

Role of Laryngeal Spasm in Negative Pressure Pulmonary Oedema.

Zara Wani¹, Dev Kumar Harkawat², Meenaxi Sharma³

¹Post Graduate, Dept. of Anaesthesia and Critical Care, NIMS Medical College & Hospital, Jaipur.

²Assistant Professor, Dept. of Anaesthesia and Critical Care, NIMS Medical College & Hospital, Jaipur.

³Professor and Head, Dept. of Anaesthesia and Critical Care, NIMS Medical College & Hospital, Jaipur.

Received: September 2016

Accepted: September 2016

Copyright: © the author(s), publisher. It is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Negative-pressure pulmonary edema (NPPE) is a clinical entity of anaesthesiologic relevance, peri-operatively caused by obstruction of the conductive airways (upper airway obstruction, UAO) due to laryngospasm in approx. 50% of the cases, its early recognition and treatment by the anaesthesiologist is mandatory. Laryngospasm, a brief closure of the vocal cords is not an uncommon peri-operative occurrence. If recognized and managed appropriately, the effects are transient and reversible. However, in rare cases where recognition and management are delayed, the consequences are associated with a high morbidity including desaturation, awareness, negative pressure pulmonary edema, and mortality.

Keywords: Negative pressure pulmonary edema, laryngospasm, anaesthesiologist, desaturation, mortality.

INTRODUCTION

Larynx is the inlet for the respiratory system. Any obstruction at the Larynx blocks the air, which enters and equalizes the negative pressure produced by the lungs during inspiration. The normal intra pulmonary pressure is -1 cm of H₂O at the start of inspiration. This pressure reaches +1mm at the start of expiration. However, intra-pleural pressure is between - 5 cm and -7.5 cm of H₂O during this process and always maintains a negativity. When an individual tries to inspire with an obstructed upper airway, this pressure drops to -50 cm of H₂O and in extreme cases have been known to exceed -100 cm of H₂O.

Name & Address of Corresponding Author

Dr. Zara Wani
Post Graduate,
Dept. of Anaesthesia and Critical Care,
NIMS Medical College & Hospital, Jaipur,

Pulmonary circulation is a low pressure system and the capillary pressure is about 10mm of mercury. The normal oncotic pressure is 25mm of mercury. Any decrease in oncotic pressure (hypo-proteinemia) or increase in hydrostatic pressure (due to increased pressure in pulmonary vessels) will produce pulmonary edema. In negative pressure, pulmonary edema (NPPE) not only does the pulmonary vascular pressure increases but some amount of damage occurs also to the alveolar membrane, which causes passage of fluid into alveoli. NPPE may develop in several clinical situations follows a sudden, severe

episode of upper airway obstruction, such as post-anaesthetic laryngospasm, post-extubation laryngospasm, epiglottitis, croup, choking and strangulation. Laryngospasm is the sustained closure of the vocal cords resulting in the partial or complete loss of the patient's airway. Laryngospasm mediated upper airway obstruction leads to forced inspiration generating excessive negative intra-thoracic pressure and causes negative pressure pulmonary edema.

The true incidence might be underestimated because many cases may be misdiagnosed or undiagnosed. The overall incidence of NPPE is less than 0.1% in all surgeries performed under general anesthesia while the incidence of development of pulmonary edema in acute upper airway obstruction (type I NPPE) ranges from 9.6-12% and that in chronic airway obstruction (type II NPPE) is 44%^[1]. In post-extubation subglottic edema, studies have reported an incidence of NPPE of upto 9.6 percent. It is estimated that 11% of all patients requiring active intervention for acute upper airway obstruction develop NPPV. Among pediatric patients, acute epiglottitis and croup leading to upper airway obstruction comprise greater than 75% of NPPE.^[2]

In the adults, risk factors include obesity with obstructive sleep apnoea, anatomically difficult intubations, the presence of airway lesions, and patients undergoing nasal, oral or pharyngeal surgery. Young male athletes are at risk because of their ability to generate significant negative intrapleural pressures. Paediatric patients also are at risk because of their extremely compliant chest walls that can generate large negative intrapleural pressures.^[3]

The symptoms of NPPE usually develop immediately after extubation, although sometimes the onset may be delayed up to several hours in the post-operative period. A possible explanation for this delayed manifestation is a positive pressure, created by forceful expiration against a closed glottis, opposing fluid transudation. As airway obstruction relieves, increased venous return causes blood shift from peripheral to central circulation and hydrostatic transudation.^[4]

Therefore, close postoperative observation must be continued for an extended time in patients experiencing respiratory difficulty. Common signs of laryngospasm include inspiratory stridor, which may progress to complete obstruction, increased respiratory effort, tracheal tug, paradoxical respiratory effort, pink frothy respiratory secretions, oxygen desaturation with or without bradycardia, crackles on chest auscultation 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.

In view of clinical feature, 77% of cases laryngospasm was clinically obvious, but 14% presented as lower airway obstruction, 5% as regurgitation or vomiting, and 4% as desaturation. Most were precipitated by direct airway stimulation (airway manipulation, regurgitation, vomiting, or secretions in the pharynx, but surgical stimulus, irritant volatile agents, and failure to deliver the anaesthetic were also precipitating factors).

DIAGNOSIS

NPPE is basically a clinical entity which can be established by diagnosis of exclusion. The main differential diagnosis includes aspiration pneumonitis, cardiogenic lung edema, and anaphylaxis. The findings of normal Brain Natriuretic Peptide (BNP) and Troponin levels, preserved systolic function on cardiac Ultrasonography, could be differentiated from cardiogenic lung edema. Chest radiograph typically demonstrates bilateral pulmonary opacities consistent with lung edema. Computed tomography of chest shows a preferential central and non-dependent distribution of ground glass attenuation.^[6]

TREATMENT

After the diagnosis of NPPE has been made, treatment is directed toward reversing hypoxia and decreasing the fluid volume in the lungs.^[7] Maintaining the airway and providing supplemental oxygen is usually all that is required for a positive outcome. If oxygenation does not improve in the

intubated patient, positive end-expiratory pressure (PEEP) should be administered to promote alveolar expansion. If oxygenation does not improve in the non-intubated patient, then immediate intubation with positive pressure ventilation and positive end-expiratory pressure is necessary.^[8] NPPE is a rapidly reversible condition with relatively simple management. Besides subsequent removal of the obstructive event, providing a patent airway and adequate oxygen saturation were the first step in the initial management. 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.

Although symptoms usually resolve with restoration and maintenance of a patent airway and supplemental O₂, they may sometimes progress to adult respiratory distress syndrome and result in death.^[9] The use of diuretic therapy (eg, furosemide, 1 mg kg⁻¹) to remove excess intrapulmonary fluid is controversial. It is possible for the patient to be hypovolemic, and consequently diuretic therapy would only worsen the clinical condition. Specifically the uses of tracheal anaesthesia, intravenous lidocaine prior to extubation, the use of lidocaine in the endotracheal cuff are current strategies employed to prevent laryngospasm after extubation.^[8] Patients in whom post obstructive pulmonary edema develops generally have an uncomplicated hospital course followed by the rapid resolution of the pulmonary edema and short hospital stays.^[10-12] Non-invasive ventilator support has gained great popularity in recent years and has replaced the traditional invasive ways. The role of non-invasive ventilation (NIV) in treatment of pulmonary edema can be significant as it dramatically reduces the work of breathing and thereby preventing muscle fatigue.^[13] Apart from this benefit, NIV may be able to reduce the ventricular after load, helps in better recruitment of alveoli, minimal disturbances of the hemodynamic parameters, and lesser incidence of ventilator associated pneumonias. The resolution of pulmonary edema usually occurs within 3-12 hours of institution of appropriate therapy.^[14] However, in a few cases the complete resolution may take up to 48 hours. The patients have good prognosis if promptly diagnosed and appropriate treatment instituted. However, a significant complication rate does exist and is generally attributed to a delay in diagnosis.

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. The inhalation route is 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) 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 pre-prepared 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. If I.V. access is secured, the use of I.V. succinylcholine in a dose of anything from 0.1 to 2 mg kg will break laryngospasm. The lower dose of 0.1 mg kg has been reported to break laryngospasm but preserve spontaneous ventilation during adult bronchoscopy cases. The I.M. dose is 4 mg kg (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 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 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 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.

CONCLUSION

In conclusion, NPPE is a relatively common and often under-diagnosed complication following an episode of upper airway obstruction. Post-anaesthetic laryngospasm is most common in adults, and acute epiglottitis by far the most common in children. This syndrome should be listed in the important differential diagnosis of peri-operative respiratory failure, and also an important clinical

entity in post-extubation respiratory distress. NPPE carries a good prognosis if promptly diagnosed and appropriately treated with or without mechanical ventilation.

NPPE, if anticipated and recognized early, will be a self-limited condition with excellent prognosis and simple management very quickly otherwise, it may have serious complications. However, both surgeons and clinicians must be provided with an understanding that any patient who is otherwise well has the potential for NPPE, which is of anaesthesiologic relevance, even late in the postoperative period. As a result, good prevention and rapid diagnosis and treatment is necessary to achieve early resolution from NPPE and avoid significant patient morbidity and mortality.

How to cite this article: Wani Z, Harkawat DK, Sharma M. Role of Laryngeal Spasm in Negative Pressure Pulmonary Oedema. *Ann. Int. Med. Den. Res.* 2016; 2(6):AN01:AN04.

Source of Support: Nil, **Conflict of Interest:** None declared

REFERENCES

1. Bajwa SJS, Kulshrestha A. Diagnosis, prevention and management of postoperative pulmonary edema. *Ann Med Health Sci Res.* 2012; 2: 180-5.
2. Robert AF, Kristen AB. Emergent management of pediatric epiglottitis. *Medscape Infectious diseases CME Update:* Nov23, 2014.
3. Sulek C. Negative-pressure pulmonary edema. In: Gravenstein N, Kirby RR. *Complications in Anesthesiology.* 2nd ed. Philadelphia, Pa: JB Lippincott; 1996: 191-7.
4. Vandes R, Kothari DS, Tripathi RS, et al. Negative pressure pulmonary edema with laryngeal mask airway use: recognition, pathophysiology and treatment modalities. *Int J Crit Ill Inj Sci.* 2012; 2:98-103.
5. Visvanathan T, Kluger MT, Webb RK, et al. Crisis management during anaesthesia: laryngospasm. *Qual Saf health Care.* 2005; 14: e3.
6. Bajwa SJS, Kulshrestha A. Diagnosis, prevention and management of postoperative pulmonary edema. *Ann Med Health Sci Res.* 2012; 2: 180-5.
7. Fistikci H, Cakar N, Kirisoglu C, Kara O, Polat S. Non-invasive Positive Pressure Ventilation in Treatment of Negative Pressure Pulmonary Edema and Alveolar Hemorrhage. *Turk Anest Rean Der.* 2008; 36: 178-81.
8. Kesimci E, Aslan B, Gümüs T, Kanbak O. Management of unpredicted postoperative negative pressure pulmonary edema: a report of two cases. *The Internet Journal of Anesthesiology* ISSN: 1092-406X 07 Sep 09.
9. Ackland GL, Mythen MG. Negative pressure pulmonary edema as an unsuspected imitator of acute lung injury/ARDS. *Chest* 2004; 125:1530-5.
10. Tarrac SE. Negative pressure pulmonary edema-a postanesthesia emergency. *J Perianesth Nurs.* 2003; 18: 317-23.
11. Westreich R, Sampson I, Shaari CM, Lawson W. Negative-pressure pulmonary edema after routine septorhinoplasty: discussion of pathophysiology, treatment, and prevention. *Arch Facial Plast Surg.* 2006; 8: 8-15.
12. Devys JM, Balleau C, Jayr C, et al. Biting the laryngeal mask: an unusual cause of negative pressure pulmonary edema. *Can J Anaesth.* 2000; 47: 176-8.
13. Hoo GW, Byrd RP, Talavera F, et al. Noninvasive ventilation. *Medscape CME Update:* Apr 4, 2014.
14. Chan SP, Liu HP, Chen CY, et al. An analysis of negative pulmonary edema cases after acute upper airway obstruction. *J Emerg Crit Care Med.* 2012; 23: 131-8.