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Association between Driving Pressure and Survival Outcomes in Mechanically Ventilated ICU Patients with Acute Respiratory Distress Syndrome

Arpita Jena[1], Manisha Mund[1]*, Deepti Swain[2], OMM Upadhyay[3]

[1]Assistant Professor, Department of Anaesthesiology and Critical Care, S.C.B. Medical College and Hospital, Cuttack, Odisha, India

[2]Assistant Professor, Department of Anaesthesiology, AHPGIC, Cuttack, Odisha, India

[3]Junior Resident, Department of Anaesthesiology and Critical Care, S.C.B. Medical College and Hospital, Cuttack, Odisha, India

***Address for Correspondence:** Dr. Manisha Mund, Assistant Professor, Department of Anaesthesiology and Critical Care, S.C.B. Medical College and Hospital, Cuttack, Odisha, India
E-mail: drmanishamund@gmail.com

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ABSTRACT

Background: Acute Respiratory Distress Syndrome (ARDS) remains a major cause of mortality in intensive care units. Driving pressure (ΔP), calculated as the difference between plateau pressure and positive end-expiratory pressure (PEEP), has emerged as a potential predictor of outcomes in mechanically ventilated patients. This study evaluated the association between driving pressure and survival outcomes in ARDS patients receiving mechanical ventilation.

Methods: A prospective observational study was conducted at a tertiary care ICU in SCB MCH, Cuttack, India, involving 100 adult patients diagnosed with ARDS according to the Berlin definition. Ventilatory parameters, including P_plat, PEEP, and ΔP, were recorded daily for the first seven days. The primary outcome was 28-day mortality. Secondary outcomes included ventilator-free days (VFD) and ICU length of stay (LOS).

Results: The overall 28-day mortality was 38%. Survivors had significantly lower mean driving pressures compared to non-survivors (12.4 pm 2.2 vs. 16.7 pm 3.1 cm H_2O; $p < 0.001$). Multivariate logistic regression identified ΔP on day 1 as an independent predictor of mortality (OR: 1.42; 95% CI: 1.18–1.71; $p = 0.0002$). A driving pressure threshold of > 15 cm H_2O was associated with a 4.5-fold increase in mortality risk. Furthermore, ΔP showed a significant negative correlation with ventilator-free days ($r = -0.68$; $p < 0.001$).

Conclusion: Higher driving pressure is strongly associated with increased 28-day mortality and reduced ventilator-free days in patients with ARDS. Monitoring ΔP provides a valuable bedside tool for individualizing PEEP and tidal volume to minimize ventilator-induced lung injury.

Key-words: Acute Respiratory Distress Syndrome; Driving Pressure; Mechanical Ventilation; Ventilator-Induced Lung Injury; Survival Analysis; Critical Care

INTRODUCTION

Acute Respiratory Distress Syndrome (ARDS) is a clinical syndrome characterized by acute onset of hypoxemic respiratory failure, bilateral pulmonary opacities, and decreased lung compliance, not fully explained by heart failure or fluid overload [1]. Despite advances in critical care, ARDS continues to carry a high mortality rate, ranging from 35% to 45% [2]. The pathophysiology involves diffuse alveolar damage leading to severe gas exchange impairment and the requirement for invasive mechanical ventilation.

However, mechanical ventilation itself can exacerbate lung damage through ventilator-induced lung injury (VILI). Historically, the ARDSNet protocol revolutionized management by advocating for low tidal volume

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ventilation (6 mL/kg of predicted body weight) to limit volutrauma and barotrauma [3]. While this strategy improved survival, it did not account for the high variability in the size of the "aerated" lung, often referred to as the "baby lung." Consequently, even low tidal volumes can cause excessive strain if delivered to a severely restricted functional lung volume.

Driving pressure (ΔP), calculated as the difference between plateau pressure (P_plat) and positive end-expiratory pressure (PEEP), represents the ratio between tidal volume and static respiratory system compliance (C_stat). In essence, it normalizes tidal volume to the functional lung capacity [4]. Seminal work by Amato et al. suggested that driving pressure is the ventilatory variable most strongly associated with survival, surpassing traditional targets like tidal volume or PEEP in isolation [5].

While the concept of ΔP has gained global attention, prospective validation in diverse clinical settings, particularly in resource-limited tertiary centers in South Asia, remains sparse. Patient-specific factors, such as high baseline comorbidity burdens and late presentation, may influence the prognostic value of ΔP. This study was designed to prospectively evaluate the association between driving pressure and 28-day survival outcomes in patients with ARDS at a major tertiary care hospital in Odisha. We hypothesized that higher driving pressures would correlate with increased mortality and worse secondary clinical outcomes.

MATERIALS AND METHODS

Study Design and Setting- This prospective observational study was conducted in the Central ICU of S.C.B. Medical College and Hospital, Cuttack, from February 2026 to August 2026. The study protocol received approval from the Institutional Ethics Committee (IEC), and informed consent was obtained from the patients' legally authorized representatives.

Patient Selection- We enrolled 100 consecutive adult patients (age >=18 years) who met the Berlin criteria for ARDS and required mechanical ventilation within 48 hours of ICU admission. ARDS was defined by: (i) onset within one week of a known clinical insult; (ii) bilateral opacities on chest imaging; (iii) respiratory failure not fully explained by cardiac failure; and (iv) a PaO_2/FiO_2 ratio ≤ 300 mmHg with PEEP >= 5 cm H_2O. We excluded patients with pre-existing chronic lung disease, those who were pregnant, and those with incomplete medical records.

Data Collection and Ventilator Management- All patients were ventilated using standard ICU ventilators (Maquet ventilator) in volume-controlled mode. Tidal volume was set at 6–8 mL/kg of predicted body weight (PBW). PEEP was adjusted by the clinical team based on FiO_2 requirements. Plateau pressure was measured daily during a 0.5-second end-inspiratory hold. Driving pressure was calculated as:

$$\mathbf{\Delta P = P\_plat - PEEP}$$

Static compliance was calculated as:

$$\mathbf{C\_stat = VT/ P\_plat - PEEP}$$

where VT is the delivered tidal volume. Demographic data, APACHE II and SOFA scores, and primary causes of ARDS (e.g., pneumonia, sepsis, trauma) were recorded at baseline. Clinical outcomes, including 28-day survival status, ICU length of stay, and ventilator-free days (defined as the number of days alive and free from mechanical ventilation at day 28), were tracked.

Statistical Analysis- Sample size was determined using the formula n = (Z^2 times p times q) / d^2 with p = 0.40, q = 0.60, and d = 0.10, resulting in a minimum of 92 patients, rounded to 100 to account for potential attrition. Data were analyzed using SPSS version 26.0. Continuous variables were expressed as Mean pm Standard Deviation (SD) or Median (Interquartile Range) based on normality testing. Categorical variables were expressed as frequencies and percentages.

Comparisons between survivors and non-survivors were performed using the Student's t-test or Mann-Whitney U test. The association between ΔP and mortality was assessed using multivariate logistic regression, adjusting for age, APACHE II score, and PaO_2/FiO_2 ratio. A Receiver Operating Characteristic (ROC) curve was constructed to determine the area under the curve (AUC) for ΔP as a predictor of mortality. A p-value < 0.05 was considered statistically significant.

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RESULTS

The study cohort comprised 100 patients with a mean age of 48.2 pm 14.5 years, of whom 62% were male. The primary causes of ARDS were pneumonia (44%), non-pulmonary sepsis (32%), and trauma (14%). At the time of enrollment, the mean APACHE II score was 18.4 pm 5.1 and the mean PaO2/FiO2 ratio was 134.6 pm 42.8 mmHg.

The 28-day mortality rate was 38%. Table 1 summarizes the baseline characteristics and ventilatory parameters stratified by ARDS severity. Patients with severe ARDS (PaO2/FiO2 ≤ 100) exhibited significantly higher driving pressures (17.8 pm 3.4 cm H2O) compared to those with mild ARDS (12.1 pm 1.9 cm H2O; p < 0.01).

Table 1: Demographic and Clinical Characteristics Stratified by ARDS Severity

Parameter	Mild (n=22)	Moderate (n=46)	Severe (n=32)	p-value
Age (years)	45.1 pm 12.3	47.8 pm 15.1	51.4 pm 13.9	0.24
Male Gender, n (%)	13 (59%)	29 (63%)	20 (62%)	0.94
APACHE II Score	14.2 pm 3.8	18.1 pm 4.5	21.7 pm 5.2	< 0.001
Mean ΔP (Day 1)	12.1 pm 1.9	14.6 pm 2.5	17.8 pm 3.4	< 0.001
Tidal Volume (mL/kg PBW)	6.4 pm 0.6	6.5 pm 0.7	6.3 pm 0.8	0.41
PEEP (cm H_2O)	8.2 pm 1.5	10.4 pm 2.1	12.6 pm 2.8	< 0.001
28-Day Mortality, n (%)	3 (13.6%)	14 (30.4%)	21 (65.6%)	< 0.001

Non-survivors presented with significantly higher mean driving pressures throughout the first 72 hours of ventilation. On Day 1, the mean ΔP in non-survivors was 16.7 pm 3.1 cm H_2O compared to 12.4 pm 2.2 cm H_2O in survivors (p < 0.001). Table 2 details the stratification of clinical outcomes based on driving pressure quartiles.

Table 2: Clinical Outcomes Stratified by Driving Pressure Quartiles

ΔP Quartile (cm H_2O)	n	Mortality (%)	VFD (Days)	ICU LOS (Days)
Q1 (< 12.5)	25	8.0%	21.4 pm 4.2	8.4 pm 3.1
Q2 (12.5–14.8)	25	24.0%	17.8 pm 5.1	11.2 pm 4.5
Q3 (14.9–17.2)	25	48.0%	12.5 pm 6.4	14.8 pm 5.9
Q4 (> 17.2)	25	72.0%	6.2 pm 5.5	19.1 pm 7.2
Trend p-value		< 0.001	< 0.001	< 0.01

Logistic regression analysis revealed that for every 1 cm H_2O increase in ΔP on Day 1, the odds of death increased by 42% (OR: 1.42; 95% CI: 1.18–1.71; p = 0.0002). In contrast, neither tidal volume (p = 0.32) nor PEEP (p = 0.11) was an independent predictor when ΔP was included in the model.

The ROC curve for driving pressure as a predictor of 28-day mortality yielded an AUC of 0.84 (95% CI: 0.76–0.92). A cutoff value of 15.0 cm H_2O provided a sensitivity of 76.3% and a specificity of 82.2%.

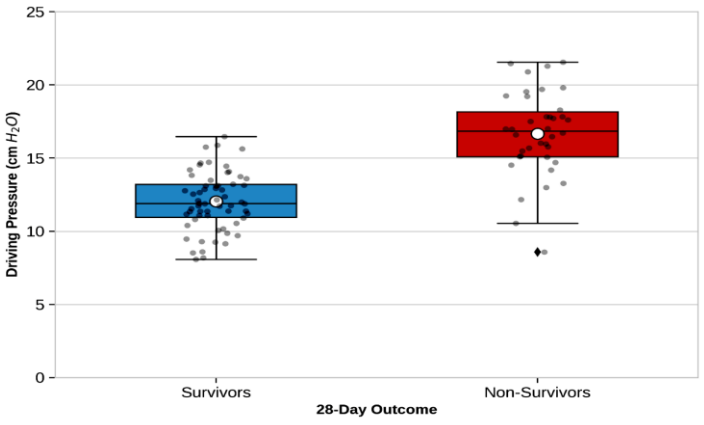

Fig. 1: Distribution of Driving Pressure in Survivors vs. Non-Survivors

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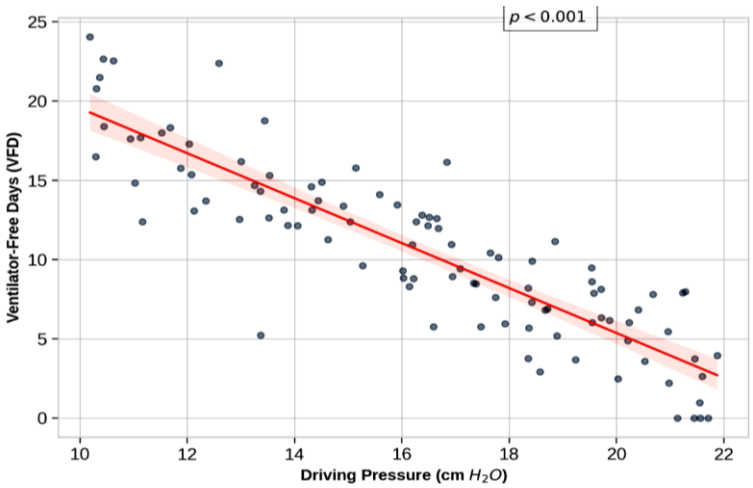

Fig. 2: Correlation Between Driving Pressure and Ventilator-Free Days

DISCUSSION

The findings of this prospective study underscore the critical role of driving pressure as a prognostic marker in ARDS patients. Our results demonstrate that higher driving pressures are independently associated with increased 28-day mortality, longer ICU stays, and fewer ventilator-free days. Specifically, a threshold of > 15 cm H_2O significantly differentiated between patients with favorable and poor outcomes.

The physiological rationale for these findings lies in the heterogeneity of lung involvement in ARDS. While the ARDSNet trial established 6 mL/kg PBW as the standard for lung protection, this fixed volume does not account for the varying size of the "functional" or "aerated" lung [6]. In patients with severe ARDS, the aerated lung volume (the "baby lung") may be as small as 20–30% of normal. In such cases, even a 6 mL/kg tidal volume can result in significant over-distension of the remaining functional alveoli, leading to VILI [7]. Driving pressure, by incorporating respiratory system compliance (ΔP = V_T/ C_stat), provides a surrogate measure of the cyclic strain imposed on the functional lung units [8].

Our data align with the retrospective analysis by Amato *et al.,* which analyzed 3,562 patients across nine RCTs and found ΔP to be the most robust predictor of mortality [5]. In our cohort, every 1 cm H_2O increase in ΔP was associated with a 42% increase in mortality risk, which is substantially higher than the 5% risk increase reported by Amato. This discrepancy may be attributed to the higher baseline severity of illness and higher APACHE II scores in our population, reflecting the challenges of tertiary care in a resource-limited setting.

The LUNG SAFE study highlighted that ARDS is often under-recognized and that clinicians frequently use suboptimal PEEP and tidal volume settings [2]. Our study observed a similar trend, where mortality was significantly higher in the severe ARDS subgroup. Interestingly, our analysis showed that PEEP itself was not a predictor of survival. This suggests that the "best" PEEP is not a fixed high or low value, but rather the level that optimizes lung recruitment without causing over-distension, ultimately resulting in a lower driving pressure [9].

One of the strengths of this study is its prospective nature and the focus on secondary outcomes like ventilator-free days. The strong negative correlation between ΔP and VFD (r = -0.68) suggests that high driving pressure not only increases the risk of death but also contributes to pulmonary morbidity in survivors, likely due to the inflammatory cascade triggered by barotrauma and biotrauma [10].

Clinical implications of our study include the suggestion that ΔP should be monitored as a primary safety target at the bedside. If ΔP exceeds 15 cm H_2O, clinicians should consider re-evaluating the ventilatory strategy, potentially by further reducing tidal volume (e.g., to 4–5 mL/kg) or adjusting PEEP to improve compliance [11,12]. Adjunctive therapies such as prone positioning, which improves lung recruitment and compliance, have been shown to reduce mortality, likely by facilitating a reduction in driving pressure [13,14].

Several limitations warrant consideration. First, this was an observational study; therefore, we cannot definitively conclude that active titration of ventilation to a specific ΔP target would improve outcomes, though the

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association is strong. Second, we measured total respiratory system driving pressure, which includes the contribution of the chest wall. In patients with high intra-abdominal pressure or obesity, transpulmonary driving pressure measured via esophageal manometry might be more accurate, though this was not feasible in our setting [15,16]. Finally, the sample size of 100, while adequate for our primary objective, may limit the power of subgroup analyses.

CONCLUSIONS

In conclusion, driving pressure is a powerful independent predictor of survival in mechanically ventilated patients with ARDS. A driving pressure greater than 15 cm H2O on the first day of ventilation is associated with a markedly higher risk of 28-day mortality and prolonged mechanical ventilation. Clinicians should incorporate the measurement of P_plat and calculation of ΔP into routine ICU care to better individualize lung-protective ventilation and improve patient outcomes. Future randomized controlled trials are needed to determine if a ΔP-limited ventilation strategy is superior to conventional tidal volume-limited strategies.

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CONTRIBUTION OF AUTHORS

Research concept- Dr Arpita Jena
Research design- Dr Manisha Mund
Supervision- Dr Deepti Swain
Materials- Dr Deepti Swain
Data collection- Dr OMM
Data analysis and interpretation- Dr Arpita, Dr Manisha
Literature search- Dr Deepti
Writing article- Dr Manisha, Dr Deepti
Critical review- Dr Arpita
Article editing- Dr Manisha, Dr Deepti
Final approval- Dr Arpita

REFERENCES

[1] ARDS Definition Task Force. Acute respiratory distres syndrome: the Berlin Definition. JAMA, 2012; 307(23): 2526-33.
[2] Bellani G, Laffey JG, Pham T, Fan E, Brochard L, Esteban A, et al. Epidemiology, patterns of care, and mortality for patients with acute respiratory distress syndrome in intensive care units in 50 countries. JAMA, 2016; 315(8): 788-800.
[3] Acute Respiratory Distress Syndrome Network. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. N Engl J Med., 2000; 342(18): 1301-08.
[4] Gattinoni L, Pesenti A. The "baby lung" became an anthropomorphic concept. Crit Care, 2020; 24(1): 234.
[5] Amato MB, Meade MO, Slutsky AS, Brochard L, Costa EL, Schoenfeld DA, et al. Driving pressure and survival in the acute respiratory distress syndrome. N Engl J Med., 2015; 372(8): 747-55.
[6] Terragni PP, Rosboch G, Tealdi A, Corno E, Menaldo E, Davini O, et al. Tidal hyperinflation during low tidal volume ventilation in acute respiratory distress syndrome. Am J Respir Crit Care Med., 2007; 175(2): 160-66.
[7] Villar J, Martín-Rodríguez C, Domínguez-Berrot AM, Fernández L, Ferrando C, Soler JA, et al. A quantile analysis of plateau and driving pressures: effects on mortality in patients with acute respiratory distress syndrome. Crit Care Med., 2017; 45(5): 843-50.
[8] Grieco DL, Chen L, Brochard L. Transpulmonary pressure: importance and limits. Ann Transl Med. 2017; 5(14): 285.
[9] Chiumello D, Carlesso E, Cadringher P, Caironi P, Valenza F, Polli F, et al. Lung stress and strain during mechanical ventilation for acute respiratory distress syndrome. Am J Respir Crit Care Med., 2008; 178(4): 346-55.
[10] Papazian L, Aubron C, Brochard L, Chiche JD, Guérin C, Guildner D, et al. Formal guidelines: management of acute respiratory distress syndrome. Ann Intensive Care, 2019; 9(1): 69.
[11] Goligher EC, Ferguson ND, Brochard LJ. Clinical challenges in mechanical ventilation. Lancet, 2016; 387(10030): 1856-66.
[12] Costa EL, Amato MB. Driving pressure: a simple and powerful tool for monitoring ventilation in ARDS. Lancet Respir Med., 2016; 4(10): 767-68.
[13] Guérin C, Reignier J, Richard JC, Beuret P, Gacouin A, Boulain T, et al. Prone positioning in severe acute

doi: 10.21276/SSR-IIJLS.2026.12.3.52

respiratory distress syndrome. N Engl J Med., 2013; 368(23): 2159-68.

[14]Beitler JR, Majumdar R, Hubmayr RD, Malhotra A, Thompson BT, Brower RG, et al. Volume-delivered fixed PEEP vs low-Pplat titration: a randomized trial. JAMA, 2019; 321(9): 846-57.

[15]Mauri T, Yoshida T, Bellani G, Goligher EC, Carteaux G, Rittayamai N, et al. Esophageal and transpulmonary pressure in the clinical setting: meaning, usefulness and perspectives. Intensive Care Med., 2016; 42(9): 1360-73.

[16]Grasso S, Terragni P, Mascia L, Fanelli V, Quintel M, Herrmann P, et al. Airway pressure-time curve profile (stress index) detects tidal hyperinflation in ARDS patients. Crit Care Med., 2004; 32(4): 1018-27.

[17]Thompson BT, Chambers RC, Liu KD. Acute Respiratory Distress Syndrome. N Engl J Med., 2017; 377(6): 562-72.

[18]Papazian L, Forel JM, Gacouin A, Penot-Ragon C, Perrin G, Loundou A, et al. Neuromuscular blockers in early acute respiratory distress syndrome. N Engl J Med., 2010; 363(12): 1107-16.

[19]Bozza FA, Salluh JI, Japiassu AM, Soares M, Assis EF, Gomes RN, et al. Outcomes of patients with acute respiratory distress syndrome in Brazilian intensive care units: a multicenter study. Crit Care Med., 2021; 49(5): e487-96.

[20]Slutsky AS, Ranieri VM. Ventilator-induced lung injury. N Engl J Med., 2013; 369(22): 2126-36.