Update in
Anaesthesia

Review

Comprehensive Review of Laryngospasm

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Abstract

Laryngospasm is a well-known entity occurring during the perioperative period, most commonly during intubation or extubation. Clinical signs are the consequence of patient effort to breath against a closed glottis.

Risk factors can be related to patient, surgery or anesthesia. They should be managed pre-operatively in order to prevent this occurrence, together with preventative drugs such as iv (intravenous) lidocaine and magnesium sulphate, iv propofol induction instead of the inhalational route in children and laryngeal aspiration before extubation.

Prompt diagnosis and management is the key to success and includes Continuous Positive Airway Pressure (CPAP) with 100% oxygen, manual maneuvers (subluxation of the temporomandibular joint and Larson's maneuver), increasing depth of anesthesia and muscle relaxation. If these measures do not succeed, forced orotracheal intubation or even cricothyroidectomy/tracheostomy are the emergency steps

INTRODUCTION

Perioperative laryngospasm is a life threatening complication during the perioperative period with an incidence of 0.78-5% depending on the surgical type, patient age, pre-existing conditions and anesthetic technique.[1]

It is defined by a sustained closure of the vocal cords as a primitive protective airway reflex to prevent tracheobronchial aspiration after an offending stimulus. The prolongation of this initial beneficial reflex after the stimulus has ceased, results in inadequate ventilation due to airway obstruction. It occurs most frequently during intubation or extubation due to a superficial level of anesthesia.[2]

The diagnosis can only be made if the closed glottis and vocal cords are visualized which is not possible in the great majority of cases. So usually it depends on the anesthesiologist's clinical judgement. Clinical signs include inspiratory stridor, paradoxical respiratory movements, suprasternal and supraclavicular retractions and rapidly decreasing oxygen saturation. As the obstruction progresses to a complete airway obstruction, the chest movements may be excessive but there is no movement of the reservoir bag and no capnogram reading. Desaturation is the most common manifestation. Other manifestations are bradycardia (6%), negative pressure pulmonary oedema (4%), cardiac arrest (0.5%), pulmonary aspiration (3%), arrhythmias and death.[3]

It is important to exclude other differential diagnoses such as: bronchospasm, supraglottic obstruction, psychogenic cause in anxious patients, vocal cord palsy, tracheomalacia, hematoma, foreign body, laryngeal edema or tracheal collapse.

PATHOPHYSIOLOGY

Causes of laryngospasm may be mechanical, chemical or thermal occurring around the glottis. They trigger the afferent fibers of the internal branch of the superior laryngeal nerve. The receptors are distributed along the glottis with the majority found on the laryngeal surface of the epiglottis.

Innervation of the supraglottic region is by the superior laryngeal nerve, while below the vocal cords it is by the recurrent laryngeal nerve. They converge in the brainstem at the tract solitary nucleus, which plays an essential role in the genesis of the upper airway reflexes.

Lateral cricoarytenoids, thyroarytenoids and cricothyroids muscles (intrinsic laryngeal muscles) are responsible for adduction of vocal cords. All of them are innervated by the recurrent laryngeal nerve except the cricothyroid, which is supplied by the external branch of the superior laryngeal nerve. Their motor neurons are located in the ambiguous nucleus and adjacent to the retroambigualis nucleus which explains why stimulation of the upper airway mucosa

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also produces cardiovascular alterations (bradycardia, changes in arterial pressure) indicating that not only skeletal muscles but also smooth muscles are involved in these reflexes.[4]

During a laryngospasm episode, either true vocal cords alone or both true and false vocal cords can be involved.[5]

RISK FACTORS

The actual known risk factors can be divided in three categories enumerated in Table 1.[6,7]

PREVENTION

In order to reduce the incidence of laryngospasm, propofol induction is the best approach as it reduces the laryngeal reflexes, particularly in children with history of asthma.

It was proven that lidocaine 1-2mg.kg^{-1} iv can be a preventive and corrective drug 2 minutes before extubation.[8] Topicalisation of the vocal cords with this agent has also been proven to be effective to prevent laryngospasm during general anesthesia in children.[9] Magnesium sulfate 15mg.kg^{-1} iv before tracheal extubation has the ability to decrease airway reflexes and cough and may play a role in laryngospasm prevention.[10]

Another important measure is removing all secretions or blood until the larynx is completely cleared before extubation.

It is debated whether tracheal extubation should be performed in awake or deeply anesthetized patients to decrease laryngospasm.[11] The literature describes the "No touch technique" which comprises the extubation of a spontaneously breathing and awakening patient, without any kind of stimulation during the emergence from general anesthesia.[12]

The artificial cough maneuver has also been described as onesingle lung inflation with 100% oxygen immediately before the removal of the endotracheal tube. It delays/prevents desaturation in the first 5 minutes after extubation and expels residual secretions in the airway decreasing the potential for vocal cord irritation.[13]

MANAGEMENT AND TREATMENT

The first step is to remove the laryngospasm stimulus, followed by a firm and vigorous mobilization of the jaw backwards with extension of neck and head, and apply CPAP with 100% oxygen via a face mask. The use of CPAP can inflate the stomach and increase the risk of gastric regurgitation. Some authors prefer the application of moderate intermittent pressure. Although airway devices can be a trigger for laryngospasm, a Guedel cannula of correct size may be helpful in providing CPAP.

Propofol in a subhypnotic dose of 0.25-0.8mg.kg^{-1} iv usually breaks the spasm. If it does not, the next step is administration of succinylcholine 0.1mg.kg^{-1} iv allowing preservation of spontaneous ventilation.[14] It has a quick onset because its ED95 is 0.3mg.kg^{-1}. Rocuronium also has an ED95 of 0.3 and will have as rapid an onset as succinylcoline and could be an option in patients who are not able to tolerate succinylcoline. Other drugs useful for treatment are alfentanil and meperidine, especially when the laryngospasm trigger was a painful stimulus. Doxapram 1.5mg.kg^{-1} can suppress laryngospasm by increasing respiratory depth. Nitroglicerin 4mcg.kg^{-1} has also been reported as effective but only acts on the smooth muscle and not on the skeletal muscle of vocal cords.[15]

The application of gentle pressure in the thoracic midline at a rate of 20-25 compressions per minute can reverse the spasm.[16] The real mechanism is unknown but some theories have been put forward. In the case of partial laryngospasm where only the true cords are involved, chest compressions will force the air through a small lumen left open at the posterior commissure of the vocal cords, ensuring ventilation and gas exchange and fast relief of partial laryngeal spasm.

Table 1. Known risk factors for larynogospasm

Patient-related	Surgery-related	Anaesthesia-related
Obesity	Nasal, oral or pharyngeal surgeries (adenoidectomy and tonsillectomy)	Laryngeal mask/Guedel airway device
Young age	GI endoscopy	Extubation
Active and passive smoking	Bronchoscopy	Suction catheter
ASA IV	Appendicetomy	Light anaesthesia plan
Gastroesophageal reflux	Anal or cervical dilatation	Blood/secretions in the airway
Obstructive sleep apnoea	Mediastinoscopy	Regurgitation
Upper airway infection	Inferior urologic surgery	Desflurane
Hypocalcaemia	Skiin transplant	Ketamine and thiopental induction
Asthma	Nociception	Nasogastric tube
Difficult airway	Surgical stimulus	Inexperience of anaesthesiologist
	Movement	Failed intubation
	Recurrent laryngeal nerve damage	Laryngoscopy
	Esophageal stimulation	
	Iatrogrnic removal of parathyroid glands	

In complete laryngospasm, in which both true and false vocal cords are opposed, this technique could help in converting to a partial spasm as air forced from below can push the area above the false vocal cords away from each other, opening the entrance of larynx. Another mechanism is the stimulation of the Hering-Breuer deflation reflex that, via the vagus nerve, may induce vocal cords relaxation.[17]

The second way is the so called Larson's Maneuver, with limited scientific evidence regarding its therapeutic use. It consists of bilateral pressure application on the mastoid processes at the level of styloid processes, between the posterior branch of the mandible and the anterior mastoid process. This results in laryngospasm cessation by provoking pain and relaxing the vocal cords.[18]

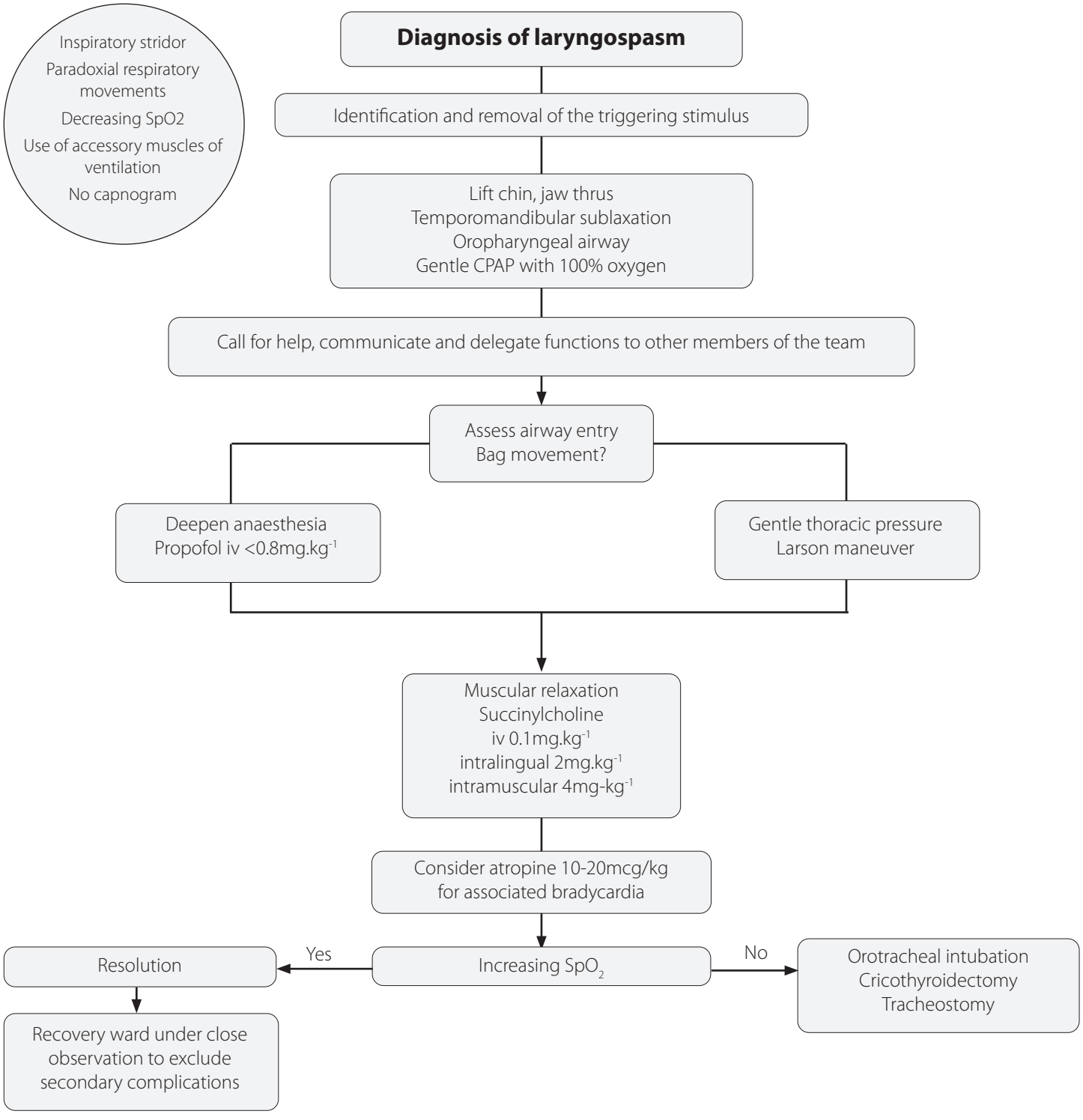

Figure 1. Simplified flowchart for laryngospasm management

One of the last measures is tracheal intubation, even with the vocal cords closed, producing trauma but rescuing the airway urgently. Cricothyroidotomy or tracheostomy are valuable procedures in extreme urgency. (Figure 1)

FOLLOW-UP

These patients should be under observation for 2-3 hours in the recovery ward to confirm a clear airway and to exclude possible complications such as pulmonary aspiration and post-obstructive pulmonary oedema. This can be a particularly harmful consequence of marked negative intrathoracic pressures due to the airway obstruction and may require intubation, ventilation and management in an ICU.[1]

CONCLUSION

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

When it occurs during the perioperative period, the priority is the prompt recognition and management according to a structured flowchart in order to minimize morbidity and mortality.

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