Laryngospasm in Paediatric Anaesthesia: A Review

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Abstract: Paediatric laryngospasm is often a serious adverse respiratory event encountered during anaesthetic care of children. This common phenomenon is dependent on multiple factors. Identifying the risk factors and taking necessary precautions are the key points in prevention of laryngospasm. Although the majority of laryngospasm episodes are self-limited or responsive to conservative maneuvers, the anaesthesiologist must be prepared to treat the episode appropriately to avoid this life-threatening complication. The present review discusses laryngospasm with the emphasis on the risk factors associated with laryngospasm and the techniques used for different preventive and treatment modalities.

Keywords: Airway management, Anaesthesia, Complication, Laryngospasm, Paediatric, Succinylcholine.

INTRODUCTION

Laryngospasm is a common and potentially life-threatening complication encountered during anaesthetic care of children. It is more prevalent in younger age group [1] with incidence ranging from 14% in less than 6 years old to 3.4% in more than 6 years old [2]. The exaggeration of this reflex may result in complete glottic closure and consequently impending respiratory arrest [3]. In the majority of patients, the prolonged hypoxia and hypercapnoea abolishes the spastic glottis reflex and the problem is self-limited [4-6]. However, in certain cases, the spasm is sustained as long as the stimulus continues and morbidity such as arrhythmia, gastric aspiration, bronchospasm, pulmonary edema, cardiac arrest may occur [6-8]. Therefore, prompt and aggressive intervention is recommended [9,10] and the use of a structured algorithm has been suggested [11]. Many authors recommended airway manipulation first, then pharmacologic agents if necessary [9,10].

The authors of this article present a review of literature to discuss the general aspects of laryngospasm with prevention and treatment modalities.

DEFINITION

Laryngospasm may be defined as glottic closure due to reflex constriction of the laryngeal muscles; it can be complete or partial.

True laryngospasm is complete closure of the larynx caused by stimulation [12]. In true laryngospasm, false cords are tightly occluded, intra-pharyngeal part of the epiglottis moves posteriorly, while there are ventral movements of both the arytenoids cartilages to effectively seal the larynx.

In *partial spasm* both vocal cords are firmly pressed against each other, leaving a small lumen open at the posterior commissure which allows minimal ventilation by the anaesthesiologist.

EPIDEMIOLOGY

Laryngospasm is probably not an unusual occurrence in most anaesthesiologist's practice. Olsson [6] quoted an incidence of laryngospasm in children of about 1.7% with a higher incidence of 2.8% in infants between 1 and 3 months. In contrast, Burgoyne [13] reports a remarkably low incidence of only 0.1% in their studied paediatric population. Similarly, the studies of Cravero *et al*. [14] and Von Ungern- Stemberg [15] reported the incidence of laryngospasm in paediatric population ranging from 0.04 to 14 %. Studies show that supervision by less experienced and non-paediatric anaesthesiologists increases incidence of laryngospasm [16]. Children passively exposed to cigarette smoke also have an increased incidence [17,18].

The incidence of morbidity resulting from laryngospasm can vary as follows: cardiac arrest (0.5%), post-obstructive negative pressure pulmonary edema (4%), pulmonary aspiration (3%), bradycardia (6%), and oxygen desaturation (61%) [6,7,11,19].

PATHOPHYSIOLOGY

Laryngospasm involves apposition of structures at three levels: (a) the supraglottic folds, (b) the false cords, and (c) the true vocal cords. A dual mechanism closes the larynx during laryngospasm: a shutter effect involving the false cords and a ball-valve effect

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involving the false cords and redundant supraglottic tissues, owing to shortening of the thyroid muscles.

The internal branch of the superior laryngeal nerve innervates the larynx from its superior boundaries to the level of the true vocal cords, while the recurrent laryngeal nerve carries the sensory elements from below the vocal cords level. The entrance to the larynx has receptors which form a protective mechanism and have greatest degree of sensitivity. In man, the posterior aspect of the true vocal cords, which is more exposed to foreign material than the anterior aspect, is a region of greater distribution of nerve endings than the anterior aspect [16]. The motor response is *via* the three main intrinsic laryngeal muscles, the lateral cricoaretenoids, thyroaretenoids (the glottic adductors), and cricoaretenoids (the vocal cord tensors). They are all supplied by the vagus nerve.

As the translaryngeal pressure gradient increases during inspiratory efforts, soft tissues of the supraglottic region are drawn into the laryngeal inlet. After laryngeal spasm develops, it can become a prolonged process because of a continuous contraction of abdominal muscles, expiratory effort, and continuing closure of the larynx. Laryngospasm may be sustained and may become progressively worse as supraglottic tissues fold over the vocal cords during forceful inspiratory efforts.

The reasons for laryngospasm include: (a) a lack of inhibition of glottis reflexes because of inadequate central nervous system depression and (b) increased stimuli. Causes of stimulation in an inappropriate depth of anaesthesia include extubation, salivary secretion, blood irritating the vocal cords, stimulation of the airway by instrumentations like artificial airway, laryngoscopic blade or suction catheter [20]. Reflex closure of the larynx is less likely during the expiratory phase of breathing and more likely during the inspiratory phase.

CLINICAL MANIFESTATION

It is difficult to distinguish between true laryngospasm and glottis spasm. Therefore, most clinicians describe both as laryngospasm, either complete or partial [21]. In *complete laryngospasm*, there is no movement of air and absence of breath sounds. Moreover, ventilation could not be done. In *partial laryngospasm*, there is inspiratory stridor and minimum movement of the reservoir bag. There is also mismatch between patients' respiratory effort and actual ventilation.

However, in both complete and partial laryngospasm, tracheal tug, paradoxical movement of the chest and abdomen are noticed [7,22]. There may be airway obstruction which does not respond to insertion of an oropharyngeal airway. Oxyhaemoglobin desaturation, bradycardia, and central cyanosis may be considered as late signs [11]. When these occur, either alone or in combination, laryngospasm is possible. Any trigger should then be removed if possible.

DIFFERENTIAL DIAGNOSIS

Laryngospasm should be distinguished from other causes of airway obstructions, such as: (1) supraglottic obstruction, and (2) bronchospasm, which are associated with similar features like - (a) inspiratory stridor, (b) retraction of intercostals and, (c) rapid deterioration in oxygen saturation.

However, supraglottic obstruction and bronchospasm can be differentiated from laryngospasm by failure of jaw thrust or head tilt maneuver and gentle positive pressure ventilation with or without oral and nasal airway to relive the airway obstruction. Performance of laryngoscopy during an episode of laryngospasm is fraught with risk and should be avoided.

RISK FACTORS

Few contributing factors were identified in children who had had a documented episode of laryngospasm: younger age, higher ASA physical status, upper respiratory tract infection (URI), URI with or without the presence of an airway anomaly, and the use of an laryngeal mask airway were identified as important factors [1,23-28]. Furthermore, there is a 10-fold increase in the incidence of laryngospasm in children who are exposed to tobacco smoke (passive smoking) [27]. History of gastro-oesophageal reflux is also a risk factor for developing laryngospasm [29,30].

Insufficient depth of anaesthesia during both induction and emergence predisposes to laryngospasm. In addition, airway irritation with volatile anaesthetics, mucous or blood, and airway manipulation with suction catheter may also induce laryngospasm [22,31]. Volatile anaesthetics have been associated with laryngospasm in paediatric anaesthesia (2.3%) [2,7].

Amongst the volatile anaesthetics, the highest incidence of laryngospasm is associated with desflurane (50%). Isoflurane is associated with

laryngospasm much more than enflurane, halothane, and sevoflurane [7,19,32,33]. However, there is no difference in the incidence of laryngospasm between sevoflurane and halothane [34].

There is a definite role of anaesthesiologists' experience in paediatric anaesthesia, as a potential risk factor for laryngospasm. Mamie *et al*. [35] reported that children not anaesthetized by paediatric anaesthesiologists have a 1.7 times greater risk of perioperative respiratory adverse events (these events included laryngospasm). Similarly, laryngospasm is more likely to occur in children who are supervised by less experienced anaesthesiologists [36]. However, Flick *et al*. [37] in their case controlled study could not confirm that procedures supervised by paediatric anaesthesiologists reduce the risk for laryngospasm. Another recent study also reported similar findings [15].

There is a close association between laryngospasm and the type of surgery [6,11]. Tonsillectomy and adenoidectomy have the highest incidence of laryngospasm (21-26%) [3, 38-44]. Hypospadias surgery and transplant surgeries in children are highly associated with laryngospasm [6]. Oesophageal procedures may cause laryngospasm secondary to stimulation of distal afferent oesophageal nerves [6]. Appendicectomy, dilatation of the anus or cervix and mediastinoscopy carry a higher risk of laryngospasm [5].

PREVENTION (TABLE 1)

There are a number of ways reported to prevent the incidence of laryngospasm [45,46]. These include:

1. (a) Identify the patients at risk for laryngospasm. In the preoperative phase, a detailed history should be taken to identify the risk factors. Household exposure to smoking should be questioned. If an adolescent is a smoker, a period of abstinence from smoking for at least 48 hours to up to 10 days may be required to reduce the risk of laryngospasm [47]. In children with upper respiratory tract infection, the use of facemask may decrease the incidence of laryngospasm by minimizing airway irritation [48,49].

   (b) To decrease the overall risk of laryngospasm, anaesthesia should be carried out by an experienced anaesthesiologist [35].

2. Inhalational induction of anaesthesia should be carried out by a nonirritant anaesthetic such as sevoflurane.

3. Laryngoscopy and tracheal intubation should be attempted after deepening the level of anaesthesia. When planning a 'deep' extubation for a tracheal tube, the airway should be suctioned first and the patient placed in the lateral position. After extubation, the patient is best left undisturbed if the airway is clear. An awake extubation, on the other hand, should occur once facial grimacing, adequate tidal volumes, a regular respiratory pattern, coughing, and preferably eye opening have returned [5].

4. Laryngospasm tends to occur more often during emergence than during induction [50] of anaesthesia. However, controversies exist about the technique of tracheal extubation which reduces the incidence of laryngospasm. Concerning tracheal extubation, Landsman [51] suggested that the endotracheal tube 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. 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.

5. Many drug have been used to prevent laryngospasm following general anaesthesia :

a) Premedication with anticholinergic agents to prevent laryngospasm is controversial. However, anticholinergics may have an indirect role by reducing the secretions which may have a triggering effect to cause laryngospasm [7,39,52,53]. Premedication with oral benzodiazepine decreases upper airway reflexes and thus may decrease laryngospasm during induction of anaesthesia [54,55].

b) The role of lignocaine (1 to 2 $mg.kg^{-1}$) in preventing laryngospasm is controversial [27]. The beneficial effects of lignocaine may be attributed to a central increase in the depth of anaesthesia. Therefore, to utilize the beneficial effect of central nervous system depression by lignocaine, extubation of trachea must be performed before signs of swallowing occur [51].

c) Gulhas *et al*. [42] recommended use of magnesium (15 $mg.kg^{-1}$ in 30 ml 0.9 % of normal saline) over 20

minutes after tracheal intubation to prevent laryngospasm during tracheal extubation. According to them, magnesium increase the depth of anaesthesia and also provide the relaxation of the muscles by which laryngospasm may be avoided.

d) Acupuncture has been described for the prevention of laryngospasm. However, this method did not totally abolish laryngospasm [38].

Table 1: A simplified Algorithm for Prevention of Laryngospasm

Anaesthesia induction
Identify the risk factors ↓ Premedication with anticholinergics and benzodiazepine ↓ Insert i.v. line 2 min after Sevoflurane / propofol induction ↓ Tracheal intubation after ensuring adequate level of anaesthesia
Emergence
Gentle suctioning of blood and secretions from oropharynx ↓ Put the patient on lateral position with head down ↓ Discontinue anaesthetic agents ↓ Give lignocaine 1 mg.kg^{-1} i.v. or propofol 0.25 mg – 0.5 mg.kg^{-1} i.v. ↓ Wait for the patient to open the eyes and spontaneously wake up ↓ Extubate the trachea using the 'artificial cough' technique

(Adapted with permission from Alalami AA, Ayoub CM, Baraka AS. Laryngospasm: review of different prevention and treatment modalities. Pediatr Anesth 2008; 18: 281-8).

MANAGEMENT (TABLE 2)

To manage laryngospasm effectively, it first needs to be recognized. Once recognized, most agree on a similar protocol. Treatment success mainly depends on the experience of the anaesthesia provider.

The treatment of laryngospasm depends on whether airway obstruction is complete or incomplete. The single diagnostic feature that distinguishes complete from incomplete airway obstruction is very simply the absence or presence of breath sound. If there are inspiratory or expiratory sounds, grunts, or whistles, then chances are that the child has incomplete airway obstruction. Because incomplete airway obstruction may rapidly become complete, signs and symptoms of obstruction (*e.g.* tracheal tug, paradoxical respiration) should be treated aggressively. Airway obstruction of either type requires initial treatment with a patency preserving maneuver such as the modified jaw thrust/chin-lift maneuver, inserting an oral or nasal airway and positive pressure ventilation with face mask and 100% oxygen. Jaw thrust including firm pressure on the ascending branch of the mandibular, triggering a severe painful stimulus ('laryngospasm notch') and slight, but frequent strokes using a reservoir bag with 100% oxygen are very useful measures in this situation till adequate ventilation is established. The 'laryngospasm notch' [56] is located just behind the earlobe. It is bordered by the base of the skull superiorly, the mastoid process posteriorly, and the ramus of the mandible anteriorly. Stimulation of laryngospasm notch can break a laryngospasm and also assist the anesthetist in initiating spontaneous respirations in the sedated patient. If these techniques suffice to treat the spasm, partial laryngospasm is diagnosed.

Table 2: A Simplified Algorithm for Treatment of Laryngospasm

Identification and removal of the offending stimulus such as secretions mucous or blood ↓ Insertion of oral or nasal airway if possible ↓ Apply jaw thrust maneuver while firmly pressing on the "laryngospasm notch" ↓ Intermittent positive pressure ventilation with face mask ↓ *If laryngospasm is not relieved*, deepen level of anaesthesia by propofol i.v. 0.25 – 0.5 mg.kg^{-1} ↓ *If laryngospasm is not relieved*, inject suxomethonium i.v. 0.1-3 mg.kg^{-1} or i.m. 3-4 mg.kg^{-1} followed by mask ventilation and / or tracheal intubation

(Adapted with permission from Alalami AA, Ayoub CM, Baraka AS. Laryngospasm: review of different prevention and treatment modalities. Pediatr Anesth 2008; 18: 281-8).

Intralaryngeal pressure becomes subatmospheric during inspiratory effort and soft tissue compression of the larynx increases. This can be improved by the application of continuous positive airway pressure (CPAP) [5]. If the obstruction is not relieved, complete obstruction should be suspected. In complete spasm CPAP may make the situation worse by forcing the area just above the false vocal cords against each other closing the entrance to the larynx [12,55]. Therefore, if complete spasm cannot be relieved,

deepening the level of anaesthesia with intravenous or inhalational anaesthetic is necessary. Propofol can be used at dose 0.25-0.8 $mg.kg^{-1}$ because of its rapid and predictable action, but if there is no intravenous line, inhalational anaesthetic (sevoflurane or halothane) can be used [10,32,57-59]. If this technique fails and oxyhaemoglobin desaturation ensues suxamethonium can be given at dose of 0.1-3 $mg.kg^{-1}$ followed by mask ventilation and, or tracheal intubation [10,60,61].

Propofol should be used prior to suxamethonium because it is successful in treating laryngospasm in 77% of cases [52,58,59]. Moreover, propofol offers other advantages like - (a) lack of interaction of a depolarizing muscle relaxant with a previously administered non-depolarizing muscle relaxant, (b) avoiding suxamethonium will eliminate the possibility of prolonged paralysis in patients with pseudocholinesterase deficiency, or (c) when suxamethonium is contraindicated (patients with recent burns, muscular dystrophy, hyperkalemia *etc*.) [58,61]. Suxamethonium and volatile agents administered individually or given together increase the possibility of malignant hyperthermia. The sustained masseter muscle spasm will make the scenario immediately life-threatening.

Management of laryngospasm becomes more of an issue when no intravenous access is available. For complete laryngospasm where there is no ability to increase the depth of anaesthesia with a volatile anaesthetic some authors advocate to use suxamethonium through intramuscular route into a large muscle bulk or *via* intra-lingual or submental routes [62-64]. In emergency situation intraosseous (IO) route should also be thought of. The only delay is the time taken to insert the IO cannula. This route is probably the most reliable route into the systemic circulation in a peri-arrest situation as may occur in severe laryngospasm. Administration of suxamethonium should not be delayed until the patient becomes severely desaturated (SpO_2 < 85%). However, in some cases, administration of suxamethonium following hypoxia may be associated with severe bradycardia and even cardiac arrest. Thus, it is highly recommended to administer atropine prior to administration of suxamethonium to treat laryngospasm [7,11,39]. Quick onset non-depolarizing muscle relaxant (rocuronium bromide) may provide an alternative to suxamethonium to provide ventilation and intubation. However, intramuscular administration of a nondepolarizing muscle relaxant is not suitable for emergency intubation mainly because of slow absorption [65,66]. Of note, shorter period of time is needed to provide adequate conditions for ventilation by mask than is needed to provide adequate conditions for tracheal intubation.

Under the extreme conditions, the vocal folds may be sprayed directly with lignocaine to relax the larynx and facilitate intubation. If, after all with these measures the airway has not been secured, then cricothyrotomy or emergency tracheostomy may be required.

Acute pulmonary oedema is often associated with acute upper airway obstruction caused by laryngospasm. Laryngospasm-induced negative pressure pulmonary oedema is most effectively managed by prompt tracheal intubation and application of continuous positive airway pressure or positive ventilation. Diuretics, morphine, and sedatives are also effective adjuncts. If the patient has no underlying cardiorespiratory problems, the pulmonary oedema resolves quickly.

The use of laryngeal mask airway (LMA) in children with URIs was associated with a higher incidences of laryngospasm, cough, and oxygen desaturation [15]. In anesthesia using LMA, there is a controversy regarding the timing of LMA removal to avoid laryngospasm. Some authors recommend awake removal of LMA in children [67,68]. However, in both sevoflurane and isoflurane anesthesia, there is a higher incidence of laryngospasm during awake compared with deep LMA removal [33]. Always remove any supraglottic source of laryngospasm like blood, secretion or vomitus. Consideration should be given to performing cautious direct laryngoscopy to gently suction the larynx clear; however, this must be performed with care as the situation may worsen.

Gentle chest compressions have been reported as a novel treatment of laryngospasm [69]. A non-randomized study with about 600 children undergoing adenotonsillectomy showed 74% were successfully treated by chest compression against 38% by the standard method (CPAP). The gentle chest compressions were delivered, while 100% O_2 *via* a tight-fitting facemask was provided, 'using the extended palm of the free hand placed on the middle of the chest, with the fingers directed caudally and performing a compression force half or less than half that used for cardiopulmonary resuscitation at a rate of approximately 20–25 compressions per min'. The mechanism of action of this maneuver may be by increasing intra-thoracic pressure, stimulation of shallow breaths, or stimulation of the vagal Hering–

Breuer deflation reflex, which may inhibit reflex glottic closure [5].

When laryngospasm is successfully treated, ventilation should be supported initially with 100% oxygen. Laryngeal suction should be considered again. Further support of the airway may be required with tracheal intubation (especially when airway soiling or pulmonary oedema has occurred). This will allow for toileting and suctioning of the airway and re-recruitment of the pulmonary alveoli to prevent postoperative secretion retention and infection. The need for prolonged or postoperative ventilation must be judged on an individual basis.

CONCLUSION

Paediatric laryngospasm is an important clinical scenario which many anaesthesiologists will encounter within their regular practice. It is a relatively common phenomenon that occurs with varying frequency dependent on multiple factors. Identifying the risk factors and taking the necessary precautions are key points in preventing laryngospasm. To overall decrease the risk of laryngospasm, anaesthesia should be carried out by an experienced anaesthesiologist. During tracheal intubation and extubation, the patient must be deeply anaesthetized to avoid laryngospasm with non-irritant volatile anaesthetics or with intravenous propofol or lignocaine. In case of failure, intravenous or intramuscular suxamethonium can be used. Rapid and effective management of laryngospasm will improve the safety of paediatric anaesthesia.

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