Negative Pressure Pulmonary Edema: Have you seen it?

Deborah A. Geisler, CRNA, MNSA
Private Practitioner
Ponte Vedra Beach, Florida

NPPE: Reported Cases

- First described in 1927
- Correlation of airway obstruction pathophysiology and pulmonary edema reported in 1942
- First Pediatric case reported in 1973
- First Adult case reported in 1977
- From 1977 to 2000 there were 102 cases reported

Pulmonary Physiology
Pulmonary Physiology

Starling's Equation

\[ Jv = (Pc - Pt) - \sigma (\pi c - \pi t) \]

PE: Determining factors

- **Pulmonary Capillary Hydrostatic Pressure** \((Pc)\)
  - Driving force to force fluid out of the capillary
  - Difficult to measure in vivo
  - Normal value is 8-10 mmHg

- **Interstitial Hydrostatic Pressure** \((Pt)\)
  - Driving force that opposes capillary hydrostatic pressure
  - Negative pressure
  - Normal value around -7 mmHg

- **Capillary Colloid Osmotic Pressure** \((\pi c)\)
  - Determined by plasma protein concentration
  - Capillary membrane relatively impermeable to filtration of plasma proteins
  - Normal value is 24 mmHg

- **Interstitial Osmotic Pressure** \((\pi t)\)
  - Determined by interstitial protein concentration
  - Lower concentration of proteins in interstitium
  - Normal value is 14 mmHg
## Types of Pulmonary Edema

**Cardiogenic Pulmonary Edema**
- Increase in pulmonary capillary hydrostatic pressures
- Fluid shift into the interstitium

**Neurogenic Pulmonary Edema**
- Vasconstriction by excessive sympathetic output
- Increase in pulmonary capillary hydrostatic pressure
- Fluid shift into the interstitium

**High Altitude Pulmonary Edema**
- Pulmonary artery vasoconstriction or cerebral hypoxia

**Opioid Induced Pulmonary Edema**
- Change in capillary permeability
- Shift of protein rich fluid out of the capillary

**Negative Pressure Pulmonary Edema**

### Definition:
A form of pulmonary edema that appears to be related to a markedly negative intrathoracic pressure due to a forced inspiration against a closed upper airway... resulting in a transudation of fluid from the capillaries into the interstitium.
“Pick a Name”

- Upper airway obstruction pulmonary edema (UAOPE)
- Noncardiogenic pulmonary edema (NCPE)
- Postextubation laryngospasm induced pulmonary edema (LIPE)
- Post-anesthetic laryngospasm pulmonary edema (PLPE)
- Negative Pressure Pulmonary Edema (NPPE)

Clinical Events of NPPE

- **First Stage**
  - Laryngospasm
  - Respiratory efforts against a closed glottis
  - Generation of a large negative intra-thoracic pressure
- **Second Stage**
  - Relief of the obstruction
  - Ensuing pulmonary edema

Causative Factors of NPPE

- **Müller Maneuver**
  - Negative intrapleural pressure
    - Normal pressure is around -5 cm
    - May generate pressures as low as -50 cm to -100 cm
  - Increase venous return to the right heart
  - Engorgement of pulmonary vasculature
  - Elevated pulmonary capillary hydrostatic pressure
  - Resulting in pulmonary edema
Causative Factors of NPPE

- Mechanical stress from Müller maneuver
  - Injury to capillary membrane
  - Change in capillary permeability
  - Hypoxemia
  - Increase in pre and post capillary resistance
  - Alteration in capillary membrane integrity

Causative Factors of NPPE

- Hyperadrenergic State
  - Resulting from hypoxic vasoconstriction
  - CNS mediated alpha adrenergic discharge with peripheral vasoconstriction
  - Increase pulmonary vascular resistance
  - Decreased left ventricular output
  - Contributes to alteration of pulmonary capillary integrity

Causative Factors of NPPE

- Metabolic Acidosis
  - Resulting from hypoxic state
  - Metabolites are direct cardiac depressants, decreasing contractility

- Auto PEEP
  - Caused by Valsalva maneuver
  - Poorly recognized complication
  - Hyperinflation of the lungs
  - Compromise ventricular function by decreasing cardiac output
Causative Factors of NPPE

- Auto PEEP
  - Reduces in ventricular pre-load magnifies the effects of auto PEEP
  - Masks the appearance of pulmonary edema until obstruction is relieved

- Relief of obstruction
  - Removes auto PEEP
  - Pulmonary edema ensues

Causative Factors of NPPE

<table>
<thead>
<tr>
<th>Time Occurrence of NPPE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fixed Obstruction</td>
</tr>
</tbody>
</table>
  - Valsalva and Müller maneuvers of same magnitude
  - Signs of NPPE occur after relief of the obstruction

- Variable Obstruction
  - Müller maneuver more predominate
  - Signs of NPPE occur during the obstruction
Upper Airway Obstruction
Predisposing Factors

- Young “fit conscience” adult males
  - Well developed anterolateral muscles of neck
  - Able to generate high negative pressures in the obstructive phase of a laryngospasm
- Anatomically difficult intubation
  - Suggests an abnormal, potentially obstructive airway

Upper Airway Obstruction
Predisposing Factors

- Nasal, Oral, or Pharyngeal Surgery
  - Potential for post operative localized edema
  - NPPE occurs over 50% of the time after an otolaryngologic procedure
- Nasal, Oral, or pharyngeal pathology
- Obesity with obstructive apnea

Classic Clinical Presentation

- “Troubled Airway”
  - Stridor
  - Respiratory distress
  - Use of accessory muscle groups for breathing
Clinical Symptoms of NPPE

- Rapid onset
- Rapid resolution
- Dyspnea
- Tachypnea
- Cyanosis
- Sudden decrease in $\text{SaO}_2$
- Vital signs stable in face of decreasing $\text{SaO}_2$

Clinical Symptoms of NPPE

- Pink frothy secretions
- Pulmonary rales
  - Bilateral or unilateral in origin
- Invasive monitoring would show:
  - No decrease in left ventricular function
  - Normal CVP
  - Normal PAP
  - No change in PCWP

Differential Diagnosis of NPPE

- Aspiration of gastric contents
  - 1st alternative diagnosis
  - Aspirate differentials:
    - pH: 2.5
    - Volume: 40 cc's
- ARDS
  - V/Q mismatch
  - VS unstable
<table>
<thead>
<tr>
<th>Differential Diagnosis of NPPE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Congestive heart failure</strong></td>
</tr>
<tr>
<td>• Suspect iatrogenic overload</td>
</tr>
<tr>
<td>• Diagnostic tool: CVP</td>
</tr>
<tr>
<td><strong>Anaphylaxis</strong></td>
</tr>
<tr>
<td>• Classic signs</td>
</tr>
<tr>
<td>• VS unstable</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>NPPE: Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary treatment modalities</strong></td>
</tr>
<tr>
<td>• Close observation</td>
</tr>
<tr>
<td>• Maintain patent airway</td>
</tr>
<tr>
<td>• Oral Airway</td>
</tr>
<tr>
<td>• Intubation</td>
</tr>
<tr>
<td>• Mechanical ventilation</td>
</tr>
<tr>
<td>• Adequate oxygenation</td>
</tr>
<tr>
<td>• CPAP mask</td>
</tr>
<tr>
<td>• PEEP with mechanical ventilation to enhance oxygenation</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Secondary treatment modalities</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>• Diuretics</td>
</tr>
<tr>
<td>• Controversial because of origin of pulmonary edema</td>
</tr>
<tr>
<td>• Beneficial in assisting mobilization of fluid as capillaries heal</td>
</tr>
<tr>
<td>• Morphine or nitroglycerine ointment</td>
</tr>
<tr>
<td>• Increase venous capacity</td>
</tr>
<tr>
<td>• Helps decrease preload in fluid mobilization stage</td>
</tr>
<tr>
<td>• Midazolam to combat situation induced anxiety</td>
</tr>
</tbody>
</table>
Discrepancies of NPPE

Onset of NPPE resembles aspiration
- Predisposing factors
  - Struggling patient
  - Difficult airway
  - Possible failed intubation
- Clinical presentation
  - Wheezing
  - Dyspnea
  - Cyanosis
- Aspiration is "known" entity... leading to a quick misdiagnosis

Mild cases do not manifest
- Minimal signs and symptoms
- Benign clinical course
- Chest x-ray or arterial blood gas seems unwarranted
- Patient would be discharged without incident
- Post operative course prolonged because of "hacking cough" which is attributed to endotracheal intubation

Preventing NPPE

Recognize potential occurrence in select patient population
- Otolaryngological procedures
- Obese with obstructive sleep
- Patients with nasal, oral, or pharyngeal pathology
- Young "fit" adult males
- Difficult intubation
Preventing NPPE

- Monitor oxygenation saturation
  - Diagnostic tool in absence of other clinical manifestations
- Oral airway
  - Instrumental in preventing upper airway obstruction
- Appropriate LOC prior to extubation

Case Study I

- 28 yo muscular male with NKDA for elective ORIF of right ankle
  - Post extubation pt developed respiratory distress with vigorous respiratory efforts
  - Attempts at mask ventilation revealed an obstructed airway with rapid desaturation to below 50%
  - Laryngospasm ensued and was relieved with succinylcholine
  - High FiO₂ rendered an SaO₂ high of 83%

- Chest exam revealed diffuse rales and wheezing
- VSB: 160/80-110-30 no murmur or gallop detected
- Chest x-ray revealed pulmonary edema
- 15 minutes after relief of laryngospasm and on 5L/min face mask:
  - ABG: pH 7.37 P₉₅ 62 P₅₃ 53
### Case Study I

- Patient began coughing up copious amount of pink frothy secretions
- Coughing had subsided within 1 hour
- 18 hours after occurrence
  - ABG on RA: pH 7.39, PaO₂ 70, PaCO₂ 47
  - Chest x-ray showed clearing
- 24 hours after occurrence
  - ABG on RA: pH 7.39, PaO₂ 83, PaCO₂ 40

### Case Study II

- 52 yo Female for TAH
  - Significant physical findings include obesity and a short thick neck
  - Uneventful induction, but unable to intubate after numerous attempts by two practitioners
  - Oral airway inserted and the patient was awakened
  - Unable to remove oral airway b/o ensuing UAO
  - Pt. became more difficult to ventilate
  - Emergency tracheostomy was performed to establish an airway
  - Copious amount of pink frothy secretions were suctioned from tracheostomy
  - Hypotension occurred requiring dopamine and transfer to ICU
  - Invasive monitors revealed:
    - PA 32/14 PCWP 14 CO 5.2 L/min
    - ABG pH 7.34, PaO₂ 83, PaCO₂ 40
  - Physical exam revealed new dense right hemiparesis
  - CT of brain showed left cerebral infarct
Case Study II

- Patient required mechanical ventilation because of ARDS
- Bilateral pneumothoraces occurred due to high airway pressures
- Upon discharge, residual effects were right hemiparesis and moderate restrictive defect on pulmonary function testing

Case Study III

- 18 yo healthy male for emergency appendectomy
  - Surgical course uneventful
  - Upon emergence the patient began to resist the OTT, and was immediately suctioned and extubated
  - Laryngospasm developed after extubation
  - "Jaw lift" maneuver was applied after 3-4 attempts at spontaneous ventilation

Case Study III

- Pt. regained independent patent airway
- Transferred to ICU with SaO₂ of 90-93% on nasal cannula at 3L/min
- 4 hours later, he developed respirations distress, coughing, hemoptysis, and dyspnea
- Clinical findings
  - ABG: pH 7.50  PaO₂ 44  PCO₂ 44
  - Chest x-ray showed diffuse pulmonary edema
Case Study III

- Treatment
  - O₂ via Venturi mask, FIO₂ 40
  - Furosemide 40mg IV
- 3 hours later
  - ABG: pH 7.48, Pao₂ 114, Paco₂ 48
  - Clinical signs of obvious improvement
- 72 hours later
  - ABGs on RA normal range
  - Normal chest x-ray

Case Study IV

- 19 yo male: Excision of Nevus
- General Anesthesia: LMA
  - Nitrous/Forane
  - Duration: 45 minutes
- Patient taken to PACU with T-Piece
- Began to swallow, cough and bite the LMA
  - 5 minutes after LMA removal
    - Dyspnea
    - Hemoptysis

Case Study IV

- Transfer to ICU:
  - ABG: 7.36/40/99 Sat: 99%
- Discharged to floor 24 hours post-op
Thank you...

For your attention,
Questions?