Rev Bras Anestesiol
2009; 59: 4: 487-495

Laringoespasmo *
Laryngospasm

Adriano Bechara de Souza Hobaika, TSA [1], Michele Nacur Lorentz, TSA [2]

RESUMO
Hobaika ABS, Lorentz MN - Laringoespasmo.

JUSTIFICATIVA E OBJETIVOS*: A manutenção das vias aéreas é de importância fundamental para os anestesiologistas, particularmente durante a indução da anestesia e após a extubação, quando ocorre mais frequentemente o espasmo da laringe ou laringoespasmo. O anestesiologista deve conhecer a fisiologia do funcionamento faríngeo-laríngeo e os fatores de risco para a obstrução das vias aéreas, pois se trata de complicação potencialmente grave que pode ocorrer durante procedimento anestésico-cirúrgico, de etiologia multifatorial e cujas consequências podem ser nefastas. O atraso no diagnóstico ou tratamento e a evolução do quadro podem levar à hipoxemia, edema agudo do pulmão e eventualmente óbito do paciente. Nesse contexto o objetivo deste artigo foi rever as medidas que devem ser tomadas em situação de laringoespasmo, já que oxigenação e ventilação adequadas podem ficar comprometidas em tal situação.*

CONTEÚDO*: Este artigo de revisão apresenta os mecanismos de manutenção das vias aéreas, discutindo seus aspectos mais relevantes e etiologia, fisiopatologia, tratamento e prevenção do laringoespasmo.*

CONCLUSÕES*: Há muitas recomendações na literatura que visam tratar ou prevenir o desenvolvimento do laringoespasmo, mas nenhuma é completamente eficaz. Devido à sua gravidade, é necessário que sejam realizados mais estudos com enfoque nas medidas de prevenção dessa complicação.*

Unitermos: COMPLICAÇÕES, Laringoespasmo: prevenção, tratamento.

SUMMARY
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BACKGROUND AND OBJECTIVES: *Airways management is fundamental for anesthesiologists, especially during induction of anesthesia and after extubation, when laryngeal spasm is more common. The anesthesiologist should know pharyngeal-laryngeal physiology and the risk factors for airways obstruction, since this is a potentially severe complication with a multifactorial etiology that can develop during anesthesia and whose consequences can be catastrophic. A delay in the diagnosis or treatment and its evolution can lead to hypoxemia, acute pulmonary edema, and, eventually, death of the patient. In this context, the objective of this report was to review the measures that should be taken in cases of laryngospasm because adequate oxygenation and ventilation may be compromised in this situation.*

CONTENTS: *This review article presents the mechanisms of airways management, discussing the most relevant aspects and etiology, pathophysiology, treatment, and prevention of laryngospasm.*

CONCLUSIONS: *The literature has several recommendations on the treatment or prevention of laryngospasm, but none of them is completely effective. Due to its severity, further studies on measures to prevent this complication are necessary.*

Keywords: COMPLICATIONS, Laryngospasm: prevention, treatment.

** Recebido da (**Received from**) CET/SBA Santa Casa de Belo Horizonte, MG*

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*Apresentado (**Submitted**) em 28 de janeiro de 2008*
*Aceito (**Accepted**) para publicação em 01 de Abril de 2009*

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Laryngospasm

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INTRODUCTION

Respiratory events are one of the greatest causes of morbidity and mortality during anesthetic-surgical procedures, especially in pediatric anesthesia because children are more susceptible to hypoxemia due to the smaller residual functional capacity and greater tendency to develop collapse of the airways [1]. Besides, children have a high vagal tonus and can rapidly evolve to apnea and laryngospasm after vagal stimulation due to irritation of receptors in the airways by secretion, tracheal intubation, or aspiration. Hypoxia and laryngospasm represent approximately 30% of respiratory events during pediatric anesthesia, difficult intubation represents 13%, and bronchospasm 7% [2].

In general, laryngospasm is considered a physiological exaggeration of the glottic closure reflex. This complication is more frequent in: 1) children, 2) airways infection, 3) manipulation of the airways, 4) use of specific anesthetics, and 5) oral or pharyngeal surgeries. Laryngospasm is a potentially severe complication with a multifactorial etiology; however, in the majority of the times it is secondary to manipulation of the airways. Several recommendations on the prevention and treatment of laryngospasm exist, and hypoxemia, vacuum-induced acute pulmonary edema, and death are among the most severe complications. Therefore, prompt diagnosis and treatment are fundamental for a good evolution of laryngospasm.

The development of laryngospasm was probably detected almost simultaneously with the discovery of anesthesia. In 1937, Guedel included laryngospasm, which is part of the phase II signs, characterized by excitement, in the description of anesthetic planes.

Laryngospasm is the most common cause of airways obstruction after extubation and, currently, electronic simulators for the training of anesthetic complications include the management of laryngospasm. Despite developments in monitoring, ventilation failure resulting from complications of the upper airways continues to be a common and avoidable cause of cardiac arrest.

PHYSIOLOGY OF THE PROTECTION REFLEX OF THE AIRWAYS

The function of intrinsic and extrinsic laryngeal muscles allows swallowing, breathing, coughing, and speech. Reflex closure of the glottis is one of the most essential reflexes for life and allows the swallowing without aspirating food, foreign bodies, or secretions. During the pharyngeal phase of swallowing, a rapid sequence of events promotes the transference of food from the oropharynx to the esophagus. This is considered a reflex phase and depends on stimuli generated in the medulla. The larynx and hyoid bone move upwards and anteriorly in the neck with the contraction of the extrinsic muscles, which helps the inversion of the epiglottis in order to occlude the airways. There is a simultaneous increase in the activity of the adductor muscles of the vocal cords helped by the constrictor muscles of the larynx: lateral cricoarytenoid, thyroaritenoid, and cricothyroid muscles, which promote the closure of the glottis[3]. The pathophysiological mechanism responsible for laryngospasm is not completely elucidated. It is believed that there is a physiological exaggeration of the glottis closure reflex and/or an intense and prolonged absence of inhibition of such reflex that prevents the air from reaching the lungs. Most reflexes involving the laryngeal muscles are generated in the medullar centers that control ventilation via the afferent superior laryngeal nerve, possibly with contribution of the recurrent laryngeal nerve. The superior laryngeal nerve is a branch of the vagus nerve and provides sensorial innervation to the vocal cords and lower aspect of the epiglottis [1]. Around the decade of 1940, it was assumed that the pressure exerted on this region of the epiglottis by the Miller laryngoscope blade frequently caused vagal reactions, such as bradycardia and laryngospasm. This was one of the reasons that motivated the use of the Macintosh blade since the valeculla where the blade exerts pressure is innervated by the glossopharyngeal nerve and triggers vagal reactions. Most of the time, laryngospasm is precipitated by glottic or supraglottic stimulation [1]. However, stimuli in other regions such as anorectal or celiac plexus procedures can also trigger laryngospasm. Initial clinical signs include stridor or respiratory silence associated with ventilatory obstruction, which can be characterized by inspiratory efforts of accessory muscles and paradoxal thoracic movements. Desaturation, bradycardia, and central cyanosis develop later especially in the absence of early detection of initial signs and institution of treatment [4].

ETIOLOGY – PATIENT-RELATED FACTORS

Some conditions can increase the incidence of laryngospasm. Children are especially at risk; from 0 to 9 years of age the incidence of laryngospasm is approximately 17 in 1,000 cases. However, in children ages 1 month to 3 months the incidence can be up to three times greater and laryngospasm is even more severe because their airways are narrow and the parasympathetic tonus is increased. In this group, laryngospasm can be fatal because desaturation develops very rapidly and tracheal intubation is more difficult[5]. Manipulation of the airways in patients with upper airways infection or asthma increases the incidence of complication in up to six times due to hyperreactivity of the airways. Several studies have confirmed that children with upper airways infection or who are recovering from it are at a higher risk for the development of laryngospasm, bronchospasm, and desaturation [6-10]. Other conditions include: Down's syndrome, Parkinson's disease, hypocalcemia, hypomagnesemia, nasogastric tubes and in children whose parents are smokers [11-13]. Obese patients with sleep apnea are also prone to develop laryngospasm [14].

ETIOLOGY – ANESTHETIC-RELATED FACTORS

Without any question, manipulation of the airways is the main anesthetic risk factor for laryngospasm and the situations with greatest risk include: 1) laryngoscopy with tracheal intubation especially when neuromuscular blockers are not used; 2) laryngoscopy in awake patients; 3) difficult tracheal intubation; and 4) tracheal extubation. Similarly, the use of a laryngeal mask and oropharyngeal cannula also increase the risk [15]. Regurgitation and superficial anesthesia can also induce laryngospasm. Some surgical procedures are more apt to trigger laryngospasm. Oral procedures that promote an increase in secretions with blood in the airways, such as tonsillectomy and laryngeal surgery, are associated with a higher risk. Some studies have reported a 21% to 26% incidence of laryngospasm after adenoidectomy and tonsillectomy. Bronchoscopy and upper gastrointestinal endoscopy can trigger the reflex by direct stimulation [16]. Certain dyestuff instilled on the eyes to evaluate the results of nasolacrimal catheterization can trigger laryngospasm. Therefore, intubation of those patients or expelling the dye through the contralateral nostril is recommended [17]. Anal dilation in adults and correction of hypospadia in children can trigger reflex laryngospasm. Thus, anesthesia with neuroaxis block, such as sacral epidural anesthesia, and maintenance of adequate general anesthesia are recommended in children ventilated with laryngeal or face mask [16].

Among inhalational anesthetic agents, desflurane and isoflurane when used for induction have an unacceptable rate of laryngospasm, around 50% [18], which is one of the motives that sevoflurane and halothane are used for inhalational anesthetic induction. But the risk is not abolished and those two anesthetic agents can trigger laryngospasm during anesthetic induction with an incidence of approximately 3% [19]. Among intravenous anesthetic agents, ketamine is frequently mentioned as a cause of laryngospasm, explained by the increase in salivation it causes, leading to ventilatory obstruction that can evolve for laryngospasm [20]. This risk can be decreased by the administration of anticholinergic agents that reduce intraoral secretions [21]. However, contrary to what would be expected, a study with 130 children with upper airways infection who underwent elective surgeries and received 0.01 $mg.kg^{-1}$ of glycopyrrolate to decrease the incidence of laryngospasm failed to demonstrate a reduction in respiratory events with the use of the anticholinergic agent [22]. Rare causes of laryngospasm have been proposed, such as the case reported by Hong and Grecu of laryngospasm attributed to perioperative blood transfusion [23]; Subramaki reported another case that developed shortly after a subarachnoid block [24] without other causes that could justify the development of laryngospasm.

DIFFERENTIAL DIAGNOSIS

Other causes of ventilatory obstruction should be considered: edema of the glottis; presence of blood and secretions in the airways; regurgitation with aspiration; foreign bodies like clothes' buttons, teeth, tonsilar tissue, and ascaris lumbricoides[25]. Large mediastinal tumors can compress the airways and trigger a clinical condition similar to laryngospasm.

PREVENTION OF LARYNGOSPASM

Both the prevention and approach of airways complications, especially in children, demand fast decisions with attention primarily on the length of surgery and anesthesia, adequate preoperative evaluation, and drugs available for immediate use. Since laryngospasm has severe and, at times, fatal repercussions and demands prompt treatment when it is diagnosed, many investigators have concentrated their efforts on preventing this reflex [3]. Since tonsillectomy is the procedure with the highest incidence of laryngospasm, it has been the focus of such studies. Topical or intravenous administration of 2% lidocaine (1 $mg.kg^{-1}$) at the time of tracheal extubation reduces the incidence of this complication [26,27]. Transtracheal laryngeal anesthesia is also indicated when planning an "awake" intubation to prevent laryngospasm. The moment of tracheal extubation is critical in the prevention of laryngospasm and the recommendations of the literature include: 1) do not stimulate the patient during extubation (no touch technique) [28]; 2) remove the tube with the patient completely awake avoiding doing it when the patient is between the states of anesthesia and consciousness; 3) avoid removal of the tube when the patient is coughing or shows reflex apnea; 4) only empty out the tube cuff at the time of extubation; and 5) extubation in the deep plane (not

recommended in children) [3,5,15]. More than 20 years ago, Lee et al. proposed the following recommendations to prevent laryngospasm in newborns and children: extubate the child when she is awake with spontaneous opening of the eyes and mouth, making an effort to remove the tube and appearing to be crying [5]. Those recommendations still seem to be the most useful and effective on preventing laryngospasm. One study reported that inhalation of 5% CO_2 at the time of extubation reduces the incidence of laryngospasm; however, this method is not easily reproducible [29]. In theory, excess CO_2 stimulates upper respiratory centers overriding the laryngospasm stimulus and inhibiting it. In reality, it is known that both hypoxia and severe hypercapnia can cause the adductor muscles of the vocal cords to relax and lessen the laryngospasm [5]. Removal of the laryngeal mask is particularly a moment of risk and some studies have focused on this subject. It is recommended to remove the laryngeal mask with the patient either on deep anesthetic plan or completely awake [30]. However, some studies have reported that removal of the tube when the patient is deeply anesthetized is associated with a lower incidence of laryngospasm. One of them recommended a sevoflurane expired fraction around 2.2% as ideal for removal of the mask [31]. The mask can also be removed with a fully inflated cuff to remove secretions from the hypopharynx. The intravenous administration of 15 $mg.kg^{-1}$ of magnesium before extubation reduces the incidence of laryngospasm probably by relaxing the laryngeal muscles [32]. A study by Batra et al. with 120 children undergoing tonsillectomy who received subhypnotic doses of propofol (0.5 $mg.kg^{-1}$) before extubation to prevent laryngospasm concluded that the incidence of laryngospasm in children who received placebo was 20%, while in those who received propofol it was 6.6% [33]. Other authors have also suggested the use of propofol with the same objective [34,35]. Acupuncture in the Shao Shang point has also proven to be very effective on the prevention of laryngospasm [36]; this point is known as the lung meridian and it is used in the treatment of lung disorders.

TREATMENT OF LARYNGOSPASM

Treatment of perioperative laryngospasm should be initiated by the removal of the stimulus, including: stopping the surgical procedure if necessary; administer CPAP with 100% O_2; deepening the plane of anesthesia with propofol or sevoflurane; if it is caused by a painful stimulus, short-acting opioids should be administered; and consider the use of succinylcholine if the measures mentioned so far were not effective.

In the postoperative period, laryngospasm is the most common cause of post-extubation airways obstruction. Therefore, extubation should be safely performed at the appropriate moment. Rassam et al. have recommended the use of 100% oxygen, peripheral nerve stimulator, reservoir bag with a closing pressure limiting valve before extubation, and transferring the patient to the post-anesthetic recovery room breathing O_2-enriched air [37]. In the case of laryngospasm, positive pressure of approximately 10 cmH_2O should be applied immediately to the airways associated with elevation of the mandible, which is enough in most cases. Anterior elevation of the mandible can help, since it weakens the contraction of thyroidal muscles and separates somewhat the vocal cords facilitating passage of air to the lungs. At this point, successive evaluations of O_2 saturation are necessary and fast ventilation with positive pressure or deepening of the anesthetic plane can be instituted [3,5]. A technique recommending applying firm pressure at a region called laryngospasm point has been described. The maneuver consists on localizing the point behind the ear lobe, between the ramus of the mandible and the mastoid process, and applying firm pressure, if possible while dislocating the mandible anteriorly. This would alleviate the laryngospasm that evolves to stridor followed by normal ventilation [38]. In the experience of the authors, applying pressure on the laryngospasm point is a valid and effective maneuver; however, a suspected case of fracture of the styloid process with the use of this maneuver has been reported.

Succinylcholine should be reserved for cases in which laryngospasm could not be alleviated by the maneuvers described. Intravenous administration should be preferred, and a dose of 0.1 $mg.kg^{-1}$ will treat this complication [39]. If a venous access is not available, which might occur during induction or if the venous access is lost when the patient awakens, the intramuscular administration of succinylcholine can be used. The intramuscular administration of succinylcholine has become more frequent in the absence of a venous access during inhalational induction. Its use, as well as the sublingual administration to treat laryngospasm when a venous access is not available, has been reported. The intramuscular route is accessible to the anesthesiologist, especially the deltoid muscle, and the anesthesiologist does not have to abandon ventilatory assistance; the recommended dose is 4 $mg.kg^{-1}$. Even so its efficacy in those cases has been questioned because the onset of action is slower (3 to 4 minutes) it has been observed that the time necessary for relaxation of central muscles such as the diaphragm and larynx is shorter, around 45 seconds, demonstrating that oxygenation is possible before the maximal response of the adductor pollicis muscle. Three cases of pulmonary edema in children after the use of intramuscular succinylcholine have been reported, although the causes were not clearly elucidated.

The irregular absorption of the drug in the presence of a low cardiac output (pre-cardiac arrest situations) is another matter to be considered. Some studies have reported that 10% of the patients did not reach more than a 30% twitch reduction, but it seems that those patients received subcutaneous, and not intramuscular, injection [40,41]. Sublingual administration can also be used because the area has a rich vascularization allowing to a fast onset of action, around

30 seconds, and lower doses of the drug can be used; however, it is associated with cardiac arrhythmias such as bigeminism, premature ventricular beats, and sinus bradycardia in up to 50% of the cases, and cases of acute pulmonary edema have also been reported [42]. The extraoral, submental administration of 3 $mg.kg^{-1}$ is an alternative to the sublingual administration. The intraosseous (IO) administration has been accepted as a fast access to the circulation in emergency situations: relaxation occurs 30 to 45 seconds after the IO administration of succinylcholine. However, approximately 60 seconds are necessary for experienced physicians to achieve this access.

It was suggested, for many years, that succinylcholine should be prepared before extubation as part of anesthetic safety, especially when the treatment of laryngospasm was considered. However, an interesting editorial in the journal Anaesthesia questioned the advanced preparation of succinylcholine as a waste and a questionable benefit. Besides, it calls the attention for the possibility of erroneous administration of the drug.

In children younger than one year, laryngospasm is associated with bradycardia in 23% of the cases and one should consider using 0.01 to 0.02 $mg.kg^{-1}$ of intravenous atropine to prevent the evolution to cardiac arrest[15].

Intravenous doxapram, 1.5 $mg.kg^{-1}$, works as a potent stimulator of upper respiratory centers and can reverse reflex laryngospasm[43]. Nitroglycerin, 4 $\mu g.kg^{-1}.min^{-1}$, can also reverse this reflex [44].

COMPLICATION OF LARYNGOSPASM

Laryngospasm can evolve with severe hypoxia, especially in the case of a late diagnosis and treatment. Hypoxia can lead to cardiac arrest with sequelae and the negative pressure secondary to respiratory efforts associated with ventilatory obstruction leads to a negative intrapleural hydrostatic pressure, which results in vacuum-induced acute pulmonary edema [50].

CONCLUSIONS

Laryngospasm is an intense and prolonged glottic closure reflex, potentially fatal if it is not diagnosed and treated immediately. Small children are affected more oftenly and it is associated with manipulation of the airway either during insertion or removal of tracheal tubes or laryngeal masks. Many recommendations to reduce the incidence of laryngospasm can be found in the literature [51,59], but none of them is completely effective. Understanding the risk factors for the development of this reflex is fundamental to design strategies for its prevention [58,59]. Due to its severity, further studies on measures to prevent the development of laryngospasm are necessary.

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RESUMEN

Hobaika ABS, Lorentz MN – Laringoespasmo.

JUSTIFICATIVA Y OBJETIVOS*: El mantenimiento de las vías aéreas tiene una importancia fundamental para los anestesiólogos, particularmente durante la inducción de la anestesia y después de la extubación, cuando ocurre más a menudo el espasmo de la laringe o laringoespasmo. El anestesiólogo debe conocer la fisiología del funcionamiento faríngeo-laríngeo y los factores de riesgo para la obstrucción de las vías aéreas, porque se trata de una complicación potencialmente grave que puede ocurrir durante el procedimiento anestésico quirúrgico, de etiología multifactorial y cuyas consecuencias pueden ser nefastas. El atraso en el diagnóstico o tratamiento y la evolución del cuadro pueden conllevar a la hipoxemia, edema agudo del pulmón y eventualmente al deceso del paciente. En ese sentido, el objetivo de este artículo fue analizar nuevamente las medidas que deben ser tomadas en una situación de laringoespasmo, ya que la oxigenación y la ventilación adecuadas pueden quedar comprometidas en esa situación.*

CONTENIDO*: Este artículo de revisión presenta los mecanismos de mantenimiento de las vías aéreas, discutiendo sus aspectos más relevantes y la etiología, fisiopatología, tratamiento y prevención del laringoespasmo.*

CONCLUSIONES*: Existen muchas recomendaciones en la literatura que objetivan tratar o prevenir el desarrollo del laringoespasmo, pero ninguna de ellas es completamente eficaz. Debido a su gravedad, se hace necesario realizar más estudios con un enfoque en las medidas de prevención de esa complicación.*