

Correlation of Positive Airway Pressure with trends of PACO₂ levels in Type 2 Respiratory Failure patients on non-invasive ventilation

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Received: 2nd Mar, 2026 | **Revised:** 14th Mar, 2026 | **Accepted:** 4th Apr, 2026 | **Available Online:** 20th Apr, 2026

ABSTRACT

Background: Type II (hypercapnic) respiratory failure, characterized by elevated PaCO₂ and respiratory acidosis, is most commonly associated with chronic obstructive pulmonary disease (COPD). Non-invasive ventilation (NIV) has proven effective in improving outcomes in these patients by reducing the need for invasive ventilation. However, there is limited evidence correlating positive airway pressure settings with trends in PaCO₂ reduction. This study aimed to evaluate the outcomes of NIV in type II respiratory failure and correlate positive airway pressure settings with arterial PaCO₂ trends.

Methods: A prospective observational study was conducted in the Department of Respiratory Medicine and ICU at Sree Balaji Medical College and Hospital. A total of 67 adult patients (>18 years) with type II respiratory failure due to COPD, bronchiectasis, restrictive lung disease, pneumonia, obstructive sleep apnea, or pulmonary edema were enrolled. NIV was initiated using standard settings (IPAP 8 cm H₂O, EPAP 4 cm H₂O), with serial arterial blood gas (ABG) analyses at 1, 4, 8, and 24 hours. Ventilator pressures were titrated based on clinical and ABG response. Patients were followed until clinical improvement, discharge, or need for intubation.

Results: Majority of patients were male (79.1%) with a mean age of 53.8 years; 44.8% were over 60 years old. Hypertension (38.8%) and diabetes (31.4%) were the most common comorbidities. COPD was the predominant diagnosis (46.3%). NIV significantly improved oxygenation (SpO₂: 83.7% to 96.5%). Strong correlation was found between PEEP and PaCO₂ at 4 hours ($r = 0.70$, $p = 0.002$) and 24 hours ($r = 0.50$, $p = 0.002$). IPAP and EPAP showed moderate correlations at initiation but declined over time.

Conclusion: Early application of NIV in type II respiratory failure improves gas exchange and reduces the need for invasive ventilation. Positive airway pressure settings, particularly PEEP, are strongly correlated with PaCO₂ reduction, highlighting their critical role in managing hypercapnic respiratory failure.

Keywords: Type II respiratory failure, Hypercapnia, Chronic obstructive pulmonary disease (COPD), Non-invasive ventilation (NIV), Positive airway pressure, Arterial blood gas (ABG).

How to cite this article: Sahana K, Sagadevan S, Charishma V, Ram Prasath S. Correlation of Positive Airway Pressure with trends of PACO₂ levels in Type 2 Respiratory Failure patients on non-invasive ventilation. *Int J Drug Deliv Technol.* 2026;16(35s):427-434. DOI: 10.25258/ijddt.16.35s.47

Source of support: Nil.

Conflict of interest: The authors declare no conflict of interest.

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INTRODUCTION:

Respiratory failure is a life-threatening clinical condition defined by the inability of the respiratory system to maintain adequate arterial oxygenation and/or eliminate carbon dioxide to meet metabolic demands. It results from an imbalance between respiratory load and ventilatory capacity and remains a major cause of intensive care unit admissions and in-hospital mortality worldwide. Based on pathophysiological mechanisms, respiratory failure is broadly classified into four types: hypoxemic (type I), hypercapnic (type II), perioperative (type III), and shock-related (type IV) respiratory failure [1,2].

Type I respiratory failure is characterized by arterial hypoxemia with normal or low PaCO₂ and is primarily attributable to impaired oxygen exchange. Common mechanisms include ventilation-perfusion (V/Q) mismatch, diffusion impairment, intrapulmonary shunting, and alveolar collapse or flooding. Conditions such as pneumonia, pulmonary edema, acute lung injury, and acute respiratory distress syndrome (ARDS) are frequently implicated. The management of type I respiratory failure is centered on improving oxygen delivery through supplemental oxygen or advanced oxygenation strategies, while addressing the underlying pathology [1,3].

In contrast, hypercapnic respiratory failure (type II), also known as ventilatory or pump failure, is defined by elevated arterial carbon dioxide tension (PaCO₂) accompanied by respiratory acidosis. This form of respiratory failure arises primarily from alveolar hypoventilation due to impaired respiratory drive, increased airway resistance, reduced chest wall compliance, respiratory muscle fatigue, or neuromuscular dysfunction. Type II respiratory failure is commonly observed in chronic obstructive pulmonary disease (COPD), obesity hypoventilation syndrome, neuromuscular disorders, chest wall deformities, and severe asthma, and may also complicate advanced ARDS [1,3,4].

Among these etiologies, COPD remains the most frequent cause of hypercapnic respiratory failure and represents a substantial contributor to global morbidity, mortality, and healthcare utilization. Acute exacerbations of COPD often precipitate acute-on-chronic ventilatory failure, characterized by PaCO₂ levels exceeding 45 mmHg and arterial pH ≤

7.35. This clinical scenario is a well-established indication for the initiation of non-invasive ventilation (NIV), supported by strong evidence demonstrating improved physiological parameters and clinical outcomes [5,6].

Ventilatory support forms the cornerstone of management in hypercapnic respiratory failure, delivered either through invasive mechanical ventilation or NIV depending on disease severity, patient cooperation, and response to therapy. Over the past two decades, NIV—particularly bilevel positive airway pressure (BiPAP)—has emerged as a first-line intervention in selected patients with acute hypercapnic respiratory failure. Its use has been shown to reduce the need for endotracheal intubation, decrease ventilator-associated complications, shorten hospital length of stay, and improve survival, especially in acute exacerbations of COPD [1,7].

BiPAP delivers two distinct levels of positive airway pressure: inspiratory positive airway pressure (IPAP), which augments tidal volume and alveolar ventilation, and expiratory positive airway pressure (EPAP), which prevents airway collapse, improves functional residual capacity, and counterbalances intrinsic positive end-expiratory pressure. The combined effect of these pressures enhances minute ventilation and promotes effective carbon dioxide elimination. Early guidelines suggested initiating NIV with IPAP levels of 8–12 cm H₂O and EPAP of 4–6 cm H₂O, followed by rapid titration toward higher pressures (15–20 cm H₂O) in patients demonstrating inadequate clinical or biochemical improvement within the first hour [5,8].

Appropriate patient selection remains critical for NIV success. Indications for initiating NIV include moderate to severe dyspnea with respiratory rate ≥ 20–25 breaths per minute, PaCO₂ > 45 mmHg with pH < 7.35, PaO₂/FiO₂ ratio < 200, use of accessory respiratory muscles, and paradoxical abdominal breathing. However, severe acidosis (pH < 7.20) is associated with increased risk of NIV failure and may warrant early consideration of invasive ventilation or management in a high-dependency or intensive care setting [5,6,9].

Despite well-defined initiation criteria, considerable variability exists in pressure titration strategies and monitoring of treatment response during NIV. While

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improvement in pH and PaCO₂ is commonly used to assess effectiveness, the relationship between applied positive airway pressure settings and the trajectory of PaCO₂ reduction has not been adequately characterized. A clearer understanding of how specific pressure adjustments influence PaCO₂ trends could facilitate more precise titration, optimize ventilatory support, and potentially reduce NIV failure rates. Accordingly, the present study aims to evaluate the correlation between positive airway pressure settings and trends in PaCO₂ levels among patients with hypercapnic (type II) respiratory failure receiving non-invasive ventilation.

Methodology

Study Design and Setting

This prospective observational study was conducted in the Department of Respiratory Medicine in collaboration with the Intensive Care Unit (ICU) at Sree Balaji Medical College & Hospital. The study was designed to evaluate real-time clinical practice without intervention or protocol manipulation, thereby reflecting routine ICU-based management of patients with hypercapnic respiratory failure. The study setting included both respiratory wards and ICU beds equipped for non-invasive ventilation (NIV), ensuring uniformity in monitoring and ventilatory support.

Ethical Considerations

The study was conducted after obtaining approval from the Institutional Human Ethics Committee, Chettinad Academy. Written informed consent was obtained from all participants or their legally authorized representatives prior to enrollment. Patient confidentiality was maintained throughout the study, and all data were anonymized during analysis. The study adhered to ethical principles outlined in the Declaration of Helsinki.

Study Population and Sample Size

A total of 67 consecutive patients diagnosed with type II respiratory failure were enrolled over the study period. Patients were recruited using a convenience sampling method based on ICU admissions meeting eligibility criteria. No prior sample size calculation was performed, as this was an exploratory observational study intended to identify clinically relevant correlations rather than test a predefined intervention.

Eligibility Criteria

Inclusion Criteria

Patients were eligible for inclusion if they were aged over 18 years, had a diagnosis of type II respiratory failure defined by elevated arterial PaCO₂ on arterial blood gas analysis, had a clinical indication for non-invasive ventilation (NIV) as per institutional ICU protocol, and provided written informed consent. Type II respiratory failure was considered in patients with hypercapnia secondary to chronic obstructive pulmonary disease, bronchiectasis, restrictive lung diseases, pneumonia, obstructive sleep apnea, and cardiogenic or non-cardiogenic pulmonary edema.

Exclusion Criteria

Patients were excluded if they refused or were unable to tolerate NIV, had facial deformities or conditions interfering with appropriate mask fitting, active or overt gastrointestinal bleeding, acute ischemic heart disease, or required immediate endotracheal intubation at presentation. Patients with hemodynamic instability, defined as persistent hypotension or requirement for high-dose vasopressor support, were also excluded. These criteria were applied to ensure patient safety and to minimize confounding factors that could independently influence PaCO₂ trends and outcomes related to NIV.

These criteria were applied to ensure patient safety and to avoid confounding factors that could independently influence PaCO₂ trends or NIV outcomes.

Baseline Assessment and Data Collection

At enrollment, detailed demographic data including age, sex, socio-economic status, and occupation were recorded. A comprehensive clinical history was obtained, documenting presenting symptoms, duration of illness, smoking history, previous hospitalizations, and relevant comorbidities such as diabetes mellitus, hypertension, ischemic heart disease, and chronic kidney disease.

Baseline neurological status was assessed using the Glasgow Coma Scale (GCS). Initial arterial blood gas (ABG) analysis was performed prior to initiation of NIV, documenting pH, PaCO₂, PaO₂, bicarbonate levels, and oxygen saturation. Additional laboratory investigations, including complete blood count, renal function tests, and chest imaging, were obtained as part of routine ICU care.

Non-Invasive Ventilation Protocol

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Patients fulfilling ICU criteria for NIV were initiated on NIV using either bilevel positive airway pressure (BiPAP) or continuous positive airway pressure (CPAP), depending on the underlying etiology and clinical indication. NIV was delivered via an appropriately sized oronasal or full-face mask to ensure optimal seal and patient comfort.

Initial ventilatory settings were determined according to institutional protocols and individualized based on patient tolerance and severity of respiratory failure. For patients on BiPAP, inspiratory positive airway pressure (IPAP) and expiratory positive airway pressure (EPAP) were titrated to achieve adequate tidal volume, reduce work of breathing, and improve gas exchange. Oxygen supplementation was adjusted to maintain target oxygen saturation.

Monitoring and Titration of NIV

Patients were closely monitored following NIV initiation. Vital parameters including oxygen saturation, heart rate, respiratory rate, and blood pressure were continuously or intermittently recorded as per ICU standards. Clinical indicators such as use of accessory muscles, patient-ventilator synchrony, and subjective comfort were assessed regularly.

NIV settings were adjusted based on clinical response and serial ABG findings. Improvement was defined by reduction in respiratory distress, stabilization of vital signs, improvement in pH, and downward trend in PaCO₂. ABG measurements were repeated at clinically relevant intervals to evaluate PaCO₂ trends in relation to applied airway pressures.

Clinical Course and Escalation Criteria

Patients demonstrating sustained clinical and biochemical improvement on NIV were gradually weaned and transitioned from ICU to ward care. These patients were counseled regarding continuation of home-based NIV where indicated, particularly in chronic hypercapnic conditions.

Patients with deterioration in neurological status, worsening acidosis, persistent or rising PaCO₂ despite optimized NIV settings, or inability to tolerate NIV were considered for escalation of care. Those with declining GCS or signs of impending respiratory arrest were intubated and managed with invasive mechanical ventilation as per ICU protocol. Such patients were included in outcome analysis but excluded from further NIV correlation assessment after intubation.

Data Management and Statistical Analysis

All collected data were entered into a structured data collection form and subsequently transferred to a secure database. Continuous variables were expressed as mean with standard deviation or median with interquartile range, as appropriate. Categorical variables were expressed as frequencies and percentages.

Correlation between airway pressure parameters and PaCO₂ trends was assessed using Pearson's/ Spearman Correlation based on data distribution. Statistical significance was defined using a two-tailed p-value threshold. Analysis was performed using standard statistical software.

RESULTS AND ANALYSIS:

Age and Gender Distribution

In the present study, the majority of patients belonged to the age group of >60 years, comprising 30 (44.8%) patients. The mean age of the study participants was 53.82 ± 18.32 years. The study population was predominantly male, with 53 (79.1%) patients, while 14 (20.9%) were female.

Comorbidities

Assessment of comorbid conditions showed that hypertension was the most frequently encountered, affecting 26 (38.8%) patients, followed by diabetes mellitus in 21 (31.4%) patients. A combination of diabetes and hypertension was identified in 11 (16.43%) cases. Other notable comorbidities included coronary artery disease in 9 (13.43%), thrombocytopenia in 4 (5.97%), and chronic kidney disease in 3 (4.47%) patients. Furthermore, 26 (38.8%) of the study participants were smokers, and 13 (19.4%) reported a history of biomass exposure. (Table 1)

Primary Diagnosis:

In this study, COPD was the most prevalent primary diagnosis, observed in 31 (46.3%) patients. Pneumonia was the second most common, affecting 14 (20.9%) patients, followed by ARDS in 11 (16.4%) cases. Other diagnoses such as septic shock, acute pulmonary edema, and acute respiratory failure were each noted in 6 (9%) patients, while pleural effusion was the least frequent, seen in 4 (6%) patients.

Table 1. Baseline Characteristics of Study Participants (N = 67)

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Variable	Category	Frequency (n)	Percentage (%)
Age Distribution	<30 years	10	14.9
	31–40 years	9	13.4
	41–50 years	8	11.9
	51–60 years	10	14.9
	>60 years	30	44.8
Mean ± SD		53.82 ± 18.32	
Gender	Male	53	79.1
	Female	14	20.9
Primary Diagnosis	ARDS	11	16.4
	Pneumonia	14	20.9
	Septic shock	6	9.0
	COPD	31	46.3
	Pleural effusion	4	6.0
	Acute pulmonary edema	6	9.0
	Acute respiratory failure	6	9.0
Comorbidities	Coronary artery disease	9	13.4
	Chronic kidney disease	3	4.5
	Thrombocytopenia	4	6.0
	Diabetes mellitus	21	31.4
	Hypertension	26	38.8
	Diabetes + Hypertension	11	16.4
	Smoking Status	Smoker	26
	Non-smoker	41	61.2
Biomass Exposure	Yes	13	19.4
	No	54	80.6

Non-Invasive Ventilation (NIV) Distribution

In this study, a total of 67 patients with type II respiratory failure were managed with non-invasive ventilation (NIV). Among them, 33 (48.3%) received continuous positive airway pressure (CPAP), while 34 (50.7%) were treated with bi-level positive airway pressure (BiPAP).

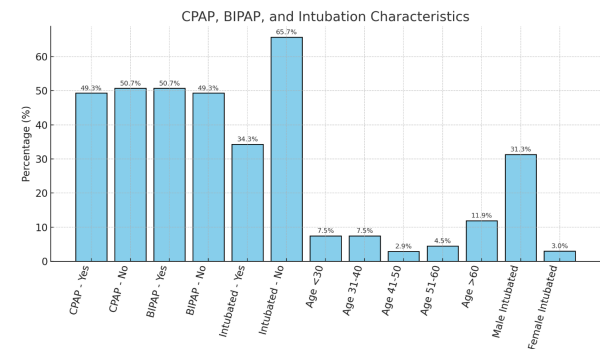
Outcome

Positive airway pressures were initiated with a PEEP of 5 cmH₂O, and arterial blood gases (ABG) were monitored on an hourly basis. PEEP was titrated upward by 1 cmH₂O in response to elevated PaCO₂ levels. The findings demonstrated that each 1 cmH₂O increase in pressure resulted in an average reduction of 10 mmHg in PaCO₂, indicating a significant correlation at both the 4th and 24th hours of monitoring. The initial IPAP:EPAP ratio was set at 6:12 and subsequently adjusted based on ABG results. A significant positive correlation was observed with IPAP (r = 0.96, p < 0.001), whereas EPAP did not show a statistically significant correlation (r = -0.12, p = 0.3).

Intubation

Out of the study population, 23 patients required intubation due to poor Glasgow Coma Scale (GCS) scores and unconsciousness at admission, following which they were transitioned to NIV. The majority of these patients were male (21; 34.3%), while 2 (2.99%) were female. Age-wise, most intubated patients belonged to the >60 years group (8; 11.94%), followed by 31–40 years (5; 7.46%) and <30 years (5; 7.46%). Other age groups included 51–60 years (3; 4.48%) and 41–50 years (2; 2.29%). (Figure 1)

Figure 1: CPAP, BiPAP and Intubation Characteristics.



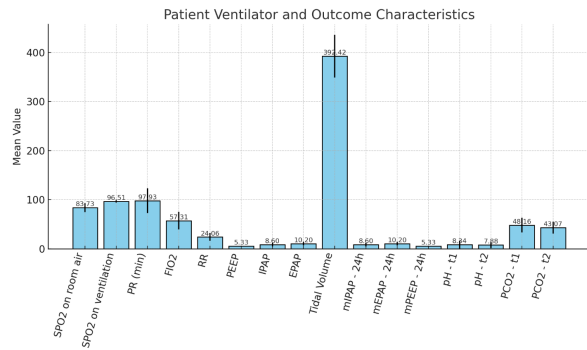
Patient Ventilator Characteristics

The mean baseline SpO₂ on room air was 83.73% ± 9.22%, which improved to 96.51% ± 2.48% with ventilation. The pulse rate (PR) had a mean of 97.93 ± 25.19/min, ranging from 40 to 150/min. The mean fraction of inspired oxygen (FiO₂) was 57.31% ± 18.05%, with values ranging between 40% and 100%. The respiratory rate (RR) averaged 24.06 ± 7.97 breaths/min (range: 14–48 breaths/min). Positive end-expiratory pressure (PEEP) had a mean of 5.33 ± 0.80 cmH₂O (range: 4–10 cmH₂O). The inspiratory positive airway pressure (IPAP) averaged 8.60 ± 3.70

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cmH₂O, while expiratory positive airway pressure (EPAP) averaged 10.2 ± 3.62 cmH₂O, with both ranging from 5 to 15 cmH₂O. The mean tidal volume delivered was 392.42 ± 43.5 mL, ranging from 300 to 450 mL. (Figure 2)

Figure 2: Patient Ventilator and Outcome Characteristics:



Correlation Between Ventilatory Pressures and PaCO₂

Analysis of correlations between ventilatory pressures and PaCO₂ revealed distinct patterns over time. PEEP: At initiation (0 hour), PEEP showed a moderate positive correlation with PaCO₂ (r = 0.49, p = 0.003), which weakened by the first hour (r = 0.20, p = 0.2). At 4 hours, the correlation was strong (r = 0.70, p = 0.002) and remained significant at 24 hours (r = 0.50, p = 0.002). PEEP at 24 hours demonstrated a very strong association with PaCO₂ at the same time (r = 0.81, p = 0.01), highlighting its sustained influence during prolonged therapy.

IPAP: Baseline IPAP correlated moderately with PaCO₂ (r = 0.49, p = 0.003) but showed no significant association at 1 hour. At 4 hours, the correlation remained moderate (r = 0.49, p = 0.003), while its association with PaCO₂ at 24 hours was weak and non-significant (r = 0.20, p = 0.2). IPAP at 24 hours only correlated significantly with PaCO₂ at 4 hours (r = 0.39, p = 0.023), suggesting a diminishing effect over time.

EPAP: Similar to IPAP, EPAP at baseline showed a moderate positive correlation with PaCO₂ (r = 0.49, p = 0.003), which was not significant at 1 hour. At 4 hours, EPAP maintained a moderate correlation (r = 0.49, p = 0.003), but its relationship with PaCO₂ at 24 hours was weak and insignificant (r = 0.20, p = 0.2). EPAP at 24 hours correlated weakly with PaCO₂ at 4 hours (r = 0.39, p = 0.023) but not at 24 hours.

Overall, PEEP demonstrated the most consistent and significant relationship with PaCO₂, particularly at later time points, whereas IPAP and EPAP showed stronger effects during early therapy that diminished by the 24th hour. (Table 2)

Table 2: Correlation of PaCO₂ with Pressure Variables

Pressure Variable	Time Point	PaCO ₂ Time	r	p-value
IPAP	Baseline	Baseline	0.96	<0.001
		1st hr	0.49	0.003
	1st hr	Baseline	0.39	0.023
		1st hr	0.20	0.20
	4th hr	4th hr	0.49	0.003
		24th hr	0.20	0.20
	24th hr	4th hr	0.39	0.023
EPAP		24th hr	0.20	0.10
	Baseline	Baseline	0.12	0.03
		1st hr	0.49	0.003
	1st hr	Baseline	0.39	0.023
		1st hr	0.20	0.20
	4th hr	4th hr	0.49	0.003
		24th hr	0.20	0.20
PEEP	24th hr	4th hr	0.39	0.023
		24th hr	0.20	0.10
	Baseline	Baseline	0.03	0.02
		1st hr	0.49	0.003
	1st hr	Baseline	0.39	0.023
		1st hr	0.20	0.20
	4th hr	4th hr	0.70	0.002
	24th hr	0.50	0.002	
	24th hr	4th hr	0.36	0.40

DISCUSSION

In the present cohort, nearly half of the patients were aged over 60 years, reinforcing the well-established association between advancing age and the development of type II respiratory failure. Age-related decline in pulmonary elastic recoil, respiratory muscle strength, and ventilatory responsiveness, combined with a higher burden of chronic comorbidities, predisposes elderly individuals to hypercapnic decompensation during acute illness [10]. The mean age of 53.8 ± 18.3 years observed in this study closely parallels that reported by Ambrosino and Vaghegginì in their 2008 evaluation of NIV use in acute exacerbations of COPD, suggesting comparable patient profiles and

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disease severity across cohorts [11]. This similarity supports the external validity of the present findings within the context of NIV-treated hypercapnic respiratory failure.

A marked male predominance was observed, consistent with prior studies evaluating NIV outcomes in acute respiratory failure. Plant et al. reported a similar male preponderance, attributing this trend to higher exposure to tobacco smoke, occupational pollutants, and biomass fuels among men [12]. The high prevalence of smoking and biomass exposure in the present cohort further supports this explanation and reflects persistent environmental and behavioral risk factors in low- and middle-income settings. Importantly, these exposures contribute not only to COPD development but also to disease severity and frequent exacerbations, thereby increasing the likelihood of hypercapnic respiratory failure requiring ventilatory support [14].

Hypertension and diabetes mellitus were the most common comorbidities identified, underscoring the intersection between chronic respiratory disease and cardiovascular-metabolic disorders. Confalonieri et al. similarly highlighted the high prevalence of cardiovascular comorbidities in patients with COPD-related respiratory failure, noting their adverse impact on clinical outcomes and ventilatory response [13]. The presence of such comorbidities may influence NIV tolerance, hemodynamic stability, and the trajectory of gas exchange improvement, emphasizing the need for individualized ventilatory strategies rather than rigid protocol-driven pressure escalation.

COPD constituted the predominant underlying diagnosis in this study, accounting for nearly half of all cases. This finding is consistent with classical and contemporary literature identifying acute exacerbations of COPD as the leading indication for NIV in hypercapnic respiratory failure [15,16]. The physiological rationale for NIV in COPD—reduction in work of breathing, unloading of fatigued respiratory muscles, and enhancement of alveolar ventilation—has been well established. In contrast, pneumonia and ARDS accounted for a substantial proportion of cases in this cohort. While NIV may be attempted in selected patients with these conditions, prior evidence indicates higher failure rates, particularly in moderate-to-severe ARDS, due to refractory hypoxemia and reduced lung compliance

[17]. The inclusion of such heterogeneous etiologies may partly explain the relatively high intubation rate observed in this study.

Ventilatory modality was almost evenly divided between BiPAP and CPAP. The physiological superiority of BiPAP in hypercapnic respiratory failure lies in its ability to deliver differential inspiratory and expiratory pressures, thereby augmenting tidal volume and facilitating carbon dioxide clearance. The observed PaCO₂ reduction with increasing IPAP is consistent with prior evidence demonstrating improved alveolar ventilation with higher inspiratory pressures [18]. However, the very strong correlation observed between IPAP increments and PaCO₂ reduction ($r = 0.96$) warrants cautious interpretation. While it underscores the importance of adequate pressure support, such a high correlation may reflect the short-term physiological response in a controlled ICU environment rather than a universally reproducible effect. Nava et al. emphasized that timely and adequate IPAP titration is crucial during the early phase of NIV, but also noted diminishing returns beyond a certain threshold due to patient discomfort and reduced compliance [19].

The need for endotracheal intubation in approximately one-third of patients aligns with real-world ICU data reported by Carron et al., who documented NIV failure rates ranging from 20% to 35% in heterogeneous hypercapnic populations [20]. In the present study, patients requiring intubation were predominantly elderly males with lower GCS scores, highlighting the critical role of neurological status in predicting NIV success. Reduced sensorium compromises airway protection and patient-ventilator synchrony, thereby increasing the likelihood of NIV failure despite optimal pressure settings.

Ventilator pressures used in this study were broadly consistent with British Thoracic Society recommendations, with mean IPAP and EPAP values falling within guideline-supported ranges [21]. These settings were associated with significant improvements in oxygen saturation, respiratory rate, and heart rate, reflecting reduced respiratory distress and improved gas exchange. Similar physiological improvements have been reported in pressure-targeted BiPAP protocols described by Ambrosino

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and Vaghegini, reinforcing the clinical relevance of guideline-concordant NIV application [11].

An important observation in this study was the sustained correlation between positive end-expiratory pressure (PEEP) and PaCO₂ reduction at 24 hours. This finding suggests that beyond early inspiratory pressure support, maintenance of alveolar recruitment and optimization of ventilation–perfusion matching play a crucial role in sustained carbon dioxide clearance. Chadda et al. previously demonstrated that while early improvements in PaCO₂ are largely driven by pressure support, longer-term trends are influenced by lung mechanics, patient effort, and disease evolution [22]. The diminishing correlation of IPAP and EPAP over time in the present study aligns with this concept and highlights the dynamic nature of NIV response.

This study has few limitations that warrant consideration. The single-center design and relatively small sample size limit generalizability. The heterogeneous etiologies of type II respiratory failure introduce confounding variables that may influence PaCO₂ trends independent of ventilatory pressures. Serial ABG measurements were performed based on clinical judgment rather than fixed time intervals, potentially introducing measurement variability. Additionally, the observational design precludes causal inference between pressure settings and PaCO₂ changes.

Despite these limitations, the findings underscore the importance of individualized NIV pressure titration guided by serial PaCO₂ trends rather than fixed pressure protocols. Early optimization of IPAP appears critical for rapid correction of hypercapnia, while sustained PEEP may contribute to longer-term ventilatory stability. These results support a dynamic, physiology-driven approach to NIV management and provide a rationale for future prospective studies evaluating pressure titration algorithms in hypercapnic respiratory failure.

CONCLUSION:

Non-invasive ventilation significantly improved gas exchange and clinical outcomes in patients with type II respiratory failure. A strong correlation was observed between PEEP levels and sustained PaCO₂ reduction, especially at 4 and 24 hours, underscoring its role in effective alveolar ventilation. While IPAP showed an early influence on PaCO₂ trends, its effect

diminished over time. These findings highlight the importance of individualized pressure titration, particularly optimizing PEEP, to enhance NIV effectiveness and patient outcomes.

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