Bruno Riou, M.D., Ph.D., Editor

Case Scenario: Perianesthetic Management of Laryngospasm in Children

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PERIOPERATIVE laryngospasm is an anesthetic emergency that is still responsible for significant morbidity and mortality in pediatric patients.[1] It is a relatively frequent complication that occurs with varying frequency dependent on multiple factors.[2–5] Once the diagnosis has been made, the main goals are identifying and removing the offending stimulus, applying airway maneuvers to open the airway, and administering anesthetic agents if the obstruction is not relieved. The purpose of this case scenario is to highlight key points essential for the prevention, diagnosis, and treatment of laryngospasm occurring during anesthesia.

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Case Report

A 10-month-old boy (8.5 kg body weight) was taken to the operating room (at 11:00 PM), without premedication, for emergency surgery of an abscess of the second fingertip on the right hand. Past medical history was unremarkable except for an episode of upper respiratory tract infection 4 weeks ago. The mother volunteered that he was exposed to passive smoking in the home. He had been fasting for the past 6 h. Preoperative evaluation was normal (systemic blood pressure 85/50 mmHg, heart rate 115 beats/min, pulse oximetry [Sp_{O_2}] 99% on room air). The procedure was expected to be very short, and general anesthesia with inhalational induction and maintenance, but without tracheal intubation, was planned. The child was placed over a forced air warmer (Bear Hugger™, Augustine Medical, Inc., Eden Prairie, MN). Anesthesia was induced by a resident under the direct supervision of a senior anesthesiologist with inhaled sevoflurane in a 50/50% (5 l/min) mixture of oxygen and nitrous oxide. Two min after loss of eyelash reflex, a first episode of airway obstruction with inspiratory stridor and suprasternal retraction was successfully managed by jaw thrust and manual positive pressure ventilation. An IV line was obtained at 11:15 PM, while the child was manually ventilated. Anesthesia was then maintained by facemask with 2.0% expired sevoflurane in a mixture of oxygen and nitrous oxide 50/50%. Sufentanil (1 mcg) was given intravenously and the surgeon was allowed to proceed 5 min later. At 11:23 PM, an inspiratory stridulous noise was noted again. Manual facemask ventilation became difficult with an increased resistance to insufflation and Sp_{O_2}

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Table 1. Risk Factors Associated with Perioperative Laryngospasm

Personal history	
	Male
	Upper respiratory tract infection present the day of surgery or within the past 2 weeks
	Wheezing at exercise or more than three times in past 12 months
	Nocturnal dry cough
	Eczema present or in the past 12 months
Family history	
	History of at least two family members having asthma, atopy (rhinitis, eczema), or smoking

Adapted from von Ungern-Sternberg BS, Boda K, Chambers NA, Rebmann C, Johnson C, Sly PD, Habre W: Risk assessment for respiratory complications in paediatric anaesthesia: A prospective cohort study. Lancet 2010; 376:773–83.

dropped rapidly from 98% to 78%, associated with a decrease in heart rate from 115 to 65 beats/min. A new episode of laryngospasm was immediately suspected. Despite a jaw thrust maneuver, positive pressure ventilation with 100% O_2, and administration of two bolus doses (5 mg) of IV propofol (0.6 mg/kg), the obstruction was not relieved and Sp_{O_2} decreased to 52%. A 0.2-mg IV bolus dose of atropine was injected and IV succinylcholine was given at a dose of 16 mg, followed by tracheal intubation. Thereafter, surgery was quickly completed, while tracheal extubation and postoperative recovery were uneventful.

Epidemiology of Laryngospasm in Pediatric Patients

Children are more prone to laryngospasm than adults, with laryngospasm being reported more commonly in children (17.4/1,000) than in the general population (8.7/1,000).[2,5–7] In fact, the incidence of laryngospasm has been found to range from 1/1,000 up to 20/100 in high-risk surgery (*i.e.*, otolaryngology surgery).[2,5–7] Many factors may increase the risk of laryngospasm. These risk factors can be patient-, procedure-, and anesthesia-related (table 1).

Patient-related Factors

Age. Young age is one of the most important risk factors. In the largest study published in the literature (n = 136,929 adults and children), the incidence of laryngospasm was 1.7% in 0–9 yr-old children and only 0.9% in older children and adults.[7] The highest incidence (more than 2%) was found in preschool age groups. In a more recent series, the overall incidence of laryngospasm was lower[8] but the predominance of such incidents at a young age was still clear: 50 to 68% of cases occurred in children younger than 5 yr. In reports addressing respiratory adverse events, including laryngospasm, the overall incidence of perioperative respiratory events as well as the incidence of laryngospasm was higher in 0–1-yr-old infants in comparison with older children.[2,5–7] The risk of perioperative respiratory adverse event was quoted as decreasing by 8% for each increasing year of age.[2] A recent large cohort study confirmed this inverse relationship between age and risk of perioperative respiratory adverse events.[5] This study showed that the relative risk for perioperative respiratory adverse events, particularly laryngospasm, decreased by 11% for each yearly increase in age.[5]

Upper Respiratory Tract Infection. Upper respiratory tract infection (URI) is associated with a twofold to fivefold increase in the risk of laryngospasm.[5,9] Anesthesiologists in charge of pediatric patients should be aware that the risks associated with a URI in an infant are magnified in this age group, especially in those with respiratory syncytial virus infection.[10] Children with URI are prone to develop airway (upper and bronchial) hyperactivity that lasts beyond the period of viral infection. Whereas epithelial damage heals in 1–2 weeks, virus-induced sensitization of bronchial autonomic efferent pathways can last for up to 6–8 weeks. Whether or not this is relevant to perioperative risk of laryngospasm has been questioned many times in the literature.[9,11] Von Ungern-Sternberg *et al.* have demonstrated an increased risk for laryngospasm only when cold symptoms are present the day of surgery or less than 2 weeks before (table 2).[5] Therefore, for children who present for elective procedures with a temperature higher than 38°C, mucopurulent airway secretions, or lower respiratory tract signs such as wheezing and moist cough, surgery is usually postponed.

Smoke Exposure. Household exposure to tobacco smoke was shown to increase the incidence of laryngospasm from

Table 2. Relative Risk (95% CI) of Laryngospasm in Children According to the Presence of Cold Symptoms

	Present	<2 Weeks	2–4 Weeks
Clear runny nose	1.98 (1.48–2.69; $P < 0.0001$)	2.04 (1.45–2.87; $P < 0.0001$)	1.16 (0.65–1.94; $P = 0.67$)
Green runny nose	4.40 (2.97–6.52; $P < 0.0001$)	6.62 (4.80–9.12; $P < 0.0001$)	0.09 (0.01–0.63; $P = 0.015$)
Dry cough	2.16 (1.50–3.10; $P < 0.0001$)	2.14 (1.38–3.30; $P = 0.001$)	0.53 (0.22–1.27; $P = 0.16$)
Moist cough	3.89 (2.89–5.23; $P < 0.0001$)	6.53 (5.01–8.53; $P < 0.0001$)	0.08 (0.01–0.58; $P = 0.012$)
Fever	2.34 (1.14–4.80; $P = 0.020$)	5.28 (3.47–8.02; $P < 0.0001$)	0.57 (0.22–1.51; $P = 0.26$)

Adapted from von Ungern-Sternberg BS, Boda K, Chambers NA, Rebmann C, Johnson C, Sly PD, Habre W: Risk assessment for respiratory complications in paediatric anaesthesia: A prospective cohort study. Lancet 2010; 376:773–83.

0.9% to 9.4% in children scheduled for otolaryngology and urologic surgery.[12] This strong association between passive exposure to tobacco smoke and airway complications in children was also observed in another large study.[13]

Procedure-related Risk Factors

The highest incidence of laryngospasm is found in procedures involving surgery and manipulations of the pharynx and larynx.[2,5–7] The incidence of laryngospasm, after tracheal extubation, has already been reported to exceed 20% and be as high as 26.5% in pediatric patients who have undergone tonsillectomy.[14–17] Urgent procedures also carry a higher risk of laryngospasm than elective procedures. In the study by von Ungern-Sternberg *et al.*,[5] emergent procedures had a moderately higher risk than elective procedures for perioperative respiratory adverse events, including laryngospasm (17% *vs.* 14%, relative risk 1.2, 95% CI 1.1–1.3; $P = 0.001$).

Anesthesia-related Risk Factors

Insufficient depth of anesthesia is one of the major causes of laryngospasm. Any stimulation in the area supplied by the superior laryngeal nerve, during a light plane of anesthesia, may produce laryngospasm. Common triggers of reflex laryngeal response during anesthesia are secretions, blood, insertion of an oropharyngeal airway suction catheter, and laryngoscopy. Inexperience of the anesthetist is also associated with an increased incidence of laryngospasm and perioperative respiratory adverse events.[2,5,18] Some factors are associated with a lower risk of laryngospasm: IV induction, airway management with facemask, and inhalational maintenance of anesthesia.[5] Induction and emergence from anesthesia are the most critical periods. However, some authors have observed that emergence from anesthesia tends to become the most critical period, possibly in relation to changes in practice including the use of laryngeal mask airway (LMA) and/or of propofol and newer inhalational agents.[8]

Morbidity Associated with Laryngospasm

Laryngospasm can result in life-threatening complications, including severe hypoxia, bradycardia, negative pressure pulmonary edema, and cardiac arrest. Laryngospasm remains the leading cause of perioperative cardiac arrest from respiratory origin in children.[1]

Pathophysiology of Laryngospasm in Children

The Upper Airway Reflexes. The upper airway has several functions (swallowing, breathing, and phonation) but protection of the airway from any foreign material is the most essential. This function involves several upper airway reflexes: the laryngeal closure reflex, which consists of vocal fold adduction; apnea; swallowing; and coughing.[19] To efficiently protect the airway, laryngeal closure reflex must be coordinated with swallowing. Both reflexes are sometimes considered as a single phylogenetic reflex.[20] The neuronal pathways underlying upper airway reflexes include an afferent pathway, a common central integration network, and an efferent pathway.[19]

Afferent Pathway. The locations of involved nerve receptors vary as a function of the upper airway reflex: pharyngeal mucosa for the swallowing reflex, supraglottic larynx for laryngeal closure reflex,[19] larynx and trachea for cough, and any part of the upper airway (but mainly nose and larynx) for apnea.

For laryngeal closure reflex, several types of receptors can be distinguished, according to their specific sensitivities to cold, pressure, laryngeal motion, and chemical agents.[19,21] The chemoreceptors are sensitive to fluids with low chloride or high potassium concentrations, as well as to strong acidic or alkaline solutions.[19,21]

The afferent nerves include the trigeminal nerve for the nasopharynx, the glossopharyngeal nerve for the oropharynx and hypopharynx, the superior and recurrent laryngeal nerves, and both branches of the vagus nerve, for the larynx and trachea. The afferent nerve involved in laryngeal closure reflex is the superior laryngeal nerve.

Common Central Integration Network. Afferent nerves converge in the brainstem nucleus tractus solitarius. The brainstem nucleus tractus solitarius is not only an afferent portal, but has interneurons that play an essential role in the genesis of upper airway reflexes.[19] Little is known about the centers that regulate and program these reflexes. They are most likely located in the medullary neuronal network rather than in the brainstem.[22–23] The higher center seems to regulate upper airway reflexes. For instance, coughing can be voluntarily inhibited.

Efferent Pathway

Principal effectors are respiratory muscles (diaphragm, intercostals, abdominals, and upper airway). More specifically, laryngeal closure reflex involves the laryngeal intrinsic muscles responsible for vocal folds adduction, *i.e.*, the lateral cricoarytenoid, thyroarytenoid, and cricothyroid muscles. Their motoneurons are located in the brainstem nucleus ambiguous and the adjacent nucleus retroambigualis. Stimulation of upper airway mucosa also produces cardiovascular (alterations of the arterial pressure, bradycardia, *etc.*) and bronchomotor reflexes, indicating that not only skeletal but also smooth muscles are involved in upper airway reflexes.[19]

Pathologic Alterations of Upper Airway Reflexes

Alterations of upper airway reflexes may occur in several conditions.

Depressed Upper Airway Defensive Reflexes with Bronchopulmonary Aspiration. This situation creates a risk of bronchopulmonary infection, chronic cough, and bronchospasm. It occurs during general or local anesthesia, natural sleep (rapid eye movement phase of sleep), hypercapnia, and hypoxia, as well as various muscular, neuromuscular junction, or peripheral nerves disorders affecting the efferent neural pathway and effector organs of upper airway reflexes.[19]

Laryngospasm. This condition arises as a result of an exaggerated and prolonged laryngeal closure reflex that can be triggered by mechanical (manipulation of pharynx or larynx) or chemical stimuli (*e.g.*, gastric acid).[24] They (mechanical and chemical stimuli) are favored by local inflammation with subsequent alteration of pharyngolaryngeal sensation (URI, gastroesophageal reflux disease, neurologic disorders)[20,25–26]; and factors influencing the central regulation system of upper airway reflexes, such as age.[20–21]

Apnea. After stimulation of the superior laryngeal nerve, apnea may result from several mechanisms: prolonged laryngeal closure reflex-related laryngeal obstruction (see the previously mentioned risk factors for increased laryngeal closure reflex); decreased swallowing reflex with accumulation of secretions in contact with the larynx vestibule and subsequent laryngeal closure reflex;[21,27] and centrally controlled apneic reflex possibly related to the "diving reflex" observed in aquatic mammals and aimed at preventing fluid aspiration in the lower airway. The apneic reflex varies as a function of age. It is frequently observed in fetuses and newborns, whereas later on, laryngeal closure reflex and cough become predominant.[21] This developmental pattern may be implicated in sudden infant death. Among all upper airway reflexes, it is the most resistant to deepening anesthesia, whereas the coughing reflex is the most sensitive. It persists for a longer period in the context of respiratory syncytial virus infection, hypoxia, and anemia.[21]

Diagnosis of Laryngospasm in Children

The diagnosis of laryngospasm depends on the clinical judgment of the anesthesiologist. Laryngospasm is usually defined as partial or complete airway obstruction associated with increasing abdominal and chest wall efforts to breathe against a closed glottis.[3,5,7] In both partial and complete laryngospasm, signs of varying degrees of airway obstruction, such as suprasternal retraction, supraclavicular retractions, tracheal tug, paradoxical chest, and abdominal movements may be seen.[3] In addition, inspiratory stridor may be heard in partial laryngospasm but is absent in complete spasm. In addition, in complete laryngospasm, there is no air movement, no breath sounds, absence of movement of the reservoir bag, and flat capnogram.[3] Finally, late clinical signs occur if the obstruction is not relieved including oxygen desaturation, bradycardia, and cyanosis.[3]

Prevention of Laryngospasm

Identifying the risk factors and planning appropriate anesthetic management is a rational approach to reduce laryngospasm incidence and severity.

Preoperative Management

A detailed history should be taken to identify the risk factors. For children with URI, cancellation of elective procedures for a period of 4–6 weeks was traditionally the rule. However, children younger than 3 yr may develop 5–10 URI episodes per year. Thus, the potential window for safe administration of general anesthesia is frequently very short. Von Ungern-Sternberg *et al.* have demonstrated an increased risk for laryngospasm only when cold symptoms were present on the day of surgery or less than 2 weeks before.[28] This finding was recently confirmed by the same team in an extensive study involving 9,297 surgical procedures.[5] Rescheduling patient 2–3 weeks after an URI episode appears to be a safe approach. Such a conservative attitude has already been proposed for otolaryngology patients, whose surgery is expected to have an effect on the recurrence of URI episodes.[11] Premedication with anticholinergic agents may decrease secretions but has no demonstrated influence on the incidence of laryngospasm.[7,29]

Anesthesia Plan

Airway Management. Manipulation of the airway at an insufficient depth of anesthesia is a major cause of laryngospasm. In children with URI, the use of an endotracheal tube (ETT) may increase by 11-fold the risk of respiratory adverse events, in comparison with a facemask.[11] Less invasive airway management could be beneficial in children with airway hyperactivity. Prospective studies supported the use of LMA over ETT in children with URI.[30–31] However, these studies were underpowered to detect differences in laryngospasm. In contrast, results from studies in children with recent URIs have shown that LMA was associated with an increased occurrence of laryngospasm.[28,32] In a recent, large, prospective study, the incidence of laryngospasm was increased after direct stimulation of the upper airway by both LMA and ETT in comparison with a facemask.[5] Therefore, LMA may be considered more stimulating than the facemask but certainly less than the ETT.

Induction Phase. There is controversy in the literature regarding the use of inhalational or IV induction agents and associated risk of laryngospasm. Only sevoflurane or halothane should be used for inhalational induction. Sufficient depth of anesthesia must be achieved before direct airway stimulation is initiated (oropharyngeal airway insertion). IV line insertion should also be delayed until deep anesthesia (regular ventilation with large tidal volume, eyeballs fixed with pupils centered in myosis or moderately dilated) is achieved. It may be difficult for a nonspecialist pediatric anesthesiologist to adequately manage an inhalational induction, because of the possibility to fail to manage the airway properly or the inability to recognize and treat early a stridor/laryngospasm. These are the reasons why inhalational induction conducted by nonspecialized anesthetists remains associated with an increased risk of laryngospasm.[2,5,18] In children with hyperactive airways, there are now several arguments in favor of IV induction with propofol *versus* inhalational induction. Experimentally, Oberer *et al.* demonstrated that in children age 2–6 yr, laryngeal and respiratory reflex responses differed between sevoflurane and propofol at similar depths of anesthesia, with apnea and laryngospasm being less severe with propofol.[33] If tracheal intubation is planned, the use of muscle relaxants prevents the risk of laryngospasm.[2] In contrast, topical anesthesia is probably not effective and the incidence of laryngospasm is even higher when vocal cords are sprayed with aerosolized lidocaine.[5]

Maintenance Phase. Laryngospasm is commonly caused by systemic painful stimulation if the anesthesia is too light during maintenance. Evidence on this subject is scarce, but the study by von Ungern-Sternberg *et al.* suggests that maintenance with sevoflurane was associated with a higher incidence of laryngospasm compared with propofol (relative risk 2.37, 95% CI 1.49–3.76; $P < 0.0001$).[5] In our case, the second episode of laryngospasm occurred while the patient was under light anesthesia. In fact, when the inspiratory stridulous noise was noted again, the patient was receiving 2% end-tidal sevoflurane and 50% N_2O, representing barely 1 minimum alveolar concentration in an infant. The use of desflurane during maintenance of anesthesia appeared to be associated with a significant increase in perioperative respiratory adverse events, including laryngospasm, compared with sevoflurane and isoflurane.[5] Isoflurane appeared to produce laryngeal effects similar to sevoflurane.[5]

Emergence. It is still debated whether tracheal extubation should be performed in awake or deeply anesthetized children to decrease laryngospasm. Several studies suggest that deep extubation reduces this incidence, whereas others observed no difference.[5,34–35] In one study, tracheal intubation with deep extubation was associated with increased respiratory adverse events rate (odds ratio = 2.39) compared with LMA removal at a deep level of anesthesia, whereas use of a facemask alone decreased respiratory adverse events (odds ratio = 0.15).[35] The difference between LMA and ETT was less evident when awake extubation was used (odds ratio = 0.65 and 1.26, respectively). In the study by von Ungern-Sternberg *et al.*, the overall incidence of respiratory adverse events seems to be higher in children who were awake when their LMA was removed and lower in those who were awake when their endotracheal tube was removed.[5] In summary, evidence seems to favor deep LMA and awake ETT removal.

In children, an "artificial cough maneuver," including a single lung inflation maneuver with 100% O_2 immediately before removal of the ETT, is useful at the time of extubation because it delays or prevents desaturation in the first 5 min after extubation in comparison with a suctioning procedure.[36] Although not demonstrated in this study, this technique could reduce laryngospasm because when the endotracheal tube leaves the trachea, the air escapes in a forceful expiration that removes residual secretions from the larynx. Usually, laryngospasm resolves and the patient recovers quickly without any sequelae. Rarely, negative pressure pulmonary edema may occur and requires specific treatment.[37] The high chest wall to lung compliance ratio observed during infancy, which disappears by the second year of life because of increased chest wall stiffness, may explain why negative pressure pulmonary edema is less frequent in infants than in older children or adults. Postoperative negative pressure pulmonary edema typically occurs in response to an upper airway obstruction, where patients can generate high negative intrathoracic pressures, leading to a postrelease pulmonary edema. This topic is beyond the scope of this article but was recently described elsewhere.[37] Eighty percent of negative pressure pulmonary edema cases occur within min after relief of the upper airway obstruction, but delayed onset is possible with cases reported up to 4–6 h later. This means that if nothing has occurred 4–6 h after the occurrence of a laryngospasm it is likely that the course will be uneventful.

Treatment of Laryngospasm

Effective management of laryngospasm in children requires appropriate diagnosis,[4] followed by prompt and aggressive management.[8] Many authors recommend applying airway manipulation first, beginning with removal of the irritant stimulus[38] and then administering pharmacologic agents if necessary.[8]

Airway Manipulation

Many methods and techniques of airway manipulation have been proposed. These interventions include removal of the irritant stimulus,[8,38] chin lift, jaw thrust,[39] continuous positive airway pressure (CPAP), and positive pressure ventilation with a facemask and 100% O_2.[3,40–43] These maneuvers are popular because they have been shown to improve the patency of the upper airway in case of airway obstruction.[42,44–45] Less commonly used airway maneuvers, such as pressure in the "laryngospasm notch"[4,44] and digital elevation of the tongue[46] also have been proposed as rapid and effective methods.[8] Overall conflicting results have been obtained regarding the best maneuver to relieve airway obstruction in children with laryngospasm. Some advocate delivery of jaw thrust and CPAP as the first airway opening maneuvers to improve breathing patterns in children with airway obstruction.[42] For others, both chin lift and jaw thrust maneuvers combined with CPAP improve the view of the glottic opening and decrease stridor in anesthetized, spontaneously breathing children.[41] It is likely that if the jaw thrust maneuver is properly applied, *i.e.*, at the condyles of the ascending rami of the mandible, then its efficacy would be improved. On the other hand, attempts to provide positive-pressure ventilation with a facemask may distend the stomach, increasing the risk of gastric regurgitation. If positive-pressure ventilation is to be performed, then moderate intermittent pressure should be applied. Recently, a new technique with gentle chest compression has been proposed as an alternative to standard practice for relief of laryngospasm.[47] In this before-after study, extubation laryngospasm was managed with "standard practice" (CPAP and gentle positive pressure ventilation *via* a tight-fitting facemask with 100% O_2 *via* facemask) during the first 2 yr of the study, whereas in the following 2 yr, laryngospasm was managed with 100% O_2 and concurrent gentle chest compression. More children who developed laryngospasm were successfully treated with chest compression (73.9%) compared with those managed with the standard method (38.4%; $P < 0.001$). None of the children in the chest compression group developed gastric distension (86.5% in the standard group). These preliminary results are interesting and need to be confirmed by further studies.

It should be noted that hypoxia ultimately relaxes the vocal cords and permits positive pressure ventilation to proceed easily. However, waiting until hypoxia opens the airway is not recommended, because a postobstruction pulmonary edema or even cardiac arrest may occur.[43]

The next step in management depends on whether laryngospasm is partial or complete and if it can be relieved or not. If complete laryngospasm cannot be rapidly relieved, IV agents should be quickly considered.

Pharmacologic Agents

Propofol. Propofol depresses laryngeal reflexes[33,48] and is therefore widely used to treat laryngospasm in children.[3,49] A study has assessed the effectiveness of a small bolus dose of propofol (0.8 mg/kg) for treatment of laryngospasm when 100% O_2 with gentle positive pressure had failed.[49] In this study, propofol was administered if laryngospasm occurred after LMA removal and if it persisted with a decrease in Sp_{O_2} to 85% despite 100% O_2 with gentle positive pressure ventilation.[49] The injection of propofol was able to relieve spasm in 76.9% of patients, whereas the remaining patients required administration of succinylcholine and tracheal intubation.[49] The success rate of propofol observed in this study is superior to the chest compression technique mentioned previously. These results are in accordance with a study showing that subhypnotic doses of propofol (0.5 mg/kg) decreased the likelihood of laryngospasm upon tracheal extubation in children undergoing tonsillectomy with or without adenoidectomy.[50] Lower doses of propofol (0.25 mg/kg) have also been used successfully to relax the larynx in a small series.[51] It should be noted that few data are available regarding the use of propofol to treat laryngospasm in younger age groups (younger than 3 yr). Furthermore, the efficacy of propofol to break complete laryngospasm when bradycardia is present has been questioned.[4] In our case, two bolus doses of 5 mg IV propofol (each representing a dose of 0.6 mg/kg) were administered but did not relieve airway obstruction. Therefore, the injection of IV succinylcholine was required to treat this persistent laryngospasm. Although the efficacy of subhypnotic doses of propofol has been suggested in children, there is a possibility that these doses are inadequate in infants, especially in those younger than 1 yr.

Muscle Relaxant. Muscle relaxants are usually administered when initial steps of laryngospasm treatment have failed to relax the vocal cords. This situation has been found to occur in approximately 50% of patients.[8] The most commonly used muscle relaxant is succinylcholine, but other agents have also been used, including rocuronium and mivacurium.[8] However, succinylcholine remains the gold standard.[4] Some authors have suggested the use of a small dose of succinylcholine (0.1 mg/kg) but there is a lack of dose-response study because the study included only three patients.[52] Therefore, we recommend using IV doses of succinylcholine no less than 0.5 mg/kg. If IV access cannot be established in emergency, succinylcholine may be given by an alternative route.[53–54] Intramuscular succinylcholine has been recommended at doses ranging from 1.5 to 4 mg/kg.[53] The main drawback of intramuscular administration is the slow onset in comparison with the IV route. However, onset time to effective relief of laryngospasm is shorter than onset time to maximal twitch depression, enabling laryngospasm relief and oxygenation (within 60 s) in less time than time to maximum twitch depression.[55] Therefore, intramuscular succinylcholine is the best alternative approach if IV access is not readily available.[56] Another alternative for succinylcholine administration is the intraosseous route. Experimental evidences and anecdotal reports indicate that intraosseous and IV injection behave similarly, resulting in adequate intubating conditions within 45 s (1 mg/kg).[57] In children in whom succinylcholine is contraindicated, rocuronium administered at a dose of two to three times the ED_{95} (0.9 to 1.2 mg/kg) may represent a reasonable substitute when rapid onset is needed.[58–60] In addition, there is a possibility to quickly reverse the neuromuscular blockade induced by rocuronium using sugammadex if necessary.[61]

The question of whether using propofol or muscle relaxant first is a matter of timing. The final decision depends on the severity of the laryngospasm (*i.e.*, partial or complete) and of the bradycardia as well as the existence of contraindication to succinylcholine.

Lidocaine. The efficacy of lidocaine to either prevent or control extubation laryngospasm has been studied since the late 1970s.[62] Some articles have confirmed the efficacy of lidocaine for preventing postextubation laryngospasm, whereas others have found the opposite results to be true.[16,63–65] A recent, well-conducted, randomized placebo-controlled trial in children undergoing cleft palate surgery demonstrated the effectiveness of IV lidocaine (1.5 mg/kg administered 2 min after tracheal extubation) in reducing laryngospasm and coughing (by 29.9% and 18.92%, respectively).[64] However, these favorable results were not confirmed in other studies.[5,65] The role of lidocaine (IV or topical) in preventing laryngospasm is still controversial. We decided to omit it in the preventive and/or treatment algorithms of laryngospasm, although other authors have included it.[3,8,66]

Other Agents. Other pharmacologic agents have been proposed for the prevention and/or treatment of laryngospasm, including magnesium,[17] doxapram,[67] diazepam,[68] and nitroglycerine.[69] However, because of the small number of

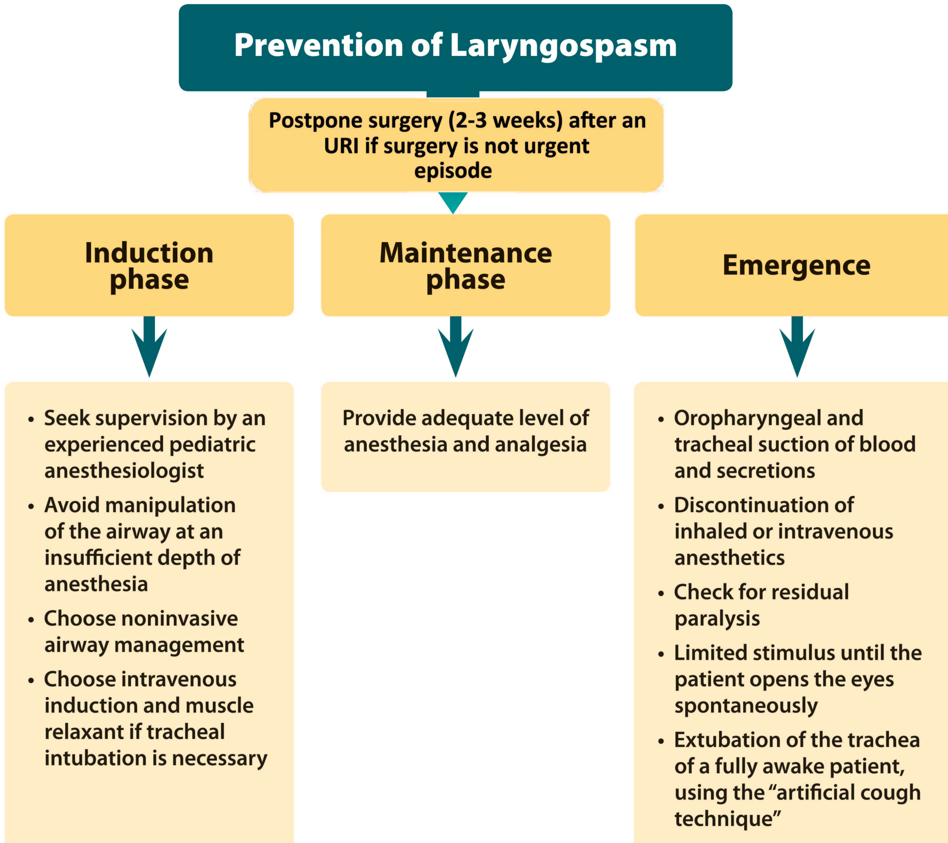

Fig. 1. Prevention of laryngospasm. URI = upper respiratory tract infection.

patients included in these series no firm conclusions can be drawn.

Suxamethonium injection in a hypoxic patient may lead to severe bradycardia and even to cardiac arrest. Therefore, giving IV atropine before IV injection of suxamethonium to treat laryngospasm is mandatory.[66]

Algorithms for Prevention and Treatment of Laryngospasm

To avoid significant morbidity and mortality, the use of a structured algorithm has been proposed.[8,70] One study suggests that if correctly applied, a combined core algorithm recommended for the diagnosis and management of laryngospasm would have led to earlier recognition and/or better management in 16% of the cases.[70] These results should encourage physicians to implement their own structured algorithm for the diagnosis and management of laryngospasm in children in their institutions. A recent retrospective study has assessed the incidence of laryngospasm in a large population and characterized the interventions used to treat these episodes.[8] The results have shown that treatment followed a basic algorithm including CPAP, deepening of anesthesia, muscle relaxation, and tracheal intubation.

The first step of laryngospasm management is prevention. Identifying patients at increased risk for laryngospasm and taking recommended precautions are the most important measures to prevent laryngospasm (fig. 1).[3] The second step relies on the emergent treatment of established laryngospasm occurring despite precautions (fig. 2).

How Can We Improve Education and Training?

The Challenge. Laryngospasm is one of the many critical situations that any anesthesiologist should be able to manage efficiently. Like any other crisis, such management requires the application of appropriate knowledge, technical skills, and teamwork skills (or nontechnical skills). However, the acquisition and the mastering of these skills during specialty training and their maintenance during continuing medical education represent a formidable challenge. For the management of laryngospasm in children, this task is complicated by two facts. First, the introduction of working hour limitations in virtually all Western countries has decreased the number of pediatric cases performed by trainees.[71] Second, most anesthetics given to children are administered by nonspecialists whose lack of experience and inability to maintain their skill set for children is a problem.

Educational Solutions. A competence-based training that includes a structured curriculum and regular workplace-based assessment may help mitigate the effects of caseload reduction. Realistic training with high-fidelity mannequins and other types of simulations represent unique educational tools that can be fully integrated in a residency program

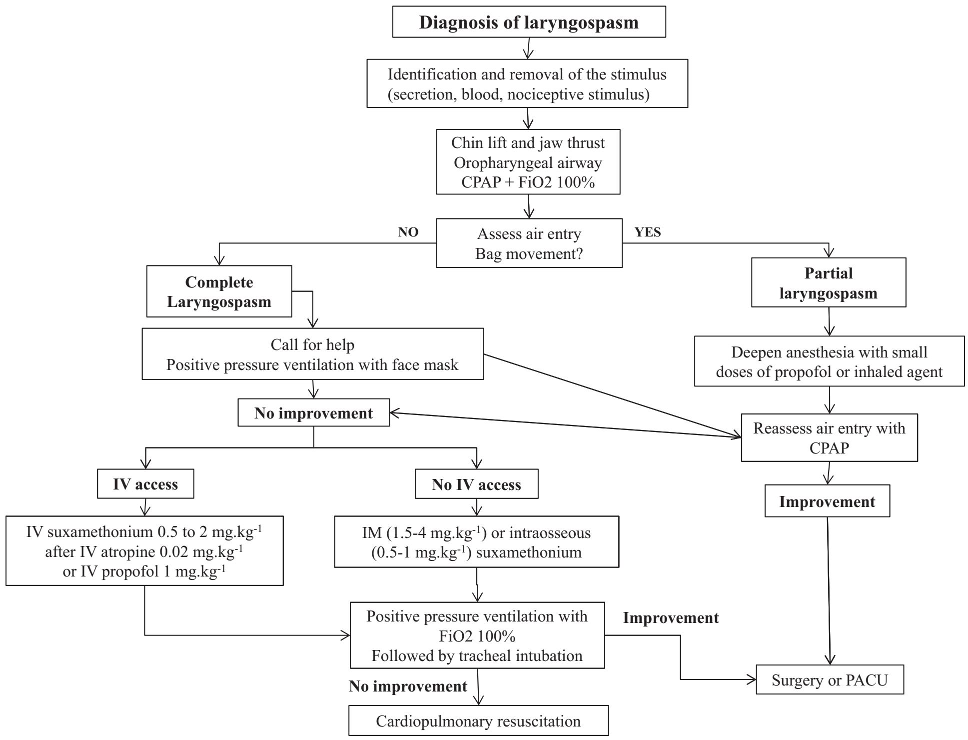

Fig. 2. Treatment of laryngospasm. CPAP = continuous positive airway pressure; Fio_2 = fractional inspired oxygen tension; IM = intramuscular; PACU = postanesthesia care unit. Adapted from Hampson-Evans D, Morgan P, Farrar M: Pediatric laryngospasm. Paediatr Anaesth 2008; 18:303–7. Used with permission of John Wiley and Sons.

based on competency.[72] Similarly, simulation-based education is being increasingly used for continuing medical education. Airway management training, including management of laryngospasm, is an area that can significantly benefit from the use of simulators and simulation.[73] These tools represent alternative nonclinical training modalities and offer many advantages: individuals and teams can acquire and hone their technical and nontechnical skills without exposing patients to unnecessary risks; training and teaching can be standardized, scheduled, and repeated at regular intervals; and trainees' performances can be evaluated by an instructor who can provide constructive feedback, a critical component of learning through simulation.[74–75]

How to Use Simulation?

Airway simulators and high fidelity mannequins are important teaching tools.[73] Simple bench models, airway mannequins, and virtual reality simulators can be used to learn and practice basic and complex technical skills. In the case of laryngospasm, basic appropriate airway manipulations such as chin lift, jaw thrust, and oral airway insertion in combination with CPAP can be demonstrated and practiced with these models.

During high-fidelity simulation, technical and nontechnical skills can then be integrated and practiced. Learning objectives should be based on recommended management algorithms and used as inputs and events embedded into one (or several) case scenario that form the basis for the simulated exercise. During the exercise, the instructor can observe and measure the performance of the trainees and compare them with the standards of performance mentioned in the algorithms. The exercise is then followed by a debriefing session during which constructive feedback is provided. An example of such a simulation-training scenario of a laryngospasm, including a description of the session and the debriefing, can be found in the appendix. In addition, a video of a simulated laryngospasm scenario is available (See video, Supplemental Digital Content 1, http://links.lww.com/ALN/A807, which demonstrates the management of a simulated laryngospasm in a 10-month-old boy). The video and the script are intended to illustrate the proper application of the management algorithm, to illustrate the technical and the nontechnical skills required in clinical practice, and to be a resource for the readers who wish to develop their own training sessions.

Knowledge Gap

There are data supporting the efficacy of structured courses that integrate airway trainers and high fidelity simulation for airway management training.[76–77] Recent evidence also supports the transfer of technical and nontechnical skills acquired during simulation to the clinical setting.[78] We therefore strongly encourage the integration of simulation-based training for pediatric airway management, including for the management of laryngospasm. However, to our knowledge, no study has evaluated the effect of such a training approach on the management of laryngospasm. There is a need to fill this knowledge gap and to answer questions about what types of clinical education and what type of management algorithm result in better outcome.

Learning outcomes are difficult to measure. However, a systematic approach based on the model of translational research has recently been proposed in medical education.[79] In this model, successive rigorous studies are conducted to evaluate the acquisition of skills and knowledge at different outcome levels. First-level studies evaluate the effect of training in a controlled environment (in simulation). Second-level studies attempt to document the transfer of skills to the clinical setting and patient care. Finally, third-level studies evaluate the effect of education on patient outcomes. Although third-level studies may prove very difficult or subject to bias, first- and second-level studies are feasible but have yet to be performed for laryngospasm and pediatric airway training. We strongly encourage future studies assessing the effect of training and simulation on the management of laryngospasm in children at various levels of outcomes.

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APPENDIX. Simulation-based Training Scenario

Laryngospasm during Induction of General Anesthesia in a 10-month-old Boy

Main Problem	Medical	Nontechnical Skills
	Intractable laryngospasm during inhaled induction and maintenance of general anesthesia	Resources management during the crisis
Learning objectives	At the end of this training session the trainees should be able to: Technical/medical knowledge and skills: 1.1. Identify the child-related risk factors of laryngospasm 1.2. Recognize a laryngospasm at induction of and during inhalational anesthesia 1.3. Manage a laryngospasm at induction and during facemask inhalation anesthesia in a child according to: 1.3.1. Prevention algorithm 1.3.2. Treatment algorithm: airway manipulation and pharmacologic treatment Nontechnical skills: 1. Announce loudly the crisis 2. Call for help early 3. Exercise good leadership 4. Communicate effectively with team members (verbalization of diagnosis and management plan, closed-loop communication)	
Brief description of the scenario	A 10-month-old boy (8.5 kg body weight) is taken to the operating room for emergency surgery of an abscess of the second right hand fingertip. Past medical history was unremarkable except for an episode of upper respiratory tract infection 4 weeks ago. The boy was exposed to home smoking. Preoperative evaluation was normal. The anesthesia team will be asked to provide him with general anesthesia using inhaled sevoflurane, oxygen, and N_2O. The anesthetic plan is then to insert an intravenous cannula and to maintain the airway using a facemask or a laryngeal mask depending on anesthetist preference. During induction of anesthesia, the child will develop a partial laryngospasm that will initially recede after simple maneuvers if properly applied (jaw thrust and manual positive pressure ventilation). During IV insertion or at the time of surgical incision, a complete intractable laryngospasm will develop and will only recede with the use of suxamethonium.	
Participants	Instructors	Learners (roles may be adapted according to local practices)
	One instructor	One anesthetist in charge
	One technical assistant	One nurse anesthetist (or second anesthetist)
	—	One anesthetist available if required (help)
Information to be given to the participants	Before the scenarios starts (briefing): You are the anesthetist on call for the day. You have been asked to take care of a 10-month-old boy (8 kg body weight) who has just been brought in the operating room for emergency surgical drainage of a second right hand fingertip abscess. The night before, he has been assessed by the anesthesiologist on call. His past medical history was unremarkable except for an episode of upper respiratory tract infection 4 weeks ago and home smoking exposure. Preoperative evaluation was normal. He has been fasting for the last 6 h and he has received no premedication. Anesthetic plan: ● Induction of anesthesia with inhaled sevoflurane, oxygen, and N_2O ● Intravenous cannulation after induction ● Control of airway using a facemask or a laryngeal mask ● Maintenance of anesthesia with sevoflurane in a mixture of oxygen and N_2O 50/50% ● Postoperative surveillance: Postanesthesia care unit	

(*Continued*)

APPENDIX. (*Continued*)

Main Problem	Medical	Nontechnical Skills
	An anesthetic record has been prepared and is given to the anesthetist on charge. On demand: If the participants ask additional information pertaining to history and physical: no other significant contributing findings During the scenario: Some information may not be available to the participants (*e.g.*, if the mannequin is not able to simulate clinical signs such as skin color, size of pupils, *etc.*) provide the participants with timely appropriate information. Let them know at the beginning that you can give them such information.	
Preparation of the simulation room	Operating room setting Pediatric equipments and drugs already prepared and/or available Appropriate anesthetic records for the case	
Preparation of the mannequin	Baby mannequin (*e.g.*: Laerdal SimBaby™ Laerdal Medical Stavanger, Norway; METI BabySIM™ Sarasota, FL ; or equivalent)	
Programming the mannequin	CAVEAT: This section needs to be tailored to the type of mannequin you are using. Instruction provided here are general guidelines to help you program your own mannequin. Initial state: Baby is alert, giggling or soft cry. Vital signs: heart rate 120/min, transcutaneous arterial oxygen saturation 97%, blood pressure 90/42 mmHg, Spontaneous breathing respiratory rate 33/min, normal respiratory and cardiac sounds. Induction of anesthesia: Initial end tidal carbon dioxide is 45 mmHg. Modify the values of the end tidal concentrations of oxygen, N_2O, and inhaled sevoflurane according to what the participants will do. The child looses consciousness. Heart rate, blood pressure, arterial saturation, and respiratory rate initially evolve as expected for a standard inhalation induction of general anesthesia in a baby. Evolution: 1. Partial laryngospasm: While the anesthetist is preparing for intravenous line insertion, the child develops a partial laryngospasm while spontaneously breathing during inhalational induction. Turn on the stridor sound. Decrease the chest compliance of the mannequin. Turn on “breathing retractions” and “seesaw respiration.” Observe the response of the anesthetic team to the new situation. → If the response is adequate (chin lift or jaw thrust, manual positive pressure ventilation, ± oropharyngeal airway, deepening of anesthesia) relieve all laryngospasm signs and symptoms and let the surgery proceed. → if the response is inadequate or inexistent: the partial laryngospasm becomes complete (see below). 2. Complete laryngospasm (Can occur either after initial laryngospasm if poorly managed, during the attempt of intravenous line insertion or at the time of surgical incision). Turn on the stridor sound. Decrease the chest compliance and turn on “breathing retractions” and “seesaw respiration.” Over the next 2 min: gradually decrease arterial saturation to 78%, increase end tidal carbon dioxide to 60 mmHg, increase respiratory rate to 45/min, decrease heart rate from 120/min to 65/min, and increase blood pressure to 110/47 mmHg. Observe what maneuvers are performed by the participants to relief the laryngospasm but maintain this state whatever they do. After 30–60 s: obstruct the airway completely, turn of stridor, the child is now apneic, capnography shows a flat line. Gradually decrease arterial saturation to 50%. Only relieve the complete laryngospasm 25–30 s after the participants have injected an appropriate dose of intravenous suxamethonium (wait longer if the injection is intramuscular).	

(*Continued*)

APPENDIX. (*Continued*)

Main Problem	Medical	Nontechnical Skills
	Adapt the vital signs according to their management. Ideal management should consist of administration of atropine concomitantly to suxamethonium, followed by mask ventilation with 100% oxygen followed by tracheal intubation. If this is performed, vital signs should be normalized rapidly. Instructions for the technician: After complete laryngospasm, if hypoxemia is not corrected efficiently within 1 min with appropriate management, bradycardia should aggravate.	
End of the scenario	The scenario ends when the baby's trachea has been intubated.	
Instruction for the debriefing	During the scenario, the instructor may use a rating form and/or a checklist with the "expected actions and behaviors." This form should reflect the learning objectives of the scenario. It is intended to be a formative document that may be used to "score" the participants performance and can be reviewed later during the debriefing process. Ideally the debriefing should be structured in the following three phases: Phase 1 reactions of the participants: This short phase is used to defuse the tension and to address issues around emotions, stress, and realism of the scenario. The instructor should attempt to control the discussion and avoiding jumping right away in the analytical phase. Phase 2 analytical phase: This phase is the most important phase during which the instructor should guide the participants in a reflective practice on what happened during the scenario. The goal is to compare their performance to the learning objectives of the session (see above) and to an ideal performance. Both technical and nontechnical skills are analyzed during the debriefing. The role of the instructor is to facilitate the process by providing constructive feedback and helping the participants to identify their strengths and weakness/areas of improvement. This guided process is at the heart of simulation-based experiential learning and should not be underestimated. Adjunct such as review of the videotape of the scenario and/or review of posters of management algorithms (such as those published in this article) are frequently used during the debriefing session. Phase 3 conclusions: During this phase, important learning points of the session are listed. Areas of improvements are translated into future learning objectives adapted to each participant (further readings or further hands-on training).	