

## Artículo de revisión

## Update on the Management of Laryngospasm.

## Actualidades en el manejo del laringoespasma en pediatría

**Enrique Hernández-Cortez MD**

Comisionado editor de la Revista Anestesia en Mexico. Federación Mexicana de Colegios de Anestesiología AC. León Guanajuato, México.

Este artículo fue publicado en el Journal Anesthesia and critical care: Open access. 2018;8(6):1-6

Fecha de recepción: Noviembre 10, 2017

Fecha de publicación: Julio 15, 2018

Email: kikinhedz@gmail.com

**Summary**

Perioperative laryngospasm is an airway emergency. It is responsible for a significant number of complications ranging from hypoxia, bradycardia, bronchial aspiration, obstructive pulmonary edema and / or cardiac arrest. It is a protective closure reflex of the glottis. Laryngospasm is a relatively frequent entity in the pediatric patient, which depends on multiple factors. The aim of the treatment is to prevent its happening. When it has already happened, it is essential to make the correct diagnosis and to start its treatment immediately to prevent patient deterioration. The treatment consists of applying effective drugs to break the spasm like propofol, magnesium sulfate, muscle relaxants and reintubation. External manoeuvres include upper chest pressure and Larson's manoeuvre.

**Keywords.** Laryngospasm, upper airway, risk factors, prevention and treatment.

**Introduction**

Laryngospasm is defined as the sustained closure of the vocal cords. It is a primitive protective airway reflex, which happens to safeguard the integrity of the airway by protecting it from tracheobronchial aspiration (1). Laryngospasm is also defined as an exaggerated response of the closure reflex or glottic muscle spasm. Essentially it is a protective reflex, which acts to prevent the entry of any foreign material into the tracheobronchial tree. The presence of this reflex results in an impediment to adequate breathing, under these conditions it becomes a sudden obstruction of upper airway. A feature of laryngospasm is that the airway closure is maintained even after the initial causal stimulus disappears. In any of the

situations mentioned above, we face a partial or total loss of the airway, and therefore an anesthetic urgency.

Forty percent of the airway obstructions are secondary to laryngospasm, and this may result in a life-threatening complication, and it is a major cause of cardiac arrest in the pediatric patient.

Laryngospasm is characterized by severe hypoxia (61%), bradycardia (6%), obstructive pulmonary edema (4%), cardiac arrest (0.5), pulmonary aspiration (3%), arrhythmias and death. It is extremely important to remember that of the complications related to anesthesia, 43% are of respiratory origin. (2).

Laryngospasm is seen mainly in the child; the most frequent cause is upper airway manipulation. Its incidence is variable, and according to the consulted author. Olsson and Hallen mentioned an incidence of 1% in adults and children, doubling in infants and schoolchildren and tripling in children under three months of age. In children with airway reactivity or asthma, incidence increases to 10%, and may rise to 25% in patients scheduled for adenotonsillectomy (3). The incidence is the highest in the child between one and three months of age. In the first nine years of age, the incidence of laryngospasm is 1.74%. For school children it is 2.8%. Likewise, there is a higher incidence of respiratory adverse events in the obese asthmatic child, but not in obese children without asthma (4).

**Mechanism of laryngospasm**

Laryngeal muscles, true and false vocal cords may be involved in laryngospasm. Most laryngeal reflexes are produced by the stimulation of the afferent fibers contained in the internal branch of the superior laryngeal nerve. These reflexes regulate laryngeal muscles

contractions which protect the airway during swallowing (5).

### Pathophysiology

The causes of laryngospasm are multiple; the presence of local, mechanical, chemical or thermal stimuli, which ascend through the superior laryngeal nerve via sensory fibres of the vagus nerve. That is, the sensitivity of the supraglottic region is given by the superior laryngeal nerve, while the sensory innervation below the vocal cords is supply by the inferior or recurrent laryngeal nerve. There is a significant number of receptors in the vocal cords as well as in the glottis and adjacent structures, but the greater density of receptors is found at the entrance of the larynx, mainly in the laryngeal face of the epiglottis.

The motor response is mainly due to the presence of three laryngeal muscles; the lateral cricoarytenoids, the thyroarytenoids (abductors of the glottis) and the cricoarytenoids (vocal cord tensor). All innervated by the inferior or recurrent laryngeal nerve, which is a branch of the upper laryngeal. In other words, the intrinsic laryngeal muscles responsible for the false vocal cords are the cricoarytenoid, thyroarytenoid and cricothyroid muscles. The true vocal cords function as unidirectional valves, they have small resistance to the pressure exerted from the inside of the thorax to the airway. Under these conditions, the airway pressure is approximately 30 mm Hg, but the pressure exerted from the outside of the airway can have a resistance equivalent to 140 mm Hg and up to 190 cm H<sub>2</sub>O in the adult. This means that it will be more difficult to overcome the resistance of the vocal cords with the pressure exerted through the face mask.

The pressure in the upper airway of the child is not known, but the false vocal cords, due to their superior location, also act as gates, preventing the escape of air from the lower respiratory system, which offers an effective resistance equivalent to 30 mm Hg, which would serve to make the cough reflex more effective.

Recent studies have shown that the shape of the airway is not that of a funnel, as it has been affirmed in the last 60 years. Now, it is known that it has an elliptical shape, and for this reason the shape and design of the pediatric tracheal tubes are with "cuff" or "tamponade". The most current publications support the use of cuffed tubes for most of the pediatric population, especially if the child is connected to a ventilator, which is also valid in newborns (6).

### Risk factor's

Traditionally risk factors for laryngospasm can be classified into three categories; related to the patient, related to anesthesia, and related to surgery.

#### Factors related to anesthesia.

The most important anesthetic factor is related to the intensity of anesthesia; a light or insufficient anesthesia leads to a predisposition to trigger laryngospasm. The induction and extubation of the patient are the critical moment, which leads to a light anesthesia, and therefore to provoke pain during this stage. For example, anesthesia with desflurane can produce cough during the emergence of anesthesia, particularly when the tracheal tube is removed as it can be correlated with a faster awakening and early perception of a foreign body in the airway. The same can occur due to the presence of secretions or blood around the larynx, factors described as strong stimuli that can end in a laryngospasm.

In pediatric anesthesia, the most frequent technique for induction is inhalation with some of the current anesthetic gases. Halothane and sevoflurane are two of the inhaled gases with a lower pungency for the airway, and there is no difference in laryngospasm between these two agents. The absence of irritant properties of halothane and sevoflurane contrasts with the irritant properties of desflurane and isoflurane, particularly in relation to the induction of anesthesia. Halothane and sevoflurane can be used with great confidence to gradually induce general anesthesia, increasing the inspired concentration progressively, resulting in a low incidence of laryngeal spasm. Desflurane, on the other hand, shows a very high pungency; 50% of patients who receive induction with this gas, can develop cough by irritation of the upper airway, followed by isoflurane and enflurane. Halothane is the least irritating inhaled agent in the airway, however this gas is practically in disuse in pediatric anesthesia (7).

Tracheal intubation is another critical moment, due to the placement of a tube inside the trachea, as well as the maneuvers of laryngoscopy. In both situations a strong stimulus can be triggered, inducing laryngospasm and/or bronchospasm.

Propofol depresses the laryngeal reflex, producing a low incidence of obstructive problems. Thiopental has been shown to increase the incidence of laryngospasm, although the possible mechanism of action is not well understood (11). Ketamine has rarely been associated with this complication (8).

The movements of the head, the placement of a nasogastric tube, or the irritation of the vocal cords by blood, mucus, vomit or other liquids, as well as suction catheters, are other causes that can produce obstruction of the superior airway. Recently, it has been shown that intubation of the trachea with the awake patient and without muscle relaxants predisposes to a higher frequency of failed laryngoscopies, a higher incidence of complicated airway, and a greater failure rate for conventional intubation.

The inexperience of the anesthesiologist or multiple attempts of tracheal intubation, and the placement of a laryngeal mask (LM) in an inadequate anesthetic plane, can be causes of laryngospasm (9). The insertion of LM triggers substantially less stimulation than tracheal intubation during anesthesia with inhaled gases.

There are contradictory reports regarding midazolam and its effects on laryngospasm. Some reports have shown an increase in the incidence of laryngospasm, however other researchers have not been able to prove this assertion. In contrast, midazolam with remifentanyl has shown a low incidence of laryngeal spasm as in ophthalmologic surgery under general anesthesia (10). Ketamine is associated with a 0.4% incidence of laryngospasm, most likely due to an increase of secretions that act as irritants of the airway or areas adjacent to the larynx (12).

### **Factors related to the patient**

There are several factors specific to the patient, with age being the most important; the younger the child, the higher the incidence of laryngospasm. The occurrence of laryngospasm followed by general anesthesia is inversely related to age. This is probably due to the anatomy of the airway structures of the newborn and the infant. The fact of finding a larynx one or two vertebral segments higher, that is to say that the larynx has a cephalic position, an epiglottis in the form of "omega", rigid (long, firm and angulated) and equally high. In the same way, a shorter

time is available for tracheal intubation, due to having a smaller volume of oxygen reserves. Newborns (RN), the ex-premature, and those patients under one year of age are more susceptible to this complication. The same thing happens when there is an infection of the upper airway, which is associated with a sensitization of the bronchial efferent pathways that can last up to four or six weeks. This type of children should receive preparatory anesthetic medication with steroids to try to reduce the possibility of laryngospasm.

The anesthesiologist should pay special attention to the risk associated with hyperreactivity of the airway, especially if it is a viral infection. The damage of the respiratory epithelium may persist for several weeks, and the viruses are responsible for increasing laryngospasm between two and five times more (13). For other authors, the infection of the upper airway has been compared to placing a very tight tube in the trachea, which can increase up to eleven times the possibility of presenting a secondary respiratory obstruction. It is suggested that in those children with a history of recent airway infection in the healing phase, the airway should be handled in a less invasive way, for example placing LM. However, the studies are contradictory since others have found a higher rate of laryngospasm after an upper airway infection (14).

An important group is the passive smokers at home, including those who are hyperreactors of the airway and asthmatic patients, who have been described up to ten times the possibility of developing laryngospasm, without forgetting the adolescent smokers, who they can also be hyperreactors of the airway (15).

Obstructive sleep apnea, obesity, complicated airway, gastroesophageal reflux, ASA IV patients, are situations capable of triggering laryngospasm (16).

### **Factors related to surgery**

There is a very close association between laryngospasm and type of surgery. The surgical procedures of the upper airway stand out; tonsillectomy and adenotonsillectomy are surgeries with a probability of developing this complication up to 21-27%. Other types of surgery are bronchoscopy and upper gastrointestinal endoscopy,



appendectomy and hypospadias repair, or inferior urological surgery such as cystoscopy, they also have a high possibility of presenting obstruction of the upper airway. Lower urinary tract procedures require a deep anesthetic plane and adequate intraoperative anesthesia, since urethral manipulation can precipitate laryngospasm, due to the activation of the Breuer-Lockhart reflex.

Cervical dilatations, and skin transplantation in children. Thyroid surgery related to upper laryngeal nerve trauma, extraction of the parathyroid glands that produces hypocalcemia, patients with cerebral palsy often have problems far swallowing and gastroesophageal reflux, poor cough reflex and decreased respiratory capacity, are situations that also predispose to laryngospasm, bronchospasm and bronchoaspiration (17,18).

#### **Time of surgery where laryngospasm can appear**

Laryngospasm can occur at any time during anesthesia-surgery, but it is most frequent during intubation or extubation, due to a light or superficial level of anesthesia. However, it can also occur during the maintenance of anesthesia because of a lightly conducted anesthesia or when the patient has pain under anesthesia.

#### **Management of laryngospasm**

The management of laryngospasm can be divided into preventive or curative as seen in table 1

*Prevention.* Preventive measures include the recognition of all risk factors already discussed. In newborn, infants and young children, airway reflexes are stimulated to a much greater degree during the induction of anesthesia with inhaled agents. Therefore, it is advisable to use inhalation anesthetics with low pungency.

The rapid inhalation technique requires high concentrations of anesthetic gas accompanied by high oxygen flows, which can cause frequent irritation of the airway that is manifested with coughing and salivation, in addition to suppressing breathing. The 6/6 induction technique described for some inhaled anesthetics, particularly in the absence of anesthetic premedication, may trigger a higher incidence of laryngospasm. Induction techniques with large boluses of inhaled anesthetics and oxygen to produce rapid induction of less than 40 seconds can result in

laryngospasm. In non-medicated adults, irritation of the airway is much greater with 2 MAC of desflurane than with 2 MAC of isoflurane or sevoflurane. There are several reasons to affirm that the best induction procedure is with propofol, especially in children with a history of asthma or those who are hyperreactors (19).

Pretreatment with morphine or fentanyl seems to decrease the incidence of cough and irritation of the airway by 10% (20). Intravenous drugs, such as propofol, produce laryngeal reflex depression (21). The use of atropine is controversial; it reduces the frequency of laryngospasm due to its antisialogogous effect by reducing the amount of pharyngeal secretions. However, it is annoying for the child the intense dry mouth, the increase in heart rate, and body temperature, in addition to the greater consumption of oxygen by the heart. Therefore, the administration of atropine is only indicated in special situations such as the newborn or the premature, with greater activity of the parasympathetic tone. The use of muscle relaxants reduces the risk of laryngospasm. Lidocaine 1 to 2 mg/kg can work both preventive and corrective of laryngeal spasm. Its administration before removing the tracheal tube has been investigated since 1970; a recent study showed that the application of lidocaine at 1.5 mg / kg, two minutes before tracheal extubation reduces the incidence of laryngospasm and cough. Lidocaine acts centrally and increases the anesthetic depth. The laryngospasm can decrease 30% vs 19% with respect to the controls (22). Recently Qi X et al performed a meta-analysis to demonstrate the efficacy of intravenous and topical lidocaine. They demonstrated that both are effective in preventing laryngospasm during general anesthesia in children (23). The administration of magnesium sulfate 15 mg/kg iv before tracheal intubation has resulted in a decrease in the incidence of laryngospasm in children. The protective effect of magnesium seems to be related to muscle relaxation and increased anesthetic depth, although more studies are required. Medication with oral benzodiazepine decreases the reflex of the upper airway and therefore decreases the incidence of laryngospasm (25).

The prevention of laryngospasm also includes removing secretions or blood, until you are sure that the larynx stays completely clean.

### **No-touch technique**

Tsui and colleagues showed that using the no-touch technique, the incidence of airway obstruction decreases. Basically, it is a technique of tracheal extubation with the awake patient, which consists of aspirating any type of pharyngeal secretion with the patient in a suitable anesthetic plane, then placing the patient in any of the lateral decubitus, and next discontinuing the anesthesia until the patient wakes up completely. Finally remove the tracheal tube gently without causing fright and without stimulating the larynx and only ventilate with 100% oxygen with face mask (24). This maneuver suggests that the tracheal tube be removed while the lungs are inflated by positive pressure, which decreases the adductor response of the laryngeal muscles and thereby reduces the incidence of laryngospasm.

### **Treatment of laryngospasm**

The proper management of laryngeal spasm requires a correct diagnosis and a rapid and aggressive intervention of the anesthesiologist. Many authors recommend first manipulating the airway, then removing those factors that act as irritants and finally administering pharmacological agents (26). The first step in the management of laryngospasm is the recognition of those patients who have risk factors, taking the most appropriate precautions, individualizing each case in particular.

The diagnosis of certainty can only be made if we can visualize the glottis or the closed vocal cords, in the great majority of cases this is not possible. We can infer the diagnosis by the clinical data. The laryngospasm is divided into partial and complete; in the first case there is little entry of air to the lungs, and from the clinical point of view it is recognized by the presence of an inspiratory stridor. When laryngospasm is complete there is no entry of air to the lungs manifested by inability to breathe and absence of breath sounds.

Respiratory effort includes inspiratory stridor, which can progress to complete obstruction in which case it will progress to a full respiratory effort. The thorax shows ineffective respiratory movements with paradoxical movements between the abdomen and the thorax. There is suprasternal and/or supraclavicular retraction with exaggerated abdominal movements, in addition to oxygen desaturation with or without bradycardia. The capnography will show a flat wave and/or absence of respiratory movement. Then general signs appear, such as desaturation, bradycardia, cyanosis, and arrhythmias until they end in cardiac arrest. If the obstruction of the airway does not respond to the placement of a Guedel cannula, the possibility of regurgitation or the presence of blood in the larynx may happen.

The diagnosis of certainty can only be made, if we can visualize the closed glottis or vocal cords, but this is not possible in the vast majority of patients suffering laryngospasm. We can infer the diagnosis by the clinical data of the patient. Laryngospasm is divided into partial and complete. In the first instance there is a certain air supply to the lungs, and from the clinical point of view it is recognized by the presence of an inspiratory stridor. In the situation of complete laryngospasm there is no entry of air to the lungs, manifested by inability to breathe and absence of breathing noises. Respiratory effort includes inspiratory stridor, which can progress to complete obstruction in which case it will progress to complete respiratory effort. There are ineffective respiratory movements with paradoxical movements between the abdomen and the thorax walls. There is suprasternal and/or supraclavicular retraction with exaggerated abdominal movements, in addition to oxygen desaturation with or without bradycardia. In the event of a complete laryngospasm there is respiratory silence. The capnography will show a flat wave, absence of movements in the rebreathing bag. After these events general signs appear; desaturation, bradycardia, cyanosis, and arrhythmias to end in cardiac arrest. If obstruction of the airway does not respond to the placement of a Guedel cannula, the possibility of regurgitation or the presence of blood in the larynx may be present.

### Management in emergency phase laryngospasm

The first maneuver to try to solve laryngospasm is the firm and vigorous mobilization of the jaw backwards with extension of neck and head, that is to say subluxating the temporomandibular joint, also known as the Esmarch-Heiberg maneuver (35). It involves pushing the jaw up and forward with the head slightly extended to retract the tongue from the back of the pharynx, which favors the mobility of the tongue towards the front and allows the laryngeal passage to open. The placement of a Guedel cannula of a correct size for the child's age may be sufficient, depending on the grade of laryngospasm. If it is possible to open the mouth, a nasal cannula can be carefully placed through the nose, avoiding nose bleeding.

Simultaneously the application of CPAP with 100% oxygen via face mask, in extreme cases can be used with two hands. It is extremely important to prevent air from passing to the stomach, as this can produce regurgitation and/or vomiting, and facilitate aspiration.

Propofol breaks the laryngospasm in 77% of cases, a sub hypnotic dose of 0.25 to 0.8 mg/kg is sufficient. Side effects are relatively benign. However, the patient may be in apnea, cyanotic and bradycardic when propofol is injected, which can increase cardiovascular depression. Propofol offers more advantages than succinylcholine (27). If the laryngospasm is not resolved, the next step is the application of succinylcholine. It is considered as the gold standard to solve this complication. Low doses of succinylcholine 0.1mg/kg have been reported effective to release laryngospasm. With this dose, ventilation is preserved during emergency situations (28).

It is important to mention that administration of succinylcholine should generally be accompanied by atropine. By the time we administer this medication, it is very possible that the child, in addition to cyanotic, is bradycardic. Succinylcholine is dangerous in a myocardium that is suffering from hypoxia and bradycardia and may end up damaging the heart. Atropine can counteract the depressant effect of succinylcholine and hypoxia. Atropine also decreases pharyngeal secretions. It is recommended that succinylcholine and atropine be administered before the oxygen saturation is below 85%.

If it is not possible to have an intravenous route we must use an alternate route, such as the intramuscular, intralingual, or intraosseal. The succinylcholine intralingual dose is at least 2 mg/kg. The intramuscular route has the disadvantage that its absorption is irregular, and a higher incidence of arrhythmias has been reported. The intramuscular dose of succinylcholine requires a higher dose up to 4 mg/kg, its main disadvantage is that it requires at least one minute for the total rupture of the laryngeal spasm. If there is not succinylcholine available, a non-depolarizing muscle relaxant such as rocuronium -1.2 mg intravenously-, is sufficient to rapidly break the laryngospasm. The problem may arise when the child does not have an intravenous line, for which the intramuscular route, specifically the deltoid muscle, can also be used. Lynne and colleagues showed that the dose of rocuronium to have a complete relaxation of the vocal cords in 2.5. minutes in children under one year is 1 mg / kg, and 1.8 mg/kg in older children (29).

Other drugs reported to be useful in the treatment of laryngospasm are alfentanil and meperidine, especially when laryngospasm was triggered by painful stimulus. Doxapram at 1.5 mg/kg can suppress laryngospasm by increasing respiratory depth. Nitroglycerin 4 µg/kg, has also been reported as effective, but this drug acts mainly on the smooth musculature and not on the skeletal muscle of the vocal cords (30). The definitive treatment for refractory laryngospasm is the blockade of the superior laryngeal nerve with local anesthetics (31).

Other maneuvers: The first maneuver is to make gentle pressure in the midline of the thorax, trying to expel the trapped air in the thorax and allow the opening of the vocal cords that works with unidirectional opening. This maneuver allows to overcome a pressure of the vocal cords of 30 mm Hg, while with the application of CPAP with 100% oxygen via face mask, we can overcome a pressure of more than 150 mm Hg (adult pressure). In other words, with this maneuver the opening of the glottis is forced by sharply increasing the intrathoracic pressure, allowing breathing or stimulating the vagal reflex of Hering-Breuer. Seventy-four % of the laryngospasm episodes were corrected with this maneuver



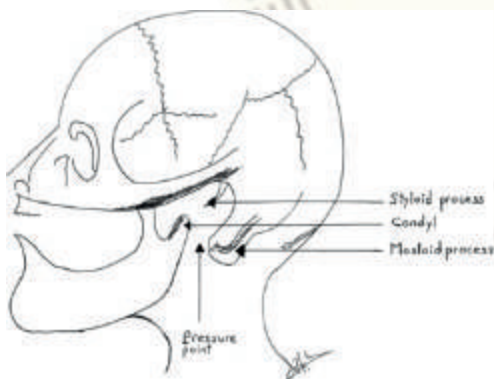
against 34% to whom conventional treatment was applied in children who received tonsillectomy (32).

The second way is a maneuver with limited scientific evidence regarding its therapeutic use. It is called the Larson maneuver. It is a technique described more than 40 years ago by Guadagni and later taken up by Larson. It is a bilateral maneuver that consists of in putting pressure on the mastoid processes at the level of the styloid processes, between the posterior branch of the mandible and the anterior mastoid process, with a small vigorous and painful force, which breaks the laryngospasm by provoking pain, relaxes the vocal cords, while moving the jaw forward and up (33). (Figure 1).

**Table 1. Classification of laryngospasm grade**

I	It is the most common grade, less risky and does not require treatment. It is a normal protection reaction
II	The arytenoepiglottic muscles are in tension and block the vision of the vocal cords. It is a more intense and lasting protection reaction. It yields with his jaw forward
III	All muscles of the larynx and pharynx are in tension, requiring the larynx to be pulled into the epiglottis, and in many cases reintubation is required.
IV	The epiglottis is trapped in the upper portion of the larynx. The use of muscle relaxants is required for resolution.

From: Hernández-Cortez E. Complications of general anesthesia in the pediatric patient. In: Hernández-Cortez E, Editor. Complications of pediatric anesthesia. Mexico: Editorial Prado; 2014. p. 63-81



The last step to rescue an emergency airway is to perform a tracheal intubation, even with the vocal cords closed, producing a trauma in them, but rescuing the airway urgently. The last manoeuvre will be cricothyrotomy or tracheostomy, as procedures of extreme urgency.

#### Follow-up after a LEP

When a patient has overcome an episode of severe laryngospasm it is recommended to leave him/her under observation for two to three hours, until it is sure that the patient does not develop one of the most frequent complications -secondary to severe laryngospasm- acute pulmonary oedema due to severe hypoxia or non-cardiogenic pulmonary oedema. It is produced by the generation of negative pressure, trying to breathe and to face a closed glottis, promoting the transudation of fluids into the alveolus, in addition to hypoxia, it increases sympathetic stimulation, causing systemic and pulmonary vasoconstriction (34).

#### Conclusions

The most important issue in laryngospasm is its prevention. The identification of the risk factors in susceptible patients can help us to avoid the obstruction of the airway. It is a priority to allow all manoeuvres that can contribute to its prevention. If the laryngeal spasm has already been established, treatment with propofol in sub hypnotic doses offers greater advantages than succinylcholine, it helps to break the spasm without myocardial depression. The administration of muscle relaxants and reintubation may be necessary advanced ways to solve the problem. The administration of muscle relaxants and hypoxia is a dangerous combination that can end in cardiac arrest, if the problem is not solved in time. Magnesium sulphate is another alternative medication, before or after the administration of other drugs, it helps to relax the bronchial musculature. Finally, the blockage of the upper laryngeal nerves can be an extreme measure in those children with a history of laryngospasm recurrence. Obstructive pulmonary oedema and bronchoaspiration are two of the most frequent complications that can happen during the period of anesthesia.

#### References

1. Gavel G, Walker RWM (2013). Laryngospasm in anaesthesia. *Critical Care & Pain* 26:1-5.
2. Olsson GL, Hallen B. Laryngospasm during anaesthesia (1984). A computer-aided incidence study in 136929 patients. *Acta Anaesthesiol Scand* 28:567-575.

Figure 1: Larsson's maneuver. *Pediatric Anesthesia* 2008; 18: 281-288.

3. El-Metainy S, Ghoneim T, Aridae E, Wahab MA (2011). Incidencia of perioperative adverse events in obese children undergoing elective general surgery. *British Journal of Anaesthesia* 106:359-363.
4. Alalami AA, Ayoub CM, Baraka AS (2008). Laryngospasm: review of different prevention and treatment modalities. *Pediatric Anesthesia* 18:281-288.
5. Subaiya L, Cosino D (2011). Pediatric cuffed endotracheal tubes: an evolution of care. *Ochsner Journal* 11:52-56.
6. Cheng EY, Mazzeo AJ, Bosnjak KJ (1996). Direct relaxant effects of intravenous anesthetics on airway smooth muscle. *Anesth Analg* 83:162-168.
7. Cavallieri BS (2014). Complicaciones anestésicas en el postoperatorio en el paciente pediátrico. En: Hernández-Cortez E, Editor. *Complicaciones de la anestesia pediátrica*. México: Editorial Prado. p. 545-560.
8. Von Ungen-Sternberg BS, Boda K, Schwab C (2007). Laryngeal mask airway is associated with an increased incidence in adverse respiratory events in children with recent upper respiratory tract infections. *Anesthesiology* 107:714-719.
9. Fick RP, Wilder RT, Pieper SF (2008). Risk factors for laryngospasm in children during general anesthesia. *Pediatr Anesth* 18:289-296.
10. Schreiner MS, O'aral, Markaris DA, et al (1996). Do children who experience laryngospasm have an increased risk of upper respiratory tract infection? *Anesthesiology* 85:475-480.
11. Mamie C, Habre W, Delhumeau C, et al (2004). Incidence and risk factors of perioperative respiratory adverse events in children undergoing elective surgery. *Pediatr Anesth* 14:218-224.
12. Cohen VG, Krauss B (2006). Recurrent episodes of intractable laryngospasm during dissociative sedation with intramuscular ketamine. *Pediatr Emerg Care* 22:247-249.
13. Von Ungern BS, Boda K, Chambers NA, Rebmann C, Johnson C (2010). Risk assessment for respiratory complications in pediatric anaesthesia: A prospective cohort study. *Lancet* 376:773-783.
14. Lakshmipathy N, Bokesch PM, Cowen DE (1996). Environmental tobacco smoke: a risk factor for pediatric laryngospasm. *Anesth Analg* 82:724-727.
15. Shott SR, Cunningham MJ. Apnea and the elongated uvula (1992). *Int J Pediatr Otorhinolaryngol* 24:183-189.
16. Bauman NM, Sandler AD, Schmidt C (1999). Reflex laryngospasm induced by stimulation of distal esophageal afferents. *Anesthesiology* 91:581-582.
17. Truong A, Truong DT (2011). Vocal cord dysfunction: An updated review. *Otolaryngology an open Access journal* S1:102.
18. Oberer C, von Ungern-Sternberg BS, Frei FJ, Erb TO (2005). Respiratory reflex responses of the larynx differ between sevoflurane and propofol in pediatric patients. *Anesthesiology* 103:1142-1148.
19. Ter Riet MF, De Souza GJA, Jacobs JS, Young D, Lewis MC (2000). Which is most pungent: Isoflurane sevoflurane o desflurane?. *Br J Anaesth* 85:305-307.
20. Betra YK, Ivanova M, Ali SS (2005). The efficacy of a sub-hypnotic dose of propofol in preventing laryngospasm following tonsillectomy and adenoidectomy in children. *Pediatr Anesth* 15:1094-1097.
21. Mihara T, Uchimoto K, Morita S (2014). The efficacy of lidocaine to prevent laryngospasm in children. *Anaesthesia* 69:1388-1396.
22. Xiaojing Qi, Zhoupeng Lai, Si Li, Xiaochen Liu, Zhongxing Wang, Wulin Tan (2016). The efficacy of lidocaine in laryngospasm prevention in pediatric surgery: a network meta-analysis. *Sci. Rep* 6:32308. doi: 10.1038/srep32308.
23. Tsui BC, Wagner A, Cave D, et al (2004). The incidence of laryngospas, with a "no touch" extubation technique after tonsillectomy and adenoidectomy. *Anesth Analg* 98:327-329.
24. Landsman IS (1997). Mechanism and treatment of laryngospasm. *Int Anesthesiol Clin* 3:67-73.
25. Burgoyne LI, Anghelescu DL (2008). Intervention steps for treating laryngospas, in pediatric patients. *Paediatr Anaesth* 18:297-302.
26. Afshan G, Chohan U, Gamar -UL-Hoda M, Kamal RS (2002). Is there a role of a small dose of propofol in the treatment of laryngeal spasm. *Paediatr Anaesth* 12:625-628.
27. Gavel G, Walker RWM (2013). Laryngospasm in anaesthesia. *Critical Care & Pain* 26:1-5.
28. Reynolds LM, Lau MBS, Brown RBS, Luks ABA, Fisher MAD (1996). Intramuscular rocuronium in infants and children. *Anesthesiology* 85:231-239.
29. Sibai AN, Yamout I (1999). Nitroglycerin relieves laryngospasm. *Acta Anesthesiol Scand* 43:1081-1083.
30. Monso A (1999). A new application for superior laryngeal nerve block treatment or prevention of laryngospasm and stridor. *Reg Anesth Pain Med* 24:186-193.
31. Gavel G, Walker RWM (2013). Laryngospasm in anaesthesia. *Critical Care & Pain* 26:1-5.
32. Johnstone RE (1999). Laryngospasm treatment an explanation. *Anesthesiology* 91:581-581.
33. Al-alami AA, Zestos MM, Baraka AS (2009). Pediatric laryngospasm: Prevention and treatment. *Current Opinion in Anesthesiology* 22:388-396.
34. Defalque RJ, Wright AJ (2003). Who invented the "JAW