RJNMSR
Review Journal of Neurological & Medical Sciences Review

Volume 4, Issue 2, 2026

COMPARATIVE ANALYSIS OF VOLUME-CONTROLLED VS PRESSURE-CONTROLLED VENTILATION IN ACUTE RESPIRATORY DISTRESS SYNDROME PATIENTS

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DOI: https://doi.org/10.5281/zenodo.18619533

Received	Accepted	Published
11 December 2025	26 January 2026	12 February 2026

ABSTRACT

Background: *Acute Respiratory Distress Syndrome (ARDS) is a major cause of acute respiratory failure with high morbidity and mortality. Mechanical ventilation is essential in ARDS management, yet inappropriate ventilatory strategies may worsen lung injury. Volume-controlled ventilation (VCV) and pressure-controlled ventilation (PCV) are commonly used modes, but evidence comparing their effectiveness, particularly in resource-limited settings, remains limited.*

Objective: *To compare the effectiveness of volume-controlled and pressure-controlled ventilation in adult patients with ARDS.*

Methods: *This cross-sectional comparative study was conducted at Lady Reading Hospital, Peshawar, over six months. Eighty-eight adult ARDS patients requiring invasive mechanical ventilation were enrolled and equally allocated to VCV (n=44) and PCV (n=44) groups. Oxygenation parameters (SpO_2, PaO_2, PaO_2/FiO_2), lung compliance, ventilatory pressures (PIP, Pplat, MAP), and $PaCO_2$ were recorded at baseline, 1, 6, 12, and 24 hours. Data were analyzed using SPSS version 27.0, and independent t-tests were applied.*

Results: *PCV was associated with significantly higher SpO_2, PaO_2, and PaO_2/FiO_2 ratios, improved lung compliance, and lower peak and plateau pressures at all time points ($p < 0.001$). Mean airway pressure was higher and $PaCO_2$ was significantly lower in the PCV group, indicating better alveolar recruitment and ventilation efficiency.*

Conclusion: *Pressure-controlled ventilation demonstrated superior oxygenation and lung-protective advantages compared to volume-controlled ventilation in ARDS patients. PCV may be a preferable ventilatory strategy during early ARDS management.*

Keywords: *ARDS, Pressure-Controlled Ventilation, Volume-Controlled Ventilation, Mechanical Ventilation*

INTRODUCTION

Acute respiratory distress syndrome (ARDS) is a severe form of acute respiratory failure associated with significant mortality and remains a considerable challenge in intensive care

medicine(1). shbaugh et al. (1967) characterized acute respiratory distress syndrome (ARDS) as severe, potentially fatal acute hypoxemia that manifests without evident cardiogenic pulmonary oedema, bilateral pulmonary infiltrates, and reduced lung compliance on chest x-ray(2). According to the PaO_2/FiO_2 ratio and the need for positive end-expiratory pressure (PEEP), the Berlin Definition of ARDS (2012) further divided ARDS into mild, moderate, and severe categories. Acute respiratory distress syndrome affects around 10% of patients in the critical care unit and almost 25% of patients who need invasive mechanical ventilation. Mortality rates remain between 30 and 45 percent despite advancements in supportive care, and survivors frequently experience long-term functional and pulmonary problems(3,4).

The pathophysiology of ARDS is characterized by alterations in the alveolar-capillary barrier, resulting in an increase in permeability and resulting in pulmonary edema. It affects the gas exchange capabilities of the lungs causing hypoxemia and reduces lung compliance(5). Through its evolution, the disease goes through three overlapping phases, namely, the exudative phase where there is flooding of the alveoli and inflammation, a proliferative phase where there is activation of fibroblasts and organization of exudates , and in some patients, a fibrotic phase where there is scarring and extensive reorganization of the lung(6). Mechanical ventilation is necessary and can be injury-generating and is therefore required because of the generation of heterogeneous lung regions with collapse, consolidation and overdistension. Mechanical ventilation is the key supportive intervention for acute respiratory distress syndrome, assisting with oxygen delivery and carbon dioxide clearance(7). However inappropriately set ventilators may worsen lung injury due to VILI. The mechanisms of ventilator-induced lung injury (VILI) include barotrauma due to excess airway pressures; volutrauma due to overdistension of alveoli; atelectrauma due to repetitive reopening and collapse of alveoli; and biotrauma due to the release of inflammatory mediators. The fact that mechanical ventilation can save a life and harm the patient drives the need to choose the best ventilatory strategy(8,9).

The two types of ventilation frequently utilized in ARDS management are volume-controlled ventilation or VCV and pressure-controlled ventilation or PCV. Volume-controlled ventilation (VCV) provides a set volume of air with each breath(10). The device guarantees that whatever volume clinicians dictate will ultimately reach the patient, irrespective of compliance/ resistance changes. As it allows clinicians to keep stable tidal volumes and predictable carbon dioxide removal, VCV is useful when precise control of minute ventilation is required. VCV is especially handy in patients who require precise control of alveolar ventilation, for example, those with acid-base disturbances(11). Another advantage of VCV is its simplicity and the fact that it is the default mode in many ICUs. However, the key limitation is that airway pressures are not controlled and may rise to quite high levels in stiff, non-compliant, acute respiratory distress syndrome lungs. High peak and plateau pressures leading to barotrauma and volutrauma is a known phenomenon. In addition, in the heterogeneous ARDS lung, VCV may unevenly distribute ventilation, with well-aerated regions receiving disproportionately high volume and diseased regions receiving less volume(12,13).

Pressure-controlled ventilation (PCV) refers to a mode of mechanical ventilation that delivers a breath to a preset peak inspiratory pressure, with the tidal volume delivered varying depending on the patient's lung compliance and airway resistance(14). PCV leads to a decelerating inspiratory flow pattern, with peak flow occurring at the start of inspiration and reducing thereafter. This leads to more uniform gas distribution and may facilitate the recruitment of alveoli, especially in non-compliant ARDS lungs(15). Using Pressure Control Ventilation diminishes the risk of barotrauma relative to volume control ventilation. Doctors tend to choose PCV when it is imperative to protect the lungs as it does not allow high airway pressure. Nevertheless, PCV has limitations. Changes in compliance or resistance will impact tidal volume as it is variable and not fixed(16). They may over-breathe (hyperventilate) and under-breathe (hypo ventilate). In a bustling or resource-limited ICU, constantly watching and changing the settings may not always be possible. In

addition, patients with quickly changing lung mechanics also do not benefit from PCV since it does not assure stable alveolar ventilation(17).

Both VCV and PCV are appropriate modes of ventilation when used with other lung protective strategies such as low tidal volumes (6 ml/kg predicted body weight) and limiting plateau pressures to < 30 cmH_2O. However, studies that compare the two modes yield conflicting results. Physiological studies indicate that peak airway pressure reduces with PCV, and improved oxygenation may result from better gas distribution(18,19). Some focus on the steady-state performance of VCV for minute ventilation and CO2 clearance. Researchers have conducted numerous randomized controlled trials comparing HFOV with conventional ventilation in a variety of patient populations (e.g. ARDS vs. ALI vs. other diagnoses). These studies have not found a survival advantage for one mode over the other. However, some studies have reported differences in secondary outcomes such as lung compliance, oxygenation indices and the incidence of ventilator-induced lung injury(20).

The choice of VCV or PCV in RICUs is made by the clinicians as per the patient's condition, but in resource-limited ICUs, the choice is often made according to physician preference, availability of ventilator functions, and local practice patterns(21). Regretfully, there aren't many good studies from low- and middle-income nations that contrast these two ventilation strategies in ARDS. The available information is mainly from Western populations that have advanced monitoring and other therapy options such as prone positioning and ECMO and recruitment maneuvers. Given the huge burden of ARDS in South Asia where pneumonia, sepsis and trauma are leading causes of ICU admission, it is important to generate evidence on the VCV and PCV as per our context(22,23).

The ventilator strategy that is employed in ARDs patients is an important determinant of outcome. Choosing the best ventilation mode may be important for gas exchange, ventilator-induced complications, intensive care unit stays, and ultimately, survival(24). As ARDS is associated with high morbidity and mortality, it is important to compare VCV with PCV in the clinical setting to develop an evidence-based protocol for better patient care(25).

In brief, ARDS is a common and devastating syndrome that requires invasive MV for ventilator supportive management. The most commonly used strategies are volume-controlled and pressure-controlled ventilation(26,27). Each has its own advantages and disadvantages. While VCV allows exact tidal volumes to be delivered and carbon dioxide control to be reliable, it has the potential for high airway pressures and possibly lung injury. On the other hand, PCV limits airway pressures and may improve oxygen distribution but does so with variable tidal volumes and requires close monitoring. Even though they are commonly used, data that compares their effectiveness is lacking, especially in low-resource contexts. Due to various reasons, almost 50% of patients with ARDS die. Early and careful application of lung protective strategies can provide maximum benefit(27). In this research we will compare clinical outcomes in an ARDS patient who will be ventilated with VCV vs PCV. With the aim to study oxygenation, respiratory mechanics, ventilator induced lung injury, duration of ICU stays and mortality in ARDS patients, it is expected that optimized ventilatory management will help in improving patient outcome.

RATIONAL:

Acute Respiratory Distress Syndrome has a very high morbidity and mortality rate despite advances in critical care. Mechanical ventilation is the mainstay in treating ARDS. When choosing between Volume-Controlled Ventilation (VCV) and Pressure-Controlled Ventilation (PCV), it is important to note that both ventilatory options have their own benefits and disadvantages. Essentially, VCV guarantees a stable tidal volume delivered but it may expose the lung to high airway pressures. On the other hand, PCV limits the airway pressures and enhances gas distribution but generates a variable tidal volume. The current data comparing the two modes is inconclusive and generated mostly in high-resource settings. Consequently, there is a significant gap in knowledge in resource-limited settings where ARDS is common. Thus, it is rationalized to bring about context-specific evidence pertaining to VCV

and PCV in the ARDS patient with a comparative objective in order to improve clinical outcome measures in our setting.

Aim and Objective:

To compare the effectiveness of volume-controlled ventilation and pressure-controlled ventilation in patients with Acute Respiratory Distress Syndrome.

MATERIAL AND METHOD

Study Design

This cross-sectional study was conducted to assess and compare volume-controlled ventilation (VCV) and pressure-controlled ventilation (PCV) in patients diagnosed with acute respiratory distress syndrome (ARDS). The research protocol was approved by the hospital ethics committee, and the study was carried out at Lady Reading Hospital, Peshawar, over a six-month period.

Participants

A total of 88 adult patients (≥18 years) requiring invasive mechanical ventilation were enrolled. Inclusion criteria required patients to be hemodynamically stable (MAP ≥60 mmHg, with or without vasopressors). Exclusion criteria included chronic lung disease, neuromuscular disease affecting respiration, pregnancy, patients requiring only non-invasive ventilation, and those with predicted survival of less than 24 hours.

Sample Size Calculation

The sample size was calculated using the formula for comparing two group means:

$$n = 2 \times (Z_{\alpha/2} + Z_{\beta})^2 \times \sigma^2 \div (\mu_1 - \mu_2)^2$$

This yielded **44 patients per group**.

Randomization and Group Allocation

After obtaining written informed consent from legally acceptable representatives, patients were allocated into two groups using a convenience sampling technique:

- **VCV group (n=44)**: Received tidal volumes of 6–8 mL/kg predicted body weight, with PEEP adjusted according to ARDSNet protocol.
- **PCV group (n=44)**: Received pressure-controlled ventilation to achieve similar tidal volumes, with decelerating inspiratory flow and I:E ratio of 1:2.

In both groups, FiO_2 and PEEP were titrated to maintain SpO_2 between 88–95% or PaO_2 between 55–80 mmHg.

Data Collection

Baseline demographic details (age, sex, weight, comorbidities) were recorded. Clinical and ventilatory parameters were measured at baseline, and then at **1 hour, 6 hours, 12 hours, and 24 hours** after initiation of mechanical ventilation. Parameters included:

- Oxygen saturation (SpO_2)
- Arterial oxygen tension (PaO_2)
- Arterial carbon dioxide tension ($PaCO_2$)
- PaO_2/FiO_2 ratio
- Lung compliance
- Peak inspiratory pressure (PIP)
- Plateau pressure (Pplat)
- Mean airway pressure (MAP)

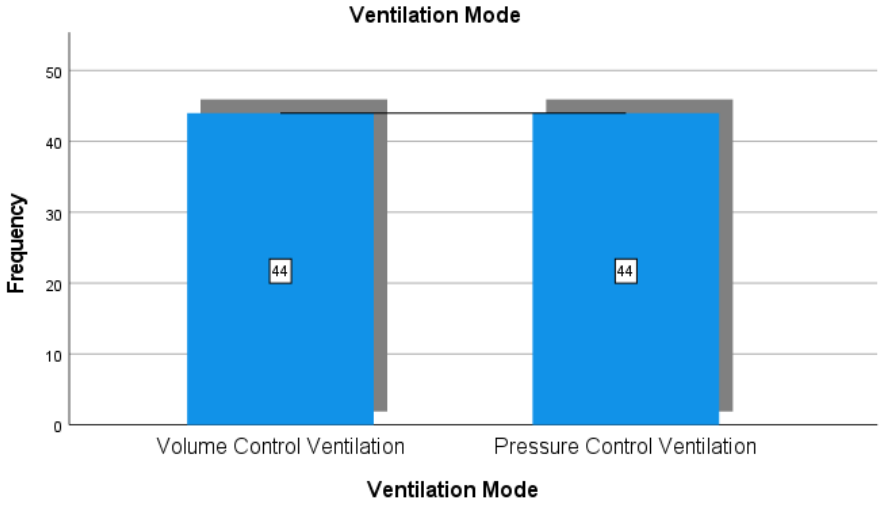
Statistical Analysis

Data were entered into a pre-designed form and analyzed using **SPSS version 27.0**. Continuous variables (PaO_2, $PaCO_2$, SpO_2, PaO_2/FiO_2 ratio, lung compliance, PIP, Pplat, MAP) were expressed as **mean ± standard deviation**. Comparisons between VCV and PCV groups were performed using the **independent t-test**. A p-value <0.05 was considered statistically significant. Results were presented in tabular format to highlight differences between the two ventilation strategies.

RESULT

A total of 88 patients were included in our study, with no missing data for ventilation mode, gender,

or comorbidity status. Patients were equally distributed between the two ventilation strategies; 44 patients (50%) received volume-controlled ventilation, while 44 patients (50%) were managed using pressure-controlled ventilation. Regarding comorbidities.



The majority of patients 62 (70.5%) had at least one comorbid condition, whereas 26 patients (29.5%) had no documented comorbidities.

Table 4.1: Distribution of Patients by Ventilation Mode and Comorbidities (n = 88)

Variable	Category	Frequency (n)	Percentage (%)
Ventilation Mode	Volume-Controlled Ventilation (VCV)	44	50.0
	Pressure-Controlled Ventilation (PCV)	44	50.0
Comorbidities	Yes	62	70.5
	No	26	29.5

COMPARISON OF SPO_2 BETWEEN VCV AND PCV GROUPS:

The comparison of oxygen saturation (SpO_2) between patients ventilated with volume-controlled ventilation (VCV) and pressure-controlled ventilation (PCV) demonstrated a consistently higher SpO_2 in the PCV group at all measured time points. At baseline, mean SpO_2 was significantly higher in the PCV group compared to the VCV group (86.36 ± 3.10 vs 84.50 ± 0.51, $p < 0.001$). This difference became more pronounced over time. At 1 hour, 6 hours, 12 hours, and 24 hours, patients in the PCV group continued to show significantly improved oxygen saturation compared to those in the VCV group, with all comparisons achieving statistical significance ($p < 0.001$). The largest difference was observed at 6 hours, where the mean SpO_2 in the PCV group was 90.11 ± 1.86 compared to 85.00 ± 1.24 in the VCV group.

Table 4.2: Comparison of SpO_2 Between VCV and PCV Groups

Time Point	VCV (Mean ± SD)	PCV (Mean ± SD)	p-value
Baseline SpO_2 (%)	84.50 ± 0.51	86.36 ± 3.10	**< 0.001**
1 Hour SpO_2 (%)	84.75 ± 1.10	87.82 ± 3.14	**< 0.001**
6 Hour SpO_2 (%)	85.00 ± 1.24	90.11 ± 1.86	**< 0.001**
12 Hour SpO_2 (%)	86.50 ± 1.68	91.05 ± 1.92	**< 0.001**

24 Hour SpO_2 (%)	87.25 ± 2.30	91.48 ± 1.91	**< 0.001**

COMPARISON OF PAO_2/FIO_2 RATIO BETWEEN VCV AND PCV GROUPS

The PaO_2/FiO_2 ratio, an important indicator of oxygenation efficiency and ARDS severity, was compared between volume-controlled ventilation (VCV) and pressure-controlled ventilation (PCV) groups at baseline and at subsequent time intervals. At baseline, the mean PaO_2/FiO_2 ratio was significantly higher in the PCV group compared to the VCV group (210.0 ± 19.3 vs 168.0 ± 8.0, $p < 0.001$). This significant difference persisted at 1 hour, 6 hours, 12 hours, and 24 hours, with the PCV group demonstrating consistently superior oxygenation throughout the observation period ($p < 0.001$ for all comparisons). The greatest improvement in PaO_2/FiO_2 ratio was observed at 12 hours, where the PCV group achieved a mean value of 220.1 ± 19.3 compared to 174.3 ± 7.1 in the VCV group.

Table 4.3: Comparison of PaO_2/FiO_2 Ratio Between VCV and PCV Groups

Time Point	VCV (Mean ± SD)	PCV (Mean ± SD)	p-value
Baseline	168.0 ± 8.0	210.0 ± 19.3	**< 0.001**
1 Hour	168.0 ± 8.0	210.7 ± 18.8	**< 0.001**
6 Hours	169.0 ± 4.7	213.9 ± 16.8	**< 0.001**
12 Hours	174.3 ± 7.1	220.1 ± 19.3	**< 0.001**
24 Hours	179.3 ± 10.9	220.8 ± 15.0	**< 0.001**

COMPARISON OF LUNG COMPLIANCE BETWEEN VCV AND PCV GROUPS

Lung compliance was compared between patients managed with volume-controlled ventilation (VCV) and pressure-controlled ventilation (PCV) at baseline and at multiple follow-up intervals. At baseline, the mean lung compliance was significantly higher in the PCV group compared to the VCV group (46.93 ± 3.69 vs 42.50 ± 2.53 mL/cmH_2O, $p < 0.001$). This statistically significant difference persisted at 1 hour, 6 hours, 12 hours, and 24 hours of ventilation. The improvement in lung compliance was more pronounced over time in the PCV group, with the greatest difference observed at 24 hours (54.30 ± 3.63 vs 46.00 ± 5.30 mL/cmH_2O, $p < 0.001$).

Table 4.4 : Comparison of Lung Compliance Between VCV and PCV Groups

Time Point	VCV (Mean ± SD) mL/cmH_2O	PCV (Mean ± SD) mL/cmH_2O	p-value
Baseline	42.50 ± 2.53	46.93 ± 3.69	**< 0.001**
1 Hour	42.50 ± 2.53	47.89 ± 3.81	**< 0.001**
6 Hours	44.00 ± 2.48	52.05 ± 5.04	**< 0.001**
12 Hours	44.75 ± 3.31	52.50 ± 4.47	**< 0.001**
24 Hours	46.00 ± 5.30	54.30 ± 3.63	**< 0.001**

COMPARISON OF PAO_2 (MMHG) BETWEEN VCV AND PCV GROUPS

Arterial oxygen tension (PaO_2) was compared between patients receiving volume-controlled ventilation (VCV) and pressure-controlled ventilation (PCV) at baseline and at multiple follow-up intervals. At baseline, the mean PaO_2 was significantly higher in the PCV group compared to the VCV group (59.80 ± 1.95 vs 50.75 ± 2.62 mmHg, $p < 0.001$). This statistically significant difference persisted at 1 hour, 6 hours, 12 hours, and 24 hours. The improvement in PaO_2 was progressive and more pronounced in the PCV group, with the largest difference observed at 24 hours (81.95 ± 2.71 mmHg in PCV vs 57.25 ± 3.31 mmHg in VCV, $p < 0.001$).

Table 4.5: Comparison of PaO_2 (mmHg) Between VCV and PCV Groups

Time Point	VCV (Mean ± SD) mmHg	PCV (Mean ± SD) mmHg	p-value
Baseline	50.75 ± 2.62	59.80 ± 1.95	< 0.001
1 Hour	52.25 ± 2.30	61.32 ± 2.41	< 0.001
6 Hours	53.25 ± 2.41	70.07 ± 2.45	< 0.001
12 Hours	55.25 ± 3.23	79.77 ± 2.94	< 0.001
24 Hours	57.25 ± 3.31	81.95 ± 2.71	< 0.001

COMPARISON OF PEAK INSPIRATORY PRESSURE (CMH_2O) BETWEEN VCV AND PCV GROUPS

Peak inspiratory pressure (PIP) was significantly lower in patients managed with pressure-controlled ventilation (PCV) compared to volume-controlled ventilation (VCV) at all measured time points. At baseline, the mean PIP in the PCV group was 27.16 ± 2.15 cmH_2O, compared to 31.50 ± 2.32 cmH_2O in the VCV group ($p < 0.001$). This statistically significant reduction persisted throughout the 24-hour observation period. The largest difference was observed at 24 hours, with the VCV group showing a mean PIP of 29.50 ± 2.72 cmH_2O compared to 23.55 ± 1.19 cmH_2O in the PCV group ($p < 0.001$).

Table 4.6: Comparison of Peak Inspiratory Pressure (cmH_2O) Between VCV and PCV Groups

Time Point	VCV (Mean ± SD)	PCV (Mean ± SD)	p-value
Baseline	31.50 ± 2.32	27.16 ± 2.15	< 0.001
1 Hour	31.00 ± 2.26	27.05 ± 1.90	< 0.001
6 Hours	31.25 ± 2.89	25.98 ± 1.70	< 0.001
12 Hours	29.75 ± 2.19	24.73 ± 1.48	< 0.001
24 Hours	29.50 ± 2.72	23.55 ± 1.19	< 0.001

Plateau pressure (Pplat) was significantly lower in patients receiving pressure-controlled ventilation (PCV) compared to volume-controlled ventilation (VCV) at all measured time points. At baseline, the mean Pplat was 24.05 ± 4.22 cmH_2O in PCV versus 29.00 ± 1.43 cmH_2O in VCV ($p < 0.001$). Similarly, mean airway pressure (MAP) was significantly higher in the PCV group at all time points, indicating better alveolar recruitment. At baseline, MAP was 12.00 ± 1.52 cmH_2O in PCV versus 10.00 ± 0.72 cmH_2O in VCV ($p < 0.001$).

Table 4.7: Comparison of Plateau Pressure (Pplat) and Mean Airway Pressure (MAP) Between VCV and PCV

Time Point	Pplat VCV (Mean ± SD)	Pplat PCV (Mean ± SD)	MAP VCV (Mean ± SD)	MAP PCV (Mean ± SD)	P-value
Baseline	29.00 ± 1.43	24.05 ± 4.22	10.00 ± 0.72	12.00 ± 1.52	< 0.001
1 Hour	28.75 ± 1.10	24.43 ± 2.36	10.00 ± 0.72	12.50 ± 1.21	< 0.001
6 Hours	28.00 ± 0.72	23.34 ± 2.12	10.75 ± 0.44	13.34 ± 1.01	< 0.001
12 Hours	27.75 ± 1.10	23.20 ± 2.70	12.50 ± 1.13	14.07 ± 1.04	< 0.001
24 Hours	27.25 ± 0.84	22.34 ± 2.87	13.75 ± 2.41	15.23 ± 1.24	0.001

COMPARISON OF PaC02 BETWEEN VCV AND PCV GROUPS

At baseline, the mean $PaCO_2$ was 48.00 ± 2.48 mmHg in the VCV group and 38.89 ± 6.20 mmHg in the PCV group, with a statistically significant difference ($p < 0.001$). After 1 hour of ventilation, $PaCO_2$ remained significantly higher in the VCV group (48.25 ± 2.52 mmHg) compared to the PCV group (39.16 ± 5.28 mmHg, $p < 0.001$). Similarly, at 6 hours, the VCV group showed a mean $PaCO_2$ of 47.50 ± 2.09 mmHg, whereas the PCV group had 38.68 ± 4.70 mmHg, again demonstrating a significant difference ($p < 0.001$). At 12 hours, the VCV group maintained a higher mean $PaCO_2$ (47.00 ± 1.75 mmHg) compared to the PCV group (39.43 ± 3.09 mmHg), with the difference remaining statistically significant ($p < 0.001$). Finally, at 24 hours, $PaCO_2$ in the VCV group was 47.00 ± 2.37 mmHg, while in the PCV group it decreased further to 36.73 ± 3.32 mmHg, indicating a sustained and significant reduction in $PaCO_2$ in the PCV group ($p < 0.001$).

Table 4.8: Comparison of $PaCO_2$ Between Volume-Controlled Ventilation and Pressure-Controlled Ventilation Groups

Time Point	VCV (Mean ± SD)	PCV (Mean ± SD)	p-value
Baseline	48.00 ± 2.48	38.89 ± 6.20	< 0.001
1 Hour	48.25 ± 2.52	39.16 ± 5.28	< 0.001
6 Hours	47.50 ± 2.09	38.68 ± 4.70	< 0.001
12 Hours	47.00 ± 1.75	39.43 ± 3.09	< 0.001
24 Hours	47.00 ± 2.37	36.73 ± 3.32	< 0.001

DISCUSSION

The present study compared the physiologic effects of pressure-controlled ventilation (PCV) and volume-controlled ventilation (VCV) in adult patients with ARDS, focusing on oxygenation parameters, lung mechanics, and ventilatory pressures at 24 hours. Overall, oxygenation was significantly better in the PCV group, accompanied by improved compliance and lower airway pressures, suggesting potential benefits of PCV in optimizing respiratory support in ARDS.

Patients ventilated with PCV demonstrated consistently higher oxygen saturation (SpO_2) and PaO_2/FiO_2 ratios at all time points compared to those managed with VCV ($p < 0.001$). The PaO_2/FiO_2 ratio, a key measure of oxygenation efficiency and ARDS severity, improved progressively in the PCV group, likely due to higher mean airway pressure (MAP), which promotes alveolar recruitment, reduces intrapulmonary shunt, and enhances ventilation–perfusion matching (28). These findings are consistent with prior studies reporting improved oxygenation with pressure-targeted modes compared to volume-targeted ventilation (29). Similar improvements in PaO_2/FiO_2 ratios with PCV have been observed in patients with reduced lung compliance (30). However, some authors caution that differences in oxygenation may not necessarily translate into long-term clinical benefits when lung-protective strategies are applied (31,32).

The average PaO_2 was significantly higher in the PCV group at baseline and subsequent time points ($p < 0.001$), reflecting enhanced alveolar ventilation. This aligns with evidence that PCV facilitates alveolar recruitment and improves gas exchange compared to volume-targeted ventilation, particularly in stiff ARDS lungs (33). In contrast, VCV may fail to adequately recruit alveoli, leading to poorer oxygenation outcomes.

Lung compliance was also significantly higher in the PCV group at all measured intervals ($p < 0.001$). Compliance reflects the ease of lung expansion, and improved values suggest more favorable distribution of ventilation. PCV may prevent regional overdistension and cyclic alveolar collapse, thereby reducing harmful mechanical stress in ARDS (34). These results support physiological studies indicating that pressure modes reduce regional lung strain compared to volume modes by allowing variable tidal volumes that adapt to patient mechanics (35).

A key finding was that peak inspiratory pressure (PIP) and plateau pressure (Pplat) were consistently lower in PCV at all time points ($p < 0.001$). Lower airway pressures are clinically important, as elevated pressures are strongly associated with ventilator-induced lung injury (VILI) and barotrauma (34). By delivering breaths at a preset pressure, PCV limits excessive airway pressures even as lung mechanics evolve. In contrast, VCV delivers fixed tidal volumes regardless of compliance, often resulting in higher PIP and Pplat in noncompliant lungs. These findings are consistent with prior studies reporting lower PIP in PCV across ARDS and other critical illness scenarios (31,30). Lower plateau pressures in PCV may also reduce alveolar overdistension, supporting lung-protective ventilation principles (36).

Interestingly, while PIP and Pplat were lower in PCV, mean airway pressure was significantly higher ($p < 0.001$). Increased MAP prolongs alveolar recruitment during the respiratory cycle, enhancing gas exchange and explaining the improved PaO_2/FiO_2 and SpO_2 values observed. Although elevated MAP can theoretically impair venous return and cardiac output, no adverse hemodynamic events were noted in this study. Nevertheless, careful monitoring of hemodynamic status remains essential when employing higher MAP strategies (37).

Taken together, the relationships between oxygenation, compliance, and ventilatory pressures suggest a physiologically coherent pattern. Improved oxygenation in PCV was associated with better compliance, lower PIP and Pplat, and higher MAP. These interactions align with lung-protective principles that emphasize limiting high airway pressures while optimizing alveolar recruitment to reduce VILI and ensure adequate oxygenation (38,34).

Despite these physiologic advantages, prior studies have reported inconsistent findings regarding long-term outcomes such as mortality or ventilator-free days. Clinical trials of PCV in acute respiratory failure have not demonstrated significant survival benefits, even though oxygenation and airway pressures were improved (32,39). This highlights the need for further research to determine whether the physiologic benefits of PCV translate into meaningful clinical outcomes. Factors such as sedation practices, adjunctive therapies (e.g., prone positioning), and individualized ventilator strategies may also influence results.

In summary, this study demonstrated that PCV provided superior oxygenation, improved compliance, and lower airway pressures compared to VCV in ARDS patients. These findings support the physiologic rationale for PCV as a potentially safer and more effective ventilation mode in ARDS. However, further large-scale studies are needed to confirm whether these advantages lead to improved survival and long-term outcomes.

Limitations

This study's strength lies in its temporal analysis of oxygenation and respiratory mechanics over 24 hours, with equal patient distribution and no missing data enhancing internal validity. However, it was single-center, limited to short-term outcomes, and lacked follow-up on ventilator-free days, ICU stay, or mortality. Larger multicenter studies with extended observation are needed to confirm whether the physiological benefits of PCV translate into meaningful clinical outcomes.

Conclusion

Findings indicate that pressure-controlled ventilation offers advantages over volume-controlled ventilation in ARDS, particularly in oxygenation and lung mechanics. PCV may provide better pulmonary protection, though ARDS remains heterogeneous and complex. Future research should explore personalized ventilator strategies and long-term outcomes to clarify the clinical impact of these physiological improvements.

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