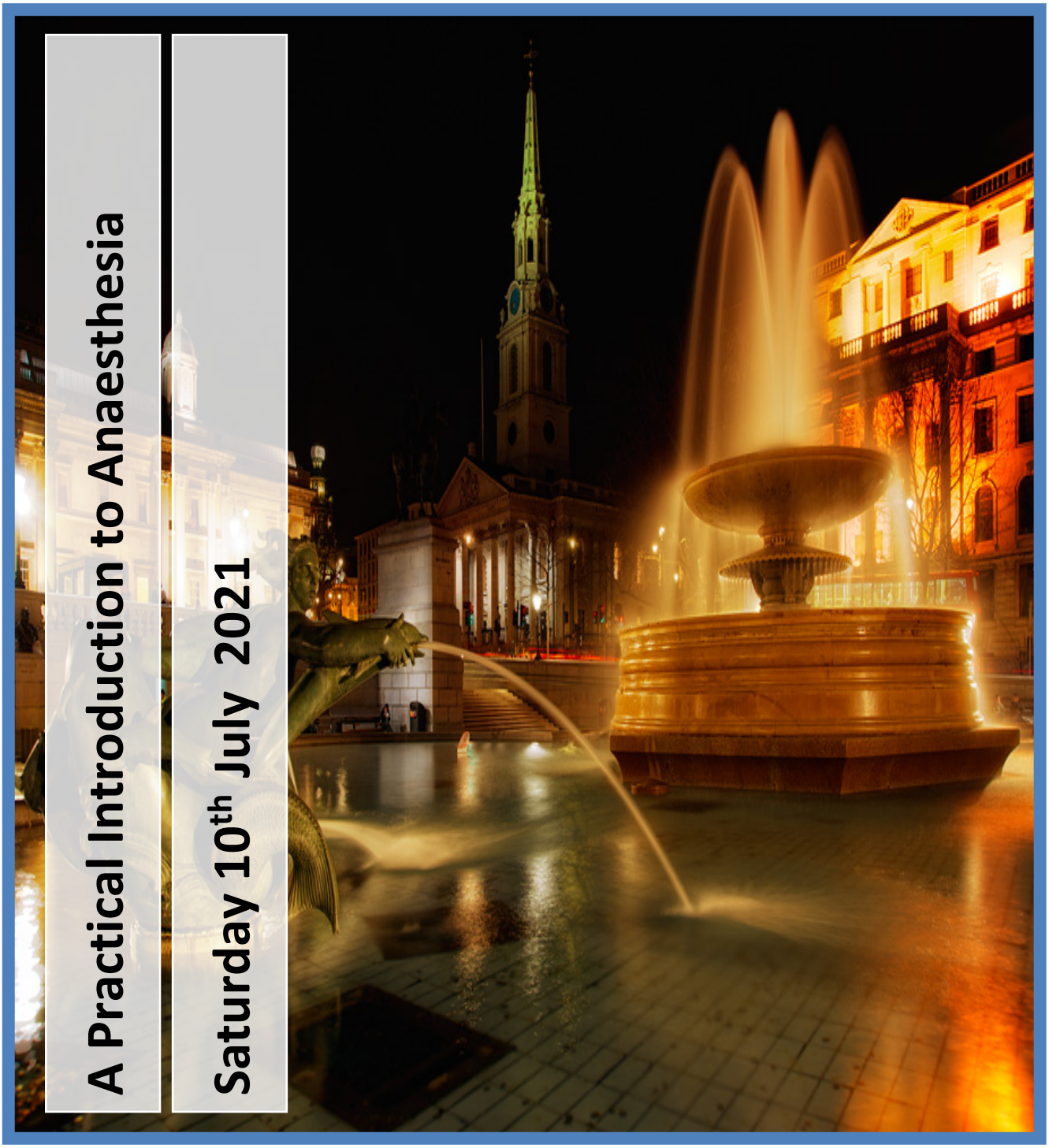
A Practical Introduction to Anaesthesia

Saturday 10th July 2021



UCL CPOM

Centre for Anaesthesia and Perioperative Medicine

The UCLH Education Centre

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www.ucl.ac.uk/anaesthesia/education/AnaesthesiaIntroduction

WELCOME AND INTRODUCTION

Dear Candidate

Welcome to "An Introduction to Anaesthesia". This is intended to be a one day survival guide for those of you new to Anaesthesia, or who want to know more about a career in Anaesthesia.

This handbook is intended to supplement the lectures. As this is the first course for those of you new to Anaesthesia, **we really want your feedback!** Is there anything you'd like in, anything not done well, or anything that was great? In addition coffee and lunch should give you a chance to discuss with the course tutors any questions you might have.

We'd recommend you start reading as soon as you start your post, as it all seems quite new. To start try using the following three books:

Anaesthesia and Intensive Care A to Z: An Encyclopedia of Principles and Practice 3rd Edition (Yentis, Hirsch & Smith)

Drugs in Anaesthesia & Intensive Care (Sasada and Smith)

Respiratory Physiology: The Essentials (John West)

We also think you should sign up to

Join the Association of Anaesthetists of Great Britain and Ireland (http://www.aagbi.org)

e-learning for Anaesthesia (www.e-lfh.org.uk/projects/ela/index.html)

Check out the Royal College of Anaesthetists (www.rcoa.ac.uk) website especially their e-learning site at http://www.rcoa.ac.uk/e-la

Good luck

The Course Directors
Rob, Mo, Hannah and Anita

If you'd like to find out more about our research and educational activities, we would love to hear from you, please contact us via the website: www.ucl.ac.uk/anaesthesia/education/AnaesthesiaIntroduction or Rob via www.ucl.ac.uk/anaesthesia/people/stephens

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THE ABC OF ANAESTHESIA

A PRACTICAL CONDUCT
DR ELISA BERTOJA

Anaesthesia: an-aesthesia from Greek meaning “without sensation”

The practical conduct of anaesthesia is just a brick in the anaesthetic wall.

Anaesthesia starts well before the anaesthetic room. Our role in the patient pathway can be divided in three stages:

PRE OPERATIVE CARE

INTRA OPERATIVE CARE

POST OPERATIVE CARE

INTRA OPERATIVE CARE is only one component of our job. Thanks to our knowledge of anatomy, physiology, pharmacology, physics and years of experience, we can run safe and effective anaesthesia in several different ways. The primary distinction is between:

GENERAL ANAESTHESIA

REGIONAL ANAESTHESIA

GENERAL ANAESTHESIA (GA)

We can divide the conduct of a GA in three main components

INDUCTION

MAINTENANCE

EMERGENCE

INDUCTION

This is the first part of the intra operative time. It usually happens in the anaesthetic room, sometimes in theatre, and in emergencies in A and E or on the Wards. By administering selective drugs we induce a loss of consciousness (hypnosis), which is temporary and reversible. The loss of consciousness can be achieved via intra venous drugs or inhalational agents.

Together with the loss of consciousness comes the loss of the airway, and loss of the airway protective reflexes. Therefore a patient undergoing GA needs an airway device to maintain airway patency (**A**) and to be able to self ventilate or to be ventilated by a machine (**B**).

The drugs used to induce and maintain anaesthesia do have an impact on the patients’ cardiovascular system(**C**). One of our roles is to preserve the patients’ cardiovascular stability.

Inducing anaesthesia means that we willingly take a patient from a self sufficient and safe condition to a state of dependency (on us, our drugs and our machines). This is pretty unique in medicine!

Lack of awareness does not imply lack of response to pain. Just because the patient is asleep does not mean that he/she is pain free. Before surgery can start we need to administer drugs that blunt the body response to pain.

MAINTENANCE

During the maintenance of anaesthesia we have to achieve two goals:

MAINTAIN SAFE AND STABLE PATIENT CONDITIONS

IMPLEMENT CONDITIONS FAVOURABLE TO THE SURGEON

A SAFE AND STABLE PATIENT

During anaesthesia a patient and his/her body do not respond to stimuli as if he/she is awake. On top of that the anaesthetic and surgical procedures implement acute changes to patients' physiology (induction agents, surgical insult, blood loss etc). As Anaesthetists, we are in charge of maintaining patients' homeostasis. For this reason we always use a variety of monitoring devices.

A patient under GA has the following standard monitoring (Association of Anaesthetists of Great Britain and Ireland (AAGB) standard guidelines)

ECG
Pulse oximeter
Blood pressure cuff
Inspiratory and expiratory airway gases, including oxygen (O_2), carbon dioxide (CO_2), and anaesthetic vapours.
Ventilatory parameters : Tidal Volume, Minute ventilation, Airways pressures,
Other devices are available to the expert Anaesthetist during major or specialized surgical procedures (Invasive pressures measurement, Doppler, Arterial Blood Gas) and Depth of anaesthesia monitoring (BI Spectral Index etc).

All procedures and data is documented on the Anaesthetic chart for intraoperative, post-operative, and future references. This is a legal document.

CONDITIONS FAVOURABLE TO THE SURGEON

Anaesthetists and surgeons work as a team for the patients' benefit. We can improve the surgical result by liaising with the surgeon before the beginning of the operation, choosing the appropriate anaesthetic technique (e.g. avoid nasal intubation for a nasal polyp extraction!), interfering with patient physiology to improve surgical conditions. (e.g. Blood pressure, muscle relaxation etc.)

EMERGENCE

The importance of emergence from anaesthesia is frequently underestimated by non-anaesthetists. It is a delicate time of the anesthetic conduit. It can be a dangerous time.

Our main goals during emergence are for the patient to:

- Regain consciousness
- Regain control on his/her airway and ventilation
- Gain satisfactory pain control

It is a challenging task and there is not just one way to achieve it. As Anaesthetists, we gradually hand over the control of the A B C back to the patient. As much as the induction, emergence can be a *grey area,* whereby complications (fail to extubate, sudden increase in BP/ HR etc.) can occur.

COMPLICATIONS OF GENERAL ANAESTHESIA

COMMON

Post Operative Nausea and Vomiting (PONV) (1 in 3)

Sore throat (1 in 4)

Chest infection (1in 5)

Cognitive dysfunction including, confusion and dizziness (1 in 5)

Muscle weakness, Aches and pain

Shivering or Itching

UNCOMMON

Awareness (1.5 in 1,000 – 1 in 42,000)

Teeth and lips damage (1 in 4500)

Breathing difficulties

Nerve Damage

RARE

Death due to Anaesthesia (1 in 185,000)

Serious allergic reaction (1: 10,000- 1:20,000)

Equipment failure

Damage to eyes

SUMMARY

TAKE HOME MESSAGE:

- ✓ **THE ANAESTHETISTS' WORK BEGINS WELL BEFORE THEATRE**
- ✓ **EACH ANAESTHESIA IS TAILORED FOR EACH PATIENT AND PROCEDURE**
- ✓ **WE WORK TOGETHER WITH THE SURGEON**
- ✓ **PATIENT SAFETY IS PARAMOUNT**

A IS FOR AIRWAY

DR ED BURDETT
DR RAVI ALAGAR

One of the consequences of general anaesthesia and deep sedation is that the patient is unable to maintain a patent airway - Anaesthetists are necessarily airway specialists.

In the emergency scenario, the airway always comes first. This is because resuscitation is futile unless you can delivery oxygen to the lungs.

A 'difficult airway' is one in which ventilating an anaesthetized patient with a mask or intubating them is difficult. Airways can be difficult at extubation too.

AIRWAY ASSESSMENT

History: Previous difficult airway

Other disorders (see below)

Examination: not very sensitive or specific

Neck flexion and extension

Mouth opening

Mallampati score (MP) Range 1-4

Thyromental distance

Atlanto-axial range of movement

Investigations: Imaging e.g. CT; flexion/extension cervical spine x-ray

Obstructive lung defects

THE DIFFICULT AIRWAY= DIFFICULT VIEW OR VENTILATION

Beware the following patients:

The multisystem disorders

Bony disorders - rheumatoid arthritis; ankylosing spondylitis

Soft tissue disorders – marfans, acromegaly scleroderma

The airway disorders:

Previous head/neck radiotherapy or surgery

Airway pathology e.g. tonsils, oral abscess, throat cancer

The acutely unwell:

Bowel obstruction – aspiration can be huge

Facial/mouth trauma or burns

Anyone in an unfamiliar area e.g. the ward, radiology etc.

Confused or aggressive patients

The others:

Pregnant women

Children with syndromes

Celebrities

This list is not exhaustive, but you get the idea.

AIRWAY MANAGEMENT

'Managing' a patient's airway is necessary if, the patient cannot maintain a patent airway, they are in respiratory failure, or they require a general anaesthetic. It includes

Supplemental oxygen

Non-invasive Manoeuvres:

Chin lift, jaw thrust

Suction, oral toilet, removal of FB etc.

Invasive manoeuvres:

Supra-glottic airways

a. Oropharyngeal / Nasopharyngeal airway
b. Laryngeal mask airway and similar

Endotracheal

a. Oral/nasal endotracheal tube
b. Tracheostomy

WHEN TO INTUBATE – A DECISION-MAKING FRAMEWORK:

The airway must be managed if:

Severe respiratory/systemic illness

Cannot maintain airway eg neurological (GCS <9)

Surgery requiring GA

Can a supra-laryngeal device (eg. LMA) be used?

Can a supra glottis device be used? Not if

Risk of aspiration e.g. Full stomach/blood in mouth

Surgery requires endotracheal seal e.g. laparotomy

Need for high pressure ventilation e.g. pneumonia

Long term e.g. ICU

Is there a possibility of a difficult facemask ventilation or direct laryngoscopy?

Acute or chronic anatomical abnormalities

Call for senior help, consider advanced technique

Is the stomach full?

Consider Rapid Sequence Induction (RSI)

It is failed oxygenation which is harmful, not failed intubation

NON-TECHNICAL SKILLS OF AIRWAY MANAGEMENT

Airway management is usually technically easy, but like any high-risk task performed under time pressure it can be stressful.

Here are a few ways of making airway management smoother.

Ensure that you and your team are aware of your limitations, both your level of experience and acutely (tired, hungry, unfamiliar with surroundings etc.)

Preparation is everything:

Consider your options - have a plan B and plan C if plan A fails. You should be calling for help before plan C.

Get ready: equipment, drugs, and anything you might need if things don't go well

Check the patient is optimised - best possible oxygenation and position

NG tube if any risk of full stomach

Check oxygen, monitoring, suction, machine and IV access beforehand

Communicate well: this is surprisingly difficult when you're under pressure. Ensure that everyone else in the team is briefed and aware of their roles.

EXTUBATION

What happens after removing your chosen airway device (extubation) can be risky - even more than intubation.

Respiratory (e.g. coughing, laryngospasm, desaturation) and cardiovascular complications (tachycardia, hypertension, arrhythmia) may happen more at extubation than at intubation.

Complication rates during intubation have fallen over recent years due to our use of a systematic approach. However there's now much more focus on optimising the way we extubate.

Consider *Identifying* who is at high risk of problems during extubation?

- Known difficult airway
- Deteriorating airway post-intubation (oedema/bleeding)
- Aspiration risk
- Cardiovascular /respiratory comorbidity

Optimisation

- Patient position
- Equipment ready
- Skilled assistance available- and a senior anaesthetist
- Awake or deep extubation

Handover and monitoring after extubation

The intubation, management and extubation guidelines by **DAS, the Difficult Airway Society** below are useful

https://www.das.uk.com/guidelines/downloads.html

B IS FOR BREATHING

DR IRENE BOURAS

WHY WE BREATHE

We breathe to enable gas exchange ie the transfer of O_2 from the air to the tissues and the removal of CO_2 from the tissues back into the air. The average adult consumes 250mls oxygen per minute and produces 200mls CO_2 per minute. Air contains 21% O_2.

OXYGEN DELIVERY

The delivery of O_2 to the tissues depends on a number of variables

O_2 delivery = arterial blood O_2 content x Hb x cardiac output

A change in any one of these variables will affect the amount of O_2 reaching the tissues

HOW WE BREATHE

There are 3 respiratory control centres in the brain which control and co-ordinate breathing. They are the:

Medullary inspiratory area

The pneumotactic area

The apneustic area

These respiratory centres receive inputs from 3 sets of sensory neurons. The central and peripheral chemoreceptors, which monitor plasma pH and CO_2 and O_2 concentrations and the stretch receptors located in bronchial walls are activated when the lungs are expanded and trigger cessation of inspiration.

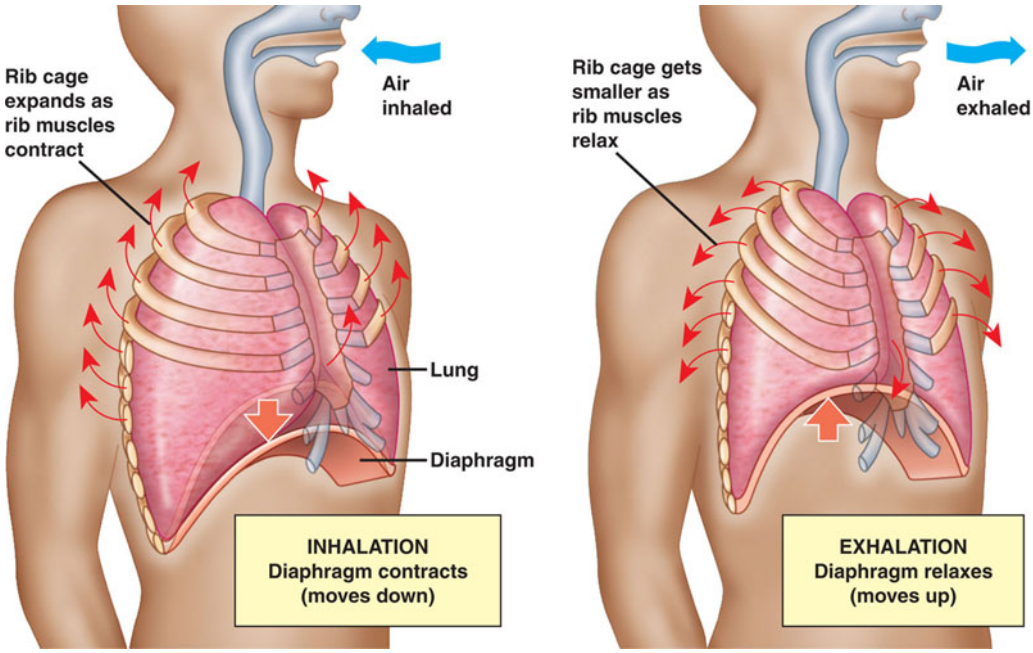
MECHANICS OF BREATHING

Inspiration:

Inspiration occurs when the inspiratory muscles (diaphragm & external intercostals) contract. This elevates the ribs and sternum and causes an increase in the size of the thoracic cavity. The sudden increase in the relative size of the thoracic cavity creates negative intra-thoracic pressure which causes air to move into the chest (from an area of high pressure to an area of lower pressure). Inspiration is an active process i.e. requires energy.

Expiration:

Expiration occurs when the diaphragm and external intercostal muscles relax. This causes the lungs to recoil back to their original volume. The reduction in volume results in a pressure increase within the lungs and expiratory gases being expelled from the lungs. Under resting conditions expiration is an entirely passive process. However, during more laboured breathing the intercostal and abdominal muscles are used which requires energy.

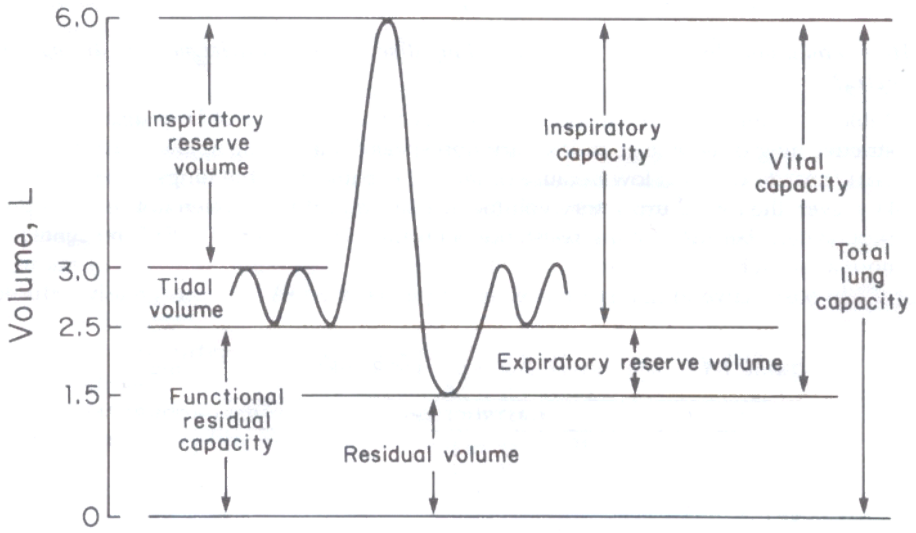


LUNG VOLUMES AND CAPACITIES

The most important lung volumes are:

Tidal Volume (the amount of air inspired during breathing at rest, approximately 500mls)

Functional Residual Capacity (the amount of air remaining in the lungs after normal expiration, approximately 2.5L). Whilst breathing room air 79% of the gas in the FRC is nitrogen, if a patient is given 100% O_2 to breathe for several minutes then all of this nitrogen will be replaced with O_2. The 2.5L O_2 in the FRC is enough O_2 to last for 10 minutes.



The reserve volumes are the volumes that can be inspired/expired over and above a normal tidal volume breath.

Not all of the inspired air takes part in gas exchange, only the air which is in the alveoli does. The air in the bronchi, bronchioles and trachea which does not contribute to gas exchange is known as dead space.

RESPIRATORY FAILURE/INSUFFICIENCY

As breathing is regulated by the nervous system and requires the use of the respiratory muscles and a patent airway any disorders which affect these may impair breathing. Problems which prevent effective gas exchange in the lungs either by preventing the inspired oxygen diffusing into the blood stream or by limiting the blood supply to the lungs (hypoperfusion) will impair ventilation. These may be congenital (e.g. motor neuron disease) or acquired (e.g. pneumonia).

Problems with breathing can occur preoperatively, intra-operatively and post-operatively. Intra and post operative breathing problems can also be attributed to:

Patient factors - a pre-existing problem which may have been worsened e.g. COPD

Anaesthetic factors – e.g. excess opiates or a high epidural block which prevents the muscles of respiration working appropriately

Surgical factors – eg diaphragmatic splinting caused by a distended abdomen or a CO_2 gas embolism during laparoscopic surgery

ASSESSING ADEQUACY OF BREATHING

This can be done either clinically, at the bedside, or with more invasive procedures. You may need to assess breathing pre-op, intra-op or post-op.

CLINICALLY

Does the breathing pattern and rate look comfortable and normal? Are accessory muscles of ventilation being used? Are they cyanosed? Is there evidence of airway obstruction? Does the chest sound normal on auscultation?

Causes of tachypnoea: compensating a metabolic acidosis (eg sepsis, DKA), hypoxeia, hypercapnia, pain, anxiety

Causes of bradypnoea: excess sedatives or opioids, CNS pathology

Oxygen saturations

Tell you only about oxygenation not ventilation; if a patient is on supplemental oxygen, oxygen saturations may mask worsening respiratory failure and hypercarbia (high CO_2).

Peak Expiratory Flow Rate/Bedside Spirometry

These tests may be useful if done serially but rarely done in practice.

CLINICAL DEFINITION OF RESPIRATORY FAILURE

Type 1 respiratory Failure = PaO_2 <8kPa, normal $PaCO_2$

Type 2 Respiratory Failure = PaO_2 <8 kPa, $PaCO_2$>6.6kPa

NON-CLINICALLY

Arterial Blood Gases

Give you important information about gas exchange and possibly the underlying cause of respiratory dysfunction.

CXR/CT - May rarely be needed as part of a pre-op assessment.

RESPIRATION/VENTILATION DURING GENERAL ANAESTHESIA

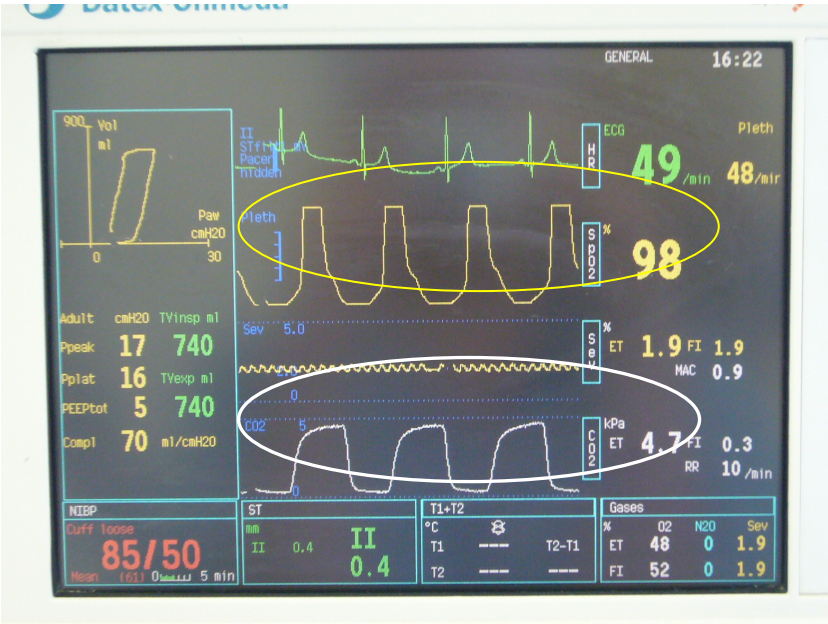
Most anaesthetic agents are respiratory depressants. Whilst spontaneously breathing during general anaesthesia both respiratory rate and tidal volume both decrease. If we want to maintain oxygen and carbon dioxide levels close to physiological levels we may chose to invasively ventilate the patient (ie to ventilate through a tracheal tube or a laryngeal mask airway). In order to take over ventilation we normally give muscle relaxant drugs which paralyse the muscles so the patient does not try to breathe against the ventilator.

Although there are many different types of ventilators and different ventilatory modes the principles are simple. You either create a negative pressure inside the chest which sucks air in:

This is an 'iron lung' that was introduced in the polio epidemics in the 1920s. It generated a negative pressure in the chest thereby 'sucking' air in. They are not practical for use today. However by creating a negative pressure inside the chest the mimic normal ventilation better than today's positive pressure ventilators.

Or you use a positive pressure to blow air into the lungs. Intermittent positive pressure ventilation (IPPV) was introduced in the 1950's and is used routinely in theatres and intensive care. Today's ventilators allow you to control the respiratory rate, tidal volume and FiO_2 (fraction of inspired oxygen).

MONITORING VENTILATION



This is a picture of a typical patient monitor on an anaesthetic machine. It includes an ECG and BP as well as oxygen saturations and capnography. The box to the left of the picture includes a flow-volume curve and also gives an estimate of lung compliance i.e. the pressure required to inflate the lungs.

CLINICAL

During anaesthesia the anaesthetist constantly observes for any change in breathing pattern, rate and asymmetrical movement of the chest. Common problems include hypoventilation and unilateral chest movement if the tracheal tube has been inserted too far into either the left or right main bronchus.

CAPNOGRAPHY

The white waveform circled in the diagram on the previous page represents end-tidal CO_2 ($ETCO_2$). This is the concentration of CO_2 which the patient breathes out. Changes in $ETCO_2$ may represent hyperventilation (in which case the $ETCO_2$ falls) hypoventilation (in which case the $ETCO_2$ rises). Sudden falls in $ETCO_2$ maybe one of the first signs of cardiac arrest or severe hypotension; the lack of lung perfusion in these conditions means that no CO_2 is delivered to the lungs and therefore cannot be exhaled.

OXYGEN SATURATIONS

The yellow waveform circled is the O_2 saturations. There must be a good waveform for the saturations recorded to be accurate. The waveform also gives you the heart rate.

CAUSES OF HYPOXAEMIA

These apply to anaesthetised and non-anaesthetised patients. They relate to the factors that determine oxygen delivery.

HYPOXIC HYPOXAEMIA: This is when not enough O_2 is transported across the lungs. It can be due to a low concentration of O_2 in the inspired air (eg anaesthetic machine failure) or impaired oxygen transfer across the lungs.

STAGNANT: If there is gross circulatory insufficiency (shock). Then there may not be sufficient cardiac output to transfer the oxyhaemoglobin to the tissues.

ANAEMIC: If there is insufficient haemoglobin then the O_2 will not be transported to the tissues.

CYTOTOXIC: This occurs when O_2 arrives at the tissues but they are unable to utilise it. Examples include cyanide toxicity which causes mitochondrial dysfunction. In this case the oxygen saturations will be normal.

As anaesthetists we assess breathing pre-operatively, intra-operatively and post-operatively. Therefore, a knowledge of respiratory physiology and pathology is important to help us optimise our patients before theatre and to explain any changes in ventilatory function. In patients with severe lung disease we may try to avoid giving a general anaesthetic by giving a regional anaesthetic (eg. a spinal). An understanding of the respiratory system along with the cardiovascular system is vital for anaesthetists as small changes in these systems can cause significant changes to the physiology of the entire body.

EVIDENCE

We know from ICU studies that over ventilation and lack of PEEP can damage already precariously functioning lungs. In 2013 Futier published a study in the NEJM ' A Trial of Intraoperative Low-Tidal-Volume Ventilation in Abdominal Surgery'. He showed that, in patients at risk of respiratory complications, those randomised to 'lung protective ventilation'

- 6-8ml/kg of ideal body weight
- 6-8cm PEEP
- recruitment manouvres

had less postoperative acute respiratory failure and shorter length of stay. Some of us use the app MedCalc to work out the ideal 6-8ml/kg values, which depend on gender and height.

C IS FOR CIRCULATION

DR ROBERT CM STEPHENS

The primary purpose of the circulation (heart, vessels & blood) is to supply oxygen to the tissues and remove waste products from cells. The Circulation in anaesthesia is a large subject: here is an introduction!

3 KEY PHYSIOLOGY EQUATIONS

Heart work ∝ Heart oxygen requirements

Heart work ∝ Preload, Afterload, Contractility, Heart rate

Message: Increasing these 4 factors may increase coronary ischaemia

Mean Arterial Pressure (MAP) = Cardiac Output (CO) x Systemic Vascular Resistance (SVR)

Cardiac Output (CO) = Heart Rate (HR) x Stroke Volume (SV)

Message: This is one way to work out why someone is hypotensive

Oxygen delivery (DO_2)= Cardiac Output (CO) x Oxygen content of blood

Oxygen Content = (SaO_2 x Haemoglobin x 1.39) + (0.003 x PaO_2)

Message: These are the ways to increase systemic oxygen delivery

EFFECTS OF ANAESTHESIA + SURGERY ON THE CARDIOVASCULAR SYSTEM

Blood is pumped (blood flow ~5L/min) down a pressure gradient from the left ventricle to the cells.

Tissues need a constant oxygen delivery to maintain oxidative metabolism.

Blood vessels vasodilate or vasoconstrict when blood pressure changes to maintain a constant blood flow. This phenomenon is known as autoregulation.

General Anaesthesia on its own (hypnosis, analgesia, paralysis) generally **reduces blood pressure** by reducing cardiac output and causing vasodilation. Fluids, vasoconstrictors and inotropes can be given to correct this. In addition most anaesthetic agents abolish the process of autoregulation, thus oxygen delivery is determined primarily by perfusion pressure (MAP)

Spinal/Epidural generally cause vasodilation, leading to **hypotension** and a compensating tachycardia

Stimulation (laryngoscopy or surgical incisions) generally opposes the effects of a general anaesthetic by increasing cardiac output (mostly heart rate) and causing vasoconstriction (see equation). Some procedures can cause vagal stimulation (e.g. dilating the cervix) which can cause bradycardias, and others have more complex effects eg inflating the peritoneum with CO_2 during laparoscopies

General and regional (spinal/epidural) anaesthesia both affect the heart and vascular resistance in different ways

There is no evidence as to the **'level' of blood pressure** (mean arterial pressure) considered acceptable under anaesthesia. Patient (Preoperative hypertension, end organ perfusion monitors etc.) and surgical (bleeding) have to be considered. For non-hypertensive patients with normal end organ perfusion under 70 years of age most anaesthetists would be happy with a mean pressure of over 60mmHg.

The degree of **acceptable heart rate change** from baseline mainly depends on the patients' cardiac condition. E.g. patients with ischaemic heart disease should not have a tachycardia as that increases cardiac O_2 demands and reduces time for coronary blood flow.

Cardiac monitors: all patients (GA, regional, sedation) should have ECG, $Sa0_2$, non-invasive BP. Invasive monitors (arterial line, CVP, cardiac output + urine output) should be placed into patients having **major surgery** (eg laparotomy or >500ml blood loss) especially if they have **limited function** (can walk up <2 flights stairs) as they are at greater risk of complications. Most Anaesthetists believe that complications result from an inability to deliver enough O_2 to the cells. Increasing Oxygen delivery to tissues (see equation) reduces complications.

KEY CARDIOVASCULAR STAGES IN 'ANAESTHESIA JOURNEY'

1 Preoperative (starvation, anxiety, anaemia, bleeding?) Fit vs unfit?

2 General Anaesthesia: induction, intubation

or Spinal/Epidural: onset of block

3 Surgical stimulation

4 Hypovolaemia, Blood loss

5 General Anaesthesia: emergence, extubation

6 Recovery including drugs

7 Complications

SUMMARY

You should consider the effects of…..

Anaesthesia
Patients' cardiovascular state
Surgery
Drugs

Upon the……..

Heart
Vessels
Blood
Cell

D IS FOR ANAESTHETIC DRUGS

DR JAMES HOLDING
DR MARYAM JADIDI

NON-ANAESTHETIC DRUGS (USUAL MEDICATION)

Patients are now encouraged to continue all their usual medications throughout the perioperative period with only a few exceptions:

DIABETIC MEDICATIONS

The aim is to maintain a perioperative BM of 4-10, and particularly to avoid hypos. And so insulin and oral hypoglycaemics should be omitted on the morning of surgery (and very long acting drugs from the evening before), and the BM monitored. Insulin sliding scale may be needed.

ANTICOAGULANTS

Aspirin – is generally now continued perioperatively, the risk of MI / stroke if it is stopped being greater than the risk of significant bleeding if it isn't, but it is stopped 7 days before some bleeding critical operations e.g. neurosurgery, arthroscopy.

Clopidogrel – is generally stopped 7-10 days preoperatively – but not lightly in patients with drug eluting coronary stents.

Warfarin – is normally stopped 5 days pre-operatively. When the INR is below 2 a daily treatment dose of unfractionated heparinmay be given for all indications except those on warfarin for simple AF prophylaxis 'bridging plan'. The last dose of heparin should be given 24 hours before surgery. If there are no concerns about bleeding post-operatively a smaller dose of unfractionated heparin is given after surgery, returning to the treatment dose on the first post-op day, which is continued until the INR has returned to a therapeutic level.

Antihypertensives – continue all, except ACE inhibitors and AT II inhibitors, which should be omitted on the morning of surgery for patients having laparoscopic surgery -there are reports of profound hypotension. Some people continue these if the patient has a diagnosis of heart failure.

Dabigatran and Rivaroxaban- newer oral anticoagulants stopped 1-2 days before surgery

PREMEDICATION

In the past all surgical patients received a heavy, long lasting premed combining anxiolysis, analgesia and a secretion drying agent. The advent of modern anaesthetic drugs and techniques has reduced the need for this kind of premed.

GENERAL ANAESTHESIC AGENTS

Here is a secret. For most operations anaesthesia is induced with a dose of short acting opiate and an intravenous induction agent. These drugs 'buy' a few minutes of unconsciousness in which time the patient is set up to breathe some anaesthetic vapour that keeps them asleep (maintenance). At the end of the operation the anaesthetic vapour is turned off, the patient breathes it out of their system, and then they wake up. Don't tell the surgeons, they still think it is some kind of magic.

Induction can also be achieved by just breathing the anaesthetic vapour, which can be useful for children, and others in whom cannulas are difficult to place. Specific muscle paralysing drugs are sometimes used to facilitate tracheal intubation, enable the respiration to be controlled, and to allow surgery within body cavities.

The following list is not exhaustive, but includes the drugs that are commonly used by UK anaesthetists in 2014. Doses have been omitted on purpose – they can be very variable. Anaesthetists tend to titrate the dose to effect. (The single bolus dose of most anaesthetic drugs (except vasopressors and remifentanil) for a standard 80 kg adult is about one ampoule).

INTRAVENOUS INDUCTION AGENTS

A single bolus dose of an induction agent quickly causes brain levels of the drug to rise and results in almost instantaneous anaesthesia. This is followed by a rapid fall in brain and blood levels as the drug is redistributed to other tissues. The result is a brief duration of anaesthesia with a rapid recovery. The emergence from anaesthesia after a single bolus dose is due to redistribution of the drug, not metabolism and elimination – which may take many hours.

PROPOFOL – which comes as a 1% emulsion (like milk), and produces a smooth and rapid induction of anaesthesia with greater laryngeal reflex suppression than other agents. It is thought to have an antiemetic effect, and its metabolism and elimination are little affected by hepatic or renal failure. The major side effect is a dose-dependant reduction in vascular tone, causing both a reduction in SVR and CVP (and preload) resulting in a reduced cardiac output and blood pressure. It can also be painful on injection, although this can be reduced by injecting into a big vein or by adding some lignocaine to the propofol. Propofol has become the dominant induction agent over the last 10 years.

THIOPENTONE – this was the universal induction agent before propofol came along. Its action is similar to propofol, although the period of unconsciousness in generally longer and there is more 'hangover', but there is less cardiovascular depression. It is the classic drug to be used in a rapid sequence induction (RSI), and is a classic trigger for porphyria.

ETOMIDATE – a cardio-stable induction agent, which has recently fallen out of favour as it inhibits corticosteroid and mineralocorticoid synthesis.

KETAMINE – a non-competitive antagonist of the calcium ion channel operated by the excitatory NMDA glutamate receptor, but also has effects at opiate and mACh receptors. It is a strange drug, which produces a strange kind of anaesthesia (dissociative) and analgesia. Laryngeal reflexes are preserved, there is bronchodilatation, and the heart rate and blood pressure are increased. It is the

only induction agent that is also effective if given intra-muscularly (IM). Its use has been limited by fears of dissociative side-effects on emergence from anaesthesia (hallucinations and nightmares).

It might be a good choice for the induction of patients with bronchospasm (e.g. acute asthma), but is more commonly used in sub anaesthetic doses for its analgesic effect.

MAINTENANCE AGENTS

The pharmacokinetics of gases and vapours when they enter a body are difficult and non-intuitive. The important value is the partial pressure, which is the driving force behind gas transfer. A vapour is a gas, and acts like a gas – but it is below its boiling point. Here are two useful concepts:

If a gas is very insoluble in blood, then not many of the gas molecules have to transfer over the gas/blood interface in the lung before equilibrium of the partial pressures is reached, and so it will be reached relatively quickly. And at the end of the operation, when the gas is removed from the breathing circuit, not many molecules have to move out of the blood before the blood partial pressure of the gas falls towards zero, and so the patient wakes up relatively quickly.

The minimal alveolar concentration (MAC) has been calculated for all anaesthetic gases / vapours, it is a measure of potency (the name is an historical error – it should be minimal alveolar partial pressure – but as atmospheric pressure at sea-level is about 100kPa it doesn't matter because the values for pressure and concentration are the same). One MAC is the concentration of anaesthetic agent, which at equilibrium will prevent a reflex response to a standardised skin incision in 50% of patients. As the alveolar concentration can be assumed to be the end expiratory concentration, which can be measured by a gas analyser, we can check that a patient is receiving enough agent to keep them anaesthetised.

ANAESTHETIC VAPOURS – their clinical effects are all broadly similar. They produce respiratory depression, bronchodilatation, and reduced blood pressure – through a mixture of reduced myocardial contractility and vasodilatation. None have analgesic properties. Isoflurane is the most soluble in blood, followed by sevoflurane and then desflurane, the least soluble

Isoflurane (ISO) – MAC = 1.1
Desflurane (DES) – MAC = 6 The most insoluble – so the fastest to equilibriate – but a respiratory irritant, so unsuitable for gaseous induction.
Sevoflurane (SEVO) – MAC = 2.2 Used for gaseous induction.

NITROUS OXIDE – a gas. MAC = 105 – so it is unable to be a sole anaesthetic agent, but it can be a carrier agent for the anaesthetic vapours. As MAC is additive a reduced amount of vapour is needed when nitrous oxide is used. It is also an analgesic, and is very insoluble in blood. It has become unfashionable recently due to its side effects of causing gas filled bowel to expand, increasing nausea, and suppressing bone marrow production of neutophils and platelets.

PROPOFOL – again. Propofol is redistributed and eliminated reliably enough to maintain anaesthesia when given as an infusion. The pharmacokinetics of infusions of drugs, which are not metabolised almost immediately, are particularly complex. Maintenance of a steady plasma level requires a continually changing infusion rate. This is achieved with a computer controlled infusion pump using an algorithm based on the patient's sex, age and weight. Unlike with MAC for the

vapours, we are unable to measure in real-time the plasma levels of propofol so can only judge the effectiveness of anaesthesia by monitoring the clinical effects.

MUSCLE RELAXANTS

These act at the post-junctional nicotinic (nACh) receptors of the skeletal muscle neuromuscular junction (NMJ). The nACh receptor is a ligand gated sodium ion channel. Binding of ACh (or nicotine) causes the channel to open, allowing sodium to move into the cell, depolarizing the membrane potential.

NON-DEPOLARIZING MUSCLE RELAXANTS

These drugs competitively and reversibly bind to the receptor preventing ACh reaching its binding site. The duration of action of a single bolus dose is: **Vecuronium** (40mins) > **Atracurium** (35mins) > **Rocuronium** (30mins). Atracurium causes more histamine release, and therefore more bronchospasm and hypotension, but it spontaneously fragments in the circulation (Hofmann degradation), and is widely metabolised, so is more suitable for patients with renal or hepatic impairment.

DEPOLARIZING MUSCLE RELAXANTS

SUXAMETHONIUM mimics the effect of ACh when it binds at the nACh receptor causing depolarisation of the cell and twitching of the muscle fibres. As these twitches are uncoordinated they result in fasciculation of the muscle body. Suxamethonium is the fastest acting of the muscle relaxants (within 30 secs), and the fastest to wear off (within 7 mins), so is the relaxant of choice for rapid sequence induction. Sux is an old, dirty drug though, with a lot of side effects. It can stimulate nACh receptors at autonomic ganglia, causing bradycardia, it causes post-op muscle pain and nausea, results in a rise in plasma potassium and it is associated with a high rate of anaphylaxis. The effect of sux is terminated by diffusion away from the NMJ and then hydrolysis by plasma cholinesterases. Plasma cholinesterase deficiency is relatively common, both genetic and acquired, and results in a prolonged block following a dose of suxamethonium (sux apnoea).

REVERSALS

NEOSTIGMINE is an acetylcholinesterase inhibitor. It is used when a non-depolarizing block has already begun to wear off to increase the NMJ synaptic concentration of ACh to help overcome the block and restore muscle power and function. It is another dirty drug, and also acts at autonomic ganglia, causing bradycardia, bronchoconstriction and smooth muscle peristalsis. To minimise these effects it is given together with an anti-cholinergic (atropine or glycopyrulate).

SUGAMMADEX is a relatively new drug that is designed to bind with the steroid non-depolarizers (rocuronium and vecuronium) and stop them acting at the NMJ. It has the potential to reverse even profound blocks with little stimulation of the autonomic system.

UPPERS AND DOWNERS

The anti-cholinergics **atropine** and **glycopyrulate** are used to reduce vagaly mediated bradycardia, and to dry secretions. Unlike atropine, glycopyrulate does not cross the blood-brain barrier, and does not cause sedation.

The pure α adreno receptor agonists **phenylepherine** and **metaraminol** predominantly cause a transient rise in blood pressure by peripheral vasoconstriction. The mixed α and β adreno agonist **ephedrine** raises BP by a combination of vasoconstriction and increased heart rate.

Most anaesthetists will reduce blood pressure by giving more anaesthetic agent or opiate, although short acting β-blockers (**labetalol**, **esmolol**), **GTN**, and the α_2 agonist **clonidine**, are also regularly used.

PAIN KILLERS

OPIOIDS

The side-effects of opioids include respiratory depression, hypotension, bradycardia, delayed gastric emptying, nausea, constipation, itching and muscle rigidity – but they are still the mainstay of surgical analgesia. By using clean short acting drugs these side-effects can be minimised. (In brackets is the equianalgesic dose to 10mg of IV morphine, and the approximate duration of action)

MORPHINE (10mg, 120mins) – metabolism produces active metabolites which can accumulate in renal failure.

FENTANYL (100mcg, 30 mins)

ALFENTANIL (1mg, 15mins)

REMIFENTANIL **–** rapidly metabolised by plasma and tissue esterases, and is very short acting. It has to be given as an infusion, and can cause severe bradycardia if given as a bolus.

CODEINE (30-60mg, 120 mins) – about 10% is metabolised to morphine, although there is genetic heterogeneity, some people unable to metabolise any, and others metabolising almost 100% to morphine. Its effects are therefore unpredictable. Use dihydrocodeine- same dose.

TRAMADOL (50-100mg, 120mins) **–** has some weak effects at opioid receptors, but also seems to modulate 5-HT, NMDA and noradrenergic receptors. It is not a wonder drug though, its nausea profile being similar to morphine.

NON-OPIOIDS

These include the usual drugs paracetamol and diclofenac, and the more unusual **ketamine** and **clonidine**.

ANTI-EMETICS

Post-operative nausea and vomiting (PONV) is common with multiple causes (opiates, stimulation of vagus, hypotension, starvation, ileus) and risk factors (females, children, surgery on the eye, inner ear, abdomen or laparoscopic). Anti-emetics are given routinely, and often 2 or 3 drugs are used together as prophylaxis from different groups eg Dexamethasone or Cyclizine and Ondansetron

CYCLIZINE – an anti-histamine (SE – tachycardia and other anti-cholinergic effects)

GRANISETRON AND ONDANSETRON – 5-HT_3 receptor antagonists (SE – constipation)

DEXAMETHASONE – a glucocorticoid (SE – deranged glucose control)

PROCHLORPERAZINE ('stematil') – a dopamine and mACh receptor antagonist (SE – extrapyramidal)

P IS FOR PREASSESSMENT

DR MARK EDWARDS

Increasing attention has been paid to preoperative assessment over the years and in many cases this is now performed on an outpatient basis weeks before surgery, followed up with a shorter visit from the anaesthetist on the day of surgery. So, why bother assessing patients before an anaesthetic?

1. Build a rapport with the patient and reduce patient anxiety
2. Assess **risk** using a structured method of patient assessment
3. Form a plan for the perioperative period and gain consent for it

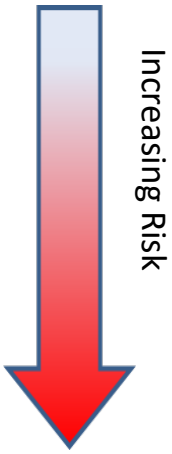
BUILDING A RAPPORT

A friendly, confident manner, a clear explanation of the expected events and an opportunity to field questions are important when seeing patients. Many patients, particularly on the day of surgery, are understandably nervous and a good preoperative visit can go a long way to reducing this anxiety.

RISK:

All operations and their anaesthetic carry risk, although for healthy patients having minor surgery the chances of serious adverse events is probably less than the chances of the patient crashing their car that year. Risk assessment informs the consent process and allows interventions which can reduce perioperative morbidity and mortality ("optimisation") to be planned. Thinking of anaesthetic risk in isolation is unhelpful – without the operation there is no anaesthetic so we should consider the effect of the whole *perioperative insult* on the patient and consider the chances of them suffering minor morbidity (e.g. a chipped tooth, sore throat or nausea) or something more major such as postoperative respiratory failure, adverse cardiac event or multiorgan dysfunction. So what dictates the risk attached to a particular operation?

Surgical factors	Patient Factors	"System" factors
Minor Surgery e.g. on the body surface, short duration	Usually fit and well, young and active	Planned surgery/experienced staff/ easy access to high quality post-op critical care
Moderate surgery	Some minor illness, well controlled	
Major surgery e.g. intra-abdominal/thoracic, major orthopaedic, long duration	Multiple cardiorespiratory or other morbidities, not well controlled, elderly patients	Emergency surgery/ junior or fatigued staff/ no or poor critical care facilities



SURGICAL FACTORS

Surgery can be divided into minor, intermediate, major and major plus as below, with examples:

Grade 1: Excision skin lesion; drainage breast abscess

Grade 2: Inguinal hernia; varicose veins; tonsillectomy; arthroscopy

Grade 3: Hysterectomy; TURP; lumbar discectomy; thyroidectomy

Grade 4: Joint replacement; thoracic operations; colonic resection; radical neck dissection

A discussion with the surgeon is very helpful in establishing whether they are expecting any particular difficulties e.g. due to previous surgery, high risk of bleeding etc.

Emergency surgery makes it much more likely that the patient already has a degree of physiological upset, such as hypovolemia, anaemia or sepsis.

PATIENT FACTORS

Many factors can influence patient risk:

AGE

Old age on its own does not automatically equate to increased risk, but there is a gradual physiological decline and the increased likelihood of overt or subclinical comorbidity with advancing age.

COMORBIDITIES

There is not enough space here to describe the effects of all illnesses on the perioperative phase, but some examples include:

Heart failure – impaired cardiac pump function will be aggravated by most anaesthetic agents, leading to possible tissue hypoperfusion. Probably the most serious common comorbidity .

Significant respiratory disease - increases the chances of intraoperative problems such as hypoventilation, quicker desaturation at intubation and extubation, difficulty ventilating the patient, postoperative respiratory insufficiency and secondary infection.

Hypertension – chronic hypertension means end organs are habituated to higher than normal blood pressures, and are more likely to suffer inadequate oxygen delivery during anaesthesia-related hypotension.

Ischaemic heart disease – if significant can lead to perioperative myocardial ischaemia in the face of the physiological stress of surgery and increased sympathetic nervous system activity.

Diabetes – fasting, omitting normal medication and the glycaemic response to the surgical stress can all lead to disruption of blood sugar control. Diabetics have a range of associated comorbidities which can also affect the perioperative course.

MEDICATION

: effects of patients' normal medication.

Some examples include:

Antihypertensives – can exaggerate intraoperative hypotension. Diuretics can cause electrolyte abnormalities.

Antiplatelets / anticoagulants – increased bleeding risk (although stopping them may lead to increased risk of cardiovascular events!).
Opiates – patients with chronic pain requiring high doses of opiates will require more

perioperative analgesia.

Steroids – long term use or recent high dose administration blunts the adrenal axis and the cortisol response to surgical stress. Additional perioperative supplementation is required.

EXERCISE TOLERANCE requires the integrated actions of the respiratory, cardiovascular and musculoskeletal systems and can be seen as a way of gauging how well a person will stand up to the stresses of surgery. Assessment can be by patient reported history e.g in terms of metabolic equivalents of task, 'METs', 1 MET being equivalent to sitting quietly.

1– 4 METs: eating, dressing, dishwashing and walking around the house

4 – 10 METs: Climbing a flight of stairs, walking on level ground at >6km/hr, running briefly and playing golf

10 METs: strenuous sports, swimming, singles tennis or football

Patients who cannot sustain 4 METs of physical activity may have worse postoperative outcomes. A more objective test of exercise tolerance is cardiopulmonary exercise testing (CPET); see box below. This advanced test may be used prior to major surgery or in patients with other risk factors. Poor CPET performance is associated with more morbidity and mortality after major surgery.

ALLERGIES, or adverse reactions to drugs used intraoperatively.

AIRWAY factors which may make controlling the patients' airway difficult.

STRUCTURED APPROACH TO PATIENT ASSESSMENT

Taking into account all the patient factors which require assessment a logical approach to the preoperative visit and assessment process is needed:

HISTORY

Previous anaesthetics – any problems?

Known family problems with anaesthetics? (rare hereditary problem e.g. suxamethonium apnoea or malignant hyperpyrexia)

Medical history, including an assessment of *severity* of the comorbidities (e.g. if asthmatic, how many exacerbations? Restricted functionally? Ever been in ITU? Ventilated? What treatment?)

Effort tolerance

Drugs – prescription, alcohol, nicotine, illegal drugs, allergies

Fasting status – a full stomach may lead to aspiration during the anaesthetic, so no solids or milk for 6 hours preop, and no water for 2 hours preop. Patients should be discouraged from excessive fasting however.

EXAMINATION

Cardiorespiratory system

Weight and BMI

Airway and dentition

INVESTIGATIONS

Should be triggered by the patient's age, severity of surgery and identified comorbidities. NICE has guidelines (www.nice.org.uk) that should be adhered to for simple tests (e.g. FBC, U+E, ECG and urine dipstick). A young fit male for minor surgery doesn't really need an ECG or routine blood tests preoperatively as the chances of finding an abnormality are extremely low, but a female will be offered Hb and Urine HCG.

ADVANCED TESTS

Imaging – e.g. CXR if active respiratory disease and no recent CXR (not for asthma). C-spine radiography if bony problem with the airway suspected (e.g. rheumatoid arthritis).

Cardiac testing – if suspected cardiac disease not already diagnosed or known disease without recent assessment. Echocardiogram? (ventricular function, murmur diagnosis etc.) Popular with anaesthetists but tells us little about how the heart will perform under stress!). Tests for ischaemic heart disease such as radionuclide scanning / stress ECHO / angiography in consultation with a cardiologist- but beware- fixing the heart in general doesn't change postoperative mortality.

Respiratory testing – arterial blood gases, pulmonary function testing. Again, may help quantify the severity of respiratory disease but are static tests.

Cardiopulmonary exercise testing

- *A dynamic, objective test demonstrating how well the patient stands up to physiological stress.*
- *Standardized exercise protocol using cycle or treadmill and gradually increasing resistance – lasts approx 10 minutes.*
- *ECG, SpO2, BP and inspired / expired O_2 and CO_2 are measured, allowing continuous calculation of oxygen uptake (VO_2) and CO_2 production.*
- *Key measures – anaerobic threshold (AT); the VO_2 at which muscular lactate production begins. VO_2peak – the maximum oxygen uptake achieved by the patient.*
- *Cardiac insufficiency, ischaemia and respiratory compromise may also be detected.*
- *AT <11ml/kg/min is associated with increased morbidity and mortality after major surgery, particularly in the presence of cardiac ischaemia.*

PUTTING IT ALL TOGETHER – RISK SCORING SYSTEMS

These aim to integrate some of the risk factors assessed above into a convenient score which gives an idea of perioperative morbidity / mortality risk. However, none has been shown to be particularly accurate when used on an individual basis for predicting perioperative events and are more useful in identifying higher risk populations.

ASA (American Society of Anesthesiologists Physiological Score) – simplest and most commonly used:

Grade 1: A healthy patient with no systemic disease

Grade 2: Mild to moderate systemic disease

Grade 3: Severe systemic disease imposing functional limitation on patient

Grade 4: Severe systemic disease which is a constant threat to life

Grade 5: Moribund patient who is not expected to survive with or without the operation

Grade 6: A brain stem dead patient whose organs are being removed for donor purposes

SORT SCORE www.sortsurgery.com- uses variables to calculate a 30 day chance of dying after surgery.

P-POSSUM score – takes into account a wide range of physiological, intraoperative and comorbidity variables to estimate postoperative risk of morbidity and mortality.

Lee's Revised Cardiac Risk Index (RCRI) – allows for age, presence of IHD, heart failure, previous strokes, diabetes and renal impairment in order to estimate the perioperative risk of *cardiac events* in non-cardiac surgery.

PERIOPERATIVE PLAN

This should be formed during the preoperative assessment. It consists of:

PREOPERATIVE

Gathering further information on patient risk factors – clinic letters, ordering relevant investigations if not done already.

Optimising comorbidities – is control of hypertension / COPD / diabetes etc. as good as it can be? Improving complex medical diseases usually requires specialist referral.

Optimising tissue oxygen delivery – see box below

Deciding on surgical timing – the urgency of surgery needs to be weighed up against the benefits of delaying to allow further patient optimisation. For example, cancelling urgent cancer surgery for two months to achieve "perfect" control of a patient's hypertension is probably inappropriate.

Practical planning – booking ITU beds, ordering cross-matched blood, ensuring the necessary expertise is available on the day of surgery. Drink fluids until 2 hours before. Carbohydrate load?

Goal-Directed Therapy

Studies by Shoemaker, Boyd and others showed that boosting high risk patients' tissue oxygen delivery using fluids, blood and inotropes preoperatively lead to improved survival after major surgery. This approach has problems such as the poor availability of preop ITU beds, the significant resources required and the need for some form of cardiac output monitor such as the - now unpopular – pulmonary artery catheter. As an alternative, giving fluids / inotropes to target physiological goals such as cardiac stroke volume can be performed intra- or postoperatively using less invasive devices, and may still reduce postoperative morbidity.

INTRAOPERATIVE

GA or regional?

If GA, spontaneously breathing or ventilated? Tracheal intubation or supraglottic or "advanced" airway technique? Method of tracheal intubation?

Volatile or IV maintenance of anaesthesia?

Need for invasive monitoring?

Type and amount of intra- and post-operative analgesia; systemic, regional block or central neuraxial e.g. epidural (or a combination!)

Once "Plan A" has been formed, backup plans B and C then need to be considered!

POSTOPERATIVE

Level of care required (related to perioperative risk) – ward, HDU or ITU?

When to extubate – immediately postoperatively or after a period of ventilation on ITU (suitable after major, prolonged surgery to allow stabilisation of body temperature, blood clotting, acid-base and electrolyte balance and ventilatory function which may all have been disturbed intraoperatively).

DISCUSSION and CONSENT

This should be informed by a description of the benefits, risks and alternatives for the main interventions planned (e.g. type of anaesthetic, analgesia plan etc.) Written information given out at preassessment clinic can help. All procedures have different risk-benefit profiles and common or rare-but-serious risks should be discussed with patients, e.g.

> *GA (sore throat, dental damage, nausea and vomiting)*
>
> *Spinal / epidural (failure, hypotension, motor block, dural puncture headache 1:100, nerve damage 1:20,000)*
>
> *Invasive lines (haematoma, infection, damage to surrounding structures)*

The risks attached to the perioperative insult for high risk patients / major surgery (may include respiratory failure, renal failure, MI, prolonged and difficult ITU stay or death – ***an honest but tactful conversation with the support of surgeon and patients relatives is required****)*

Signed consent is not currently required for anaesthesia, but clear documentation of what was discussed is vital.

Patients also need a general description of events, particularly if you will be doing anything which the patient might not expect e.g. rapid sequence induction ("someone will press on your throat as you go off to sleep!"), ventilating for a while on ITU postoperatively, using suppositories!

R IS FOR REGIONAL ANAESTHESIA AND PAIN

DR MAYA NAGARATNAM

Pain

An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.

Acute pain is of short duration, and warns of imminent tissue damage (less than 3 months)

Chronic pain persists longer than normal tissue healing time (more than 3 months)

Nocioceptive - pain resulting from somatic and visceral stimulation/injury

Neuropathic - pain resulting from injury to the nervous system

WHY MANAGE PAIN?

Pain is still undermanaged in hospital simply because it is not assessed well. Doctors and nurses tend to underestimate the level of pain experienced by patients. Managing patients' pain during the perioperative period is important for humanitarian reasons and because good pain relief has significant physiological benefits:

REDUCED

Sympathetic activity

Incidence of acute coronary syndromes

Risk of tachycardia and dysrhythmias

Respiratory complications

Thromboembolic events

Chronic pain syndrome

IMPROVED

Patient satisfaction

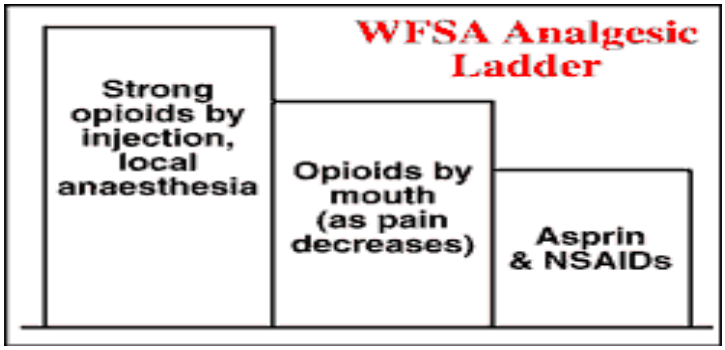
Wound healing

Mobilization

Earlier hospital discharge

METHODS OF MANAGING PAIN

ENTERAL AND PARENTERAL



The World Federation of Societies of Anaesthesiologists (WFSA) or WHO Analgesic Ladder has been developed to treat acute pain. Initially, the pain can be expected to be severe and may need controlling with strong analgesics in combination with local anaesthetic blocks and peripherally acting drugs.

The oral route for the administration of drugs may be denied because of the nature of the surgery and drugs may have to be given by injection. Normally, postoperative pain should decrease with time and the need for drugs to be given by injection should cease. The second rung on the postoperative pain ladder is the restoration of the use of the oral route to deliver analgesia. Strong opioids may no longer be required and adequate analgesia can be obtained by using combinations of peripherally acting agents and weak opioids. The final step is when the pain can be controlled by peripherally acting agents alone

PATIENT-CONTROLLED ANALGESIA

Patient-controlled analgesia (PCA) is an effective way of providing opioid analgesia where the patient titrates the dose to his/her need by pressing a button that delivers a small bolus (e.g. 1 mg morphine). It is safe, has a high patient satisfaction and is usually set up by the anaesthetist in theatre.

Managed by the acute pain team it is used postoperatively until the patient can tolerate oral analgesia. For safety, a separate IV line is required with a non-return valve and crystalloid infusion at 30 ml/hour to keep the line patent.

REGIONAL ANAESTHESIA AND ANALGESIA

PERIPHERAL NERVE BLOCKS

When surgery is performed on the extremities (arms and legs), the innervation of which is derived centrally from the spinal nerves. These nerves coalesce into plexuses, and finally divide into terminal nerves supplying the bones and muscles and innervating the skin of the arm and leg. At certain points along their path these nerves can easily be identified and blocked with local anaesthetic, achieving analgesia and anaesthesia. Peripheral nerve blocks are usually used as adjuncts to anaesthesia for pain relef and rarely used as solo technique.

CENTRAL NERVE BLOCKADE

Epidural

Epidurals can be used as sole anaesthetics and analgesic tool or as a way to provide analgesia intra and post operatively. The doses for anaesthesia are of greater concentration than for pure analgesia.

It works by diffusion of local anaesthetics in the epidural space into the CSF and disrupting the transmission of pain in the spinal pathway.

Epidural infusions of local anaesthetic (often with an opioid, e.g. fentanyl) provide great pain relief, decrease respiratory complications, the risk of venous thrombosis and short-term morbidity.

They are usually sited by the anaesthetist in theatre under strict aspesis and left in for less than 4 days after surgery.

They are the analgesic of choice during labour.

Coagulation must be normal before their insertion or removal to prevent an epidural haematoma, so low molecular weight heparin should not be given within 12 hours of either event. The acute pain team usually manages the infusion rate (typically 5–15 ml/hour). Intravenous (IV) fluids need to be given and urinary catheterization may be required. Before removal, alternative analgesia needs to be started.

Spinals

Used as anaesthetic technique particularly in operations below the umbilical and anaesthetic of choice in obstetrics.

Local anaesthetic is placed directly in the CSF and therefore much lower concentrations than that used for epidurals required.

INDICATIONS AND CONTRAINDICATIONS FOR REGIONAL ANAESTHESIA

Relative Indications	Relative Contraindications
Contraindications to GA Elderly patients with systemic disease esp. respiratory Morbidly obese patients	Coagulopathy Inadequate resuscitation Patient refusal Septicaemia Neurological disease Anatomical deformities of spine

CONTROVERSIES IN REGIONAL ANAESTHESIA

There is no evidence of any improvement in patient outcomes, but satisfaction may be better.

Difficult airway: Initially, spinal anaesthesia may appear to offer an ideal solution to the problem of a patient with a potentially difficult airway if anatomically appropriate however always bear in mind the risk of failure of regional or an ascending block neccesiting airway securing urgently losing the element of planning.

Spinal with sedation: The optimal level of sedation can be difficult to judge as too much sedation can lead to hypoventilation, hypoxia or silent regurgitation of gastric contents. Conscious sedation is a very fine art.

Advantages

Avoidance of general anaesthesia in high-risk patients
Arguably more stable intraoperative conditions (i.e. cardiovascular)
Effective perioperative pain control, extending into the postoperative period
Reduced nausea and vomiting
Antithrombotic, therefore reduced risk of DVT
Avoidance of opiates in elderly, confused or opiate sensitive patients
Preoperative pain control (e.g. pre-amputation ischaemic pain)

Disadvantages

Time consuming – most regional blocks take 15-30 mins to be effective
Failure rate 1-50% dependent upon expertise of operator
Little evidence that outcomes are improved
Risk of undetected epidural haematoma or compartment syndrome
Delayed post operative mobility

NAP 3 study

The largest ever prospective study into the major complications of epidurals and spinal anaesthetics concluded that previous studies have over-estimated the risks of severe complications of these procedures. NAP3 concluded that the estimated risk of permanent harm following a spinal anaesthetic or epidural is lower than 1 in 20,000 and in many circumstances the estimated risk is *considerably* lower.

But there were *big differences* in the risks between those having spinals or epidurals for Obstetric, Pain and Surgery (Perioperative) probably due to the differences in patient fitness and the nature of the surgical insult.

You can look up the precise numbers and download an app from www.rcoa.ac.uk/nap3

SUMMARY

Regular simple analgesia is useful in pre-empting or anticipating pain.

Paracetamol is a very effective analgesic.

Non-steroidal anti-inflammatory drugs are opioid sparing, use the lowest possible effective dose with a mucoprotective agent.

Regular review of the analgesic regimen is important.

Do not be afraid to give intravenous morphine according to your local hospital guidelines: stay with the patient and titrate in small boluses.

Always try to maintain the oral analgesic route especially with drug problem patients.

Liaise with the acute pain team early.

References:

M Nagaratnam et al Prescribing analgesia for the surgical patient British Journal of Hospital Medicine, January 2007, Vol 68, No 1

E IS FOR EMERGENCIES

DR JANE LOWERY

An introduction to the development of crisis management skills.

WHAT CONSTITUTES AN ANAESTHETIC EMERGENCY?

An unexpected rapidly evolving event requiring immediate intervention to prevent disaster.

Emergencies can be simple and easily treatable e.g. hypotension secondary to induction of anaesthesia treated with ephedrine, or more complex e.g. the difficult airway that may require a range of equipment and several pairs of hands.

WHY DO EMERGENCIES OCCUR?

PATIENT FACTORS:

Obesity – desaturate quickly, difficult to intubate and ventilate, increased incidence of reflux, poor IV access, difficult to move and position patient, usually associated co-morbidities.

Undiagnosed conditions – dysrhythmias, thyroid disease, malignant hyperpyrexia, suxamethonium apnoa, bleeding disorders.

Difficult airways – Rheumatoid arthritis, Ankalosing spondylitis, receding jaws etc.

Allergies – anaphylaxis

Not appropriately starved in emergencies – increased incidence of aspiration

ANAESTHETIC FACTORS:

Inadequate preoperative assessment – may lead to incorrect anaesthetic plan

Too much induction agent – hypotension, bradycardia, poor organ perfusion

Too little induction agent – awareness, difficult airway, laryngospasm, bronchospasm

Regional blocks – all associated with their own risks and the generic risk of LA toxicity

Fatigue – end of a long night shift?

Distractions – people entering/leaving, mobile phones

Equipment failure – not checked, no replacement, not available

Poor communication – between multidisciplinary team

Hypothermia – can lead to coagulopathies, arrythmias

SURGICAL FACTORS:

Unexpected severe haemorrhage – accidently severed arteries

Positioning – proning required, head down, sitting up

Poor communication – of the extent of surgery, expected blood loss, difficult aspects

HOW CAN WE PREVENT EMERGENCIES OCCURRING?

Optimise your environment – familarise yourself with theatre/anaesthetic room

Check your equipment – daily machine check, dates on drugs, laryngoscopes, airway adjuncts, drugs

Anticipate and plan – a good pre-operative history should allow you to anticipate potential difficulties and therefore make a plan B and have relevant drugs and equipment 'handy'. You may even need and discuss with the team a plan C- what you'll do if things fail.

Take a leadership role – allocate tasks if necessary. Try to be specific: e.g. 'John- could you get 2 units of blood from the fridge' etc. Distribute workload and use all available resources.

Communicate effectively – introduce yourself, express your concerns to others in theatre. Make it clear what your plan is and what equipment you want ready or on standby.

Call for help early – you can never do this early enough.

Minimise distractions – mobiles on silent, prevent people from walking in and out

Don't rush – don't succumb to production pressures. Remember patient safety comes first.

Never work alone if feeling fatigued or ill

Always ensure the **WHO** Surgical Safety Checklist is completed before surgery begins

HOW DO WE APPROACH THE MANAGEMENT OF A DEVELOPING CRISIS?

We use effective practical and communication skills. Both are absolutely key in the management of an emergency situation and most of us have a precompiled response that we revert to when things start to go wrong.

KEY PRINCIPLES:

Stay calm

Express your concerns to others in the anaesthetic room/theatre so they don't divert their attention

Call for senior help if none present – get someone else to do this

Revert to plan B or C if problem anticipated

If you don't know what's happening, go through your precompiled response e.g. SOS COVER ABCD A SWIFT CHECK

Once problem identified allocate tasks effectively to resolve the situation quickly and efficiently

Over the next few pages the following anaesthetic emergencies are covered:

Anaphylaxis, Bradycardia, Massive haemorrhage, Malignant Hyperpyrexia, Laryngospasm, Can't intubate can't ventilate and Local anaesthetic toxicity. These are only guidelines and local protocols at your hospital must be consulted

SOS COVER ABCD A SWIFT CHECK

S – Stay calm

O – 100% Oxygen

S – Shout for help

C – Circulation, Capnograph, Colour (sats)

O – Oxygen (supply, quantity)

V – Ventilation (airway pressures, volumes, adequacy)

E – Endotracheal tube (dislodgement, kinks)

R – Review monitors and equipment, machine (connections)

A – Airway

B – Breathing

C – Circulation (in more detail)

D – Drugs (too much too little, finished)

A – Anaphylaxis, Awareness, Air embolism, Air in pleura (pneumothorax)

SWIFT CHECK – of patient, surgeon and surrounds

NB – for a spontaneously breathing patient change to:

AB COVER CD A SWIFT CHECK

ANAPHYLAXIS UNDER ANAESTHESIA

DEFINITION: Severe generalised life-threatening hypersensitivity reaction

INCIDENCE: 50 – 100 cases related to anaesthesia per year

CAUSES: Most commonly - Neuromuscular blocking drugs, Latex, Antibiotics, Hypnotics, Colliod, Opioids.

SIGNS: Hypotension, loss of peripheral pulse, desaturation, bronchospasm, flushing, urticaria, swelling, ECG abnormalities, tachycardia initially, bradycardia pre terminal sign

MANAGEMENT:

Stop administration of all possible causative agents

CALL FOR HELP

100% oxygen. Lie patient flat with legs elevated

Adrenaline – do not hesitate

IM = 0.5 – 1 mg (0.5 – 1 ml of 1:1000) repeat every 10 mins as necessary

IV = 50 – 100 mcg (0.5 – 1 ml of 1:10 000) titrating as required

Adrenaline infusion may be required (refer to hospital protocol)

Establish large bore IV access if not already present and administer colloid/crystalloid. May need 2-4 litres of fluid

Secondary treatment:

Chlorpheniramine 'Piriton' 10 – 20mg IV

Hydrocortisone 200mg

Bronchodilators (salbutamol) as required

Take blood for mast cell tryptase – immediately (although this must not interfere with initial resuscitation) at 1 hour post anaphylaxis and at 6 – 24 hours post anaphylasis (plain red top sample tube). Label clearly and call lab.

Follow-up. Refer to an allergist. Report to the Committee on Safety of Medicines by filling in a "Yellow Card" (found in back of BNF)

Talk to the patient once recovered about what happened

Issue a medic alert bracelet or card

BRADYCARDIA

DEFINITION: Heart rate less than 50 beats per minute

CAUSES:

Surgical

Pneumoperitoneum

Cervical dilatation (gynaecology)

Stretching of spermatic cord (Urology)

Anaesthetic

Hypoxia

Hypotension

Regional anaesthesia

Drugs e.g. metaraminol/phenylephrine

Patient

Perioperative cardiac event

MANAGEMENT:

Do not hesitate to treat as cardiac arrest if necessary

CALL FOR HELP

100% oxygen and ensure adequate ventilation

If hypotensive inform surgeon and turn off vaporiser.

Give fluid bolus (colloid or crystalloid)

Atropine 600mcg or could try glycopyrollate first 200mcg

If unresponsive to the above may need Adrenaline 0.5 – 1ml of 1 in 10 000 solution as bolus (may need to follow with infusion)

If still no response external pacing is required

LARYNGOSPASM

DEFINITION: Glottic closure due to reflex constriction of laryngeal muscles

CAUSES:

Light anaesthesia

Soiling of the larynx e.g. secretions, blood clot post tonsillectomy

Strong stimuli e.g. anal stretch, cervical dilatation

Tracheal extubation

Insertion/removal of airway devices

Recurrent laryngeal nerve damage

Upper respiratory tract infections

More common in children

RECOGNITION/SIGNS:

Stridor during inspiration, however severe laryngospasm will be quiet.

Respiratory distress, sea-saw abdominal movements, tracheal tug

Flat capnograph

Unable/difficult to ventilate

Hypoxaemia/falling oxygen saturation

MANAGEMENT:

Stop stimulus

100% oxygen

CALL FOR HELP

Open airway - chin lift, jaw thrust

Apply CPAP via tight fitting bag and mask

Deepen anaesthesia e.g. propofol (50 – 200mg bolus)

If still no relief, give suxamethonium 100mg (adults)

Consider crycothroidotomy if extremely severe

F IS FOR FLUID AND BLOOD TRANSFUSION

DR JASMEET KAUR

Surgical patients receive approximately 40% of the transfused allogenic blood in the UK. Anaesthetists are involved in the prescription and administration of much of this blood.

The purpose of a red cell transfusion is to improve the oxygen carrying capacity of the blood- see Circulation Chapter 'C' in Handout

TRANSFUSION TRIGGERS

RBC Transfusion not indicated when Hb >100g/L

Hb < 70g/L- strong indication for transfusion

(Transfusion should be given according to the rate of ongoing loss)

Transfusion essential when Hb< 50 g/L

RBC Transfusion less clear when Hb between 70-100 g/L

Available evidence suggests often not justified

Cardiopulmonary reserve needs to be assessed.

Symptomatic patients should be transfused.

WHEN TO TRANSFUSE?

Decision to transfuse should be made on an individual patient basis.

Not all patients agree to transfusion.

Patients need not be transfused to achieve a "normal" haemoglobin concentration.

Need to consider factors such as

- Cause and severity of anaemia
- Patient's ability to compensate for anaemia
- Rate of ongoing blood loss
- Likliehood of further blood loss
- Risk of coronary artery disease
- Balance of risk vs benefits of transfusion

RISKS OF RBC TRANSFUSION

IMMEDIATE IMMUNE REACTIONS Acute haemolytic transfusion(ABO incompatibility) Febrile non haemolytic transfusion reaction Allergic reaction 'TRALI'	**DELAYED IMMUNE REACTION** Graft vs Host Disease Delayed Haemolytic transfusion reaction Alloimmunisation
NON –IMMUNE IMMEDIATE REACTIONS Volume overload ARDS Massive transfusion: coagulopathy, hypothermia, hyperkalaemia, acidosis, citrate toxicity. Bacterial infection	**NON-IMMUNE DELAYED REACTION** Viral infection: HIV, HepB, Hep C, CMV, vCJD Iron overload

BLOOD COMPONENT THERAPY

Red cell transfusion is not the whole story, to achieve haemostasis platelets, clotting factors, fibrinogen also needed.

PLATELETS

Obtained from either pooled plasma or from individual donor

INDICATION:

Need to consider endogenous platelet function as well as actual count

Stable patients: Platelets>$10x10^9$/L in absence active bleeding does not warrant transfusion

Invasive procedures eg: surgery, chest drains, tracheostomy with platelet count< $50X\ 10^9$/L requires platelet transfusion to increase count> $50x10^9$/L

Actively bleeding patient, platelet transfusion required to keep count> $50x10^9$/L

DOSE:

Platelets come in adult bag equivalents. Each dose should raise platelet count by approx. $20x10^9$/L in most adult patients

NB. In many hospitals platelets are ordered from an external site, if you think you might need them order them early

FRESH FROZEN PLASMA (FFP)

Human donor plasma obtained from whole blood. A typical unit is 150-200ml

FFP is stored frozen, need to factor in thawing time when ordering (20mins)

Contains all clotting factors and components of fibrinolytic and complement systems

INDICATION:

May be necessary as empirical treatment of acquired coagulopathy with prolonged INR/APTT given either therapeutically in face of bleeding or prophylactically in non-bleeding subjects prior to surgery/invasive procedures

NB. Little evidence to support prophylactic use of FFP

DOSE:

Acute massive blood loss:

FFP recommended to keep PT/APTT< 1.5x mean control (12-15ml/kg)

CRYOPRECIPITATE

Cryoglobulin fraction of plasma.

Contains: fibrinogen (150-300mg), FVIII, FXIII,VWF

INDICATION:

Aquired coagulopathy related to haemorrhage, trauma, sepsis

Aim is to keep fibrinogen >1.5g/L

DOSE:

Usually issued as 10 pack dose: contains 1.5-3g fibrinogen

PATIENTS PREDISPOSED TO INCREASED BLEEDING

Liver disease

Renal disease

Congenital coagulopathies

Patients taking antiplatelet drugs

Patients taking anticoagulants

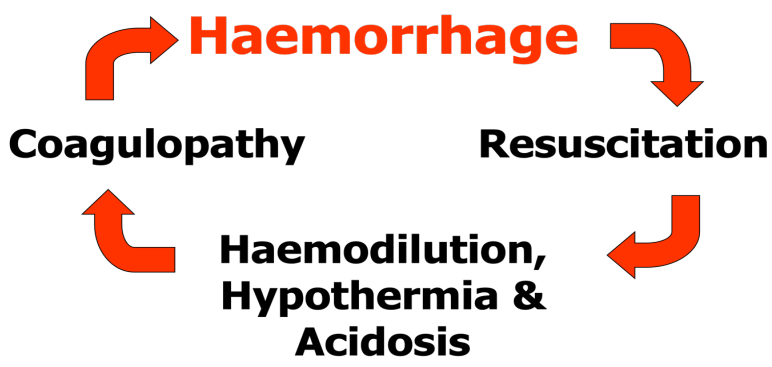
Jehovah's Witnesses- likely not to want any blood products. Discuss in depth first what's OK/not.

MASSIVE HAEMORRHAGE

DEFINITION:

Loss of 1 blood volume within 24hrs
50% blood volume loss within 3 hours
Rate of blood loss of 150ml/min

"Bloody Vicious Cycle" can develop following initial resuscitation of haemorrhage.



Aim of management should be to avoid haemodilution, hypothermia and acidosis by managing aggressively and appropriately.

MANAGEMENT OF MASSIVE BLOOD LOSS

Maintain Hb >80g/L

Maintain platelet count>75 x 10^9/L

Anticipate platelet count <50x10^9/L after 2x volume replacement

Maintain PT and APTT <1.5 x mean control

Give FFP 12-15ml/kg

Maintain fibrinogen >1.5g/L

Avoid DIC

(Based on previous guidelines by British Committee for Standards in Haematology- guidelines have now expired with no replacement however are still a good rule of thumb to follow))

Newer thoughts: Give RBC:FFP, 1:1 if anticipating massive blood loss- but little evidence for this

SUMMARY

GOAL	PROCEDURE	COMMENTS
Restore circulating volume	Insert wide bore peripheral cannulae Give enough warmed crystalloid, colloid, blood Aim to maintain normal BP and urine output >30ml/hr	14G or larger Monitor CVP Blood loss is often underestimated Keep patient warm
Contact key personnel	Clinician in charge & Blood bank Duty anaesthetist, haematologist	Co- ordinator should communicate & document notes
Arrest bleeding	Early intervention	Surgical, obstetric or interventional radiology
Request laboratory investigations	FBC, PT, APTT, Fibrinogen; blood bank sample, biochemical profile, blood gases or pulse oximetry Ensure correct sample identity Repeat FBC,PT, APTT, Fibrinogen every 4 hrs, or after 1/3 blood vol replacement Repeat after blood component infusion	Take samples at earliest opportunity as results may be affected by colloid infusion Misidentification is commonest transfusion risk. May need to give component before results available
Request suitable red cells	**Un-crossmatched group O Rh neg** in extreme emergency No more than 2 units Un-crossmatched ABO group specific when blood group known **Fully crossmatched** If irregular antibodies present When time permits use blood warmer and/or rapid infusion device Employ blood salvage if available and appropriate	Rh pos is acceptable if patient is male or postmenopausal female Lab will complete crossmatch after issuing blood Further crossmatch not required after replacement of 1 blood volume (8 - 10 units) Blood warmer indicated if flow rates >50 ml/kg/hr in adult Salvage contra- indicated if wound heavily contaminated
Request platelets	Allow for delivery time from blood centre Anticipate platelet count <50 x 10^9/L after 2 x blood volume replacement	Target platelet count >100 x 10^9 /L for multiple/CNS trauma or if platelet function abnormal >50 x 10^9/L for other situations
Request FFP	12-15 ml/kg =c750ml= c3 units for an adult 12-15 Aim for PT & APTT < 1.5 x control Allow for 30 mins thawing time	PT/APTT >1.5 x mean control correlates with increased surgical bleeding
Request cryoprecipitate (1-1.5 packs/10kg body wt)	To replace fibrinogen & FVIII Aim for fibrinogen > 1.5g/L Allow for delivery time + 30 mins thawing time	Fibrinogen <0.5 g/L =↑ bleeding Fibrinogen deficiency develops early when plasma poor RBCs used for replacement
Suspect DIC	Treat underlying cause if possible	Shock, hypothermia, acidosis lead to risk of DIC Mortality of DIC is high

Acute Massive Blood Loss Template Guideline. BJA 2000;85:487

CRYSTALLOIDS + COLLOIDS

CRYSTALLOIDS: HARTMANN'S, SALINE, 5% GLUCOSE & 4% GLUCOSE WITH 1/5 SALINE

Pass freely through a semipermeable membrane

Contain water and dissolved electrolytes

Many of them are isotonic with extracellular fluid

Saline based crystalloids (e.g. Hartmann's) will distribute within the *extracellular* space.

5% glucose is effectively giving free water as the glucose is wholly metabolised+ the resulting water will redistribute into all the compartments going mainly intracellularly.

Hypertonic saline (1.8%) is under investigation for use in trauma and resuscitation.

COLLOIDS: GELOFUSIN AND GELOPLASMA

Are larger molecular wt substances suspended, not dissolved, in a carrier solution

Don't pass through a semipermeable membrane

Are less readily filtered at the kidney

Stay in the intravascular compartment longer than crystalloids.

Carrier solution can be saline (gelofusin, voluven) or Hartmann's-l ike (ie balanced eg geloplasma)

STARCH COLLOID CONTROVERSIES- NOW NOT USED IN EUROPE

In the UK we have had 2 colloid types- gelatins (bovine) and starches (plant) suspended in saline or 'balanced' (Hartmann's-like) carriers. In the last few years there have been several ICU/sepsis studies that have had excess renal complications and mortality in the starch group compared to crystalloids. This has led to the withdrawal of the license for starch colloids in the UK, leaving us with gelatins and crystalloids only.

Many people feel unconcerned about the starch withdrawal as there is overall no evidence to say colloids are better, but others feel there is evidence starches are safe in elective surgical (as opposed to ICU) patients.

COMPOSITION

	Na	Cl^-	K^+	Lactate	Ca^{++}	Mg^{++}	Other
Crystalloids							
Hartmann's Solution (CSL)	131	111	5	29	2		
Saline	150	150					
5% Glucose	0	0					Glucose 50 g/l
4% Glucose Saline	30	30					Glucose 40 g/l
Colloids							
Gelofusin, Elohaes(S), Voluven(S), Volplex, Haesteril(S), Albumin	150	120-150					
Haemacell	145	145	5		6		
Geloplasma, Isoplex	150	100	5	30		1-1.5	
Volulyte- a starch	137		4		110	1.5	Acetate 34

From BNF, approximate values in mmol/l Note: Starch based products no longer in use

CRYSTALLOIDS VS COLLOIDS

A controversial subject

Crystalloids	Colloids
Cheap	More expensive
Easy to manufacture and store	Small risk of anaphylaxis (1/2000-3000)
Long shelf life	Smaller volume of infusion needed
No anaphylaxis/allergic reaction	Remain longer in the intravascular space

WHAT + HOW MUCH FLUID IN THEATRE?

There is little evidence to favour colloids or crystalloids, however, recent studies including the 6S trial and the CHEST study have pushed the balance of opinion against starch based colloids and these have been withdrawn in a number of countries. Gelatin based colloids remain in use throughout the UK.

NICE 2013 guidelines govern ward based fluid-

ASSES

500ml resuscitation bolus (of Saline/Hartmann's) over 15 mins , repeated x4 if needed

MAINTAINENCE - see NICE for the formula

Should consider:

History, examination (measures of perfusion)

Deficit (measured + insensible) & ongoing losses (measured + insensible)

Intravascular vs Cellular dehydration

Electrolyte levels

Speed of fluid loss (days/hours/minutes)

Vasodilated / ill patients may need several litres of fluid *before surgery*

Intraoperative: should use Cardiac output monitor for urgent/emergency or major surgery

Serial 200ml colloid challenges with stroke volume (e.g. Oesophageal Doppler) measurement

Ongoing Hartmanns' solution in addition to colloid

Warm fluid to reduce hypothermia

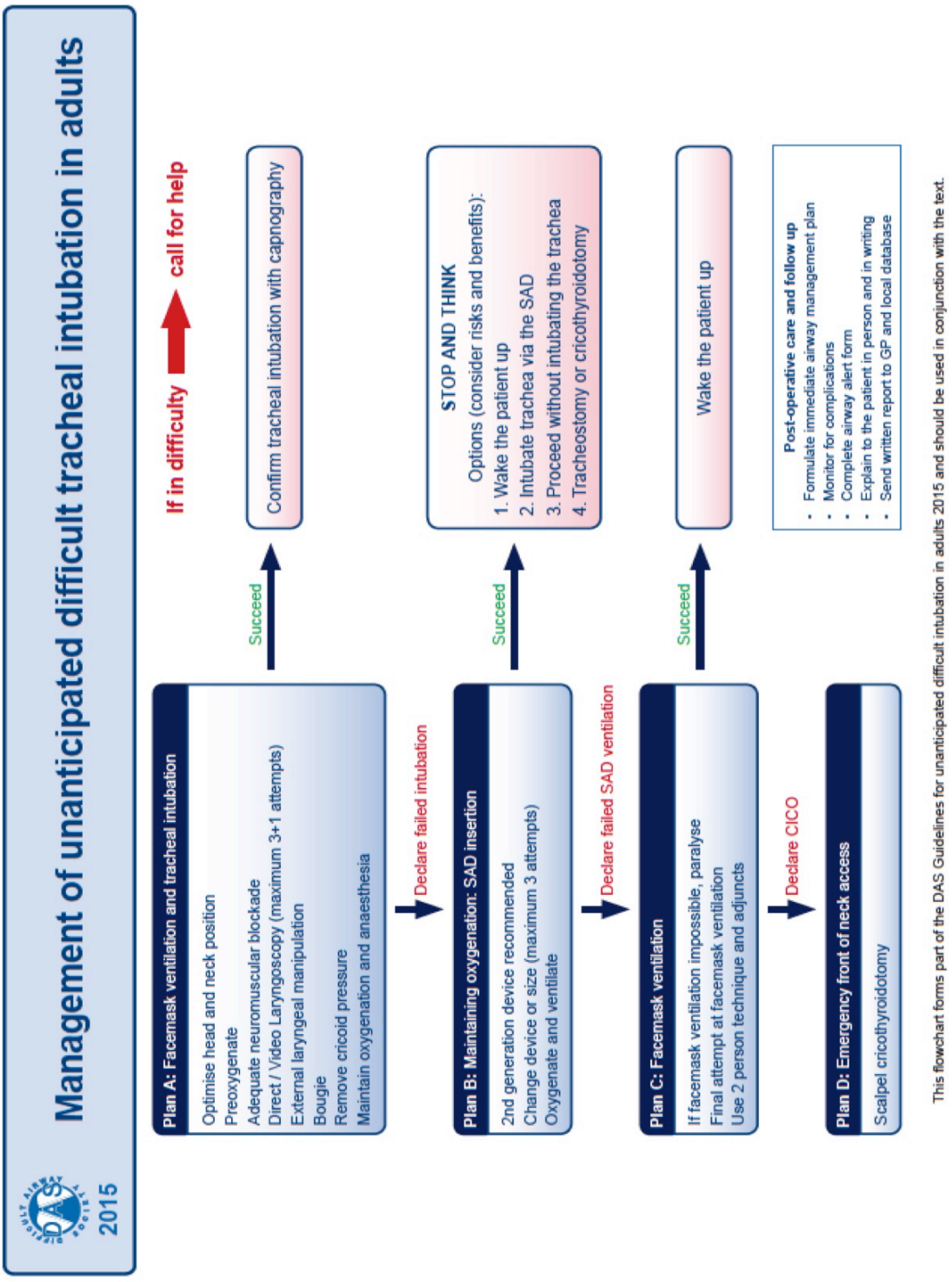
Prescribe postoperative fluids

HYPOVOLAEMIA

History	Clinical signs	Laboratory markers
Vomiting, diarrhoea Intestinal obstruction Fluid intake Thirst	Low urine output Increased capillary refill time Tachycardia Postural hypotension (late sign) Decreased conscious level Low cvp	Raised haematocrit Increased serum lactate Increased urea disproportionate to creatinine Metabolic acidosis Increased plasma osmolarity SV rises on colloid challenge

APPENDIX 1 UNANTICIPATED DIFFICULT INTUBATION

‘Plan A B C D’ from the Difficult Airway Society



APPENDIX 2 MANAGEMENT OF LOCAL ANAESTHESIA TOXICITY

AAGBI Safety Guideline

Management of Severe Local Anaesthetic Toxicity

1 Recognition

Signs of severe toxicity:

- Sudden alteration in mental status, severe agitation or loss of consciousness, with or without tonic-clonic convulsions
- Cardiovascular collapse: sinus bradycardia, conduction blocks, asystole and ventricular tachyarrhythmias may all occur
- Local anaesthetic (LA) toxicity may occur some time after an initial injection

2 Immediate management

- Stop injecting the LA
- Call for help
- Maintain the airway and, if necessary, secure it with a tracheal tube
- Give 100% oxygen and ensure adequate lung ventilation (hyperventilation may help by increasing plasma pH in the presence of metabolic acidosis)
- Confirm or establish intravenous access
- Control seizures: give a benzodiazepine, thiopental or propofol in small incremental doses
- Assess cardiovascular status throughout
- Consider drawing blood for analysis, but do not delay definitive treatment to do this

3 Treatment

IN CIRCULATORY ARREST

- Start cardiopulmonary resuscitation (CPR) using standard protocols
- Manage arrhythmias using the same protocols, recognising that arrhythmias may be very refractory to treatment
- Consider the use of cardiopulmonary bypass if available

GIVE INTRAVENOUS LIPID EMULSION
(following the regimen overleaf)

- Continue CPR throughout treatment with lipid emulsion
- Recovery from LA-induced cardiac arrest may take >1 h
- Propofol is not a suitable substitute for lipid emulsion
- Lidocaine should not be used as an anti-arrhythmic therapy

WITHOUT CIRCULATORY ARREST
Use conventional therapies to treat:

- hypotension,
- bradycardia,
- tachyarrhythmia

CONSIDER INTRAVENOUS LIPID EMULSION
(following the regimen overleaf)

- Propofol is not a suitable substitute for lipid emulsion
- Lidocaine should not be used as an anti-arrhythmic therapy

4 Follow-up

- Arrange safe transfer to a clinical area with appropriate equipment and suitable staff until sustained recovery is achieved
- Exclude pancreatitis by regular clinical review, including daily amylase or lipase assays for two days
- Report cases as follows:
  in the United Kingdom to the National Patient Safety Agency (via **www.npsa.nhs.uk**)
  in the Republic of Ireland to the Irish Medicines Board (via **www.imb.ie**)

If Lipid has been given, please also report its use to the international registry at **www.lipidregistry.org**. Details may also be posted at **www.lipidrescue.org**

Your nearest bag of Lipid Emulsion is kept.....

Malignant Hyperthermia Crisis

AAGBI Safety Guideline

Successful management of malignant hyperthermia depends upon early diagnosis and treatment; onset can be within minutes of induction or may be insidious. The standard operating procedure below is intended to ease the burden of managing this rare but life threatening emergency.

1 Recognition

- Unexplained increase in $ETCO_2$ **AND**
- Unexplained tachycardia **AND**
- Unexplained increase in oxygen requirement
  (Previous uneventful anaesthesia does **not** rule out MH)

2 Immediate management

- **STOP** all trigger agents (anaesthetic vapours, etc.)
- **CALL FOR HELP.** Allocate specific tasks (action plan in MH kit)
- Install clean breathing system and **HYPERVENTILATE** with **100% O_2 high flow**
- Maintain anaesthesia with intravenous agent
- **ABANDON/FINISH** surgery as soon as possible

3 Monitoring & treatment

- Give **dantrolene**
- Initiate active **cooling** avoiding vasoconstriction
- **TREAT:**
  - **Hyperkalaemia:** calcium chloride, $NaHCO_3^-$, glucose/insulin
  - **Arrhythmias:** magnesium/amiodarone/metoprolol **AVOID** calcium channel blockers - interaction with dantrolene
  - **Metabolic acidosis:** hyperventilate, $NaHCO_3^-$
  - **Myoglobinaemia:** forced alkaline diuresis (mannitol/frusemide + $NaHCO_3^-$) may require RRT later
  - **DIC:** FFP, croyprecipitate, platelets
- Check plasma CK as soon as able

DANTROLENE
2.5mg/kg immediate iv bolus.
Repeat 1mg/kg boluses as required to max 10mg/kg

For a 70kg adult

- **Initial bolus: 9 vials dantrolene** 20mg (each vial mixed with 60ml sterile water)
- Further boluses of 4 vials dantrolene 20mg repeated up to 7 times.

Continuous monitoring
Core & peripheral temperature
$ETCO_2$
SpO_2
ECG
Invasive blood pressure
CVP

Repeated bloods
ABG
U&Es (potassium)
FBC (haematocrit/platelets)
Coagulation

4 Follow-up

- Continue monitoring on ICU, repeat dantrolene as necessary
- Monitor for renal failure and compartment syndrome
- Repeat CK
- Consider alternative diagnoses (sepsis, phaeochromocytoma, thyroid storm, myopathy)
- Counsel patient & family members
- Refer to MH unit (see contact details below)

The UK MH Investigation Unit, Academic Unit of Anaesthesia, Clinical Sciences Building, St James's University Hospital Trust, Leeds LS9 7TF. Direct line: 0113 206 5270. Fax: 0113 206 4140. Emergency Hotline: 07947 609601 (usually available outside office hours). Alternatively, contact Prof Hopkins or Dr Halsall through hospital switchboard: 0113 243 3144.

Your nearest MH kit is stored .....

APPENDIX 4 MACHINE CHECKLIST

Checklist for Anaesthetic Equipment 2012

AAGBI Safety Guideline

Checks at the start of every operating session
Do not use this equipment unless you have been trained

Check self-inflating bag available	
Perform manufacturer's (automatic) machine check	
Power supply	• Plugged in • Switched on • Back-up battery charged
Gas supplies and suction	• Gas and vacuum pipelines – 'tug test' • Cylinders filled and turned off • Flowmeters working (if applicable) • Hypoxic guard working • Oxygen flush working • Suction clean and working
Breathing system	• Whole system patent and leak free using 'two-bag' test • Vaporisers – fitted correctly, filled, leak free, plugged in (if necessary) • Soda lime - colour checked • Alternative systems (Bain, T-piece) – checked • Correct gas outlet selected
Ventilator	• Working and configured correctly
Scavenging	• Working and configured correctly
Monitors	• Working and configured correctly • Alarms limits and volumes set
Airway equipment	• Full range required, working, with spares
RECORD THIS CHECK IN THE PATIENT RECORD	
Don't Forget!	• Self-inflating bag • Common gas outlet • Difficult airway equipment • Resuscitation equipment • TIVA and/or other infusion equipment

CHECKS BEFORE EACH CASE

Breathing system	Whole system patent and leak free using 'two-bag' test Vaporisers – fitted correctly, filled, leak free, plugged in (if necessary) Alternative systems (Bain, T-piece) – checked Correct gas outlet selected
Ventilator	Working and configured correctly
Airway equipment	Full range required, working, with spares
Suction	Clean and working

THE TWO-BAG TEST

A two-bag test should be performed after the breathing system, vaporisers and ventilator have been checked individually

i. Attach the patient end of the breathing system (including angle piece and filter) to a test lung or bag.

ii. Set the fresh gas flow to 5 $l.min^{-1}$ and ventilate manually. Check the whole breathing system is patent and the unidirectional valves are moving. Check the function of the APL valve by squeezing both bags.

iii. Turn on the ventilator to ventilate the test lung. Turn off the fresh gas flow, or reduce to a minimum. Open and close each vaporiser in turn. There should be no loss of volume in the system.