Anaesthesia

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Lec 3

General Anaesthesia

Intro

Anaesthetists have to be familiar with a wide range of drugs that (unlike other in most branches of medicine) are almost always given parenterally: either IV or via inhalational. In addition to their effect on the CNS, most drugs have undesirable actions on many other body systems, of which the anaesthetists must be fully aware.

Just like the airplane trip (take off, flying and landing), the steps of general anaesthesia are induction, maintenance and recovery.

IV induction of anaesthesia

This is most frequently achieved in adults by the IV injection of drug. Consciousness is lost rapidly as the concentration of the drug in the brain rises very quickly. The drug is then redistributed to other tissues and plasma concentration falls; this is followed by a fall in brain concentration and the patient recovers consciousness. Despite a short duration of action, elimination, usually by hepatic metabolism, may take considerably longer and lead to accumulation. Consequently most drugs are not given repeatedly to maintain anaesthesia (only exception is propofol).

1. **Ketamine (Ketalar)**
   - May cause tachycardia and HT, precaution use in cardiovascular disease patient, useful in hypovolemic (shocked) patients.
   - Bronchodilator, minimal depression of respiration and laryngeal reflexes are well preserved.
   - Increase intracerebral and intraocular pressures.
   - Profound analgesia (especially in sub-anaesthetic doses)
   - Useful in remote areas (prehospital).
   - Vivid hallucination.

2. **Thiopentone (Pentothal)**
   - Dose dependent hypotension, worse in hypovolemic and cardiac disease patients.
   - Apnea and depress respiration.
   - Decrease intracranial pressure and considered powerful anticonvulsant.
   - Patients may 'taste' garlic or onion!
   - Cumulative, delayed recovery after repeated doses.

3. **Propofol (Diprivan)**
   - Hypotension, worse in hypovolemic and cardiac disease patients.
   - Apnea (up to 60 s) and depress respiration.
   - Reduce cerebral blood flow and ICP.
   - Pain on injection, involuntary movements and hiccough.
   - Non-accumulative, repeated injections or infusion used to maintain anaesthesia (TIVA : Total IV Anaesthesia).

Maintenance of Anaesthesia

This can be achieved either by using one of a variety of inhalational anaesthetics in oxygen, or by an IV infusion of a drug, most commonly Propofol.

Inhalational Anaesthesia

Inhalational anaesthetic is a group of halogenated hydrocarbons with a relatively low boiling point. A 'vaporizer' is used to produce an accurate concentration in the inspired gas mixture. There are two concepts that will help in understanding the use of inhalational anaesthetics; minimum alveolar concentration and solubility.

1. Minimum Alveolar Concentration

To compare potencies and side-effects of the inhalational anaesthetics, MAC is used; it is the concentration required to prevent movement in 50% of patients following a surgical stimulus. At 1 MAC or multiples, the anaesthetic effect will be the same and a comparison of the side-effects can be made. Compounds with a low potency (Desflurane) will have a high MAC, those with high potency (Halothane) will have a low MAC. The value of MAC is reduced in elderly, in those with hypotension, hypothermia, concurrent use of opioids and acute alcoholism. Otherwise it is increased in infants and young age and in those with chronic alcoholic or drug abusers.

2. Solubility

Inhalational anaesthetics exert their effects on the CNS. It is the partial pressure in brain that is responsible for the anaesthetic effect and this follows closely the partial pressure in the alveoli. One of the determinants of the alveolar partial pressure is how soluble the inhalational anaesthetic in the blood. Relatively *insoluble* anaesthetic (Desflurane) is removed slowly from the alveoli by pulmonary blood. The alveolar (and brain) partial pressures rise quickly and anaesthesia is induced rapidly. In contrast, a *soluble* anaesthetic (Halothane) is removed rapidly from the alveoli into the pulmonary blood, limiting the rate of rise of alveolar and brain partial pressures. Consequently, induction will be slower. Recovery from anaesthesia follows similar principles in reverse.

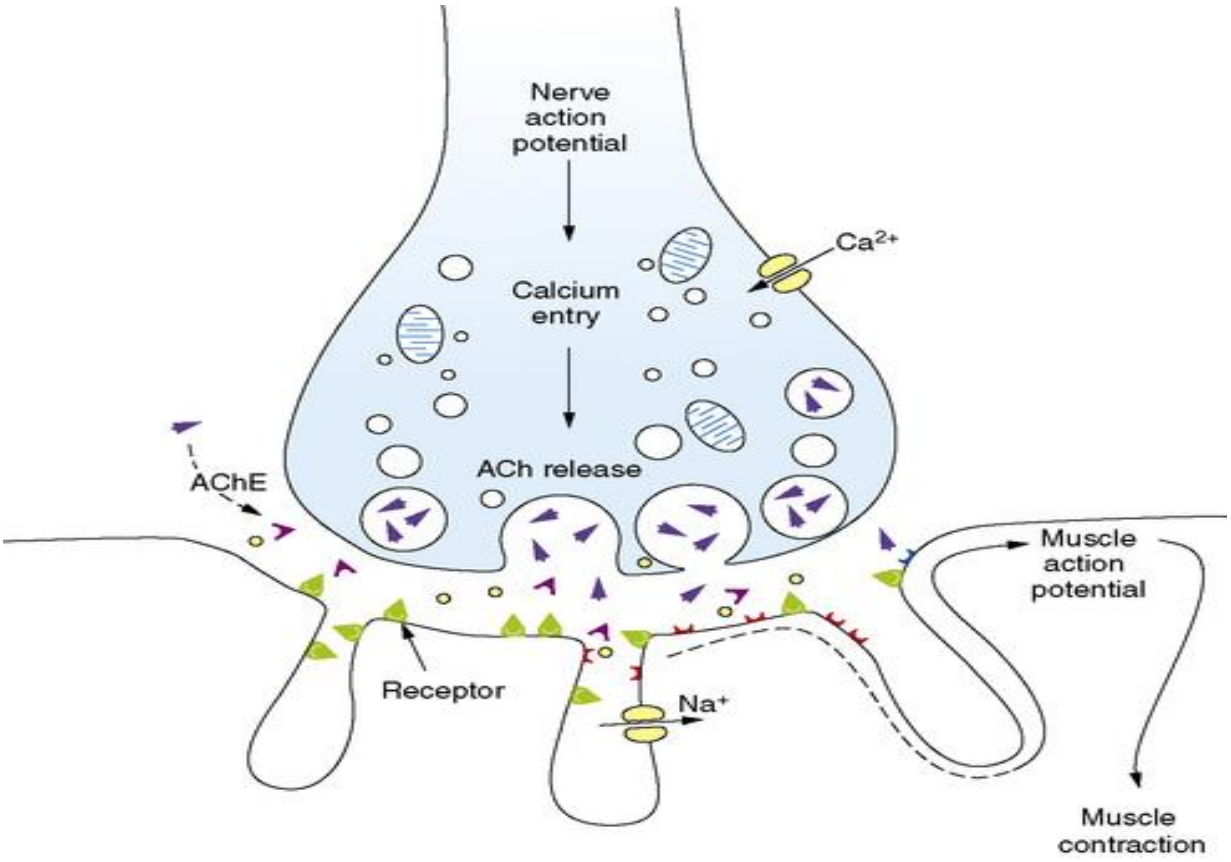
Characteristics of ideal volatile anaesthetic agent

- Ample potency.
- Pleasant smell.
- Low soluble in blood.
- Resistant to physical and metabolic degradation.
- No or negligible side-effects on vital organs.
- Non-toxic.
- Non-epileptogenic.
- Non-irritant to the respiratory tract.
- Neither circulatory stimulation nor inhibition.
- Non-inflammable.
- Low cost.

Halothane

- Thyme like odor.
- Highly potent.
- Highly soluble.
- Reduce blood pressure, myocardial depression and may cause arrhythmia.
- Depress respiration.
- Bronchodilator.
- Increase cerebral blood and ICP.
- May cause hepatitis on repeated exposure.

Neuromuscular junction NMJ



- ACh : Acetylcholine (Neurotransmitter)
- ACh receptor (Nicotinic receptor)
- AChE: Acetylcholine esterase (Degradation enzyme)
- NMB: Neuromuscular blocking agent
- Anti-AChE or Anti-ChE: Anticholinestrase (drug to inhibit AChE)
- AntiCholiergics: reduce ACh (autonomic) effects.

Neuromuscular Blocking Drugs

These work by interfering with the normal action of acetylcholine at the motor end-plate, blocking the receptors on the post-synaptic muscle membrane. Muscle relaxants are divided into two groups, the names of which are thought to reflect their mode of action.

Depolarizing neuromuscular blocking drugs

Suxamethonium

This is the only drug of this type in regular clinical use. After injection there is a short period of muscle fasciculation (visible light skeletal muscle contractions) due to depolarization of myocytes, followed by muscle paralysis in 40-60 s. Recovery occurs as a result of hydrolysis by plasma (pseudo-) cholinesterase, with restoration of normal neuromuscular transmission after 4-6 mins. This rapid onset makes it the drug of choice to facilitate tracheal intubation in patients likely to regurgitate and aspirate.

Systemic effects

- No direct effect on the cardiovascular, respiratory or CN systems. Bradycardia secondary to vagal stimulation is common after very large or repeated doses, necessitating pretreatment with atropine.
- A massive rise in serum potassium may provoke dysrhythmias in certain patients with:

1. Burns, maximal 2nd day to 3 months after burn.
2. Denervation injury (spinal cord injury), muscle dystrophies (Duchenne's) and rush injury.

- Increased intraocular pressure which may cause loss of vitreous in penetrating eye injury.
- Muscle pain most common 24 h after administration in young adults.
- Prolonged apnea in patients with pseudocholinestraes deficiency.

Non-depolarizing blocking drugs

These drugs compete with ACh and block its access to the post-synaptic receptor sites on the muscle but do not cause depolarization. They are sometimes referred to as *competitive neuromuscular blockers*. The time to maximum effect, that is when relaxation is adequate to allow tracheal intubation, is relatively slow compared with suxamethonium, generally 1.5-3 mins.

They are used in two ways:

- Following suxamethonium to maintain muscle relaxation during surgery.
- To facilitate tracheal intubation in non-urgent situation.

Duration of these drugs varies from short-acting up to 15 mins (Mivacurium), intermediate-acting up to 30 mins (Atracurium) and long-acting up to 45 mins (Pancuronium).

Although recovery of normal neuromuscular function eventually occurs spontaneously after the use of these drugs, it is often accelerated by the administration of an *anti-cholinestrase*.

Anticholinestrase

The action of all the neuromuscular blocking drugs wears off spontaneously with time, but this is not always clinically appropriate. In patients who require reversal of NMB drugs, an anticholinestrase is given. This inhibits the action of the enzyme acetylcholinesterase, resulting in an increase in the concentration of acetylcholine at the neuromuscular junction NMJ (nicotinic effect). The speed of recovery will depend upon the intensity when reversal is attempted- the more intense the block the slower the reversal.

Anticholinestrases also function at parasympathetic nerve endings (muscarinic effect), causing bradycardia, spasm of bowel, bladder and bronchi, increased bronchial secretions, etc. therefore they are always administered with a suitable dose of atropine or glycopyrrolate to block the unwanted muscarinic effects.

The most commonly used anticholinestrase is *Neostigmine*.