Laryngospasm in anaesthesia

Gil Gavel FRCA
Robert WM Walker FRCA

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Key points

Direct laryngeal or distant visceral stimulation can produce laryngospasm in light planes of anaesthesia.

Recognition of at-risk patients will help to prevent laryngospasm and avoid potential significant morbidity.

Prompt recognition and early correction is essential to re-establish ventilation and oxygenation as soon as possible.

Treatment requires opening and clearing the oropharynx, applying continuous positive airway pressure with 100% oxygen, followed by deepening of anaesthesia with propofol, and/or paralysing with succinylcholine.

When i.v. access is not present, succinylcholine can be administered i.m. in a dose of 4 mg kg^{-1}.

Gil Gavel FRCA
Fellow in Paediatric Anaesthesia
Royal Manchester Children's Hospital
and
ST7 Anaesthesia
East of Scotland
UK

Robert WM Walker FRCA
Consultant Paediatric Anaesthetist
Royal Manchester Children's Hospital
Oxford Road
Manchester M13 9WL
UK
Tel: +44 161 7011263
Fax: +44 161 7014875
E-mail: robert.walker@cmft.nhs.uk
(for correspondence)

Laryngospasm is the sustained closure of the vocal cords resulting in the partial or complete loss of the patient's airway. Although described in the conscious state and associated with silent reflux, laryngospasm is a problematic reflex which occurs often under general anaesthesia. It is a primitive protective airway reflex that exists to protect against aspiration but can occur in light planes of anaesthesia.

The overall incidence has been reported by Olsson and Hallen at just under 1% in both adult and paediatric practice.[1] The incidence doubles in children and trebles in the very young (birth to 3 months of age). They also report an incidence for laryngospasm of 10% in the very young paediatric patient with reactive airways, either due to upper respiratory infection or asthma. The incidence of laryngospasm has been reported in the literature as high as 25% in patients undergoing tonsillectomy and adenoidectomy.[2]

Laryngospasm can rapidly result in hypoxaemia and bradycardia. In order to re-establish oxygenation, a clear management plan is required to avoid significant morbidity and even mortality.

Pathophysiology

Closure of the glottic opening by constriction of the intrinsic laryngeal muscles is a protective airway reflex to prevent against pulmonary aspiration.[3] It is normally triggered by a peri-glottic stimulus mediated via the vagus nerve. Sensory fibres from laryngeal mechanical, chemical, and thermal receptors ascend via the vagus nerve, via the internal branch of the superior laryngeal nerve. The highest receptor density exists posteriorly at the true vocal cords, where foreign material is most likely to spill into the airway. The motor response is via the three main intrinsic laryngeal muscles, the lateral cricoaretenoids, thyroaretenoids (the glottic adductors), and cricoaretenoids (the vocal cord tensors). They are all supplied by the vagus nerve via the recurrent laryngeal nerve (the external branch of the superior laryngeal nerve supplies only the cricothyroid muscle). Glottic closure occurs by either true vocal cord adduction alone or in conjunction with adduction of the false vocal cords. Additionally, the supraglottic soft tissues are thought to impact into the glottis as they are pulled down by an increasing translaryngeal pressure gradient during obstructed inspiratory effort. The soft tissue compression of the larynx, as the intralaryngeal pressure becomes subatmospheric, can be improved by the application of continuous positive airway pressure (CPAP).

In the conscious state, the laryngeal closure reflex has a degree of voluntary control from higher cerebral centres, therefore enabling the human being to regain control of his or her airway soon after a potential aspiration episode. This theory is loosely supported by the observation that mongrel puppies aged 50–70 days are prone to laryngospasm, at a time when cortical maturation is not complete. After maturation of the cortical centres, they grow out of this tendency. Therefore, during anaesthesia, laryngospasm may be more likely due to the dampening of central inhibitory mechanisms.

Animal work on the glottic closure reflex shows that reflex closure of the larynx is less likely during the expiratory phase of breathing and more likely during the inspiratory phase. Hypercapnia protects against reflex glottis closure by depressing adductor activity, and hypocapnia makes prolonged glottis closure more likely. Hypoxia (Pa_{O_2} <50 mm Hg) also has a depressant effect on the adductor neurones, but a Pa_{O_2} >50 mm Hg has only a minimal effect on the glottis closure reflex. These observations add weight to the statement that 'laryngospasm will break under severe hypoxia', but it must be noted that this is not a sensible approach to management.

Morbidity

A review of the Australian Incident Monitoring Study reports of laryngospasm in 2005 revealed significant morbidity associated with laryngospasm in paediatric and adult anaesthetic practice.[4] Although the most prominent finding was

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significant hypoxaemia (61%), bradycardia occurred in 6% overall but in 23% of patients <1 yr old. Post-obstructive pulmonary oedema occurred in 4% and pulmonary aspiration in 3%.

Risk factors

An increased risk of laryngospasm may be due to a combination of anaesthetic, patient, or surgery-related factors[5] (Table 1).

Common anaesthetic factors include light anaesthesia at the time of stimulus, the use of a potentially more irritant volatile anaesthetic such as isoflurane or desflurane, the presence of blood or secretions in the airway, and instrumentation of the airway at light planes of anaesthesia. The use of i.v. anaesthetic agents has been associated with a lower incidence of laryngospasm. The use of the laryngeal mask airway (LMA) and the inexperience of the anaesthetist, especially when dealing with children, have been associated with a greater incidence of laryngospasm.

Young children with hypersensitivity of the airway (from infective, inflammatory, or other irritation such as passive smoking) have a 10-fold increase in the risk of laryngospasm. If possible, an anaesthetic should be delayed for at least 4 weeks after an upper respiratory tract infection (URTI) for that reason. Pre-existing airway abnormalities and gastrooesophageal reflux are also important risk factors.

Tonsillectomy and adenoidectomy have been associated with a >20% incidence of laryngospasm. Appendicectomy, dilatation of the anus or cervix, mediastinoscopy, and hypospadias repair all carry a higher risk.

Management of laryngospasm

The management of laryngospasm consists of its prevention, recognition, treatment, and post-anaesthetic care. Other novel treatments are also discussed.

Prevention

Anaesthetic technique

Recognition of patients at higher risk of laryngospasm (Table 1) will ensure that an adequate depth of anaesthesia is attained before any potential triggering stimulus.[6] Clear communication and understanding within the anaesthetic and surgical teams of these risks is imperative—therefore, the 'tradition' of the surgeons asking whether it is safe to start their procedure should continue!

Table 1 Details of risk factor predisposing to triggering laryngospasm

Anaesthetic-related factors	
Insufficient depth of anaesthesia	
Induction/maintenance	Especially with a facemask or LMA
Emergence	Especially after tracheal extubation
Airway irritation	
Volatile anaesthetics	Halothane and sevoflurane least irritant
Mucous	
Blood	
Manipulation	Laryngoscopy, suction catheter
Airway device	
LMA	Greater risk than TT
I.V. induction agents	
Thiopental	Lack of suppression of airway reflexes (unlike propofol)
Volatile anaesthetic agents	
Desflurane>isoflurane>enflurane>halothane/sevoflurane	
Experience of anaesthetist	In children inexperienced anaesthetists are more likely to cause laryngospasm
Patient-related factors	
Age	Inverse correlation with age: young children at greatest risk
Airway hyper-reactivity	
Asthma	Approximate 10 times increased in risk, if active asthma
URTI	10-fold risk for up to 6 weeks Delay elective anaesthetic for at least 2 weeks LMA lower risk than TT in URTI
Tobacco smoke	
Chronic use	Abstain for at least 2 days to reduce risk
Passive exposure	10 times increase in risk in children
Obesity with obstructive sleep apnoea	
Gastrooesophageal reflux	May be a primary aspiration or related to chronic inflammation of the upper airway
Airway anomaly	Subglottic stenosis or cysts, laryngeal pappilomatosis, cleft palate, vocal cord paralysis, laryngomalacia, tracheal stenosis, Pierre Robin syndrome
Others	Elongated uvula, history of choking while sleeping, febrile non-haemolytic transfusion reaction, Parkinson's disease (especially on withdrawal of treatment), psychogenic
Surgical-related factors	
Shared airway surgery	Tonsillectomy and adenoidectomy carry greatest risk
Thyroid surgery	Due to superior laryngeal nerve injury, or hypocalcaemia secondary to accidental parathyroid gland excision
Oesophageal surgery	Thought to be due to stimulation distal afferent oesophageal nerves
Others	Appendicectomy, cervical dilatation, hypospadias repair, skin grafting

It is mainly during induction and emergence that a patient is at risk of laryngospasm due to the changing levels of anaesthesia. Inhalation induction should always be carried out using a non-irritant agent such as sevoflurane. I.V. induction with propofol is smoother and less problematic.

During the emergence phase, patients should be extubated either in a deep plane of anaesthesia or fully awake but not in-between. This holds for tracheal tubes and supraglottic airway devices (SADs). Both techniques have their advantages and disadvantages, but neither technique is superior in terms of the development of laryngospasm. When planning a 'deep' extubation for a tracheal tube, the airway should first be suctioned and the patient placed in the lateral position. After extubation, the patient is best left undisturbed if the airway is clear. An awake extubation, on the other hand, should occur once facial grimacing, adequate tidal volumes, a regular respiratory pattern, coughing, and preferably eye opening have returned. The 'No Touch' technique is essentially an 'awake' extubation. It consists of pharyngeal suctioning and lateral positioning while anaesthetized, followed by avoidance of any stimulation until eye opening when extubation takes place. The 'No Touch' technique has been specifically studied as a means of reducing laryngospasm and has been shown to be associated with a low incidence of post-extubation laryngospasm.

Tracheal extubation during forced positive pressure inflation decreases laryngeal adductor excitability, decreasing the likelihood of laryngospasm, and also clears the airway of secretions or blood.

Pharmacological prevention

The following studies have been carried out in intubated patients. There is little information available for the prevention of laryngospasm with the use of SADs.

Magnesium (15 mg kg^{-1}) administered i.v. intraoperatively has been shown to reduce the frequency of laryngospasm after awake extubation in a small but well-designed study of paediatric adenotonsillectomy patients. The postulated protective effect of magnesium is due to both an increased depth of anaesthetic and muscle relaxation. This agent may have a role to play in the future prevention of laryngospasm, but more studies are needed.

Lidocaine has been studied both topically and i.v. to prevent laryngospasm. There are only a few studies looking at i.v. lidocaine in a dose of 1.5–2 mg kg^{-1} given before extubation to prevent laryngospasm. The results from the available studies are conflicting with one study showing a positive effect and the other similar rates of laryngospasm between the groups. Topical lidocaine (4 mg kg^{-1}) applied to the larynx before intubation is used often when manipulating the larynx and has been studied as an aid to prevent laryngospasm. The only available study shows a slight decrease in the incidence of laryngospasm.

Atropine is thought to reduce the risk of laryngospasm by its antisialogue action reducing the amount of pharyngeal secretions. Its use to prevent laryngospasm is unproven.

Recognition

Any episode of airway obstruction in an anaesthetized patient may be due to laryngospasm. This possibility becomes greater if basic airway manoeuvres and adjuncts have failed to relieve the airway obstruction. Apnoea, breath holding, bronchospasm, or pulmonary aspiration may all present similarly to laryngospasm.

Laryngospasm is obvious in 75% of cases, but the study by Visvanathan showed that 25% present atypically. These patients may present as simple airway obstruction, regurgitation and vomiting, or desaturation.

Common signs of laryngospasm include inspiratory stridor which may progress to complete obstruction, increased respiratory effort, tracheal tug, paradoxical respiratory effort, oxygen desaturation with or without bradycardia, or airway obstruction which does not respond to a Guedel airway. When these occur, either alone or in combination, laryngospasm is possible. Any trigger should then be removed if possible. The possibility of regurgitation or blood in the airway should also be considered and the plane of anaesthesia altered if necessary.

Treatment

It is important to have rehearsed a clear plan of action.[7] After exclusion of other obvious causes of airway obstruction, this plan should be put into action. An oxygen saturation which continues to decrease below 80% with or without an accompanying bradycardia should prompt the anaesthetist to act quickly to regain oxygenation of the patient. Good communication to other immediate team members is vital to ensure success.

Initial treatment of laryngospasm classically consists of:

(i) removing any triggering stimulation;
(ii) ensuring a clear larynx, that is, checking for blood or stomach contents;
(iii) relieving any possible supra-glottic component to the airway obstruction;
(iv) application of CPAP with 100% oxygen.

Help should be requested if required. This is mandatory for the inexperienced anaesthetist. Consideration should be given to performing cautious direct laryngoscopy to gently suction the larynx clear of secretions, blood, or gastric contents; however, this must be performed with care as the situation may worsen.

As part of the initial treatment, a vigorous jaw thrust will lift the tongue off the pharyngeal wall and potentially help lift the supraglottic tissues from the false vocal cords. Placement of an appropriately sized Guedel oropharyngeal airway will help to ensure patency of the supraglottic airway. If the mouth does not open, a nasopharyngeal airway can be placed carefully to avoid the risk of bleeding. Meanwhile, CPAP with 100% oxygen via a tight-fitting facemask (using two hands if necessary) should be maintained. At this point, it is important to avoid vigorous attempts at ventilation as this will only inflate the stomach and cause diaphragmatic splinting.

The action plan may vary slightly if the patient is in the induction phase or emergence phase. During the emergence phase, one may be tempted to 'sit it out' after ensuring the upper airway is clear. However, if laryngospasm is not rapidly settling, the only options are to rapidly deepen anaesthesia or to paralyse. This is also the situation during induction. Should laryngospasm not break, after efforts to deepen anaesthesia, paralysis may be necessary. The inhalation route is of course of limited use in this situation as a means of deepening the plane of anaesthesia and an i.v. bolus of a rapid onset anaesthetic agent is preferred. I.V. propofol (0.5 mg kg^{-1} increments) is the drug of choice. Cardiovascular adverse effects at this dose are relatively minor, although the patient may become apnoeic. If apnoeic, generally laryngospasm will have settled and ventilation usually easily supported. Propofol has been reported to relieve laryngospasm in just more than 75% of cases. Propofol can be used alone or followed by the use of succinylcholine. Its use can avoid the need for paralysis and in some instances the potential side-effects of succinylcholine. Therefore, in paediatric and adult anaesthetic practice, when planning either a gaseous induction or a spontaneously breathing technique, it is always useful to have some preprepared syringes of propofol as 'emergency' drugs, in addition to atropine and succinylcholine.

Succinylcholine is the drug of choice if propofol fails to relieve laryngospasm, although many may prefer to use succinylcholine as first line. It can be given i.v. with rapid effect or by an alternative route if i.v. access is not present.[8] If i.v. access is secured, the use of i.v. succinylcholine in a dose of anything from 0.1 to 2 mg kg^{-1} will break laryngospasm. The lower dose of 0.1 mg kg^{-1} has been reported to break laryngospasm but preserve spontaneous ventilation during adult bronchoscopy cases.

Should i.v. access not be available then succinylcholine can be given by the i.m. route, the intralingual route (i.l.), or the intraosseous route (i.o.). The i.m. dose is 4 mg kg^{-1} (suggested maximum dose 200 mg). Although the time taken for full paralysis is 3–4 min, the time taken to break laryngospasm will be 45 s–1 min. Studies have shown that relaxation of the laryngeal muscles occurs before skeletal muscles and thus i.m. succinylcholine is a reasonable option. The i.m. route is easily accessible (either deltoid or the lateral quadriceps) and use of i.m. succinylcholine given when the oxygen saturations are continuing to decrease will gain control within 1 min. However, if the administration is given late when perfusion through the skeletal muscles is poor, the uptake will be variable.

I.L. succinylcholine is essentially an i.m. injection into the body of the tongue. An i.l. injection of succinylcholine of 2 mg kg^{-1} has been studied in children. Full relaxation occurs in 75 s, and therefore, relaxation of laryngospasm will be quicker than an i.m. injection in the skeletal muscles. The use of i.l. succinylcholine has been associated with arrhythmias and this is unexplained. Practically, i.l. succinylcholine requires removal of tight-fitting CPAP to administer it into the centre of the tongue with a small gauge needle. To avoid the need for this, submental intralingual succinylcholine has also been studied. Using this approach, a dose of 3 mg kg^{-1} is injected into the tongue underneath the jaw in the centre of the tongue base. Relaxation using this approach is more variable and onset of action and duration of action are similar to the i.m. route.

The use of i.o. infusions has gained popularity for rapid access to the circulation in all age groups. I.O. succinylcholine in a dose of 1 mg kg^{-1} has been verified in animal studies as similar in onset to i.v. succinylcholine (35 s). The only delay is the time taken to insert the i.o. cannula. This route is probably the most reliable route into the systemic circulation in a peri-arrest situation as may occur in severe laryngospasm. In this situation, an i.m. injection of succinylcholine loses nothing and may have good effect, but if the situation is deteriorating, then the insertion of an i.o. needle will allow for the administration of resuscitation drugs if necessary.

When laryngospasm is successfully treated, ventilation should be supported initially with 100% oxygen. Laryngeal suction should be considered again. Further support of the airway may be required with tracheal intubation (especially when airway soiling or pulmonary oedema has occurred). This will allow for toilet and suctioning of the airway and re-recruitment of the pulmonary alveoli to prevent postoperative secretion retention and infection. The need for prolonged or postoperative ventilation must be judged on an individual basis.

Anecdotal treatment options with limited evidence base

Larson's manoeuvre is bilateral firm digital pressure on the styloid process behind the posterior ramus of the mandible. It is essentially a vigorous jaw thrust with pressure between the posterior ramus of the mandible and anterior to the mastoid process. This manoeuvre is reported to break laryngospasm anecdotally but has not been subjected to study. This is a variation of a vigorous jaw thrust and its use is reported only in correspondence in the literature.

Superior laryngeal nerve blocks have been described to successfully treat recurrent laryngospasm in a small case series.

Doxopram or nitroglycerin infusions have each been reported as case reports to treat laryngospasm.

Gentle chest compressions have been reported as a novel treatment of laryngospasm.[9] A non-randomized study with about 600 children undergoing adenotonsillectomy in each arm showed an almost doubling in success in treating laryngospasm using this technique over the traditional one of CPAP. Although the methodology could be criticized, the results are interesting. There was approximately an 8% incidence of laryngospasm in both groups. Seventy-four per cent were successfully treated by chest compression against 38% by the standard method.

The gentle chest compressions were delivered, while 100% O_2 via a tight-fitting facemask was provided, 'using the extended palm of the free hand placed on the middle of the chest, with the fingers directed caudally and performing a compression force half or less than half that used for cardiopulmonary resuscitation at a rate of approximately 20–25 compressions per min'. Three possible explanations suggested for the success at treating laryngospasm are forcing open the glottis by increasing intra-thoracic pressure, stimulation of

shallow breaths, or stimulation of the vagal Hering–Breuer deflation reflex, which may inhibit reflex glottic closure. There was also minimal gastric distension in the chest compression group. Caution should be exercised in the use of this technique and treatment of deteriorating hypoxaemia not delayed.

Conclusion

Laryngospasm can be prevented by paying attention to the depth of anaesthesia and recognition of risk factors.

Treatment of laryngospasm should proceed traditionally by clearing supraglottic airway obstruction and soiling, CPAP with 100% O_2, deepening of anaesthesia i.v., and paralysis using succinylcholine by the i.v., i.m., or i.o. route as appropriate. Effective team communication and leadership is essential during this emergency situation.

Declaration of interest

None declared.

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Please see multiple choice questions 1–4.