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Non-Invasive Ventilation In Patients With Acute Pulmonary Edema

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Abstract

Acute pulmonary edema (APE) is a life-threatening condition with a high mortality rate. It has two types: cardiogenic, which usually happens when a previously asymptomatic condition decompensates, and non-cardiogenic, which is brought on by other factors. Hypoxemia and decreased pulmonary compliance are the results of both types of ventilation impairment. In order to reduce venous return, lower left ventricular afterload, and relieve pulmonary edema, management involves medication and noninvasive ventilation (NIV). As a result, vital signs improve, and the chance of needing intubation decreases. BiPAP and CPAP, primarily used in cardiogenic APE, enhance hemodynamics, oxygenation, and breathing effort. They are more successful in symptom relief than oxygen therapy alone, but no conclusive evidence exists that they lower short-term mortality. Both cardiogenic and non-cardiogenic APE patients can benefit from NIV since it lowers respiratory effort and dyspnea without requiring endotracheal intubation. By increasing intrathoracic pressure through positive pressure, NIV improves pulmonary compliance and reduces respiratory effort. Studies indicate that Noninvasive Pressure Support Ventilation (NIPSV) and Continuous Positive Airway Pressure (CPAP) are equally effective in reducing mortality and intubation rates. However, NIPSV is associated with faster resolution of respiratory failure, though it does not significantly improve final clinical outcomes over CPAP. Thus, while both modalities are effective, NIPSV may be preferred for quicker recovery. In order to maximize oxygenation, lessen respiratory effort, and enhance cardiac function, initiating NIV in APE necessitates precise ventilator settings and vigilant monitoring. Despite its benefits, NIV is contraindicated in cases of airway protection failure, severe hemodynamic instability, facial trauma, severe gastrointestinal issues, massive hemoptysis, excessive secretions, cardiac arrest, and upper airway obstruction. NIV remains a first-line treatment for APE due to its effectiveness and safety profile. It is a well-tolerated intervention with adverse event rates comparable to drug therapy alone, making it a key component in the management of acute pulmonary edema.

Keywords: Acute Pulmonary Edema, APE, Non-Invasive Ventilation, NIV, Continuous Positive Airway Pressure, CPAP, Cardiogenic Pulmonary Edema, Non-Cardiogenic Pulmonary Edema, BiPAP.

Introduction

Acute pulmonary edema (APE) is a life-threatening condition associated with high global mortality rates. Characterized by hemodynamic instability and impaired gas exchange, APE can rapidly progress to acute respiratory failure, necessitating urgent intervention . Current management combines pharmacological therapies - such as opioids, vasodilators, and diuretics - with noninvasive ventilation (NIV) [1, 2].

NIV modalities, including bilevel positive airway pressure (BiPAP) and continuous positive airway pressure (CPAP), are pivotal in managing APE. The use of noninvasive positive pressure

ventilation traces back to the early 20th century, initially applied in thoracic surgery and later adopted for cardiac patients in the 1930s. However, its integration into emergency care for respiratory failure began in the 1990s [3].

As a therapeutic tool, NIV aims to prevent respiratory collapse, reduce the need for invasive procedures like orotracheal intubation (OTI), shorten hospital stays, and lower mortality rates (2–4). By decreasing systemic venous return and left ventricular afterload, NIV reduces pulmonary congestion, stabilizes vital signs, and reduces the likelihood of OTI [4].

While NIV accelerates symptom resolution and corrects metabolic disturbances more rapidly than standard oxygen therapy, its impact on short-term mortality remains unclear . This study evaluates the efficacy of NIV in APE and identifies factors influencing its success, providing insights to optimize clinical outcomes [5, 6].

Pathophysiological Basis For Niv In Ape

Cardiogenic and non-cardiogenic pulmonary edema differ fundamentally in etiology. Cardiogenic edema arises from left ventricular (LV) dysfunction, leading to elevated pulmonary capillary pressure and subsequent fluid transudation into alveolar spaces. In contrast, non-cardiogenic edema [e.g., acute respiratory distress syndrome (ARDS)] results from increased alveolar-capillary membrane permeability due to systemic inflammation or direct lung injury [7].

Although cardiogenic and non-cardiogenic pulmonary edema may exhibit similar clinical manifestations, their management differs significantly. NIV is more commonly applied in cardiogenic cases, and the selection of NIV mode and settings should be tailored to the patient's hemodynamic condition and potential complications.

NIV improves ventilation and oxygenation in acute cardiogenic pulmonary edema through multiple mechanisms. Intrathoracic pressure (ITP), elevated by positive pressure ventilation, plays a central role. By reducing venous return, NIV lowers cardiac preload, alleviating pulmonary congestion – a critical benefit in heart failure. However, hypovolemic patients may experience excessive preload reduction, leading to hypotension from inadequate LV filling. Simultaneously, NIV reduces LV afterload by decreasing the transmural pressure gradient across the LV wall, facilitating ejection and improving cardiac output in heart failure. This dual effect on preload and afterload underscores NIV's hemodynamic benefits in cardiogenic edema [8-11].

The impact of ITP on right ventricular (RV) function is more nuanced. Elevated ITP increases pulmonary vascular resistance, potentially exacerbating RV afterload in patients with pre-existing RV dysfunction. Thus, careful patients' selection and monitoring are essential.

Beyond hemodynamic effects, NIV enhances gas exchange by recruiting collapsed alveoli, decreasing ventilation-perfusion mismatch and expanding the surface area available for gas exchange. Additionally, elevated ITP redirects edema fluid from the alveolar space back into the vascular system, improving oxygenation [12-14].

The hemodynamic effects of positive pressure ventilation are complex and influenced by the patient's volume status, baseline cardiac function, and specific ventilation settings such as positive end-expiratory pressure (PEEP). For example, preload reduction may dangerously reduce cardiac output in hypovolemia, while afterload reduction optimized cardiac performance in heart failure. Thus, meticulous titration on ventilatory settings is essential to optimize hemodynamic results and prevent complications such as hypotension [15-17].

Collectively, these mechanisms – preload/afterload modulation, alveolar recruitment, and edema redistribution – improve ventilation, oxygenation, and cardiac function in acute cardiogenic pulmonary edema, reducing respiratory effort and stabilizing hemodynamics.

Modalities and Clinical Evidence of Niv In Ape

The two primary NIV modalities are CPAP and pressure support with PEEP; the latter often delivered as BiPAP. CPAP increases ITP, improving oxygenation, reducing respiratory effort, and lowering LV afterload. BiPAP combines inspiratory pressure support with PEEP, when combined with conventional pharmacotherapy, accelerates the recovery of vital signs and arterial blood gas parameters in cardiogenic APE, reducing the need for invasive mechanical ventilation [18, 19].

In hypoxemic respiratory failure, NIV aims to maintain adequate PaO2 until the underlying cause resolves. For cardiogenic pulmonary edema, goals extend to improving cardiac output by optimizing preload and afterload. While CPAP focuses on stabilizing alveolar recruitment, BiPAP provides additional inspiratory support, directory reducing respiratory effort [20].

Compared to CPAP, BiPAP produces greater improvements in oxygenation, carbon dioxide clearance, and respiratory effort reduction. Berbenetz et al. found no significant difference in myocardial infarction rates between BiPAP and CPAP when adjusted for baseline risk, suggesting that earlier observations of harm may have been confounded by disease severity. In the same study Berbenetz et al. highlight reductions in in-hospital mortality and adverse outcomes [21].

The 3CPO trial (ISRCTN07448447), a landmark study investigating NIV (CPAP and NIPPV) compared to standard oxygen therapy in the treatment of acute cardiogenic pulmonary edema, demonstrated that while NIV rapidly improves symptoms and corrects physiological derangements, it does not reduce short-term mortality. This finding contrasts with earlier meta-analyses suggesting mortality benefits, underscoring NIV's role as adjunctive therapy for severe respiratory distress or pharmacological non-responders, rather than a survival-enhancing intervention [22].

Comparative studies of ventilatory modalities reveal distinct efficacy profiles. In acute cardiogenic pulmonary edema, NIPPV (BiPAP) outperforms both CPAP and standard oxygen therapy, significantly reducing intubation rates and accelerating improvements in respiratory rate, PaO_2, $PaCO_2$, and pH (19). CPAP shows intermediate efficacy, while oxygen therapy alone yields the poorest outcome. These findings align with Brito et al.'s analysis of CPAP and BIPAP in acute COPD exacerba-

tions, where both modalities improved lung function, but BiPAP uniquely reduced dyspnea without affecting hospitalization or mortality [23].

Nouira'sSS research corroborates these trends, showing comparable mortality and intubation rates between CPAP and NIPPC (specifically non-invasive pressure support ventilation, NIPSV). However, NIPPV achieved faster resolution of respiratory failure, suggesting its preference for rapid clinical stabilization. The study also found no increased risk of myocardial infarction with NIPSV, reinforcing its safety profile [24].

Consistent trends across studies highlight that both CPAP and NIPPV (BiPAP) are effective in cardiogenic pulmonary edema, reducing invasive interventions and improving physiological parameters. NIPPV, however, offers superior symptomatic physiological relief and faster recovery, likely due to its dual pressure support. Despite these benefits, neither does the mortality improve survival rates, emphasizing their role in symptom management rather than curative treatment [25].

The successful implementation of NIV also depends on selecting an appropriate patient interface, such as a facial or nasal mask. Facial masks provide higher pressure and allow oral breathing but are less comfortable, while nasal masks require a closed mouth to prevent leaks and are better tolerated in chronic respiratory failure. However, NIV interfaces such as facial masks are more invasive than conventional oxygen therapy (e.g., nasal cannula or simple facial masks) and may present limitations in emergency settings, particularly in patients with poor adherence, excessive mucus secretion, altered consciousness, or facial anatomical abnormalities [26].

High-flow nasal cannula (HFNC) therapy has emerged as a less invasive alternative. The 2021 ESC guidelines for acute heart failure acknowledge HFNC as a reasonable option for patients' intolerant to NIV, particularly in mild cases . HFNC improves respiratory rate, lactate levels, and oxygenation/ventilation in patients with heart failure and APE in the emergency department. HFNC can effectively replace conventional oxygen therapy as the initial treatment for cardiogenic pulmonary edema. While HFNC is valuable for initial stabilization, NIV (CPA/BiPAP) remains superior for rapid symptom relief in acute settings [27, 28].

Indications and Contraindications

NIV, including CPAP and BiPAP, is a first-line treatment for acute hypercapnic respiratory failure in COPD exacerbations or weaning, as well as hypoxemic conditions such as acute cardiogenic pulmonary edema and respiratory failure in immunocompromised patients. By improving respiratory mechanics and reducing left ventricular afterload, NIV stabilizes hemodynamics and gas exchange . The 2017 European Respiratory Society/ American Thoracic Society guidelines recommend NIV for:

- COPD exacerbations,
- Cardiogenic pulmonary edema,
- Post-extubation respiratory failure,
- Thoracic trauma (with pressure precautions),
- Respiratory acidosis ($PaCO_2 > 45$ mmHg, pH < 7.35),
- Persistent hypoxemia unresponsive to supplemental oxygen.

NIV is contraindicated in scenarios where safety and efficacy are compromised. Absolute contraindications include:

- Inability to protect the airway (e.g., Glasgow < 8, bulbar stroke, severe agitation),
- Severe hemodynamic instability (e.g., refractory shock, uncontrolled arrhythmia),
- Facial trauma, anomalies or surgery,
- Severe gastrointestinal conditions (active vomiting, obstruction, bleeding),
- Massive hemoptysis, excessive secretions, cardiac arrest, or upper airway obstruction, requiring immediate intubation [29].

Relative contraindications include mild hypotension, inability to expectorate secretions, uncooperative patients, and isolated RV failure. In clinical practice, patient selection must balance the urgency of respiratory support against risks such as aspiration or hemodynamic compromise, emphasizing individualized assessment.

Initial Ventilator Settings and Titration

The initiation of NIV in patients with APE requires careful selection of ventilator settings and close monitoring to optimize oxygenation, reduce the work of breathing, and improve cardiac function. CPAP is typically initiated at a pressure range of 5-10 cm H20, while BiPAP is started with an inspiratory positive airway pressure (IPAP) of 10-15 cm H2O and an expiratory positive airway pressure (EPAP) of 5cm H2O to support ventilation and alleviate pulmonary congestion . The fraction of inspiratory oxygen (FiO2) is usually set at 100% initially to rapidly improve oxygenation, with subsequent titration to maintain an oxygen saturation (SpO2) of ≥ 92%, thus minimizing the risk of hyperoxia-related vasoconstriction. A full-face mask is preferred as the initial interface to reduce air leaks and ensure effective pressure delivery. Careful titration of pressure is crucial, as excessive positive pressure may reduce cardiac output, whereas insufficient pressure may fail to relieve symptoms or improve gas exchange. Regular reassessment of respiratory rate, gas exchange, and hemodynamic parameters is essential to guide further adjustments and avoid complications such as barotrauma or hypotension. Clinical guidelines recommend close monitoring during the first hour of therapy, as the majority of improvements in respiratory distress occur within this time [30-34].

Complications

During NIV application, continuous monitoring is essential to assess treatment effectiveness (2). Key aspects to monitoring include altered consciousness, respiratory discomfort, patient tolerance, ventilatory parameters, ventilatory asynchrony, vital signs, and laboratory tests (e.g., arterial blood gas analysis). If any of these signs appear, the medical team should reassess whether to continue NIV or consider OTI [35]. After the first hour of NIV application, compared to drug therapy alone, no significant changes in blood pressure (systolic, diastolic, or mean) were observed. However, positive pressure reduced respiratory rate and increased arterial oxygen pressure. Even though NIV is usually well tolerated and effective, there is a chance that it will cause mild to more serious problems. The mask itself is typically the cause of minor issues like skin irritation, pressure sores, or general discomfort brought on by an improper fit [36, 37].

Gastric insufflation is a more serious issue because it can raise the risk of aspiration, especially in patients who have trouble protecting their airways . NIV can also affect blood flow and heart function, sometimes leading to low blood pressure—especially in patients who already have cardiovascular instability. Delaying intubation when a patient is not responding well to NIV carries a significant risk as well. When misapplied and for an extended period, it can have the opposite effect of what is intended. Although there is a decreased risk of hospital-acquired infections with NIV compared to invasive ventilation, careful patient selection and close monitoring are essential to minimizing complications [38].

When side effects like mask-related problems, gastric insufflation, or hypotension outweigh the advantages of continuing NIV, it is time to stop using it. If left untreated, mask-related problems like skin deterioration or discomfort should be discontinued, or other interfaces should be considered as they can result in poor tolerance. Stopping NIV may be necessary if gastric insufflation - which can happen when air leaks into the stomach - causes severe discomfort or increases the risk of aspiration. Another serious consequence that may necessitate stopping NIV is hypotension, which is caused by decreased preload because of elevated intrathoracic pressure. If this condition results in hemodynamic instability, it may be necessary to stop NIV [39].

The switch from NIV to a nasal cannula or reservoir mask must be handled carefully to ensure proper ventilation and oxygenation. During this transition, the NIV settings are gradually decreased while the patient's hemodynamics and respiratory condition are continuously monitored. Once the patient is stable on lower NIV settings, they can be moved to a nasal cannula or reservoir mask to maintain oxygen saturation within target ranges. Throughout this transition, ongoing observation is necessary to identify any indications of respiratory distress or decline quickly.

In situations where NIV is more detrimental than helpful, a therapeutic trial with a time limit (usually less than an hour) can help assess whether NIV should be continued. Close observation for clinical deterioration is essential if NIV is stopped, particularly in patients with chronic hypercapnic respiratory failure, who may develop respiratory muscle weakness and worsening arterial blood gas levels [40, 41].

Discussion

The success of NIV depends on factors such as synchronized breathing, lower disease severity (lower SAPS or APACHE scores), minimal air leaks and secretions, hypertension at admission, hypercapnia, and a positive response within the first hour. Effective therapy is also associated with pH correction, reduced respiratory rate, and an increased PaO_2/FiO_2 ratio. Additionally, proper interface selection, optimal patient-ventilator synchrony, patient comfort, and active participation play crucial roles in maximizing NIV effectiveness. Ventilation failure can be predicted by several criteria, such as severe disease severity (APACHE II, SAPS II, SOFA), advanced age, no improvement after 1 hour, and multiorgan involvement. Pre-morbid conditions, pH < 7.25, and $PaCO_2 \geq 75$ mmHg after 2 hours also indicate failure. Other causes include uncertain etiology of respiratory failure, ARDS, pneumonia, $PaO_2/FiO_2 < 150$ mmHg, and excessive respiratory effort.

Future developments and approaches to treating APE with NIV center on maximizing its effectiveness while reducing any possible side effects. In this situation, the main objectives of NIV are to increase ventilation and oxygenation by lowering cardiac preload and afterload, which can be accomplished by applying positive intrathoracic pressure [42].

One promising area of innovation is the refinement of NIV interfaces and modes. To improve patient comfort and lower the chance of air leaks, HFNO and helmet NIV are being investigated as potential substitutes for conventional face masks . By lowering left ventricular afterload and increasing cardiac output, these interfaces may also make it possible to administer PEEP more consistently, which is essential for treating acute pulmonary edema [43, 44].

The development of more protective NIV strategies is another area of focus. This includes preventing self-inflicted lung injury (P-SILI), which can happen when patients with lung injury make vigorous inspiratory efforts using higher PEEP levels and longer sessions. Studies have also investigated synchronized intermittent mandatory ventilation (SIMV), which may help treat hypercarbia and altered consciousness in acute cardiogenic pulmonary edema [45].

Incorporating sophisticated monitoring methods, like echocardiography, can also help customize NIV settings to meet the needs of specific patients by evaluating cardiopulmonary interactions and guaranteeing ideal preload and afterload circumstances . This individualized approach may improve NIV's efficacy and safety when treating acute pulmonary edema. NIV is still a mainstay in the treatment of acute pulmonary edema, but new research and technology are being developed to improve patient outcomes, lower the risk of complications, and fine-tune its use. The current knowledge of the physiological effects of NIV and the necessity of cautious patient selection and monitoring lend support to these innovations [46, 47].

Conclusion

The literature strongly supports the use of positive pressure as a first-line treatment for APE, as it is a safe therapeutic approach with adverse event rates like those of drug therapy alone. The positive pressure generated by NIV helps reduce dyspnea and respiratory effort, increases lung compliance, and has beneficial cardiac effects, such as improved cardiac output. NIV prevents alveolus collapse, maintains alveolar patency, and optimizes gas exchange. e reduces left ventricular afterload and improves cardiac output by decreasing venous return. This study reveals that NIV reduces mortality rates, the need for orotracheal intubation, and hospital length of stay. Finally, the study suggests that NIV should be standard practice for this patient population.

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