Inhalation (gas) Induction

By the perioperativeCPD team

Introduction
Inhalation or gas induction is the use of volatile anaesthetic gases only to induce anaesthesia. Although it is routine to induce general anaesthesia by the intravenous (IV) route, the inhalational route remains an acceptable method and is preferred in certain circumstances. This can include paediatrics or where IV access is not possible. Like IV induction inhalational induction has both risks and benefits.

Historical Perspective
Historically, anaesthesia was inhalation based. Nitrous oxide was first used in 1844, ether in 1846 and chloroform the following year.

IV induction was introduced in the early 20th Century with the synthesis of barbiturates. Thiopental (Pentothal sodium) was the first IV anaesthetic introduced into clinical practice in the 1930s. It revolutionized anaesthesia by reducing the time of induction, so that less time was spent in the traumatic second stage of anaesthesia. Thiopental is still used today along with propofol for IV induction.

Note: although technically ‘inhalational induction’ is the correct term, ‘gas induction’ is frequently used and both terms will be used in this module.
Why an inhalational induction/ what are the advantages?
For most patients without airway pathology, IV agents provide for more rapid definitive airway control and avoid the difficult second stage of anaesthesia. In this group IV induction is safer. It is also more convenient and pleasant for the patient. However, there are certain reasons why an inhalation induction may be used to good effect, or may be the preferred technique:

**Less traumatic**
Inhalation induction may be less traumatic than venous cannulation in:
- Children
- Needle phobic adults
- Adults with learning disabilities

**Where venous access is difficult**
Inhalation induction is valuable where venous access is difficult. After induction, peripheral venous vasodilation will facilitate cannulation. This needs to be balanced against the risk of inducing anaesthesia without IV access.

**Spontaneous ventilation**
With inhalation induction spontaneous ventilation is maintained. This is valuable where a difficult airway is being managed, or where positive pressure ventilation is to be avoided. Caution must be shown with deep levels of inhalation anaesthesia where apnoea can still occur.

**Airway tone**
Airway tone is better maintained than with IV induction. This is useful with difficult airway management. Caution once deep volatile anaesthesia is achieved, as airway tone may be reduced.

**Depth of anaesthesia**
It is possible to alter depth of anaesthesia during the course of induction. This compares with IV induction, where the dose cannot be changed once administered.

**Acute anaphylaxis**
Acute anaphylaxis does not seem to occur with inhaled volatile agents. This may confer an advantage in patients with a history of anaphylaxis where the precipitating agent(s) has not been identified.

**Bronchodilator effect**
The bronchodilator effect of volatile agents may confer an advantage in brittle asthmatics. It appears to be mediated by lowering calcium concentrations in smooth muscle. Caution with desflurane and isoflurane where the pungent smell may neurally mediate bronchoconstriction.

**Brief outpatient anaesthesia effect**
For brief outpatient anaesthesia, e.g. dental extractions in children, rapid elimination of volatile agents via the lungs may provide more rapid recovery than elimination of IV drugs. (This idea is now being challenged by short acting IV agents, e.g. propofol and remifentanil.)
Disadvantages/risks of a inhalational induction

**Smell of agent**
The smell may cause anxiety or refusal of the technique.

**Airway irritation**
The use of some induction agents (isoflurane, desflurane) may result in coughing and laryngospasm.

**Traumatic second stage of anaesthesia**
Traumatic second stage of anaesthesia can be particularly difficult in adults, and difficult also for the anaesthetist. It is advisable to forewarn those working with you, and to seek their assistance if necessary.

**Cardiovascular and respiratory depression**
This is dose related and therefore occurs at deeper levels of anaesthesia.

**Delayed definitive airway control**
This is a potential problem in a high risk, vomiting patient.

**Theatre pollution**
Scavenging during inhalation induction is difficult in children, where there is a refusal of face mask application.

**Malignant hyperthermia**
All volatile agents can precipitate malignant hyperthermia in susceptible individuals.

**Emergence issues**
There can be issues with sevoflurane emergence after an inhalational induction. Emergence delirium is a transient state of marked irritation and disassociation upon awakening which does not respond to consoling measures.

Which gases are used for an inhalational induction?
Although theoretically any volatile agent can be used to induce anaesthesia, only sevoflurane, halothane and nitrous oxide are commonly used. Desflurane is a faster acting agent but is irritant to the airway with associated breath holding and laryngospasm.

The following properties make ‘ideal’ inhalation agents:

- Low blood:gas partition coefficient allowing rapid induction
- A low minimum alveolar concentration (MAC) allowing it to be administered in high concentrations of oxygen
- Non-flammable or explosive (no currently used anaesthetics are flammable)
- Non-pungent or odourless
- Non-irritant to the airway
- Non-cardiac depressant
- Non-respiratory depressant
- Non-toxic to liver and kidneys, not metabolised by the body

A note about MAC values: You may see slightly varying values in different books because MAC has a range of values. The values here are those most commonly quoted.
Sevoflurane
Currently, sevoflurane is the inhalational agent of choice, with halothane as a less commonly used alternative, mainly in low resource countries.

The properties of sevoflurane come close to the ideal inhalational agent:

- Low blood: gas partition coefficient
- It has a relatively low MAC of 2.0%
- Non-flammable
- Sweet smelling (although always a matter of debate)
- Non-irritant to the airways
- Non-toxic to the liver and kidneys
- Cardiac and respiratory depression is dose dependent

Halothane
Halothane is still used in developing countries due to its lower cost. Although it does have many properties that make it suitable for gas induction i.e. pleasant smell, low MAC and minimal respiratory depression, it can cause hepatic problems ‘halothane hepatitis’ and arrhythmias.

Halothane is also relatively soluble in blood, so gas induction can be slow compared to sevoflurane, because it takes longer to build up a sufficient concentration in the lungs to achieve anaesthesia. This can be overcome by a technique called ‘over-pressure’ in which high inspired concentrations are given at the start of induction. The vaporiser can give 8x the MAC of halothane. (Sevoflurane can go to 4x MAC)

Nitrous Oxide
Nitrous oxide (N₂O) has been used since the 1840s although its high MAC (104%) means that it cannot be used as the sole agent to achieve a gas induction, especially as it is hypoxic in high concentrations. Nitrous oxide is often used as a carrier gas due to its role in the second gas effect (see below).

Second gas effect of nitrous oxide
Nitrous oxide is rapidly absorbed from the alveoli when it is administered in a high concentration (e.g. 70% with oxygen). If it is co-administered with a volatile agent, e.g. sevoflurane, the rapid absorption of nitrous oxide will result in an increased alveolar concentration of the volatile agent in the lungs. This increase in concentration will result in a more rapid onset of the volatile agent. The actual difference this makes clinically is debatable.

Fig. 1: The second gas effect of nitrous oxide
**Inhalation Induction Techniques**

Two methods of inhalation induction are used:
- **Tidal volume breathing**
- **Vital capacity breath**

They both use either oxygen/volatile agent or oxygen/nitrous oxide/volatile agent mixture.

**Tidal Volume Breathing**

Tidal volume breathing involves the introduction of the inhalation agent (or agents) with the patient breathing normally through the facemask. The volatile agent can either be introduced gradually, or a high concentration may be used from the onset.

This technique is much slower than an intravenous induction. It requires a quiet environment, although gentle verbal reassurance to the patient is valuable.

**Vital Capacity Breath**

Vital capacity breath induction requires:

- Patient co-operation
- A tight sealing face mask (to ensure no atmospheric air is inhaled)
- Preferably, a large (2-3 L) bag on the anaesthetic breathing system

The bag is primed with a high concentration of volatile agent (i.e. 8 % sevoflurane) in oxygen. The patient first empties their lungs down to residual volume and then takes in a vital capacity breath, holding it for as long as possible before resuming normal tidal volume breathing.

As patient co-operation is required, this technique can only be used in older children and adults. However, it is quicker than the tidal volume inhalational method (but not as quick as intravenous induction).

It is advisable to do a practice with the patient using 100 % oxygen, so they can perfect the technique before the real thing.

*Fig 2: Vital Capacity Breath Induction*
Some useful tips to consider when performing a gas induction:

**Premedication.** Consider a lower threshold for using a sedative premedication. You may struggle with a highly anxious patient. Short acting benzodiazepines are often used for this purpose. Although opioids have a sedative effect, they cause respiratory depression, which reduces minute ventilation and slows the delivery of volatile agents to the lungs.

**Keeping children calm.** Get as much parental cooperation as you can. If an infant is happy in the parent’s arms, leave the child there and be ready to transfer to the bed when asleep.

**Keeping children still.** Wrap infants and small children in a blanket if possible, to keep arms under wraps. The child is then less likely to pull and disconnect various connections of the airway.

**Boundaries.** Be aware of the boundary between achieving anaesthesia and overuse of coercion or restraint.

**The excitation phase.** Heads and arms wriggle during the excitation phase. Do not try to hold the head still. Stand behind the patient, keep the mask on the face and rotate the mask with the head as it wriggles.

**Assistance.** Do not be afraid to have a second anaesthetist, who can rapidly secure intravenous access once the patient is asleep, so that you can concentrate on maintaining the airway. As with any anaesthetic, inhalation induction should be conducted with a trained assistant and full monitoring.

**Drugs** Intravenous emergency drugs, should be readily available.

What factors influence the speed of induction?

When a volatile agent is used for induction, anaesthesia occurs when a certain concentration of volatile agent is achieved in the brain; the site of action. This concentration of a volatile agent is usually expressed as a partial pressure.

The speed of onset of any inhaled agent depends mainly on the:

**Inspired concentration (high equals fast)**
Although pungency or tolerability can limit the inspired concentration of agent.

**Cardiac output (low equals fast)**
Higher cardiac output tends to slow induction due to faster clearance into the pulmonary blood flow.

**Gas solubility in blood (low equals fast)**
The more soluble the agent is in blood, i.e. the higher the blood/gas solubility, the faster the uptake of the agent. If an agent is quickly removed from the lungs it takes longer for the concentration to rise.

When understanding this is important to remember that the alveolar concentration equals the brain concentration as the partial pressures are virtually the same in the brain and alveolus. The faster the concentration in the lungs increases the faster the concentration in the brain increases.

So, gas induction is slow if the agent has a high blood/gas solubility, e.g. halothane, because fast removal from the lungs delays the build-up of the alveolar concentration that is required to induce anaesthesia. Conversely, if an agent is insoluble, less agent is absorbed, the concentration in the lungs rises rapidly and induction is faster.

So, gas induction is fast if the agent, such as sevoflurane, is relatively insoluble in blood because removal of agent from the lungs is slow.
**MAC (low equals fast)**
Potency of anaesthetic agents is compared using Minimum Alveolar Concentration (MAC). MAC is the alveolar concentration at one atmosphere which abolishes motor response to a standardized incision in 50% of patients. The MAC of an agent is inversely proportional to potency; i.e. more potent agents require smaller alveolar concentrations to produce anaesthesia.

The MAC of halothane is 0.75% which makes it of high potency. This is an advantage as the vaporiser can give 8x the MAC speeding up an otherwise slow induction. This technique is called over-pressure.

<table>
<thead>
<tr>
<th>Anaesthetic</th>
<th>MAC%</th>
<th>Blood: Gas Coefficient</th>
<th>Rate of onset</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitrous Oxide</td>
<td>104</td>
<td>0.47</td>
<td>Fast</td>
</tr>
<tr>
<td>Desflurane</td>
<td>6</td>
<td>0.45</td>
<td>Fast</td>
</tr>
<tr>
<td>Sevoflurane</td>
<td>2</td>
<td>0.65</td>
<td>Fast</td>
</tr>
<tr>
<td>Isoflurane</td>
<td>1.15</td>
<td>1.4</td>
<td>Medium</td>
</tr>
<tr>
<td>Halothane</td>
<td>0.75</td>
<td>2.3</td>
<td>Slow</td>
</tr>
</tbody>
</table>

**Guedel’s stages of anaesthesia**

In August 1917, Dr Arthur Guedel arrived in France as part of the American Expeditionary Force (AEF). Of the 491 medical officers in the AEF, none had specialist anaesthetic training. Guedel was one of a team of four doctors who worked continuously for 72 hours in theatre, due to the sheer number of casualties.

To train lay staff and nurses to administer an anaesthetic safely, Dr Guedel devised a wall chart that illustrated four stages of anaesthesia. By the end of the war he was using his chart to supervise the delivery of anaesthesia at 40 sites, and he and his staff dealt with many of the 250 000 American casualties of World War I.

*Fig 3 Dr Arthur Guedel and his 4 stages of anaesthesia.*
Guedel’s revolution was to include the pupillary changes that occurred with increasing doses of ether. In an age without monitors, pupil changes were easily observable physiological monitors of depth of anaesthesia. Guedel’s original eye signs included the activity of the motor muscles of the eye (deviant gaze) and pupillary dilation. The eyelid, corneal and eyelash reflexes were added later.

**Guedel’s Classical Stages**

**Stage One:**
Stage 1, also known as *induction*, is the period between the administration of induction agents and loss of consciousness. During this stage, the patient progresses from analgesia without amnesia to analgesia with amnesia. Patients can carry on a conversation at this time.

**Stage Two:**
Stage 2, also known as the excitement stage, is the period following loss of consciousness and marked by excited and delirious activity. During this stage, the patient’s respiration and heart rate may become irregular. In addition, there may be uncontrolled movements, vomiting, suspension of breathing, and pupillary dilation. The combination of spastic movements, vomiting, and irregular respiration may compromise the patient's airway. Modern rapidly acting drugs used today minimize time in this stage and reach Stage 3 as fast as possible. IV anaesthetics such as propofol bypass this stage totally.

**Stage Three:**
In Stage 3, also known as *surgical anaesthesia*, the skeletal muscles relax, vomiting stops, respiratory depression occurs, and eye movements slow and then stop. The patient is unconscious and ready for surgery. This stage is divided into four planes:

1. The eyes roll, then become fixed;
2. Corneal and laryngeal reflexes are lost;
3. The pupils dilate and light reflex is lost;
4. Intercostal paralysis and shallow abdominal respiration occur.

**Stage Four**
Stage 4, also known as overdose, occurs when too much anaesthetic medication is given relative to the amount of surgical stimulation and the patient has severe brainstem or medullary depression, resulting in a cessation of respiration and potential cardiovascular collapse. This stage is lethal without cardiovascular and respiratory support, but should not happening with modern anaesthetic monitoring.

**Guedel’s Stages Today**
All anaesthetists must be able to recognize the clinical signs of an anaesthetic which is either too light or too deep. At the time Guedel developed his classification of the stages and planes of anaesthesia, his main concern was anaesthetic overdose causing cardio-respiratory collapse and death, as they had no electronic monitoring.

Guedel’s observed physical signs corresponded with depth of anaesthesia as long as only a single anaesthetic agent was used, as depth of anaesthesia was dose-dependent. Several features of anaesthesia today can adversely affect the observation of such physical signs including the use of premedication, opiates and muscle relaxants.
References and other reading


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