International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
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editorial-office@sciformat.ca

ARTICLE TITLE	NON-INVASIVE VENTILATION IN CARDIAC PULMONARY EDEMA – NARRATIVE REVIEW
DOI	https://doi.org/10.31435/ijitss.2(50).2026.5832
RECEIVED	25 March 2026
ACCEPTED	23 June 2026
PUBLISHED	29 June 2026
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NON-INVASIVE VENTILATION IN CARDIAC PULMONARY EDEMA – NARRATIVE REVIEW

Izabela Szubert *(Corresponding Author, Email: izabelaszubert2312@gmail.com)*
Independent Public Provincial Integrated Hospital in Szczecin, Szczecin, Poland
ORCID ID: 0009-0006-7162-6651

Julia Suchecka
Independent Public Provincial Integrated Hospital in Szczecin, Szczecin, Poland
ORCID ID: 0009-0001-9975-355X

Jakub Lamorski
Independent Public Provincial Integrated Hospital in Szczecin, Szczecin, Poland
ORCID ID: 0009-0004-5456-7413

Gabriela Stępień
University Clinical Hospital No. 2 PMU in Szczecin, Szczecin, Poland
ORCID ID: 0009-0006-4505-5162

Wiktor Nesterak
Pomeranian Medical University, Szczecin, Poland
ORCID ID: 0009-0009-5322-903X

Michał Chylewski
Pomeranian Medical University, Szczecin, West Pomerania, Poland
ORCID ID: 0009-0007-4789-1903

Karolina Chudecka
Independent Public Provincial Integrated Hospital in Szczecin, Szczecin, Poland
ORCID ID: 0009-0004-0262-5036

Aleksandra Kędzierska
Pomeranian Medical University, Szczecin, Poland
ORCID ID: 0009-0008-7050-4139

Urszula Przewoźna
University Clinical Hospital No. 2 PMU in Szczecin, Szczecin, Poland
ORCID ID: 0009-0003-2186-6604

Katarzyna Józwa
Independent Public Provincial Integrated Hospital in Szczecin, Szczecin, Poland
ORCID ID: 0009-0003-5615-2388

ABSTRACT

Non-invasive ventilation (NIV), including continuous positive airway pressure (CPAP) and bilevel positive airway pressure (BiPAP), is a cornerstone in the management of acute cardiogenic pulmonary edema (CPE). NIV improves oxygenation, reduces the work of breathing, and exerts favorable hemodynamic effects by decreasing preload and afterload. These physiological mechanisms contribute to rapid clinical stabilization in critically ill patients.

Current estimates suggest that the condition affects approximately 1 million patients with pulmonary edema secondary to cardiac causes. (1) Patients with CPE present with a wide range of symptoms, most notably dyspnea, reflecting the acute nature of the condition, underscoring the severity and clinical importance of the issue.

Overall, NIV represents an effective first-line supportive therapy in patients with acute CPE, although further research is needed to clarify its effect on long-term outcomes. Available data consistently demonstrate that NIV reduces the need for endotracheal intubation and improves short-term physiological parameters, particularly when initiated early. (2) However, its impact on mortality remains inconsistent across studies.

This review synthesizes current evidence on NIV in cardiogenic pulmonary edema, focusing on underlying mechanisms, clinical effectiveness, and comparative data on CPAP and BiPAP, with emphasis on their role in emergency and intensive care settings.

KEYWORDS

Cardiogenic Pulmonary Edema; Non-Invasive Ventilation; Acute Respiratory Failure; Heart Failure; CPAP; BiPAP; Emergency Care

CITATION

Izabela Szubert, Julia Suchecka, Jakub Lamorski, Gabriela Stępień, Wiktor Nesterak, Michał Chylewski, Karolina Chudecka, Aleksandra Kędzierska, Urszula Przewoźna, Katarzyna Józwa. (2026) Non-Invasive Ventilation in Cardiac Pulmonary Edema – Narrative Review. *International Journal of Innovative Technologies in Social Science*. 2(50). doi: 10.31435/ijitss.2(50).2026.5832

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Introduction

Cardiogenic pulmonary edema (CPE) is a common and life-threatening manifestation of acute heart failure, characterized by rapid deterioration in gas exchange and hemodynamic instability requiring urgent intervention. Early diagnosis and prompt initiation of appropriate therapy are essential for improving patient outcomes.

The use of NIV has shown a similar relative growth pattern across diverse causes of respiratory failure. Non-invasive ventilation (NIV) has become a cornerstone in the management of acute respiratory failure, particularly when avoidance of endotracheal intubation is desirable. One of its most well-established indications is the treatment of CPE

Between 2000 and 2009, the population-based utilization of NIV among patients hospitalized with acute respiratory failure in the absence of COPD increased from 6 to 39 cases per 100,000 U.S. residents, representing a 560% rise.(3)

NIV is delivered as continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BiPAP), which differ in pressure delivery and physiological effects. CPAP provides continuous positive airway pressure, primarily improving oxygenation and reducing cardiac preload. In contrast, BiPAP delivers both inspiratory and expiratory pressure support, enhancing ventilation in addition to oxygenation. (4)

Despite widespread clinical use, optimal patient selection, timing of initiation, and choice of ventilatory mode remain subjects of ongoing debate. Therefore, this narrative review aims to critically evaluate the current evidence on the use of NIV in CPE, with particular emphasis on physiological mechanisms, clinical outcomes, and the comparative effectiveness of different ventilatory strategies in emergency and intensive care settings.

Methods

This structured narrative review was conducted using the PubMed, Scopus, and Google Scholar databases. The search strategy included the following terms: *cardiogenic pulmonary edema*, *non-invasive ventilation*, *CPAP*, *BiPAP*, and *acute heart failure*. Studies published in English between 1991 and 2025 were considered.

Eligible studies included randomized controlled trials, meta-analyses, clinical guidelines, and observational studies with substantial sample sizes. Case reports, conference abstracts, and non–peer-reviewed articles were excluded.

Study selection was based on relevance to the topic, with emphasis on clinical outcomes associated with the use of non-invasive ventilation in cardiogenic pulmonary edema. Primary outcomes of interest included mortality, the need for endotracheal intubation, and intensive care unit (ICU) length of stay.

Due to heterogeneity in study design and outcome reporting, a qualitative synthesis of the evidence was performed. No formal assessment of study quality or risk of bias was performed, consistent with the narrative nature of the review.

Cardiogenic Pulmonary Edema: Pathophysiology

Acute cardiogenic pulmonary edema most commonly occurs in elderly patients and remains a frequent clinical emergency. Pulmonary edema is broadly classified into cardiogenic and non-cardiogenic forms. The condition is commonly precipitated by acute cardiovascular events, including acute coronary syndrome (ACS), tachyarrhythmias, valvular heart disease, and uncontrolled hypertension (5).

Cardiogenic pulmonary edema results from a complex interplay of hemodynamic overload, neurohormonal activation, and microvascular dysfunction, rather than solely from elevated hydrostatic pressure. Acute increases in left ventricular filling pressure lead to elevated pulmonary capillary hydrostatic pressure, exceeding the capacity of alveolar fluid clearance mechanisms.(6)

Under physiological conditions, the pulmonary interstitium maintains fluid balance maintained by Starling forces. An acute elevation in pulmonary capillary pressure

(>18 mm Hg) can lead to increased fluid filtration into the lung interstitium, exceeding the compensatory capacity of lymphatic drainage. That may contribute to progressive fluid accumulation in the interstitial space and subsequently the alveoli. The transition from interstitial edema to alveolar flooding depends on the integrity of the alveolar epithelial barrier. In CPE, sodium transport and water clearance may be impaired due to mechanical stress and neurohormonal activation.(7)

Neurohormonal activation plays a central role in disease progression. Sympathetic stimulation increases catecholamine release, leading to vasoconstriction, elevated afterload, and increased myocardial oxygen demand. Activation of the renin–angiotensin–aldosterone system further promotes sodium retention and intravascular volume expansion, exacerbating pulmonary congestion. The hyperadrenergic state is enhanced by hypoxemia. (8)

Inflammatory mechanisms may also contribute to disease progression. Although CPE is primarily considered a hydrostatic process, emerging evidence suggests that cytokine release, oxidative stress, and endothelial dysfunction may increase capillary permeability, particularly in advanced stages. These pathophysiological mechanisms provide a rationale for the use of non-invasive ventilation.

CPE – symptoms, diagnosing and common treatment

Clinically, CPE presents with acute dyspnea, orthopnea, tachypnea, pulmonary crackles, and signs of respiratory distress, potentially progressing to respiratory failure or cardiogenic shock. Common physical findings include a third heart sound, jugular venous distension, pulmonary rales, peripheral edema, wheezing, and ascites (9). Prompt reduction in the severity of dyspnea represents a key therapeutic goal and provides a clinically relevant measure of treatment effectiveness.(10)

Diagnosis is based on clinical assessment supported by arterial blood gas analysis, natriuretic peptides, imaging (chest radiography, echocardiography), and lung ultrasound, which provides a rapid and reliable bedside tool.

Treatment focuses on rapid stabilization of respiratory function and correction of hemodynamic abnormalities. First-line therapy includes diuretics, vasodilators, and non-invasive ventilation. Loop diuretics are widely used, although reduced responsiveness may occur in chronically treated patients. Vasodilators such as nitroglycerin effectively reduce cardiac filling pressures and improve symptoms. In selected patients with hypotension and hypoperfusion, inotropic support may be required.(11) The use of opioids remains controversial due to potential adverse effects and should be used with caution.(12)

Physiological Rationale for NIV

Non-invasive ventilation (NIV), including CPAP and BiPAP, exerts its therapeutic effects through combined respiratory and hemodynamic mechanisms. It is widely used in conditions such as obstructive sleep apnea, asthma exacerbations, and chronic obstructive pulmonary disease. In the 1990s, non-invasive ventilation was first introduced as a therapeutic ventilatory strategy for patients with acute respiratory failure (ARF) secondary to severe cardiogenic pulmonary oedema. (13) NIV is particularly relevant in acute CPE, where impaired gas exchange and cardiac dysfunction coexist and patients gain benefit from NIV on the grounds that it induces a more rapid improvement in respiratory distress and metabolic disorder than does standard oxygen therapy. (14)

A key mechanism of NIV is the application of positive end-expiratory pressure (PEEP), which promotes alveolar recruitment and improves ventilation–perfusion matching, thereby enhancing oxygenation. In pulmonary edema, fluid accumulation leads to alveolar collapse and reduced lung compliance; NIV counteracts these processes and helps restore functional residual capacity while reducing intrapulmonary shunting.

It also reduces the work of breathing by decreasing inspiratory effort and improving respiratory mechanics. This is particularly important in patients with respiratory distress, in whom excessive respiratory effort may lead to muscle fatigue and ventilatory failure.(15)

CPAP acts directly on alveolar units by maintaining positive pressure throughout the respiratory cycle. This effect increases end expiratory lung volume through reopening collapsed alveoli, which leads to increased lung compliance and mitigates pulmonary shunt. (16) Therefore, ventilation/perfusion match is enhanced and gas exchange improved.

In addition, NIV exerts important hemodynamic effects through increases in intrathoracic pressure. By elevating intrathoracic and right atrial pressures, PEEP reduces the pressure gradient between mean systemic filling pressure and right atrial pressure, leading to decreased venous return and reduced stroke volume. The use of NPPV resulted in decreased right and left ventricular preload, indicating improved cardiac function.(17) Continuous positive airway pressure (CPAP) reduces left ventricular afterload through attenuation of the negative intrathoracic pressure fluctuations associated with spontaneous breathing, as well as by decreasing transmural pressure (18)

CPAP provides a constant level of positive airway pressure throughout the respiratory cycle and is primarily associated with improved oxygenation. BiPAP, by delivering separate inspiratory (IPAP) and expiratory (EPAP) pressures, offers additional ventilatory support and is particularly beneficial in patients with hypercapnia or increased work of breathing. CPAP may also facilitate improved carbon dioxide clearance and normalization of pH, likely through optimization of respiratory mechanics and improved ventilation–perfusion coupling. (19)

Furthermore, positive airway pressure may contribute to the reduction of interstitial edema and airway resistance, thereby improving overall lung mechanics.

The effectiveness of NIV depends on appropriate patient selection, correct interface fitting, and close clinical monitoring. Failure of NIV requires timely escalation to invasive mechanical ventilation.

CPAP vs BiPAP: Clinical Considerations

CPAP provides continuous positive airway pressure and is primarily indicated in hypoxemic respiratory failure associated with pulmonary congestion. It is particularly effective in patients without significant hypercapnia.

BiPAP delivers both inspiratory and expiratory pressure support and is more suitable for patients with ventilatory failure, increased work of breathing, or coexisting hypercapnia. Bilevel positive airway pressure involves the application of two pressure levels: an inspiratory pressure delivered in synchrony with the patient's inspiratory effort (pressure support) and an expiratory pressure maintained throughout the respiratory cycle (positive end-expiratory pressure, PEEP). (20)

Current evidence does not demonstrate clear superiority of either modality in terms of mortality outcomes. However, non-invasive positive pressure ventilation (NPPV), as a class, has been shown to reduce hospital mortality. For example, Vital et al. demonstrated that NPPV significantly reduced hospital mortality compared with standard treatment (RR 0.66, 95% CI 0.48–0.89). (18) In contrast, Bersten et al. found no significant difference in in-hospital mortality between treatment groups. Relative to continuous positive airway pressure, bilevel positive airway pressure is associated with greater minute ventilation, improved carbon dioxide elimination, and reductions in both inspiratory effort and work of breathing. Bersten et al.

demonstrated that, after 30 minutes, arterial carbon dioxide tension decreased to a greater extent in patients receiving oxygen therapy combined with continuous positive airway pressure compared with those receiving oxygen alone.(13)

Both CPAP and BiPAP are equally effective in managing acute respiratory failure. (19)

Therefore, the choice of ventilatory mode should be guided by individual patient characteristics rather than a fixed protocol.

In patients requiring endotracheal intubation, non-invasive ventilation may be used for preoxygenation due to its ability to reduce the risk of hypoxemia. High-flow nasal oxygen represents a viable alternative in non-hypoxemic patients or in cases where non-invasive ventilation is contraindicated. (20)

Clinical Evidence

NIV consistently reduces the need for endotracheal intubation, particularly when initiated early in prehospital or emergency department settings. Thompson et al. (21) demonstrated an absolute reduction in intubation rates and mortality in selected out-of-hospital patients treated with CPAP compared with standard therapy.

Evidence regarding mortality benefit, however, remains inconsistent across studies.

Meta-analyses suggest that NIV may reduce hospital mortality and shorten ICU stay in selected populations, although findings are not uniform. (22) Some studies report no significant difference in overall hospital length of stay, whereas others demonstrate reduced ICU admission rates and shorter ICU duration among patients treated with NIV. According to Roessler et al. (23), patients receiving standard therapy were more frequently admitted to the intensive care unit and experienced prolonged ICU stays.

In contrast, patients treated with CPAP showed a statistically significant reduction in ICU length of stay (mean difference: −1.3 ± 2.6 days) compared with those managed without NPPV."

Multiple randomized controlled trials have shown that NIV significantly improves physiological parameters in acute CPE, including oxygenation, respiratory rate, and dyspnea severity.(24) Patients receiving NIV require continuous monitoring to assess response to therapy and detect early signs of treatment failure. In a retrospective ICU-based study, Kallio et al. (25) reported that CPAP use in patients with presumed acute pulmonary oedema was associated with improved oxygenation (77% ± 11% vs. 90% ± 7%), as well as reductions in respiratory rate (34 ± 8 vs. 28 ± 8 breaths/min), heart rate (108 ± 25 vs. 100 ± 20 beats/min), and systolic blood pressure.

Compared with invasive mechanical ventilation, NIV is associated with fewer complications, including lower rates of ventilator-associated pneumonia and ICU-acquired infections. Observational studies have also suggested improved survival in selected patient populations treated with NIV as first-line therapy. In the study by Schnell et al. (26), first-line noninvasive ventilation (NIV) was linked to higher 60-day survival and a decreased incidence of ICU-acquired infections compared with primary endotracheal intubation.

Post-extubation respiratory failure is a recognized complication of endotracheal intubation. Non-invasive ventilation (NIV) has been shown to reduce the risk and incidence of this adverse outcome.(27)

An increase in myocardial infarction was seen in the NIV group although this was based on a very low certainty of evidence (OR 1.18, 95% CI 0.95–1.48). (28)

The most consistent benefit of NIV in CPE is rapid physiological stabilization rather than uniform reduction in mortality.

Variability in outcomes is likely related to differences in timing of NIV initiation, baseline disease severity, and heterogeneity of study populations.

Therefore, NIV should be considered primarily as an early stabilization strategy rather than a universally mortality-reducing intervention. Nevertheless, Garuti et al. (29), in a prospective observational (non-randomised) study performed by trained nurses, reported that helmet CPAP (prehospital and in-hospital), compared with standard therapy, reduced mortality in patients with acute respiratory failure (ARF) by 77% ($p = 0.005$), with prehospital use alone associated with a 94% reduction after adjustment ($p = 0.011$).

Overall, variability in outcomes likely reflects differences in patient populations, disease severity, timing of intervention, and ventilatory strategies.

Discussion

Non-invasive ventilation remains one of the most important supportive interventions in the management of acute cardiogenic pulmonary edema. The available evidence consistently demonstrates that NIV improves oxygenation, reduces respiratory distress, and decreases the need for endotracheal intubation, particularly when initiated early in the course of acute respiratory failure. These benefits are likely related to both respiratory and hemodynamic mechanisms, including alveolar recruitment, reduction of work of breathing, and decreased cardiac preload and afterload.

Despite substantial evidence supporting the physiological and short-term clinical benefits of NIV, its impact on mortality remains less consistent across studies. Several factors may explain these discrepancies. First, significant heterogeneity exists among study populations, including differences in severity of heart failure, presence of hypercapnia, associated ischemic heart disease, and baseline hemodynamic instability. Second, studies differ in the timing of NIV initiation, ventilatory settings, and criteria for escalation to invasive mechanical ventilation. Early initiation of NIV, particularly in prehospital and emergency department settings, appears to be associated with improved outcomes compared with delayed implementation.

Current evidence does not clearly demonstrate superiority of either CPAP or BiPAP in terms of mortality reduction. However, important physiological differences between these modalities may influence patient selection. CPAP is generally preferred in patients with predominantly hypoxemic respiratory failure and pulmonary congestion, whereas BiPAP may provide additional benefit in patients with hypercapnia, ventilatory fatigue, or increased work of breathing. Therefore, selection of ventilatory strategy should be individualized according to the patient's respiratory and hemodynamic profile rather than based on a universal protocol.

Compared with invasive mechanical ventilation, NIV is associated with lower rates of ventilator-associated pneumonia, reduced ICU-related complications, and improved patient comfort. Avoidance of endotracheal intubation may also reduce the need for sedation and shorten intensive care unit stay in selected patients. Nevertheless, NIV failure remains an important clinical concern. Delayed recognition of treatment failure and postponed intubation may worsen outcomes, particularly in patients with progressive hypoxemia, altered mental status, or cardiogenic shock. Continuous clinical monitoring and timely reassessment are therefore essential during NIV therapy.

Interpretation of the available evidence is limited by heterogeneity in study design, variable definitions of treatment success, and inconsistent outcome reporting across trials. In addition, many studies included relatively small patient populations or differed in baseline pharmacological management. The narrative nature of this review and the absence of formal risk-of-bias assessment also represent important limitations.

Current international guidelines support the use of NIV as first-line respiratory support in appropriately selected patients with acute cardiogenic pulmonary edema and respiratory distress. Future research should focus on optimization of ventilatory strategies, identification of predictors of NIV failure, and clarification of the role of emerging approaches such as high-flow nasal oxygen therapy in comparison with conventional non-invasive ventilation.

Conclusion

Non-invasive ventilation is an effective and physiologically sound intervention in acute cardiogenic pulmonary edema. It provides rapid improvement in oxygenation and hemodynamics while reducing the need for invasive mechanical ventilation.

Current evidence supports NIV as a first-line therapy in appropriately selected patients, particularly when initiated early. Further large-scale randomized studies are needed to better define optimal treatment strategies and identify patient subgroups most likely to benefit.

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