Perioperative Laryngospasm - Review of literature

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Abstract

Laryngospasm is an often encountered complication perioperatively. A phenomenon that occurs with varying severity and signs. Multiple factors have been attributed to its cause. Management using different medications and maneuvers has been resorted to. This article reviews literature on this long studied anesthetic emergency. Various instances of laryngospasm with different situations and different methods of avoiding and overcoming it have been reviewed.

INTRODUCTION

Laryngospasm is one of the complication seen in the perioperative period especially during induction of anaesthesia or during extubation. It consists of prolonged glottis closure reflex mediated by the superior laryngeal nerve [1]. The common inciting factors are hyperactive airway like in case of upper respiratory tract infection. Other common triggering factors are painful stimulation, primary vagal hypertonicity, insufficient depth of anaesthesia on endotracheal intubation, light anaesthesia on tracheal extubation or combination of either preceding with or without some irritant such as blood, mucus, laryngoscope blade, suction catheter, surgical debris or other foreign body [1].It can be serious causing fatal cardiac or cerebral complications. Olsson and Hallen observed 136,929 patients, an incidence of 8.6/1000 in adults and an even higher incidence of 27.6/1000 of laryngospam in children was observed[2].

ANATOMY AND MECHANISM OF LARYNGOSPASM []

Laryngospasm involves three structures – the aryepiglottic folds, false vocal cords and the true vocal cords. The intrinsic muscles of the larynx are normally concerned in the movement of the laryngeal cartilages relative to one another. The muscles most involved in the _aryngospasm are the lateral cricoarytenoid and the thyroarytenoids (adductors of the glottis) and the cricothyroid (a tensor of the vocal cords). During a _aryngospasm either the true vocal cords alone or the true and false vocal cords both become apposed in the midline and close the glottis.

INNERVATION OF THE LARYNX

The intrinsic muscles receive a motor supply from the external branch of the superior laryngeal nerve and recurrent laryngeal nerve one on each side. The recurrent nerve supplying all intrinsic muscles other than the cricothyroid, which is supplied by the external branch of the superior laryngeal nerve.

A number of afferent pathways form a part of the laryngeal reflex arc according to the site of stimulation and the nature of the stimulus.

Stimulation of the nasal mucosa, soft palate and the pharynx. Animal studies have shown that superior laryngeal nerves mediated a minor portion of the pharyngeal inhibitory reflex, But that the main component was mediated through the pharyngeal branch of the vagus nerve.

Stimulation of the epiglottis and larynx. The internal branch of the superior laryngeal nerve innervates the larynx from its superior boundaries to the level of the true vocal cords. Whereas below the vocal cords level the recurrent laryngeal nerve carries the sensory elements. The entrance to the larynx has receptors which form a protective mechanism and have greatest degree of sensitivity. In man, the posterior aspect of the true vocal cords, which is more exposed to foreign material than the anterior aspect, was a region of greater distribution of nerve endings than the anterior aspect.

Simulation of the tracheobronchial tree – Mechanical stimulation of the larger passages elicits a forced expiratory response and showed that the afferent nerve fibers are in the vagi as seen in the study done in animals. Rex found that apnea and bronchospasm occurred even though an isolated

segment of the trachea with its nerve and blood supply was intact, and was exposed to ether. This suggested stimulation of the chemoreceptors in the lung.

Abdominal viscera and diaphragm stimulation – Pressure, tension and friction applied to the deep surfaces of the parietal peritoneum caused periods of apnea and the efferent pathway was said to be in the intercostals nerves.

In laryngeal spasm in man, either the true vocal cords alone or the true and false vocal cords become opposed in the midline and close the glottis. The reflex bronchiolar constriction was best observed by stimulation of the nasal mucous membrane [3].

LARYNGEAL CLOSURE AND ITS FUNCTION

There is double valve mechanism with in the larynx which is capable of controlling both the entrance and the exit of air [3]. When the true vocal cords were in apposition they would prevent the entrance of air, but not its exit, whereas apposition of false vocal cords was capable of preventing even a powerful current of air passing through from below.

On observation of action of larynx in quiet respiration in man, the dorsal cricoarytenoid muscles are normally in a state of partial contraction, which is tonic in nature and that the afferent impulses involved in this reflex are conducted along the vagi [3]. The adductor muscles have no role in respiration but protect the lower airway against foreign body and in modified forms of expiration as in coughing and laughing.

CLINICAL SIGNIFICANCE OF LARYNGOSPASM

Laryngospasm is commonly perceived to be a significant problem by anaesthesiologists, with an incidence of 0.78%-5% depending on surgical type, patient age, pre-existing condition and anaesthetic technique [1]. It presents a potential danger which often becomes a real danger in the maintenance of a clear airway during general anaesthesia. Laryngospasm is the occlusion of the glottis due to contraction of the intrinsic muscles of the larynx, which is essentially considered a protective reflex to prevent any foreign body reaching the tracheobronchial tree and lungs. Bronchospasm is the contraction of the bronchial musculature which causes constriction of the smaller air passages and may be associated with laryngospasm in some cases. The proposed causes of Laryngospasm includes:

Direct irritation of the vocal cords, occurring when there is sudden increase in the concentration of the irritating vapor at

the mask and damage to the tissues with traction on abdominal or pelvic viscera being of particular importance [3].

Mechanical stimulation of the epiglottis during attempts at intubation, especially in light planes of anaesthesia.

Under conditions of light thiopentone narcosis, there being an apparent increase in the sensitivity of the laryngeal reflexes than under inhalational anaesthesia. This was more due to the failure of the barbiturates to depress laryngeal reflexes than to any other stimulant action

Consensus opinion is that atropine does not prevent laryngospasm, although it may remove some of the preoperative causes by suppressing the secretion of saliva and mucous which may stimulate a spasm if they enter any part of the upper airway.

Bauman et al observed that stimulation of the distal esophageal afferent neurons evoked a sustained laryngeal adductor response in canines [4]. Alternate pathway is mediated via the vagus nerve. This reflex is different from that seen in laryngeal chemoreflex [4].

Respiratory infection dramatically increases the incidence of laryngospasm specially in children. Application of topical ligdocaine 4% to the larynx at the time of intubation has shown to decrease the incidence of laryngospasm in tonsillectomy patients [5]. Laryngospasm has been described with the elongated uvula [6], as sleep-related [7] and evoked by distal esophageal afferent [4], and even post-operatively causing pulmonary edema [8910]. The development of marked negative intrathoracic pressures due to airway obstruction is believed to be the primary pathological event in the development of pulmonary edema [8910].

LARYNGOSPASM IN CHILDREN

Cravero et al and Burgoyne et al reported 0.43/1000 and 1/1000 incidence of laryngospasm in children respectively [1112]. Although a protective reflex, it can persist to cause hypoxia, hypercapnea, cyanosis, desaturations, arrhythmias, pulmonary edema, bronchospasm, cardiac arrest or gastric aspiration [113]. It is often self limiting as hypoxia and carbon-di-oxide retention abolishes the reflex [14].

Olsson et al found the overall incidence of laryngospasm in the largest 11 year prospective study (of 136929 patients) to be 7.9/1000 anaesthetics or 8.7/1000 patients[2]. The incidence in children being higher especially in infants1-3

months of age. Some of the precipitating factors were extubation, presence of a nasogastric tube, oral endoscopy, esophagoscopy and majorly in children with respiratory tract infections [215]. Some authors have proposed laryngeal spasm to be a complication of barbiturate induced parasympathetic activity [16]. Amongst the inhalational agents isoflurane showed greater incidence of laryngospasm than halothane, enflurane and sevoflurane [17].

Laryngospasm needs to be differentiated from bronchospasm, supraglottic obstruction, a psychogenic cause in anxious adolescents and young adults (in response to exercise and emotional stress) [1819], a paradoxical vocal cord movement (post-extubation vocal cord dysfunction) [2021] and episodic laryngeal spasm subsequent to superior laryngeal nerve injury after thyroid surgery [222324]. Other causes to be excluded are foreign body, epiglottic impaction, laryngeal edema and tracheal spasm or collapse.

Incomplete airway obstruction or partial obstruction is generally associated with an audible inspiratory or expiratory sound. The stridulous noise mismatches with bag movement and the patient's respiratory effort. If the obstruction worsens, tracheal tug and paradoxical respiratory movements of the thorax and the abdomen develop. Audible sounds cease with complete obstruction resulting in no bag movement and no ventilation.

PRECIPITATING RISK FACTORS

Patient-related: Paediatric population is more susceptible [25], especially those with upper respiratory tract infection or asthma having an irritable airway [262728]. Airway hyperactivity stays for 4-6 weeks and elective surgery must be delayed for 6 weeks. Chronic smokers have increased airway sensitivity and need abstinence for 48 hours at least [29]. Studies done by Lyons et al and by Lakshmipathy et al on passive smoking and tobacco smoke respectively also proved to be risk factors for paediatric laryngospasm [3031]. History of gastroesophageal reflux [32], patients with long uvula [6] and with history of choking during their sleep [7] may have more chances of developing laryngospasm under general anesthesia. Upper airway anomalies was a significant risk factor as seen in the study done by Flick et al [33]. Activation of laryngeal thermoreceptors, chemoreceptors or both by hyperventilation can result in a spasm as studied by Ambroglio et al [34]. Nishino et al put forth that hypercapnea attenuates airway defensive reflexes in patients anaesthetized with enflurane [35].

Anaesthesia-related factors – Inadequate depth of anaesthesia during induction and emergence, while holding a mask on spontaneous breathing, the usage of a laryngeal mask airway may precipitate a laryngospasm [136]. Volatile anaesthesic like isoflurane may be irritant as compared to other agents like halothane, sevoflurane or enflurane [11737]. Mucus, secretions, blood, laryngoscope, suction catheter or any other foreign body in the laryngopharynx may trigger a laryngospasm specially in light plane of anaesthesia. Among the intravenous induction agents barbiturates like thiopentone have shown to increase laryngospasm [13153839]. Ketamine although not usually associated with laryngospasm, produces secretions which can play a trigger by irritating the vocal cords [4041]. Laryngospasm is seen more with sevoflurane than propofol induction [2542] and amongst the inhalational agents maximum with desflurane. In reducing order of association with laryngospasm inhalational agents are isoflurane, enflurane, halothane and sevoflurane [173743]. A relatively less experienced anaesthesia provider also encounters more number of laryngospasms [15].

Surgery-related factors: Upper airway surgeries are associated with a larger incidences (21-26%) of laryngospasms, that is tonsillectomy and adenoidectomy[444546474849450]. Other surgeries like appendicectomy, dilatation of anal sphincter or cervix, mediastinoscopy, hypospadias surgery and skin transplant in children are also highly associated with laryngospasm [236]. Damage to the superior laryngeal nerve after a thyroid surgery or iatrogenic removal of the parathyroid glands cause hypocalcemia that has predisposed to laryngospasm [222324]. Stimulation of the distal afferent nerves in esophageal procedures cause reflex laryngospasm as studied by Bauman et al [24].

Rare associations - Hong and Grecu reported a case of febrile non-hemolytic transfusion reaction which presented as laryngospasm after autologous blood transfusion [51]. Inapparent regurgitation and aspiration of gastric contents although not commonly noted may be an early indicator of laryngospasm [52]. Primary laryngospasm is a known complication of Parkinson's disease and acute withdrawal of medication can precipitate upper airway obstruction [5354]. Laryngospasm during a sub-arachanoid block due to increased vagal activity [55].

MANAGEMENT

Laryngospasm, if not promptly managed effectively may lead to increased morbidity and mortality. Help should be

sought early as these patients can deteriorate easily. If there is incomplete airway obstruction, remove irritant stimulus (eliminating surgical stimulation of visceral nerve endings), deepen anaesthetic plane, apply jaw thrust maneuver, insert an oral or a nasal airway and provide gentle continous positive airway pressure with 100% oxygen. Pressing firmly at the 'laryngospasm notch' helps relieving the spasm partly because the forward displacement of the mandible prevents tongue fall as advocated by Guadagni and Larson [56]. Much contrary to the recommendation that pain should be avoided, severe pain is an essential component of this maneuver. Most likely explanation being that the painful stimulus (periosteal pain caused by pressing on the styloid process) helps relaxing the vocal cords by the autonomic nervous systems [5657]. Mark recommended manual elevation of the tongue to relieve laryngospasm, by removing the obstruction caused by the tongue falling backwards into the larngopharynx [58]. Some of these patients who present with the history of snoring and airway obstruction-a sleep study (polysomnography) may help outlining the cause.

If not relieved by above maneuvers suspect complete laryngospasm and call for help, deepen anaesthesia with intravenous or non-irritant inhalational agents. Propofol in doses sub-hypnotic of 0.25 to 0.8 mg/kg show rapid action and can be used as alternative in cases where suxamethonium is contraindicated as in burns, muscular dystrophy and hyperkalemia to name a few conditions [82559]. Afshan reported 77% success in treating patients with 0.8mg/kg propofol for laryngospasm [44]. Even then if no success, use of suxamethonium in the dose 0.1-3mg/kg intravenous or 4mg/kg intramuscular or even intralingual (if no intravenous access available) followed with mask ventilation or if needed tracheal intubation relieves laryngospasm [60616263 64]. A small dose of suxamethonium intravenously causes relaxation immediately facilitating intubation of the larynx. In event the laryngospasm occurs before an intravenous access can be taken (especially in paediatrics), other alternative routes can be tried to expedite and relieve it. Although 4mg/kg suxamethonium, intramuscular takes 4 minutes for maximal twitch depression, it was observed that the laryngospasm was relieved in much lesser time as laryngeal muscles are very sensitive [64]. Intraosseous route is faster than intramuscular route and comparable to intravenous route [64].

In case of complete airway obstruction forced inflation of pharynx distends the pyriform fossa which subsequently presses the aryepiglottic folds more tightly against each other. This further causes stomach inflation rather than of the lungs. Dislocation of the temporomandibular joint anteriorly by application of pressure to the ascending rami of the mandible lengthens the thyrohyoid muscle and unfolds the soft supraglottic tissue. If this fails atropine and suxamethonium is given intravenous. If no intravenous access suxamethonium is given intramuscular 4mg/kg. Pulmonary edema has been reported following administration of intramuscular succinylcholine [65]. Atropine is avoided in patients on halothane as ventricular arrhythmias are reported. On becoming hypoxic and having bradycardia, the child may need to be intubated without muscle relaxation, than to wait for the effects of succinylcholine [66]. The vocal cords can be sprayed with lidocaine, in order to bring relaxation and facilitate intubation [5]. If these methods fail; emergency cricothyrotomy or emergency tracheostomy may be required.

Lee J et al concluded that the laryngeal tube removal in the anaesthetized state reduced cough, hypersalivation and prevented tube displacement and hypoxia [67].. They suggested upper airway obstruction in the anaesthetized state must be predicted and managed with chin or jaw lifting Various other studies done, showed that extubation or removal of LMA in anaesthetized state is associated with far fewer complications than in awake state [67686970]. Despite the fact that deep extubation might afford some protection against coughing and straining, the risk of aspiration and inadequate airway protection in this vulnerable period is a cause of concern.

Gulhas et al reported 25% incidence of laryngospasm at extubation in deep plane of anaesthesia in the control group as compared to nil in the group treated with 15mg/kg magnesium sulphate [71]. Tsui et al advocated the 'no touch technique', that is avoidance of disturbing or stimulating the patient till fully awake thus preventing premature bucking when tube is in situ [45]. The patients were placed in recovery position (lateral) after careful suctioning, volatile agents discontinued and were allowed to spontaneously wake up avoiding any kind of stimulation. No laryngospasm was reported using this 'no touch' technique. Authors suggest removing the tube while the lungs are inflated by positive pressure, thus decreasing the adductor response of the laryngeal muscles and subsequently reducing the incidence of laryngospasm. This positive pressure is followed by an 'artificial cough' (forced exhalation) after extubation which expels any secretions or blood in turn decreasing the

irritation to the vocal cords [14].

Although, Leicht et al [48] and Lee CK et al [49] and Koc et al [50] used awake tracheal extubations in their studies, an incidence of laryngospasm was reported as 22%, 23.7% and 27% respectively in their control groups post tonsillectomy. The incidence was higher probably as they used swallowing as an indication of consciousness, which rather signifies return of laryngeal reflexes.

Several other modalities have been proposed and have been used for the attenuation of laryngospasm, including anticholinergics which reduce secretions like atropine [132572], nebulised lidocaine (reducing sensitivity of upper airway reflexes) [73], cocaine [74], benzodiazepines like diazepam (decrease upper airway reflexes) [7576], doxapram in dose 1.5mg/kg over 20 secs (increased respiratory drive) [77], propofol (depresses airway reflexes) [25]. Sibai et al reported the use of intravenous nitroglycerine 4 mcg/kg may be effective in the management of post extubation partial laryngospasm (nitroglycerine stimulates formation of cGMP which is a mediator for non-adrenergic and non-cholinergic nerves which cause relaxation of the airway smooth muscles [7879]. The use of acupuncture by proposed mechanism of anti-nociception and variety of actions on the CNS, is studied by Lee et al and may prevent and treat laryngospasm occurring after tracheal extubation in children [49]. In this study, the control group showed a 23.7% incidence of laryngospasm as compared to 5.3% in the acupuncture group.

In case of recurrent postoperative laryngospasm, a differential of psychogenic/hysterical stridor must be considered and often reassurance and benzodiazepines help [1819]. Instructions to breathe in a slow pattern minimizes inspiratory obstruction. These patients have normal clinical and endoscopic findings in between episodes. On laryngoscopy, these may present with fully adducted vocal cords, hypopharyngeal spasm and paradoxical vocal cord movement [18]. Flow volume loops show paroxsysmal extra-thoracic airway obstruction. The mechanism of paradoxical vocal cord movements is unclear, but involves brain stem inspiratory centre and phase reversal occurs between inspiratory neuron and vocal cord motor neurons [2180]. It is essential to rule out inadequate neuromuscular relaxant reversal, alteration of consciousness due to benzodiazepines, volatile agents, opioids and look for underlying pulmonary or cardiac cause.

In patients of Parkinson's disease, optimization of their treatment especially during an acute infection and in the perioperative period is essential to avoid laryngospasm [5354]. Usage of 5% carbon dioxide 5 minutes prior to tracheal extubation, stimulates the respiratory drive to exhale carbon-di-oxide overrides the laryngeal reflexes [3581].

Baraka et al [46] and Gefke et al [47] reported no laryngospasm in the groups given intravenous lidocaine 2mg/kg given two and one minute prior respectively but Leicht et al said it may not always prevent laryngospasm [46474850]. Authors suggest to extubate before the signs of swallowing activity. The mechanism of action of lidocaine may be central interruption of the reflex pathway, or direct peripheral action on the sensory or motor nerve terminals.

Finally, laryngospasm was successfully treated with superior laryngeal nerve blocks as reported by Monso et al [82]. Mevorach suggested usage of the superior laryngeal nerve block which helps in interrupting the reflex arc and helps dissipating the stimuli causing the laryngospasm [1]. Importantly, the damage to the recurrent laryngeal nerve could be masked if this block were to be given bilaterally, thus leaving the adductors of the cords unopposed. In occurrence of severe repeated postoperative laryngospasm a fiberoptic pharyngeal and laryngeal examination must be conducted to rule out the pathology [1]. This should be done after the superior laryngeal block has dissipated.

To summarize, prevention is the best therapy. Awareness about the various precipitating factors which increase the risk of laryngospasm is necessary. History of prior anaesthesia, complications, respiratory problems, surgeries should be noted. Patient should be intubated in deeper planes of anaesthesia. Extubation can be tried with various medications. If this finds no avail, resort to using suxamethonium should be made if not contraindicated. After laryngospasm, signs of aspiration, pulmonary edema must be sought and treated [89105283].

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