

In the name of God

NIV in Acute Respiratory Failure

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NIV in acute respiratory failure

Noninvasive ventilation (**NIV**) refers to the delivery of positive pressure ventilation through a noninvasive interface (e.g, nasal mask, face mask, or nasal plugs), rather than an invasive interface (endotracheal tube, tracheostomy).

NIV can be used as ventilatory support for patients with acute or chronic respiratory failure.

NIV in acute respiratory failure

There are two type of acute respiratory failure;

TYPE I; ACUTE HYPOXEMIC RESPIRATORY FAILURE

This type of respiratory failure occurs with alveolar flooding and subsequent ventilation-perfusion mismatch and intrapulmonary shunt physiology.

Alveolar flooding may be a consequence of pulmonary edema, lung injury, pneumonia, or alveolar hemorrhage. (PaO₂ < 60 mmHg)

TYPE II; RESPIRATORY FAILURE: HYPERCAPNEIC RESPIRATORY FAILURE

This type of respiratory failure is a consequence of alveolar hypoventilation and results from the inability to eliminate carbon dioxide effectively. (Pco₂ > 45 mmHg,

PH < 7.35)

Clinical indications for NIV in adults with acute respiratory failure ^{3, 4, 6-8, 11-13}

- Acute exacerbation of COPD – in the context of acute hypercapnic respiratory failure (pH <7.35 and elevated PaCO₂)
- Obesity hypoventilation syndrome (OHS) – in the context of acute hypercapnic respiratory failure (pH <7.35 and elevated PaCO₂)
- Neuromuscular disease (NMD) – consider NIV in acute respiratory distress, with a high work of breathing or difficulty with mucus clearance.
- Acute cardiogenic pulmonary oedema, CPAP (hypoxic respiratory failure) or AHRF
- Immunocompromised patients with acute respiratory failure
- Acute pneumonitis, including COVID-19, moderate-to-severe hypoxic respiratory failure PaO₂/FiO₂ >150mmHg, CPAP
- Weaning high-risk patients from mechanical ventilation
- Post-extubation management
- Post-operative acute respiratory failure
- Chest trauma

Contraindications to noninvasive ventilation

Absolute

The need for emergent intubation (eg, cardiac or respiratory arrest, severe respiratory distress, unstable cardiac arrhythmia)

Relative

Nonrespiratory organ failure that is acutely life-threatening

Severe encephalopathy (eg, GCS <10)

Severe upper gastrointestinal bleeding

Hemodynamic instability

Facial or neurological surgery, trauma, or deformity

Significant airway obstruction (eg, laryngeal mass or tracheal tumor)

Inability to cooperate, protect airway, or clear secretions (eg, patients at high risk of aspiration)

Anticipated prolonged duration of mechanical ventilation (eg, ≥ 4 to 7 days)

Recent esophageal or gastric anastomosis*

Multiple contraindications

Insufficient staffing support

NIV in acute respiratory failure

The best candidates for NIV in Acute Respiratory Failure;

Acute cardiogenic pulmonary edema (ACPE)

Acute exacerbation of chronic obstructive pulmonary disease (AECOPD) with hypercapnic respiratory acidosis

Acute exacerbation of chronic obstructive pulmonary disease (AECOPD)

Evidence from large randomized trials and meta-analyses consistently indicates that **BPAP** improves important clinical outcomes in patients having an AECOPD complicated by hypercapnic acidosis;

50 percent reduction in mortality

Reduced the rate of intubation by 65 percent

Reduced hospital length of stay

Reduced complications unrelated to NIV (e.g. ventilator-associated pneumonia, multiorgan failure)

Improve indices of gas exchange.

Acute exacerbation of chronic obstructive pulmonary disease (AECOPD)

The mechanism by which **BPAP** improves hypercapnic respiratory failure in **AECOPD** is due to *improved alveolar ventilation* as evidenced by improved respiratory mechanics (eg, decreased respiratory rate, an increased tidal volume, and an increased minute ventilation) and improved gas exchange parameters (eg, increase in arterial oxygen tension [PaO_2] and decrease in the PaCO_2).

While data in the past suggested that patients with severe exacerbations were more likely to benefit from BPAP, a 2017 Cochrane analysis of 17 trials reported no differences between those with mild (defined as a pH between 7.3 and 7.35) or severe exacerbations (defined by those with a pH <7.3) .

Acute exacerbation of chronic obstructive pulmonary disease (AECOPD)

While **BPAP** is the mode of choice, continuous positive airway pressure (**CPAP**), pressure support ventilation (**PSV**), and other modes (eg, neurally adjusted ventilatory assist [**NAVA**]) have also been shown to result in success in patients with acute hypercapnic respiratory failure due to AECOPD. However, these modes are less likely to improve alveolar ventilation and do not have a strong body of evidence to support their use in patients with AECOPD. Modes like PSV and NAVA may be suitable for those who find BPAP uncomfortable or who are dyssynchronous with the ventilator.

Acute exacerbation of chronic obstructive pulmonary disease (AECOPD)

Initial settings

Bilevel NIV

- Start with low pressure in spontaneously triggered mode with backup rate: Inspiratory pressure at 8 to 12 cm H₂O; expiratory pressure at 3 to 5 cm H₂O
- Gradually increase inspiratory pressure (10 to 20 cm H₂O) as tolerated to achieve alleviation of dyspnea, decreased respiratory rate, increased tidal volume (if being monitored), and good patient-ventilator synchrony
- Provide O₂ supplementation as needed to keep O₂ saturation >90%

NIV in acute respiratory failure

Acute cardiogenic pulmonary edema (ACPE)

The mechanism by which NIV improves ACPE is thought to be due to preload reduction, the prevention of alveolar collapse at end expiration, and decreased left ventricular afterload.

NIV decreases the need for intubation,
improves clinical and laboratory indices of respiratory failure (e.g, heart rate, dyspnea, hypercapnia, acidosis), and
improves mortality
but did not have any impact on hospital length of stay.

Acute cardiogenic pulmonary edema (ACPE)

CPAP

- CPAP level at 5 to 8 cm H₂O
- Gradually increase CPAP level as tolerated (up to 20 cm H₂O) to achieve improvement in dyspnea and reduction in respiratory rate
- Provide O₂ supplementation as needed to keep O₂ saturation >90%

NIV in acute respiratory failure

After NIV is initiated, the patient should be observed closely for the first few minutes to troubleshoot any initial problems . Many patients do not initially tolerate NIV due to **discomfort** or **ventilator dyssynchrony** (i.e, the phases of ventilator breath do not match that of the patient) . Ventilator dyssynchrony may be due to one or more issues including auto-positive end-expiratory pressure (auto-PEEP), reduced flow due to an interface leak, or discomfort and anxiety.

NIV in acute respiratory failure

Once the patient is comfortable, vital signs, oxygenation, and mental status are observed for the next one to two hours in a closely monitored setting . At that point, we typically re-evaluate the patient clinically and obtain arterial blood gases. When the patient demonstrates improvement in clinical signs and symptoms and gas exchange, we typically persist with NIV . However, if they do not improve or show signs of deterioration at any time during the trial, NIV should be abandoned and the patient promptly intubated.

NIV in acute respiratory failure

Clinical criteria suggesting failure include;

worsening gas exchange, increasing respiratory rate, worsening encephalopathy or agitation, inability to clear secretions, inability to tolerate any of the interfaces, or hemodynamic instability .

NIV in acute respiratory failure

In practice, a significant amount of clinical judgement is required when assessing the response to NIV. For example, for patients who do not demonstrate improvement and who have a do-not-intubate order in place, increasing the settings or changing to a full-face mask may be appropriate with a view to re-evaluation in another one to two hours. Similarly, in patients who are minimally improved but in whom further optimization of medical therapy is pending (e.g, further diuresis, bronchodilators, inotropic support, or ultrafiltration), it is not unreasonable to persist with NIV provided the delay in administering treatment is relatively short. Some patients may demonstrate partial improvement, such as improved respiratory acidosis but no improvement in oxygenation, in which case increasing the fraction of inspired oxygen (FiO_2) or switching to a mode that provides PEEP may help.

NIV in acute respiratory failure

Weaning

A patient is deemed ready to wean when gas exchange and clinical parameters of acute respiratory failure have improved dramatically and the cause of respiratory failure has improved. While there are no universal physiologic parameters that support a readiness for weaning from NIV, we consider the following as reasonable:

A respiratory rate ≥ 12 and ≤ 22 breaths per minute.

Peripheral oxygen saturation (SpO_2) ≥ 90 percent on ≤ 60 percent FiO_2 or predicted needs can be met with oxygen delivered via high flow nasal cannulae (HFNC) or low flow oxygen.

Hemodynamic stability (preferably off or on low dose vasopressors and heart rate ≥ 50 and ≤ 120 beats per minute).

The pH is preferably > 7.25 and the patient should ideally be afebrile, awake and alert, or easily arousable.

Minimal NIV settings (eg, bilevel positive airway pressure 10 cm H_2O /5 cm H_2O or continuous positive airway pressure ≤ 10 cm H_2O).

NIV in acute respiratory failure

there is no universal protocol for weaning and in general, it is individualized and dependent upon the patient's response to decreased support. Weaning from NIV may be accomplished by progressively decreasing the amount of positive airway pressure, by permitting the patient to be disconnected from the NIV for progressively longer durations, or a combination of both.



Thank you