An introduction to anaesthesia

Introduction

Anaesthetic experience in the undergraduate timetable is often very limited so it can remain somewhat of a mysterious practice well into specialist training. This introduction to the components of an anaesthetic will help readers to get more from clinical attachments in surgery and anaesthetics or serve as an introduction to the topic for novice or non-anaesthetists.

Types and sites of anaesthesia

The term anaesthesia comes from the Greek meaning loss of sensation. Anaesthetic practice has evolved from a need for pain relief and altered consciousness to allow surgery. Early anaesthetics used plant derivatives with later introduction of ether, inhaled gases and chloroform. Modern anaesthesia has been developed and refined to enable surgery, interventions, pain relief and stabilization, and organ support.

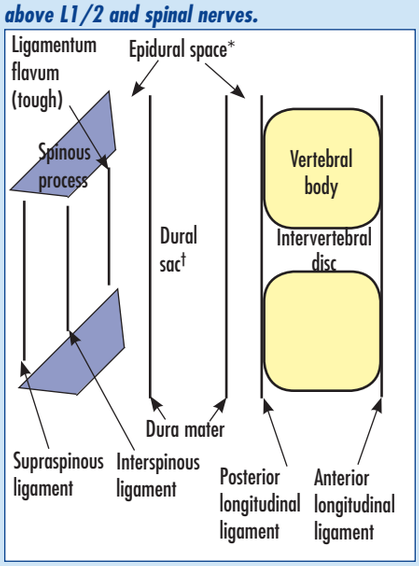
Various forms of anaesthesia are conducted throughout the hospital and beyond. The operating theatres are the most common venue but anaesthetics are delivered on the labour ward, day surgery, intensive care, the emergency room, interventional radiology, computed tomography and magnetic resonance imaging, and on the wards during emergency care and transfer of acutely unwell patients. Certain regional procedures may take place in pain clinics and outpatient settings.

In general anaesthesia a reversible state of unconsciousness is achieved. It can be divided into three stages: induction, maintenance and emergence.

In regional anaesthesia, nerve transmission is blocked, and the patient may stay awake or be sedated or anaesthetized during a procedure. Techniques used include:

- Local anaesthetic field block
- Peripheral nerve block
- Nerve plexus block
- Central neuraxial block, e.g. spinal or epidural (*Figure 1* and *Table 1*).

Figure 1. Schematic vertical longitudinal section of vertebral column and structures encountered when performing central neuraxial blocks. * negative pressure space filled with fat and venous plexi. † extends to S2, containing arachnoid mater, CSF, pia mater, spinal cord above L1/2 and spinal nerves.



Components of a general anaesthetic

A general anaesthetic always involves an hypnotic agent, usually an analgesic and may also include muscle relaxation. The combination is referred to as the 'triad of anaesthesia'.

The relative importance of each component depends on surgical and patient factors: the intervention planned, site, surgical access requirement and the degree of pain or stimulation anticipated. The technique is tailored to the individual situation.

Induction

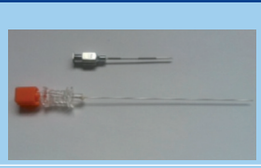
The induction of anaesthesia refers to the transition from an awake to an anaesthetized state. This end point can be ill defined and the process of induction is a time of physiological disruption with multi-system effects.

Standard induction

Intravenous

The standard induction is with the intravenous agent propofol. A calculated by weight dose is delivered and the effects reviewed before further titration of the drug. Delays in inducing anaesthesia may represent slow arm–brain circulation time (e.g. elderly, cardiovascular disease),

Table 1. Characteristics of different central neuraxial blocks

Subarachnoid (spinal)		Epidural	
Injection through dura into CSF		Catheterization of potential space outside dura	
Low volume (up to 3 ml)		High volume (>10 ml)	
High concentration local anaesthetic 0.5% bupivicaine		Variable concentration local anaesthetic, analgesia 0.1% bupivicaine, anaesthesia up to 2% lignocaine	
Rapid onset dense sensorimotor block		Gradual titration of block density, may be motor sparing	
Profound vasodilation causing haemodynamic instability		Gradual titration causing less haemodynamic disturbance	

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patient anxiety, recreational drug use or extravasation. An opioid is often given to reduce the dose of induction agent needed and smooth the induction process. A muscle relaxant is usually given if intubation is required.

Inhalational induction

An alternative method of inducing anaesthesia is with a volatile agent, e.g. sevoflurane. The concentration of volatile delivered is gradually increased with the patient spontaneously breathing. Common uses include paediatric practice, cases of difficult airway, difficult venous access or inhaled foreign body where maintaining spontaneous ventilation is preferable.

Intubation of the trachea can be achieved under deep inhalational induction without muscle relaxation.

Rapid sequence induction: when and why?

A specifically adapted induction process is used when rapid intubation of the trachea is required to minimize risk of regurgitation and aspiration (*Table 2*). Such instances include intestinal obstruction or intra-abdominal pathology, an un-fasted patient in an emergency or trauma situation, obstetric emergency or a strong history of reflux. Pre-oxygenation plus rapid induction and paralysis obviate the need for bag mask ventilation before securing the airway, so the risk of gastric insufflation and regurgitation is reduced (Sinclair and Luxton, 2005).

Table 2. Rapid sequence induction

Preparation	Trained staff	
	Emergency drugs and equipment	
	Tipping trolley	
	Suction on under pillow	
	Aspiration of nasogastric tube	
Pre-oxygenation	Fraction of inspired oxygen 100%	
	3 minutes regular breathing or five vital capacity breaths	
Cricoid pressure	Pressure over cricoid cartilage	
	Compression of underlying oesophagus	
	Prevents regurgitation of gastric contents soiling oropharynx or airway	
	Release pressure if vomiting	
Drugs	No co-induction opioid	
	Thiopentone 3–5 mg/kg	
	Suxamethonium 1–2 mg/kg	
Intubation	Once tracheal intubation confirmed:	Ventilation commenced
		Cricoid pressure released

Muscle relaxation

If intubation is required, it may be necessary to paralyse the patient using:

- Depolarizing muscle relaxants (e.g. suxamethonium)
- Non-depolarizing muscle relaxants (benzylisoquinoloniums, e.g. atracurium, or aminosteroids, e.g. rocuronium).

Normally, an action potential reaching the nerve terminal of the neuromuscular junction causes calcium influx and acetylcholine to be released pre-synaptically. Acetylcholine crosses the cleft and binds to postsynaptic nicotinic acetylcholine receptors causing opening of these ion channels and depolarization of the motor end plate. If a sufficient end plate potential is achieved, an action potential is generated leading to muscle contraction (King and Hunter, 2002).

A depolarizing agent such as suxamethonium (biochemically two acetylcholine molecules) binds to the postsynaptic acetylcholine receptors, resulting in transient receptor agonism and muscle contraction followed by a refractory period of muscle relaxation within 30–60 seconds lasting several minutes. Its relatively short-lived effects are the result of its metabolism by plasma cholinesterase.

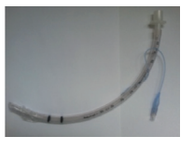
Non-depolarizing agents are competitive antagonists of acetylcholine at the postsynaptic nicotinic receptor and are used for more prolonged paralysis. Blocking the ion channel, their main action is to prevent end plate depolarization and propagation of the impulse. Atracurium undergoes spontaneous degradation in the plasma known as 'Hoffman' degradation, while some is hydrolysed by esters so it is a useful agent in patients with hepatic and renal impairment as offset is not reliant on organ function. As the percentage acetylcholine receptor occupancy falls, competitive antagonism is lost and acetylcholine can once again bind to receptors to generate an end plate potential and reach the threshold for transmission. Neuromuscular function is restored (Appiah-Ankam and Hunter, 2004).

Neuromuscular junction function should be monitored using a peripheral nerve stimulator and observing response to stimulations over a peripheral nerve (e.g. ulnar). The stimulation is supramaximal in order to stimulate all the nerve fibres and produce a consistent muscular response. The number and strength of resultant muscle twitches gives information about the recovery of the neuromuscular junction (Davis and Kenny, 2003). In order to enhance neuromuscular recovery post non-depolarizing relaxation at the end of surgery, the amount of acetylcholine in the synapse is increased by inhibiting the acetylcholinesterase enzyme using a reversal agent such as neostigmine.

Airway maintenance

Under anaesthesia the soft tissues of the airway relax and patency may be lost. Protective airway reflexes are also suppressed. Manual manoeuvres and simple adjuncts such as a chin tilt, jaw thrust and Guedel airway are used as soon as the patient begins to lose airway tone to prevent obstruction. Conventionally the options for maintaining the airway of an anaesthetized patient are a supraglottic device (e.g. laryngeal mask airway) or endotracheal intubation (*Figure 2*). At the

Figure 2. Supraglottic and endotracheal airways.

Device	Supraglottic	Endotracheal
		
Features	Sits above vocal cords	Passes through vocal cords
	Maintains airway	Inflated cuff
	No airway protection against aspiration	Airway protected

preoperative visit, history, examination and review of investigations and previous anaesthetic charts contribute to the assessment of the airway and perioperative planning (Cranshaw and Cook, 2011).

Maintenance

Maintenance of anaesthesia refers to keeping a patient unconscious and can be achieved using inhaled volatile agents or continuous infusion of intravenous agents.

Volatile agents are most commonly used, delivered via vaporisers found on the 'back bar' of the anaesthetic machine which feed into the breathing circuit. The concentrations of the inhaled agents are measured and displayed. Expired end tidal concentration is equivalent to the alveolar concentration which in turn represents the concentration at the site of action (CNS). This gives the anaesthetist an idea of the amount of anaesthetic agent reaching the patient and the likely depth of anaesthesia. The minimal alveolar concentration is the alveolar concentration of a volatile agent which when given alone prevents movement in 50% of healthy volunteers to a standard surgical stimulus (e.g. skin incision). The minimal alveolar concentration varies between different volatile agents inversely related to their potency (as their structures vary) and is also affected by other pharmacological and physiological variables (Yentis et al, 2009).

Intravenous maintenance of anaesthesia can be achieved with infusions of propofol with or without an opioid delivered via a pump. Several pharmacokinetic models have been developed which map the theoretical body compartments among which a drug distributes. The desired plasma or effector site concentration can be dialled up alongside basic patient demographics (age, sex, weight) and the pump adjusts the rate of infusion to achieve the specified drug concentration. This is known as a target controlled infusion. Effective secure intravenous access is crucial.

The choice of maintenance technique may be determined by surgical and patient factors and the experience of the anaesthetist. Total intravenous anaesthesia is often used in day surgery, neurosurgery or if patients get severe postoperative nausea and vomiting as it avoids emetogenic volatiles and enables rapid recovery with minimal hangover effect (Yuill and Simpson, 2002).

Systemic effects of general anaesthesia

General anaesthesia leads to multi-system physiological changes (*Tables 3* and *4*). The systemic effects of anaesthesia vary with the drugs used so different agents are favoured in different clinical contexts. In general, intravenous (propofol and thiopentone) and volatile agents all reduce blood pressure as a result of vasodilation, and negative inotropy and chronotropy. Starting positive pressure ventilation (i.e. ventilating someone) can impede venous return to the heart, reducing preload and cardiac output. The sympathetic stimulation from surgery opposes these changes.

Intravenous (propofol, thiopentone and etomidate) and volatile agents are all respiratory depressants and depress airway reflexes to differing degrees. Propofol is particularly effective at inducing transient apnoea and depressing airway reflexes facilitating placement of supraglottic devices post induction. Of the volatile agents, sevoflurane is the least irritant to airways making it particularly suitable for

Table 3. Systemic effects of general anaesthesia

System	Common anaesthetic agents	Ketamine
Cardiovascular	Hypotension: mean arterial pressure = (heart rate x stroke volume) x systemic vascular resistance	Normotension or hypertension
	Vasodilation (↓systemic vascular resistance)	Tachycardia
	Negative chronotropy (↓heart rate)	
	Negative inotropy (↓stroke volume)	
Respiratory	Loss of airway reflexes and tone	Airway reflexes and tone maintained
		Bronchodilation
Gastrointestinall	Propofol = antiemetic	Salivation
	Volatiles = emetogenic	Emetogenic
CNS	Hypnosis	Dissociative anaesthesia, analgesia, hallucinations

From Sasada and Smith (2008)

Table 4. Stages of a general anaesthetic: an A, B, C, D approach

Stage of general anaesthesia	Airway	Breathing	Circulation	Drugs
Induction	Plan for securing, maintaining and protecting airway as soft tissue tone and reflexes are lost	High flow oxygen at induction, consider pre-oxygenation	Vasodilation leads to reduced systemic vascular resistance and mean arterial pressure, intubation can cause sympathetic hypertensive response	Intravenous: analgesia (opioid co-induction, e.g. fentanyl) then hypnotic agent (e.g. propofol, thiopentone) with or without muscle relaxation or volatile gas induction (sevoflurane)
Maintenance	Maintain airway position and patency	Maintain saturations, ventilatory strategies, lung protection	Maintain adequate cardiac output and tissue perfusion, fluid balance	Volatile (e.g. sevoflurane, isoflurane, desflurane) Intravenous (total intravenous anaesthesia, e.g. propofol +/- remifentanil), analgesia, antiemesis
Emergence	Suction secretions, as airway tone and reflexes return plan for safe removal of supraglottic device or extubation	Increase fraction of inspired oxygen, ensure adequate spontaneous tidal volumes	Time of haemodynamic instability	Reversal of neuromuscular block

gaseous induction and paediatrics. Desflurane is irritant and is therefore often used once anaesthesia and airway have been established. Both sevoflurane and isoflurane are bronchodilators and may even have a role in the management of brittle asthma.

Ketamine is an exceptional intravenous agent in that it maintains cardiovascular stability and preserves muscular tone, airway patency and bronchodilates in addition to its analgesic properties. It is termed a 'dissociative anaesthetic', meaning the patient may be unaware and detached from his/her surroundings but not completely unconscious. Its attributes make it useful in haemodynamically unstable patients, the developing world and field anaesthesia (Peck et al, 2008).

Emergence and recovery

Once anaesthesia is no longer required, maintenance agents can be switched off. Before emergence, adequate analgesia and anti-emesis should be ensured and neuromuscular junction function restored if a muscle relaxant has been used.

Like induction, emergence can be a time of physiological disturbance. As patients start to wake from anaesthesia or 'lighten' they may develop agitation, laryngospasm and breath-holding. Conventionally extubation is performed following oropharyngeal suction, once the patient is generating good tidal volumes and is awake, ensuring airway reflexes have returned and the patient will protect his/her own airway. In certain circumstances extubation may be performed 'deep', i.e. with the patient still under anaesthesia. Under anaesthesia, airway reflexes will remain suppressed, reducing the risk of coughing, laryngospasm and hypertension associated with extubation. This may be preferable in certain neurosurgical and cardiac patients in whom surges in intracranial or systemic blood pressure should be avoided. However, the airway will be unprotected against aspiration until the patient is awake.

The recovery room is an intermediate place of safety between theatre and the ward where immediate surgical or anaesthetic complications can be detected and managed. Vital signs, pain scores and other potential problems such as postoperative nausea and vomiting are monitored.

Perioperative care

Warming

As homeothermic mammals, our core temperature is designed to be around 36.5°C. Patients are susceptible to hypothermia under anaesthesia as a result of vasodilation causing redistribution of heat from core to periphery, convection, radiation (exposed areas), conduction (contact with cold metal objects), evaporation (endotracheal tube bypasses nasopharyngeal humidification, exposed moist mucosal surfaces) and loss of compensatory heat-preserving or heat-generating mechanisms, e.g. shivering.

Hypothermia can cause coagulopathy, perioperative cardiac events, increased risk of postoperative infection and can prolong recovery and hospital stay. Exposure should be minimized and temperature monitored pre-, intra- and postoperatively. Warm air devices and warmed fluids can be used to offset heat loss and maintain optimal body temperature (Harper et al, 2008).

Fluid balance

The anaesthetist needs to be an expert in fluid resuscitation, using crystalloids, colloids and blood products where appropriate. The aim is to ensure good tissue perfusion and hence oxygenation. Rather than give a fixed fluid dose, monitors (e.g. oesophageal doppler) are often used and fluid challenges given to achieve a set endpoint, aiming to avoid hyper- or hypovolaemia (Doherty and Buggy, 2012).

Positioning

Patients are vulnerable to nerve and pressure point injury under anaesthesia and protection of these areas is the responsibility of the anaesthetist. Patients should ideally be in a neutral position with padding used to support at-risk areas (Knight and Mahajan, 2004).

Awareness

Awareness is the unplanned recall of events under anaesthesia and is often one of the complications patients fear most. It can be implicit or explicit, from a vague sense of having been awake through to specific memories of events and conversations respectively. Awareness is distressing and can lead to post-traumatic syndromes. Monitoring for awareness can include clinical observation (such as papillary dilatation, lacrimation, sweating) and measurement (heart rate, blood pressure, end tidal volatile concentration and depth of anaesthesia monitors). Most depth of anaesthesia monitors interpret patterns of neuronal electrical activity to deduce the level of conscious state (Al-Shaikh and Stacey, 2007).

Other drugs

Aside from the traditional triad of anaesthesia drugs, the cardiovascular system is often manipulated to offset the effects of anaesthesia or surgical stimulation. Heart rate may be increased by an anti-muscarinic (e.g. atropine or glycopyrrolate) or a mixed beta-adrenoceptor agonist (e.g. ephedrine) or reduced by beta-blockers. Blood pressure can be increased by vasoconstricting with an alpha 1 adrenoceptor agonist such as metaraminol or reduced with an alpha antagonist (e.g. phentolamine).

Analgesia

Pain relief is very important for patients and features in the triad of anaesthesia. Despite a patient being unconscious and unaware intraoperatively, stimulation (e.g. surgery) will still elicit a sympathetic response which analgesia can desirably attenuate. Appropriate analgesia is also essential for smooth emergence and comfort immediately after surgery. Analgesia is typically multi-modal with opioids titrated to extent of stimulation and predicted postoperative pain.

Safety

Patient safety is crucially important. The World Health Organization surgical safety checklist is a tool to attempt to make the perioperative journey safer and enhance team communication. The entire team must ensure the correct patient is consented for the correct procedure and that any allergies or potential complications are acknowledged and shared among the team.

The World Health Organization surgical safety checklist has three components which are completed on arrival to the anaesthetic room, before the start of surgery or intervention and at the end of the procedure (Walker et al, 2012).

Conclusions

Anaesthesia is an enormous subject and this article is merely a tip of the iceberg introduction to some types of regional and general anaesthesia.

Anaesthetists, while developing specialized airway skills and a deep understanding of physiology and pharmacology, need an holistic approach and broad knowledge base because of the varied nature of their role. Anaesthetists will come into contact with approximately two thirds of hospital patients in a diverse range of clinical contexts and environments. Hopefully this article has whetted your appetite to know more or given you a fresh insight into a specialty which is taking place in all corners of your hospital. **BJHM**

Conflict of interest: none.

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KEY POINTS

- Anaesthesia means loss of sensation and can be divided into regional anaesthesia (blockade of nerve transmission) or general anaesthesia (a reversible state of unconsciousness).
- General anaesthesia often comprises a triad of hypnosis, analgesia and muscle relaxation.
- General anaesthesia can be divided into three stages: induction, maintenance and emergence.
- Under general anaesthesia airway tone and reflexes are lost and the airway must be maintained with manual manoeuvres, adjuncts (Guedel, laryngeal mask airways) or definitive devices which also protect the airway from regurgitation and aspiration (e.g. endotracheal tubes).
- General anaesthesia leads to multi-system physiological changes particularly at induction and emergence.
- Other aspects of perioperative care central to anaesthetic practice include thermal homeostasis, fluid balance, positioning, avoidance of awareness, analgesia and patient safety.