NIV use in ED

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SQUH
Outline

• History & Introduction
• Overview of NIV application
• Review of proven uses of NIV
History of Ventilation

• 1940’s: Polio epidemics
• 1960’s: Rise of positive pressure ventilation
• 1980’s: Resurgence of interest in NIV in Treatment of hypoventilation & neuromuscular syndromes
• 1990’s: use of NIVs in acute respiratory failure
NIV decrease rate of intubation

NIV decrease mortality

Advantages of NIV

• Easy removal & application.
• Avoid complications of ETI
  – Barotrauma
  – Ventilator associated Pneumonia
  – Sedation
• Airway reflexes preserved
• Comfortable to patient
• May allow eating, talking, coughing
Selecting patients for NIV

- Know conditions NIV works for the best
  - COPD
  - Acute pulmonary edema
- Subjective dyspnea with respiratory rate > 25 breaths /min
- Use of accessory muscles
- PaCO2 > 45 mm Hg with pH ≤ 7.35
- PaO2/FiO2 < 200 mm Hg
- Conscious and cooperative (with possible exception of COPD: see text)
- Proper mask fit
CPAP

• CPAP: Continuous Positive Pressure Ventilation:
  – A single set pressure applied during all phases of the respiratory cycle
**BiPAP**

- **BiPAP**: Bi-level Positive Airway Pressure
  - Delivers 2 levels of pressure to the patient: IPAP & EPAP.
  - IPAP = EPAP + PS.
Basics of respiratory mechanics important to mechanical ventilation

- Pressure Difference
- Gas Flow
- Volume Change
PHYSIOLOGIC: IPAP

• IPAP: inspiratory cycle
  – augments respiratory effort: Increases Vt, decreases RR
  – Unload respiratory muscles
PHYSIOLOGY: CPAP

• Stent open upper airway
  – reduce obstruction
• Stent open lower airway expiratory cycle
  – overcome atelectasis
• Increase size of RV & FRC
  – Improve oxygenation
• Overcome intrinsic peep
  – intrinsic peep>> difficulty in generating pressure gradient for flow

- CPAP is most useful for patients who primarily have hypoxic respiratory failure.
Respiratory Failure

Hypoxemic
- Pulmonary Edema (CHF)
- ARDS
- Pneumonia
- Pulmonary Fibrosis
- Pulmonary Trauma
- Pulmonary Hemorrhage

Hypercapnic
- Obstructive airway
  - COPD
  - Asthma
- Neuropathy/myopathy:
  - Myasthenia gravis
  - Guillain-Barre
- OSA
Normal Inspiration
During a normal inspiration, the diaphragm descends, pulling the pleura apart, producing a negative pressure in the alveoli. This causes a pressure gradient from the upper airway leading to gas flow.

Normal End Expiration
Normally at end expiration the pressure in the alveoli is equal to atmospheric pressure.

Gas Trapping at End Expiration (Auto PEEP)
Gas is trapped in the alveoli at end expiration (due to obstruction) applying a positive pressure.

Auto PEEP - Inspiration
In order to permit gas flow when gas is trapped in the alveoli, the pleura and diaphragm must work very hard to generate a pressure such that the alveolar pressure becomes negative in relation to atmospheric pressure: a large imposed work of breathing.
The use of external PEEP in the setting of auto-PEEP may be conceptualized by the "waterfall over a dam" analogy. In this analogy, the presence of dynamic hyperinflation and 10 cmH2O of auto-PEEP is represented in the top panel by the reservoir of water trickling over the dam represented by the solid block. In the middle panel, as long as the external PEEP is less than or equal to the amount of auto-PEEP, the amount of water in the upstream reservoir, representing dynamic hyperinflation, does not increase. However, once the amount of water in the reservoir does increase (bottom panel), dynamic hyperinflation worsens.
Giving CPAP to a patient who has auto-PEEP

- The increased work of breathing associated with auto-PEEP can be offloaded by applying CPAP to the trachea/mouth, and splinting open the connecting airways.
NIV modes

• Pressure modes

• Volume modes

• **Pressure-cycled vents are better tolerated than volume-cycled vents**
Spontaneous Modes

- Analogous to PS in invasive ventilation.
- Spontaneous mode depends on patient effort to trigger inhalation.
- The patient's inspiratory effort triggers the switch from EPAP to IPAP. The inspiratory phase cycles off, and the machine switches back to EPAP when it detects a cessation of patient effort.
- Tidal volume ($V_t$) varies breath to breath and is determined by degree of IPAP, patient effort, and lung compliance.
Timed mode

• the IPAP/EPAP cycling is purely machine-triggered, at a set rate, typically expressed in breaths per minute (BPM).

• Similar to PC
Spontaneous/timed (ST) mode

- In spontaneous/timed mode a "backup" rate is also set to ensure that patients still receive a minimum number of breaths per minute if they fail to breathe spontaneously.

- Similar to SIMV
S/T mode

- CPAP 5-8
- IPAP 12-15
- RR 8-15
- Ti 1s
- FiO2 0.4-1.0 (titrate to SpO2)
Protocol for initiation of noninvasive positive pressure ventilation

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<tbody>
<tr>
<td>1.</td>
<td>Appropriately monitored location, oximetry, respiratory impedance, vital signs as clinically indicated</td>
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<tr>
<td>2.</td>
<td>Patient in bed or chair at &gt;30-degree angle</td>
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<td>3.</td>
<td>Select and fit interface</td>
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<tr>
<td>4.</td>
<td>Select ventilator</td>
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<tr>
<td>5.</td>
<td>Apply headgear; avoid excessive strap tension (one or two fingers under strap)</td>
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<tr>
<td>6.</td>
<td>Connect interface to ventilator tubing and turn on ventilator</td>
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<tr>
<td>7.</td>
<td>Start with low pressure in spontaneously triggered mode with backup rate; pressure limited: 8 to 12 cmH₂O inspiratory pressure; 3 to 5 cmH₂O expiratory pressure</td>
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<td>8.</td>
<td>Gradually increase inspiratory pressure (10 to 20 cmH₂O) as tolerated to achieve alleviation of dyspnea, decreased respiratory rate, increased tidal volume (if being monitored), and good patient–ventilator synchrony</td>
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<td>9.</td>
<td>Provide O₂ supplementation as needed to keep O₂ saturation &gt;90 percent</td>
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<td>10.</td>
<td>Check for air leaks, readjust straps as needed</td>
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<td>11.</td>
<td>Add humidifier as indicated</td>
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<td>12.</td>
<td>Consider mild sedation (e.g., intravenously administered lorazepam 0.5 mg) in agitated patients</td>
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<td>13.</td>
<td>Encouragement, reassurance, and frequent checks and adjustments as needed</td>
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<td>14.</td>
<td>Monitor occasional blood gases (within 1 to 2 hours) and then as needed</td>
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Setting EPAP

• It is reasonable to start higher, around 8 to 12 cm H2O, when providing CPAP therapy for ACPE.
• studies that found a benefit to CPAP used these initial levels
Mask intolerance

- Patients often complain about the tightness of the interface when it is first applied. Allowing the anxious patient to hold the mask in place while low amounts of PEEP are first applied (3–5 cm H2O) is a technique these authors have used with some anecdotal success.
BiPAP Graphics

Patient Monitoring

- Inspiratory Pressure
- Expiratory Pressure
- Breath Rate
- Duration of Inspiratory Phase for Timed Breath
- Exhaled Tidal Volume
- Exhaled Minute Ventilation
- % Patient Triggered Breaths
- Inspiratory Time/Total Cycle Time
- Patient Leak
- Measured Peak Inspiratory Pressure
- Oxygen Concentration
- Time to Reach Inspiratory Pressure
NIV unlikely to work

- Severe hypoxemia (PaO2/FiO2 <75),
- Severe acidemia
- Multi organ failure or slowly reversible disease (in short term)
- Uncooperative patient
- Encephalopathy with inability to protect airways and a high risk of aspiration
- Increased risk of aspiration: copious secretions, vomiting or severe gastrointestinal bleeding
- Recent airway or gastrointestinal surgery
- Inability to fit mask
Criteria for discontinuation of NIPPV and intubation

• Mask intolerance and poor adherence
• Failure to improve dyspnea, gas exchange (e.g., PaO2/FiO2 ≤146 [or ≤175 for ARDS] after 1 hr of NIPPV)
• Failure to improve mental status within 30 min
• Hemodynamic instability, cardiac ischemia or arrhythmias
• Difficulties with secretions management
Disadvantage of NIV

• Delay in intubation
• Acute unrecognized deterioration
• Aspiration
• Poor tracheal toileting
• Abdominal distention (GE sphincter pressure up to 25 cmH₂O)
• Difficult transport
• Poor tolerance
• Facial pressure necrosis, local barotrauma.
NIV effect on Hemodynamics

• Decrease Preload:
  – Increases in intrathoracic pressure impede venous return.
  – Hypovolemic patients are the mostly

• Left Ventricular output:
  Increases in intrathoracic pressure also assist the left ventricle by lowering cardiac afterload.
<table>
<thead>
<tr>
<th>Level of Evidence</th>
<th>Strength of Recommendation†</th>
<th>Location</th>
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<tbody>
<tr>
<td><strong>A: High</strong></td>
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<tr>
<td>Severe exacerbation of COPD (pH &lt; 7.35 with hypercarbia)</td>
<td>Recommend NIPPV</td>
<td>ICU, RCU, ward</td>
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<tr>
<td>Cardiogenic pulmonary edema ‡</td>
<td>Recommend NIPPV or CPAP</td>
<td>ICU, RCU</td>
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<tr>
<td><strong>B: Moderate</strong></td>
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<tr>
<td>Immunocompromised with hypoxemic failure</td>
<td>Suggest NIPPV</td>
<td>ICU, RCU</td>
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<tr>
<td>Facilitate weaning in patients with COPD</td>
<td>Suggest NIPPV</td>
<td>ICU, RCU</td>
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<tr>
<td>Preventive after extubation in patients with high reintubation risk (chronic lung disease/ PaCO₂ &gt; 45)</td>
<td>Suggest NIPPV</td>
<td>ICU</td>
</tr>
<tr>
<td>Extubation failure in patients without COPD</td>
<td>Suggest against use of NIPPV</td>
<td>ICU</td>
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<tr>
<td><strong>C: Low</strong></td>
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<tr>
<td>ALI/ARDS</td>
<td>Recommend against CPAP</td>
<td>ICU</td>
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<td>Option for NIPPV</td>
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<tr>
<td>Asthma/status asthmaticus</td>
<td>Option for NIPPV</td>
<td>ICU, RCU</td>
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<td>Extubation failure in patients with COPD</td>
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<td>Preventive after extubation in patients with low reintubation risk</td>
<td>Suggest against use of NIPPV</td>
<td>ICU</td>
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Non-invasive positive pressure ventilation for treatment of respiratory failure due to exacerbations of chronic obstructive pulmonary disease.

Ram FS, Piot J, Lightowler J, Wedzicha JA.

**Abstract**

**BACKGROUND:** Non-invasive positive pressure ventilation (NPPV) is being used increasingly in the management of patients admitted to hospital with acute respiratory failure secondary to an exacerbation of chronic obstructive pulmonary disease (COPD).

**OBJECTIVES:** To determine the efficacy of NPPV in the management of patients with respiratory failure due to an acute exacerbation of COPD.

**SEARCH STRATEGY:** An initial search was performed using the Cochrane Airways Group trials register and other relevant electronic databases. An updated search was conducted in September 2003 and another in April 2004.

**SELECTION CRITERIA:** Randomised controlled trials comparing NPPV plus usual medical care (UMC) versus UMC alone were selected. Trials needed to recruit adult patients admitted to hospital with respiratory failure due to an exacerbation of COPD and with PaCO\(_2\) > 6 kPa (45 mmHg).

**DATA COLLECTION AND ANALYSIS:** Two reviewers independently selected articles for inclusion, evaluated methodological quality of the studies and abstracted the data.

**MAIN RESULTS:** Fourteen studies were included in the review. NPPV resulted in decreased mortality (Relative Risk 0.52; 95%CI 0.35 to 0.76), decreased need for intubation (RR 0.41; 95%CI 0.33 to 0.53), reduction in treatment failure (RR 0.48; 95%CI 0.37 to 0.63), rapid improvement within the first hour in pH (Weight Mean Difference 0.03; 95%CI 0.02 to 0.04), PaCO\(_2\) (WMD -0.40 kPa; 95%CI -0.78 to -0.03) and respiratory rate (WMD -3.08 bpm; 95%CI -4.26 to -1.89). In addition, complications associated with treatment (RR 0.38; 95%CI 0.24 to 0.60) and length of hospital stay (WMD -3.24 days; 95%CI -4.42 to -2.08) was also reduced in the NPPV group.

**REVIEWERS’ CONCLUSIONS:** Data from quality randomised controlled trials show benefit of NPPV as first line intervention as an adjunct therapy to usual medical care in all suitable patients for the management of respiratory failure secondary to an acute exacerbation of COPD. NPPV should be considered early in the course of respiratory failure and before severe acidosis ensues, as a means of reducing the likelihood of endotracheal intubation, treatment failure and mortality.
Non-invasive positive pressure ventilation (CPAP or bilevel NPPV) for cardiogenic pulmonary oedema.

Vital FM¹, Ladeira MT, Atallah AN.

Abstract

BACKGROUND: This is an update of a systematic review previously published in 2008 about non-invasive positive pressure ventilation (NPPV). NPPV has been widely used to alleviate signs and symptoms of respiratory distress due to cardiogenic pulmonary oedema. NPPV prevents alveolar collapse and helps redistribute intra-alveolar fluid, improving pulmonary compliance and reducing the pressure of breathing.

OBJECTIVES: To determine the effectiveness and safety of NPPV in the treatment of adult patients with cardiogenic pulmonary oedema in its acute stage.

SEARCH METHODS: We searched the following databases on 20 April 2011: CENTRAL and DARE, (The Cochrane Library, Issue 2 of 4, 2011); MEDLINE (Ovid, 1950 to April 2011); EMBASE (Ovid, 1980 to April 2011); CINAHL (1982 to April 2011); and LILACS (1982 to April 2011). We also reviewed reference lists of included studies and contacted experts and equipment manufacturers. We did not apply language restrictions.

SELECTION CRITERIA: We selected blinded or unblinded randomised or quasi-randomised clinical trials, reporting on adult patients with acute or acute-on-chronic cardiogenic pulmonary oedema and where NPPV (continuous positive airway pressure (CPAP) or bilevel NPPV) plus standard medical care was compared with standard medical care alone.

DATA COLLECTION AND ANALYSIS: Two authors independently selected articles and abstracted data using a standardised data collection form. We evaluated study quality with emphasis on allocation concealment, sequence generation allocation, losses to follow-up, outcome assessors, selective outcome reporting and adherence to the intention-to-treat principle.

MAIN RESULTS: We included 32 studies (2916 participants), of generally low or uncertain risk of bias. Compared with standard medical care, NPPV significantly reduced hospital mortality (RR 0.66, 95% CI 0.48 to 0.89) and endotracheal intubation (RR 0.52, 95% CI 0.36 to 0.75). We found no difference in hospital length of stay with NPPV; however, intensive care unit stay was reduced by 1 day (WMD -0.89 days, 95% CI -1.33 to -0.45). Compared with standard medical care, we did not observe significant increases in the incidence of acute myocardial infarction with NPPV during its application (RR 1.24, 95% CI 0.79 to 1.95) or after (RR 0.70, 95% CI 0.11 to 4.26). We identified fewer adverse events with NPPV use (in particular progressive respiratory distress and neurological failure (coma)) when compared with standard medical care.

AUTHORS’ CONCLUSIONS: NPPV in addition to standard medical care is an effective and safe intervention for the treatment of adult patients with acute cardiogenic pulmonary oedema. The evidence to date on the potential benefit of NPPV in reducing mortality is entirely derived from small-trials and further large-scale trials are needed.
Randomized, prospective trial of bilevel versus continuous positive airway pressure in acute pulmonary edema.

Mehta S¹, Jay GD, Woolard RH, Hipona RA, Connolly EM, Cimini DM, Drinkwine JH, Hill NS.

Abstract

OBJECTIVE: To evaluate whether bilevel positive airway pressure, by actively assisting inhalation, more rapidly improves ventilation, acidemia, and dyspnea than continuous positive airway pressure (CPAP) in patients with acute pulmonary edema.

DESIGN: Randomized, controlled, double-blind trial.

SETTING: Emergency department in a university hospital.

PATIENTS: Twenty-seven patients, presenting with acute pulmonary edema, characterized by dyspnea, tachypnea, tachycardia, accessory muscle use, bilateral rales, and typical findings of congestion on a chest radiograph.

INTERVENTIONS: In addition to standard therapy, 13 patients were randomized to receive nasal CPAP (10 cm H2O), and 14 patients were randomized to receive nasal bilevel positive airway pressure (inspiratory and expiratory positive airway pressures of 15 and 5 cm H2O, respectively) in the spontaneous/timed mode that combines patient flow-triggering and backup time-triggering.

MEASUREMENTS AND MAIN RESULTS: After 30 mins, significant reductions in breathing frequency (32 +/- 4 to 26 +/- 5 breaths/min), heart rate (110 +/- 21 to 97 +/- 20 beats/min), blood pressure (mean 117 +/- 28 to 92 +/- 18 mm Hg), and Paco2 (56 +/- 15 to 43 +/- 9 torr [7.5 +/- 2 to 5.7 +/- 1.2 kPa]) were observed in the bilevel positive airway pressure group, as were significant improvements in arterial pH and dyspnea scores (p < .05 for all of these parameters). Only breathing frequency improved significantly in the CPAP group (32 +/- 4 to 28 +/- 5 breaths/min, p < .05). At 30 mins; the bilevel positive airway pressure group had greater reductions in Paco2 (p = .05), systolic blood pressure (p = .005), and mean arterial pressure (p = .03) than the CPAP group. The myocardial infarction rate was higher in the bilevel positive airway pressure group (71%) compared with both the CPAP group (31%) and historically matched controls (38%) (p = .05). Duration of ventilator use, intensive care unit and hospital stays, and intubation and mortality rates were similar between the two groups.

CONCLUSIONS: Bilevel positive airway pressure improves ventilation and vital signs more rapidly than CPAP in patients with acute pulmonary edema. The higher rate of myocardial infarctions associated with the use of bilevel positive airway pressure highlights the need for further studies to clarify its effects on hemodynamics and infarction rates, and to determine optimal pressure settings.

Comment in

Changes in cardiac output do not explain the higher rate of myocardial infarction associated with the use of bilevel compared with continuous positive airway pressure. [Crit Care Med. 1998]
Acute Asthma Exacerbation

• Few small, randomized trials evaluated BPAP in asthma.
  – No decrease in intubation rate
  – No patient died in these trials.

• Although the benefit of NIV in asthma has not been demonstrated in large, multicenter randomized trials, no demonstrable harm from this intervention has been detected.

• Its routine use cannot be recommended, but, in select cases of severe asthma, NIV should be considered.
Pneumonia

• the literature on NIV for patients with CAP has produced mixed results.
• Prospective trials have demonstrated failure rates as high as 50%.
• A single, randomized, controlled trial evaluating standard therapy and standard therapy plus NIV in 56 patients with CAP found no improvement in the mortality rate. Patients who received NIV did have lower rates of intubation and shorter lengths of stay in an intermediate care unit.
• An additional randomized, controlled trial evaluated patients with ARF of varying causes. In this study, patients with CAP who were treated with NIV had lower intubation rates and a lower mortality rate in the intensive care unit.
ALI/ARDS

- Agarwal et al. in a meta-analysis concluded that NIV is unlikely to have any significant beneficial outcomes.
- Few small prospectine & randomised trial have found trend toward avoiding intubation and improving mortality.
- Most of these studies done in ICU setting.
- Careful selection and close monitoring when NIV is used should be implemented.
Pre-oxygenate & delayed sequence intubation
Delayed sequence intubation: a prospective observational study.

Weingart SD¹, Trueger NS², Wong N³, Scoff J⁴, Singh N⁵, Rudolph SS⁶.

Study Objective: We investigate a new technique for the emergency airway management of patients with altered mental status preventing adequate preoxygenation.

Methods: This was a prospective, observational, multicenter study of patients whose medical condition led them to impede optimal preintubation preparation because of delirium. A convenience sample of emergency department and ICU patients was enrolled. Patients received a dissociative dose of ketamine, allowing preoxygenation with high-flow nonrebreather mask or noninvasive positive pressure ventilation (NIPPV). After preoxygenation, patients were paralyzed and intubated. The primary outcome of this study was the difference in oxygen saturations after maximal attempts at preoxygenation before delayed sequence intubation compared with saturations just before intubation. Predetermined secondary outcomes and complications were also assessed.

Results: A total of 62 patients were enrolled: 19 patients required delayed sequence intubation to allow nonrebreather mask, 39 patients required it to allow NIPPV, and 4 patients required it for nasogastric tube placement. Saturations increased from a mean of 89.9% before delayed sequence intubation to 98.8% afterward, with an increase of 8.9% (95% confidence interval 6.4% to 10.9%). Thirty-two patients were in a predetermined group with high potential for critical desaturation (pre-delayed sequence intubation saturations ≤93%). All of these patients increased their saturations post-delayed sequence intubation; 29 (91%) of these patients increased their post-delayed sequence intubation saturations to greater than 93%. No complications were observed in the patients receiving delayed sequence intubation.

Conclusion: Delayed sequence intubation could offer an alternative to rapid sequence intubation in patients requiring emergency airway management who will not tolerate preoxygenation or peri-intubation procedures. It is essentially procedural sedation, with the procedure being preoxygenation. Delayed sequence intubation seems safe and effective for use in emergency airway management.

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Take home messages

• Applicability of NIV is expanding to many causes of ARF in ED.
• Awaiting better evidence in some conditions it still could be used as a bridging device before intubation.
• careful selection and close monitoring of patients will increase chance of successful NIV trial.