Physiologic Wins Without Survival Gain? Prone Positioning in VV-ECMO-Treated ARDS

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Abstract

Aim: Prone positioning (PP) is a well-established strategy to improve oxygenation in acute respiratory distress syndrome (ARDS) patients; however, its role during Venovenous Extracorporeal Membrane Oxygenation (VV-ECMO) remains uncertain. The aim of this study was to evaluate the impact of PP on Intensive Care Unit (ICU) survival and respiratory parameters in patients with ARDS supported by VV-ECMO.

Methods: This retrospective observational study included 100 adult patients who underwent VV-ECMO for respiratory failure between 2015 and 2024. Patients were divided into two groups: Those who received prone positioning during ECMO (≥16 hours) and those who remained supine. Clinical characteristics, ventilatory parameters, and outcomes were compared.

Results: Forty-five patients (45%) underwent PP during ECMO. Although ICU survival was higher in the prone group (53.7% vs. 46.3%), the difference was not statistically significant (p=0.246). However, PP was associated with significant improvements in driving pressure, mechanical power, compliance, and gas exchange indices in the the period following PP.

Conclusions: While PP during VV-ECMO did not significantly improve survival, it contributed to favorable physiological changes, supporting its use as an adjunctive therapy in ARDS management under ECMO.

Keywords: *Prone positioning; ECMO; respiratory mechanics; ARDS; lung protective ventilation*

1. Introduction

Extracorporeal membrane oxygenation (ECMO) has become a pivotal intervention for managing refractory acute respiratory failure and cardiogenic shock, particularly in patients with severe acute respiratory distress syndrome (ARDS) [1]. Although ECMO delivers indispensable support for gas exchange, it does not replace the need for concomitant lung and diaphragm protective ventilation (LDPV) strategies [2]. Moreover, multiple randomized controlled trials have consistently demonstrated that prone positioning (PP) markedly enhances oxygenation and reduces mortality in ARDS patients [3,4]. The physiological rationale underpinning the efficacy of PP comprises three principal mechanisms: (1) improved ventilation perfusion (V/Q) matching via dorsal lung recruitment; (2) enhanced clearance of pulmonary secretions through gravitational drainage; and (3) attenuation of dorsal lung compression and atelectasis by promoting a more uniform distribution of transpulmonary pressures [5–7].

Nevertheless, implementation protocols and the clinical benefits of PP in ECMO-supported patients remain poorly defined, as these individuals were systematically excluded from the major trials evaluating PP. Specific challenges in this population include logistical barriers, the risk of circuit dislodgement, and potential hemodynamic instability. Recent studies and growing clinical experience, however, indicate that PP can be performed safely and effectively in the ECMO setting when conducted by trained teams following standardized protocols [8,9].

Despite its increasing use, there remains no consensus or guideline endorsed recommendation for routine PP during ECMO. The primary objective of this study was to determine whether PP during veno-venous ECMO (VV-ECMO) is associated with a difference in ICU mortality. Secondary objectives included evaluating the effects of PP on respiratory mechanics and gas exchange parameters.

2. Materials and Methods

Following approval by the Bakırköy Dr. Sadi Konuk Training and Research Hospital Ethics Committee (Decision No. 2025-202), we conducted a retrospective observational study in the hospital's ICU. Patient data between June 2015 and December 2024 were extracted from the ImdSoft Metavision/QlinICU Clinical Decision Support System (ImdSoft, Israel) using structured query language queries. Inclusion criteria were: age ≥18 years, patients under invasive mechanical ventilation and ECMO treatment for respiratory failure, and availability of complete ventilatory, ECMO and clinical data.

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The primary evaluation endpoint of the study was ICU mortality. Secondary endpoints included changes in respiratory mechanics and gas exchange parameters, including driving pressure, mechanical power, lung compliance, oxygenation index, oxygen saturation index, respiratory rate, and lactate levels. Physiological variables were evaluated as secondary outcomes to explore mechanistic effects of prone positioning during ECMO.

Patients were stratified into two cohorts. The prone cohort comprised patients who underwent at least one PP session during ECMO, with a cumulative duration ≥ 16 hours, whereas the supine cohort included those maintained exclusively in the supine position throughout ECMO support. Mechanical ventilation was delivered via Maquet Servo-i ventilators (Getinge, Sweden), utilizing both pressure-controlled ventilation (PCV) and volume-controlled ventilation (VCV) modes according to standard protocols. All patients were managed according to institutional protocols for PP and ECMO support. Ultra-protective MV settings including tidal volumes <4 mL kg^{-1} predicted body weight, positive end-expiratory pressure (PEEP)> 10 cm H_2O, plateau pressures ≤ 24 cmH_2O, and respiratory rates (RR) ≤ 20 breaths min^{-1} were maintained in both cohorts throughout ECMO therapy. Both groups followed a standardized ECMO weaning protocol. Clinical assessments were performed every 12 hours once patients met at least one of the following criteria:

- Increased respiratory system compliance
- Improved arterial oxygenation
- Favorable chest imaging findings

However, any patient who exhibited a decrease in respiratory system compliance exceeding 10% or deterioration in arterial oxygenation on blood-gas analysis over a continuous 12 hour period despite adherence to the ultra protective MV strategy and an ECMO delivered FiO_2 of 100% was repositioned into the PP at the attending clinician's discretion. Prone positioning was not applied according to a fixed or protocolized algorithm. Instead, it was implemented as a clinician-driven, time-dependent intervention based on serial physiological assessments during ECMO support.

Minute time slots were transferred from the data pool to Microsoft Excel as hourly time slots through the functions of SQL queries. The hourly mechanical ventilation parameters in the Excel dataset were calculated at 24-hour intervals (days) using the LEFT function of the Excel program as follows:

It was calculated by taking the difference of the first 10 characters of the signal date (Time 1) of the parameters transferred to the software from the mechanical ventilator and the first 10 characters of the patient's admission to the ICU (Time 2) (=LEFT (Time 1;10) - (LEFT(Time 2;10)).

Data Collection

Patients were categorized as prone and non-prone groups.

The following variables were collected:

- Demographics: Age, sex, body mass index (BMI), ICU survival status.

Clinical scores: Acute physiology and chronic health evaluation (APACHE) II, Sequential organ failure assessment (SOFA) (initial and final).

- ARDS etiology: Covid-19, H_1N_1, Bacterial, etc
- Anticoagulation: Bivalirudin, heparin
- PP duration (hours)
- PP before ECMO (yes/no)
- Ventilatory parameters at two time points: 1.before PP, 2.after PP
    - Driving pressure (DP)
    - Mechanical power (MP)
    - RR, compliance, PEEP
    - Gas exchange indices: lactate, oxygenation index (OI), oxygen saturation index (OSI)

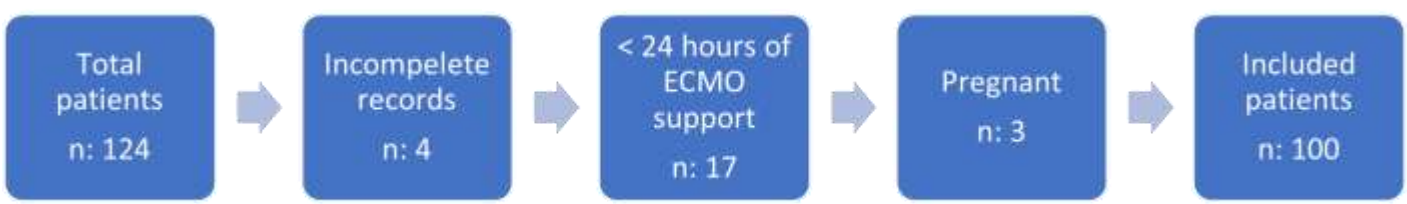
A priori sample size estimation was performed using G*Power software (v. 3.1, Düsseldorf, Germany). Based on a two-sided significance level of 0.05, a statistical power of 80%, an assumed common variance of 1, and an expected maximum difference of 2.5 units in mean driving pressure between prone and supine groups, a minimum sample size of 100 patients was estimated. The a priori sample size calculation was designed to detect differences in physiological parameters, particularly driving pressure, and not to evaluate mortality outcomes, which were analyzed as secondary exploratory endpoints.

Statistical Analysis

The data were analyzed using IBM SPSS ver. 27 (IBM Corp., Armonk, NY, USA). The categorical variables are expressed as proportions and frequencies. The Kolmogorov-Smirnov test was applied to test the normality distribution. The quantitative variables are summarized as Mean±Standard deviation (SD). To explore the impacts of categorical independent variables, the chi-square test was used. Mean values were compared between two groups using the independent or paired t-test, as well as using non-parametric tests, such as the Mann-Whitney U-test. Friedman test was applied to comparison clinical measurements during times in each group. P-values less than 0.05 were considered to indicate statistical significance. Effect size estimates (Cohen's d) were calculated and reported for continuous physiological variables to support interpretation of the magnitude of observed changes. The general guidelines for interpreting the effect size are as follows: 0.0- 0.5 = small effect, 0.5- 0.8 = moderate effect, >=0.8 = large effect.

Figure 1

Flow diagram showing the number of patients assessed for VV-ECMO



3. Results

Patients with incomplete records (n = 4) or <24 hours of ECMO support (n = 17), and pregnant patients (n = 3) were excluded from the study. After applying exclusion criteria, 100 patients were included in the final analysis, of whom 45 (45%) underwent PP during ECMO and 55 (55%) remained exclusively in the supine position (Figure 1).

Baseline characteristics including age, sex, BMI, duration of ECMO support, APACHE II score, and initial SOFA score did not differ significantly between the cohorts. In contrast, the final SOFA score was significantly lower in the prone cohort compared with the supine cohort (p = 0.022) (Table 1).

Both cohorts demonstrated temporal improvements in ventilatory and gas exchange parameters during ECMO support; however, at the post-prone assessment, the prone cohort exhibited significantly greater improvements than the supine cohort, with lower driving pressure (p = 0.041), reduced mechanical power (p = 0.021), a greater increase in static compliance (p = 0.040), lower oxygenation index (p = 0.036) and oxygen saturation index (p = 0.023), reduced respiratory rate (p = 0.029), lower PEEP levels (p = 0.014), and decreased lactate concentrations (p = 0.021) (Table 2).

No significant intergroup differences were observed for these parameters at the pre-prone time point, indicating that the divergences emerged specifically after the initiation of PP.

ICU survival was 53.7% (n : 22) in the prone cohort versus 46.3% (n : 19) in the supine cohort; this difference did not reach statistical significance (p = 0.246). Likewise, no significant association was observed between PP duration and survival status (p = 0.803) (Table 3).

The distribution of different causes of ARDS was similar between groups (Table 4).

Likewise, no statistically significant differences were observed in comorbidity profiles, including pulmonary, cardiovascular, metabolic, and renal diseases (Table 5).

Table 1

Baseline and demographic characteristics comparison between two groups

	Non-prone n=55 Mean±SD	prone n=45 Mean±SD	p value	Cohen's d
Gender, Male n (%)	62%	58%	0.672	0.08
Age (Year)	45.2 ± 14.3	42.5 ± 12.1	0.321	0.20
BMI (Kg/m2)	27.9 ± 6.1	30.2±7.3	0.716	0.36
ECMO Duration (Hour)	298 ± 142	275 ± 158	0.519	0.15
APACHE II	23.1 ± 7.2	24.5 ± 6.7	0.384	0.20
SOFA initial	9.8 ± 3.4	10.2 ± 3.1	0.541	0.12
SOFA final	8.1 ± 3.2	6.4 ± 2.8	0.022*	0.56

**significant at 0.05 level*

BMI: Body mass index, ECMO: Extracorporeal membrane oxygenation, APACHE: Acute Physiology and Chronic Health Evaluation, SOFA: Sequential Organ Failure Assessment.

Table 3

The comparison of prone position duration between survived and non-survived groups and survival status of patients in prone and non-prone groups.

	Non-survived	Survived	p value
Prone group (n) (%)	23 (39.0)	22 (53.7)	0.246
Non-prone group (n) (%)	36 (61.0)	19 (46.3)	
Prone Duration (Hour) Mean±SD	22.9±8.7	23.3±7.6	0.803
Cohen's d		0.29	

Table 2

Respiratory mechanics and laboratory parameters between prone and non-prone group during pre-prone and post-prone periods.

Pre-prone period	Non-Prone n=55 Mean±SD	Prone n=45 Mean±SD	p value	Cohen's d	Post-prone period	Non-Prone n=55 Mean±SD	Prone n=45 Mean±SD	p value	Cohen's d
DP	19.5 ± 5.3	18.2 ± 4.1	0.231	0.27	DP	14.7 ± 4.2	12.1 ± 3.8	0.041*	0.65
MP	21.4 ± 7.1	19.8 ± 6.2	0.156	0.24	MP	9.8 ± 4.3	7.5 ± 3.1	0.021*	0.60
Compliance	26 ± 10	28 ± 11	0.320	0.19	Compliance	35 ± 13	29 ± 12	0.040*	0.58
OI	13.8 ± 6.3	12.4 ± 5.1	0.188	0.24	OI	8.9 ± 3.5	6.2 ± 2.8	0.036*	0.84
OSI	10.3 ± 4.2	9.5 ± 3.8	0.278	0.20	OSI	7.6 ± 3.1	5.1 ± 2.3	0.023*	0.90
RR	19.5 ± 5.3	18.2 ± 4.1	0.154	0.27	RR	16.7 ± 4.2	14.1 ± 3.8*	0.029*	0.65
PEEP	10.8 ± 3.1	10.2 ± 2.8	0.312	0.20	PEEP	10.1 ± 2.8	8.5 ± 2.3	0.014*	0.62
Lactate	3.6 ± 2.1	3.4 ± 1.9	0.611	0.09	Lactate	3.0 ± 1.7	2.1 ± 1.3*	0.021*	0.59

**significant at 0.05 level.*

DP: Driving pressure, MP: Mechanical power, OI: Oxygenation index, OSI: Oxygen saturation index, RR: Respiratory rate, PEEP: Positive end-expiratory pressure.

Table 4

Etiology of ARDS patients in prone and non-prone group.

Diagnosis	Prone Group (n=45)	Non-Prone Group (n=55)	p value	Cohen's d
COVID-19	23 (51.1%)	26 (47.3%)	0.701	0.07
Bacterial	5 (11.1%)	6 (10.9%)	0.972	<0.05
Aspiration	4 (8.9%)	6 (10.9%)	0.725	0.06
H1N1 Influenza	2 (4.4%)	3 (5.5%)	0.814	0.05
Other Infectious ARDS	2 (4.4%)	1 (1.8%)	0.466	0.15

ARDS: Acute respiratory distress syndrome.

Table 5

Comorbidities between prone and non-prone group.

Comorbidity	Prone Group (n=45)	Non-Prone Group (n=55)	p value
Pulmonary	15 (34%)	16 (29%)	0.478
- COPD/Asthma	8 (18%)	8 (15%)	0.616
- Interstitial Lung disease	4 (8%)	4 (7%)	0.825
- Cystic Fibrosis	3 (8%)	4 (7%)	0.822
Cardiovascular	12 (27%)	14 (25%)	0.750
- Hypertension	7 (15%)	8 (14%)	0.846
- CAD	4 (9%)	4 (8%)	0.801
- Heart Failure	1 (3%)	2 (3%)	1.004
Metabolic	10 (22%)	10 (18%)	0.491
Diabetes	8 (19%)	9 (16%)	0.586
-Obesity (BMI ≥30)	2 (3%)	1 (2%)	0.674
Other	8 (17%)	15 (28%)	0.067
- CKD	2 (5%)	4 (7%)	0.577

COPD: Chronic obstructive pulmonary disease, CAD: Coronary artery disease, BMI: Body mass index, CKD: Chronic kidney disease.

4. Discussion

This study investigated the role of PP in ECMO patients, with particular emphasis on survival outcomes and physiological effects. PP was not associated with a statistically significant difference in ICU mortality, acknowledging the limitations imposed by time-dependent exposure and confounding by indication, but it was consistently associated with marked improvements in respiratory mechanics and oxygenation parameters. These results suggest that while PP may not enhance survival in this population, it retains clinical value as an adjunctive therapy for optimizing physiological parameters in ARDS patients with ECMO support.

Prone positioning in ECMO supported patients significantly improves respiratory mechanics, including reduced DP and MP, as well as enhanced lung compliance. These benefits likely arise through two primary mechanisms:(1) mitigation of ventilator-induced lung injury (VILI) via optimized stress/strain distribution, and (2) promotion of pulmonary recovery through improved alveolar recruitment and more homogeneous ventilation [10,11]. Consistent with these physiological effects, patients undergoing PP demonstrate marked improvements in OI and OSI, reflecting enhanced gas exchange efficiency due to reduced intrapulmonary shunting and better alveolar recruitment [5,12]. These findings suggest that the clinical value of PP during ECMO may be greatest when applied early and in carefully selected patient subgroups, an aspect that warrants further investigation in prospective studies.

Despite these advantages, PP has not been associated with significant survival benefits in ECMO patients as it observed both in our cohort and prior studies [13,14]. Several factors may explain this finding. First, the timing of PP initiation during ECMO was not standardized and was left to clinician discretion; delayed initiation may attenuate any potential survival benefit, as earlier application of PP has been associated with more pronounced effects in non-ECMO ARDS populations. Second, ECMO duration varied substantially among patients, reflecting differences in disease severity and recovery trajectories, which may have diluted the impact of PP on mortality outcomes. Third, the heterogeneous nature of the study population including variability in ARDS etiology (notably COVID-19 versus non-COVID ARDS), baseline organ dysfunction, and comorbidity burden suggests that distinct patient phenotypes may respond differently to PP. Importantly, mortality in ECMO-supported patients is frequently driven by extrapulmonary factors such as multiorgan failure, cardiovascular dysfunction, and infectious complications, which may overshadow improvements in respiratory mechanics and gas exchange. Nearly half of the study population consisted of patients with COVID-19–related ARDS, a condition that has been suggested to exhibit distinct pathophysiological features compared with non–COVID-19 ARDS. Although baseline characteristics and the distribution of COVID-19 infection were similar between the prone and non-prone groups, the present study was not designed or powered to support phenotype-specific subgroup analyses. Therefore, COVID-19 status was considered a source of clinical heterogeneity rather than a stratification variable for outcome assessment. Consequently, the observed physiological effects of PP should be interpreted in the context of a mixed ARDS population, and caution is warranted when extrapolating these findings to specific ARDS phenotypes. Collectively, these factors indicate that PP primarily optimizes pulmonary physiology during ECMO, while its impact on survival may depend on early application and careful patient selection. Nevertheless, the robust physiological improvements such as reduced DP and MP support PP's role in facilitating LDPV and creating optimal conditions for recovery [5,15,16].

Observational studies by Giani et al. and Zaaqoq et al., confirms that PP safely enhances respiratory parameters without increasing complications. While its impact on survival remains neutral, the intervention's ability to improve lung mechanics and oxygenation underscores its clinical utility in managing ECMO supported patients [17,18].

Recent meta-analyses have further strengthened the evidence base. Chan et al. and Navalesi et al. systematically demonstrated PP's capacity to improve oxygenation (P/F ratios), reduce DP, and potentially decrease ECMO duration while maintaining an excellent safety profile [19, 20].

Despite demonstrating favorable physiological effects, prone positioning was not independently associated with improved ICU mortality in this cohort. This finding underscores the complex and multifactorial nature of outcomes in ECMO-treated ARDS, where survival is influenced by multiple factors beyond short-term improvements in respiratory mechanics, including underlying disease severity, extrapulmonary organ dysfunction, and complications related to ECMO support. Accordingly, the absence of a survival benefit in our study should not be interpreted as a lack of physiological efficacy, but rather as a reflection of the limitations inherent to observational analyses of critically ill populations.

This study has several limitations that should be acknowledged. First, the observational and single-center design introduces the possibility of selection bias and limits generalizability. Unmeasured

confounders, including clinician-driven decisions regarding PP and undocumented disease severity markers, may have influenced both treatment allocation and outcomes. In addition, PP was applied as a time-dependent and clinically driven intervention during ECMO support. Patients were required to survive long enough and to exhibit clinical indications to receive PP, which may have introduced confounding by indication as well as immortal time bias. Accordingly, comparisons of ICU mortality between prone and non-prone groups should be interpreted descriptively, and causal inference regarding survival cannot be established.

The study focused on ICU mortality and short-term physiological parameters; long-term survival, functional outcomes, and quality-of-life measures were not assessed. In addition, a substantial proportion of patients had COVID-19 related ARDS, which may represent a source of clinical heterogeneity. Finally, the absence of propensity score matching or multivariable adjustment represents an additional limitation, and residual confounding cannot be excluded despite comparable baseline characteristics between groups.

5. Conclusion

In VV-ECMO supported ARDS patients, PP was not associated with reduced ICU mortality but led to significant improvements in respiratory mechanics and gas exchange, including lower driving pressure and mechanical power and improved lung compliance. These findings suggest that PP during ECMO should be considered a physiological optimization strategy to facilitate lung- and diaphragm-protective ventilation rather than a survival-directed intervention. When applied by experienced teams, PP may be beneficial in selected patients with persistent lung stress or impaired oxygenation despite ultra-protective ventilation. Further prospective studies are needed to refine patient selection and treatment protocols.

Statement of ethics

The study received ethical approval by the Bakırköy Dr. Sadi Konuk Training and Research Hospital Ethics Committee (Decision No. 2025 202) and was conducted in accordance with the principles of the Declaration of Helsinki

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Conflict of interest statement

The author declare that they have no conflict of interest.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

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