

Paediatric anaesthetic emergencies part I

Original Article by: Dr Claire Todd, South West School of Anaesthesia
Dr James Cockcroft, South West School of Anaesthesia
Dr Sarah Rawlinson, Derriford Hospital, Plymouth, UK

Introduction

- Laryngospasm
- Suxamethonium Apnoea
- Malignant Hyperthermia
- Anaphylaxis

Each of these presents a complex, potentially life-threatening anaesthetic emergency, which all anaesthetists (particularly paediatric) should be aware of and able to treat. Part one of this tutorial pair will deal with laryngospasm and suxamethonium apnoea.

Laryngospasm

Laryngospasm is a reflex closure of the upper airway caused by adduction of the vocal cords due to glottic muscular spasm. This narrowing of the laryngeal aperture results in partial or complete airway obstruction. If the spasm is sustained, it can result in morbidity such as hypoxia, gastric aspiration, arrhythmia, pulmonary oedema and cardiac arrest. The absolute incidence of laryngospasm is difficult to determine but it occurs more frequently in children compared to adults with figures commonly quoted around 1-2%. The incidence increases in the presence of an upper respiratory tract infection and when the anaesthetist is inexperienced in managing a paediatric airway. It can occur at any point during anaesthesia, but is most common during induction of anaesthesia, laryngoscopy & following tracheal extubation.

Risk factors for laryngospasm can be divided into three categories:

Anaesthetic factors:

- Inadequate depth of anaesthesia
- Irritant volatile agents
- Inexperienced anaesthetist

Local stimulation of the larynx by:

- Saliva
- Blood
- Vomitus
- Foreign bodies
- Instruments: including laryngoscope, laryngeal mask airways (LMAs) and suction catheters

External factors:

- Surgical stimulation
- Moving / transferring patient
- Anal / cervical stimulation (the Brewer-Luckhardt reflex)

Differential diagnosis

Bronchospasm

Inhaled foreign body

Laryngeal oedema or trauma

Recurrent laryngeal nerve damage

The clinical features can vary depending on the severity of the laryngospasm and the degree of airway obstruction. Often the initial clinical features are subtle but the clinical picture can evolve quickly, so when anaesthetising children a high index of suspicion is useful. Potential features include:

- Stridor: a harsh high pitched noise usually heard on inspiration
- Use of accessory muscles causing tracheal tug, intercostal and subcostal recession
- Paradoxical respiratory pattern
- Decreased tidal volumes
- Difficulty in ventilating patient through facemask or LMA
- Desaturation and cyanosis

- Bradycardia

Management

Early recognition and prompt treatment is key as oxygen desaturation occurs quickly in children. The aim is to maintain oxygenation until the laryngospasm resolves. It is important to call for help early as the clinical picture can progress rapidly from mild laryngospasm to complete airway obstruction and cyanosis.

- Switch to 100% oxygen via an anaesthetic breathing circuit
- Open the airway with a firm jaw thrust (this may break the laryngospasm due to a combination of airway opening and stimulation)
- Deliver Continuous Positive Airway Pressure (CPAP) if possible by closing the APL valve or partially occluding the reservoir bag opening on the Mapleson F circuit (T piece)
- Attempt gentle bag mask ventilation, ensuring that the stomach is not inflated in the process, as this will further obstruct ventilation and increase the risk of regurgitation
- Eliminate the cause if easily identifiable
- Ask surgeon to stop
- Deepen anaesthesia
- Remove blood/secretions from airway
- If laryngospasm fails to break with above methods give Suxamethonium (up to 2mg/kg IV). An alternative is propofol 0.5mg/kg IV. ¹
- Intubation of the trachea may be necessary
- Beware hypoxic bradycardia: this may resolve with re-oxygenation, however one should always have atropine (20mcg/kg) to hand

If laryngospasm occurs during inhalational induction without IV access, I.M. suxamethonium can be given (up to 4mg/kg) into a large muscle, e.g. upper arm/thigh. The intraosseous route has also been shown to be effective.

Once the laryngospasm has resolved, consider inserting a nasogastric tube to decompress the stomach (especially if prolonged CPAP was administered). Patients can develop negative pressure pulmonary oedema so they should be monitored closely (including oxygen saturation) in the post-operative period.

Suxamethonium apnoea

Suxamethonium is a depolarising muscle relaxant, which was introduced into clinical use in the 1950's. It consists of 2 acetylcholine molecules and binds to the post-synaptic acetylcholine receptor at the neuromuscular junction (NMJ). This produces rapid depolarisation and muscle relaxation. It is usually rapidly hydrolysed by plasma cholinesterase to inactive succinylcholine and choline, resulting in a short duration of action (3-5 minutes).

Its main use in clinical practice in the UK is during rapid sequence induction (RSI), due to its rapid onset time. It is also used as treatment for emergency airway problems, such as laryngospasm, especially in children.

Dose

Adult:

1 - 1.5mg/kg intravenously

Produces paralysis within 30 – 90 seconds

Paediatric:

1 - 2mg/kg intravenously

Can also be given intramuscularly (usually reserved for airway emergencies such as laryngospasm with no IV access) at a dose of 2 – 5mg/kg.²

Side effects

The side effects of suxamethonium include:

- Hyperkalaemia
- Myalgia
- Bradycardia
- Malignant hyperthermia
- Anaphylaxis
- Increase in intraocular pressure
- Prolonged neuromuscular blockade

Prolonged neuromuscular blockade (suxamethonium apnoea)

As outlined above, suxamethonium is hydrolysed in the NMJ by the enzyme plasma cholinesterase. If the activity of this enzyme is reduced then the duration of action of suxamethonium will increase. The activity of plasma cholinesterase can be reduced because of acquired conditions or genetic variability.

Causes of acquired plasma cholinesterase deficiency:

- Hepatic failure
- Pregnancy
- Renal failure
- Malignancy
- Burns
- Malnutrition
- Hypo-proteinaemia

- Drugs, including ketamine, oral contraceptive pill, lithium, some cytotoxic drugs

These acquired causes have a variety of different underlying mechanisms. These include reduced enzyme production, altered enzyme binding in the blood and altered enzyme activity.

Genetic variability

Single amino acid substitutions in the genetic coding for plasma cholinesterase can result in altered activity of the enzyme. There are four alleles (different forms of the gene) for plasma cholinesterase:

- Usual (normal)
- Atypical
- Silent
- Fluoride-resistant

These four alleles make up 10 different genotypes (as each individual inherits two alleles, one from each parent). These different genetic combinations result in the following phenotypes (how the genetic make-up is expressed clinically):

- **Normal Enzyme**
95% of the population are homozygous for the normal gene and metabolise suxamethonium rapidly.
- **Heterozygous** for one of the abnormal genes
Approximately 4% of population possess one usual (normal) gene and one of the three abnormal genes. These patients clinically have a prolonged block to suxamethonium, which typically lasts for 10 – 20 minutes.
- **Homozygous** for 2 abnormal genes
These patients have no copies of the usual (normal) gene in their genetic makeup. These make up a very small proportion of the population (<0.01%) and clinically they have a prolonged block after the administration of suxamethonium, which lasts several hours until excreted by the kidney.

Presentation

Patients tend to present with an absence of muscle function after the effects of anaesthesia have worn off. Commonly this is as an inability to breathe, at the end of a procedure, which involved the administration of suxamethonium.

In heterozygotes for one abnormal gene, the suxamethonium apnoea effect is likely to wear off before the effects of any non-depolarising relaxant, therefore having no detrimental effects to the patient.

With homozygotes to 2 abnormal genes, the effect of suxamethonium apnoea may well last well beyond the effects of any non-depolarising relaxant used.

The potential for any patient to suffer from a prolonged neuromuscular block is used as an argument for ensuring the actions of suxamethonium have worn off before administering a longer acting muscle relaxant. This can be detected clinically or by the use of a peripheral nerve stimulator. If this is not done, there is the potential for unintentional awareness to occur at the end of the procedure when the anaesthetic agent is withdrawn but normal muscle function has not yet returned.

Management

Management is primarily supportive in nature and should consist of the following steps:

- ABC
- Maintain a patent airway and ensure adequate ventilation
- If prolonged block becomes apparent during failed intubation follow failed intubation algorithm ³
- Once airway secure maintain anaesthesia with volatile or IV agent
- Monitor neuromuscular function with peripheral nerve stimulator
- Consider transfer to ICU for continued sedation and ventilation
- Consider the administration of fresh frozen plasma (if available) to provide a source of plasma cholinesterase
- After recovery and if available, refer for investigation.
- Explanation to child and parents / guardians. Consider the need for counselling if the patient was aware.

Investigation

If a child has had an episode of suxamethonium apnoea then this must be explained to the family, and they should be advised that both the patient and their direct family should have a blood test to measure the level of plasma cholinesterases to confirm whether they have the inherited form or the acquired. The test involves adding dibucaine, an amide local anaesthetic that inhibits normal plasma cholinesterase. Its inhibition of the abnormal forms of the enzyme is less effective. The percentage inhibition of the plasma cholinesterase is measured and expressed as the 'Dibucaine number'.

The normal enzyme has a dibucaine number of 80 (i.e. 80% of the enzyme activity is inhibited). Heterozygotes for the abnormal plasma cholinesterase gene will have a dibucaine number of 45-65 and homozygotes a number less than 30.

References:

1. Alalami AA, Ayoub CM, Baraka AS. Laryngospasm: review of different prevention and treatment modalities. *Paediatric Anaesthesia* 2008; **18**: 281-288
2. British National Formulary (BNF). Access online, <http://www.bnf.org>
3. Difficult Airway Society (DAS) guidelines. Access online <http://www.das.uk.com/guidelines/downloads.html>

Original article found at: <http://www.wfsahq.org/resources/anaesthesia-tutorial-of-the-week>

Copyright remains with original authors