Induction of anaesthesia

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Abstract

General anaesthesia is a temporary state of unconsciousness that is induced to facilitate a therapeutic procedure. Induction is the first stage of a sequential process. It commences with patient preparation and assessment away from theatre, and continues in the safe and monitored environment of the anaesthetic room or operating theatre, where the administration of drugs and airway interventions take place. The anaesthetic then transits through maintenance, emergence and recovery phases. The exact mechanism of induction-whether it be intravenous, inhalational or rapid sequence induction-depends on the needs of the patient and the procedure planned. As general anaesthesia is seldom a therapeutic intervention in itself, it is essential that inherent risks to the patient are minimized.

Keywords Complications of induction; inhalational induction; intravenous induction agents; mind the gap; pre-oxygenation; rapid sequence induction

Royal College of Anaesthetists CPD matrix: 1A02, 1E06, 2A03

Learning objectives

After reading this article, you should be able to:

- demonstrate the preparation required prior to induction of anaesthesia
- compare the different methods of induction
- identify the appropriate induction for each individual patient
- recognize and manage the complications associated with induction of anaesthesia

Introduction

Induction of anaesthesia is the initiation of a temporary state of unconsciousness, amnesia, analgesia and muscle relaxation. Rarely is it performed as a therapeutic procedure in itself, but rather as a means to make a therapeutic procedure more tolerable. It is therefore imperative it is performed in the safest way possible. It is the duty of every anaesthetist to pick the most suitable agent, via the most suitable route, to ensure these endpoints are met whilst making every effort to minimize the inherent risks to the patient.

Before induction

Preoperative assessment

The aims of the preoperative assessment are twofold. Firstly, it serves to identify the high-risk patient (and arrange appropriate investigations or treatment pre-emptively); secondly, it enables the anaesthetist to design a comprehensive perioperative strategy. This should be explained to and agreed with the patient, thereby gaining informed consent and facilitating planning of the case with the surgical team.

During the preoperative assessment a focused anaesthetic history should be taken as detailed in Box 1. An assessment of the airway and a relevant systemic examination should follow. Many patients are unaware of the details of their diagnoses and investigation results. A review of their hospital notes is often necessary to reveal further vital information relevant to planning their anaesthetic.

In the theatre complex

The anaesthetic equipment should be checked in a thorough and systematic way as recommended in the AAGBI guidelines before the patient arrives in the anaesthetic room (http://www.aagbi.org/sites/default/files/checking_anaesthetic_equipment_2012.pdf). The anaesthetist must have access to, and be aware of the location of, the difficult airway equipment, defibrillator and emergency drugs such as dantrolene, intralipid™ and sugammadex™.

The anaesthetist should be accompanied in the anaesthetic room (AR) by a trained assistant, either an anaesthetic nurse or operating department practitioner (ODP). The World Health Organization checklist for safer surgery should be a part of the pre-induction sequence for every case. Monitoring standards are specified by the AAGBI (Box 2), and should be applied to every patient unless likely to cause distress, such as patients with special needs and young children.

There are several scenarios in which such preparation prior to induction is not feasible. Emergency surgical procedures such as ruptured abdominal aortic aneurysms and category 1 Caesareans will bypass the anaesthetic room altogether. In these circumstances, or when called to the emergency department for emergency induction of anaesthesia, an AMPLE history (allergies, medication, past medical history, last meal intake, events leading to illness) may be more appropriate.

Induction techniques

Intravenous induction

Induction via intravenous agents is the most commonly used method for induction of anaesthesia. Administered either as a manual bolus or via a syringe driver as part of total intravenous anaesthesia (TIVA), they cause loss of consciousness in approximately one arm–brain circulation time. Since the 1930s there have been many IV induction agents but only four are in common use today: propofol, thiopentone, etomidate and ketamine.[1]

Propofol: (2,6-di-isopropyl phenol) is a highly lipid soluble phenolic derivative. Its mechanism of action is largely unknown, but the accepted theory is potentiation of the inhibitory activity

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Anaesthetic history

- Problems with previous anaesthetics
- Family history of problems with anaesthetics
- Co-morbidites and the impact these have on their functional capacity
- Allergies
- Medication, including alternative remedies, and which have been taken or omitted
- Any smoking, alcohol or illicit drug use
- When they last ate or drank
- Dentition, establish any loose teeth or dental work

Box 1

of the GABA A receptor. It is presented as a 1% or 2% lipid and water emulsion containing soya bean oil and purified egg phosphatide, but is not held to be allergenic to those allergic to egg or soya. Propofol is the most commonly used IV induction agent in the world and is also used for maintenance during TIVA and for sedation in theatre and the intensive care unit (ITU). The induction dose is usually 1–3 mg/kg and its short duration of action is due to its large volume of distribution. In adults it should be injected into a steadily running drip at 1–2 ml per second and titrated to effect. In children or in a rapid sequence induction, a predetermined dose should be injected at 5 ml per second straight into the cannula. Propofol is painful on injection. The most effective method of reducing the pain is to use a large antecubital vein or inject 20 mg of lidocaine while preventing drainage with venous occlusion.[2]

Thiopentone: is a short-acting barbiturate, and in modern anaesthesia is predominantly used for rapid sequence induction at a dose of 3–7 mg/kg. It is also used on intensive care for the management of status epilepticus and as an infusion to reduce intracranial pressure. It is presented as a pale yellow powder that is dissolved in 20 ml of water to provide a 2.5% alkaline solution of pH 10.5. Being so alkaline, inadvertent intra-arterial injection is limb threatening. After bolus injection, recovery of consciousness is even more rapid than propofol and is again due to drug redistribution. In the 5th National Audit Project (NAP5), thiopentone was identified as a risk factor for accidental awareness during general anaesthesia (AAGA) owing to this shorter duration of anaesthesia from a single bolus so it is recommended that additional doses be given if there is any delay in securing the airway due to unanticipated difficulties.[3] By contrast, thiopentone is metabolized by the liver to several active metabolites that will accumulate during an infusion due to hepatic enzyme saturation and zero order kinetics. The use of thiopentone has reduced significantly since the introduction of propofol, largely due to favourable laryngeal conditions provided by propofol for the insertion of laryngeal masks and the clear headed recovery associated with propofol.

Monitoring for induction and maintenance of anaesthesia

1 Pulse oximeter
2 Non invasive blood pressure monitor
3 Electrocardiograph
4 Airway gases: oxygen, carbon dioxide and vapour
5 Airway pressure

Box 2

Etomidate: is an ester imidazole derivative. At an induction dose of 0.3 mg/kg its high lipid solubility and large non-ionized fraction cause rapid unconsciousness with little effect on systemic vascular resistance, myocardial contractility or heart rate. After gaining popularity in the 1970s, it became clear that etomidate infusions used as sedation on ICU resulted in increased mortality due to adrenocortical suppression, specifically the P450 enzymes 11-β and 17-α hydroxylase involved in steroidogenesis.

Ketamine: is a phencyclidine derivative that antagonizes N-Methyl-D-aspartic acid receptors. At an induction dose of 1–2 mg/kg it causes dissociative anaesthesia, analgesia, amnesia, anxiolysis and bronchodilatation. Unlike other IV induction agents it stimulates the sympathetic nervous system making it suitable for hypotensive patients but should be used with caution in those with ischaemic heart disease. Despite the drugs attractive profile its popularity in the developed world has been limited by unpleasant psychogenic side effects. In the past, ketamine has been avoided in head injuries due to concerns it would raise intracranial pressure. It is now clear that this is not the case; indeed there is the possibility that it has neuroprotective activity in its reduction of the neurotoxin glutamate.

Future agents: PFO713 has a similar profile to propofol, producing reliable, rapid anaesthesia without pain on injection and with improved cardiovascular stability. Carboetomidate is an etomidate analogue that has reduced ability to inhibit the enzyme 11 β-hydroxylase, and hence reduced adrenocortical suppression. Both agents are awaiting further clinical trials.[3]

Co-induction agents: IV induction agents are very rarely given in isolation. Adjuncts such as short-acting opioids (e.g. fentanyl, alfentanil and remifentanil) are given to obtund the response to laryngoscopy, reduce the dose of the induction agent and hence reduce side effects, and to provide intra-operative analgesia. Midazolam provides anxiolysis prior to induction, and is synergistic with induction agents in that the depth of anaesthesia is more than the sum of each of the individual agents. In cases where elevation in blood pressure or intracranial pressure is of particular concern, lidocaine 1.5 mg/kg or a beta-blocker such as esmolol at a dose of 2 mg/kg IV can be given 3 minutes prior to intubation.

Pre-oxygenation

Pre-oxygenation is the process of replacing nitrogen with oxygen within the lungs, which maximizes haemoglobin and plasma saturation and more importantly creates an oxygen reservoir within the functional residual capacity (FRC). This oxygen reservoir will extend the time until desaturation during apnoeic periods.

A standard anaesthetic induction in a healthy, slim patient involves the administration of a hypnotic agent followed immediately by airway maintenance and ventilation (either