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Paediatric airway emegencies

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Southern African Journal of Anaesthesia and Analgesia. 2021;27(6 Suppl 1):S1-55
https://doi.org/10.36303/SAJAA.2021.27.6.S1.2697
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South Afr J Anaesth Analg
ISSN 2220-1181 EISSN 2220-1173
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FCA REFRESHER COURSE

Paediatric airway emergencies

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Keywords: paediatric airway emergencies, perioperative complications, airway management

Perioperative complications of airway management are more common in children than adults, which may result in critical events including cardiac arrest.[1-3]

Paediatric airway emergencies cover a vast array of topics. These include:

1. The difficult airway society guidelines for paediatrics.
   Please refer to the guidelines online at:
   https://das.uk.com/guidelines/paediatric-difficult-airway-guidelines
2. Airway-related complications:
   a. Hypoxia
   b. Laryngospasm
      Refer to S74 of the FCA 2 supplement of 2017 – Laryngospasm in anaesthesia by Dr S Spijkerman.
      https://www.sajaa.co.za/index.php/sajaa/issue/view/124
   c. Bronchospasm (and anaphylaxis)
   d. Aspiration
   e. Postintubation croup
3. Paediatric airway infections
4. Foreign body aspiration
   Refer to S42 of the FCA 2 supplement of 2017 – Anaesthetic considerations for inhaled and ingested foreign bodies in children: What? Where? When? by Dr AZ Bhettay.
   https://www.sajaa.co.za/index.php/sajaa/issue/view/124

We will mainly focus on airway-related complications in these notes.

Please refer to two excellent reviews covered in the 2017 FCA 2 supplement as referred to above which has provided comprehensive reviews of Laryngospasm and Foreign Body Aspiration in Children.

It is stated that "Anaesthesiologists who perform fewer than 100 paediatric cases on infants and young children per year are five times more likely to experience complications compared with anaesthesiologists who do more than 200 cases per year".[4] Paediatric respiratory events carry a higher rate of mortality than in adults.[5]

Incidence of airway-related complications

Respiratory and airway events account for the most common perioperative complications in paediatric patients.[1] The Paediatric Perioperative Cardiac Arrest (POCA) registry and the American Society of Anesthesiologists (ASA) closed claim analysis found that cardiac arrest and brain death in paediatric patients are still mainly caused by airway problems despite improvements in monitoring and anaesthetic drugs.[4] Twenty-seven percent of all paediatric perioperative cardiac arrests were initiated by a respiratory event. Laryngospasm was the most common cause. Other aetiologies included difficult intubation, oesophageal intubation, airway obstruction and aspiration.[5,6] In the Anaesthesia Practice In Children Observational Trial (APRICOT Study) 60% of all anaesthesia-related complications were respiratory and airway events.[7] In all the anaesthetics laryngospasm occurred in 1.2%, bronchospasm in 1.2%, postoperative stridor in 0.7% and aspiration in 0.1%. The highest rate of events occurred in neonates and infants. Other risk factors included prematurity and reactive airways.[7]

A. Hypoxaemia

Paediatric patients are particularly prone to rapid oxygen desaturation due to little respiratory reserve. The infant or toddler has a higher respiratory rate and heart rate to compensate for smaller tidal volumes, larger dead space, higher oxygen consumption and higher carbon dioxide production. The paediatric chest wall is mechanically less efficient and limits potential lung expansion.[1,4] Infants also have a high vagal tone which predisposes them to experience bradycardia and cardiac arrest in response to hypoxaemia especially when combined with stimulation of the airway.[1]

B. Laryngospasm

(I will only be doing a summary of laryngospasm as Dr S Spijkerman did a detailed review of this topic in the FCA 2 supplement of 2017 (S74) – Laryngospasm in Anaesthesia by Dr S Spijkerman. https://www.sajaa.co.za/index.php/sajaa/issue/view/124)

Laryngospasm is mainly seen in children and is a life-threatening complication during the perioperative period. Incidence ranges

from 1.7–25%.[1] Laryngospasm is the reflex closure of the false and true vocal cords, accompanied by the descent of the epiglottis over the laryngeal orifice. Laryngospasm occurs during induction, maintenance, or emergence from anaesthesia. It must be recognised and treated rapidly to prevent complications. Most of the time laryngospasm responds to treatment with no sequelae but oxygen desaturation (61%), bradycardia (6%), negative pressure pulmonary oedema (4%), aspiration (3%) and cardiac arrest (0.5%) can occur.[1,8] It may progress from inspiratory stridor, retractions, and rocky chest wall movements with inspiration (partial laryngospasm) to complete cessation of air movement (complete laryngospasm).

There are four mechanisms by which closure of the larynx occurs:[4]

i. Closure of the vocal cords, both by pulling them together and by tensing
ii. Closure of the false vocal cords
iii. Mounding of the paraglottic tissues by elevation of the larynx
iv. Folding of the epiglottis over the glottic opening

Risk factors for laryngospasm can be assessed by three categories related to patient, anaesthesia and surgical factors as discussed in Table I.[3]

Table I: Risk factors for laryngospasm[3]

Patient-related factors	Anaesthetic-related factors	Surgical-related factors
Age: • Inverse correlation with age: younger children at greater risk	Insufficient depth of anaesthesia: • Induction/maintenance – Face mask/ supraglottic airway (SGA) Emergence especially post- tracheal extubation	Shared airway surgery: • Tonsillectomy and adenoidectomy – greatest risk
Airway Hyperactivity: • Asthma – 10 x > risk with active asthma • URTI – 10 x risk for up to 6 weeks, delay elective surgery for at least 2 weeks, SGA < ETT	Airway irritation: • Volatile anaesthetics • Mucous • Blood • Manipulation/instrumentation	Thyroid surgery: • Due to superior laryngeal nerve injury/ • Hypocalcaemia secondary to parathyroid gland excision
Tobacco smoke: • Chronic use – abstain for at least 2 days to reduce risk • Passive exposure – 10 x increased risk in children	Airway device: • SGA > Endotracheal tube (ETT)	Oesophageal surgery: • Due to stimulation of distal afferent oesophageal nerves
Obesity with OSA: • Gastro-oesophageal reflux • Airway anomaly	IV induction agents: • Thiopentone doesn't suppress airway reflexes	Others: • Appendicectomy • Cervical dilatation • Hypospadias repair • Skin grafting • Mediastinoscopy/bronchoscopy/GI endoscopy
	Volatile anaesthetic agents: Desflurane > isoflurane > enflurane > Halothane/sevoflurane	Moving or transferring a patient
	Experience of anaesthetist – more likely with inexperience	

Table II: Prevention of laryngospasm

Identify the patients at risk and take recommended precautions

↓

Postpone surgery (2–3 weeks) after an URTI if surgery is not urgent

Induction phase	Maintenance phase	Emergence
• Experienced paediatric anaesthetist	• Provide an adequate depth of anaesthesia and analgesia	• Suction nasal and oropharyngeal secretions/ blood
• Avoid manipulation of the airway at an insufficient depth of anaesthesia		• Check for residual paralysis
• Choose a non-invasive airway device where possible		• Limited stimulus until the patient opens the eyes spontaneously
• Choose a non-irritant volatile agent (sevoflurane) with inhalational inductions and an intravenous induction agent with smooth properties (propofol) and muscle relaxation if tracheal intubation is necessary		• Extubate patients either in a deep plane of anaesthesia "No Touch" * technique or fully awake, using the "artificial cough technique"** (not in-between)

* The 'No Touch' technique is essentially an awake tracheal extubation.[3,8] This technique consists of suctioning of the blood and secretions from the pharynx, turning the patient to the lateral position while anaesthetised, discontinuing the volatile anaesthetics and avoiding any stimulation until the patients open their eyes and spontaneously wake up to be followed by tracheal extubation.[3,8]
** Tracheal extubation: it has been suggested that the tracheal tube should be removed while the lungs are inflated by positive pressure; this technique decreases the adductor response of the laryngeal muscles and reduces the incidence of laryngospasm.[3,8] Positive pressure inflation of the lungs before tracheal extubation is followed by forced exhalation 'artificial cough' after extubation which expels any secretions or blood and this in turn decreases vocal cord irritation and laryngospasm.[3,8]

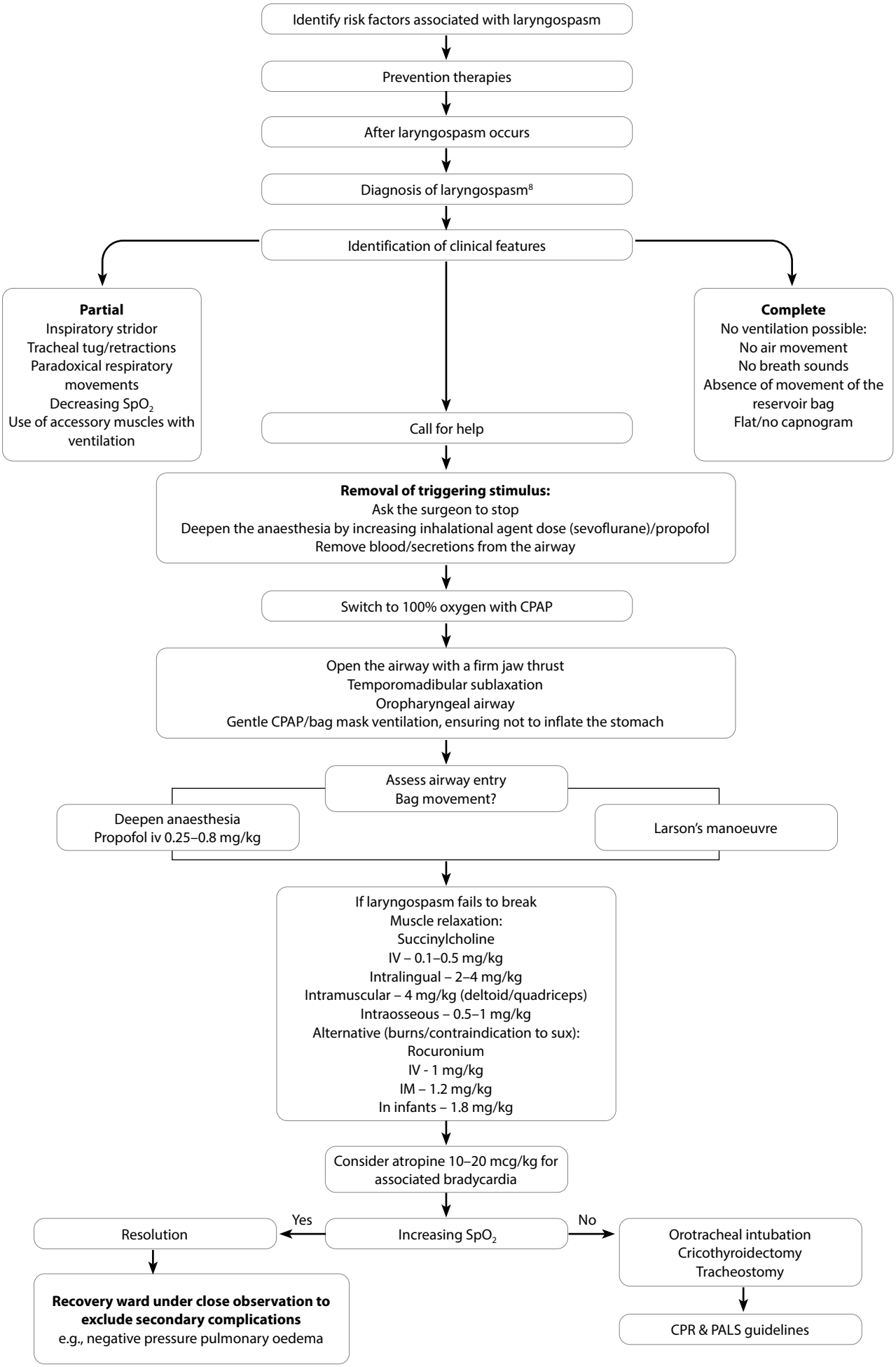

Figure 1: The management of intraoperative laryngospasm in children[1,2,9,10] (Adapted from Update in Anaesthesia WFSA-D-18-00036)

Prevention of Laryngospasm in discussed in Table II and has been based on the different phases during anaesthesia.[3,9]

Many other pharmaceutical agents (magnesium sulphate, lignocaine, atropine, and benzodiazepines) have been studied but more studies are necessary, and their use remains controversial.

The management of intraoperative laryngospasm in children is depicted in the flow diagram of Figure 1.[1,2,9,10]

Early recognition and prompt treatment are key as desaturation and potential complications occur quickly in children.[2]

C. Intraoperative bronchospasm

Bronchospasm is a relatively common event during general anaesthesia. It can present in isolation (stimulation from an airway device) or as a part of a more serious underlying pathology such as anaphylaxis, aspiration of gastric contents or underlying reactive airway disease.[1,11]

Bronchospasm is a reversible reflex constriction of the smooth muscle lining the bronchioles.[12] It is a common feature of reactive airways disease.

The APRICOT study reported the incidence of intraoperative bronchospasm to be around 0.3–3.2% in children and 96% of cases occurred in theatre.[1] The incidence is highest is asthmatic patients (6%) but life-threatening bronchospasm can still occur in healthy patients without any underlying lung pathology.[12]

Pathophysiology[12,13]

Intraoperative bronchospasm is a reflex that is mediated via the vagus nerve.[12] A noxious stimulus, such as endotracheal intubation, activates afferent sensory fibres in the vagus nerve that stimulate neurons within the nucleus of the solitary tract.[12] These neurons then stimulate efferent fibres through the vagus nerve to bronchiolar smooth muscle. Released acetylcholine neurotransmitters then bind to the M3 muscarinic receptor, resulting in an increase in cyclic guanosine monophosphate and inducing bronchiolar smooth muscle contraction.[12] Other mediators that may participate in this reflex include histamine, tachykinins, vasoactive intestinal peptide, and calcitonin gene-related peptide.[12]

Most causes of perioperative bronchospasm involve a nonallergic mechanism.[11,12]

Clinical signs of bronchospasm under anaesthesia:[1]

- Auscultation – wheezing
- Inspection – slow expiration
- Ventilation – decreased tidal volume and high inspiratory pressure
- Monitoring:
  - Oxygen saturation – decreased
  - Capnography[14] – upsloping (delayed rise) of end-tidal carbon dioxide ($EtCO_2$) (due to prolonged expiration) producing a characteristic "shark-fin" appearance and in severe cases a decreased value or absent $EtCO_2$ waveform (Figures 2 and 3).

Essentially transient increases in airway resistance lead to an obstruction of both expiratory and inspiratory airflow.[12]

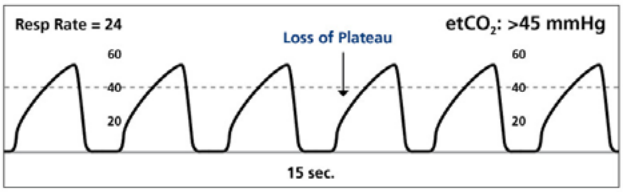

Figure 2: "Shark fin" appearance[14]

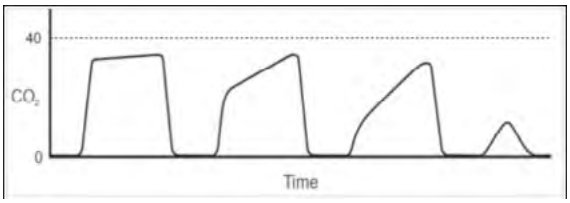

Figure 3: Bronchospasm[15]

Table III: Differential diagnosis of bronchospasm

Causes of wheeze during general anaesthesia[11]	Causes of increased peak airway pressures during IPPV[11]	
Problems with the endotracheal tube: • Malpositioned (endobronchial, oesophageal, abutting the carina) • Obstructed (mucous plug, foreign body, cuff herniation) • Kinked	Anaesthetic equipment	• Excessive tidal volume • High inspiratory flow rates
Bronchospasm	Airway device	• Small diameter ETT • Endobronchial intubation • ETT kinked/blocked
Pulmonary oedema		
Aspiration of gastric contents		
Pulmonary embolism	Patient	• Obesity • Head down position • Pneumoperitoneum • Tension pneumothorax • Bronchospasm
Anaphylaxis		
Tension pneumothorax		
Foreign body in the tracheobronchial tree		
Laryngospasm		
Obstruction in the breathing circuit		

Table IV: Prevention of bronchospasm

Comorbidity	Signs of control	Pre-op considerations
Airway disease (Asthma/COPD): Assess for degree of medical optimisation and disease control	Exclude any signs of poor control: • Active wheezing • Cough • Increased sputum production • Shortness of breath • Diurnal variability in peak expiratory flow rate (PEFR) • Recent/frequent exacerbations or admission to hospital	• Continue medication until time of surgery • Careful medication history for NSAID-induced bronchospasm (15%)
Smoking		Stop smoking preoperatively ideally 6–8 weeks to reduce the risk of respiratory complications
Upper respiratory tract infection		Postpone surgery for at least 2 weeks for complete resolution of symptoms

Bronchospasm is not the only cause for wheeze/increased peak airway pressures during anaesthesia.[11]

Differential diagnosis

Bronchospasm occurs commonly during induction (airway irritation usually intubation) and maintenance (anaphylactic or serious allergic reaction) of anaesthesia and less often during emergence and the recovery stages.[11,12] The differential diagnosis of bronchospasm is discussed in Table III.

Bronchial hyperactivity[13]

1. Airway irritation in patients known to be at higher risk of bronchial hyperreactivity, such as those with poorly controlled reactive airway disease (asthma and COPD)
2. An upper respiratory tract infection, and
3. History of smoking
4. History of atopy

Pharmacological

There are pharmacological causes of bronchospasm: desflurane, β-blockers, NSAIDs, cholinesterase inhibitors (neostigmine), and histamine-releasing drugs (atracurium, mivacurium, sodium thiopental, morphine).[11,12] Use these drugs with caution in at-risk patients.

Airway soiling

Unexplained bronchospasm that occurs after induction in a patient without risk factors for airway hyperreactivity may be the result of pulmonary aspiration of gastric contents. Aspiration may involve active vomiting or passive regurgitation. In addition to the classic signs of bronchospasm, the patient who aspirated typically develops hypoxaemia. Aspiration can occur in a patient receiving general anaesthesia with a face mask, laryngeal mask, and endotracheal tube.[11,12]

Anaphylaxis

At any stage of anaesthesia, bronchospasm may be one of several manifestations of a serious allergic reaction or anaphylactic shock.[13]

Bronchospasm can represent either an anaphylactoid reaction or Ig E-mediated anaphylaxis.[13]

The most common allergens responsible are:

- muscle relaxants (rocuronium, succinylcholine)
- antibiotics (penicillins, cephalosporins)
- latex, and
- blood products (red blood cells, fresh frozen plasma)

In addition to the usual presentation of bronchospasm, anaphylaxis typically includes cutaneous signs such as an urticarial rash and angioedema as well as severe haemodynamic aberrations (tachycardia, hypotension, circulatory collapse).[11,12]

Inadequate depth of anaesthesia – stimulates the bronchospasm reflex.[12]

Therefore, surgical stimulation or mechanical manipulation of the airway (especially endotracheal intubation), in conjunction with an inadequate depth of anaesthesia, significantly increases the chance of bronchospasm.[11]

An approach to the prevention of bronchospasm is shown in Table IV.

Measures to lower the risk of precipitating intraoperative bronchospasm include:[11,12]

- Administration of preoperative inhaled bronchodilators (β2 adrenergic agonists) and steroids (inhaled and IV) about 30 minutes prior to surgery.
- Use of regional techniques where appropriate can avoid the need for general anaesthesia and intubation.
- Ensure adequate depth of anaesthesia before airway instrumentation.
- Consider the use of a laryngeal mask airway rather than endotracheal intubation.
- Consider the use of ketamine.
- Avoid drugs that cause histamine release.
- Consider topical lignocaine to the airway.
- Consider deep extubation.

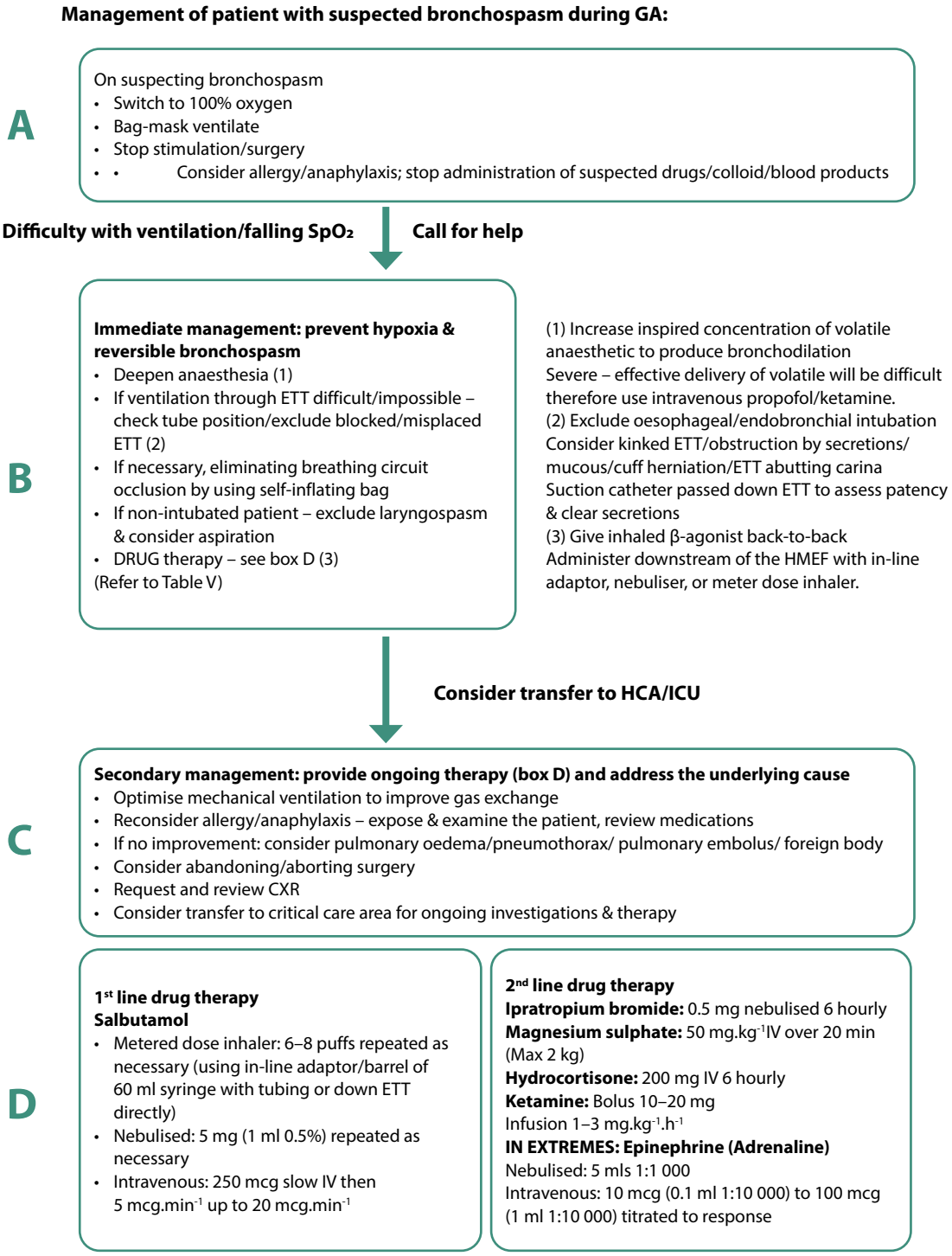

Figure 4: Adapted from Update in Anaesthesia 2011: Management of bronchospasm during general anaesthesia by Alex Looseley[11]

Management of a patient with suspected bronchospasm during general anaesthesia (GA) is depicted in Figure 4.[11]

https://e-safe-anaesthesia.org/e_library/05/Bronchospasm_during_anaesthesia_Update_2011.pdf

Aims:

- To restore adequate ventilation and oxygenation.
- Treat rapidly while simultaneously identifying the underlying cause.
- Reverse airflow obstruction and hypoxaemia before irreversible ischaemia ensues.

Table V shows the drugs used in the treatment of bronchospasm.[11]

Mechanical ventilation

The primary aim of mechanical ventilation in acute bronchospasm is to prevent or correct hypoxaemia.[11]

- Tidal volumes – reduce to avoid high peak airway pressures and barotrauma.
- Permissive hypercapnia is tolerated if oxygenation is adequate – as long as severe acidosis does not develop (pH < 7.15).
- Long expiratory time to allow complete exhalation and reduce 'breath-stacking' and intrinsic PEEP.

Table V: Drug doses for use in Bronchospasm

Drug	Paediatric dose
Salbutamol	MDI 6–8 puffs Nebulised < 5 yrs 2.5 mg, > 5 yrs 2.5–5 mg IV – 4 mcg.kg^{-1} slow IV then 0.1–1 mcg.kg^{-1}.min^{-1}
Epinephrine (Adrenaline)	IV – 0.1–1.0 mcg.kg^{-1} (0.01–0.1 ml.kg^{-1} of 1:100 000) IM - < 6 months 50 mcg, 6 months - 6 years 120 mcg, 6–12 years 250 mcg, > 12 years 500 mcg Nebulised 0.5 ml.kg^{-1} 1:1 000 (max 5 mls)
Ipratropium bromide	Nebulised (2–12 years) 0.25 mg 6 hourly
Magnesium sulphate	50 mg.kg^{-1} IV over 20 min (max 2 g, unlicensed)
Ketamine	Infusion 1–3 mg.kg^{-1}.h^{-1}
Aminophylline	5 mg.kg^{-1} IV over 20 min then 1 mg.kg^{-1}.h^{-1} (< 9 years), 0.8 mg.kg^{-1}.h^{-1} (9–16 years) infusion Omit loading dose if taking theophylline
Hydrocortisone	< 1 years 25 mg, 1–5 years 50 mg, 6–12 years 100 mg 6 hourly
Chlorphenamine	< 6 months 250 mcg.kg^{-1} IV, 6 months – 6 years 2.5 mg IV, 6–12 years 5 mg IV

Adapted from Update in Anaesthesia 2011: Management of bronchospasm during general anaesthesia by Alex Looseley[11]

- Intrinsic PEEP can increase intra-thoracic pressure, decrease venous return and cause hypotension.
- Minimising intrinsic PEEP is best achieved with a slow respiratory rate, an inspiratory: expiratory ratio of at least 1:2 to 1:3 (will help prolong the expiratory rate).
- If bronchospasm is severe, only 3–4 breaths per minute may be possible if you allow full expiration.

Complications of bronchospasm

1. Respiratory deterioration[1]
   a. Tension pneumothorax – with IPPV
   b. Atelectasis – due to mucous plugging
2. Cardiovascular deterioration[1]
   a. Reduced venous return – due to high airway pressures
   b. Anaphylaxis – hypotension
   c. Adrenal insufficiency – insidious hypotension secondary to glucocorticoids

Emergence and extubation

Bronchospasm can occur on emergence from anaesthesia when ETT is insitu and when the level of anaesthesia is reduced.[1,11] The aim is for a smooth controlled emergence. Whereas deep removal of airway devices may result in a smoother emergence than awake removal, the risk of perioperative respiratory adverse events (PRAEs) may be similar.[16] In a single-centre randomised controlled trial that compared awake versus deep extubation of the trachea in 100 children with increased airway susceptibility undergoing adenotonsillectomy, the rate of PRAEs was similar in the two groups.[16] Children who were extubated awake showed a tendency toward more and longer episodes of very brief (< 10 seconds) desaturations to < 95 per cent, but overall, there was no evidence for an increased risk of oxygen desaturation following either technique. There was also no difference in the occurrence of laryngospasm or bronchospasm between children who were extubated awake as compared with those who were extubated while still at a surgical level of anaesthesia.[1,16] Patients with a complicated intraoperative course may benefit from continued postoperative intubation and ventilation.[1,11]

D. Aspiration

The incidence of aspiration of gastric contents ranges from 0.02–0.1% of all anaesthetics in children making this a rare complication of paediatric airway management.[1]

Fasting guidelines are usually created on an institutional basis, and often represent multidisciplinary consensus among surgery,

Table VI: PACSA fasting guidelines[17]

• Pre-operative fasting for elective procedures in children 0–16y of age		
Solid food, formula milk	Breast milk	Clear fluids
6h	4h	1h
• Clear fluids are defined as water, clear (nonopaque) fruit juice or squash/cordial, ready diluted drinks, and non-fizzy sports drinks. Non thickened, non-carbonated		
• Contra-indications to be considered by the anaesthetist and surgical team include: Gastro-oesophageal reflux (GORD) (either on treatment or under investigation), renal failure, severe cerebral palsy, some enteropathies, oesophageal strictures, achalasia, diabetes mellitus with gastroparesis, and/or surgical contra-indications		

Table VII: Fasting guidelines of international anaesthesia societies[1]

Country, year	Fasting requirements at time of induction	Comments
American Society of Anaesthesiologists, 2017[18]	• 2 hours clear liquids, not including alcohol • 4 hours breast milk • 6 hours nonhuman milk, infant formula, light meal • 8 hours or more fried or fatty food or meat	• Healthy patients, elective surgery, pregnant patients not in labor • Light meal defined as toast or cereal with clear liquid
European Society of Anaesthesiology, 2011[17,19]	• 2 hours clear liquids • 1 hour clear liquids for children • 4 hours breast milk • 6 hours, infant formula, solid food • Chewing gum and sucking hard candy allowed up until time of induction	• Applies to patients with obesity, diabetes, GORD, nonlaboring pregnant patients • Encourages oral fluid up to 2 hours prior to induction
Canadian Anaesthesiologists' Society, 2014[20,21]	• 2 hours clear liquids • 1 hour clear liquids for children • 4 hours breast milk • 6 hours light meal, infant formula, and nonhuman milk • 8 hours meat, fried, or fatty food	
Association of Anaesthetists in Great Britain and Ireland, 2010[17,22]	• 2 hours clear liquids • 1 hour clear liquids for children • 4 hours breast milk • 6 hours solid food, infant formula, and cow's milk	• Gum chewing should be treated as clear
Scandinavian Society of Anaesthesiology and Intensive Care Medicine, 2005[23]	• 2 hours clear liquids • 4 hours breast milk and infant formula • 6 hours solid food and cow's milk • 2 hours chewing gum and any form of tobacco • Up to 1 hour prior to induction, 150 ml water	• 2 hours for preoperative carbohydrate drinks intended for preoperative nutrition
German Society of Anaesthesiology and Intensive care, 2004[24]	• 2 hours clear liquids • 4 hours breast milk and infant formula • 6 hours meal	
Australian and New Zealand College of Anaesthetists, 2016[25]	• 2 hours clear liquids, all ages • 3 hours breast milk for infants < 6 months of age • 4 hours formula for infants < 6 months of age • 6 hours breast milk, formula, limited solid food for children > 6 months of age and adults	• Encourages oral fluid up to 2 hours prior to induction • Up to 200 ml clear liquids per hour up until two hours prior to induction for adults

anaesthesia, endoscopy, radiology, and nursing services. Our PACSA guidelines as shown in Table VI[17] are as follows:

Preoperative fasting guidelines or consensus statements have been developed by anaesthesia societies and organisations around the world (Table VII).[1] All rely on the physiology of gastric emptying and expert opinion, without evidence, that following recommendations improves clinical outcomes.[1]

Increasingly, recommended fasting intervals for liquids are being reduced, with some institutions allowing clear liquids almost immediately prior to surgery.[26]

Regardless of institution-specific guidelines, every effort should be made to avoid prolonged unplanned fasting because of schedule delays, especially in very young children, and clear fluid intake should be encouraged, within the bounds of the established guidelines.[1,27] Fasting and the management of anaesthesia for children at high risk of aspiration and/or abnormal stomach emptying should be individualised.

Risk factors for aspiration

Risk factors for aspiration[7,28-30] in children include:

- American Society of Anesthesiologists (ASA) physical status of III or IV.
- Emergency procedures.
- Abnormal stomach emptying: children with diabetes, gastro-oesophageal reflux, bowel or gastric outlet obstruction, trauma.

While most studies find the highest rates of aspiration at induction and laryngoscopy, up to one-half of these events may occur during maintenance of anaesthesia or at extubation.[7,28] Light anaesthesia and high intra-abdominal pressure (e.g. due to lithotomy positioning) are additional risk factors for aspiration during maintenance with a SGA in place.[28]

Aspiration events during maintenance of anaesthesia are more common in patients managed with an SGA, mask anaesthetic, or natural airway rather than an ETT.

Prevention[26,27]

- Strict adherence to fasting guidelines and recognition of patients with delayed gastric emptying or with a full stomach.
- Patients with intestinal obstruction require a naso-gastric tube.
- Choice of appropriate induction technique and airway device.
- Controlled Rapid Sequence Induction and Intubation requires rapid induction of anaesthesia, deep muscle relaxation and gentle face mask ventilation to maintain oxygenation, ventilation, and anaesthesia.

- Ensure sufficient depth of anaesthesia (+/- muscle relaxation) to avoid coughing, bucking, straining resulting in regurgitation of gastric content and pulmonary aspiration.

Treatment of aspiration

Subsequent management depends on the severity of signs and symptoms, the timing of the aspiration, the perceived cause of aspiration, and the urgency of the procedure. If an aspiration event is suspected, immediate management should include the following steps:[1,28,31]

- Call for assistance and help.
- Place patient in lateral and head down position.
- Suction clearance of airway content, administer 100% oxygen by face mask and secure airway with tracheal intubation. Remove a SGA, if used, as it can trap gastric contents at the glottic opening.
- Evaluate for laryngospasm and bronchospasm and treat as necessary.
- No broncho-alveolar lavage.
- Solid foreign body aspiration may require bronchoscopy and extraction.
- Lung recruitment manoeuvre if required.
- Postpone non-emergency surgery if severe aspiration suspected.
- Chest x-ray, steroids or prophylactic antibiotics are not routinely required unless symptoms persist.
- Attempt early extubation.

Post-op care

Take home message – hospital discharge is possible if asymptomatic after two hours.[28,30,31]

Morbidity from pulmonary aspiration varies widely, ranging from no observable sequelae to severe hypoxaemia and acute respiratory distress syndrome.[1] Based on large prospective observational studies of anaesthesia-related aspiration in paediatric patients, pulmonary sequelae occur in one-third to three-fifths of patients who aspirate.[28,30] If pulmonary sequelae occur, they do so within two hours of aspiration. Therefore, patients may be discharged from the hospital two hours after suspected aspiration if they have no new pulmonary symptoms (e.g. cough or wheeze) and have normal oxygen saturation on room air.[28,30]

Children with mild symptoms who maintain oxygen saturation (SpO_2) > 90% with low-level oxygen supplementation via nasal cannula can be observed on a patient ward. Children who require mechanical ventilation or high fraction of inspired oxygen should be admitted to the intensive care unit. Recovery in severe cases may take days to weeks, though death due to aspiration in otherwise healthy children is extremely rare.[29,30]

E. Postintubation croup

Postintubation croup may occur in the post-anaesthesia care unit (PACU) in recently extubated children. The overall incidence is around 1%, occurring more commonly in children aged between one and four years.[32] This occurs due to local oedema and inflammation caused by pressure of the ETT on laryngeal or subglottic structures. Even a small amount of oedema can cause a significant increase in airway resistance because of the small internal diameter of the trachea in young children. Clinical signs usually present within 30 minutes of extubation.[33] Symptoms may include barking cough, inspiratory stridor, suprasternal or subcostal retractions, respiratory distress, and cyanosis.

Risk factors for postintubation croup

The risk factors (Table VIII) included are those related to the patient, airway management (usually related to factors that increase trauma to the larynx), and the procedure.[32,34,35]

Prevention of postintubation croup

To minimise the need for reintubation for an incorrectly sized tube, use an appropriately sized cuffed ETT in all children.[1,32,33]

The goal for all children should be to achieve a smooth, controlled emergence from anaesthesia without laryngospasm or bronchospasm, oxygen desaturation, coughing, or vomiting.[1,32]

For children who undergo oropharyngeal or neck surgery or have other risk factors for postintubation croup (multiple intubation attempts, absence of an airway leak), dexamethasone 0.5 to 0.6 mg/kg intravenous (IV) is routinely administered intra-operatively. Dexamethasone may also decrease postoperative nausea and vomiting and pain.[1,32,36]

Treatment of postintubation croup

Generally there is a good response to treatment with IV dexamethasone and nebulised epinephrine.[33] This is primarily based on clinical experience and the treatment of infectious croup.

- Dexamethasone: 0.6 mg/kg IV to a maximum 10 mg, if not given intraoperatively.[36] A dose as low as 0.15 mg/kg has also been shown to be just as effective.[37]
- Nebulised epinephrine administered over 15 minutes, as follows:[38]
  - Racemic epinephrine 0.05 mL/kg per dose (maximum of 0.5 ml) of a 2.25% solution diluted to 3 ml total volume with normal saline, OR
  - L-epinephrine 0.5 ml/kg per dose (maximum of 5 ml) of a 1:1 000 dilution.

Racemic and L-epinephrine are equally effective. The treatment may be repeated every 15 to 20 minutes if necessary.[38]

Outcomes after postintubation croup

Typically the effects of racemic epinephrine last for two hours, after which symptoms may recur.[1,33,38] Children who receive

Table VIII: Risk factors for postintubation croup

Patient	Airway management	Procedure
Age: 1–4 years old	Traumatic/repeated intubation attempts	Non-supine position
	Oversized ETT	Excessive movement of the tube (e.g. coughing with the tube in place, moving the patient's head)
	Coughing with an ETT in place	Prolonged surgery
	Lack of an airway leak with > 25 cm H_2O airway pressure	Head and neck surgery
	Intubation longer/> one hour	

epinephrine should be observed in the hospital for at least three to four hours after administration for the "rebound effect" and recurrence of symptoms.[1]

- Children who at 3–4 hours are breathing comfortably, without stridor, and have normal oxygen saturation (SpO_2) on room air may be discharged home with instructions to return to the emergency department if symptoms recur.[39,40]
- Children who have continual airway obstruction signs or if repeated doses of epinephrine are required, should be admitted to the hospital or intensive care unit, as indicated, for further monitoring and treatment.[1,33]

Conclusion

Paediatric patients commonly present with airway problems which can result in serious morbidity and mortality. A detailed history especially of any airway issues and proper risk stratification is important, especially in children at high risk for perioperative airway obstruction. The take home message is to be vigilant and prepared to manage any potential airway complications.

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