Research Based Guidelines for Treatment of a Laryngospasm

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Abstract
The anesthesia practitioner must respond quickly and effectively with corrective treatment to prevent patient harm associated with a laryngospasm. An objective review of current literature provides assistance in formulating a plan to manage a laryngospasm. This case report discusses an active laryngospasm and the actions taken for resolution. Familiarity with best practice recommendations will prepare the anesthesia practitioner to act during this airway emergency.

**Keywords:** laryngospasm, vocal cords, airway emergency, stridor, larynx
In the event of a laryngospasm, patency of the larynx is compromised. This glottic closure may result in negative pressure pulmonary edema and hypoxic cardiac arrest. The anesthesia practitioner must rapidly respond with corrective treatment to prevent patient harm. With varying treatment modalities being utilized for a laryngospasm, it is important to review current literature to determine the most effective management. This case report discusses an active laryngospasm and the actions taken for resolution.

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**Case Report**

A 17-year-old male patient weighing 72 kg and 175 cm tall presented for an inguinal hernia repair. The patient had no past medical or surgical history. He was not taking any medications. Airway assessment revealed a thyromental distance greater than 3 fingerbreadths and mallampati class I. The patient underwent an uneventful intravenous induction sequence using fentanyl 100 mcg, lidocaine 60 mg, propofol 160 mg, rocuronium 40 mg followed by endotracheal intubation. Grade 1 view of the larynx was noted during intubation. General anesthesia was maintained with sevoflurane 2.5% inspired concentration in oxygen 2 L/min. The surgery was uneventful and the patient incurred minimal blood loss. Post procedure, the neuromuscular relaxation was reversed using neostigmine 3 mg and glycopyrrolate 0.4 mg. The patient met extubation criteria with eye opening on command, spontaneously ventilation rate of 12 breaths per minute, and tidal volumes greater than 300 ml. End-tidal sevoflurane observed at .4% prior to
extubation. The patient’s oral pharynx was suctioned, balloon cuff was deflated and the patient was extubated. Anesthesia circuit mask applied immediately after extubation. A laryngospasm was suspected after removal of the endotracheal tube. This was determined by lack of end tidal CO₂ and absence of movement in the breathing bag when circuit mask sealed tightly to patients face. First action taken was positive pressure via anesthesia circuit mask using 100% O₂. Next intervention taken was insertion of an oral airway and jaw thrust with firm pressure at the laryngospasm notch. Despite these interventions the laryngospasm persisted. This prompted administration of lidocaine 100 mg bolus. No change in mask ventilation occurred. The patients SpO₂ remained greater than 90 percent throughout these interventions. Once the patients SpO₂ declined to 74 percent succinylcholine 20 mg intravenous bolus was given. In less than 10 seconds mask ventilation became possible and end tidal CO₂ noted. After approximately 6 minutes of breathing spontaneously with adequate tidal volumes the patient was transferred to the post anesthesia care unit (PACU). Upon arrival, laryngospasm reoccurred and repeat succinylcholine 20 mg was given. Bag mask ventilation was resumed until the patient displayed adequate spontaneously ventilation. After approximately 5 minutes of assisted ventilation the patient displayed purposeful movement and sustained eye opening to command. The patient was monitored for the following hour in the PACU to assess for airway patency and observation of postoperative complications. For the duration of time in the PACU vital signs were stable. The patient had no signs of cardiac or pulmonary complications. The patient reported no recollection of the events during emergence. The patient was discharged without further complications.
Discussion

Laryngospasms most commonly occur during induction and extubation though may occur several minutes after extubation.\(^2\) A laryngospasm occurs due to either a lack of inhibition of glottis reflexes resulting from inadequate central nervous system depression or because of an increased stimulus. Closure of the glottis occurs as afferent fibers of the internal branch of the superior laryngeal nerve become stimulated thus initiating laryngeal muscle contraction.\(^3\) Factors that increase stimulus such as laryngoscopy, suction catheters, extubation, and blood or secretions in the laryngopharynx can irritate the vocal cords and precipitate a laryngospasm.\(^4\) The most significant risk factor identified is any stimulation during a light plane of anesthesia.\(^5\)

Early recognition of a laryngospasm involves detecting a lack of air movement. This can be assessed by feeling for air passage by the anesthesia practitioner over the mouth, breath sounds, inspiratory stridor, tracheal tug, or paradoxical movements of the chest and abdomen. Late signs of a laryngospasm include decreased O\(_2\) saturation, bradycardia, and cyanosis.\(^3\)

Muscle paralysis with succinylcholine is widely accepted as the most effective treatment for laryngospasm. An intubating dose of succinylcholine administered in emergency situations to both adults and pediatrics is viewed as the gold standard. However, succinylcholine administration may be unfavorable as the first line treatment due to its several contraindications. Bradycardia in pediatrics and the potential for prolonged paralysis in patients with unknown pseudocholinesterase deficiency are among the potential risks. Intravenous doses of succinylcholine as low as 0.1 mg/kg have been
shown to be effective in termination of laryngospasm. Benefits of this low dose include maintenance of spontaneous breathing and a decreased incidence of bradycardia. No contraindication to the use of succinylcholine existed for the case study patient. Because the patient was showing signs of inadequate tissue perfusion due to inability to oxygenate, utilizing succinylcholine was appropriate and necessary.

Two studies on laryngospasm treatment concluded that less than 50% of laryngospasms required paralysis. As such, alternative maneuvers and medications were explored as first response options. Multiple research studies support preforming a jaw thrust along with firm pressure at the laryngospasm notch as a highly effective treatment to successfully terminate a laryngospasm. The laryngospasm notch is located between the mastoid process and the lobule of the ear. Inward pressure applied at the styloid process may produce severe periosteal pain that can relax the vocal cords. This maneuver in combination with positive pressure ventilation using 100% O₂ enhances the chance of successful cessation of the laryngospasm. When performed correctly, the maneuver may be the only necessary intervention to break a laryngospasm and is highly recommended to perform before pharmacologic interventions.

Propofol is among the various intravenous agents recommended for laryngospasm treatment after airway manipulation techniques prove unsuccessful. A low-dose propofol, 0.25 mg/kg IV, may be attempted. A larger dose of propofol, 1-2 mg/kg IV, may be necessary if initial low dose is unsuccessful. Although propofol is not widely administered for this purpose, current research suggests propofol be utilized before
resorting to succinylcholine as it has been successful in treating laryngospasm in 76.9% of cases.³

Conflicting research exists regarding the use of lidocaine IV for the prevention or treatment of laryngospasm. A recent study on pediatrics undergoing cleft palate repair surgery demonstrates the use of lidocaine 1.5 mg/kg administered 2 minutes prior to extubation decreases the incidence of laryngospasm by 29.9%.⁶ Additional studies have found no significant difference in prevention of laryngospasm. Lidocaine, therefore, is generally not recommended as a first line IV agent for the treatment of laryngospasm.³

Various techniques should be considered for cases of refractory laryngospasm as in the case study scenario. The anesthesia practitioner should consider a superior laryngeal nerve block, as well as transtracheal administration of lidocaine. The latter can be achieved with direct injection of lidocaine through the cricothyroid membrane.⁵

One recent study of children undergoing tonsillectomy discovered the mechanism of chest compression to be highly efficacious in relieving laryngospasms. Laryngospasms termination with gentle chest compression and 100% O₂ were successful in 73.9% of patients. Further research will aid in determining the role of chest compressions for treatment of laryngospasms.⁷

After analysis of the sequence of events and efforts to relieve the laryngospasm in the case study, propofol may have been a more appropriate first pharmacologic intervention instead of licocaine. Due to the progressive decline in oxygen saturation, the literature supports the timely administration of succinylcholine. This case study is unique in that
few laryngospasms are refractory. Unfortunately, minimal research has been conducted on treatment modalities for refractory laryngospasm. Nearly all the literature agrees that less aggressive techniques to break the laryngospasm should be explored unless the patient requires immediate treatment. In the event of an emergency or prior unsuccessful treatment, succinylcholine IV should be administered without hesitation.
References


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