Continuing Medical Education Article

Laryngospasm in paediatric anaesthesia

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Laryngospasm occurs frequently during the administration of anaesthesia to paediatric patients.[1] The reported incidence of laryngospasm in patients aged 0–9 years is 17.4/1,000 patients.[1] This incidence is almost twice that of the total population studied (8.7/1,000 patients). Although most instances of laryngospasm resolve without serious complications, complete laryngeal obstruction can result in intense laryngospasm, gastric aspiration, cardiac arrest and death.[1] Because of the high incidence of laryngospasm and the severity of potential complications, a review of laryngospasm in children, its etiology, treatment and prevention, is presented.

Pathophysiology

The pathophysiological mechanism of laryngospasm during anaesthesia is unclear.[2] Most practising anaesthetists believe that laryngospasm is precipitated by closure of the true vocal cords. This closure results in either complete or incomplete airway obstruction. However, Keating[3] questioned the mechanism of laryngospasm. He stated that either the true vocal cords alone or both the true and false cords become apposed in the midline and close the glottic inlet during laryngospasm. The role of the false vocal cords in laryngospasm is unclear.

Debate over the mechanism of laryngospasm continues from both anatomical and neurophysiological points of view. We found it most helpful to return to Fink's early review of laryngospasm[2] for a clearer description of this entity. Laryngeal closure involves apposition of structures at three levels: (1) the supraglottic folds, (2) the false vocal cords, and (3) the true vocal cords. With the aid of lateral *x*-rays of the neck, tomography and electromyography, Fink found that the human larynx was more than just a shutter. He proposed a dual mechanism for closure of the larynx: first, a shutter effect (the vocal cords) and secondly, a ball valve effect (the false cords and the redundant supra-glottic tissue). The ball valve effect depends on the shortening of the thyrohyoid muscle, an extrinsic muscle (Figure 1). The soft tissues of the supra-glottic region then become rounded and redundant and are drawn into the laryngeal inlet as the translaryngeal inspiratory pressure gradient increases. Fink differentiated stridor from laryngospasm – the former being an intermittent closure of the glottis alone and the latter, a prolonged interruption of respiration due to a ball valve mechanism of the vocal cords, false cords and supra-glottic tissue. Fink noted that "when laryngeal spasm sets in, the expiratory effort becomes prolonged and closure of the larynx and contraction of the abdominal wall become continuous." Laryngospasm in fact may be an extreme form of cough.

Clinicians commonly observe prolonged glottic closure in response to glottic or supra-glottic mucosal stimulation. Laryngeal closure continues long after cessation of mucosal stimulation. Suzuki believed that laryngospasm was an exaggerated laryngeal response to stimulation of the superior laryngeal nerve.[4] Although his studies involved animals, he made some interesting observations which might very well have clinical relevance to humans. He noted that synaptic transmission in the superior laryngeal nerve was reduced during deep barbiturate anaesthesia. This contradicted the belief that barbiturates actually aggravated laryngospasm. He also observed that hypoxia abolished post-synaptic potentials and led to an overall depressive effect on all reflex neural activity in the animal model. This latter observation supported the notion that severe hypoxia may actually terminate laryngospasm. It appears that the results of additional animal studies explain in part our clinical observations.[5,6] Laryngospasm is a sustained event, which can be abolished by deep anaesthesia, severe hypoxia and hypercarbia.

Etiology

Several investigators have studied laryngospasm during anaesthesia.[1,7] Olsson *et al.*,[1] during an 11 year prospective study, found an overall incidence of 7.9/1,000 anaesthetics or 8.7/1,000 patients. The incidence in children 0–9 years of age was higher, 17.4/1,000 patients, and within this age group, infants 1–3 months of age had the greatest incidence (more than three times the rate in any other age group). Other factors associated with an increased incidence of laryngospasm in children were:

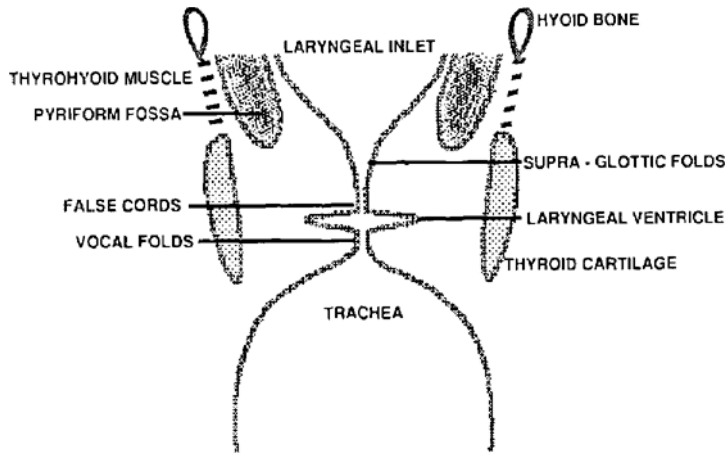

FIGURE 1 Sectional view of larynx (adapted from Fink BR. The Etiology and Treatment of Laryngeal Spasm. Anesthesiology 1956; 17: 569–77, with permission).

extubation (42/1,000), presence of a nasogastric tube (48.5/1,000) and oral endoscopy and esophagoscopy (48.5/1,000). The incidence of laryngospasm was highest (95.8/1,000) in children with respiratory tract infections.

Barbiturates have been identified as a group of drugs which predispose to the occurrence of laryngospasm. Olsson and Hallen[1] found a slightly greater incidence of laryngospasm in children who were anaesthetized with a barbiturate compared to those anaesthetized without a barbiturate. Some authors have suggested that laryngeal spasm is a direct complication of the use of thiopentone.[8–10] They have speculated that laryngospasm is due to parasympathetic overactivity in the airway inlet resulting from a direct action of thiopentone. Others have proposed that parasympathetic overactivity increases as the dose of the barbiturate increases.[11] Animal studies have been contradictory. In 1937, an examination of the effect of short-acting barbiturates on the glottic opening in cats suggested a relationship between barbiturates and parasympathetic-induced laryngospasm.[12] A more recent study in 1977 found that barbiturates depressed the reflex closure of the glottis in cats.[4] The debate concerning the effect of short-acting barbiturates on the laryngeal reflex mechanism continues unresolved.

Volatile anaesthetic agents have also been associated with laryngospasm.[13,14] In the cat, laryngospasm can be triggered by instilling ether or halothane directly into the trachea: an effect not seen with methoxyflurane.[13] It was suggested that laryngospasm might be avoided if the concentrations of these volatile anaesthetics were restricted. However, there are no data to support such a notion. Compared to halothane and enflurane, isoflurane has been associated with a significantly greater incidence of laryngospasm during induction of anaesthesia in children.[14]

Light anaesthesia is another important cause of laryngospasm. In 1939, Cole addressed the problem of respiratory obstruction during thyroidectomy and concluded that "if a patient is anaesthetized deeply before the operation is begun, laryngeal spasm will be much less likely to develop."[15] Reflex closure of the larynx was thought to be a response to stimulation of visceral nerve endings in the pelvis, abdomen, thorax, or larynx itself; and when such stimulation led to respiratory obstruction this was most often the result of indequate anaesthesia.[2] Because many patients are intubated during anaesthesia, the risk of laryngospasm due to light anaesthesia during surgery is minimal. However, laryngospasm may occur following extubation during light anaesthesia in children.[16] Sadly, our technology has not eliminated this serious complication but has simply deferred it from the intraoperative period to the postoperative period.

Management

Incomplete airway obstruction is generally associated with an audible inspiratory or expiratory sound, which is best heard with a precordial stethoscope. If the obstruction becomes progressively worse, tracheal tug, and paradoxical respiratory movements of the thorax and abdomen will develop. Should the obstruction progress

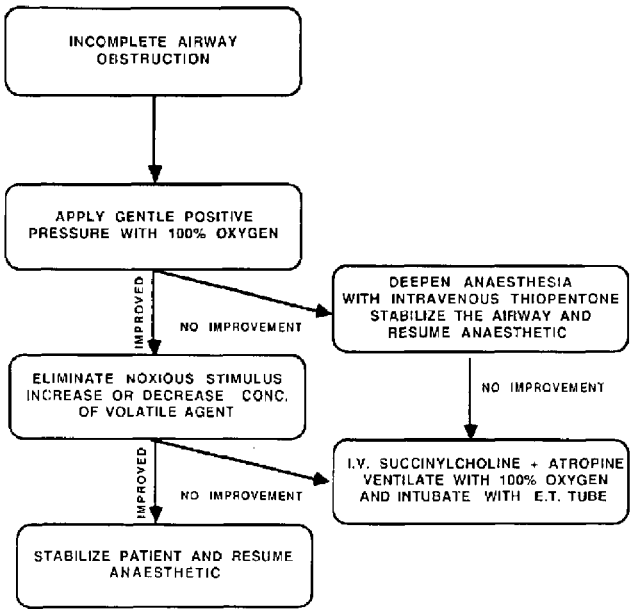

FIGURE 2 Algorithm displaying course of management for incomplete airway obstruction.

from incomplete to complete, the audible sounds cease, leaving only visible signs of airway obstruction.

Incomplete upper airway obstruction can often be relieved with one of several therapeutic manoeuvres (Figure 2). The first is to remove the irritant stimulus,[2] by terminating surgical stimulation of visceral nerve endings or by removing debris from the larynx. The second is to deepen the level of anaesthesia. The third is to facilitate ventilation by applying gentle continuous positive airway pressure with 100 per cent oxygen via a tight-fitting face mask.

Complete airway obstruction shares many of the clinical signs with incomplete obstruction – tracheal tug, indrawing of the chest wall and marked abdominal respiration. However, the absence of respiratory sounds is the "sine qua non" of complete laryngospasm. Continuous monitoring of respiratory sounds with a precordial stethoscope will lead to an early diagnosis of airway obstruction and should result in early therapeutic intervention. A precordial stethoscope should be a routine monitor during the administration of anaesthesia to paediatric patients.

When complete obstruction of the upper airway occurs, what is the best management? Fink[2] wrote: "Such a spasm cannot be broken by bag pressure through a mask. Forced inflation of the pharynx distends the pyriform fossas on either side of the larynx and presses the aryepiglottic folds more firmly against each other" (Figure 1). This manoeuvre is commonly taught as an effective means of managing laryngeal spasm. However, in the presence of complete obstruction, oxygen will fill the stomach instead of the lungs. In the presence of complete obstruction of the airway, the temporomandibular joint should be dislocated anteriorly by applying pressure to the ascending rami of the jaw.[2] This manoeuvre lengthens the thyrohyoid muscle and unfolds the soft supraglottic tissue. If dislocation of the tempero-mandibular joint fails, then atropine and succinylcholine should be administered intravenously. However, laryngospasm may occur when intravenous access is not available. In this instance, intramuscular succinylcholine (4 $mg \cdot kg^{-1}$) is recommended.[17] After intramuscular succinylcholine, the vocal cords will usually relax sufficiently within one minute to permit ventilation, and within several minutes to facilitate tracheal intubation. Pulmonary oedema has been reported following the administration of intramuscular succinylcholine.[18] The mechanism of this is unclear. Intralingual atropine and succinylcholine are not recommended in children anaesthetized with halothane/nitrous oxide/oxygen[19] since ventricular arrhythmias have been reported. If the laryngospasm is sustained and the child becomes hypoxic, it may

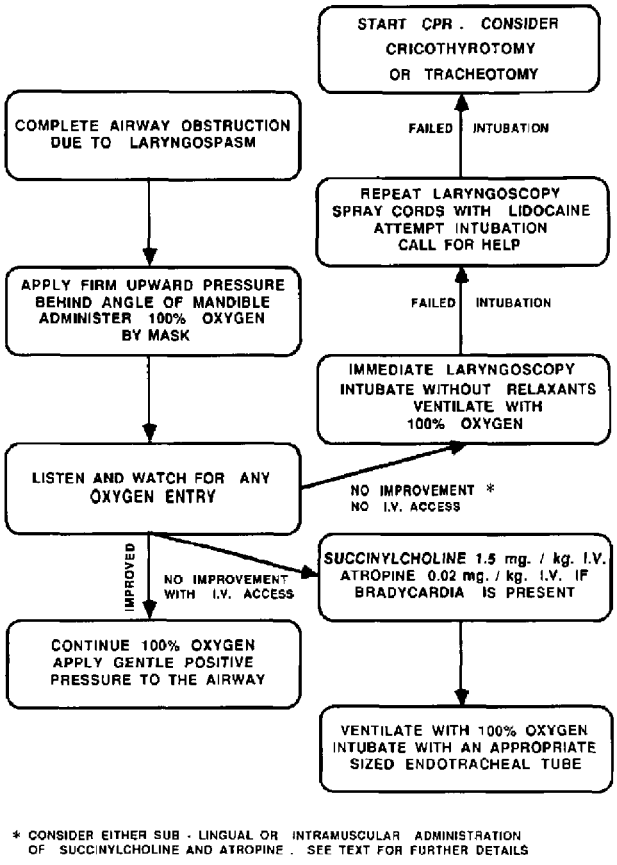

FIGURE 3 Algorithm displaying course of management for complete airway obstruction.

be necessary to intubate without muscle relaxation.[20] This may be preferable to waiting for the effects of succinylcholine in a child who has laryngospasm and who is bradycardic. Under these extreme conditions the vocal folds may be sprayed directly with lidocaine (Figure 3), in order to relax the larynx and facilitate intubation. The mechanism of this relaxation is unknown but may be due to the direct effect of local analgesia or to a prolonged effect of hypoxia. If after all of these measures the airway has not been secured, then cricothyrotomy or emergency tracheostomy may be required (Figure 3).

Several other therapeutic modalities have been proposed for the treatment of laryngospasm, including hypoxia, doxapram, diazepam and chest compression.[4,21–24] We do not condone these treatments.

Prevention

Prevention, of course, is the best therapy for laryngospasm. Olsson *et al.*[1] stated that "to minimize the risk of perioperative laryngospasm, it is necessary to be aware of the factors which increase the risk of laryngospasm." The high risk factors Olsson enumerated included pre-existing respiratory problems, a history of previous anaesthetic complications, surgery including endoscopy and hypospadias repair, and those with a nasogastric tube *in situ*. Intravenous lidocaine 2 $mg \cdot kg^{-1}$ given one minute before extubation may prevent or attenuate laryngospasm.[16] However, the effectiveness of lidocaine in this regard is controversial.[25,26] Leicht *et al.*[26] suggest that intravenous lidocaine does not prevent laryngospasm if the patients are extubated when they begin to swallow. The

authors also suggest that to derive any benefit from the lidocaine one must extubate before signs of swallowing activity. The mechanism of action of intravenous lidocaine may be a central interruption of the reflex pathway, or a direct peripheral action on the sensory or motor nerve terminals. Although the effectiveness of lidocaine is still questioned, we believe that in patients who are at high risk of developing laryngospasm, lidocaine should be administered intravenously slowly over a 30-second period.[27]

Lee and Downes[28] proposed the following guidelines to diminish the likelihood of post-extubation laryngospasm. "The infant or child, before tracheal extubation, should open his eyes and mouth spontaneously, move all extremities vigorously and resume a normal breathing pattern after a cough." At the completion of surgery the children should remain on the operating room table with all monitors in place until the child is awake and fulfills the above criteria. Furthermore, during emergence, some anaesthetists avoid disturbing, touching, or stimulating the patient until he/she awakens. This "no touch" technique is believed to prevent or reduce premature coughing or bucking while the endotracheal tube is in place. This also allows the child to awaken fully before extubation and thus may further reduce the likelihood of extubation laryngospasm.

Another preventative measure is to directly apply local anaesthetic to the supraglottic mucosa. Cocaine (four per cent solution) has been effective in reducing the incidence of laryngospasm when applied topically to the vocal cords, arytenoids and false cords at the end CO_2 laser surgery.[29]

Vagolytic drugs have been recommended as premedicants in order to prevent laryngospasm. The results of a survey in 1978 revealed that 21 per cent of anaesthetists advocated the use of IM atropine as premedication in order to prevent laryngospasm.[30] Although the mechanism of action of IM atropine is unclear, presumably it is achieved through the elimination of undesirable secretions. However, a conflicting report found that the incidence of laryngospasm in those patients receiving vagolytics in their premedication was twice that in patients who did not receive such therapy.[1] Intramuscular vagolytic premedication is no longer a routine at our institution. Extubation while the patient is still deeply anaesthetized is a method advocated by some. While no data are available, it is our impression that the incidence of laryngospasm may be reduced. However, we are concerned that this practice will lead to the premature transfer of children to the Recovery Room where they may be prone to hypoventilation, aspiration or respiratory obstruction. We currently extubate patients who are still deeply anaesthetized only when there is a specific indication to do so.

Conclusion

Laryngospasm is seen most frequently in paediatric anaesthesia. Although laryngospasm has been regarded as a self-limiting complication, statistically unlikely to produce severe complications, 5.0/1,000 patients who develop laryngospasm have a cardiac arrest.[1] In less severe instances, laryngospasm may be complicated by bronchospasm, hypoxia, gastric aspiration, arrhythmias, and delayed recovery. Pulmonary oedema following laryngospasm has recently been reported as a significant complication.[28] A high level of preparation and forethought can lead to a reduction in morbidity and mortality as a result of laryngospasm.

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