

Review article

Laryngospasm: review of different prevention and treatment modalities

ACHIR A. ALALAMI MD, CHAKIB M. AYOUB AND
ANIS S. BARAKA MD

Department of Anaesthesia, American University of Beirut Medical Center, Beirut, Lebanon

Summary

Laryngospasm is a common complication in pediatric anesthesia. In the majority of cases, laryngospasm is self-limiting. However, sometimes laryngospasm persists and if not appropriately treated, it may result in serious complications that may be life-threatening. The present review discusses laryngospasm with the emphasis on the different prevention and treatment modalities.

Keywords: laryngospasm; review; anesthesia; treatment; prevention; modalities

Introduction

Laryngospasm is mainly seen in children. It is a reflex closure of the upper airway as a result of the glottic musculature spasm. It is essentially a protective reflex that acts to prevent foreign material entering the tracheobronchial tree. The exaggeration of this reflex may result in complete glottic closure and consequently impeding respiration (1). This leads to hypoxia and hypercapnea. In the majority of patients, the prolonged hypoxia and hypercapnea abolishes the spastic reflex and the problem is self-limited (2–4). However, in certain cases, the spasm is sustained as long as the stimulus continues and morbidity such as cardiac arrest, arrhythmia, pulmonary edema, bronchospasm or gastric aspiration may occur (4–6).

The present review discusses the general aspects of laryngospasm with the emphasis on the different prevention and treatment modalities.

Epidemiology

The overall incidence of laryngospasm is 0.87%. The incidence in children in the first 9 years of age is 1.74% with a higher incidence of 2.82% in infants between 1 and 3 months (4).

The incidence of morbidity resulting from laryngospasm can vary as follows: cardiac arrest 0.5%, postobstructive negative pressure pulmonary edema 4%, pulmonary aspiration 3%, bradycardia 6% and oxygen desaturation 61% (4,6–8).

Mechanism

Most laryngeal reflexes are elicited by stimulation of the afferent fibers contained in the internal branch of the superior laryngeal nerve. These reflexes control the laryngeal muscle contractions which protect the airway during swallowing. Laryngospasm may occur secondary to loss of inhibition of the laryngeal closure reflex as a result of abnormal excitation (3).

Clinical manifestation

Laryngospasm can be partial, when there is some degree of air entry and it is recognized by

Correspondence to: Achir A. Alalami MD, Chief resident of Anaesthesiology, Department of Anaesthesiology, American University Hospital, Riad El Solh, 1107-2020, Beirut, Lebanon (email: achiralami@gmail.com).

inspiratory stridor (6). Laryngospasm can be complete, with no air movement and absence of breath sounds. It is early recognized by feeling with the hand or placing the ear over the mouth (9,10). In both partial and complete laryngospasm, signs of airway obstruction such as tracheal tug, paradoxical movement of the chest and abdomen are noticed (6,11,12). Late signs such as oxyhemoglobin desaturation, bradycardia and central cyanosis may ensue (8).

Differential diagnosis

Laryngospasm should be differentiated from other causes of airway obstruction such as bronchospasm or supraglottic obstruction (8). Both supraglottic obstruction and partial laryngospasm are associated with inspiratory stridor and intercostal retractions with rapidly deteriorating oxygenation. These two complications can be differentiated by directly visualizing the vocal cords while the patient is making inspiratory efforts (9). However, in the clinical situation, inserting a laryngoscope to visualize the vocal cords in such situation may worsen laryngospasm. Jaw thrust or head tilt maneuver with or without insertion of oral or nasal airway while applying gentle positive pressure would relieve both supraglottic obstruction and partial laryngospasm. If these fail to treat the obstruction, complete laryngospasm should be suspected and appropriate treatment should be initiated (10). In this issue if the journal, Anghelescu and Burgoyne (13) discuss the incidence and treatment of laryngospasm at the St Jude Children's Research Hospital in Memphis. Anesthesiologists must also differentiate laryngospasm from breath-holding by the presence of rocking-type movement of the chest wall and abdomen (14).

Laryngospasm can be psychogenic which is most often seen in anxious adolescents and young adults. It presents with sudden paroxysms of stridor or wheezing in response to exercise or emotional stress and is treated with reassurance and midazolam sedation (15,16).

Risk factors

Risk factors can be classified into three categories: anesthesia-related factors, patient-related factors and surgery-related factors:

Anesthesia-related factors

Insufficient depth of anesthesia during both induction and emergence predisposes to laryngospasm. During anesthesia including tracheal intubation, laryngospasm tends to occur after extubation, while anesthesia by spontaneous breathing using a face or laryngeal mask may result in laryngospasm during induction or maintenance (8,11,17).

In addition, airway irritation with volatile anesthetics, mucus or blood and airway manipulation with suction catheter or laryngoscope blade may also induce laryngospasm (11,17).

Among the intravenous (i.v.) induction agents, barbiturates such as thiopentone have been shown to increase the incidence of laryngospasm (4,6,18–22). Ketamine is rarely associated with laryngospasm (0.4%) (5,23). However, Ketamine may cause hypersalivation which can precipitate laryngospasm by irritating the vocal cords (23). Also, anesthesia induction with propofol is less associated with laryngospasm than sevoflurane induction (24).

Volatile anesthetics have been associated with laryngospasm in pediatric anesthesia (2.3%) (5,6). Among all the volatile agents, the highest incidence of laryngospasm is associated with desflurane (50%). Isoflurane is more associated with laryngospasm than enflurane, halothane and sevoflurane (6,7,25,26). There is no difference in the incidence of laryngospasm between sevoflurane and halothane (27).

Finally, laryngospasm is more likely to occur in children which are supervised by less experienced anesthesiologists (28).

Patient-related factors

The incidence of laryngospasm following general anesthesia is inversely correlated with age (29). Children with upper respiratory tract infection or active asthma have irritable airway and are approximately 10-fold more prone to develop laryngospasm.

Airway hyperactivity lasts for up to 6 weeks after respiratory infection, thus elective surgery can be delayed for 6 weeks (4,5,18,30–32).

Chronic smokers have increased airway reflex sensitivity and are more prone to develop laryngospasm. A period of abstinence from smoking of at

least 48 h and possibly up to 10 days may be required to reduce the risk of airway problems (33). There is a 10-fold increase in the incidence of laryngospasm in children who are exposed to tobacco smoke. Therefore, preoperative visit should include questioning about 'passive smoking' (20). History of gastroesophageal reflux is also a risk factor for developing laryngospasm. (34,35). Finally, patients with elongated uvula and those with history of choking during sleep may also develop laryngospasm under general anesthesia (11,36,37).

Surgery-related factors

There is a close association between laryngospasm and the type of surgery (4,8). Tonsillectomy and adenoidectomy have the highest incidence of laryngospasm (21–26%) (1,19,38–43). Other types of surgery such as appendectomy, cervical dilation, hypospadias surgery and skin transplant in children are highly associated with laryngospasm (4). Thyroid surgery has been associated with laryngospasm secondary to superior laryngeal nerve injury or to (44) iatrogenic removal of parathyroid glands causing hypocalcemia that predisposes to laryngospasm (45). Esophageal procedures may cause laryngospasm secondary to stimulation of distal afferent esophageal nerves (4,46).

Prevention

Identifying the patients at risk for laryngospasm and taking the necessary precautions are the most important measures to prevent laryngospasm.

Inhalational induction of anesthesia should be carried out by a nonirritant anesthetic such as sevoflurane. Also, during sevoflurane induction of anesthesia, it has been recommended to insert the i.v. line 2 min after the loss of lid reflex to ensure an adequate level of anesthesia and to decrease the incidence of laryngospasm (47). Laryngoscopy and tracheal intubation should also be attempted after deepening the level of anesthesia in order to avoid laryngospasm.

Many controversies exist among anesthesiologists about the best technique of tracheal extubation which reduces the incidence of laryngospasm. Both awake and anesthetized extubation have advantages and disadvantages.

The advantage of deep tracheal extubation over awake extubation in children undergoing tonsillectomy is that patients are less likely to cough and strain which can cause bleeding from the tonsillar bed and consequently increasing the risk of laryngospasm. On the other hand, awake tracheal extubation offers the advantage of protecting the airway against aspiration during this vulnerable period.

Patel *et al.* (48) undertook a study comparing awake vs anesthetized tracheal extubation of patients after tonsillectomy and adenoidectomy. They used the criteria of awake extubation when patients demonstrated facial grimace, had adequate tidal volumes and respiratory rate, coughed with open mouths or opened their eyes. They concluded that there is no difference in laryngospasm incidence between the two techniques. On the other hand, Pounder *et al.* (49) studied the incidence of respiratory complications in children undergoing minor surgery and found greater oxyhemoglobin desaturation in the awake extubation group than in the deep extubation group ($P < 0.05$). They also found 16% incidence of laryngospasm in the awake group vs 4% incidence in the anesthetized group ($P > 0.05$). The 'No Touch' technique was initially described by Lee (6,50). It is virtually an awake tracheal extubation. This technique consists of suctioning of the blood and secretions from the pharynx, turning the patient to the lateral position while anesthetized, discontinuing the volatile anesthetics and avoiding any stimulation until the patients open their eyes and spontaneously wake up to be followed by tracheal extubation. Tsui *et al.*'s (40) study applied this technique to 20 children undergoing tonsillectomy and adenoidectomy and found no incidence of laryngospasm. However, this study has several shortcomings. First, there was no control group with the deep extubation technique. Second, the size of the group was small (20 patients).

Concerning tracheal extubation, Lee suggested that the tracheal 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 (12). Also, 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.

Many drugs have been used to prevent laryngospasm following general anesthesia.

Premedication with anticholinergic agents to prevent laryngospasm is controversial. However, anticholinergics decrease secretions which play a role in triggering laryngospasm and thus they play an indirect role in reducing the incidence of laryngospasm (6,29,38,51). Also, premedication with an oral benzodiazepine decreases upper airway reflexes and thus may decrease laryngospasm during induction of anesthesia (47,51,52,53).

The role of lidocaine in preventing laryngospasm is controversial. The i.v. administration of lidocaine for prevention of laryngospasm was initially described by Baraka. He studied 40 children undergoing tonsillectomy and adenoidectomy and found that none of the 20 children who had received an i.v. injection of lidocaine $2 \text{ mg}\cdot\text{kg}^{-1}$ 1 min before extubation developed laryngospasm after tracheal extubation, while four of 20 children of the control group developed severe laryngospasm after extubation of the trachea (42). However, Leicht *et al.* studied 100 children after tonsillectomy in which i.v. lidocaine $1.5 \text{ mg}\cdot\text{kg}^{-1}$ was administered before tracheal extubation when patients started swallowing. They found no difference in the incidence of laryngospasm between the study and control groups. They suggested that the beneficial effects of lidocaine demonstrated by Baraka may be attributed to a central increase in the depth of anesthesia. Thus, in order to benefit from the effect of central nervous depression produced by lidocaine, tracheal extubation must be performed before signs of swallowing occur (43). Koc *et al.* have also reported that 2% topical lidocaine sprayed to the glottis at $4 \text{ mg}\cdot\text{kg}^{-1}$ or 2% intravenous lidocaine given at $1 \text{ mg}\cdot\text{kg}^{-1}$ 5 min before extubation are fairly effective in preventing laryngospasm following tonsillectomy and adenoidectomy (39). Also spraying the glottis with 2% lidocaine at $4 \text{ mg}\cdot\text{kg}^{-1}$ has an important clinical application in decreasing the incidence of laryngospasm during awake intubation in neonates (54).

Gulhas *et al.* described the use of magnesium to prevent laryngospasm after tonsillectomy and adenoidectomy in children. They gave $15 \text{ mg}\cdot\text{kg}^{-1}$ magnesium sulphate in 30 ml 0.9% NaCl over 20 min after tracheal intubation in 20 patients and found that the incidence of laryngospasm during tracheal extubation in this group was 0% compared with

25% in the control group ($P < 0.05$). They suggest that magnesium acts by both increasing anesthesia depth and providing muscle relaxation in preventing laryngospasm (41).

Few anesthesiologists in modern practice would use 5% carbon dioxide (CO_2) for preventing laryngospasm, assuming that it could be found on the anesthesia machine. This can be performed by inhaling the 5% CO_2 for 5 min prior to tracheal extubation. The mechanism is that the respiratory drive to exhale the carbon dioxide overrides the laryngospasm reflex. This was a study performed on cats and thus human studies are needed to prove its efficacy and safety (55).

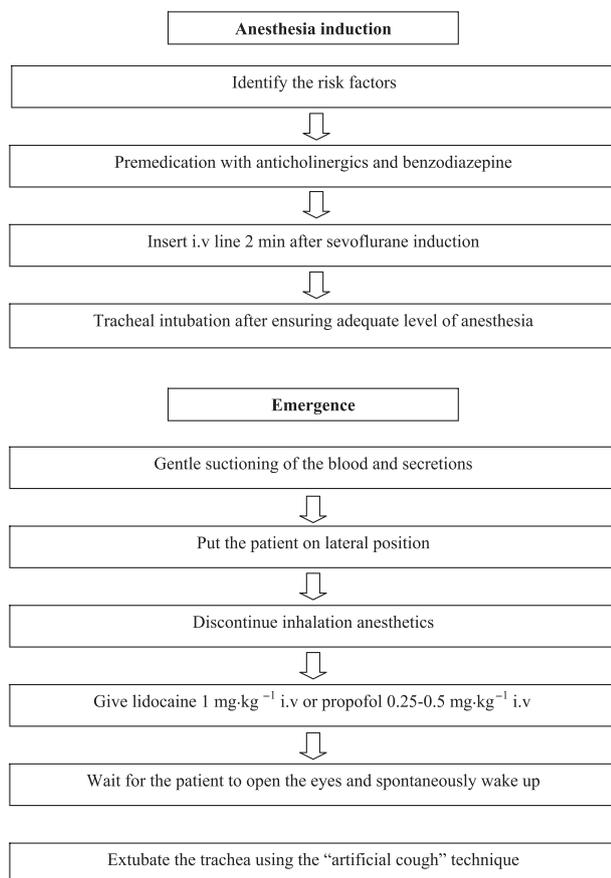
Finally, acupuncture has been described for the prevention of laryngospasm. The incidence of laryngospasm of was 5.3% in the study group compared with 23.7% in the control group ($P < 0.05$). This method although reducing the incidence of laryngospasm, it did not totally abolish it. In addition, the anesthesiologist would need to learn the proper techniques of acupuncture (19) (Table 1).

Treatment

For laryngospasm which occurs during anesthesia induction or emergence, the treatment is the same: identifying and removing the offending stimulus, applying jaw thrust maneuver, inserting an oral or nasal airway and positive pressure ventilation with 100% oxygen. If these techniques suffice to treat the spasm, partial laryngospasm is diagnosed. If the obstruction is not relieved, complete laryngospasm should be suspected and the next step should be calling for help and deepening the level of anesthesia with i.v. or inhalational anesthetic. Propofol can be used at doses $0.25\text{--}0.8 \text{ mg}\cdot\text{kg}^{-1}$ because of its rapid and predictable action, but if there is no i.v. line inhalational anesthesia can be used (29,56–59). If this technique fails and oxygen-hemoglobin desaturation ensues ($\text{SpO}_2 < 85\%$) suxamethonium can be given at doses of $0.1\text{--}3 \text{ mg}\cdot\text{kg}^{-1}$ followed by mask ventilation and, or tracheal intubation (60–62).

The use of propofol at $0.5 \text{ mg}\cdot\text{kg}^{-1}$ i.v. to treat laryngospasm has been shown to be safe and free of cardiovascular events, however, some patients may develop transient apnea which needs airway support and ventilation (29). The question of whether to

Table 1
A simplified algorithm for prevention of laryngospasm



use propofol or suxamethonium is a matter of timing. Propofol should be used prior to suxamethonium because it is successful in treating laryngospasm in 76.9% of cases (29,58,59). In addition, propofol offers many advantages over suxamethonium. First, is the lack of interaction of a depolarizing drug with a previously administered nondepolarizing muscle relaxant. Second, avoiding suxamethonium will eliminate the possibility of prolonged paralysis in patients with pseudocholinesterase deficiency. Finally, propofol can be used when suxamethonium is contraindicated, such as in patients with muscular dystrophy, recent burns, spinal cord transection or hyperkalemia (58,61). Suxamethonium still has a crucial role when propofol is unsuccessful (59) and its administration should not be delayed until the patient becomes severely desaturated ($\text{SpO}_2 < 85\%$). This is because

suxamethonium administration following hypoxia may be associated with severe bradycardia and even cardiac arrest. Thus, it is highly recommended to give atropine at $0.02 \text{ mg}\cdot\text{kg}^{-1}$ i.v. prior to administration of suxamethonium to treat laryngospasm (6,8,38).

In case of cardiac arrest following suxamethonium administration, tracheal intubation should be followed by ventilation with 100% oxygen and epinephrine should be given at $5\text{--}10 \mu\text{g}\cdot\text{kg}^{-1}$ i.v. in incremental doses until there is a response (61).

An intubating dose of suxamethonium ($1\text{--}3 \text{ mg}\cdot\text{kg}^{-1}$ i.v.) has been used to treat laryngospasm until a report by Chung and Rowbottom showed that the use of $0.1 \text{ mg}\cdot\text{kg}^{-1}$ i.v. of suxamethonium was successful in treating laryngospasm. The two advantages of this small dose of suxamethonium are the maintenance of spontaneous breathing thus avoiding further hypoxia and the avoidance of bradycardia following repeated doses (60).

When laryngospasm occurs during inhalational induction without previous i.v. access several options can be used. Many studies have shown that the intramuscular (i.m.) administration of a nondepolarizing muscle relaxant is not suitable for emergency intubation mainly because of slow absorption (63–66). Warner recommends that i.m. suxamethonium can be administered at $4 \text{ mg}\cdot\text{kg}^{-1}$ followed by tracheal intubation, claiming that maximum relaxation of i.m. suxamethonium that takes 3 or 4 min is not necessary to treat laryngospasm because the onset of neuromuscular blockade is more rapid in the larynx compared with peripheral muscles (67). However, Donati *et al.* (64) advise against the use of i.m. suxamethonium for intubation without i.v. access. They suggest establishing an i.v. access for the administration of drugs to treat laryngospasm. If this does not succeed, help should be sought to establish an i.v. access including the femoral vein. Weiss *et al.* (68) recommend using the intraosseous route as an efficient and quick access to give neuromuscular blocking drugs with faster central circulation times and better pharmacokinetics compared with a peripheral i.v. route.

For laryngospasm which occurs after tracheal extubation, Owen showed that infusing doxapram at $1.5 \text{ mg}\cdot\text{kg}^{-1}$ over 20 s to five patients rapidly abolished postextubation laryngospasm. He suggested that doxapram abolishes laryngospasm by

increasing the respiratory drive. However, the effect of doxapram was seen in only five patients and a study of large number of patients would be needed to confirm its efficacy and practicability (69).

Sibaii also, reported two cases of postextubation laryngospasm that were treated with i.v. nitroglycerin at $4 \text{ g}\cdot\text{kg}^{-1}$. However, whether laryngospasm was self-limited or was actually treated by nitroglycerin needs to be answered. It is well known that nitroglycerin relaxes the smooth muscles, but the laryngeal muscles are skeletal muscles and may not be a target of nitroglycerin action (17).

There is a technique which was first described 40 years ago by Guadagni and was later described by Larson. It involves placing the middle finger of each hand in what they term the 'laryngospasm notch' (Figure 1). This technique consists of firmly pressing inward toward the base of the skull with both fingers, while at the same time applying jaw thrust maneuver (27). This opens the airway and induces periosteal pain by pressing on the styloid process which helps relaxing the vocal cords by the autonomic nervous system (27,70).

Finally, Monso *et al.* reported three cases of postextubation laryngospasm that were successfully treated with superior laryngeal nerve block (Figure 2). Also, Mevorach described the successful use of superior laryngeal nerve block in treating postextubation laryngospasm that was refractory to medical treatment (11,71). The superior laryngeal

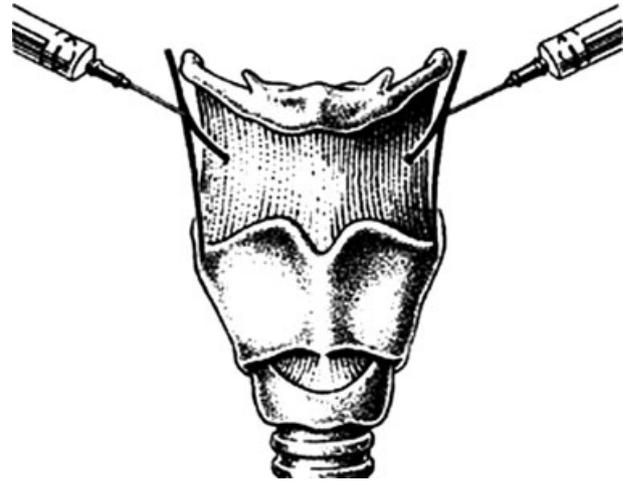


Figure 2
Superior laryngeal nerve block (71). (A 25 G needle is used to make contact with the hyoid bone, walking off the inferior margin of the bone and advancing 1–2 mm until it pierces the hyoid ligament).

nerve, a branch of vagus nerve, provides anesthesia for supraglottic mucosa via its internal branch. Blocking this nerve may be useful for both prevention and treatment of laryngospasm (71). These are case reports; hence a study of large number of patients is needed to confirm its efficacy (Table 2).

In conclusion, knowledge of the risk factors whether patient-related, anesthesia or surgery-related is the most important factor for prevention of laryngospasm. During tracheal intubation, the

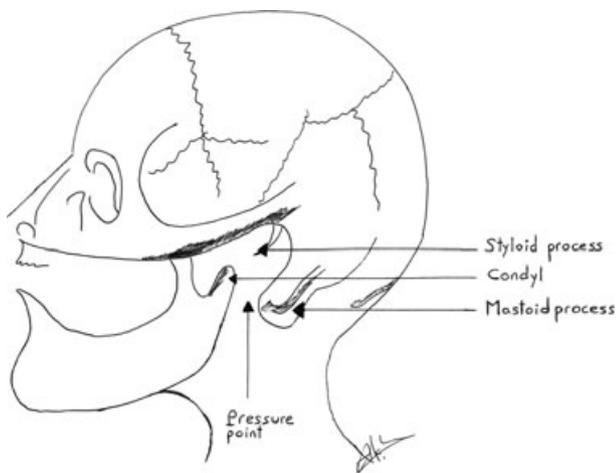


Figure 1
Laryngospasm notch (located behind the lobule of the pinna of the ear, bounded anteriorly by the ascending ramus of the mandible adjacent to the condyle, posteriorly by the mastoid process of the temporal bone and cephalad by the base of the skull).

Table 2
A simplified algorithm for treatment of laryngospasm

Identification and removal of the offending stimulus such as secretions, mucus or blood
↓
Inserting an 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 the level of anaesthesia by propofol i.v $0.25\text{--}0.8 \text{ mg}\cdot\text{kg}^{-1}$
↓
If laryngospasm is not relieved, inject suxamethonium i.v $0.1\text{--}3 \text{ mg}\cdot\text{kg}^{-1}$ or i.m $3\text{--}4 \text{ mg}\cdot\text{kg}^{-1}$ followed by mask ventilation and/or tracheal intubation

patient must be deeply anesthetized to avoid laryngospasm. There is still controversy whether awake including the 'no touch technique' or anesthetized tracheal extubation should be used in order to prevent laryngospasm. i.v. lidocaine or propofol administration before tracheal extubation may decrease the incidence of laryngospasm. Also, propofol can relieve laryngospasm in most of the patients. In case of failure, i.v. or i.m. suxamethonium can be used. Finally, the patients who have suffered laryngospasm should be assessed for the possibility of developing pulmonary aspiration or postobstructive negative pressure pulmonary edema.

Acknowledgements

We would like to thank Dr Maud Nawfal for contributing to the revision of the final manuscript and Dr Marie Aouad for helping us in the literature review.

References

- 1 Van der Walt J. Laryngospasm. In: Bissonnette B, Dalens B, eds. *Pediatric Anesthesia: Principles & Practice*. New York: McGraw Hill, 2002: 644.
- 2 Suzuki M, Sasaki CT. Laryngospasm: a neurophysiologic redefinition. *Anesthesia for eye, ear, nose and throat surgery*. In: Miller RD, ed. *Miller's Anesthesia*, 6th edn. Philadelphia: Elsevier Churchill Livingstone, 2005: 2538.
- 3 Fink BR. The etiology and treatment of laryngeal spasm. *Anesthesiology* 1956; **17**: 569–577.
- 4 Olsson GL, Hallen B. Laryngospasm during anaesthesia. A computer-aided incidence study in 136,929 patients. *Acta Anaesthesiol Scand* 1984; **28**: 567–575.
- 5 Green SM. Ketamine sedation for pediatric procedures: Part 2, review and implications. *Ann Emerg Med* 1990; **19**: 1033–1046.
- 6 Roy WL, Lerman J. Laryngospasm in paediatric anaesthesia. *Can J Anaesth* 1988; **35**: 93–98.
- 7 Da Silva PSL. Negative-pressure pulmonary edema. A rare complication of upper airway obstruction in children. *Pediatr Emerg Care* 2005; **21**: 751–754.
- 8 Visvanathan T, Kluger MT. Crisis management during anaesthesia: laryngospasm. *Qual Saf Health Care* 2005; **14**: e3.
- 9 Rajan GRC. Supraglottic obstruction versus true laryngospasm: the best treatment. *Anesthesiology* 1999; **91**: 581–582.
- 10 Burwell DR, Jones JG. The airway and anesthesia. In: Miller RD, ed. *Miller's Anesthesia*, 6th edn. Philadelphia: Elsevier Churchill Livingstone, 2005: 1618.
- 11 Mevorach DL. The management and treatment of recurrent postoperative laryngospasm. *Anesth Analg* 1996; **83**: 1110–1111.
- 12 Landsman IS. Mechanism and treatment of laryngospasm. *Int Anaesthesiol Clin* 1997; **3**: 67–73.
- 13 Anghelescu DL, Burgoyne LL. Intervention steps for treating laryngospasm in pediatric patients. *Pediatr Anesth* 2008; **18**: 297–302.
- 14 Miller R. Older pediatric patients. Pediatric anesthesia. In: Miller RD, ed. *Miller's Anesthesia*, 6th edn. Philadelphia: Elsevier Churchill Livingstone, 2005: 2382.
- 15 Golden SE. The management and treatment of recurrent postoperative laryngospasm. *Anesth Analg* 1997; **84**: 1389–1396.
- 16 Patterson D. The management and treatment of recurrent postoperative laryngospasm. *Anesth Analg* 1996; **83**: 1110–1111.
- 17 Sibai AN, Yamout I. Nitroglycerin relieves laryngospasm. *Acta Anaesthesiol Scand* 1999; **43**: 1081–1083.
- 18 Schreiner MS, O'Hara I, Markakis DA *et al*. Do children who experience laryngospasm have an increased risk of upper respiratory tract infection? *Anesthesiology* 1996; **85**: 475–480.
- 19 Lee CK, Chien TJ, Hsu JC *et al*. The effect of acupuncture on the incidence of postextubation laryngospasm in children. *Anaesthesia* 1998; **53**: 910–924.
- 20 Horita A, Dille JK. Observation on the action of thiopental on the laryngeal reflex. *Anesthesiology* 1955; **16**: 848–853.
- 21 Lakshmi N, Bokesch PM, Cowen DE *et al*. Environmental tobacco smoke: a risk factor for pediatric laryngospasm. *Anesth Analg* 1996; **82**: 724–727.
- 22 Heard KM. Benefits and hazards of pentothal anesthesia. *Anesthesiology* 1944; **5**: 448–464.
- 23 Mcglone RG, Howes MC, Joshi M. The Lancaster experience of 2.0 to 2.5 mg/kg intramuscular ketamine for paediatric sedation: 501 cases and analysis. *Emerg Med* 2004; **21**: 290–295.
- 24 Oberer C, von Ungern-Sternberg BS, Frei FJ *et al*. Respiratory reflexes responses of the larynx differ between sevoflurane and propofol in pediatric patients. *Anesthesiology* 2005; **103**: 1142–1148.
- 25 Doi M, Ikeda K. Airway irritation produced by volatile anaesthetics during brief inhalation: comparison of halothane, enflurane, isoflurane and sevoflurane. *Can J Anaesth* 1993; **40**: 122–126.
- 26 Fisher D, Robinson S. Comparison of enflurane, halothane and isoflurane for diagnostic and therapeutic procedures in children with malignancies. *Anesthesiology* 1985; **63**: 647–650.
- 27 Miller R. Sevoflurane. Pediatric anesthesia. In: Miller RD, ed. *Miller's Anesthesia*, 6th edn. Philadelphia: Elsevier Churchill Livingstone, 2005: 2373.
- 28 Larson PC. Laryngospasm—the best treatment. *Anesthesiology* 1998; **89**: 1293–1294.
- 29 Batra YK, Ivanova M, Ali SS *et al*. The efficacy of a subhypnotic dose of propofol in preventing laryngospasm following tonsillectomy and adenoidectomy in children. *Pediatr Anesth* 2005; **15**: 1094–1097.
- 30 Nandwani N, Raphael JH, Langton JA. Effects of upper respiratory tract infection on upper airway reactivity. *Br J Anaesth* 1997; **78**: 352–355.
- 31 Tait AR, Knight PR. Intraoperative respiratory complications in patients with upper respiratory tract infections. *Can J Anaesth* 1987; **34**: 300–303.
- 32 Levy L, Pandit UA, Randel GI *et al*. Upper respiratory tract infections and general anaesthesia in children. *Anaesthesia* 1992; **47**: 678–682.
- 33 Erskine RJ, Murphy PJ, Langton JA. Sensitivity of upper airway reflexes in cigarette smokers: effect of abstinence. *Br J Anaesth* 1994; **73**: 298–302.
- 34 Gallivan GJ, Hoffman L, Gallivan KH. Episodic paroxysmal laryngospasm: voice and pulmonary function assessment and management. *J Voice* 1996; **10**: 93–105.

- 35 Loughlin CJ, Koufman JA, Averill DB *et al.* Paroxysmal laryngospasm secondary to gastroesophageal reflux. *Laryngoscope* 1996; **106**: 1502–1505.
- 36 Aloe FS, Thorpy MJ. Sleep-related laryngospasm. *Arq Neuropsiquiatr* 1995; **53**: 46–52.
- 37 Shott SR, Cunningham MJ. Apnea and the elongated uvula. *Int J Pediatr Otorhinolaryngol* 1992; **24**: 183–189.
- 38 Hartley M, Vaughan RS. Problems associated with tracheal extubation. *Br J Anaesth* 1993; **71**: 561–568.
- 39 Koc C, Kocaman F, Aygenc E *et al.* The use of perioperative lidocaine to prevent stridor and laryngospasm after tonsillectomy and adenoidectomy. *Otolaryngol Head Neck Surg* 1998; **118**: 880–882.
- 40 Tsui BCH, Wagner A, Cave D *et al.* The incidence of laryngospasm with a “No Touch” extubation technique after tonsillectomy and adenoidectomy. *Anesth Analg* 2004; **98**: 327–329.
- 41 Gulhas N, Durmus N, Demirbilek S *et al.* The use of magnesium to prevent laryngospasm after tonsillectomy and adenoidectomy: a preliminary study. *Paediatr Anaesth* 2003; **13**: 43–47.
- 42 Baraka A. Intravenous lidocaine controls extubation laryngospasm in children. *Anesth Analg* 1978; **57**: 506–507.
- 43 Leicht P, Wisborg T, Chraemmer-Jorgensen B. Does intravenous lidocaine prevent laryngospasm after extubation in children? *Anesth Analg* 1985; **64**: 1193–1196.
- 44 Mortero RF, Orahovac Z, Tsueda k *et al.* Severe laryngospasm at tracheal extubation in a patient with superior laryngeal nerve injury. *Anesth Analg* 2001; **92**: 271–272.
- 45 Miller R. Thyroid surgery. Anesthesia for eye, ear, nose and throat surgery. In: Miller RD, ed. *Miller's Anesthesia*, 6th edn. Philadelphia: Elsevier Churchill Livingstone, 2005: 2540.
- 46 Bauman NM, Sandler AD, Schmidt C *et al.* Relex laryngospasm induced by stimulation of distal afferents. *Laryngoscope* 1994; **104**: 209–214.
- 47 Schwartz D, Connelly NR, Gutta S *et al.* Early intravenous cannulation in children during sevoflurane induction. *Pediatr Anesth* 2004; **14**: 820–824.
- 48 Patel RI, Hannallah RS, Norden J *et al.* Emergence airway complications in children: a comparison of tracheal extubation in awake and deeply anaesthetized patients. *Anesth Analg* 1991; **73**: 266–270.
- 49 Pounder DR, Blackstock MB, Steward DJ. Tracheal extubation in children: halothane versus isoflurane, anesthetized versus awake. *Anesthesiology* 1991; **74**: 653–655.
- 50 Lee KWT, Downes JJ. Pulmonary edema secondary to laryngospasm in children. *Anesthesiology* 1983; **59**: 347–349.
- 51 Rosen M. Atropine in the treatment of laryngeal spasm. *Br J Anaesth* 1960; **32**: 190–191.
- 52 Murphy PJ, Langton JA, Barker P *et al.* Effect of oral diazepam on the sensitivity of upper airway reflexes. *Br J Anaesth* 1993; **70**: 131–134.
- 53 Gilbertson AA. Laryngeal spasm. *Br J Anaesth* 1993; **71**: 168–169.
- 54 Barash PG. Neonatal anesthesia. *Clinical Anesthesia* 5th Edition. Philadelphia: Lippincott Williams & Wilkins, 2006: 1199.
- 55 Nishino T, Yonezawa T, Honda Y. Modification of laryngospasm in response to changes in PaCO₂ and PaO₂ in the cat. *Anesthesiology* 1981; **55**: 286–291.
- 56 Miller R. Laryngoscopy and intubation. Airway management. In: Miller RD, ed. *Miller's Anesthesia*, 6th edn. Philadelphia: Elsevier Churchill Livingstone, 2005: 1648.
- 57 Nawfal M, Baraka A. Propofol for relief of extubation laryngospasm. *Anaesthesia* 2002; **57**: 1028–1044.
- 58 Fodale V, Pratico C, Leto G *et al.* Propofol relieves post-extubation laryngospasm in obstetric anesthesia. *Int J Obstet anesth* 2004; **13**: 196–200.
- 59 Afshan G, Chohan U, Qamar-Ul-Hoda M *et al.* Is there a role of a small dose of propofol in the treatment of laryngeal spasm? *Paediatr Anaesth* 2002; **12**: 625–628.
- 60 Chung DC, Rowbottom SJ. A very small dose of suxamethonium relieves laryngospasm. *Anaesthesia* 1993; **48**: 229–230.
- 61 Miller R. Depolarizing muscle relaxants. Muscle relaxants. In: Miller RD, ed. *Miller's Anesthesia*, 6th edn. Philadelphia: Elsevier Churchill Livingstone, 2005: 486.
- 62 Miller R. Upper airway obstruction. Airway management. In: Miller RD, ed. *Miller's Anesthesia*, 6th edn. Philadelphia: Elsevier Churchill Livingstone, 2005: 1619.
- 63 Denman WT, Kaplan RF, Goudsouzian NG *et al.* Intramuscular rapacuronium in infants and children. *Anesthesiology* 2001; **94**: 3–7.
- 64 Donati F, Guay J. No substitute for the intravenous route. *Anesthesiology* 2001; **94**: 1–2.
- 65 Reynolds LM, Lau M, Brown R *et al.* Intramuscular rocuronium in infants and children: dose ranging and tracheal intubating conditions. *Anesthesiology* 1996; **86**: 231–239.
- 66 Cauldwell CB, Lau M, Fisher DM. Is intramuscular mivacurium an alternative to succinylcholine? *Anesthesiology* 1994; **80**: 320–325.
- 67 Warner DO. Intramuscular succinylcholine and laryngospasm. *Anesthesiology* 2001; **95**: 1039–1040.
- 68 Weiss MM, Gerber A. The substitute for the intravenous route. *Anesthesiology* 2001; **95**: 1040–1041.
- 69 Owen H. Postextubation laryngospasm abolished by doxapram. *Anaesthesia* 1982; **37**: 1112–1114.
- 70 Johnstone RE. Laryngospasm treatment—an explanation. *Anesthesiology* 1999; **91**: 581–582.
- 71 Monso A, Riudebas J, Palanques F *et al.* A new application for superior laryngeal nerve block: treatment or prevention of laryngospasm and stridor. *Reg Anesth Pain Med* 1999; **24**: 186–193.

Accepted 24 September 2007