

Running head: NEGATIVE PRESSURE PULMONARY EDEMA

Negative Pressure Pulmonary Edema

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### Abstract

The purpose of this project was to investigate Negative Pressure Pulmonary Edema (NPPE) with special consideration related to its prevention, recognition and treatment. Furthermore, the focus of the research was intent on measuring the rudimentary knowledge of the Adventist University student nurse anesthesia (SRNA) population regarding NPPE and then determining whether the students became more knowledgeable about the topic. A lecture was provided utilizing a PowerPoint presentation, as well as a pre-test and post-test in order to assess whether comprehension of NPPE increased, decreased, or remained consistent in order to better understand the topics relevancy to anesthesia practice. All three points are imperative for overall positive patient outcomes.

A provider must be able to prevent NPPE, however if it is not preventable then a provider must be able to recognize its presentation and appropriately intervene to prevent potential morbid outcomes. Literature was not definite on how to prevent it, nevertheless it can be prevented most of the time with diligence. Diligently preventing laryngeal irritation through suctioning and anesthetic depth was shown to be key. The goal was to observe an increase from the pre-test scores by at least a 10% margin. The post-test resulted in an overall increase by 30% from the pre-test thus surpassing the 10% goal with a statistical significance of  $P < .05$ . The pre-test and post-test results indicated a substantial increase in understanding and provided proof of the validity of the PowerPoint learning tool in teaching future SRNAs about prevention, recognition and treatment of NPPE.

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Negative pressure pulmonary edema (NPPE) is a serious medical problem that can occur at any stage throughout the perioperative process once anesthesia has been induced. The development of NPPE can be arrested or prevented completely with a number of different strategies. Unrecognized NPPE can quickly turn into a medical emergency while NPPE itself can significantly prolong one's hospital stay. Anesthesia providers and postoperative care unit (PACU) nurses should be alert to the potential development of NPPE and what to do in the event that it occurs.

The purpose of this project was to highlight, after an extensive literature review, how to prevent, recognize and treat episodes of NPPE during the perioperative period. Although this is a fairly rare occurrence, it is important to educate medical professionals on the condition so that there will be some familiarity should it happen. Gupta (2012) states, "The present case emphasizes the importance of constant vigilance and early diagnosis. Immediate re-establishment of the airway, adequate oxygenation, and application of positive airway pressure represent the cornerstones of the therapy" (p) 648. This showed that this topic is of the utmost importance to anesthesia providers as well as nurses caring for patients who have been recently anesthetized.

### **Review of Literature**

A preliminary literature review was done; articles from within the last five years were identified and reviewed. Articles were chosen based on their information covering the prevention, recognition and treatment of NPPE in the perioperative period. The plan involved acquiring a solid body of journal articles and case studies covering this topic. Taking these articles, an in-depth review of the findings was done.

Negative pressure pulmonary edema or NPPE is a rare nevertheless severe postoperative complication that is poorly understood and grossly misdiagnosed amongst anesthesia providers. NPPE is used interchangeably with several different terminologies one being term post extubation pulmonary edema. The use of the term, post-extubation pulmonary edema, stems from the likelihood of this phenomenon occurring during emergence from general anesthesia (Pathak, Rendon, & Ciubotaru, 2011). According to Bhattacharya et al. (2016), the incidence of NPPE occurrence is less than 0.1% of all NPPE recorded cases, however, it is projected that this number could be as high as up to 11% following acute upper airway obstruction.

Although much research has been conducted on the topic of NPPE, considerable debate remains about the definitive causes and pathophysiology of this phenomenon. Education on NPPE remains deficient amongst anesthesia providers despite published data and research (Lemyze & Mallat, 2014). The need for educating anesthesia providers is a critical step in preventing the deleterious effects of NPPE with particular attention focused on the post-extubation period. It is estimated that up to 70% off all NPPE cases occur immediately after extubation (Gupta, Ardavan, Hall, & Palese, 2012). One common cause of NPPE that many researchers agree on is the occurrence of laryngospasm immediately following extubation.

Laryngospasms are common perioperative events that often result without long-term negative patient outcomes. However, when laryngospasms are not detected early and treated

aggressively NPPE may ensue, triggering detrimental health outcomes (Ghofaily, Simmons, Chen, & Liu, 2013). Statistics have shown that over 10 % of patients who experience a laryngospasm perioperatively will develop NPPE although these occurrences often remain undetected (Singh, Nakra, Shankar, & Jacob, 2012). Utilization of an LMA during general anesthesia may be identified as a possible triggering agent in inducing laryngospasms. Other risk factors for laryngospasms include but are not limited to the presence of an upper respiratory infection, male gender, young age, and a history of increased reactive airway disease. Laryngospasms are further exacerbated by the presence of irritating stimuli to the mucosal lining of the nasopharynx, which may trigger the closure of the vocal cords.

Although there are many conflicting theories on the etiology and pathophysiology of NPPE, the majority of researchers agree that upper airway obstructions against a closed glottis opening have a strong correlative function in the provocation of NPPE. Literature shows that one of the main causes of NPPE is laryngospasm. Laryngospasms result through a mechanism that involves the laryngeal reflex arc. This reflex arc consists of nerve pathways, which include the trigeminal, glossopharyngeal and superior and inferior laryngeal branches of the vagus nerve via afferent pathways, which innervate the mucosal linings of the nasopharynx to the vocal cords. Any stimulation of this reflex pathway can quickly cause the vocal cords to close. Stimuli include secretions, blood, temperature changes and aspiration of gastric contents. During a prolonged laryngospasm, clinical manifestation will be such that the patient becomes bradycardic followed by tachycardia (Ghofaily et al., 2012).

Normal intrathoracic pressure ranges from -3 – 10cmH<sub>2</sub>O. However, negative pleural pressures produced by forced inspiration against a closed glottis can generate greater than -100 cmH<sub>2</sub>O (Pathak, Rendon, & Ciubotaru, 2011). The ability to generate enough negative force

against an upper airway obstruction is known to produce a marked increase in the intrapulmonary hydrostatic pressure causing a transcapillary flux of fluid from the capillary interstitial space into the alveoli. Interestingly enough, the drastic increase in hydrostatic pressure within the capillary membrane bases does not increase intrapulmonary arterial pressure as it is safeguarded against changes in pressure by compensatory systemic venous return.

Despite the severity of NPPE, the occurrence is often short lived usually resolving within 48 hours after the initial insult. The inability of the negative pressure pulmonary episode to last is largely due in part of the inability of the lungs to maintain a state of constant hydrostatic pressure. Resolution of the intrapulmonary edema is aided by the reuptake of sodium into the lung interstitium via sodium channels found in type 1 and type 2 epithelial cells lining the alveoli. This shift of sodium restores the osmotic gradient of the intrapulmonary capillary bed allowing for edematous fluid to be reabsorbed into the lymphatic system. (Bhattacharya, Kallet, Ware, & Matthay, 2016) NPPE can affect unilateral or bilateral lungs after the manifestation of an upper airway obstruction.

NPPE is largely considered a non-cardiogenic pulmonary edema, which causes a shift of intrapulmonary fluid into the interstitium of the lungs following the generation of large amounts of negative pressure against an obstructed airway during attempts of respiratory inspiration. Although little change is seen in the pulmonary arterial pressure there is a noted increase in the overall pulmonary vascular resistance formed by inevitable periods of hypoxia. Ultimately, hypoxia may result in strain on the right ventricle of the heart and may cause left ventricular diastolic dysfunction secondary to a leftward shift of the intra-ventricular myocardial septum (Pathak, Rendon, & Ciubotaru, 2011).

Bajwa & Kulshrestha (2012) identified nine different factors that could potentially lead to NPPE. These factors include hanging, laryngeal tumor, strangulation and sleep apnea. Other factors include biting of the endotracheal tube as well as croup and epiglottitis in children. The administration of paralyzing agents at the start of an inhalational induction has also been implicated in NPPE due to paralysis of the glossal muscles before the diaphragm. Despite these many factors, the common factors remain to be post-extubation laryngospasm. Ghofaily, Simmons, Chen & Liu (2012) identified several risk factors for perioperative laryngospasm are male gender, history of reactive airway disease, the presence of a dry cough, young age, and a recent upper respiratory tract infection.

Preventative measures against NPPE revolve mainly around prophylaxis. Understanding populations that are the greatest risk for NPPE can vastly decrease the occurrence of events leading to high negative intrathoracic pressures. Bhaskar & Fraser (2011) further differentiate NPPE into two main categories, Type I or Type II. Type I NPPE most commonly occurs following an acute airway obstruction. Causes of Type I NPPE include hanging, upper airway tumor, strangulation, foreign body aspiration, epiglottitis, choking, croup and strangulation. In addition, near drowning, goiter, endotracheal tube obstruction and mononucleosis have all been identified as causative factors in Type I NPPE.

In contrast to Type I NPPE, Type II occurs as a result of relief of a chronic upper airway obstruction such as removal of large tonsils, hypertrophic adenoids or a redundant uvula. According to Bhaskar & Fraser (2011) “The incidence of developing Type I NPPE associated with acute postoperative upper airway obstruction is 9.6–12%, whereas the incidence of developing Type II NPPE is 44%. In adults about 50% of NPPE occurrences are due to postoperative laryngospasm” (p. 309).



Prompt recognition of laryngospasms should first be treated by discontinuation of any stimuli followed by administration of 100% oxygen with continuous positive pressure ventilation. Inability to open the vocal cords with this traditional method further necessitates pharmacological intervention. According to Ghofaily et al. (2013), administration of an IV anesthetic induction agent should be administered followed by the intravenous administration of succinylcholine at a dose of 0.5 mg/kg. If there is a contraindication to the usage of succinylcholine then atropine 0.01mg/kg can be used. Failure of positive pressure and pharmacological agents to break a laryngospasm should be followed by immediate endotracheal intubation with the addition of PEEP in order to maximize alveolar surface area to increase oxygen saturation and promote gas exchange while preventing further alveolar damage.

Clinical manifestations of an acute airway obstruction that may prompt early recognition of NPPE include but are not limited to suprasternal and supraclavicular retractions, panicked facial expressions, intercostal retractions and stridor. Signs and symptoms of NPPE can soon follow the relief of a prolonged laryngospasm. Respiratory distress with an inability to maintain an  $SPO_2 > 95\%$  along with the presence of the hallmark signs of pink frothy sputum can be the first indication of NPPE. Auscultation findings of coarse rhonchi and wheezing along with diffuse bilateral interstitial and alveolar infiltrates are also telltale signs of NPPE after the occurrence of a laryngospasm (Ghofaily, Simmons, Chen, & Liu, 2013).

Further management of NPPE includes maintaining mechanical ventilation in the Intensive Care Unit (ICU) setting. Pulmonary edema can be managed with the use of diuretics and steroids although this treatment method remains debatable. Computed Tomography (CT) can aid in differential diagnosis with evident preferential central and nondependent dissemination of ground-glass attenuation noting areas of edema and microhemorrhage. Although NPPE most

commonly occurs after immediate post-extubation laryngospasm, some studies have reported NPPE occurring up to twelve hours later. NPPE generally resolves within twenty-four hours.

### **Project Description**

After submission of the project proposal to the Institutional Review Board (IRB) and the Scientific Review Committee (SRC), a PowerPoint presentation was done, using the information retrieved from the extensive literature review, to disseminate these findings to the focus group. The focus group consisted of ADU NAP students graduating in 2017 and 2018.

Before the presentation, a pre-test was administered to the above-mentioned ADU NAP 2017 and 2018 cohorts in order to discover what the baseline knowledge on this topic is. The goal of the presentation was to inform and educate SRNA's on negative pressure pulmonary edema in a manner that is applicable to practice. Once the presentation was done a post-test was given to the same group to determine if knowledge was increased thus legitimizing the project.

### **Evaluation Plan**

Before the presentation, a pre-assessment of the 2017 and 2018 ADU NAP classes was conducted to determine baseline knowledge on the presented topic. Once the pretest was completed a PowerPoint was discussed covering the gathered evidence-based findings retrieved from extensive research and literature review. Immediately following a brief question and answer period a post-test was administered to the group. The pretest and the posttest were in a multiple-choice format, consisting of ten questions. Both tests had the same questions and answers.

The data from the pre and posttests was analyzed for their statistical value as it pertained to increasing the working knowledge of the topic. Students were also be given access to the PowerPoint presentation in order to review the core concepts of the project in order to reinforce the teaching points. A comparison of the pretest and posttest scores was executed. A ten percent improvement in scores from the pretest to the posttest was the bar for determining if there was worthwhile learning from the presentation. Although many recommendations and cited literature has been studied to recognize and treat NPPE, no single intervention has been proven in the prevention of it. Perhaps the most vital intervention in preventing the incidence of negative pressure pulmonary edema is to recognize and avoid laryngeal irritants in order to reduce the occurrence of laryngospasm (Bhaskar & Fraser, 2011).

### **Results and Conclusions**

In reviewing the results of the pre-test amongst 42 SRNA's surveyed, it was determined that the student base knowledge in regards to negative pressure pulmonary edema was elementary. The pre-test consisted of ten questions, which included multiple choice, true/false, and multiple-select options. The pre-test mean result was 42.24% with a standard deviation of 17.873 and a standard error mean of 2.758. Consequently, pre-test results substantiated the need

for an expansion in knowledge in the areas of prevention, recognition, and treatment of NPPE. After completion of the power point presentation, all 42 SRNA's were given a post-test which consisted of the same identical ten questions presented in the pre-test in order to assess the quality of erudition.

Post-test results were collected upon completion by all participants. A significant increase in the post-test mean score was noted as 71.19% from the pre-test score of 42.24%. The post-test standard deviation was 22.654 with a mean standard error of 3.496. The acquired t-value of the presentation was -23.601 with an associated p-value of <.05 level of confidence, therefore it could be concluded that the results were statistically significant. Based on the pre-test and post-test statistical analysis it is possible to conclude that an increase in overall SRNA knowledge in regards to prevention, recognition, and treatment of NPPE was attained.

Capstone project data analysis:

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair1	PreTest	40.24	42	17.873	2.758
	PostTest	71.19	42	22.654	3.496

Paired Samples Test

		Paired Differences				t	df	Sig. (2-tailed)	
		Mean	Std.Devi- -ation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	PreTest- PostTest	-30.952	8.500	1.312	-33.601	-28.304	-23.601	41	.000

The initial desired outcome was that there would be an overall 10% increase from the pre and post-test scores. The final difference between pre-test and post-test scores was determined to be a total of 28.95%, impressively surpassing the 10% goal. However, this project is not without

limitations. All 42 participants were seated in close proximity in a large classroom with connecting tables which left an opportunity for open and unmonitored discussion which could significantly skew the results of both the pre and post test. Furthermore, there was only one hour of total class time in which consents had to be passed out signed and returned before the pre-test was distributed, possibly creating a sense of hurriedness amongst students to complete the pre-test without carefully reading questions the questions that were being asked.

This was particularly noted in the pre-test in which a question was asked for the students to select two answers with 25 students only selecting one answer. Only four students selected one option to the same question in the post-test. It should be noted that two students were unable to take the pre and post test due to late attendance during the pre-testing, therefore, indicating that pretest and post-test may have been higher than initially reported if these students had been allowed to participate. Overall, students appeared engaged and were able to actively participate during the presentation when prompted. Audiovisual teaching techniques were included in the PowerPoint in order to help incorporate different learning styles amongst students.

A plethora of interesting findings was found in researching NPPE. Prior to the study, the researchers were unaware of the vast clinical underestimation of the occurrence of NPPE amongst patients who experienced perioperative laryngospasms. Furthermore, it was unbeknown to the researchers that NPPE was classified into two different types, which included an array of different etiologies. Perhaps the greatest lesson learned by the researchers was that no specific methodology was proven to prevent NPPE, which indicates that further academic and clinical research is needed on the subject matter.

There are many implications to nurse anesthesia practice based on the research conducted. SRNA's should be able to recognize the patients who are at higher risks for acute

airway obstructions based on the different etiologies listed in the PowerPoint under type 1 vs type 2 NPPE. In addition, practitioners should also understand the measures to take when confronted with an active laryngospasm and that the presence of pink frothy sputum is the hallmark sign of NPPE. Prompt recognition, as well as prompt intervention, are perhaps the most important implications to practice as these measures may help to drastically reduce future morbidity and sequelae associated with NPPE.

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## Appendix A

### Survey Questions

1. When monitoring a patient during a suspected active laryngospasm with an LMA, the anesthesia provider can expect to see which initial correlative changes on the monitors?
  - A. Tachycardia followed by bradycardia and hypoxia
  - B. Initial rapid desaturation followed tachycardia and hypertension
  - C. Bradycardia followed by compensatory tachycardia and hypoxia
  - D. Severe hypoxia followed by bradycardia and asystolic arrest
  
2. A 50-year-old male patient is undergoing general anesthesia with an LMA for correction of an inguinal hernia. Upon emergence, the patient briefly bites down on the LMA before relaxing once again. The anesthesia provider notes an increase in peak airway pressures and a loss of ETCO<sub>2</sub>, saturation percentage begins to fall into the low 80's. The anesthesia provider suspects a laryngospasm but is not able to release the spasm with continuous positive pressure, what is the next course of action that should be taken?
  - A. IV administration of an induction agent to deepen the anesthetic
  - B. IV administration of Succinylcholine 0.1-0.5 mg/kg
  - C. IV administration Atropine 0.01mg/kg
  - D. Clearance of any airway secretions and immediate intubation with an ETT
  
3. The hallmark sign of negative pressure pulmonary edema (NPPE) is

- A. Cessation of ETCO<sub>2</sub> waveform with suprasternal and subclavicular retractions
  - B. Laryngospasm unrelieved by continuous positive pressure ventilation.
  - C. Sudden increase in peak pressure and decrease in minute ventilations.
  - D. The appearance of pink frothy sputum with concurrent auscultation of rales or rhonchi.
4. The main difference between Type I NPPE and Type II NPPE is (Select 2)
- A. Type I NPPE commonly develops after relief of a chronic upper airway obstruction
  - B. Type II NPPE develops immediately after the onset of an acute airway obstruction
  - C. Type I NPPE develops immediately after the onset of an acute airway obstruction
  - D. Type II NPPE commonly develops after relief of a chronic upper airway obstruction
5. All of the followings are preoperative risk factors for developing NPPE except for
- A. Presence of airway lesions
  - B. Upper airway surgery
  - C. Obesity
  - D. Obstructive sleep Apnea
  - E. All of the above are risk factors for NPPE
6. True or False. NPPE typically occurs immediately after extubation but can occur several hours into the postoperative phase.
- A. True
  - B. False
7. With prompt recognition and treatment NPPE generally resolves within
- A. 2-12 hours postoperatively
  - B. 24-48 hours postoperatively



- C. 48- 72 hours postoperatively
  - D. 72 hours – 1 week postoperatively
8. The most common cause of NPPE is
- A. Biting on the endotracheal tube during emergence
  - B. Postextubation laryngospasm
  - C. Increased neck circumference
  - D. Aspiration of gastric contents
9. Upon review of a Computed Tomography scan performed for differential diagnosis of a patient suspected of having developed NPPE, which radiological results are consistent characteristic findings in patients with NPPE?
- A. Preferential central and nondependent distribution of ground glass attenuation.
  - B. Preferential peripheral and dependent distribution of ground glass attenuation.
  - C. Preferential central and dependent distribution of ground glass attenuation.
  - D. Preferential Peripheral and dependent distribution of ground glass attenuation.
10. Attempts to inspire against an upper airway obstruction can generate intrathoracic negative pressures up to
- A. -3 to 10 cmH<sub>2</sub>O
  - B. -10 to -40 cmH<sub>2</sub>O
  - C. -3 to -40 cmH<sub>2</sub>O
  - D. -50 to -100 cmH<sub>2</sub>O

Answer Key

1. C

2. A

3. D

4. C & D

5. E

6. A (True)

7. B

8. B

9. A

10.D

## Appendix B

**ADU NAP CAPSTONE PROJECT – INFORMED CONSENT**

Our names are Mohammed Ahmad & Joshua Saladino and we are MSNA students in the Nurse Anesthesia Program (NAP) at Adventist University of Health Sciences (ADU). We are doing a Capstone Project called Prevention, Recognition & Treatment of Negative Pressure Pulmonary Edema. This project is being supervised by Manuel Tolosa. We would like to invite you to participate in this project. The main purpose of this form is to provide information about the project so you can make a decision about whether you want to participate.

**WHAT IS THE PROJECT ABOUT?**

The purpose of this project is to increase awareness about prevention, recognition, and treatment of NPPE among anesthesia providers

**WHAT DOES PARTICIPATION IN THIS PROJECT INVOLVE?**

If you decide to participate in this project, you will be asked to complete an anonymous pre-assessment, attend a classroom presentation, and then complete an anonymous post-assessment. The assessment methods to prevent, recognize and treat episodes of NPPE during the perioperative period. Your participation by attendance at the presentation and completion of the survey is anticipated to take approximately 40 minutes.

**WHY ARE YOU BEING ASKED TO PARTICIPATE?**

You have been invited to participate as part of a convenience sample of students currently enrolled in the ADU NAP. Participation in this project is voluntary. If you choose not to participate or to withdraw from the project, you may do so at any time.

**WHAT ARE THE RISKS INVOLVED IN THIS PROJECT?**

Although no project is completely risk-free, we don't anticipate that you will be harmed or distressed by participating in this project.

**ARE THERE ANY BENEFITS TO PARTICIPATION?**

We don't expect any direct benefits to you from participation in this project. The possible indirect benefit of participation in the project is the opportunity to gain additional knowledge how to prevent, recognize and treat episodes of negative pressure pulmonary edema during the perioperative period.

**HOW WILL THE INVESTIGATORS PROTECT PARTICIPANTS' CONFIDENTIALITY?**

The results of the project will be published, but your name or identity will not be revealed. To maintain the confidentiality of assessments, the investigators will conduct this project in such a way to ensure that information is submitted without participant identification by using a randomized numbering system that includes the same numerical identification for the pre-test, as

well as the post-test. By using a numerical identification system instead of participant names, the investigators will not have access to any participants' identities.

**WILL IT COST ANYTHING OR WILL I GET PAID TO PARTICIPATE IN THE PROJECT?**

Your participation will cost approximately 40 minutes of your time, but will require no monetary cost on your part. You will not be paid to participate.

**VOLUNTARY CONSENT**

By signing this form, you are saying that you have read this form, you understand the risks and benefits of this project, and you know what you are being asked to do. The investigators will be happy to answer any questions you have about the project. If you have any questions, please feel free to contact Mohammed Ahmad at Mohammed.ahmad@my.adu.edu or Joshua Saladino at Joshua.Saladino@my.adu.edu If you have concerns about the project process or the investigators, please contact the Nurse Anesthesia Program at (407) 303-9331.

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**Participant Signature**

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**Date**

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**Participant Name (PRINTED LEGIBLY)**

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 Project Chair: Dr. Manuel Tolosa, DNAP, CRNA  
 Nurse Anesthesia Program, Adventist University of Health Sciences

### History

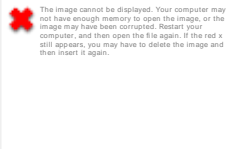
- Negative pressure pulmonary edema (NPPE), was first recognized in 1927 in spontaneously breathing dogs exposed to airway resistance.
- 1942 – Pathophysiological link between negative pressure and pulmonary edema.
- Later researchers discovered correlation between pulmonary edema and upper airway obstruction.

### What is NPPE?

- AKA post-obstruction pulmonary edema (POPE)
- Occurs in 0.05% - 0.1% of all anesthetic cases
- Thought to occur in up to 11% of patients needing intervention for acute upper airway obstructions
- Can be classified as Type 1 or Type 2 NPPE
- Cause is multifaceted but has a central mechanism

### What is NPPE?

- Pathak, Rendon, & Ciubotaru, 2011 states, "the ability to generate enough negative force against an upper airway obstruction is known to produce a marked increase in the intrapulmonary hydrostatic pressure causing a transcapillary flux of fluid from the capillary interstitial space into the alveoli".



### What is NPPE

- Upper airway obstruction causes increased pre/post capillary pulmonary vascular resistance in a nonuniform fashion creating overall increase in pulmonary vascular resistance & hyper-adrenergic state precipitating pulmonary edema.
- Generates a sudden large increase in preload
- SNS stimulation causes increased MAP/afterload leading to decreased stroke volume

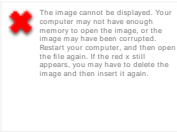
### Video

- [https://youtu.be/8puGL9\\_fJSs?t=3m23s](https://youtu.be/8puGL9_fJSs?t=3m23s)

1/13/17

### Pressures

- Normal intrathoracic pressures range from -3 to -10 cmH2O
- Forceful inspiration against closed glottis can generate pressures from -50 up to -100 cmH2O
- Disruption of the alveolar-capillary endothelial membrane allowing protein escape into alveoli




### Type 1

- Occurs after acute upper airway obstruction
- Commonly referred to as "laryngeal spasm-induced pulmonary edema".
- Can also occur after surgical manipulation of the airway
- Seen in young, healthy, athletic males

### Causes of Type 1 NPPE

<ul style="list-style-type: none"> <li>• Hanging</li> <li>• Strangulation</li> <li>• OSA</li> <li>• Obesity</li> <li>• Upper airway lesions/surgery</li> <li>• Foreign body</li> </ul>	<ul style="list-style-type: none"> <li>• Epiglottitis</li> <li>• Croup</li> <li>• Intraoperative ETT suctioning</li> <li>• Biting on LMA</li> <li>• ETT obstruction</li> <li>• Goiter</li> </ul>
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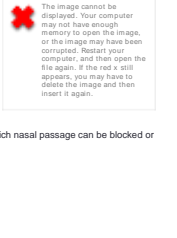


### Type 2

- Occurs after sudden relief of chronic upper airway obstruction and subsequent release of built-up auto-peep.
- Rapid return of chronically elevated lung pressures generates negative intra-pulmonary shifts of interstitial fluid into the lung interstitium.
- Less prevalent than type 1
- Seen in patients of extreme ages (very young and old).

### Causes of Type 2 NPPE

- Removal of large tonsils
- Hypertrophic adenoids
- Redundant uvula
- Choanal stenosis (Genetic disorder in which nasal passage can be blocked or narrowed)
- Removal of upper airway tumor



### Prevention


- Laryngospasm is most common cause of NPPE
- Laryngospasms are not uncommon, but if recognized and managed correctly, effects will be brief and reversible.
  - Mediated by reflex arc via cranial nerves V, IX, X which provide afferent pathways from nasopharynx to vocal cords

**Prevention**

- Factors increasing risk for laryngospasm
  - Recent upper respiratory infection
  - Male gender
  - Young age
  - Dry cough
  - History of reactive airway disease

**Prevention**

- Common stimuli of laryngospasm
  - Secretions
  - Blood
  - Gastric fluid
  - Pressure & Temperature changes
  - Smooth & skeletal muscle stimulation




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**Prevention**

- Suction airway before patient becomes "light" (avoid stage 2)
- Utilize soft bite block
- Maintain proper plane of anesthesia relative to current stimuli
- Utilize LTA (1% Lidocaine or 2% Tetracaine)
- Consider low dose (0.5mg/kg) Propofol prior to removing advanced airway

**Prevention**


- Remember- there is no intervention proven to prevent NPPE.
- So be diligent and prevent laryngeal irritation



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**Recognition**

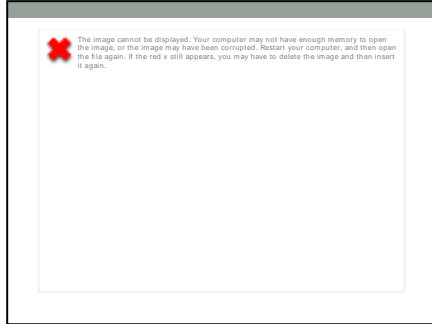
- Manifestation
  - Desaturation
  - Agitation
  - Tachypnea
  - Tachycardia
  - Rales on auscultation
  - Accessory muscle use
  - Panicked expression
  - Pink frothy sputum (Hallmark sign)
- **When in doubt, listen to the patient!**



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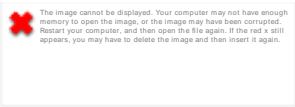
**Recognition**

- According to Bhaskar & Fraser (2011),
  - Classic CT finding "Preferential central and nondependent distribution of ground-glass attenuation (edema/hemorrhage)."



### Recognition

- Patient laryngospasms, what do you see?
  1. Sudden loss of ETCO2
  2. Initial bradycardia (common in children) due to vagus nerve innervation
  3. Hypoxia from continued laryngospasm
  4. Tachycardia & hypertension caused by override of PNS by SNS
  5. Bradycardia & asystolic arrest due to prolonged hypoxemia

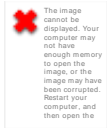


### Recognition

- Onset of NPPE can present within minutes to thirty hours after extubation or relief of obstruction
- Literature recommends post-operative monitoring of high risk patients for 2-12 hours

### Treatment

- Goal is to relieve the upper airway obstruction which will lead to NPPE
  - Stop stimulation
  - Suction airway
  - Continuous positive pressure with 100% O2
  - Deepen anesthetic with IV induction agent (Propofol 0.25-2mg/kg)
  - Succinylcholine 0.1-0.5mg/kg (IM 4mg/kg)
    - If succs contraindicated can give atropine 0.01mg/kg
  - Consider intubation if all else fails



### Treatment of NPPE

- Treatment is mainly supportive aimed at reducing hypoxemia
- Intubation and mechanical ventilation with PEEP
- Obtain CXR or chest CT
- Obtain ABG
- Diuretics and steroids (controversial)
- Scheduled respiratory breathing treatment
- NPPE usually resolves within 24-48 hours without sequelae

### Review

- Know your at risk populations
- Best treatment is prevention and awareness of predispositions
- Type 1 vs. Type 2 causes of NPPE
- Hallmark sign of NPPE is pink-frothy sputum
- Differential diagnosis with Chest CT




### Review

- Management of NPPE is largely supportive and aimed at reversing hypoxemia
- Post operative monitoring for 2 to 12 hours recommended
- Commonly associated with use of LMA
- Outcome depends on immediate recognition and treatment of upper airway obstruction
- Never extubate patient in Stage II of anesthesia


### Review

- Onset of NPPE occurs minutes to hours after relief of upper airway obstruction
- Anticipate and have ready necessary tools to relieve and treat airway obstruction and NPPE
- No concrete method proven to prevent NPPE
- Can lead to ARDS and multisystem failure.
- NPPE generally resolves within 24-48 hours postoperatively without further sequelae



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### Questions



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