

WARWICK

WARWICK MEDICAL SCHOOL

HANDBOOK OF PERI-OPERATIVE MEDICINE

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Preface

This handbook aims to provide a comprehensive knowledge on peri-operative care of surgical patients, including principles of anaesthetic management to phase 2 and phase 3 students of Warwick Medical School. In recent years, in view of improving patient care and safety during surgery, emphasis has been placed on preoperative preparation, optimisation of co-existing medical diseases and improving postoperative outcomes through enhanced recovery pathways. The chapters in this book are selected from the learning outcomes of care of the surgical patient specialty block. In this handbook we have tried to modernise the way in which we teach and learn the principles underpinning much of Medicine. In order to simplify and help you learn, at your own pace, we have added multiple links to videos and online material which should facilitate understanding of the concepts we present.

This handbook is a starting point for you to begin to understand the concepts, principles and importance of peri-operative care and is complimented by the teaching you will receive during your placement in peri-operative medicine. We encourage you to read the book and view the videos to further your knowledge and hope this will invigorate your curiosity in the presented topics.

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

We value your feedback, please email your comments and suggestions to C.Mendonca@warwick.ac.uk

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The pre-operative assessment helps to identify and optimise co-existing medical conditions. It minimises the risk of cancellations on the day of surgery, by ensuring that the patient is optimally prepared and essential resources are identified and co-ordinated.

The key components of preoperative assessment

- A detailed history and clinical examination to identify present and past medical conditions.
- Appropriate investigations based on both medical conditions and proposed surgical procedure
- Optimisation of medical conditions by appropriate multi-disciplinary management
- Decision to continue or discontinue any medications
- Decision to perform the procedure as day case
- Providing detailed patient information on proposed surgical procedure, type of anaesthesia and any special postoperative pain relief techniques

In scheduled and elective cases ideally, an initial pre-operative assessment should be performed immediately following the decision to operate. If the patient is fit and undergoing simple surgery, this can be a one-stop service. Early pre-operative assessment ensures that patients with medical conditions requiring further investigation or treatment are identified early and appropriate action is taken.

In an emergency situation, obviously, the available time is limited and resuscitation and assessment of vital parameters should happen concurrently.

NCEPOD classification of surgical procedures

Table 1.1 The NCEPOD (National Confidential Enquiry into Patient Outcome and Death) categories (Dec 2004):

Category	Description	Timing
NCEPOD 1 Immediate	Immediate life saving operation, resuscitation simultaneous with surgical treatment (e.g. trauma, ruptured aortic aneurysm)	Within mins
NCEPOD 2 Urgent	Operation as soon as possible after resuscitation (e.g. irreducible hernia, intussusception, oesophageal atresia, intestinal obstruction, major fractures)	Within hours
NCEPOD 3 Expedited	An early operation, but not immediately life saving (e.g. malignancy)	Within days.
NCEPOD 4 Elective	Operation at a time to suit both patient and surgeon (e.g. cholecystectomy, joint replacement)	At time to suit patient & surgeon

Fasting guidelines

In view of preventing pulmonary aspiration of gastric contents, patients should not eat or drink prior to anaesthesia. The AAGBI recommends the minimum fasting periods based on the American Society of Anaesthesiologists (ASA) guidelines:

- 6 hours for solid food, infant formula, or other milk.
- 4 hours for breast milk.
- 2 hours for clear non-particulate and non-carbonated fluids

Prolonged fasting can result in dehydration and increases the stress. Therefore, it is encouraged to drink water up to an hour before surgery.

Preoperative investigations

The request for pre-operative investigations should be based on:

- Factors apparent from the clinical assessment
- The likelihood of asymptomatic abnormalities
- The severity of the surgery contemplated

The National Institute of Clinical Excellence (NICE) has developed more elaborate guidelines for preoperative investigations in various patient populations. These are determined by different surgical procedures based on the patient's age and co-morbid conditions.

Table 1.2 Indications for preoperative investigations (NICE 2016)

Investigation	Indication
Full blood count	All adult women Men over the age of 60 years Cardiovascular, Respiratory, Renal or Haematological disease Major surgery
Urea & electrolytes	All patients over 60 years Cardiovascular, Respiratory and renal disease

	<p>Diabetics</p> <p>Patients on steroids, diuretics, ACE inhibitors</p> <p>Cardiovascular and major surgery</p>
ECG	<p>All patients over 60 years</p> <p>Cardiovascular, Renal disease</p> <p>Diabetics</p> <p>Cardiothoracic surgery</p>
Chest X-ray	<p>Cardiovascular and respiratory disease</p> <p>Malignancy</p> <p>Major thoracic and upper abdominal surgery</p>
Sickle cell test	<p>Should be considered in patients from following ethnic origins</p> <p>North Africa, west Africa, South/Sub Saharan Africa</p> <p>Afro Caribbean</p>
Blood group and cross match	<p>If the anticipated blood loss is < 15% of total blood volume and the Hb>13 gm/dl, group and save the sample.</p> <p>If the anticipated blood loss is > 15% of total blood volume then cross matched blood should be available for peri-operative period. One should refer to the local guidelines on Maximum Blood Ordering Schedule (MBOS).</p>

Evaluation of patients with cardiovascular disease

Preoperative cardiac evaluation must be carefully tailored to the patient.

- Acute surgical emergency: rapid assessment of cardiovascular vital signs, volume status, and electrocardiogram (ECG).
- In less urgent circumstances, the situation may include cancellation of an elective procedure, as well as consideration of the special needs of patients with co-morbid disease who undergo surgery

History

- Identify serious cardiac conditions such as prior angina, recent or past MI, congestive cardiac failure, and symptomatic arrhythmias.
- Risk factors such as peripheral vascular disease, cerebrovascular disease, diabetes mellitus, renal impairment, and chronic pulmonary disease.
- Any recent change in symptoms.
- Current medications and dosages.
- Functional capacity (exercise tolerance).

Examination

- General appearance, cyanosis, pallor, dyspnoea during conversation or with minimal activity, poor nutritional status, obesity, skeletal deformities, tremor and anxiety.
- Acute heart failure: pulmonary crackles and chest x-ray evidence of pulmonary congestion correlate well with elevated pulmonary venous pressure
- Chronic heart failure: An elevated JVP or a positive hepatojugular reflux are more reliable signs. Peripheral edema is not a reliable indicator.
- Cardiac auscultation
 - A third heart sound at the apical area suggests a failing left ventricle.
 - A murmur may suggest the presence of valvular disease.
 - Significant aortic stenosis poses a high risk for noncardiac surgery.
 - Significant mitral stenosis or regurgitation increases risk of cardiac failure.
 - Aortic regurgitation and mitral regurgitation predispose the patient to infective endocarditis. If mitral regurgitation is rheumatic in origin or due to mitral valve prolapse, consideration must be given to endocarditis prophylaxis.

Clinical Predictors of Increased Perioperative Cardiovascular Risk

Major

- Unstable coronary syndromes.
 - Recent MI (>7 days but ≤30 days) with evidence of important ischemic risk by clinical symptoms or noninvasive study.
 - Unstable or severe angina. May include “stable” angina in patients who are unusually sedentary.
- Decompensated congestive heart failure.
- Significant arrhythmia.
 - High-grade atrioventricular block.
 - Symptomatic ventricular arrhythmias in the presence of underlying heart disease.
 - Supraventricular arrhythmias with uncontrolled ventricular rate.
- Severe valvular disease.

Intermediate

- Mild angina pectoris (Canadian Cardiovascular Society Class I or II).
- Prior myocardial infarction by history or pathological waves.

- Compensated or prior congestive heart failure.
- Diabetes mellitus.

Minor

- Advanced age.
 - Abnormal electrocardiogram (LVH, LBBB, ST-T abnormalities).
 - Rhythm other than sinus (e.g. atrial fibrillation).
 - Low functional capacity (e.g. Unable to climb one flight of stairs with a bag of groceries).
 - History of stroke.
 - Uncontrolled systemic hypertension.
- **Major predictors** mandate intensive management, which may result in delay or cancellation of surgery unless it is emergent.
 - **Intermediate predictors** are well-validated markers of enhanced risk of perioperative cardiac complications and justify careful assessment of the patient's current status.
 - **Minor predictors** are recognized markers for cardiovascular disease that have not been proven to independently increase perioperative risk.

Functional Capacity (MET levels)

Functional capacity is a measure of exercise tolerance and can usually be estimated from the ability to perform the activities of daily living. It is expressed in metabolic equivalent (MET) levels:

- the oxygen consumption (VO_2) of a 70-kg, 40-year-old man in a resting state is 3.5 ml/kg per minute or 1 MET.

Functional capacity has been classified as

- *excellent* (greater than 7 METs).
- *moderate* (4 to 7 METs).
- *poor* (less than 4 METs). or
- *unknown*.

(Climbing a flight of stairs corresponds roughly to 4 METs)

Evaluation of patients with respiratory disease

Postoperative pulmonary complications following major surgical procedures can be as high as 25-50%. In particular, cardiothoracic surgery and abdominal procedures carry more risk of morbidities due to respiratory complications. Identification of the high risk group and proactive strategies to modify the risks can minimise these morbidities and improve outcome.

Preoperative respiratory risk factors:

- Age > 60 years
- Smoking
- Obesity
- Chronic lung disease, in particular, if the patient is symptomatic at the time of surgery
- Abnormal chest signs
- Abnormal chest radiograph
- PaCO₂ > 6 kPa
- Impaired cognitive function

Pre-operative Investigations

The pulmonary investigations can be divided into:

Static: Lung volumes.

Dynamic: Peak expiratory flow rate (PEFR), FEV₁/ FVC, Flow-volume loop.

Gas exchange: Carbon monoxide transfer factor (DLCO)

Cardiopulmonary exercise testing.

Table 1.3 Restrictive and obstructive lung disease based on FEV₁ and FVC

Lung disease	FVC	FEV ₁	FEV ₁ / FVC %
Restrictive	↓↓	↓↓	Normal
Obstructive	Normal	↓↓	↓↓

In normal healthy subjects, 75 to 80 percent of the FVC is exhaled during the first second; the remaining volume is exhaled in 2 or 3 additional seconds.

FEV₁/FVC ratio gives an indication on the severity of obstructive lung disease

<70 %: mild obstruction

<60%: moderate obstruction

<50%: severe obstruction

Following tests indicate severe lung disease

- FVC < 50% of normal
- FEV₁ <1 Litre.
- FEV₁ / FVC < 50%
- PaCO₂ > 6 kPa

- Carbon monoxide transfer (DLCO) < 55%

Chest radiography

- Rarely reveal anything that might change the decision to perform an operation in patients without other risk factors.
- Indicated in new or changing lung disease and in patients believed to be at high risk for pulmonary complications.

Strategies to improve outcome

- Cessation of smoking
- Treat airflow obstruction with bronchodilators
- Antibiotics: in active infection
- Delay surgery: if surgery is elective and chest / systemic symptoms are still active
- Chest physiotherapy
- Patient education: breathing exercises, continuous positive airway pressure (CPAP) etc.

Preoperative assessment of diabetic patients

When considering the diabetic patient for surgery it is essential to determine:

- Type of diabetes (type 1 or 2)
- Stability of the disease
- Diabetic complications
- Surgical procedure (minor, intermediate, major)

The complications in a diabetic patient can grossly be classified into

- Acute metabolic complications e.g. hypo and hyperglycaemia, dehydration, diabetic ketoacidosis, non-ketotic diabetic acidosis, hypokalaemia
- Chronic end organ damage due to micro and macrovascular disease e.g. nephropathy, autonomic neuropathy, coronary artery disease, peripheral vascular disease

Recognition of these complications is by history, examination and investigation and management involves a multidisciplinary approach involving surgical, diabetes specialist and the anaesthetic team.

Poorly controlled diabetes also increases the risk of post-operative wound infection. HbA1c estimation is a reflection of the integrated blood glucose control over the preceding two to three months. HbA1c values more than 70 nmol/mol are associated with four-fold increase in mortality after cardiac surgery. Preoperative optimization of glycaemic control should aim at achieving HbA1C value less than 69 nmol/mol.

Pre-operative management of the diabetic patient

Three key principles for management of the diabetic patient are:

1. Diabetic medication should be omitted on the morning of surgery.

2. The procedure should be scheduled as early as possible on the list, preferably first.
3. Aim to return the patient as soon as possible to their normal diet and routine medication.

Blood glucose should be maintained at 6 -10 mmol/L during peri-operative period. Hyperglycaemia can occur due to release of catabolic hormones as a result of stress induced by surgery. In situations where, a patient is expected to resume oral intake within an hour of surgery, can be managed by simply postponing their usual diabetic treatment (insulin or oral hypoglycaemic drugs) until they take a delayed meal after surgery. Obviously, it is important that blood glucose is monitored closely. Vigilance is necessary to avoid the risk of hypoglycaemia caused by the delayed action of insulin or oral hypoglycaemic agents taken on the day before surgery or on the morning of surgery in the case of afternoon operations. Patients who are likely to have prolonged starvation period will require a variable rate intravenous insulin infusion (VRIII). The choice of the glucose/insulin infusion regimen is guided by local hospital policy and guidelines.

Perioperative management of anticoagulation

- Surgery should be safely performed without increasing risk of haemorrhage or thromboembolism. A multi-disciplinary policy and guidelines available should be followed and further advice can be obtained from cardiologists or anti-coagulation team.
- Most patients can undergo dental extractions, arthroscopy, arthrocentesis, biopsies, ophthalmic operations and diagnostic endoscopy without alteration of their regimen.
- For invasive surgical procedures, oral anticoagulation needs to be withheld and the decision whether to pursue an aggressive strategy of perioperative administration of intravenous (IV) heparin or subcutaneous (SC) low-molecular-weight heparin (LMWH) should be individualized. Invasive surgery is generally safe when the INR ~1.5.
- It takes approximately 4 days for the INR to reach 1.5 once oral anticoagulant is stopped preoperatively.
- Appropriate guidance should be followed for other newer oral anticoagulants (NOACs). NOACs such as dabigatran, Rivaroxiban and Apixiban are usually stopped 2-3 days prior to the surgery.

Pre-operative drug therapy

- Majority of the routine medication including chronic pain medication should be continued during peri-operative period
- Oral hypoglycaemic drugs and insulin should be omitted on the morning of the surgery
- Patients receiving more than 10mg/day of prednisolone for 3 months or should receive peri-operative supplementary hydrocortisone

- Patients taking oral contraceptive pills and hormone replacement therapy have an increased risk of venous thromboembolism.

Pre-operative risk assessment

ASA classification

The commonly used risk assessment tool is The American Society of Anesthesiologists' (ASA) classification. Based on the severity of co-morbidities, the associated risk is graded into five classes. The addition of 'E' denotes emergency surgery, defined as threat to survival or body part if delayed. Increasing physical status is associated with increasing mortality. Emergency surgery increases risk dramatically, especially in patients in ASA class 4 and 5.

Table 1.4 ASA Classification

Class	Definition	Examples, including, but not limited to:
ASA I	A normal healthy patient	Healthy, non-smoking, no or minimal alcohol use.
ASA II	A patient with mild systemic disease	Mild diseases only without substantive functional limitations. Examples include (but not limited to): current smoker, social alcohol drinker, pregnancy, obesity (BMI < 40), well-controlled diabetes, hypertension and mild lung disease.
ASA III	A patient with severe systemic disease	Substantive functional limitations; one or more moderate to severe diseases. Examples include (but not limited to): poorly controlled diabetes or hypertension, COPD, morbid obesity (BMI ≥40), active hepatitis, alcohol dependence or abuse, implanted pacemaker, moderate reduction of ejection fraction, end-stage renal disease undergoing regularly scheduled dialysis, history of MI > 3 months ago), CVA, TIA, or coronary artery disease/stents.
ASA IV	A patient with severe systemic disease that is a constant threat to life	Examples include (but not limited to): recent history of MI (< 3 months), CVA, TIA coronary artery disease, ongoing cardiac ischaemia, severe valve dysfunction, severe reduction of ejection fraction, sepsis, DIC, end stage renal

		disease not undergoing regularly scheduled dialysis.
ASA V	A moribund patient who is not expected to survive without the operation	Examples include (but not limited to): ruptured abdominal or thoracic aortic aneurysm, massive polytrauma, intracranial bleed with mass effect, ischaemic bowel in the face of significant cardiac pathology or multiple organ/system dysfunction.
ASA VI	A declared brain-dead patient whose organs are being removed for donor purposes	

Other Risk Assessment Tools

American College of cardiology /American Heart Association Cardiac risk stratification for non-cardiac surgical procedures

High (Reported cardiac risk >5%)

- Emergent major operations, particularly in the elderly.
- Aortic and other major vascular surgery.
- Peripheral vascular surgery.
- Anticipated prolonged surgical procedures associated with large fluid shifts and / or blood loss.

Intermediate (risk generally <5%)

- Carotid endarterectomy.
- Head and neck surgery.
- Intraperitoneal and intrathoracic surgery.
- Orthopedic surgery.
- Prostate surgery.

Low (cardiac risk generally <1%)

- Endoscopic procedures.
- Superficial procedures
- Cataract surgery.
- Breast surgery.

Surgical Outcome Risk Tool (SORT) (www.sortsurgery.com) is a pre-operative risk prediction tool for death within 30 days of surgery. It incorporates a wide range of surgical procedures, urgency of procedure and ASA physical status. In addition, age, the presence of cancer also included. The other scoring tools include Physiological and Operative Severity Score for the Enumeration of Mortality and Morbidity (**POSSUM**) and its derivative, Portsmouth Physiological and Operative Severity Score for the Enumeration of Mortality and Morbidity (P-POSSUM).

Further Reading

Barnard NA, Williams RW, Spencer EM. Preoperative patient assessment: a review of the literature and recommendations. *Ann R Coll Surg Eng* 1994; 76: 293-297.

Management diabetes during surgery

<https://bnf.nice.org.uk/treatment-summary/diabetes-surgery-and-medical-illness.html>

Management of adults with diabetes undergoing surgery and elective procedures:
Improving standards

<https://www.diabetes.org.uk/resources-s3/2017-09/Surgical%20guideline%202015%20-%20summary%20FINAL%20amended%20Mar%202016.pdf>

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https://anaesthetists.org/Portals/0/PDFs/Guidelines%20PDFs/Guideline_preoperative_assessment_patient_preparation_anaesthetist_2010_final.pdf?ver=2018-07-11-163756-537&ver=2018-07-11-163756-537

Routine preoperative tests for elective surgery. NICE guidelines 2016
<https://www.nice.org.uk/guidance/ng45>.

BASIC AIRWAY MANAGEMENT

The aim of basic airway management is to provide a patent passage for air to be able to move freely from outside the body through to the trachea or to bypass an obstructed airway. Management of the airway should also provide a means of assisted ventilation or controlled ventilation and should protect the airway from aspiration of gastric contents.

Rapid assessment and management of the airway is crucial in a patient who is critically unwell to prevent hypoxia due to a lack of oxygenation of the brain. Basic airway management allows rapid restoration of oxygen to the vital organs of the body as well as clearance of CO₂ (ventilation) which can lead to severe acidosis.

The Unconscious Patient

An unconscious patient is at risk of losing their airway at any point between their mouth and their trachea. This obstruction may be partial or total. Most commonly the level of obstruction is in the pharynx caused by the tongue. Airway obstruction can be caused by foreign bodies, food boluses or blood clots. In a sub-conscious state laryngospasm can cause severe airway obstruction either due to stimulation or due to laryngeal oedema (burns, inflammation or anaphylaxis). Infra-glottic airway obstruction (below the level of the glottis) may occur in the semi-conscious patient due to excessive secretions, mucosal oedema, bronchospasm or aspiration of gastric contents.

Assessment of the airway should be conducted using a *look, listen* and *feel* approach.

The use of accessory muscles may be evident in a spontaneously breathing patient with airway obstruction. In a patient with complete airway obstruction there is paradoxical movement of the chest and abdomen, this is sometimes known as 'see-saw' breathing. In complete airway obstruction, breath sounds may be absent.

Partial airway obstruction can lead to noisy breathing and the type of noise may suggest the level of the obstruction:

- *Inspiratory stridor* – caused by obstruction at or above the glottic level
- *Expiratory wheeze* – obstruction of the lower airways
- *Gurgling* – due to presence of liquid or semisolid material in the major airways
- *Snoring* – due to partially occluded pharynx by the tongue or palate

It is important to remember that normal breathing is quiet, partially obstructed breathing is noisy while complete airway obstruction is silent. Airway obstruction should be relieved promptly to facilitate adequate oxygen delivery to the lungs.

Basic Airway Manoeuvres

Airway assessment begins by verifying if the patient is able to talk to you. If the patient can talk then their airway is patent. If the airway is noisy or there is no response to voice then certain basic airway manoeuvres can help.

First check that the airway is clear by visually inspecting the oropharynx, this is done by opening the mouth and checking for any foreign body or secretions. This step should take place before attempting any airway manoeuvres as the debris may become an aspiration hazard if the airway is opened prior to suctioning (only suction as far as the eye can see).

There are three manoeuvres that can be used to open the airway and relieve any obstruction:

- *Head tilt* – can be attained by placing one hand on the patient's forehead and tilting the head backwards gently (figure 1). This manoeuvre is contraindicated in the presence of suspected cervical spine disease or injury.



Figure 1: A head tilt demonstrated on a manikin

- *Chin lift*: The patient's chin is lifted to open the airway using the fingertips of the other hand (figure 2).



Figure 2: A chin lift demonstrated on a manikin

- *Jaw thrust*: After identifying the angle of the mandible, the ring and little fingers are placed behind the angle, the index and middle finger is placed over the body of the mandible to apply steady upwards and forward pressure to lift the mandible. The thumbs are used to open the mouth slightly by downward displacement of the chin (figure 3). This is the technique of choice to open the airway in order to facilitate facemask ventilation.



Figure 3: A jaw thrust demonstrated on a manikin

Basic Airway Adjuncts

If the basic airway manoeuvres relieve the airway obstruction it is then important to use an adjunct to maintain airway patency, such as a nasopharyngeal airway (contraindicated in basal skull fracture) or an oropharyngeal airway.

The following videos include indications, technique and complications of insertion of both oropharyngeal and nasopharyngeal airways.

Video on [Oropharyngeal airway insertion](#)

Video on [Nasopharyngeal airway insertion](#)

Oxygen

Oxygen should always be administered in the highest concentration available. A mask with a reservoir bag can give an oxygen concentration of about 85% at flows of 10-15 litres/min. With high flows, an ordinary oxygen mask should deliver oxygen up to 50%, a venturi mask delivers 24-60% depending on the mask chosen. Oxygen administration should not be discontinued except for the brief period for airway interventions designed to improve the patency of the airway. Oxygenation refers to the delivery of oxygen to the lungs.

Ventilation

Ventilation is the term applied to the clearance of carbon dioxide from the lungs. It may be necessary to provide artificial ventilation in a patient who is unable to maintain spontaneous

ventilation by themselves. This can be provided either as assisted ventilation or total ventilation. Ventilation is provided using a facemask (bag-mask) device, i-gel or endotracheal tube. The self-inflating bag is connected to an oxygen source and the reservoir bag allowed to fill. As the bag is squeezed, its contents are delivered to the patient's lungs. As the bag is released the expired air from the patient is vented to the atmosphere and a one way valve allows the reservoir bag to fill with 100% oxygen from the oxygen source. The bag-valve device can be used to ventilate the lungs using just air with an oxygen concentration of 21%.

Airway Management Techniques

There are a number of airway techniques, both basic and advanced that may be required to support a patient's airway:

- Face mask ventilation (basic)
- Insertion of an i-gel (basic)
- Laryngoscopy and tracheal intubation (advanced)
- Fibre-optic intubation (advanced)
- Tracheostomy (advanced)
- Crico-thyroidectomy (advanced)

This [video](#) describes the use of a facemask including the features of a bag-valve system.

i-gel – supraglottic device

The i-gel or Supraglottic Airway Device encompass a number of devices that are designed to facilitate oxygenation and ventilation of the lungs without directly intubating the trachea. Over the years these devices have evolved, initially just consisting of a silicone tube and inflatable cuff to more recently incorporating a gastric bypass channel to provide an escape channel for gastric contents. The second generation devices (such as an i-gel) are available on all resus trolleys across the hospital. They are easy to insert by non-Anaesthetists, provide good airway management in emergency situations and can be life saving devices.

Endotracheal tubes are classed as definitive airway devices. Any cuffed tube that sits within the trachea is a definitive airway as it provides mechanical protection of the airway from soiling (mainly from gastric aspiration).

An i-gel is indicated for managing a difficult airway, where mask ventilation or endotracheal intubation may be difficult, indeed it is part of the Difficult Airway Society (DAS) guidelines (see below) for difficult airway management.

Table 1. Selection of appropriate size of LMA according to patient's weight.

Patients wt. (kg)	<5	5-10	10-20	20-30	30-50	50-70	70-100
LMA size	1	1 ¹ / ₂	2	2 ¹ / ₂	3	4	5
Max. cuff inflation volume (ml)	4	7	10	14	20	30	40

This [video](#) describes the i-gel device, the technique for insertion and common uses.

Advanced Airway Management

The Difficult Airway Society publish guidelines for advanced airway management. These guidelines relate to common scenarios that Anaesthetists may be confronted with in their routine practice. The guidelines are available [here](#).

Endotracheal intubation

Endotracheal intubation is considered to be the optimal form of airway management and is to be used only when trained personnel are available to carry out the procedure. Tracheal intubation is considered superior to other advanced airway management techniques for the following reasons:

1. The airway is reliably isolated from foreign material in the oropharynx.
2. Suction of inhaled particles from the lower respiratory tract is possible.
3. More effective ventilation of lungs.

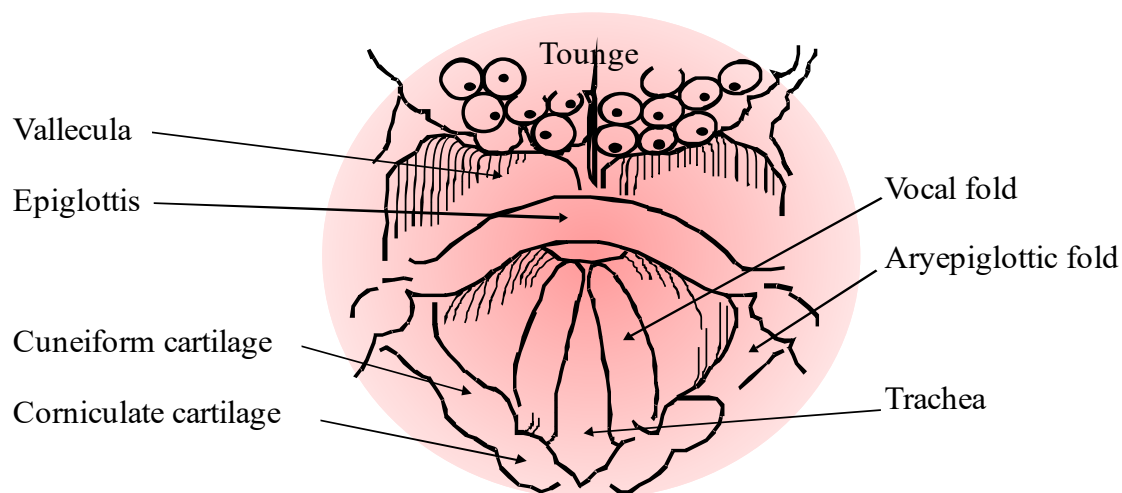


Figure 2.4 Larynx as visualized during direct laryngoscopy

A laryngoscope is needed as well as an appropriately sized endotracheal tube. In some situations, laryngoscopy and intubation may prove impossible as in epiglottitis, pharyngeal pathology etc for which anaesthetic drugs or fiberoptic laryngoscopy may be required. The following are potential complications that can occur:

1. Difficult or impossible to intubate – can occur with epiglottitis, pharyngeal pathology, anatomical abnormalities like short obese neck, receding chin, buck teeth, pathology such as maxillo-facial fractures, facial burns etc. It is important to remember that failure to oxygenate the patient in a case of difficult intubation by bag and mask or any other means can lead to hypoxia and brain damage.
2. Cardiovascular response: Laryngoscopy and tracheal intubation produces sympathetic stimulation resulting in tachycardia and hypertension. In children it can be associated with bradycardia.
3. Damage to the teeth, soft tissue injury of the oropharynx, trauma to the larynx, and trachea.
4. Pulmonary aspiration during the process of induction of general anaesthesia

5. Oesophageal intubation – endotracheal intubation should be confirmed by capnometry as well as visualising the tube passing through the vocal cords wherever possible. Failure to recognise oesophageal intubation can lead to hypoxia and brain damage.

6. Tracheal tube displacement and blockage of the tube with secretions.

7. In patients with unstable cervical spine, laryngoscopy and tracheal intubation may cause further damage.

Extensive training and regular practice is required to acquire and maintain the skills of intubation.

The following [video](#) explains the basics of endotracheal intubation.

Video-laryngoscopes

A recent addition to airway equipment available for intubation is the video laryngoscope used for indirect laryngoscopy. There are various designs, makes and models of video laryngoscopes and some anaesthetic departments in the UK have moved to a universal video laryngoscope model. A video laryngoscope contains a small camera in the blade of the scope and affords the user several advantages over a direct laryngoscope. It provides a greater field of view, requires less manipulation of the airway structures and allows more than one person to learn from the laryngoscopy (as there is a screen to look at). A video laryngoscope can be used for the intubation of a known difficult airway and is cited as an adjunct in the DAS guidelines (see below).

The following [video](#) describes how to use a video-laryngoscope for intubation.

Awake tracheal intubation

Awake tracheal intubation is usually considered in an anticipated difficult tracheal intubation. After explaining the technique, the patient's airway (nasopharynx, oropharynx, larynx and part of trachea) is anaesthetised using local anaesthetic. Tracheal intubation is performed using a flexible intubating fiberoptic scope, before induction of general anaesthesia

The following [video](#) shows a patient's perspective of awake tracheal intubation.

Tracheostomy

Tracheostomy is a surgically created opening in the neck into the trachea. The following are the main indications for tracheostomy.

- To bypass the airway obstruction
 - For certain surgical procedures – e.g. laryngectomy.
 - Trauma involving upper airway when tracheal intubation is likely to be impossible.
 - Inhalation of hot smoke or corrosives – e.g. after burns.
 - Laryngeal dysfunction due to vocal cord palsy or tracheomalacia.
 - Congenital anomalies such as glottic stenosis.

- For tracheal toilet – for long term care of patients with neuromuscular weakness or coma.
- In ICU – for mechanical ventilation lasting more than a few days.

There are no absolute contraindications for tracheostomy, though conditions like coagulopathy warrant special attention before the procedure. Tracheostomy can be done by open surgical method commonly in the operating theatre or percutaneous surgical tracheostomy usually done at the bedside in ICU. Percutaneous tracheostomy offers advantages in that seriously ill patients in ICU do not have to be transferred to the operating theatre, it is quicker and has a lower incidence of bleeding and infection.

There are various complications of a tracheostomy which can be classified as early and late. The main early complication is haemorrhage. Other important complications include misplacement of the tracheostomy tube, perforation of the posterior wall of the trachea and oesophagus and scarring. Tracheo-oesophageal fistula is a rare complication resulting from trauma or necrosis of the posterior wall of the trachea. The tracheostomy stoma will be formed within 3 days, this allows safe replacement of a tracheostomy tube. If the tube is dislodged during the initial 48 hrs, a tracheal dilator is required to replace the tube.

Following a tracheostomy, appropriate care should be taken to prevent infection and atelectasis. Artificial humidity, breathing exercises and regular tracheal suction is required.

Management of a difficult airway

The report of American Society of Anaesthesiologists (ASA) Task Force on the management of difficult airway defines a difficult airway as “the clinical situation in which a conventionally trained anaesthesiologist experiences difficulty with mask ventilation, difficulty with tracheal intubation or both”.

A careful airway examination can predict difficulty in both mask ventilation and tracheal intubation. When a difficult airway is anticipated during the preoperative assessment, there is enough time available to plan the management. As every patient is unique, every patient having an Anaesthetic requires an airway strategy, a method to deal with the unexpected. The airway strategies need to be dynamic to ensure maximal patient safety. These strategies need to be part of the communication between Anaesthetic teams and Surgeons and should be included in the team brief, as part of the WHO Surgical Checklist. In certain circumstances one may not predict a difficult airway but after induction of general anaesthesia it may turn out to be difficult to manage the airway both in terms of bag-mask ventilation (can't ventilate) and in terms of tracheal intubation (can't intubate).

General anaesthesia is a triad of amnesia (unconsciousness), analgesia, control of autonomic reflexes. In addition, neuromuscular blocking drugs (muscle relaxants) are used to facilitate tracheal intubation, mechanical ventilation or the surgical procedure.

Amnesia or unconsciousness may be produced by various drugs, which depress the central nervous system (CNS). Commonly used anaesthetic agents (e.g. propofol, thiopentone) produce unconsciousness in one arm-brain circulation time with transient depression of cardio-respiratory function.

In patients with appropriate fasting, general anaesthesia is induced, usually, by administering a short acting opioid analgesic agent (alfentanil/ fentanyl or IV infusion of remifentanyl) followed by a slow injection of intravenous anaesthetic agent (propofol), with simultaneous assessment of verbal response or eyelash reflex of patient. When unconsciousness is induced, if needed, a neuromuscular blocking drug is administered. The patient's airway is maintained using a supraglottic airway device (laryngeal mask airway/ I-Gel) or through tracheal intubation. General anaesthesia is commonly maintained by inhalational anaesthetic agent and ventilating the lungs with a mixture of oxygen and air.

This [video](#) explains further on process of induction of general anaesthesia. Once the patient arrives to anaesthetic room, a WHO sign in process is completed where identity of patient, consent form and satisfactory functioning of anaesthetic equipment is confirmed. This is followed by appropriate monitoring, preoxygenation and induction of general anaesthesia

In patients with risk of aspiration, a 'rapid sequence induction' (RSI [Video](#)) is performed. In a classical RSI of general anaesthesia, following pre-oxygenation, a predetermined dose of intravenous anaesthetic agent is administered. This is followed immediately by injecting a rapidly acting neuromuscular blocking drug such as suxamethonium. Therefore, airway is secured with shortest time interval following induction, minimising the risk of aspiration. Often pressure is applied to the cricoid cartilage to occlude the oesophagus, to avoid regurgitation of stomach contents into the oropharynx and subsequent aspiration. Due to advances in pharmacology, currently a modified rapid sequence induction is increasingly performed where propofol and rapidly acting intermediate duration neuromuscular blocking drugs such rocuronium is used.

Intravenous anaesthetic agents

Intra-venous anaesthetic agents are used for the following reasons:

- To induce general anaesthesia
- To maintain anaesthesia after induction through an intravenous infusion
- To provide sedation, e.g. in critical care and as an adjunct to regional anaesthesia
- To treat status epilepticus

Thiopentone sodium: It is an ultra short acting barbiturate available in powder form. It is dissolved in water that gives a solution with a pH of 10.5, this causes extreme irritation if injected extra-vascularly or intra-arterially. The dose of thiopentone is 4-7mg/kg. It produces rapid loss of consciousness and induction of anaesthesia is smooth & is rarely associated with involuntary movements or pain on injection.

Recovery from amnesia usually occurs in 5-10 minutes after a single dose. It decreases the cerebral metabolic rate and intracranial pressure. It is anticonvulsant and used in treatment of status epilepticus. Thiopentone causes depression of myocardial contractility and peripheral vasodilatation. Arterial blood pressure decreases with reflex tachycardia. It causes dose-related depression of the respiratory centre. Apnoea is common. Laryngospasm may be precipitated by various stimuli in the oropharynx or larynx.

Propofol: It is a phenol derivative, and the most commonly used intravenous anaesthetic agent because of its faster recovery profile and its antiemetic effect. Propofol is formulated in white aqueous emulsion containing soyabean oil & purified egg phosphatide. The dose is 1.5-2.5mg/kg for induction of anaesthesia. It induces anaesthesia very rapidly (20-40 seconds) and recovery of consciousness is quick (3-5 minutes) with minimal 'hangover' effect. Propofol frequently causes excitatory phenomenon on induction of anaesthesia. Compared to thiopentone it causes greater hypotension, predominantly due to vasodilatation. Propofol causes apnoea more commonly and for a longer duration, than after thiopentone. The greater suppression of laryngeal reflexes enables the insertion of supraglottic airway device with low incidence of coughing or laryngospasm.

Propofol causes pain on injection. Propofol is the most suitable of the agents currently available for use in total intravenous anaesthesia (TIVA) and sedation in ICU because of its significantly less cumulation on prolonged use.

Ketamine: It is a phencyclidine derivative. It produces 'dissociative anaesthesia' rather than generalised depression of central nervous system. Ketamine is presented as a solution in a vial. The dose is 1-2mg/kg. After i.v. injection, it induces anaesthesia in 30-60 seconds and last for 10-15 minutes. Ketamine is also effective within 3-4 minutes after i.m. injection and has a duration of action of 15-25 minutes.

Ketamine is a potent analgesic. Ketamine increases intracranial pressure and it should not be used in patients with intracranial pathology (e.g. - head injury). Recovery from ketamine anaesthesia may be associated with restlessness, agitation and disorientation. Unpleasant nightmares and hallucinations may occur during recovery and up to 24 hours later. Ketamine causes tachycardia, a rise in arterial blood pressure and cardiac output. Muscle tone increases with ketamine and this helps to maintain the airway. Ventilation is well maintained and pharyngeal & laryngeal reflexes are preserved. Ketamine causes bronchodilatation. Ketamine is a favourite induction agent for pre-hospital situations as an analgesic and anaesthetic. It's also useful for induction of anaesthesia in hypotensive patients (e.g. trauma).

Inhalational anaesthetic agents

Inhalational and gaseous anaesthetic agents are mainly used for maintenance of general anaesthesia. Inhalational anaesthetic agents are sometimes used for induction of anaesthesia (e.g. difficult i.v. access). Inhalational anaesthetic agents are delivered through a vaporiser, equipment used to vaporise liquid volatile anaesthetic agent and deliver it to patient in a controlled fashion. Vaporised anaesthetic agent is delivered through breathing circuit to the lungs.

The Minimum alveolar concentration (MAC) of an inhalational agent is an index of its potency. The MAC of volatile anaesthetic agent approximately equals the brain concentration and this can be measured. The oil/gas solubility of an inhalational agent determines its potency. The higher the

oil/gas solubility of an agent, the more potent the drug. The blood/gas solubility of a volatile anaesthetic agent determines the onset & offset of action. The lower the blood/gas solubility of an agent, the faster is the onset & offset of action.

Commonly used inhalational anaesthetic agents in UK are isoflurane, sevoflurane, desflurane. Most of the inhalational anaesthetic agents increase intracranial pressure and cause myocardial and respiratory depression. The degrees by which these effects occur vary with different agents and their concentration. All currently used inhalational anaesthetic agents cause malignant hyperpyrexia in susceptible patients.

Isoflurane: This is a colourless, volatile liquid with a slightly pungent odour. The pungency of the vapour causes coughing or breath holding on induction and therefore isoflurane is not used for induction of anaesthesia. The MAC of isoflurane is 1.15% in oxygen. Isoflurane causes dose dependent depression of ventilation. It increases the respiratory rate but depresses the tidal volume. Isoflurane causes hypotension, predominantly as a result of reduction in systemic vascular resistance. It is metabolised in the liver, minimally, by oxidation.

Sevoflurane: This is a methyl propyl ether. It is non-flammable and has a pleasant smell. The blood/gas partition coefficient of Sevoflurane (0.69) is about half of that of Isoflurane (1.43). This leads to faster onset/offset of action as compared to isoflurane. The MAC of sevoflurane is about 2% in oxygen.

The pleasant smell, which is non-irritant to the respiratory tract and has a low blood/gas partition coefficient – these properties make sevoflurane a popular choice for the induction of anaesthesia. Sevoflurane causes dose dependent depression of ventilation. It relaxes bronchial smooth muscles. It decreases arterial blood pressure, mainly by reducing the systemic vascular resistance, but cardiac output is well maintained in normal anaesthetic concentrations. It is more cardio-stable than isoflurane. Sevoflurane is metabolised in the liver to organic and inorganic fluoride ions. It is absorbed and degraded by soda lime. One of the breakdown products is 'Compound A'. Normally the concentration of 'compound A' produced is well below toxic levels.

Desflurane: It has ethereal, a more pungent odour than isoflurane, and is irritant to the upper respiratory tract. It is therefore not recommended for gaseous induction of anaesthesia. Desflurane has a MAC of 6% in oxygen.

Desflurane has a boiling point of 23.5°C and has a very high vapour pressure at room temperature. These properties demand use of a special vaporizer (TEC 6) which requires a source of electric power to heat and pressurize it. Desflurane has a very low blood/gas partition coefficient (0.42). This, in theory, allows extremely rapid induction of anaesthesia but this is limited by a pungent odour and respiratory irritation. However, it is possible to alter the depth of anaesthesia rapidly and the recovery from anaesthesia is faster than that following any other inhalational anaesthetic agent.

Desflurane is irritant to the respiratory tract and causes coughing, breath holding and laryngospasm and therefore is not preferred for induction of anaesthesia. It decreases myocardial contractility and systemic vascular resistance. Heart rate increases with higher concentration. Desflurane undergoes minimal biodegradation.

Nitrous oxide: It is a sweet smelling, non-irritant colourless gas. It is stored in compressed form as a liquid in cylinders. In the UK, the cylinders are painted blue. The cylinder of nitrous oxide contains liquid and vapour. Nitrous oxide is a good analgesic but a weak anaesthetic. The use of 100% oxygen as the inspired gas may lead to absorption atelectasis in lung units distal to the site of airway closure. Therefore, during general anaesthesia, oxygen is delivered along with nitrous oxide or air to achieve an inspired oxygen concentration of about 33%.

Nitrous oxide has the following disadvantages:

- Nitrous oxide diffuses 25 times faster than nitrogen. So, it rapidly diffuses in to closed spaces. It increases in size of compliant spaces, e.g. pneumothorax, intestinal distension, air embolism. It increases the pressure in non compliant spaces e.g.: middle ear and the intracranial space
- It contributes to post-operative nausea and vomiting
- On prolonged exposure can interact with vitamin B₁₂ and inhibit DNA synthesis, megaloblastic anaemia, fetotoxic effects and neuropathy
- Provides a potential room for error to administer a hypoxic mixture through the anaesthetic machine.

Analgesic drugs

Analgesia is one of the components of general anaesthesia. There are various groups of analgesics used in clinical practice. They are broadly classified in the following groups:

- Simple analgesics
- Opioid analgesics
- Local anaesthetics drugs

Simple analgesics

Most drugs from this group of analgesics also possess a variable degree of anti-inflammatory action and therefore are frequently referred to as NSAIDs (non-steroidal anti-inflammatory drugs). They are mainly administered through oral or rectal routes. They are principally used in the post-operative period for the treatment of mild or moderate pain associated with somatic structures. They may eliminate the need for opioid analgesia in minor surgery and may significantly reduce opioid requirement after major surgery.

e.g. Paracetamol, Diclofenac, Ibuprofen

Advantages: lack of respiratory depression, low incidence of nausea/vomiting and simple to administer.

Disadvantages: gastritis, exacerbation of asthma (in 5-10% of asthmatics), impairment of renal function in susceptible patients and increase bleeding tendency.

Opioid Analgesics

Opioid analgesics are used for the treatment of moderate or severe pain. They are used in the intra-operative as well as the post-operative period. The most commonly used opioid analgesics are alfentanil, fentanyl, morphine and remifentanyl. They are administered mainly by the intravenous route during the intra-operative period. Remifentanyl is an ultra short acting analgesic administered as a continuous infusion. It is an ideal intra-operative analgesic, however its use necessitates that post-operative pain be controlled with some other methods such as regional analgesia.

Advantages: They are very strong analgesics and can be used safely in patients with a history of gastric ulcer or asthma, they have no effect on platelet function.

Disadvantages: Side effects of opioids include nausea, vomiting, sedation, respiratory depression, tolerance, dependence.

Further details of simple analgesics and opioids are considered in chapter 7.

Local Anaesthetics

They are used to block peripheral nerves (e.g. brachial plexus block for upper arm surgery) or to block nerve conduction at spinal cord level (e.g. spinal or epidural block). They are also infiltrated at the site of incision for post-operative pain relief.

e.g. lidocaine, bupivacaine, prilocaine

Advantages: very good quality pain relief, side effects associated with opioids or NSAIDs are absent.

Disadvantages: experience and skill is required to perform nerve blocks, can be associated with motor block during postoperative period.

Further details on pharmacology of local anaesthetics are discussed in chapter 6.

Neuromuscular blocking drugs

Muscle relaxation is one of the three components of general anaesthesia. Muscle relaxation is not always required during general anaesthesia. Muscle relaxant is used during general anaesthesia if the patient needs tracheal intubation, mechanical ventilation or the surgical procedure demands muscle relaxation. These drugs act at the neuromuscular junction.

Depolarising neuromuscular blocking drugs

This group of muscle relaxant is similar in structure to acetylcholine and therefore causes depolarisation at the neuromuscular junction. Only difference is that it acts for a prolonged duration as compared to acetylcholine. Prolonged action leads to exhaustion of the ion channels transmitting signals and muscle relaxation follows. Initial depolarisation causes muscle fasciculations which is a feature of depolarising block.

Suxamethonium: It is the only depolarising muscle relaxant used in current practice. Its use is limited for intubation in patients at increased risk of aspiration. Occasionally it is used for treatment of laryngospasm. Suxamethonium is presented in a 2ml ampoule. The dose is 1-1.5mg/kg. It has a fast onset of action (30-45 seconds) and its action lasts for 3-5 minutes. In recent years, rocuronium has been used as an alternate to suxamethonium for RSI.

Suxamethonium causes muscle fasciculations. Muscle fasciculations results in some degree of muscle tissue breakdown leading to a rise in K^+ level and myalgia in the post-operative period. The myalgia commonly occurs a day after exposure and is described as aches and pains in major muscle groups especially around the neck and shoulders.

Suxamethonium is metabolised by pseudocholinesterase enzyme. In presence of abnormal or deficient pseudocholinesterase enzyme, action of suxamethonium is prolonged for more than 30 minutes and this condition is called suxamethonium apnoea. In susceptible patients, suxamethonium causes malignant hyperpyrexia.

Non-depolarising neuromuscular blocking drugs

This group of muscle relaxants act by competing with acetylcholine at the neuromuscular junction and thereby cause muscle paralysis. Nicotinic receptors are present at the neuromuscular junction and have a role in transmission of impulses from nerve to muscle. Acetylcholine is a neurotransmitter at the neuro-muscular junction.

All non-depolarising muscle relaxants possess at least one quaternary ammonium group to bind to post-synaptic nicotinic receptors at the neuromuscular junction.

As the term suggests, they do not cause depolarisation and so fasciculations are not seen with non-depolarising blockers. In absence of fasciculations, adverse effects like hyperkalemia or myalgia do not occur.

Non-depolarising agents are classified depending on their duration of action.

- Short acting: mivacurium
- Intermediate acting: atracurium, vecuronium, rocuronium
- Long acting: pancuronium

Vecuronium: This is a monoquaternary amine. It is presented in powder form. An intubating dose is 0.1mg/kg. The onset of action is within 2-3 minutes and action last for about 30 minutes. Vecuronium, normally, does not produce histamine release, nor does it have any cardiovascular effects.

Rocuronium: Rocuronium is a monoquaternary amine with a very rapid onset of action for a non-depolarising muscle relaxant. It is presented in a vial as colourless solution. The dose is 0.6-0.9mg/kg. The onset of action is within 60-90 seconds. It is used as an alternative to suxamethonium (if no difficulty anticipated in intubation) in rapid sequence induction of anaesthesia. Its duration of action is about 30 minutes. In most other respects, rocuronium resembles vecuronium.

Atracurium: Atracurium is presented as a colourless solution in an ampoule and used in a dose of 0.5mg/kg. The onset of action is 2.0-2.5 minutes and it lasts for about 20 minutes. Atracurium is unique amongst muscle relaxants as it is metabolised in the body spontaneously by Hoffman degradation (pH & temperature dependent degradation) and ester hydrolysis. Its metabolism is not

dependent on liver or kidney function. Atracurium is relaxant of choice in sick patients with impaired liver or kidney functions.

Atracurium may release histamine and may produce local wheals and flaring around the injection site. This may be associated with a slight fall in blood pressure. A metabolite of Hoffaman degradation, laudanosine, has epileptogenic properties, although this complication has never been reported in humans.

Cis-atracurium: This is a specific isomer of atracurium developed to produce a drug with desired clinical actions but with reduced side effects. It is more potent than atracurium and has a slightly delayed onset & longer duration action.

Its main advantage is that it does not release histamine and therefore is more cardio-stable. It also undergoes spontaneous degradation.

Mivacurium: This drug has the shortest duration of action (~15 minutes) amongst non-depolarising muscle relaxants. So mivacurium is preferred in patients undergoing short duration surgical procedures. The dose is 0.15mg/kg. It is metabolised by plasma cholinesterase enzyme. In the presence of reduced plasma cholinesterase activity due to acquired or inherited factors, the duration of action of mivacurium, similar to that of suxamethonium, may be increased. It also produces histamine release.

Anticholinesterase Drugs

These drugs inhibit the action of acetylcholinesterase at the neuromuscular junction. By inhibiting acetylcholinesterase at the neuromuscular junction they prolong the half-life of acetylcholine. Thus, an increased concentration of acetylcholine, competitively antagonise the action of non-depolarising agents.

Anticholinesterase drugs are used at the end of surgery to reverse the residual neuromuscular block. The most commonly used anticholinesterase during anaesthesia is neostigmine. Anticholinesterases are also used in the treatment of Myasthenia gravis.

Neostigmine is a commonly used anticholinesterase used to reverse the residual effect of non-depolarising muscle relaxants at the end of a surgical procedure. Neostigmine potentiates the action of acetylcholine. The action of acetylcholine at the nicotonic receptor helps to antagonise the effects of muscle relaxants. Acetylcholine by its action on muscarinic receptors produces undesirable effects like bradycardia, salivation, sweating, bronchospasm, increased intestinal motility and blurred vision. These undesirable effects are reduced by the simultaneous administration of an anticholinergic agent such as glycopyrrolate or atropine.

Suggammadex: Suggammadex is a drug that can be used to rapidly reverse the effects of neuromuscular blockade after administration of aminosteroid non-depolarising drugs such as Vecuronium and Rocuronium. This is a modified cyclodextrin that encapsulates the neuromuscular blocking drugs to render them inactive.

Total intravenous anaesthesia (TIVA)

Anaesthesia is induced with an intravenous anaesthetic agent and then maintained with continuous infusion of an intravenous anaesthetic agent instead of a volatile anaesthetic agent.

All the components of general anaesthesia are provided by selecting specific intravenous agents. Syringe pumps (figure 3.1) programmed with pharmacokinetics of specific intravenous agents for target-controlled infusion (target plasma or effect site concentration) are now available.

A combination of propofol and remifentanyl are commonly used. This technique avoids hazards associated with volatile anaesthetic agents (respiratory irritation, operating room pollution, malignant hyperpyrexia). TIVA using propofol is also associated with reduced incidence of postoperative nausea and vomiting.



Figure 3.1 Intravenous infusion through TCI infusion

Anaesthetic machine

It safely delivers oxygen, anaesthetic gases and volatile agents at a desired concentration, as controlled by the operator, to the patient's lungs through a breathing system. It provides a supply of anaesthetic gases, regulates the pressure of anaesthetic gases and oxygen, allows mixing of gases and anaesthetic agents. A basic anaesthetic machine consists of the following components.

- Rigid metal framework with compressed gas source
- Pressure regulators: regulates the pressure of oxygen, air and nitrous oxide
- Pressure gauges: Measures the pressure of oxygen, air and nitrous oxide both in cylinder and pipelines.
- Flowmeters: Reads the flow of oxygen, air and nitrous oxide, recent machines have electronic flow meters.

- Back bar system with vaporiser attachment
- Antihypoxic device: ensures a minimum concentration of oxygen at least 25%.
- High pressure release valve: protects the anaesthetic machine from high pressure due to downstream obstruction.
- Oxygen flush: enables to deliver high flow of oxygen at a rate of 35 L/min.
- Common gas outlet: final mixture of gases and volatile agents leaves the anaesthetic machine and enters the breathing system.



Figure 3.2 A modern anaesthetic machine

Anaesthetic machines should be regularly checked according to the guidelines published by Association of Anaesthetists of Great Britain and Ireland.

A breathing system delivers the mixture of anaesthetic gases from the machine to the patient. A Circle breathing system, Bain system, Magill system and modified T piece system are commonly used. All the waste anaesthetic gases (exhaled from patient and spilled over from anaesthetic machine) are scavenged using a specific scavenging system. Modern anaesthetic machines also incorporate a ventilator, monitor and several safety mechanisms that enables early warning of any problems and possible critical incidents. Recent advances in hardware and software mean that Anaesthetic machines are able to perform self-checks to aid safety and are able to deliver set concentrations of volatile agents to patients.

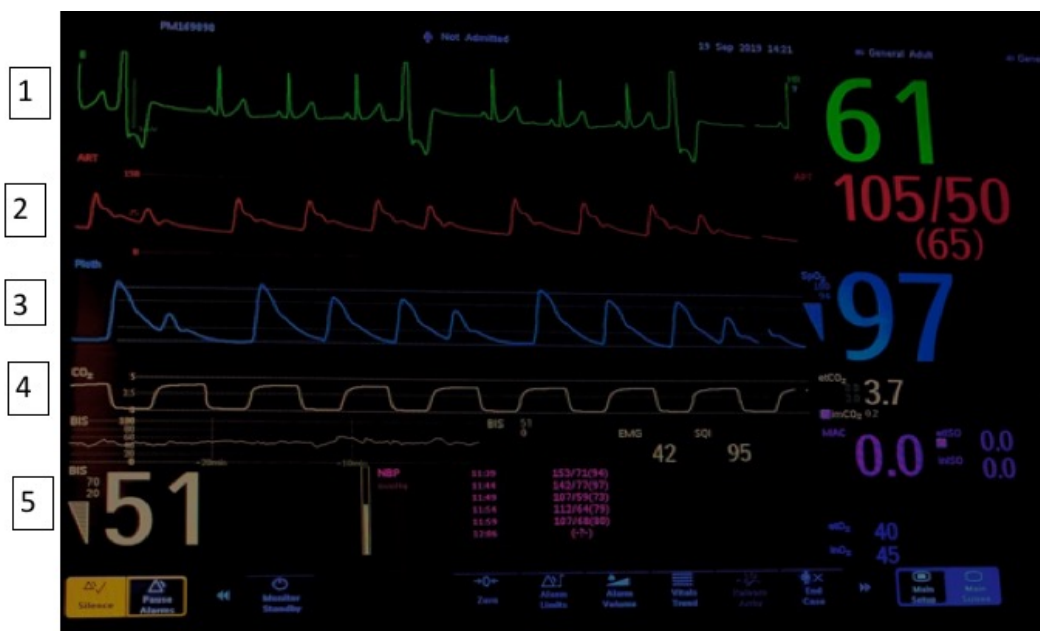
Further reading

C Mendonca, Vaidyanath C. Handbook of Anaesthesia and Peri-operative Medicine. TFM Publishing, Shrewsbury, UK. 2017.

To ensure optimum patient safety certain core standards of monitoring should be used during peri-operative period. Although monitoring provides information that facilitates early recognition of critical incidents, an experienced and vigilant anaesthetist is the most important monitor. However, it is impossible to prevent all adverse events, as human error is inevitable.

During general anaesthesia the inspired oxygen concentration, peripheral oxygen saturation (pulse oximetry), expired carbon dioxide concentration (capnograph) and electrocardiogram (ECG) are monitored continuously. Blood pressure and vapour concentration should also be monitored. In a ventilated patient ventilatory parameters such as tidal volume, minute ventilation and airway pressures should also be monitored. Core temperature, invasive blood pressure, central venous pressure, cardiac output, neuromuscular block, blood loss and urine output monitoring is dictated by the nature of surgery and physical status of the patient. The following monitoring should be established during induction of anaesthesia.

- Pulse oximeter
- Blood pressure
- ECG
- Capnograph
- Neuromuscular monitoring when neuromuscular blocking agent is used.
- Depth of anaesthesia monitoring when TIVA is used



Intra-operative monitoring of vital parameters and anaesthetic agent concentration.

1: ECG, 2: invasive (arterial BP), 3: Pulse oximetry, 4: End tidal CO₂, 5: Depth of anaesthesia (BIS)

During local anaesthesia and sedation continuous ECG, pulse oximetry, non-invasive blood pressure and respiration should be monitored. In addition, a verbal contact should be maintained throughout the procedure.

Clinical monitoring

Despite advances in technology, clinical monitoring plays a vital role because machines that provide us with information may fail. Information provided from the machines should be confirmed by clinical means. This includes extensive monitoring of the anaesthetic machine and equipment, close observation of the patient and the events in the operating theatre. Depth of anaesthesia may be monitored by clinical parameters such as movements, lacrimation, sweating, increase in heart rate and blood pressure. Cardiovascular parameters may be monitored by feeling peripheral pulses, capillary refill and auscultating heart sounds. Respiratory parameters are monitored by observing chest movements, movement and feel of the reservoir bag, auscultating lung fields and observing lip and nail bed colour for cyanosis.

Monitoring with special equipment

In recent years, monitors have become popular due to advances in technology. The use of monitors decrease the cognitive load of the anaesthetist allowing them to perform various other tasks such as preparing drugs, administration of drugs and writing notes.

Pulse oximetry

The pulse oximeter was developed in early 1970s by Aoyagi in Japan. It is the greatest advance in patient monitoring. Its' documented benefit in improved patient safety has led to the widespread use of this technology in anaesthesia. It gives a value of oxygen saturations of the haemoglobin in the arterial blood. It is a simple, reliable and continuous non-invasive method of detecting hypoxaemia.

The pulse oximeter probe consists of two light emitting diodes and a photo-detector. One of the photo-detectors emits light in the red region (660 nm wave length) and the other at the infrared region (940 nm wavelength). De-oxyhaemoglobin (reduced Hb) absorbs maximum light at the red region (660 nm wavelength). At 940 nm absorbance of oxy-haemoglobin is greater than de-oxy haemoglobin. There is a microprocessor in the main unit with an inbuilt algorithm in which the ratio of absorption at red region to infra-red region corresponds to an empirically found saturation value. It produces a waveform of pulsatile flow. It also displays the heart rate derived from the pulse wave form. Pulse oximeters are accurate in the range of saturations of 70 – 100% (+/-2%).

In the following situations the pulse oximeter may not be accurate.

- Presence of abnormal haemoglobin such as carboxy haemoglobin and methaemoglobin.
- Anaemia (below 8g/dl)
- Dyes like methylene blue leads to false low readings
- Reduced peripheral circulation due to vasoconstriction (hypovolaemia, hypotension, cold) or peripheral vascular disease results in an inaccurate reading.
- Venous congestion may result in low readings.
- Bright ambient light can affect the accuracy of the pulse oximeter.
- Motion artefacts such as shivering or seizure activity can result in inaccurate readings.
- Presence of nail varnish may cause falsely low readings.

Electrocardiogram (ECG)

The ECG is a surface recording of the electrical activity of the myocardium. It is recorded by connecting various electrodes through which electrical potentials are measured. The ECG provides information on heart rate, rhythm and some indication of myocardial ischaemia. It doesn't provide any indication about the adequacy of circulation.

The ECG monitoring system consists of the following three components:

- Skin electrodes detect the electrical activity of the heart.
- An amplifier to boost the ECG signal.
- An oscilloscope which displays the amplified signal.

The ECG is recorded at a speed of 25 mm per second and at a standardisation of 1 cm height representing one mV of amplitude. On a standard ECG recording the paper width of a small square (1 mm) represents 0.04 seconds and a large square (5 mm) 0.2 seconds. Five big squares represent a time scale of 1 second and 300 big squares represent a time scale of 60 seconds or one minute. Hence if the rhythm is regular, the heart rate can be calculated by dividing 300 by the number of big squares within one cardiac cycle (between two consecutive R waves).

The lead system: There are 12 conventional leads, 6 in frontal plane (I, II, III, aVR, aVL, aVF) and 6 in horizontal plane (V1-V6). The heart is situated in the centre of the electrical field which it generates. The electrical intensity diminishes as the distance increases from the centre. The lead axis from three standard leads (lead I, II & III) form a triangle known as an Einthoven triangle. In routine practice, monitors with three limb leads are used. Three electrodes are placed as follows

- one on the left arm (LA), usually colour coded as yellow
- one on the right arm (RA), usually colour coded as red
- and one on the left leg (LL), usually colour coded as green or black.

For convenience, during intra-operative monitoring the left leg electrode is often placed over the left side of chest, near the apex beat

Lead I measures the potential difference between the right arm electrode and the left arm electrode. Lead II is derived from negative electrode on the right arm and positive electrode on the left leg, it measures the potential difference between the right arm and left leg electrode. It is usually the best lead for detecting rhythm disturbances. Lead III measures the potential difference between the left arm and the left leg. For detecting ischaemic changes, ST segment should be monitored in appropriate leads. The ST segment changes in lead V1-V4 usually monitor the left anterior descending artery territory, V4-V6 circumflex artery and lead II, III, aVF monitor the right coronary artery territory. When only bipolar leads are used then a modified V5 lead may be used for detecting ischaemia. CM5 is a modified V5 lead configuration where the right arm electrode of lead I is placed over the manubrium sternum, the left arm electrode is placed over the left anterior axillary line in the 5th intercostal space and the ground electrode is placed on the left shoulder.

In a normal ECG, the P wave represents atrial depolarisation, the QRS complex ventricular depolarisation and the T wave represents ventricular repolarisation. The P-R interval represents the time taken for the depolarisation to travel from SA node to the ventricles (via AV node and bundle of His-Purkinje system). The QRS duration represents the time taken for depolarisation to travel through the bundle of His-Purkinje system and ventricular muscles.

- Normal P-R interval is 0.12 -0.2 seconds.
- Normal QRS duration is less than or equal to 0.1 seconds.
- ST segment depression more than 2 mm represents myocardial ischaemia. and the ST segment is elevated in myocardial infarction or myocarditis.

Continuous ECG monitoring should be used for all cases during anaesthesia and sedation. Knowledge of the ECG is essential for appropriate diagnosis of peri-operative arrhythmias and ischaemic changes.

Monitoring blood pressure

Blood pressure is an indirect measure of blood flow and functioning of the circulatory system. It can be measured non-invasively using a cuff and manometer or an automated oscillometric method and invasively by placing a catheter in a peripheral artery.

Non-invasive technique of measuring blood pressure

An aneroid manometer or mercury type manometer (sphygmomanometer) can be used for manual measurement of blood pressure. Mercury sphygmomanometers need regular maintenance with suitable safety procedures in place for using mercury. Automated devices are increasingly used in current clinical practice. The cuff is inflated to a pressure above the expected systolic pressure and then slowly deflated at a rate of 2-3 mmHg per beat. At systolic pressure peripheral pulse appears which can be detected by palpation of radial or brachial artery. On auscultation over the brachial artery characteristic sounds (described by Korotkoff) can be heard. These sounds are described in 5 phases. Phase I begins with the appearance of sounds and corresponds to systolic pressure; phase II, it becomes louder; in phase III there is a rise in volume; phase IV is muffling of sound and phase V is the total disappearance of sounds. Phase V is accepted as diastolic pressure.

For an accurate reading an appropriately sized pneumatic cuff should be used. The maximal occlusion pressure under the cuff is proportional to the inflation pressure and cuff width. A too narrow cuff over estimates the blood pressure and a too large cuff under estimates the blood pressure. The width of the cuff should be 20% greater than the diameter of the arm and it should cover two-thirds of the upper arm. The arm should be supported at heart level.

In an automated BP measurement device a pressure transducer measures the pressure and oscillations. A microprocessor controls the inflation, deflation and display of numerical value.

Disadvantages of non-invasive technique.

- Inaccurate in the presence of arrhythmias.
- Not possible to have a continuous measurement.
- Not reliable in extremes of BP (underestimates when too high and vice versa).
- Pressure effects when used for a prolonged time and frequent readings resulting in petechiae, potentially even nerve palsy.

Invasive technique of measuring blood pressure

The technique of invasive pressure measurement involves placing a cannula in the peripheral artery. Radial, dorsalis pedis, brachial and femoral arteries are commonly used. The system includes a cannula placed in the artery connected to a transducer (figure 4.3). In the transducer, mechanical energy of movement of the diaphragm due to arterial pulsations is converted in to electrical energy and displayed as a blood pressure reading on the monitor. The cannula is continuously flushed with normal saline to prevent clotting.

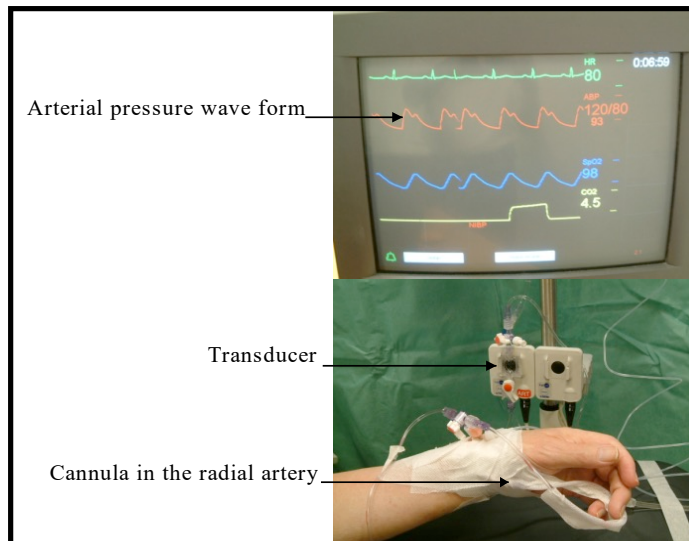


Figure 4.2 Arterial cannula (radial artery) and transducer system.

Indications for direct arterial blood pressure measurement

- Cases where rapid blood pressure changes are anticipated as in cardiovascular disease, major blood loss, cardiac surgery, intracranial surgery and induced hypotension
- Need for frequent arterial blood gas analysis
- Cases where non-invasive blood pressure may be inaccurate: arrhythmias, morbidly obese patients

Disadvantages of invasive technique

- Arterial obstruction and distal ischaemia can occur due to thrombus, haematoma
- Bleeding
- Infection
- Accidental injection of drugs.

Capnography

This is the measurement of carbon dioxide concentration (ETCO₂) in each breath of the respiratory cycle. Following tracheal intubation, continuous waveform of ETCO₂ confirms correct placement of tracheal tube. This [video](#) explains the significance of capnography in confirming tracheal intubation.

Capnograph displays a wave form, from which the respiratory rate and ETCO_2 can be measured. Monitoring expired carbon dioxide concentration provides useful information about the respiratory system, cardiovascular system, metabolism and integrity of the breathing system. During induction of anaesthesia it is used to confirm the placement of a tracheal tube. The contour and pattern of the waveform also provides additional information regarding re-breathing, airway obstruction and wearing off of neuromuscular block.

In healthy patients at normal physiological parameters, ETCO_2 approximates to the partial pressure of arterial carbon dioxide (PaCO_2). In normal lungs the partial pressure ETCO_2 is about 0.5 to 0.8 kPa less than the PaCO_2 .

Hyperventilation, reduced perfusion of alveoli due to reduced cardiac output, reduced metabolism or pulmonary embolism leads to decrease in the ETCO_2 . Increased metabolism, fever, re-breathing and hypoventilation results in high ETCO_2 .

Infrared analysers are most commonly used to measure end-tidal carbon dioxide concentration. Gas molecules having two or more dissimilar atoms absorb infrared light. CO_2 has a strong absorption band at a wavelength of 4.26 μm . The amount of infrared radiation absorbed is proportional to the carbon dioxide concentration.

Monitoring of anaesthetic agent concentration

Most often general anaesthesia is maintained using inhalational (volatile) anaesthetic agents. Inspired and expired concentration of the anaesthetic agent should be continuously monitored. Again infrared analysers are commonly used for measuring anaesthetic concentration.

Monitoring inspired oxygen concentration

In view of avoiding hypoxia it is important to continuously monitor inspired or expired oxygen concentration. Oxygen concentration in a gas mixture can be measured using a paramagnetic technique, or a fuel cell. The partial pressure of oxygen in the blood can be measured using a Clark electrode.

Monitoring depth of anaesthesia

Depth of anaesthesia can be monitored using electro encephalogram (EEG) which involves surface recording of the electrical activities from the cerebral cortex. In practice the information obtained from an EEG is difficult to interpret and the pattern of EEG is also affected by various pathophysiological events like hypoxia, hypotension and hypercarbia. Recently Bispectral index (BIS) monitoring has been used for depth of anaesthesia ([video](#)). This monitor generates a number called the bispectral index on a continuous scale of 0 to 100. 100 represents normal cortical activity and 0 represents no cortical activity. BIS values of 40-60 imply adequate depth of anaesthesia.

Monitoring neuromuscular function

This helps to assess the onset of neuromuscular block, depth of neuromuscular block and adequacy of recovery from neuromuscular block. The principle involves transcutaneous electrical stimulation near a nerve and assessment of muscle contraction. The muscle contraction by visual inspection or by palpation of the muscle or acceleromyography or mechanomyography.

Monitoring temperature

Body temperature usually decreases during the intra-operative period. There is heat loss from radiation, convection, evaporation and respiration. General anaesthesia depresses the thermoregulatory centre and most of the anaesthetic agents produce vasodilatation, facilitating heat

loss. Other factors such as infusion of cold i.v. fluids, exposure of the body cavity, low room temperature all can further increase heat loss. Temperature should be monitored during major surgery and in all cases where heat loss is anticipated.



Figure 4.3 Tympanic thermometer

Temperature probes that are based on the principle of thermocouples are used in clinical practice to measure temperature. Most commonly used sites for temperature measurement are nasopharynx, and tympanic membrane.

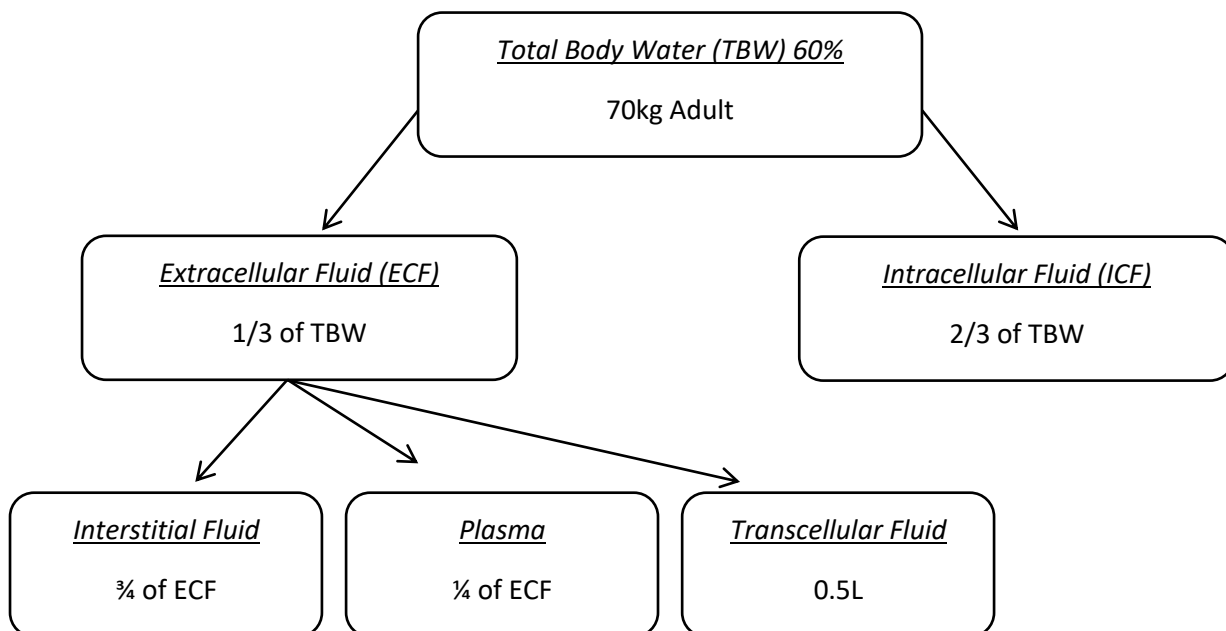
Measurement of central venous pressure, cardiac output and pulmonary artery wedge pressure are sometimes used for major surgery and in patients with cardiovascular disease. Central venous pressure is monitored by placing a long catheter in the internal jugular vein or subclavian vein. A balloon-tipped pulmonary artery catheter is used to assess left atrial pressure and pulmonary artery pressure. Cardiac output can be measured using oesophageal Doppler or by analysing the arterial waveform.

Further reading

Checketts MR, Alladi R, Ferguson K, et al. Recommendations for standards of monitoring during anaesthesia and recovery 2015: Association of Anaesthetists of Great Britain and Ireland. *Anaesthesia*. 2016;71(1):85–93. doi:10.1111/anae.13316

INTRAVENOUS FLUID THERAPY AND BLOOD TRANSFUSION

Intravenous fluid therapy restores the normal circulating blood volume and helps to maintain the perfusion of organs. Total body water in normal adults is around 60% of body weight in men. This proportion is lower in obesity as adipose contains less water than lean tissue. In children, a slightly higher proportion of body weight is represented as water (in infants about 80% of body weight is water).



Maintenance of Fluid Compartments

Semi-permeable membranes separate the above fluid compartments with an active Sodium pump.

- As a result, sodium is the major determinant of the ECF.
- Large anions (glycogen and plasma proteins) are unable to escape from the cell through these membranes so draw Potassium ions into the cell to maintain electrical neutrality.
- Fluid moves across the membranes as a result of osmotic differences
- Under normal conditions, fluid movement across these compartments is governed by ECF Sodium concentration.
- Osmotic pressure and hydrostatic are two of the main mechanisms which control the movement of water.

Regulation of ECF volume

Thirst - Hypovolaemia and changes in osmolarity are detected by osmoreceptors in the Hypothalamus. This leads to an increase in thirst, and an increase in hydration

Osmolality – When increased, the thirst mechanism is activated and ADH is released from the posterior pituitary, which retains fluid from the kidney and dilutes the hypertonic plasma.

Role of the kidney – Thirst and ADH control the intake and excretion of water and the electrolyte composition of the urine is governed by the kidneys. In situations whereby there is reduced Total Body Water (TBW), the kidney aims to reabsorb sodium, which leads to a greater degree of water retention. Changes in the Glomerular Filtration Rate (GFR) and Renin- Angiotensin-Aldosterone axis are paramount in leading to sodium and therefore in turn water conservation.

Pathophysiological effects of fluid imbalance

Illness and injury can affect fluid and electrolyte balance in a number of ways:

Non Specific Metabolic Response to Stress/Injury

Injury or surgery leads to an activation of the sympathetic nervous system that drives a catabolic phase, leading to an increase in cortisol, ADH and aldosterone. When there has been a reduction in ECF volume, Sodium and water are retained.

Transcapillary escape of Albumin – In injury capillary membrane pores increase in size, and albumin no longer remains in the vasculature. This alters fluid retention and leads to a reduction in fluid in the ECF

Specific organ Dysfunction

Cardiovascular – Increased vulnerability of fluid overload

Renal – Impaired clearance or excessive excretion

GI – Increased or decreases GI losses. Ileus can sequester large fluid volumes

Liver – Impaired albumin production and electrolyte balance

Respiratory – High respiratory fluid losses

Neurological – Hypothalamic/Pituitary disorders. Deranged ADH secretion

Dermatological – Burns/Inflammation leads to high loss

Endocrine – Diabetes, Addisons's, SIADH

Assessment of Fluid Balance

Clinically - includes heart rate, skin turgor, hydration of mucous membranes, core-peripheral temperature gradient, pulse rate and volume, BP and urine output. Blood pressure is not a reliable sign, as compensatory mechanisms produce vasoconstriction and tend to maintain blood pressure until severe hypovolaemia has occurred. Hourly urine output of less than 0.5ml/kg is observed in severe hypovolaemia.

Biochemically – Acute dehydration can lead to a haemoconcentration of remaining electrolytes, such as sodium. Haemoglobin can be seen to be elevated alongside haematocrit.

[video1](#) and [video 2](#) explains general assessment of fluid status

Fluid Administration

Before prescribing or administering fluids, it is important to assess the degree of fluid loss or the existing fluid balance. Normal physiological input and output (Table 1.) is a useful starting point, and can help identify the potential cause of disturbance.

Intravenous fluids can be administered for the following three main reasons:

Maintenance: includes a basic requirement. Maintenance fluids are designed to replace water normally lost through GIT, kidneys and normal evaporative losses from the respiratory tract.

Replacement: includes replacing on-going losses. Electrolyte composition of these fluids is equivalent to that of extracellular fluid.

Resuscitation: includes restoration of already lost fluid or blood.

The following factors should be considered during fluid resuscitation.

1. Consider fluid and electrolyte requirements:

Approximate Daily Water Balance in Health

Intake (ml)		Output (ml)	
Water from beverages	1200	Urine	1500
Water from food	1000	Faeces	500-1000
Water from metabolism	300	Insensible losses (skin/lungs)	100

Average Daily Intake

Water	25-30 ml/kg/day
Sodium	1-2 mmol/kg/day
Potassium	0.5-1 mmol/kg/day

2. Consider previous fluid and electrolyte status: The factors leading to fluid depletion include things such as starvation, vomiting, diarrhoea, bowel preparation, burns, pyrexia, haemorrhage and drugs (diuretics) should be considered.

3. *Anticipate ongoing excess losses:* Surgical losses include external losses from the surgical field and fluid sequestration, colloquially called “third space loss” which is found in wound or burns, oedema, ascites and in the gastrointestinal tract.

4. *Consider Repeated reassessment:* Fluid balance should be frequently reassessed during the peri-operative period.

Excessive intravenous fluids during the peri-operative period in patients undergoing major surgery can lead to increased morbidity and prolonged stay in intensive care. The following are recognized consequences of excessive intravascular volume:

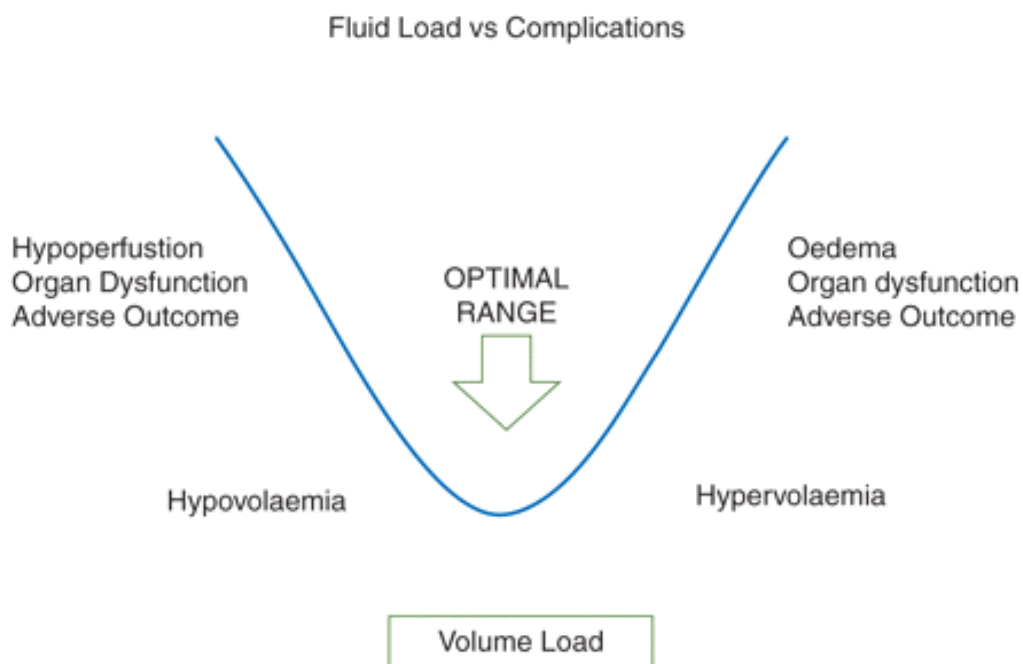
It increases demand on cardiac function and may result in myocardial dysfunction

It increases extravascular lung water and can predispose to pneumonia and respiratory failure.

Excessive intravascular volume increases the workload of kidneys.

It can lead to oedema of the gut which can result in gastrointestinal dysfunction.

Excessive crystalloids and some of the colloids can also cause coagulation abnormality.



Choice of fluid therapy

Crystalloids: Electrolyte solutions that are best used to replace losses from perspiration, respiration and urine output.

Advantages: Cheap, replaces extravascular loss.

Disadvantages: Only $\frac{1}{3}$ of the volume is distributed into the intravascular space, hence a larger volume is needed for replacing the losses. When used in large quantities can result in peripheral and pulmonary oedema

Colloids: Solutions of macromolecular solutes that exert a colloid oncotic pressure to retain fluid within the vascular system. Colloid can increase intravascular volume, preload, cardiac output and tissue perfusion

Advantages: Smaller volumes needed, stays in the vascular compartment for a longer time, reduced peripheral oedema and higher systemic O₂ delivery.

Disadvantages: Risk of anaphylaxis, relatively expensive, can interfere with coagulation system. Hence routine use of colloids is discouraged and are only used with specific indication.

Commonly used crystalloids

Normal saline (0.9% NaCl solution): An isotonic solution that distributes throughout the ECF compartment. Therefore, about ¼ of the administered normal saline will eventually remain in the circulation. **A potential problem of normal saline is hyperchloraemic metabolic acidosis which is more likely in patients with renal insufficiency.**

5% Dextrose: It is an electrolyte-free solution and less than 10% stays in the intravascular space. The glucose component is metabolised and only water remains. This equally distributes into the total body water (TBW). ECF being 1/3 of TBW, only about 333 ml of the administered 1000ml remains in ECF. Of this 333 ml only ¼ remain in intravascular compartment, which is about 84 ml. Intravascular resuscitation is minimal and cellular swelling occurs.

The administered free water causes a decrease in the serum and interstitial electrolyte concentrations (dilutional effect) and may lead to symptomatic hyponatraemia.

Fluid	Osmolality (mOsm/l)	pH	Na + (mmol/l)	K + (mmol/l)	Ca ++ (mmol/l)	Lactate (mmol/l)	Cl – (mmol/l)
Plasma	285 to 295	7.4	142	4	5	27	1
0.9% Saline	308	5.5	154				154
Lactate Ringer's	273	6.5	130	5.4	2.7	29	109

Fluid Challenge

This is a useful tool at assessing fluid responsiveness. In order to assess the response to the change in preload (via fluid administration), cardiac output and stroke volume changes need to be measured. However, in practice, this level of measurement is unavailable, so blood pressure is used as a metric for changes in cardiac output.

Ultimately a successful fluid challenge, whereby the cardiac output increases, indicates the capacity of the system to accommodate further increases in preload to increase the cardiac output. Fluid challenges should be delivered using volumes of 250-500ml fluid depending on the patient's cardiovascular status and co morbidities.

Blood Transfusion

The primary purpose of transfusing blood is to improve organ perfusion and tissue oxygenation through increasing O₂ carrying capacity.

Ultimately one haemoglobin molecule can bind four molecules of oxygen for transport around the body.

The decision to transfuse should take into account several factors.

- Acute or Chronic blood loss (haemodynamic status)
- The expected amount of further blood loss
- The current Haemoglobin level
- The presence of coexisting cardiovascular disease
- Anaemia

The WHO defines anaemia as a condition whereby the number of Red Blood Cells (RBC) and their oxygen carrying capacity is insufficient to meet the body's physiological requirements. Current definitions of anaemia:

Men - <130g/dl, Women (Not pregnant) - <120g/dl

Transfusion Targets

Large randomized controlled trials have indicated a restrictive blood transfusion target is as effective as the use of using liberal targets, therefore aim for the following transfusion targets:

- 70g/dl, aiming for a haemoglobin concentration target of 70-90g/dl
- 80g/dl, aiming for a haemoglobin concentration target of 80-100 g/dl for patients with acute coronary syndrome.

A single unit policy of blood transfusion is now widely accepted for stable patients without active bleeding, as this helps to minimize the risk of transfusion, and improves the economics associated with blood administration.

Blood Components

All blood components supplied by the UK transfusion services are leucodepleted to decrease the risk of potential transfusion transmitted variant Creutzfeldt-Jakob (vCJD) disease. Other benefits of leucodepletion include a reduced incidence of non-haemolytic febrile transfusion reaction as well as reduced transmission of other leukocyte associated viruses such as cytomegalovirus.

Packed red cells: In an emergency and extreme situations, it may be necessary to use uncrossed matched group O blood. In pre-menopausal females if the blood group is not known, O Rh negative blood should be given to reduce the risk of haemolytic disease of the newborn in subsequent pregnancy.

Platelets: platelets are collected from the pooled buffy coats of the whole blood or by individual donor apheresis. They may be stored up to 5 days on an agitator at 22°C. During surgery and invasive procedures platelets should be transfused if the platelet count is less than 50 x 10⁹/L. In a stable patient, in the absence of bleeding a platelet count > 10 x 10⁹/L may be accepted.

Fresh Frozen Plasma: This is produced by centrifugation of whole blood and frozen to minus 30°C to achieve factor VIII concentration > 0.7iu/ml. It should be thawed before transfusion and thawed FFP is best used immediately, although it may be stored at 4°C and transfused within 24 hrs. FFP is used to treat acquired coagulopathy with prolonged INR, PT or APTT to 1.5 times the normal.

Cryoprecipitate: This is the cryoglobulin fraction of the plasma obtained by thawing a single donation of FFP at 4°C. It is rich in factor VIII, von Willebrand factor, factor XIII and fibrinogen. It is

used if the fibrinogen level is less than 1 g/dl in acquired coagulopathy related to haemorrhage, sepsis and trauma.

ABO System

Blood Type	Incidence	Antigens on RBC	Antigens in plasma	Packed RBC Compatibility
O	45%	No antigens	A and B	O
A	40%	A	B	A, O
B	10%	B	A	B, O
AB	5%	A and B	No antigens	A, B, AB, O

O- is the universal donor group, whereas AB+ is the universal acceptor group

Complications of blood transfusion

Most transfusion reactions are mild and can be treated symptomatically. They can broadly be classified into immunological and non-immunological;

Immunological

- *Acute Haemolytic Transfusion Reaction (1 in 250,000 transfusions)*
- ABO Incompatibility – leads to severe destruction of RBC
- *Non haemolytic febrile transfusion reaction (3% of transfusions):* Due to cytokine release from donor RBCs. Onset within 1-6hours of start
- *Anaphylaxis (1 in 50,000 transfusions):*As a result of recipient IgA deficiency. Treated with Adrenaline
- *Transfusion Related Acute Lung Injury (<0.08% of transfusions):* Activation of recipients granulocytes. Usually due to massive FFP/Platelet transfusion
- Delayed Haemolytic Transfusion Reaction (Rare)
- Seen in patients previously sensitised to specific RBC antigens

Non – Immunological

- *Transfusion Associated Circulatory Overload (TACO):* Clinical state of fluid overload as a result of massive transfusion. Patients develop signs of hypervolaemia, and imaging can often demonstrate pulmonary oedema. Treatment is usually supportive with diuretics
- *Hyperkalaemia:* Plasma concentration of RBC increases during storage. However, this only becomes an issue if the patient is also hypothermic and acidotic.
- *Hypothermia:*RBCs are stored at 4°C, and administration can lower the core temperature, impairing coagulation, and reducing tissue oxygen delivery.
- *Hypocalcaemia:* Citrated complexes form with storage medium that reduces overall Calcium
- *Infections*
Viral – HIV/Hepatitis A, B, C
Bacterial
Parasitic – Malaria
- *Acute Haemolytic Transfusion Reaction:* This is one of the most feared reactions and causes significant systemic derangement. Clinical features include fever, chills, nausea, flushing, pruritis, urticaria, flank pain and dyspnoea. Renal failure, DIC and jaundice are significant associated complications of haemolytic transfusion reaction.

- **Management:**
Immediate cessation of transfusion
- Call for help, and inform on call haematologist
- Confirm Diagnosis
- Repeat cross-matching
- Coomb's test
- Supportive Care
- Immediate infusion of saline to promote diuresis and manage hypotension
- Consider vasopressors to maintain blood pressure
- Cardiac monitoring (significant risk of hyperkalaemia)
- Repeat coagulation studies (administer FFP/platelets if DIC develops)

Vigilance is key, as is clear patient identification before drawing blood for cross matching and administration of blood products.

Massive haemorrhage

Usually defined as the loss of one blood volume within a 24 hour period (blood volume in an adult is 70 ml/kg body weight and in infants 80-85 ml/kg body weight). It can also be defined as more than 50% of blood volume loss within 3 hours or blood loss at a rate more than 150 ml/min.

Management of massive blood loss

- Airway, Breathing, Circulation
- Summon for help
- Restore the circulating volume immediately (with crystalloids, colloids) to maintain tissue perfusion and oxygenation.
- Surgical control of bleeding
- Treatment of coagulopathy with blood components. Once stabilized patient will require more intensive monitoring in a controlled environment.

Major haemorrhage and massive transfusion associated with several complications and increased mortality. Appropriate locally agreed guidelines are useful in early identification and effective management.

Further reading

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Regional anaesthesia involves the introduction of drugs with the intention of blocking the nerve supply to a specific part of the body such as a limb. In most cases it provides a safer alternative to general anaesthesia as well as prolonged postoperative analgesia. Regional anaesthesia is achieved by using local anaesthetic drugs that block nerve conduction.

Types of Regional Anaesthesia

Central Neural Blocks

Spinal anaesthesia (intrathecal or subarachnoid block)

Epidural anaesthesia

Caudal anaesthesia

Peripheral Nerve Blocks

Intravenous Regional Anaesthesia (IVRA)

Topical and Infiltration anaesthesia

Others: Intrapleural analgesia, ophthalmic anaesthesia

Advantages of regional anaesthesia

Conscious patient - able to warn of adverse effects (during carotid surgery, and trans-urethral resection of prostate), less interruption of oral intake.

Avoidance of adverse effects of general anaesthesia like nausea, vomiting, sore throat and hang over.

Effects of general anaesthesia, respiratory function and mechanics can be avoided when appropriate regional technique is chosen.

Avoids hazards of unconsciousness like aspiration of gastric contents, anatomical damage to skin, joints, nerves etc.

Improved postoperative pain relief, decreased narcotic use, faster recovery.

It reduces stress response to surgery.

Reduced blood loss particularly with pelvic and hip surgery.

Decreased incidence of pneumonia, and DVT.

Complications of regional anaesthesia

- Technical: failure of the technique, direct trauma to nerves and blood vessels (bleeding and haematoma), pneumothorax with intercostal and intrapleural block.
- Excessive local anaesthetic volume can result in total spinal during epidural and phrenic nerve block during brachial plexus block.
- Those related to specific technique: Hypotension, bradycardia and headache following spinal or epidural analgesia. Rare possibility of nerve injury with peripheral nerve blocks.
- Drug related: Local anaesthetic toxicity due to intravascular injection or systemic absorption, overdose of local anaesthetic, anaphylactoid reaction and methaemoglobinaemia (prilocaine)

Contraindications

Absolute: Patient refusal, anaesthetist's inexperience and localised infection at the

site.

Relative: Abnormal anatomy or deformity, coagulation disorders, neurological disease.

Pharmacology of local anaesthetic drugs

- Local anaesthetic drugs can reversibly block nerve conduction and produce loss of sensation. They can be classified into amides or esters depending upon the chemical link between the amino and aromatic chain.
- Esters contain ester linkage and are relatively unstable in solution. They are hydrolysed in the body by plasma esterases. They are more likely to produce hypersensitivity reactions due to para-amino benzoic acid which is one of the breakdown products. Examples: cocaine, procaine and amethocaine
- Amides contain amide linkages and are stable in solution. They are metabolised by amidases in liver. Hypersensitivity reactions to amides are very rare.
- Examples: lignocaine, prilocaine, bupivacaine and ropivacaine.

Mechanism of action

Most local anaesthetics are weak bases. When deposited in tissues (which normally have alkaline pH) they dissociate into ionised and unionised forms. The unionised form can cross biological membranes and enter into the neurons. Within the nerve cell the molecules again dissociate into ionised and unionised form. Here the ionised component blocks the sodium channels from inside and blocks the conduction of impulses.

Clinical features produced by the block are affected by:

- Patient variables like age, fitness, pregnancy etc.
- Individual drug characteristics
- Concentration and dose used: Higher concentration and dose reduces the onset time, and leads to increased density and duration of block
- Site of injection: Injection to the site with high vascularity results in increased systemic absorption of the drug.
- Additives: vasoconstrictors such as adrenaline and felypressin reduce absorption and prolong the block
- Hyaluronidase increases the tissue penetration and improves the spread of local anaesthetic, used in eye blocks.
- Dextrose is used in spinal anaesthetic solution to increase baricity.

Toxicity of local anaesthetics

Systemic or localised toxicity usually occurs due to accidental intravascular injection of local anaesthetic, subarachnoid injection or use of excessive dose. It primarily involves the central nervous system and cardiovascular system. Initial symptoms in an awake patient include feeling of light headedness, dizziness and circumoral numbness. This then progresses to drowsiness, muscle twitching and generalised convulsions. The respiratory centre may be involved, resulting in respiratory arrest. Cardiovascular toxicity usually occurs at a higher dose than that needed for CNS toxicity. It depresses the pacemaker activity and results in bradycardia and sinus arrest.

Treatment of local anaesthetic toxicity

- Summon for help
- Stop injecting the drug

- Airway, breathing and circulation: The airway should be maintained and 100% oxygen should be administered by facemask. Check pulse, blood pressure, oxygen saturation and ECG.
- Treat the convulsions using diazepam 2.5 mg i.v or lorazepam 4 mg i.v. or thiopental 50 mg i.v.
- Manage bradycardia, hypotension and associated arrhythmias.
- Occasionally adrenaline may be necessary to treat hypotension.
- Any concurrent acid base and electrolyte abnormalities should be corrected.
- **In severe toxicity leading circulatory arrest**, start CPR and give intravenous lipid emulsion (Intralipid). Initial dose is 1.5ml/kg over 1 minute of 20% lipid emulsion, followed by IV infusion of 15ml/Kg/hour. Bolus dose can be repeated at 5 minute interval (maximum three doses)

Commonly used local anaesthetics

Lignocaine: It is an amide local anaesthetic with fast onset of action. It has a moderate duration of action, about 1-2 hours. It produces moderate vasodilatation. It is less toxic than bupivacaine. It is used for infiltration of surgical wound sites, epidural anaesthesia and for selected nerve blocks.

Maximum dose: 3 mg/kg for plain solution and 7 mg/kg with adrenaline

Bupivacaine: It is an amide local anaesthetic with moderate onset of action. It has a long duration of action, about 2-4 hours. It is more cardio-toxic than other local anaesthetics.

It is more potent than lignocaine. It is used for infiltration, epidural, spinal and peripheral nerve blocks.

Maximum dose: 2mg /kg

Levobupivacaine: It is a levorotatory enantiomer of racemic bupivacaine. Clinically it is similar to bupivacaine. The important difference is that it is less cardiotoxic.

Maximum dose: 2mg /kg

EMLA cream is a **Eutectic Mixture of Local Anaesthetics**. It is a mixture of 2.5% prilocaine and 2.5% lidocaine, used for topical anaesthesia. It should remain in contact with the skin for 60 minutes to produce adequate analgesia. Commonly used in children to provide analgesia during vene-puncture.

Amethocaine is an ester, used similar to EMLA to produce topical anaesthesia. It has faster onset and longer duration of action as compared to EMLA cream.

Practical aspects of using local anaesthetics

Dose: Should not exceed maximum allowable dose to avoid toxicity.

Toxicity also depends on the vascularity of the site of injection and metabolic status of the patient.

Should choose the drug with least toxicity. For example, levobupivacaine instead of bupivacaine or lignocaine instead of bupivacaine.

Dose calculation: Local anaesthetic drugs are presented as percentage solutions. For example, 1% lignocaine contains 1gm of lignocaine in 100ml of solution or 10mg per each ml of solution. For a patient weighing 70 kg, a total dose of 210 mg for plain lignocaine, one can use 21 ml of 1% lignocaine or 42ml of 0.5% lignocaine. Adrenaline is usually added at a dilution of 1:200,000. That

means each ml of solution contains 5 microgram of adrenaline (1gm in 200,000ml, 1mg in 200ml, 1000microgram in 200ml).

Spinal Anaesthesia

Spinal anaesthesia was first performed for surgery by August Bier in 1899. It can be used for surgical procedures to the lower part of the body, usually below the level of the umbilicus. It is commonly used for Caesarean section, inguinal hernia repair, pelvic surgery, transurethral resection of prostate and lower limb surgery. It can be used in combination with general anaesthesia to provide intra-operative and postoperative analgesia. Spinal anaesthesia is produced by injecting a small volume of local anesthetic in to the cerebrospinal fluid (CSF) in the subarachnoid space.

Mechanism of Action

Spinal anaesthesia results in a rapid onset of block, usually within 3-5 minutes, depending on the local anaesthetic drug used. Maximal effects may take up to 30 minutes. The following effects are produced by blocking various nerve fibres during the onset of spinal anaesthesia.

Autonomic block: Smaller sympathetic fibres are more easily blocked than larger sensory and motor fibres. Hence, the 'sympathetic' block appears earlier than sensory or motor block. Block of thoracolumbar (T1 –L2) sympathetic outflow produces vasodilatation, reduced venous return and hypotension.

Sensory block: level of block produced depends on the volume of drug injected into the CSF. Sensory block results in loss of pain, temperature sensation. Pressure sensation is usually preserved.

Motor block: Results in weakness of lower limbs, abdominal muscles and if it extends to the thoracic segments produces weakness of the intercostals muscles. As the diaphragm is innervated by the phrenic nerve (C3-5), respiratory function can still be maintained.

Anatomy

The spinal cord terminates at L1 or L2 in adults and L3 in infants. The line joining the top of the iliac crests corresponds to L4 vertebral level and is called Tuffier's line (figure 6.1). The subarachnoid space ends at S2 in adults. The subarachnoid space extends laterally along the nerve roots to the dorsal root ganglia.

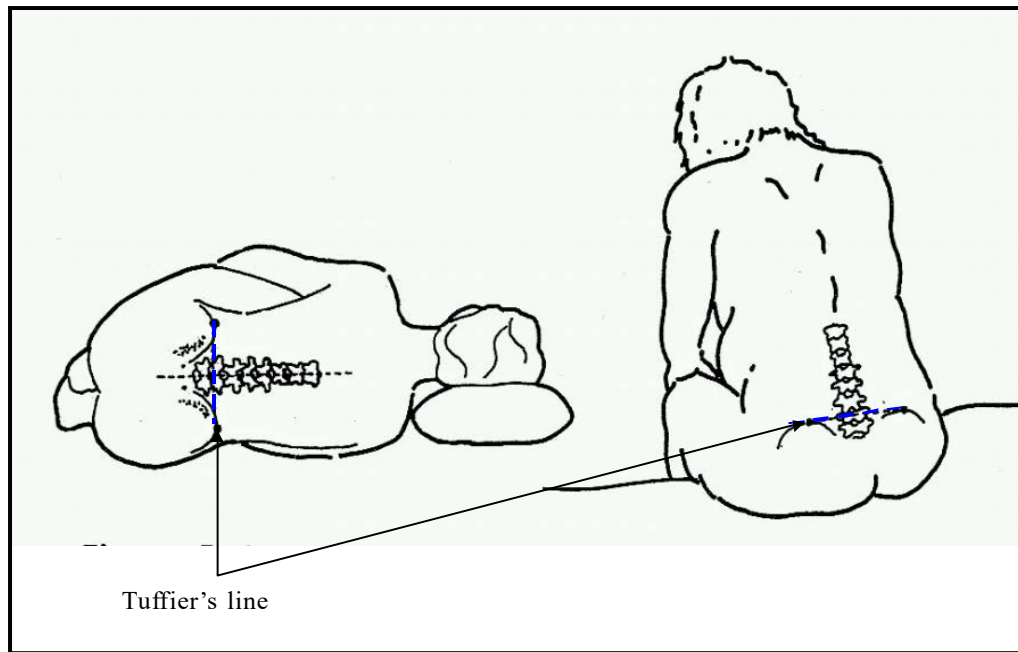


Figure 6.1 Landmarks for spinal and epidural anaesthesia

Technique

A complete preoperative assessment of the patient should be performed and informed consent should be obtained. Facilities for resuscitation and progression to general anaesthesia must be available. Intravenous access should be secured before commencing the block and standard monitoring should be established.

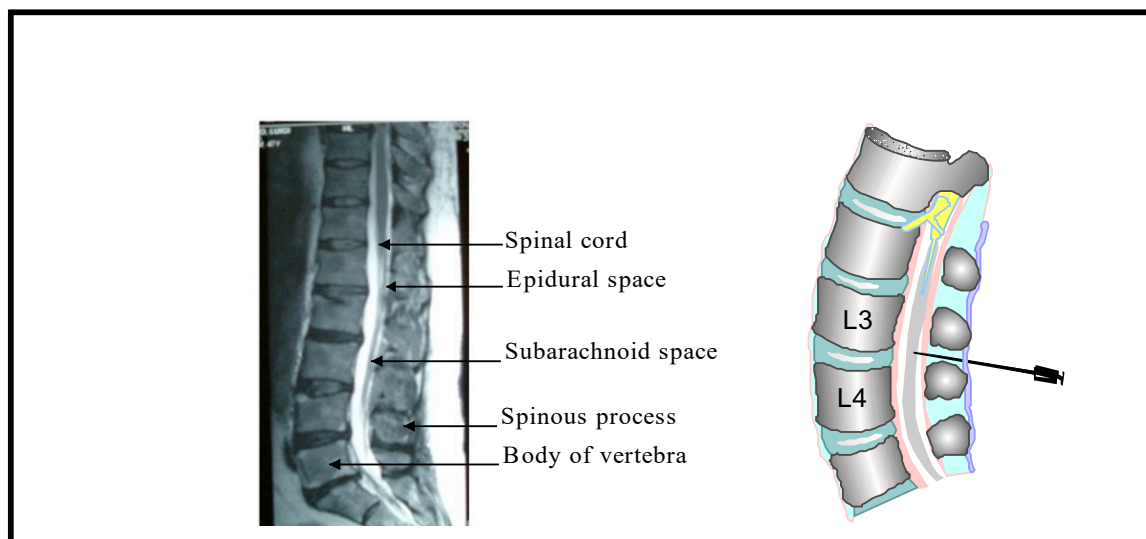


Figure 6.2 MRI scan: Anatomy of epidural and subarachnoid space.

The patient should be sitting or lying on their side. Flexion of the lumbar spine opens the intervertebral spaces. The appropriate interspinous space should be identified (usually L3-4 or L4-5). The Anaesthetist scrubs and dons a sterile gown and gloves. The back is cleaned using standard antiseptic solution and draped. The chosen interspace is infiltrated with local anaesthetic. The spinal needle is inserted in the midline, aiming slightly cranially. Resistance increases as the ligamentum flavum is entered and with further advancement of the needle the dura is encountered, with a sudden "give" as the dura is pierced. Correct placement of the needle is confirmed by cerebrospinal fluid at the hub. Spinal injection can also be performed using the para-median approach. After confirming the correct placement of local anaesthetic drug, it is injected into the CSF. Usually a volume of 2 to 3 ml is injected depending on the level of block required and the physical status of the patient. Either heavy bupivacaine 0.5% or plain bupivacaine 0.5% is commonly used. Fentanyl 15 to 25 micrograms is commonly added to improve the quality of block. Other opioids such as morphine or diamorphine are also used for major abdominal or pelvic surgeries to provide post-operative analgesia. In situations where long acting opioids are administered into the spinal space, patient should be closely monitored for respiratory depression in high dependency care unit or post anaesthesia care unit. This [video](#) describes the procedure of spinal anaesthesia.

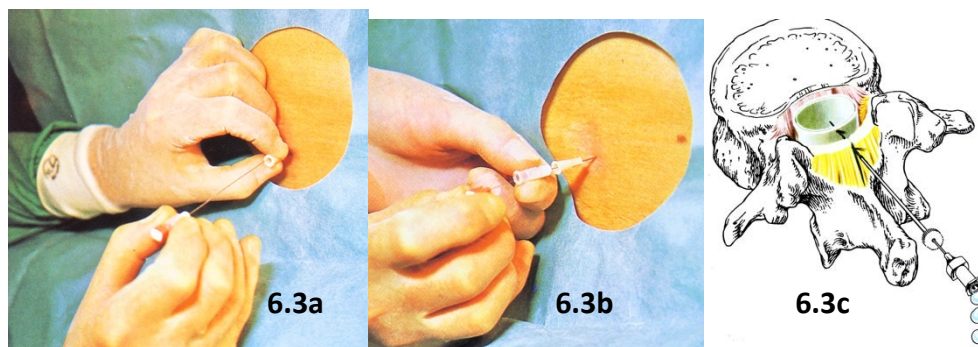


Figure 6.3a, 6.3b and 6.3c Technique of spinal anaesthesia

Epidural Anaesthesia

Epidural anaesthesia is produced by injecting a large volume (10-20ml) of local anaesthetic drug in the epidural space. The epidural space is a potential space that lies between the dura and the periosteum lining the inner aspect of the vertebral canal. On the posterior aspect, the ligamentum flavum completes the boundary between the lamina. It extends from the foramen magnum to the sacral hiatus. The epidural space contains fat, areolar tissue, lymphatics and internal vertebral venous plexus.

Epidural versus Spinal anaesthesia

Spinal anaesthesia is usually used as a single injection (although not commonly used, a catheter can be inserted into the subarachnoid space and continuous spinal anaesthesia is possible). Spinal anaesthesia produces dense, rapid onset of block with a small dose of local anaesthetic. As a single dose the duration of action is limited.

In epidural anaesthesia a catheter is usually inserted and a continuous infusion or intermittent top ups of local anaesthetic can be used to extend the duration. Compared to spinal anaesthesia it

requires a large volume (10-20 ml of local anaesthetic). The onset of block is slower, over 15 to 30 minutes and hence provides better cardiovascular stability compared to spinal anaesthesia (slow onset of cardiovascular effects). It may result in patchy block and missed segments are possible.

Indications

It can be used as the sole anaesthetic for orthopaedic procedures on the lower limb, gynaecological, caesarean section, vascular reconstructive surgery of the lower limbs and urological procedures. It is commonly used in combination with general anaesthesia for upper abdominal and thoracic surgery. When used in combination it helps to reduce the stress response and can be extended for the post operative period to provide analgesia.

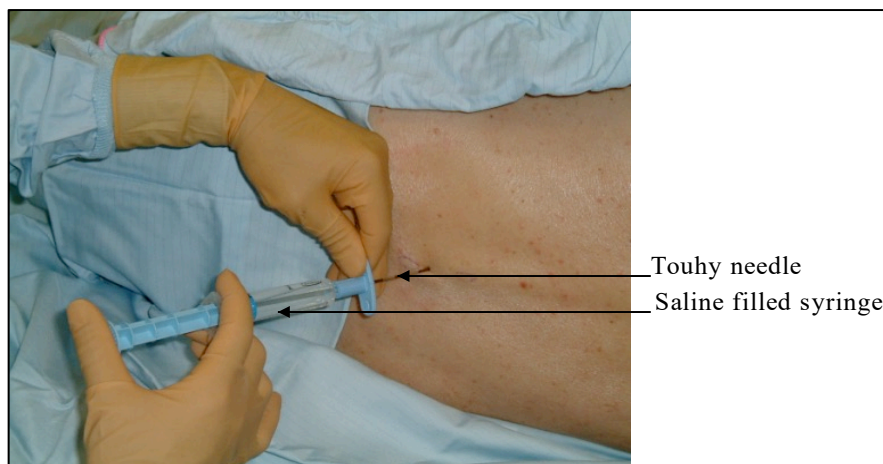


Figure 6.4 Technique of epidural anaesthesia, patient on right lateral position.

Technique

The patient position, monitoring and preparation is as mentioned for spinal anaesthesia. The chosen inter-space is infiltrated with local anaesthetic (1% lidocaine). A midline or para-median approach is chosen. A Touhy needle is inserted into the skin and then advanced to a depth of 2-3 cm until a distinct sensation of increased resistance is felt. Then the trocar is removed and a saline filled syringe is attached. The needle and syringe is slowly advanced, continuously checking for the loss of resistance, which will be felt as the needle exists through the ligamentum flavum and enters in to the epidural space (saline is injected). At this stage the syringe is removed and the catheter inserted for about 15-18 cm at the hub. The depth of the needle in the epidural space is noted and the needle is gradually withdrawn. About 3-5 cm of catheter should be left inside the space.

This [video](#) describes the anatomical basis of epidural anaesthesia. Watch the technique of epidural anaesthesia [here](#)

Complications:

Hypotension
Post-dural puncture headache
Missed segments
Epidural haematoma
Total spinal anaesthesia

Hypotension

Vasodilation is due to the sympathetic block and results in reduced systemic vascular resistance and a reduction in effective circulating volume. 40 to 60% of oxygen should be administered via a face mask, administration of vasopressor and intravenous fluids should correct the hypotension. Ephedrine, metaraminol, phenylephrine are commonly used vasopressors.

Ephedrine: It is a plant alkaloid, an indirectly acting sympathomimetic agent. It has both alpha and beta adrenoreceptor effects. It is available as 30mg/ml in an ampoule. It is usually diluted to 10ml with saline or water and given in increments of 1-2 ml (3-6mg) i.v. It causes vasoconstriction and increase in heart rate. Hence it increases blood pressure both by increasing cardiac output and systemic vascular resistance.

Metaraminol: It has both direct and indirect actions. It has predominant alpha effects and can produce a reflex bradycardia. It is supplied as 10 mg ampoules and usually diluted to 20 mls and given in increments of 1-2 ml (0.5-1mg) i.v. It is slower in onset than ephedrine (about 2 minutes) and causes less tachycardia than ephedrine.

Phenylephrine: It is a potent, pure vasoconstrictor which is available in 10 mg ampoules. It should be given in increments of 100 -200 mcg i.v.

Post-dural puncture headache

Dural puncture can be accidental during an epidural block or deliberate during spinal anaesthesia. Leakage of cerebrospinal fluid through the dural hole can lead to intracranial hypotension and stretching of the meninges and cranial nerve roots. This can result in headache which is described as a severe headache, usually frontal and bilateral, worsened by standing and relieved by lying. It is also associated with visual disturbances and photophobia. Management involves adequate hydration, simple analgesics and epidural blood patch.

Total spinal anaesthesia

It is very rare but can be catastrophic if not diagnosed early enough. It usually results from inadvertent injection of a planned epidural dose of local anaesthetic into the sub arachnoid space. It is characterised by severe hypotension, bradycardia, weakness of upper limbs, inability to talk and respiratory arrest.

Treatment: ABC approach, 100% oxygen, summon for help, rapid infusion of intravenous fluids and vasopressors. Inadequate breathing or respiratory arrest will need ventilatory support until the spinal block wears off completely.

Assessing the height of block

The block height should be tested and documented before starting the surgical procedure. Most lower abdominal surgeries require adequate anaesthesia between T6 –T8 dermatomes. Certain anatomical landmarks are used to approximate the level of dermatome.

Table 6.1 Dermatome levels

<u>Level</u>	<u>Dermatome</u>
T4-5	Nipple
T6-8	Xiphisternum
T10	Umbilicus
L1	Groin
S2	Perineum

The level of sensory block is usually tested for cold and touch sensation. An ice cube or ethyl chloride spray is used for testing cold sensation and cotton wool is used for testing the touch sensation. One should first test the sensation on the chest or arm (where sensation is normal). Then start working upwards from the feet and lower abdomen until the patient appreciates the sensation. If this is inconsistent or equivocal, the patient can be gently pinched with artery forceps or fingers on blocked and unblocked segments and asked if they feel pain. Patients should be instructed that they may still be aware of touch and pulling sensation but will not feel pain.

Central neuraxial block and anticoagulation

- Patients presenting for surgery can be on anticoagulant therapy or on other drugs that can affect clotting mechanisms. In the presence of abnormal coagulation, an increased risk of epidural or spinal heamatoma is associated with central neuraxial block.
- Central neuraxial block is contraindicated if the patient is on full oral anticoagulation or standard heparin.
- If the patient is on warfarin and epidural or spinal anaesthesia is highly indicated, then one should consider discontinuing warfarin 3-4 days prior to the surgery. The INR must be less than 1.5.
- Low dose standard heparin (5000 units s/c, bd): One should wait at least 4 hrs after a dose before performing Epidural or spinal injection. Heparin should not be administered until one hr following epidural or spinal injection.
- Low molecular weight heparin: Epidural or spinal anaesthesia can be performed 12 hrs after the last dose.
- If intra-operative anticoagulation is required, it should not be given until two hour after the spinal or epidural injection.
- Central neuraxial block is generally avoided if the platelet count is less than $100 \times 10^9/L$.
- Fibrinolytic or thrombolytic therapy is a contraindication for central neuraxial block.

The above precautions also apply for the removal of an epidural catheter. Clotting parameters should be near normal before removing epidural catheters.

Although NSAID's such as aspirin have an effect on platelet function, low dose aspirin does not increase the risk of epidural haematoma. Clopidogrel, an antiplatelet agent should be discontinued about a week before performing epidural or spinal anaesthesia.

Many new anticoagulation drugs have come into existence, many of which negate the need for regular blood tests (such as INR testing with warfarin). These drugs (such as Rivaroxiban) have no specific blood test that can detect levels and may require specific haematological intervention for reversal (if at all possible).

Peripheral nerve blocks

Minor surgical procedures and procedures on the limbs can be performed using peripheral nerve blocks alone. Most often peripheral nerve blocks are used in combination with general anaesthesia or spinal anaesthesia to extend the analgesia through postoperative period. Most of the peripheral nerve blocks provide analgesia for a duration of 4 -16 hours depending on the type and concentration of local anaesthetic and any other additive drugs used. Duration of the block can also be extended using a catheter technique where a continuous infusion of local anaesthetic can be used.

The following are the common peripheral nerve blocks used in clinical practice.

Upper limb blocks:

- Brachial plexus block
- Ulnar nerve block at elbow
- Median nerve block at elbow
- Wrist block
- Digital nerve blocks

Lower limb blocks:

- Sciatic nerve block
- Femoral nerve block
- Lateral cutaneous nerve of thigh block
- Ankle block

Trunk blocks:

- Intercostal nerve block
- Thoracic paravertebral block
- Ilioinguinal and iliohypogastric nerve block

Ophthalmic blocks:

- Peribulbar block
- Sub-Tenon's block

Intravenous regional anaesthesia (IVRA): This technique involves exsanguination of limb and then injection of local anaesthetic into the veins of the limb. Minor superficial surgery of the forearm and hand can be performed. Prilocaine 0.5% and lignocaine 0.5% are suitable local anaesthetics. Long acting local anaesthetic such as bupivacaine, ropivacaine and levobupivacaine are contraindicated, as their systemic absorption can result in cardiac toxicity.

Further Reading

<https://anaesthetists.org/Home/Resources-publications/Guidelines/Management-of-severe-local-anaesthetic-toxicity>.

Local Anaesthetics: In TE Peck and SA Hill. Pharmacology for anaesthetists and intensive care, 4th edition, 2014; Cambridge University Press

A good postoperative management plan is important to minimize complications and improve the outcome after surgery. It depends on

- Patient factors: age, comorbid conditions and current physiology.
- Surgical factors: site and nature of surgery, fluid and blood loss
- Anaesthetic factors: type of anesthesia and pain management

The common problems are briefly discussed below

Pain: Effective relief of pain is of paramount importance following surgery. This should be achieved for humanitarian reasons, but there is evidence that pain relief has significant physiological benefit.

The site of the surgery has a profound effect upon the degree of postoperative pain. Surgical procedure on the thorax and upper abdomen are more painful than operations on the lower abdomen which, in turn, are more painful than peripheral operations on the limbs. The result is an inability to cough and clear secretions, which may lead to lung atelectasis and pneumonia.

Pain causes an increase in the sympathetic response of the body with subsequent rises in heart rate, cardiac work and oxygen consumption. Prolonged pain can reduce physical activity and lead to venous stasis and an increased risk of deep vein thrombosis and consequent pulmonary embolism. In addition, there can be widespread effects on the gut and urinary tract motility which may lead, in turn, to postoperative ileus, nausea, vomiting and urinary retention. These problems are unpleasant for the patient and may prolong hospital stay.

The choice of pain-relieving techniques may be influenced by the site of surgery. Equally, it may be influenced by drug availability and familiarity with different methods of analgesia.

Management of pain

The World Health Organization Analgesic Ladder was introduced to improve pain control in patients with cancer pain. However, it also has lessons for the management of acute pain. As originally described, the ladder has three steps.

1. Simple analgesics (paracetamol \pm NSAIDs)
2. Weak opioids (like codeine) \pm Simple analgesics
3. Strong opioids (like morphine) \pm Simple analgesics

The important concept reinforced by WHO is to give the analgesics by clock, not prn (prn often means pain relief nil!). There should be regular analgesics on board along with 'as required analgesics' for breakthrough pain.

If the initial presentation of acute postoperative pain is severe and difficult to control, the alternate analgesic ladder recommended by The World Federation of Societies of Anaesthesiologists (WFSA) is useful. Here the initial control of severe pain is accomplished with stronger opioids \pm judicious use of local anaesthetic techniques. Then switch over to opioids by mouth and finally to simple analgesics when the situation permits.

Pharmacology of analgesics

The analgesics commonly used for post operative pain management can be grouped as follows:

1. Paracetamol
2. Non steroidal anti-inflammatory drugs (NSAIDs)
3. Opioids
4. Miscellaneous: Tramadol

Regional anaesthetic techniques like epidural and peripheral nerve blocks can be used as a sole technique to manage postoperative pain or as an adjunct to the above mentioned drugs.

Paracetamol

Paracetamol has analgesic and antipyretic properties but little anti-inflammatory effect. It is well absorbed orally and is metabolised almost entirely in the liver. It has few side effects in normal dosage and is widely used for the treatment of minor pain. It causes hepatotoxicity in overdose. It is a good analgesic when prescribed on regular basis.

Drug	Route	Dose	Frequency	Max. daily dose
Paracetamol	Per oral / I.V	1G	4-6 hourly	4G (in adults)

Non-steroidal anti-inflammatory drugs

NSAIDs have both analgesic and anti-inflammatory actions. Their mechanism of action is predominantly by inhibition of prostaglandin synthesis by the enzyme cyclo-oxygenase which catalyses the conversion of arachidonic acid to the various prostaglandins that are the chief mediators of inflammation. All NSAIDs work in the same way and thus prescribing more than one NSAID at the same time is unsafe. NSAIDs may be usefully combined with opioids due to their different modes of action.

Relative contraindications to the use of NSAIDs

- History of peptic ulceration, gastrointestinal bleeding or bleeding diathesis;
- Surgical procedures associated with high blood loss;
- Asthmatics with history of sensitivity to aspirin or other NSAIDs;
- Renal impairment

NSAIDs are available in a variety of formulations: tablet, injection, topical cream and suppository. The incidence of side effects and adverse reactions with an individual drug is similar regardless of the route of delivery.

Drug	Route	Dose	Frequency	Max. daily dose
Ibuprofen	Per oral	400 – 600 mg	4-6 hourly	2400 mg
Diclofenac	Per oral / Per rectal	50 mg	8 hourly	150 mg
Diclofenac	Per rectal	100 mg	16 hourly	150 mg

Ketorolac	Intravenous	10-30 mg	8 hourly	90 mg (max. 2 days)
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The new COX-2 inhibitors may be a better choice in patients where postoperative bleeding and effects on gastrointestinal tract is of concern. They are still unsafe in NSAID sensitive asthmatics and in presence of renal impairment.

Codeine

Codeine is a weak opioid analgesic which is derived from opium alkaloids (as is morphine). Codeine is markedly less active than morphine, has predictable effects when given orally and is effective against mild to moderate pain. It may be combined with paracetamol but care should be taken not to exceed the maximum recommended dose of paracetamol when using combination tablets.

Drug	Route	Dose	Frequency	Maximum daily dose
Codeine	Per oral	30-60 mg	4-6 hourly	300 mg

Morphine

Severe pain arising from deep or visceral structures requires the use of strong opioids. Appropriate treatment begins with an understanding of the correct drug, route of administration and the mode of action. Early administration will achieve effective analgesic concentrations and make it easier to maintain the therapeutic level of the drug in the blood. Once a satisfactory level of pain relief has been achieved this can be maintained by regular administration of opioid. Administration of adequate doses of analgesic may be inhibited because of side effects, notably nausea and vomiting.

Though there are other strong opioids like pethidine their analgesic effect is less satisfactory than morphine and is often reserved for patients with documented allergy to morphine.

Routes of administration of opioid drugs

The oral route of administration is the most widely used route and most acceptable for the patient. Disadvantages of the oral route to treat acute pain are that absorption of opioids may be reduced by the delay in gastric emptying that follows surgery. Nausea and vomiting may prevent absorption of drugs administered orally.

Intramuscular administration represents the optimum technique for strong opioids. This method of analgesia may be associated with peaks and troughs in effect.

Intravenous administration that involves incremental administration of small doses of strong opioids has the advantage of faster onset and predictable effect. But, by convention, this route is not preferred in general wards for the fear of respiratory depression. The person administering intravenous opioids should have sufficient knowledge and familiarity with the concerned drug and should be skilled enough to recognize and manage respiratory depression.

Drug	Route	Dose	Frequency
Morphine	Per oral	10 – 20 mg	2 hourly

Morphine	Intramuscular	10 – 15 mg	2 hourly
Pethidine	Intramuscular	50 – 100 mg	2 hourly

Patient Controlled Analgesia (PCA)

The PCA became popular when it was realised that individual requirements for opioids varied considerably. A system was devised whereby patients could administer their own intravenous analgesia and so titrate the dose to their own end-point of pain relief. In theory, the plasma level of the analgesic will be relatively constant and side effects caused by fluctuations in plasma level will be eliminated.

Drug	Bolus	Lock-out interval	Maximum hourly dose
Morphine	1-2 mg	5 – 10 min	10 - 20 mg
Fentanyl	10-20 mcg	5-10 min	100 -200 mcg

Monitoring of patients on PCA

Patients on PCA and those receiving strong opioids should be carefully monitored for side effects. Normal postoperative observation of blood pressure, heart rate, temperature and oxygen saturation should be continued. Respiratory rate should be monitored hourly whilst on PCA. In addition, volume of morphine/ fentanyl solution infused, pain scores and nausea vomiting score should be documented. Supplementary oxygen should be administered whilst on a PCA. Concurrent administration of other opioids or the same drug via other routes should be avoided as it will increase the risk of respiratory depression.

Side effects and toxicity of opioids

Opioid analgesics share many side effects, though the degree may vary between agents. The most common include nausea, vomiting, constipation and drowsiness. Larger doses produce respiratory depression and hypotension. The specific antidote naloxone is indicated if there is coma or very slow respiration. Because of its short action, repeated injections of 100 – 200 mcg intravenously or an infusion may be necessary.

Tramadol

Tramadol is a weak opioid (μ) receptor agonist. It also acts through other pathways involving serotonin and norepinephrine. Though the analgesic effect is only one-tenth of morphine in some instances tramadol is a good alternative to use of stronger opioids. Care needs to be taken if patients have a history of epilepsy. Nausea and vomiting are frequent side effects of this drug.

Drug	Route	Dose	Frequency	Maximum daily dose
Tramadol	Per oral	50 – 100 mg	4-6 hourly	400 mg
Tramadol	Intramuscular	50 – 100 mg	4-6 hourly	400 mg

Local Anaesthetics

Regional anaesthetic techniques used for surgery will provide near perfect postoperative pain relief if it can be prolonged beyond the duration of the surgery. There are many local anaesthetic techniques which can be continued into the postoperative period to provide effective pain relief. Most of these can be carried out with minimal risk to the patient and include local infiltration of incisions with long-acting local anaesthetics, blockade of peripheral nerves or plexuses and continuous block techniques peripherally or centrally (spinal or epidural).

Continuous infusion of a combination of local anaesthetics with opioids given epidurally produces very effective analgesia. They may also produce undesirable side effects such as hypotension, sensory and motor block, nausea, vomiting and urinary retention.

Care of patients with local anaesthetic infusion in post-operative ward

Patients with continuous local anaesthetic infusion, either peripherally or through epidural catheters usually come with clear prescription and instruction from the operating room by the anaesthetist involved. Still, awareness about some possible complications that can be encountered in the postoperative period can help in early intervention and safe clinical practice.

As far as peripheral catheter techniques are concerned, the common cause for which the doctors will be involved is for incomplete block or failed block with poor pain relief. The pain team can be called and in the meantime the management of acute pain can be as discussed earlier with analgesic medications. Other than this the personnel involved in the management of postoperative patients should be aware of local anaesthetic toxicity.

Patients with epidural infusion: Observations should include pain score, level of sedation, respiratory rate, nausea and vomiting and epidural pump observations. Apart from local anaesthetic toxicity, epidural infusion has its own complications that warrant immediate attention:

- Hypotension
- Respiratory insufficiency due to high block or epidural opioids.
- Post-operative Respiratory Insufficiency
- Postoperative respiratory insufficiency can be precipitated by a variety of factors.

Other common problems during immediate post-operative period

Anaesthesia related: Residual effects of anaesthetic medications, opioids, muscle relaxants; Spinal or epidural opioids can cause delayed respiratory depression. Epidural infusion of local anaesthetics can cause weakness of intercostal muscles.

Patient related: Hypotension, hypothermia, metabolic derangements like hypoglycaemia, acidosis can impair respiratory function; Existing cardio respiratory problems can get exacerbated due to perioperative stress and manifest as respiratory failure; Other pathologies can include pneumothorax, pulmonary embolism etc.

Surgery related: Surgeries involving the thorax and upper abdomen, laparoscopic procedures can predispose to mechanical factors that can reduce vital capacity, lead to atelectasis and manifest as hypoxaemia.

Irrespective of the underlying cause for the respiratory insufficiency the initial management involves assessment and management of airway, breathing and circulation. The epidural infusion can be discontinued while the cause of respiratory insufficiency is investigated.

If the clinical situation suggests opioid as the cause for respiratory depression, then intravenous administration of naloxone can be tried to reverse the effects. Hypotension should be managed with fluid boluses and judicious use of vasopressors like ephedrine or metaraminol. Basic monitoring such as ECG, non-invasive blood pressure, and pulse oximeter should be instituted simultaneously.

If the situation warrants, summon help from the resuscitation team which should include anaesthetists who are generally capable of evaluating the condition, identify the underlying cause and manage appropriately.

Post-operative nausea and vomiting (PONV)

Nausea and vomiting in the postoperative period may be the most unpleasant memory for patients in association with the hospital stay. Often the experience is described as worse than having pain.

Risk factors

Patients undergoing gynecological and urological procedures, bowel and gall-bladder surgeries, ENT and eye procedures and prolonged operations are at a higher risk.

Patient factors include females of child bearing age, obesity, previous history of PONV, migraine, motion sickness etc.

Some anaesthetic drugs including nitrous oxide, inhalational agents can increase the incidence. Both pain as well as strong analgesics like morphine can cause PONV.

Effects of PONV

Other than the unpleasantness, PONV is a common cause for unplanned admission following day surgeries and increased length of hospital stay.

Retching and forceful vomiting in severe cases can increase blood loss, predispose to wound dehiscence and incisional hernia, aspiration pneumonia. Persistent vomiting can lead to dehydration and metabolic derangements.

Management

There are simple, but important, measures to be taken to minimize the incidence of PONV.

These include:

- Avoiding hypoxaemia
- Avoiding dehydration and hypotension
- Avoiding too early feeding in the postoperative period
- Pain management with judicious mix of analgesics including local anaesthetic techniques when appropriate.

There is no single drug that is superior to another in management of PONV. In high risk groups, it has been consistently shown that a combination of antiemetics is more effective than single drugs. Interestingly there is evidence that the 'conventional' antiemetics like metaclopramide and droperidol have limited or no effect in the management of PONV.

The drugs available to treat PONV are often classified based on their mechanism of action. These include antidopaminergics, antihistamines, anticholinergics, antiserotonergic (5-HT₃ antagonists) and miscellaneous drugs like dexamethasone.

Commonly used drugs in the management of PONV.

Drug	Route	Dose	Frequency
Cyclizine	IM or IV	50 mg	8 hourly
Prochlorperazine	buccal	3-6 mg	12 hourly
Ondansetron	IM, IV or PO	4 – 8 mg	8 hourly
Dexamethasone	IV	8 mg	---

Unexpected pain or persistent severe pain or postoperative nausea and vomiting despite conventional management may be an early signal for a potential underlying problem that requires senior help and possible further investigation.

Further reading

- Schug, S. A. *et al.* (2016) 'Acute pain management: scientific evidence, fourth edition, 2015.', *The Medical journal of Australia*. Australia, 204(8), pp. 315–317. doi: 10.5694/mja16.00133.
- Best practice in the management of continuous epidural analgesia in the hospital setting (2010) The Royal College of Anaesthetists Publication
https://www.rcoa.ac.uk/system/files/FPM-EpAnalg2010_1.pdf

Enhanced recovery after surgery (ERAS) facilitates early mobilization after surgery by maintaining normal physiological function and reducing stress related to surgery. The ultimate aim is to reduce morbidity and length of stay in the hospital. The protocol based perioperative care focused at pre-operative preparation, intra-operative management, surgical technique and minimizing post-operative complications helps achieve these goals.

Pre-operative preparation

Preadmission counselling provides information relating to surgery and helps to reduce fear and anxiety related to surgery. Medical optimisation of comorbidities, abstinence of alcohol or tobacco has shown to reduce postoperative morbidity.

Complex carbohydrate drinks, containing 12.5% maltodextrins, drunk up to 2 hours pre-operatively have been found to reduce pre-operative thirst, hunger, anxiety and insulin resistance, without increasing aspiration risk. Carbohydrate drinks are thus a recommended part of enhanced recovery.

Antimicrobial agents are indicated where contamination into a sterile area may occur. The optimum time for administration is 30-60 minutes prior to skin incision and the choice of antibiotics should be made in conjunction with local guidelines.

Intra-operative management

Anaesthetic technique: The newer and short-acting anaesthetic agents with more rapid clearance and minimal hangover effects facilitate rapid recovery. For major surgeries, goal directed fluid therapy as guided by non-invasive cardiac output monitoring (such as oesophageal Dopplers) ensure optimum fluid balance and maintenance of cardiac output.

Identification of patients' risk for postoperative nausea and vomiting (PONV) and prophylaxis using multimodal approach is an important element of ERAS.

Surgical technique: The stress response to surgery is directly related to the extent of tissue trauma. Minimally invasive surgical approach such as laparoscopic and robot-assisted procedures have shown to reduce the length of hospital stay. Tranexamic acid has been routinely used for knee and hip replacement procedures to reduce the blood loss.

Nasogastric tubes: The use of nasogastric intubation in routine colorectal surgery has been demonstrated to increase the incidence of fever, atelectasis and pneumonia. Hence routine use should be avoided.

Maintenance of normothermia: Peri-operative hypothermia increases the incidence of wound infection, cardiac events, haemorrhage and worsens postoperative pain scores. Shivering

dramatically increases total body oxygen consumption at a time when oxygen consumption is already raised by tissue trauma. It is therefore important to ensure patients arrive into theatre normothermic and are kept around normal temperature (>36°C). Techniques commonly utilised include forced air warming devices, underbody warming mattresses and warmed fluids, while also limiting heat loss through evaporation by increasing theatre humidity and applying hydrophobic filters to breathing circuits to retain respiratory humidity.

Post-operative management

Analgesia: A good post-operative pain relief regimen using multimodal approach such as local infiltration of local anaesthetics, low dose epidural analgesia where appropriate, use adjunctive analgesics (Gabapentine, IV lidocaine, Magnesium sulphate) facilitates early mobilisation and improves postoperative morbidity.

Venous thromboembolism (VTE) prophylaxis: Venous thromboembolism (deep vein thrombosis and pulmonary embolism) is recognised complication following major surgery and carries a significant morbidity and mortality. A combination of methods involving intermittent pneumatic calf compression intraoperatively, compression stockings and low-molecular-weight heparin (LMWH) administration has shown to reduce the risk of postoperative DVT.

Postoperative nutrition: Pre-operatively, it is important to maintain normal nutrition and avoid excessive fasting. Postoperatively, the patient should be allowed to drink immediately after surgery and eat as soon as possible. Though this is associated with higher rates of vomiting due to ileus, it does reduce length of hospital stay and infection, and is not associated with anastomotic dehiscence.

Glucose control: Insulin resistance is a known consequence of the stress response to surgery, and thus the measures taken in the enhanced recovery programme to reduce the surgical stress response should also reduce insulin resistance. In situations where hyperglycaemia is persisting (>10 mmol/L) insulin infusion may be necessary to control hyperglycaemia.

Early mobilisation: Early mobilisation is associated with a reduced incidence of chest complications and in combination with good nutrition, can improve muscle strength in the early postoperative phase.

Further reading

Place K, Scott NB. Enhanced recovery for lower limb arthroplasty. *Br J Anaesth* CEACCP 2014; 14: 95-9.

Gustafsson UO, Scott MJ, Schwenk W, et al. Guidelines for perioperative care in elective colonic surgery: enhanced recovery after surgery (ERAS®); Society recommendations. *World J Surg* 2013; 37: 259-84.

Khan SK, Malviya A, Muller SD, et al. Reduced short-term complications and mortality following enhanced recovery primary hip and knee arthroplasty: results from 6000 consecutive procedures. *Acta Orthopaedica* 2014; 85: 26-31.

ACID-BASE DISORDERS

A normal acid-base balance which ensures optimal function of enzymes at a cellular level.

This chapter looks to explore the following:

- Basic principles of acid-base balance
- What is the anion gap
- What is an arterial blood gas (ABG) and what can it tell us?
- Diabetic Ketoacidosis

Basic Principles of acid-base balance

The basic principles of acid-base balance are dependent on understanding of substances known as acid or alkali. An acidic substance is one which releases H^+ and an alkali is one which binds with H^+ ions. pH is the negative logarithm to the base 10 of hydrogen ion concentration. Acid is quantified by the number of H^+ ions present in solution. The relationship between H^+ and pH is as follows:

pH 7.0 = 100 nmol/L i.e., 10^{-7} mol/L

pH 8.0 = 10 nmol/L

pH 6.0 = 1000 nmol/L

There are several equations that are important to understanding acid-base balance in the body. These equations highlight the importance of Carbon Dioxide (CO_2) as a prime contributor to acid in the body.

- $CO_2 + H_2O = H_2CO_3 = H^+ + HCO_3^-$
- $H_2O = OH^- + H^+$
- $OH^- + CO_2 = HCO_3^-$

The Henderson-Hasselbalch equation relates pH to the bicarbonate buffer system and H^+ ions. This equation is better described in this [video](#).

$pH \propto HCO_3^- / H_2CO_3$ $pH = 6.1 + \log [HCO_3^- / H_2CO_3]$

Normal acid-base balance

H^+ concentration is normally maintained within strict limits by:

1. Buffering of H^+ ions
2. Elimination of H^+ ions

The two major organs that enable the human body to maintain acid-base balance are the lungs and the kidneys. Both systems have unique characteristics that work synergistically to maintain tight control on acid levels in the human body. The lungs provide rapid (in seconds) potential to excrete CO_2 or acid from the body, hence there is a significant increase in your minute ventilation when you exercise. The kidneys adapt more slowly to changes in acid-base status (over several days) and can increase the elimination of H^+ ions as well as retaining bicarbonate to buffer increased acid (such as in chronic obstructive airways disease).

Please watch this [video](#) to better understand the basics of acid-base balance.

Buffers

A buffer is a substance that resists change in acid-base status. Multiple buffering systems exist in the human body, usually consisting of a weak acid and its salt with its conjugate base.

Buffers can broadly be classified as extracellular and intracellular:

Extracellular Buffers:

- 1) Bicarbonate (65% of total buffering capacity)
- 2) Haemoglobin (can also be considered as intracellular as it is within red cells) (29% of total buffering capacity)
- 3) Phosphate in urine
- 4) Calcium bicarbonate in bone

Intracellular Buffers:

- 1) Proteins (5% of total buffering capacity)
- 2) Phosphates

Anion gap

The anion gap is the difference between the measured cations (positive) and anions (negative).

$(\text{Na}^+ + \text{K}^+) - (\text{Cl}^- + \text{HCO}_3^-)$; Normal value is 12-18 mmol/L

There is normally a difference between cations and anions in the body due to unmeasured anions (proteins in the anionic form, phosphates, sulphates and organic acids).

Acidosis with normal anion gap

In patients with a normal anion gap the drop in HCO_3^- is compensated for (almost completely) by an increase in Cl^- . Causes are hyperchloremic acidosis e.g. gastrointestinal loss of HCO_3^- in diarrhoea, renal dysfunction and renal tubular acidosis.

A high anion gap indicates that there is loss of HCO_3^- without a concurrent increase in Cl^- (measured anion). HCO_3^- is replaced by the unmeasured anion resulting in a high anion gap (refer to table below) .

Arterial blood gas

An arterial blood gas is a sample of arterial blood that is analysed for its composition. The details that an arterial blood gas provides can be correlated with the patient's history to aid in diagnosis.

In order to correctly analyse an arterial blood gas sample, the temperature and the inspired oxygen of the patient need to be noted. The basic components of an arterial blood gas are:

- pH – This gives information about whether the patient is acidotic or alkalotic. Normal values are 7.35 – 7.45
- PCO_2 – The partial pressure of carbon dioxide present in the sample. A raised carbon dioxide indicates a respiratory problem. Normal values 4.5 – 6.0 kPa
- PO_2 – The partial pressure of oxygen present in the sample. A low oxygen content indicates hypoxia. Normal values >10.6 kPa.
- HCO_3^- – This is the bicarbonate content of the sample. A low bicarbonate indicates metabolic compensation of an acidosis. A raised bicarbonate indicates chronic renal compensation (for example in COPD). Normal values 22 – 28 mmol/L

There are other components of the arterial blood gas that can be very useful, these include:

- Hb – Haemoglobin
- HCT – Haematocrit
- Glucose
- Lactate. Normal value 0.5 – 2.2 mmol/L
- Base Excess – This indicates the amount of acid or base you would have to add to the sample to return the pH back to normal.

Please watch the following [video](#) to help understand how to interpret arterial blood gases.

An easy way of interpreting arterial blood gases is using a flow chart such as this:

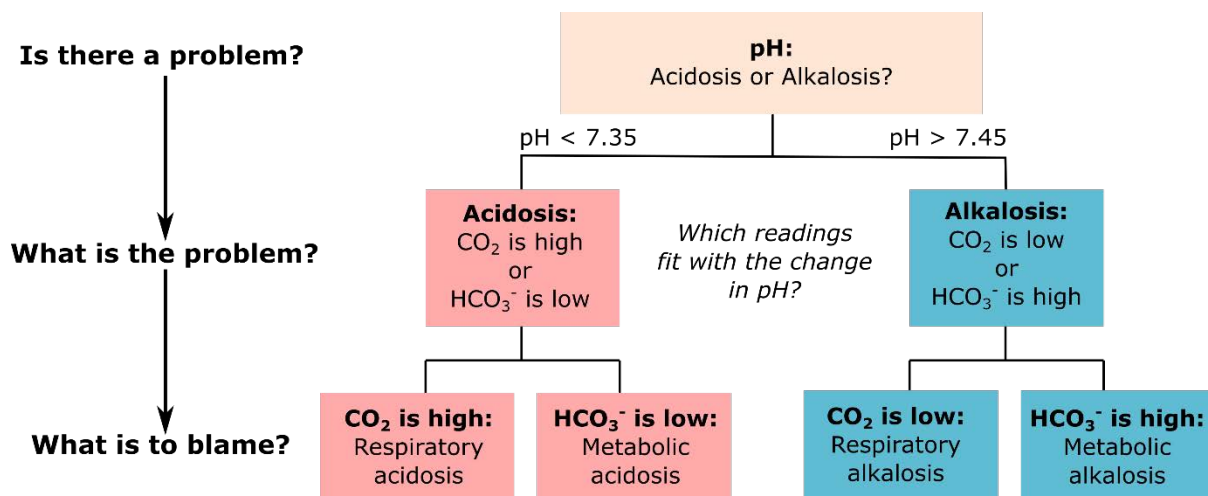


Figure 8.1: Easy interpretation of an arterial blood gas

Table 8.1 Common Causes of Metabolic Disturbance

	Acidosis (pH<7.35)	Alkalosis (pH>7.45)
Respiratory (Caused by hypo or hyperventilation)	Caused by hypoventilation : Acute: <ul style="list-style-type: none"> • Depression of central respiratory centre (cerebral disease or drugs) • Neuromuscular disease (myasthenia gravis, Guillain-Barré syndrome) Chronic: <ul style="list-style-type: none"> • COPD • Pickwickian syndrome 	Caused by hyperventilation : <ul style="list-style-type: none"> • Panic attacks • Heart attack • Pain • Drug use • Asthma • Pulmonary Embolism • Pregnancy • High altitude • Salicylate poisoning
Metabolic (caused by retention or loss of H ⁺ ions)	Increased Anion Gap: M -Methanol	Normal Anion Gap: Chloride responsive: Loss of H ⁺ ions: <ul style="list-style-type: none"> • Vomiting

	U -Uraemia (chronic kidney failure) D -Diabetic ketoacidosis P -Paraldehyde (rare) I -Infection, Iron, Isoniazid, Inborn errors of metabolism L -Lactic acidosis E -Ethylene glycol S -Salicylates	H - Hyperalimantation A - Addison disease R - Renal tubular acidosis D - Diarrhoea A - Acetazolamide S - Spironolactone S - Saline infusion	<ul style="list-style-type: none"> • Via the kidney Other causes: <ul style="list-style-type: none"> • Cystic Fibrosis • Alkalotic agents Chloride resistant: <ul style="list-style-type: none"> • Retention of bicarbonate • Hyperaldosteronism • Low magnesium levels in the blood
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For further details, please refer to following video links

Respiratory Acidosis Video	Respiratory Alkalosis Video
Metabolic Acidosis Video	Metabolic Alkalosis Video

Diabetic Ketoacidosis

Pathophysiology

Due to a lack of insulin, glucose cannot enter cells, and carbohydrate-based metabolism is changed over to fat oxidation. This is an extension of normal physiological mechanisms that compensate for starvation. Free fatty acids are produced in fat cells and transported to the liver. In the liver, they are broken down into acetate, then ketoacids (acetoacetate and beta-hydroxybutyrate). The ketoacids are then exported from the liver to peripheral tissues (notably brain and muscle) where they can be oxidised.

Both absences of insulin and excess glucagon result in inhibition of glycolysis. Such inhibition not only raises glucose levels but stimulates ketone formation. Insulin deficiency and other stress hormones also promote increased glucose production via glycogen breakdown. A combination of reduced cellular uptake of glucose and gluconeogenesis increases the plasma glucose. Hyperglycaemia promotes osmotic diuresis with loss of water, sodium, potassium and phosphate. Further associated nausea and vomiting exacerbate dehydration.

The main causes of death in diabetic ketoacidosis include severe hyperkalaemia, hypokalaemia, aspiration of gastric contents and cerebral oedema. Poor prognostic features include impaired conscious level, acidosis with pH <7.0, oliguria and hypokalaemia.

Management

A patient who has diabetic ketoacidosis needs assessment and resuscitation in a critical care environment. Airway, breathing and circulation should be assessed, and supplemental oxygen should be administered. The aim is to control the blood glucose, correct dehydration, electrolyte and acid-base imbalance.

Monitoring should include continuous ECG, pulse oximetry, invasive arterial blood pressure, central venous pressure, urine output, and temperature. Further investigations such as urine analysis for ketone bodies, arterial blood gas for pH, serum electrolytes (K^+), full blood count, urine, and blood cultures should also be performed.

Control of blood glucose: A short acting insulin (Actrapid) infusion should be started as a sliding scale regimen. There are several locally agreed protocols for insulin infusion. The aim is to maintain the blood glucose between 4 – 8 mmol/L. Once the blood glucose is less than 12 mmol/L, a 5% dextrose infusion should be started at a rate of 125 ml/hour to prevent hypoglycaemia.

Serum potassium should be monitored every two hours; it should be maintained between 4 – 5 mmol/L.