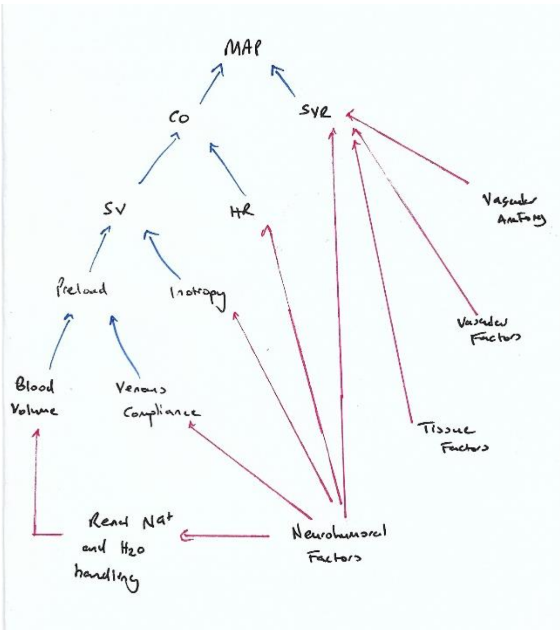
Cardiac Output (Q): Maintaining perfusion

Cardiac output needs to be maintained to ensure an adequate perfusion pressure to organs and prevent organ dysfunction from developing. Two equations help us understand the physiology of perfusion pressure:

Blood Pressure= Systemic vascular resistance (SVR) x Cardiac Output

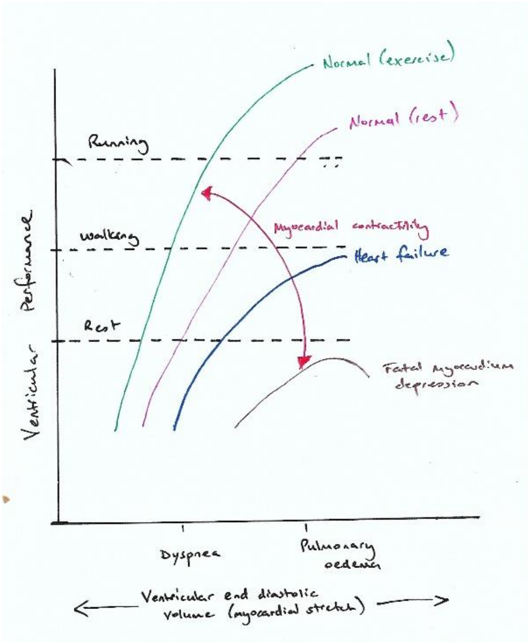
Cardiac Output= SV (stroke volume) x HR (heart rate)

A reasonable Mean Arterial Production (MAP) is required to perfuse our brains and other vital organs. We aim to keep MAP above 65mmHg in normotensive patients. We can manipulate vascular resistance, heart rate, stroke volume and contractility using IV fluids and vasoactive/inotropic drugs.



Contractility: Maintaining perfusion

Cardiac muscle can alter its contractile strength based on load applied. This is called the Frank-Starling Relationship.



This relationship describes how the strength of cardiac contraction (contractility) is dependent upon the initial muscle fibre length in the left ventricle.

We can increase the initial fibre length by giving IV fluids (increasing the preload). This is akin to the ventricular end-diastolic volume LVEDP.

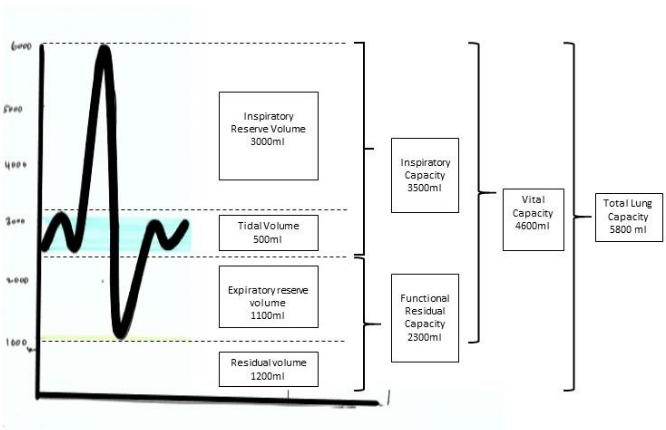
If excessive fluids are given, the heart cannot generate proportional inotropy. The curve reaches a plateau and the heart goes into failure.

Respiratory Mechanics: Volumes and Ventilation

Why do anaesthetists pre-oxygenate patients? The figure below figure represents the respiratory volumes of a 70kg male.

Functional residual capacity (FRC) represents the largest oxygen (O_2) store in the body. Room air contains 21% O_2, so an FRC of 2400ml contains 504ml O_2. Normal O_2 consumption is approximately 250ml/min, so this store lasts 2 minutes.

If we pre-oxygenate with 100% oxygen the 79% of nitrogen in the FRC is washed out, 2300ml of O_2 increases apnoea time to 9 minutes and 12 seconds. Alveolar O_2 content is much lower, and O_2 consumption can also be higher so this time is usually an overestimate. Longer apnoeic time helps in securing the airway with a reduced risk of hypoxia.



Patients are frequently ventilated using positive pressure (PPV). Lung injury is caused if patients are over-ventilated (barotrauma/volutrauma/ARDs). If the patient is under-ventilated, they can become hypercarbic and acidotic. A basic ventilator is either ‘pressure’ or ‘volume’ controlled.

With **pressure control** the ventilator delivers a fixed pressure on every breath, this determines the volume delivered. This causes less barotrauma, but volumes change significantly as lung compliance varies; patients can be **under-ventilated**.

With **Volume control** the ventilator delivers a fixed volume on every breath, this determines the peak pressure. It allows more precise control of CO2 but causes **more barotrauma**. Modern ventilators use a sophisticated mix of both modes to achieve gentle but effective ventilation.

A Career in Anaesthesia?

Anaesthesia is a rewarding and interesting career with many avenues of general and specialist interest. As an anaesthetist there is scope to work in perioperative medicine, critical care, A&E resuscitation, day surgery units, transport of critically ill patients, pain medicine and many other avenues. There is scope for research, management, and teaching roles as part of the job.

Recruitment in the UK occurs after the foundation program. The training lasts for 7 years and consists of ‘core’, ‘intermediate’ and ‘higher’ stages. There are some tricky exams which occur in two stages, the ‘primary’ during core training and the ‘final’ in intermediate training.

The Royal College of Anaesthetists (RCoA) is the governing body for the specialty. They offer both an undergraduate and foundation membership service. The Association of Anaesthetists of Great Britain and Ireland (AAGBI) also offer student memberships. Most medical schools now have anaesthetic/intensive care societies.

If you want to be an anaesthetist consider looking at the ‘person specification’ provided on the RCoA website for recruitment. Anaesthetists are keen to recognize the value of good generic qualities in potential trainees, this includes an interest in the specialty and an enthusiasm for audit, research, learning, teaching, and personal development.

We hope you have found this handbook helpful during your time in perioperative medicine! Hopefully, it will have helped demystify some of the work we do. Having some understanding of the world of anaesthesia can be useful in your everyday life as well as when working as a doctor in any hospital or interventional specialty.

All the best, and good luck!

Publication of this handbook has been supported by the Royal College of Anaesthetists through the Belfast Prize. We thank the College for their contribution towards undergraduate training in anaesthesia!