# **Supporting the Transition into Anaesthesia:** Development of a Study Guide for New Trainees

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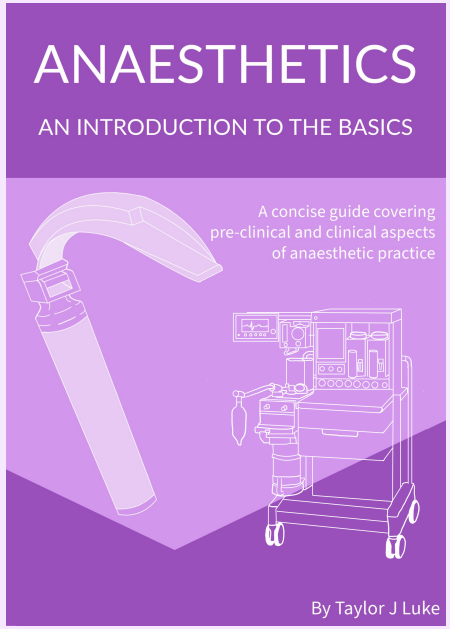
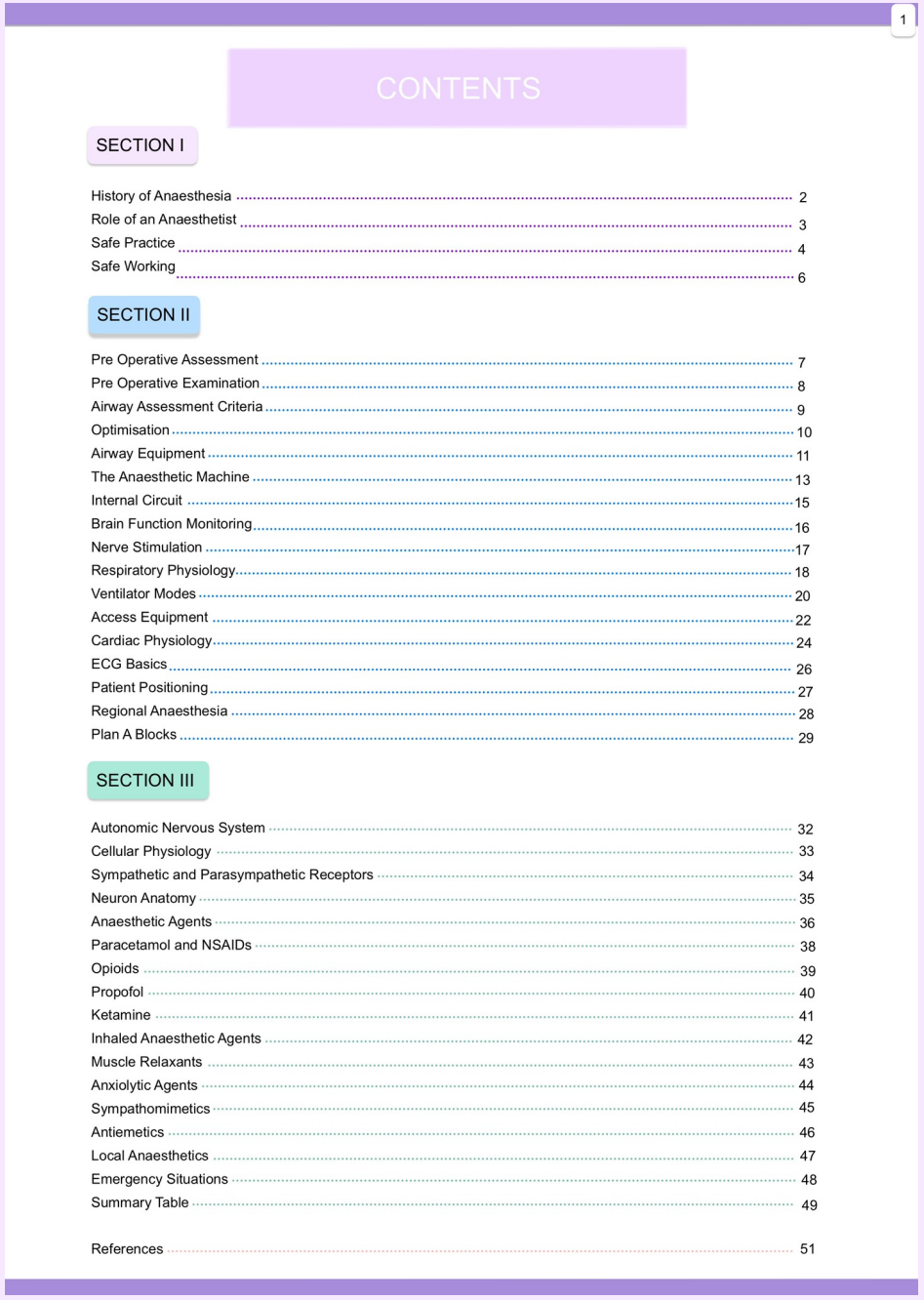
## Background:

- In the UK, transition from foundation to anaesthetics training is a steep learning curve with trainees facing an extensive curriculum.
- The plethora of formal resources can be overwhelming at the beginning of training and shortlisting useful resources is often time intensive, increasing stress and anxiety.
- This self-directed project aimed to develop a concise, practical study guide to introduce the basics of anaesthesia in a visually engaging format.




## Methods:

- Experience of receiving limited exposure to anaesthesia within undergraduate medical education, combined with an early interest, motivated research into the foundational principles and UK training structure of anaesthetics.
- Research of literature identified key concepts and practical knowledge required at the beginning of anaesthetic training. Using personal perspective, as an individual not versed in the specialty, to determine relevant and useful topics for beginners to learn.
- Numerous sources of literature were collated in a study guide, focussing on hand drawn illustrations to enhance understanding.
- Completion of a six-week hands-on anaesthetic placement provided insight into key knowledge requirements for daily practice and facilitated situational awareness of different challenges faced by trainee anaesthetists in real time.
- Verbal feedback and teaching from practicing anaesthetists assisted in ensuring the study guide contained comprehensive content, differentiating between literature and clinically relevant information.



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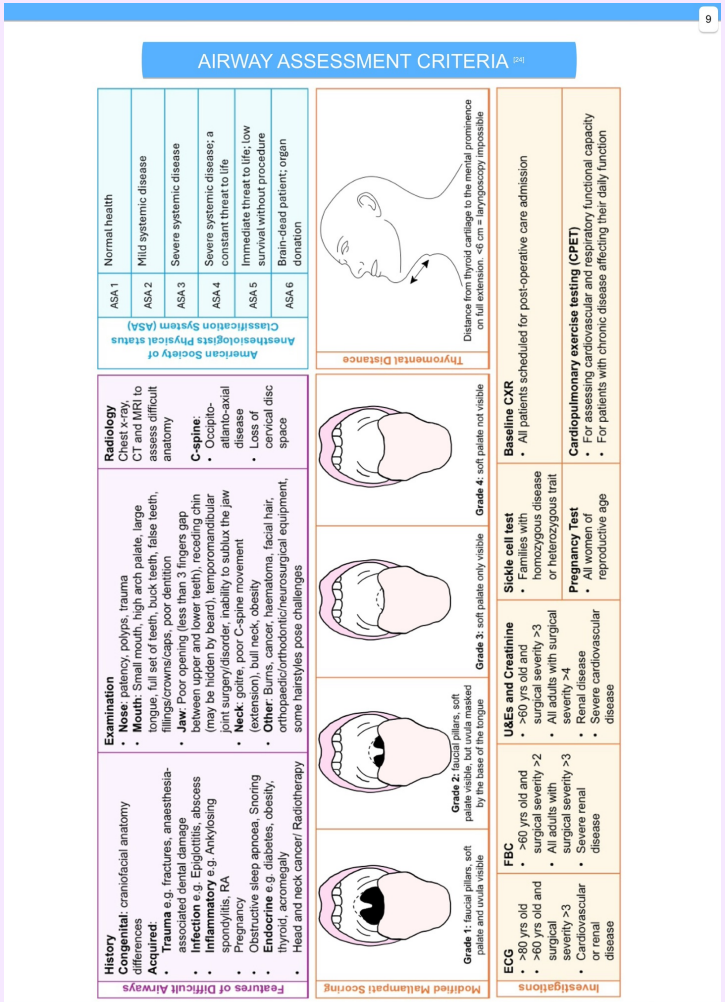
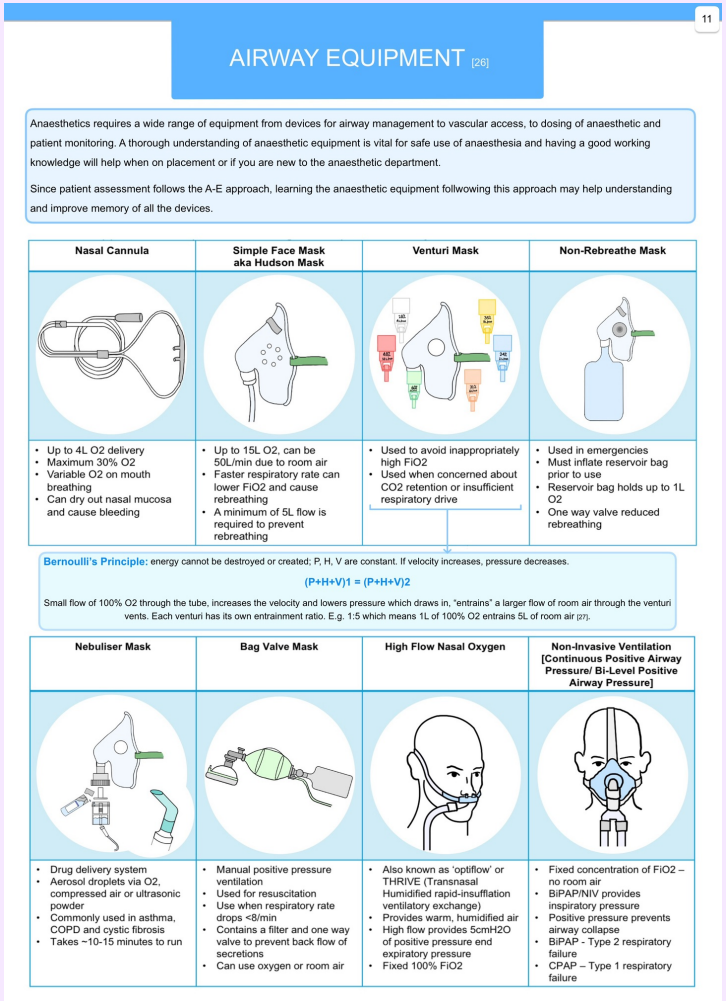
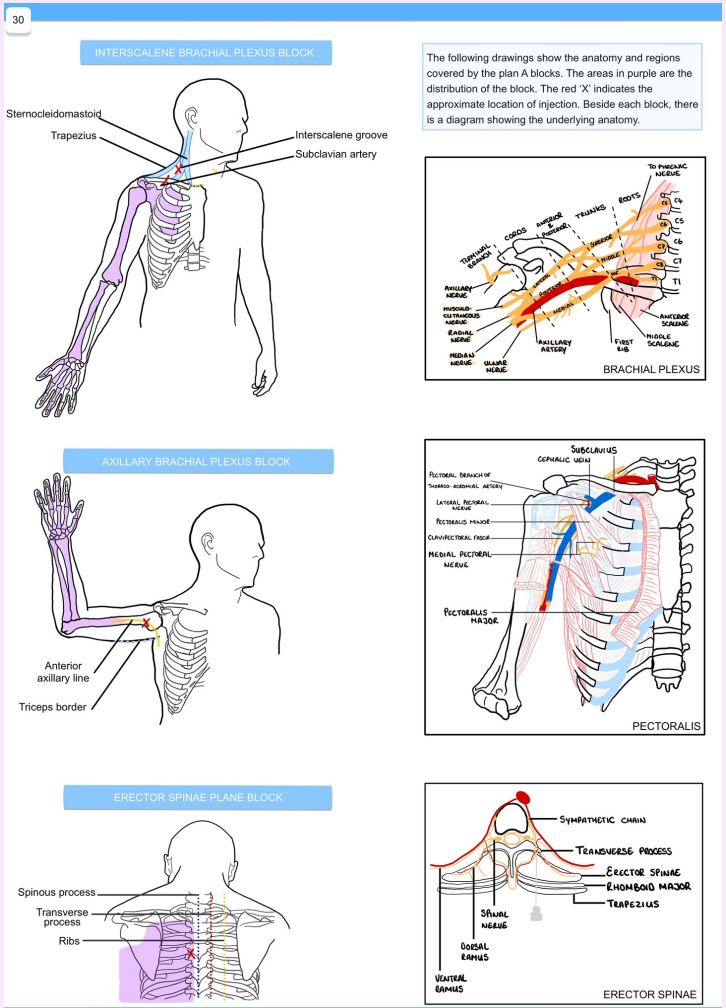
## Results:

- A 53 page study guide was created, using 53 different sources of literature including textbooks, academic papers, online platforms (e-Learning Anaesthesia) and the Royal College of Anaesthesia (RCoA) website.
- Included content was selected based upon recurring themes across the literature and referenced to the RCoA curriculum.
- Hand drawn illustrations were used to enhance content delivery and account for different learning styles.

Included are images of pages from the study guide. The contents page indicates the topics covered and the other pages are examples of the visual illustrations used in the guide. The pharmacology pages are colour coded to the syringe label guidelines of the Association of Anaesthetists of Great Britain and Ireland.



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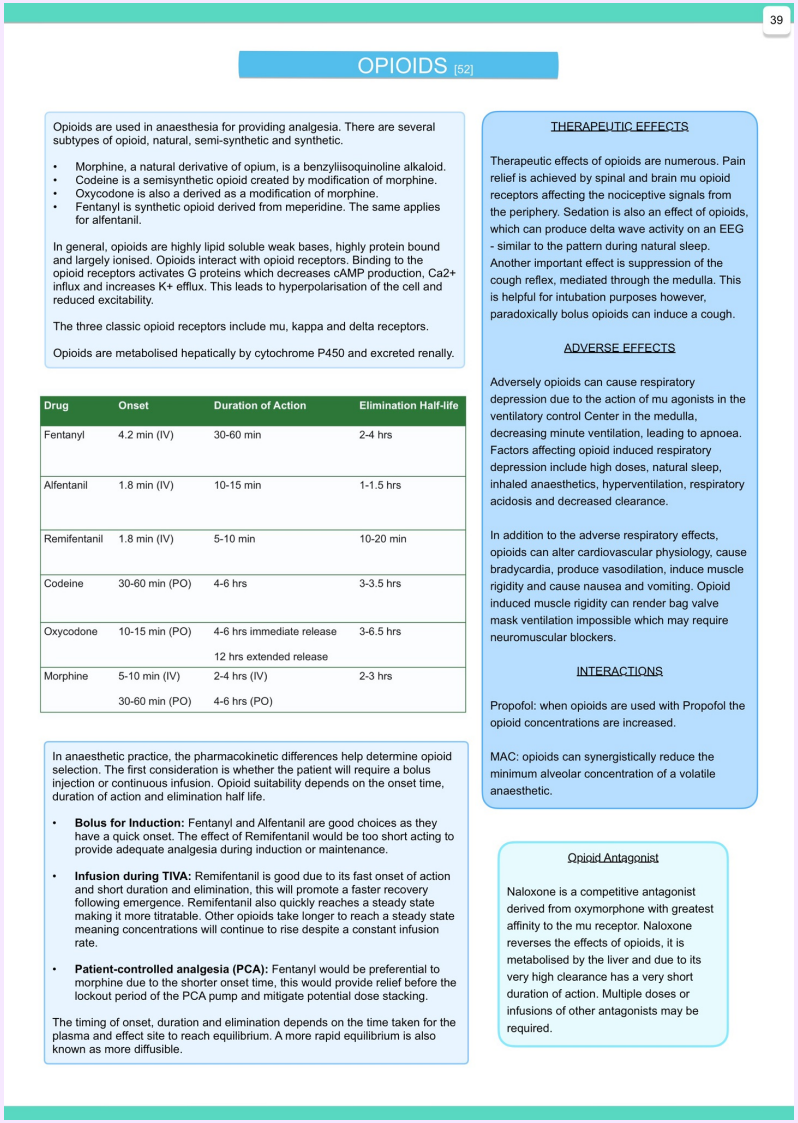

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## OPIOIDS [52]

Opioids are used in anaesthesia for providing analgesia. There are several subtypes of opioid, natural, semi-synthetic and synthetic.

- Morphine, a natural derivative of opium, is a benzyliisoquinoline alkaloid.
- Codeine is a semisynthetic opioid created by modification of morphine.
- Oxycodone is also a derived as a modification of morphine.
- Fentanyl is synthetic opioid derived from meperidine. The same applies for alfentanil.

In general, opioids are highly lipid soluble weak bases, highly protein bound and largely ionised. Opioids interact with opioid receptors. Binding to the opioid receptors activates G proteins which decreases cAMP production, Ca2+ influx and increases K+ efflux. This leads to hyperpolarisation of the cell and reduced excitability.

The three classic opioid receptors include mu, kappa and delta receptors.

Opioids are metabolised hepatically by cytochrome P450 and excreted renally.

| Drug | Onset | Duration of Action | Elimination Half-life |
|---|---|---|---|
| Fentanyl | 4.2 min (IV) | 30-60 min | 2-4 hrs |
| Alfentanil | 1.8 min (IV) | 10-15 min | 1-1.5 hrs |
| Remifentanil | 1.8 min (IV) | 5-10 min | 10-20 min |
| Codeine | 30-60 min (PO) | 4-6 hrs | 3-3.5 hrs |
| Oxycodone | 10-15 min (PO) | 4-6 hrs immediate release<br>12 hrs extended release | 3-6.5 hrs |
| Morphine | 5-10 min (IV)<br>30-60 min (PO) | 2-4 hrs (IV)<br>4-6 hrs (PO) | 2-3 hrs |

In anaesthetic practice, the pharmacokinetic differences help determine opioid selection. The first consideration is whether the patient will require a bolus injection or continuous infusion. Opioid suitability depends on the onset time, duration of action and elimination half life.

- **Bolus for Induction:** Fentanyl and Alfentanil are good choices as they have a quick onset. The effect of Remifentanil would be too short acting to provide adequate analgesia during induction or maintenance.
- **Infusion during TIVA:** Remifentanil is good due to its fast onset of action and short duration and elimination, this will promote a faster recovery following emergence. Remifentanil also quickly reaches a steady state making it more titratable. Other opioids take longer to reach a steady state meaning concentrations will continue to rise despite a constant infusion rate.
- **Patient-controlled analgesia (PCA):** Fentanyl would be preferential to morphine due to the shorter onset time, this would provide relief before the lockout period of the PCA pump and mitigate potential dose stacking.

The timing of onset, duration and elimination depends on the time taken for the plasma and effect site to reach equilibrium. A more rapid equilibrium is also known as more diffusible.

### THERAPEUTIC EFFECTS

Therapeutic effects of opioids are numerous. Pain relief is achieved by spinal and brain mu opioid receptors affecting the nociceptive signals from the periphery. Sedation is also an effect of opioids, which can produce delta wave activity on an EEG - similar to the pattern during natural sleep. Another important effect is suppression of the cough reflex, mediated through the medulla. This is helpful for intubation purposes however, paradoxically bolus opioids can induce a cough.

### ADVERSE EFFECTS

Adversely opioids can cause respiratory depression due to the action of mu agonists in the ventilatory control Center in the medulla, decreasing minute ventilation, leading to apnoea. Factors affecting opioid induced respiratory depression include high doses, natural sleep, inhaled anaesthetics, hyperventilation, respiratory acidosis and decreased clearance.

In addition to the adverse respiratory effects, opioids can alter cardiovascular physiology, cause bradycardia, produce vasodilation, induce muscle rigidity and cause nausea and vomiting. Opioid induced muscle rigidity can render bag valve mask ventilation impossible which may require neuromuscular blockers.

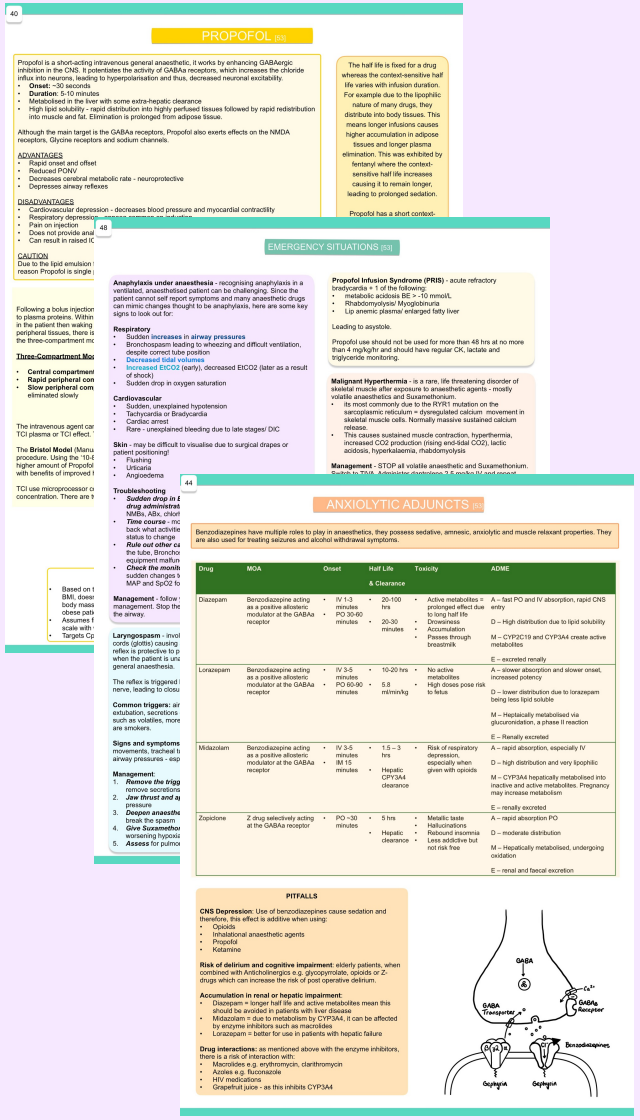
### INTERACTIONS

Propofol: when opioids are used with Propofol the opioid concentrations are increased.

MAC: opioids can synergistically reduce the minimum alveolar concentration of a volatile anaesthetic.

### Opioid Antagonist

Naloxone is a competitive antagonist derived from oxymorphone with greatest affinity to the mu receptor. Naloxone reverses the effects of opioids, it is metabolised by the liver and due to its very high clearance has a very short duration of action. Multiple doses or infusions of other antagonists may be required.



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## PROPOFOL [53]

Propofol is a short-acting intravenous general anaesthetic, it works by enhancing GABAergic inhibition in the CNS. It potentiates the activity of GABAa receptors, which increases the chloride influx into neurons, leading to hyperpolarisation and thus, decreased neuronal excitability.

- **Onset:** ~30 seconds
- **Duration:** 5-10 minutes
- Metabolised in the liver with some extra-hepatic clearance
- High lipid solubility - rapid distribution into highly perfused tissues followed by rapid redistribution into muscle and fat. Elimination is prolonged from adipose tissue.

Although the main target is the GABAa receptors, Propofol also exerts effects on the NMDA receptors, Glycine receptors and sodium channels.

ADVANTAGES
- Rapid onset and offset
- Reduced PONV
- Decreases cerebral metabolic rate - neuroprotective
- Depresses airway reflexes

DISADVANTAGES
- Cardiovascular depression - decreases blood pressure and myocardial contractility
- Respiratory depression
- Pain on injection
- Does not provide anal
- Can result in raised IC

CAUTION
Due to the lipid emulsion
reason Propofol is single

The half life is fixed for a drug whereas the context-sensitive half life varies with infusion duration. For example due to the lipophilic nature of many drugs, they distribute into body tissues. This means longer infusions causes higher accumulation in adipose tissues and longer plasma elimination. This was exhibited by fentanyl where the context-sensitive half life increases causing it to remain longer, leading to prolonged sedation.

Propofol has a short context-

Following a bolus injection to plasma proteins. Within in the patient then waking peripheral tissues, there is the three-compartment mo

Three-Compartment Mo

- Central compartment
- Rapid peripheral com
- Slow peripheral com eliminated slowly

The intravenous agent car TCI plasma or TCI effect.

The Bristol Model (Manu procedure. Using the "10-L higher amount of Propofol with benefits of improved t

TCI use microprocessor c concentration. There are t

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## EMERGENCY SITUATIONS [53]

**Anaphylaxis under anaesthesia** - recognising anaphylaxis in a ventilated, anaesthetised patient can be challenging. Since the patient cannot self report symptoms and many anaesthetic drugs can mimic changes thought to be anaphylaxis, here are some key signs to look out for:

**Respiratory**
- Sudden increases in airway pressures
- Bronchospasm leading to wheezing and difficult ventilation, despite correct tube position
- Decreased tidal volumes
- Increased EtCO2 (early), decreased EtCO2 (later as a result of shock)
- Sudden drop in oxygen saturation

**Cardiovascular**
- Sudden, unexplained hypotension
- Tachycardia or Bradycardia
- Cardiac arrest
- Rare - unexplained bleeding due to late stages/ DIC

**Skin** - may be difficult to visualise due to surgical drapes or patient positioning!
- Flushing
- Urticaria
- Angioedema

**Troubleshooting**
- *Sudden drop in E drug administrat* NMBs, ABx, chlor
- *Time course* - mc back what activitie status to change
- *Rule out other ca* the tube, Broncho equipment malfun
- *Check the monit* sudden changes t MAP and SpO2 fo

**Management** - follow management. Stop the the airway.

**Laryngospasm** - invol cords (glottis) causing reflex is protective to p when the patient is un general anaesthesia.

The reflex is triggered nerve, leading to obstru

**Common triggers:** air extubation, secretions such as volatiles, more are smokers.

**Signs and symptoms** movements, tracheal t airway pressures - esp

**Management:**
1. *Remove the trigg* remove secretions
2. *Jaw thrust and a* pressure
3. *Deepen anaesthe* break the spasm
4. *Give Suxamethon* worsening hypoxia
5. *Assess* for pulmo

Based on t BMI, doesn body mass obese pati Assumes t scale with Targets Cp

**Propofol Infusion Syndrome (PRIS)** - acute refractory bradycardia + 1 of the following:
- metabolic acidosis BE > -10 mmol/L
- Rhabdomyolysis/ Myoglobinuria
- Up anemic plasma/ enlarged fatty liver

Leading to asystole.

Propofol use should not be used for more than 48 hrs at no more than 4 mg/kg/hr and should have regular CK, lactate and triglyceride monitoring.

**Malignant Hyperthermia** - is a rare, life threatening disorder of skeletal muscle after exposure to anaesthetic agents - mostly volatile anaesthetics and Suxamethonium.
- its most commonly due to the RYR1 mutation on the sarcoplasmic reticulum = dysregulated calcium movement in skeletal muscle cells. Normally massive sustained calcium release.
- This causes sustained muscle contraction, hyperthermia, increased CO2 production (rising end-tidal CO2), lactic acidosis, hyperkalaemia, rhabdomyolysis

**Management** - STOP all volatile anaesthetic and Suxamethonium.

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## ANXIOLYTIC ADJUNCTS [53]

Benzodiazepines have multiple roles to play in anaesthetics, they possess sedative, amnesic, anxiolytic and muscle relaxant properties. They are also used for treating seizures and alcohol withdrawal symptoms.

| Drug | MOA | Onset | Half Life & Clearance | Toxicity | ADME |
|---|---|---|---|---|---|
| Diazepam | Benzodiazepine acting as a positive allosteric modulator at the GABAa receptor | • IV 1-3 minutes<br>• PO 30-60 minutes | • 20-100 hrs<br>• 20-30 minutes | • Active metabolites = prolonged effect due to long half life<br>• Drowsiness<br>• Accumulation<br>• Passes through breastmilk | A – fast PO and IV absorption, rapid CNS entry<br>D – High distribution due to lipid solubility<br>M – CYP2C19 and CYP3A4 create active metabolites<br>E – excreted renally |
| Lorazepam | Benzodiazepine acting as a positive allosteric modulator at the GABAa receptor | • IV 3-5 minutes<br>• PO 60-90 minutes | • 10-20 hrs<br>• 5.8 ml/min/kg | • No active metabolites<br>• High doses pose risk to fetus | A – slower absorption and slower onset, increased potency<br>D – lower distribution due to lorazepam being less lipid soluble<br>M – Heptaically metabolised via glucuronidation, a phase II reaction<br>E – Renally excreted |
| Midazolam | Benzodiazepine acting as a positive allosteric modulator at the GABAa receptor | • IV 3-5 minutes<br>• IM 15 minutes | • 1.5 – 3 hrs<br>• Hepatic CPY3A4 clearance | • Risk of respiratory depression, especially when given with opioids | A – rapid absorption, especially IV<br>D – high distribution and very lipophilic<br>M – CYP3A4 hepatically metabolised into inactive and active metabolites. Pregnancy may increase metabolism<br>E – renally excreted |
| Zopiclone | Z-drug selectively acting at the GABAa receptor | • PO ~30 minutes | • 5 hrs<br>• Hepatic clearance | • Metallic taste<br>• Hallucinations<br>• Rebound insomnia<br>• Less addictive but not risk free | A – rapid absorption PO<br>D – moderate distribution<br>M – Hepatically metabolised, undergoing oxidation<br>E – renal and faecal excretion |

**PITFALLS**

**CNS Depression**: Use of benzodiazepines cause sedation and therefore, this effect is additive when using:
- Opioids
- Inhalational anaesthetic agents
- Propofol
- Ketamine

**Risk of delirium and cognitive impairment**: elderly patients, when combined with Anticholinergics e.g. glycopyrrolate, opioids or Z-drugs which can increase the risk of post operative delirium.

**Accumulation in renal or hepatic impairment**:
- Diazepam = longer half life and active metabolites mean this should be avoided in patients with liver disease
- Midazolam = due to metabolism by CYP3A4, it can be affected by enzyme inhibitors such as macrolides
- Lorazepam = better for use in patients with hepatic failure

**Drug interactions**: as mentioned above with the enzyme inhibitors, there is a risk of interaction with:
- Macrolides e.g. erythromycin, clarithromycin
- Azoles e.g. fluconazole
- HIV medications
- Grapefruit juice - as this inhibits CYP3A4




## Discussion:

- This project provides a basic introduction to anaesthesia, covering topics that arise in day to day practice.
- It can be used as a supplementary aid alongside existing material, helping guide trainees to discover knowledge gaps and provide a foundation for their learning and future development.
- The learning process is unique to every individual, the visual impact of this guide with illustrations, bright colours and colour-coded pharmacology may help trainees to digest the extensive curriculum. As well as increasing accessibility to learners with specific learning differences such as dyslexia and ADHD.

## Conclusion:

- The transition into anaesthetic training is a large undertaking and a resource containing the basics can help mitigate stress and anxiety around starting a new, exciting career.
- Further development of the guide could include addition of more content and illustrations, with potential distribution regionally and nationally. This has potential to improve trainee confidence, competence and well-being in a standardised manner.
- To ensure the guide is fit for purpose, a project to disseminate the material and collect feedback from current trainees and clinicians would help tailor the guide, ensuring the user's needs are met appropriately.

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