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Conventional protective ventilation with elevated mechanical power: Migration to safer values with small ventilatory adjustments

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Abstract

Background
Mechanical power (MP) integrates multiple ventilatory variables into a single measure of the energy delivered to the respiratory system and has been associated with ventilator-induced lung injury and adverse outcomes. However, it remains unclear whether ventilatory observations meeting conventional protective criteria also exhibit low MP and whether small ventilatory adjustments can meaningfully reduce MP in severely burned patients undergoing pressure-controlled ventilation (PCV).
Methods
This interventional repeated-measures study with randomized sequence of ventilatory adjustment protocols included adult burn patients receiving PCV in an intensive care unit. A total of 123 paired ventilatory observations were screened. Three reductive ventilatory strategies were assessed: reduction of inspiratory pressure variation by 2 cmH_2O (ΔPinsp), reduction of respiratory rate by 2 breaths/min (RR), and combined reductions of 1 cmH_2O and 1 breath/min (RR–ΔPinsp). Thirty-nine ventilatory observations meeting predefined protective tidal-volume and pressure criteria constituted the analytical sample. MP was calculated using the simplified Becher equation. Clinical, arterial blood gases, and ventilatory effects were evaluated at baseline and one hour after the interventions, as well as migration between MP categories (<1 8 and ≥ 18 J/min).
Results
Oxygenation remained stable, with no significant changes in PaO_2, SpO_2, or PaO_2/FiO_2. $PaCO_2$ increased modestly and arterial pH decreased slightly, but both remained within physiological limits. No desaturation, clinically relevant acidosis, hemodynamic instability, or ventilatory asynchrony was observed. Among the 39 ventilatory observations, 17 (43.6%) had MP ≥ 18 J/min at baseline. MP decreased significantly from 17.39 to 15.02 J/min overall ($P < 0.001$). The greatest reduction was observed in the ΔPinsp group (3.00 J/min), followed by RR–ΔPinsp (2.34 J/min) and RR (1.78 J/min), with significant differences among strategies. Ventilatory observations with MP <18 J/min increased from 22/39 (56.4%) to 34/39 (87.2%), whereas those with MP ≥ 18 J/min decreased from 17/39 (43.6%) to 5/39 (12.8%) ($P < 0.001$).
Conclusions
A substantial proportion of ventilatory observations meeting conventional protective criteria still exhibited elevated MP. Small ventilatory adjustments during protective PCV were associated with significant reductions in calculated MP and migration toward values below 18 J/min without compromising short-term arterial blood gases or clinical stability. Reduction in inspiratory pressure variation produced the greatest effect on MP.

Keywords: Mechanical Power, Burns; Mechanical Ventilation; Pressure-Controlled Ventilation; Ventilator-Induced Lung Injury.

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Conflict of interest/Disclosures: None
This study is derived from the same study cohort and experimental protocol of previously published study in “Burns Open 14 (2026) 100445”. The present manuscript has a distinct analytical focus, specifically examining the subgroup of ventilatory observations that already met conventional protective criteria, but nevertheless had elevated MP, to analyze the migration from ≥18 to <18 J/min after the adjustments

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Introduction

Respiratory failure may develop soon after severe burns, often necessitating mechanical ventilation due to loss of consciousness, facial edema, inhalation injury, or hypoxia. [1] Although essential, mechanical ventilation can also contribute to ventilator-induced lung injury (VILI). [2] Conventional ventilatory management emphasizes static parameters: plateau pressure (Pplateau), positive end-expiratory pressure (PEEP), driving pressure (ΔP), and tidal volume (VT) but neglects dynamic factors. [3] Therefore, lung-protective strategies are recommended, with tidal volumes of 4–8 mL/kg predicted body weight, plateau pressure < 30 cmH_2O, driving pressure < 15 cmH_2O, [4] and individualized PEEP titration according to oxygenation and lung mechanics. [4]

VILI results from tissue vulnerability, the number of high-energy cycles applied per unit of time, and the duration of exposure. [5] Mechanical power (MP) integrates respiratory rate (RR), strain, and stress as determinants of the total energy delivered to the lungs. [6] It has emerged as a comprehensive index for optimizing protective ventilation, [2] although its long-term clinical effects remain uncertain. [7] In burn patients, mean MP values around 22.8 J/min have been reported, [8] exceeding thresholds associated with greater VILI risk (> 12 J/min), mortality (> 17 J/min), [9] and a 0.1% hourly rise in mortality when values surpass 18 J/min. [8] In acute respiratory distress syndrome (ARDS), survivors spent a mean of 151 hours with MP >18 J/min, compared with 275 hours in non-survivors. [10]

MP may therefore reflect excessive ventilator settings and increased VILI risk. [11] Ultimately, the therapeutic goal of mechanical ventilation to rest and protect the lungs may be undermined when parameters exceed safe thresholds, thereby increasing MP. [12] Refining ventilator settings to minimize MP while maintaining adequate oxygenation and safety is therefore essential. [13] However, the relative contributions of VT, pressures, and RR to MP and VILI risk remain uncertain. [14,15] Although tidal volume and pressure thresholds are widely used to define protective ventilation, it remains unclear whether ventilations that satisfy these conventional criteria also exhibit low mechanical power. Consequently, a proportion of patients may remain exposed to elevated MP despite apparently protective ventilatory settings. In addition, it remains uncertain whether small reductions in ventilatory load are sufficient to meaningfully reduce MP and facilitate migration toward values below 18 J/min, a threshold previously associated with more favorable prognostic profiles.

The primary objective of this study was to evaluate the effect of three small reductive ventilatory strategies on mechanical power (MP) in severely burned patients undergoing pressure-controlled ventilation (PCV) with protective pressure and tidal-volume settings. Secondary objectives were to assess the clinical and arterial blood gas effects of these interventions and to determine whether modest ventilatory adjustments could promote migration from MP ≥ 18 J/min to MP < 18 J/min. The primary outcome was the change in calculated MP from baseline to 1 h after the assigned ventilatory adjustment. Secondary outcomes included changes in arterial blood gas variables, short-term clinical tolerance, and migration between MP categories (< 18 and ≥ 18 J/min).

Methods

The study protocol was approved by the Ethics Committee of Mackenzie Evangelical College of Paraná (protocol no. 7.151.837). Written informed consent was obtained from the legal representatives of all patients before enrollment.

Study design and participants

We conducted an analytical, interventional repeated-measures study in adult burn patients, in which the order of the ventilatory adjustment protocols was randomized. Patients were admitted to the Intensive Care Unit (ICU) of Hospital Universitário Evangélico Mackenzie (HUEM) between 1 February and 10 July 2025. All patients were under pressure-controlled ventilation (PCV) using the Magnamed® FlexiMagMax 700 ventilator (Magnamed, São Paulo, Brazil), under deep sedation and continuous analgesia, targeting a Richmond Agitation and Sedation Scale (RASS) score of –5, indicating unresponsiveness to verbal or physical stimuli [18]. Inclusion criteria were: (1) total body surface area (TBSA) burned ≥ 20% in adults (≥ 18 years) or ≥ 15% in older adults (> 60 years); (2) requirement for invasive mechanical ventilation due to acute respiratory failure within 48 h of admission; and (3) maintenance of PCV mode for at least one hour before and one hour after ventilatory adjustment.

Exclusion criteria included pre-existing chronic pulmonary disease (COPD, asthma, or restrictive disorders), hemodynamic instability requiring escalating vasopressor support, severe metabolic acidosis (arterial pH < 7.20), incomplete ventilatory or arterial blood gas data, severe ARDS according to the Berlin definition, [19] the need for neuromuscular blockade, or requirement for prone positioning.

In our ICU, patients with PaO_2/FiO_2 < 150 mmHg are frequently managed in the prone position as part of routine care for moderate to severe hypoxemia. Because prone positioning would prevent standardized assessment of the ventilatory responses evaluated in the present study, these

patients were not eligible for inclusion. Importantly, no screened patient required prone positioning at the time of evaluation. Oxygenation impairment was primarily attributed to burn-related systemic inflammatory response and/or inhalation injury. Pneumonia and sepsis-related ARDS were ruled out through clinical and laboratory evaluation before enrollment.

Repeated-measures protocol and ventilatory interventions

The study included 41 patients, each evaluated over three consecutive mornings according to a repeated-measures design. Each patient underwent only one ventilatory adjustment per day. The order in which the three ventilatory adjustment protocols were applied was randomized for each patient by simple lottery, so that the three intervention conditions were performed on different study days. The sequence of interventions was randomized for each patient by simple lottery. Because ventilatory adjustments were applied directly at the bedside, allocation concealment and blinding were not feasible. Because ventilatory adjustments were applied directly at the bedside, allocation concealment and blinding of the intervention were not feasible.

The interventions consisted of: (1) reduction of inspiratory pressure above PEEP (ΔPinsp) by 2 cmH_2O, (2) reduction of respiratory rate (RR) by 2 breaths/min, or (3) combined reduction of ΔPinsp by 1 cmH_2O and RR by 1 breath/min. All adjustments were implemented from the baseline ventilatory settings established by the ICU clinical team. Fraction of inspired oxygen (FiO_2), positive end-expiratory pressure (PEEP), and inspiratory time (Tinsp) were kept constant throughout each intervention period. To avoid interference with the protocol, no practical clinical procedures, such as dressing changes, were performed during the one-hour intervention periods.

Data collection and measurements

Demographic and anthropometric variables (age, sex, height, weight, and body mass index [BMI]) were recorded at admission. Burn extent and depth were quantified as percentage of total body surface area burned (%TBSA) and classified as second- or third-degree burns. The Abbreviated Burn Severity Index (ABSI) and corresponding predicted mortality were also determined for each patient.

Arterial blood gas sampling and ventilatory measurements were obtained under baseline conditions and again one hour after each ventilatory adjustment. Across the three study days, a total of 123 paired ventilatory observations were collected, each consisting of two time points (baseline and 1 h) for a given intervention in each patient. All procedures were conducted under stable clinical conditions, and the same team performed all measurements to ensure protocol consistency.

Arterial blood gases were analyzed using a Stat Profile Prime® analyzer (Biomedical, Waltham, MA, USA) to obtain PaO_2, $PaCO_2$, and arterial pH. Peripheral oxygen saturation (SpO_2) was recorded simultaneously. All measurements were performed while patients were within the physiological thermal range. The PaO_2/FiO_2 ratio was calculated according to the Berlin definition of ARDS.

Mechanical power (MP) was estimated using the simplified formula for PCV described by Becher et al. [13]

$$MP = 0.098 \times RR \times VT \times (PEEP + \Delta Pinsp)$$

where RR is respiratory rate (cycles·min^{-1}), VT is tidal volume (L), PEEP is positive end-expiratory pressure (cmH2O), and ΔPinsp is inspiratory pressure above PEEP (cmH2O). The constant 0.098 converts $cmH_2O \cdot L \cdot min^{-1}$ to joules per minute (J/min). Under PCV, inspiratory flow follows a decelerating pattern, and peak inspiratory pressure may approximate plateau pressure under passive conditions; therefore, ΔPinsp was operationally considered the lung-inflating pressure responsible for generating VT. Patients were continuously monitored for asynchrony, desaturation, acidosis, and hemodynamic instability.

Analytical sample and statistical analysis

The analytical unit of the study was the ventilatory observation rather than individual patients. Therefore, the same patient could contribute more than one ventilatory observation to the overall dataset and, when eligible, to the protective ventilation subset.

For the present analysis, we selected ventilatory observations meeting predefined protective ventilation criteria at baseline: tidal volume (VT) < 8 mL/kg predicted body weight, peak inspiratory pressure (Ppeak) < 30 cmH_2O, and inspiratory pressure variation (ΔPinsp) < 15 cmH_2O. These protective observations constituted the analytical sample for the mechanical power analysis.

Although the primary endpoint was the change in calculated MP, clinical interpretation focused on short term physiological tolerance, including preservation of oxygenation, arterial pH, and overall ventilatory stability.
No formal sample size calculation was performed, as this was an exploratory physiological study.

Statistical analysis

Continuous variables are presented as mean ± standard deviation or median, as appropriate, and categorical variables as absolute and relative frequencies. Because multiple observations could originate from the same patient, within-patient correlation was considered in the longitudinal analysis. Changes in continuous variables between baseline and 1 h were analyzed using mixed-effects models, with patient included as a random effect and time as a fixed effect. Comparisons in the magnitude of MP reduction among intervention strategies were performed using the Kruskal–Wallis test, followed by Dunn's post-hoc test with Holm–Bonferroni adjustment when appropriate. These between-strategy comparisons should be interpreted with caution because some patients contributed more than one ventilatory observation to the analytical sample. Changes in MP category (< 18 vs ≥ 18 J/min) between baseline and 1 h were assessed using McNemar's exact test. A two-sided P-value <0.05 was considered statistically significant. Statistical analyses were performed using GraphPad Prism (GraphPad Software, San Diego, CA, USA).

Results

Patient enrollment and derivation of ventilatory observations can be summarized as follows: 41 patients were included in the repeated-measures protocol and evaluated over three consecutive mornings, yielding 123 paired ventilatory observations. Among these, 39 ventilatory observations met the predefined protective ventilation criteria at baseline and constituted the analytical sample.

Because the analytical unit was the ventilatory observation rather than the individual patient, the same patient could contribute more than one observation across the repeated-measures protocol. These 39 protective ventilatory observations were derived from 25 unique patients and were distributed across the intervention groups as follows: 12 in the

ΔPinsp group, 12 in the RR group, and 15 in the RR–ΔPinsp group, corresponding to 31.7% of all ventilatory observations. The study included 41 patients, with ventilatory observations obtained on a median of day 3 after burn injury (IQR, 3–4; range, 3–6). The mean predicted body weight was 65.51 ± 9.78 kg (median, 66 kg; IQR, 57–76), and the mean BMI was 26.13 ± 2.80 kg/m² (median, 24.87 kg/m²; IQR, 23.25–28.21); 9 patients (22.0%) had a BMI > 30 kg/m². Mean TBSA burned was 37.6 ± 11.9% (median, 35%; IQR, 30–45%; range, 20–60%), and the mean ABSI score was 8.69 ± 2.69 (median, 9; IQR, 7–11).

Before intervention, the median tidal volume was 0.48 L (IQR, 0.42–0.54), corresponding to 7.2 mL/kg predicted body weight (IQR, 6.4–8.3). Mean peak inspiratory pressure was 24.08 ± 3.24 cmH_2O, with a median of 24 cmH_2O (IQR, 22–26). The median respiratory rate was 20 breaths/min (IQR, 18–22), and mean PEEP was 8.69 ± 1.43 cmH_2O (median, 8 cmH_2O; IQR, 8–10). The median inspiratory pressure above PEEP (ΔPinsp) was 15 cmH_2O (IQR, 14–17), with a mean of 15.39 ± 2.49 cmH_2O. Respiratory system compliance was 0.032 L/cmH_2O (IQR, 0.027–0.037), with a mean of 0.0321 ± 0.0070 L/cmH_2O. Median FiO_2 was 0.30 (IQR, 0.25–0.40), with a mean of 0.34 ± 0.11.

At baseline, 17 of the 39 protective ventilatory observations (43.6%) had mechanical power (MP) ≥ 18 J/min, whereas 22 (56.4%) had MP < 18 J/min. Mean baseline MP was 17.39 ± 4.08 J/min, with a median of 17.08 J/min.

After one hour of the reductive ventilatory interventions, MP decreased from 17.39 to 15.02 J/min overall, corresponding to a mean reduction of 2.37 J/min (P <0.001). According to intervention strategy, the mean MP reduction was 3.00 J/min (95% CI, 2.46–3.55; P < 0.001) in the ΔPinsp group, 1.78 J/min (95% CI, 1.56–1.99; P < 0.001) in the RR group, and 2.34 J/min (95% CI, 1.79–2.89; P < 0.001) in the RR–ΔPinsp group. The magnitude of MP reduction differed significantly among strategies (Kruskal–Wallis P = 0.00136), with a greater reduction in the ΔPinsp group than in the RR group after Holm–Bonferroni adjustment (adjusted P = 0.00090) (Table 1, Figure 1).

The ventilatory adjustments were accompanied by reductions in ΔPinsp, RR, and minute ventilation, while VT/kg remained within the predefined protective range. VT showed a small reduction from 0.441 to 0.421 L (P < 0.05), whereas VT/kg remained stable (6.7 vs 6.4 mL/kg, not significant), indicating preservation of the protective tidal-volume range (Figure 2).

Regarding arterial blood gas response, oxygenation remained stable after the interventions, with no significant changes in PaO_2, SpO_2, or PaO_2/FiO_2. In contrast, $PaCO_2$ increased modestly from 36.82 ± 7.36 to 38.81 ± 7.05 mmHg (P = 0.006), and arterial pH decreased slightly from 7.43 ± 0.13 to 7.42 ± 0.10 (P = 0.026), while remaining within the physiological range. No significant changes were observed in bicarbonate or lactate during the one-hour observation period (Table 2, Figure 3).

No clinically relevant deterioration in oxygenation or acid–base status was observed during follow-up. No episodes of ventilatory asynchrony, desaturation, hemodynamic instability, or clinically relevant acidosis were recorded.

Migration analysis showed that, among the 17 observations with baseline MP ≥ 18 J/min, 12 (70.6%) migrated to MP < 18 J/min after one hour, whereas 5 (29.4%) remained ≥18 J/min.

No observation with baseline MP < 18 J/min migrated to MP ≥ 18 J/min. Overall, the proportion of protective ventilatory observations with MP < 18 J/min increased from 22/39 (56.4%) at baseline to 34/39 (87.2%) after one hour, while the proportion with MP ≥ 18 J/min decreased from 17/39 (43.6%) to 5/39 (12.8%) (Figure 4).

The change in MP category was statistically significant (McNemar's exact test, P <0.001) (Table 3).

Table 1 Mechanical power before and after one hour, according to intervention strategy

Intervention group	N	Baseline MP (J/min)	MP after 1 h (J/min)	Mean MP reduction (J/min)	95% CI	P-value
ΔPinsp	12	17.83	14.82	3.00	2.46–3.55	< 0.001
RR	12	17.37	15.60	1.78	1.56–1.99	< 0.001
RR–ΔPinsp	15	17.04	14.70	2.34	1.79–2.89	< 0.001
Overall	39	17.39	15.02	2.37	—	< 0.001

Data are presented as mean values. Mean MP reduction was calculated as baseline MP minus MP after 1 h. Within-group p-values refer to the effect of time in the longitudinal analysis. The overall comparison among intervention strategies was performed using the Kruskal–Wallis test, followed by Dunn's post-hoc test with Holm–Bonferroni adjustment.
MP, mechanical power; ΔPinsp, inspiratory pressure above PEEP; RR, respiratory rate; CI, confidence interval.

Table 2: Arterial blood gas and physiological variables before and one hour after reductive ventilatory adjustments.

Variable	N	Baseline	After 1 h	P-value
pH	39	7.43 ± 0.13	7.42 ± 0.10	0.026
PaO_2 (mmHg)	39	99.39 ± 20.38	97.41 ± 17.56	0.487
$PaCO_2$ (mmHg)	39	36.82 ± 7.36	38.81 ± 7.05	0.006
SpO_2 (%)	39	97.29 ± 1.86	97.00 ± 1.87	0.103
Bicarbonate (mmol/L)	39	26.05 ± 5.17	26.38 ± 4.84	0.485
Lactate (mmol/L)	39	2.25 ± 1.51	2.28 ± 1.41	0.694
PaO_2/FiO_2 (mmHg)	39	330.32 ± 96.35	323.64 ± 88.52	0.496

Data are presented as mean ± standard deviation. P-values refer to the comparison between baseline and 1 h in the longitudinal analysis. PaO_2, arterial oxygen tension; $PaCO_2$, arterial carbon dioxide tension; SpO_2, peripheral oxygen saturation; FiO_2, fraction of inspired oxygen.

Table 3 Migration between mechanical power categories before and one hour after reductive ventilatory adjustments.

Baseline MP category	After 1 h: MP < 18 J/min	After 1 h: MP ≥ 18 J/min	Total
MP < 18 J/min	22 (56.4%)	0 (0.0%)	22 (56.4%)
MP ≥ 18 J/min	12 (30.8%)	5 (12.8%)	17 (43.6%)
Total	34 (87.2%)	5 (12.8%)	39 (100%)

Data are presented as number (% of the analytical sample). McNemar's exact test was used to compare MP categories between baseline and 1 h. MP: mechanical power.

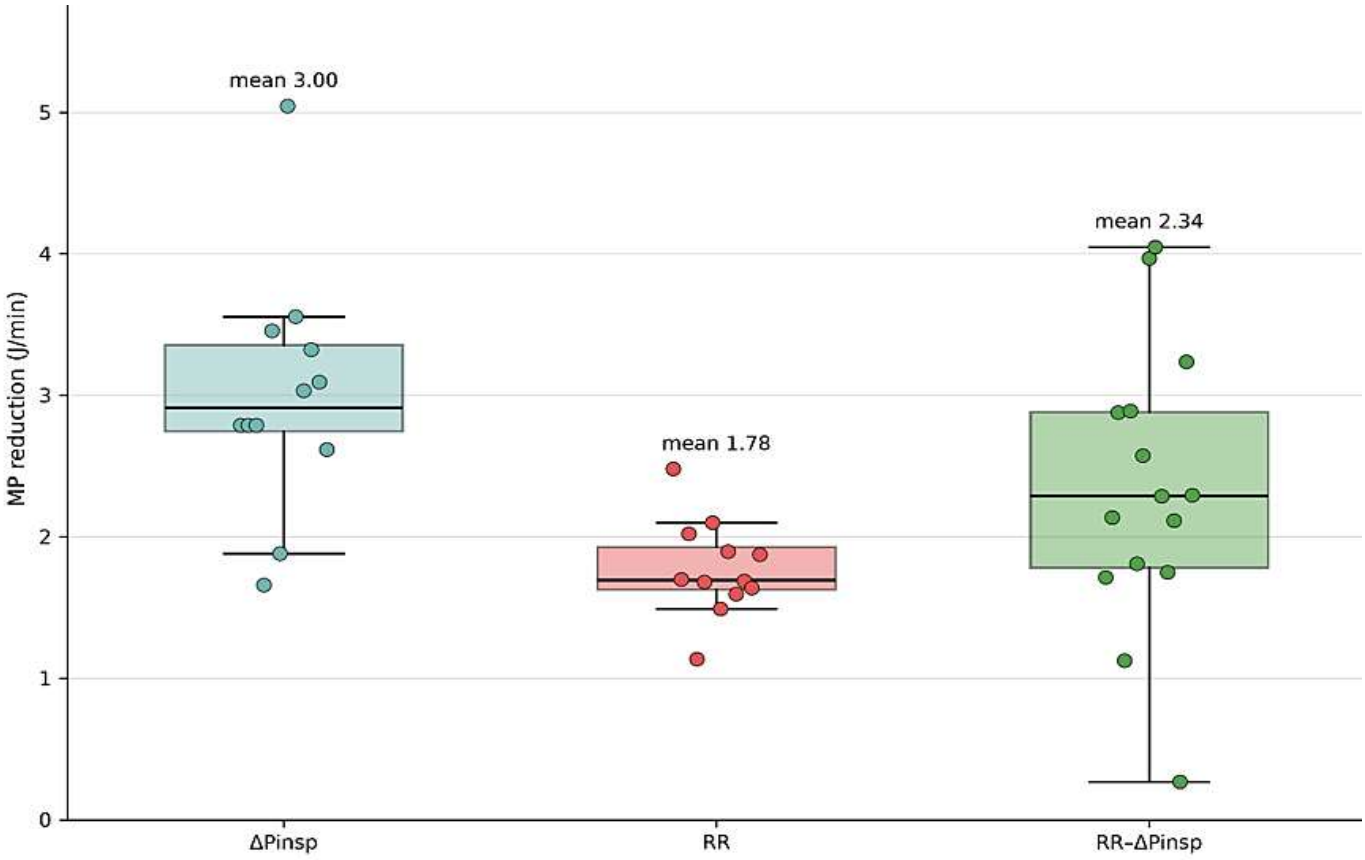


Figure 1: Reduction in mechanical power according to intervention strategy. The greatest reduction was observed in the ΔPinsp group, followed by the RR–ΔPinsp group and the RR group.

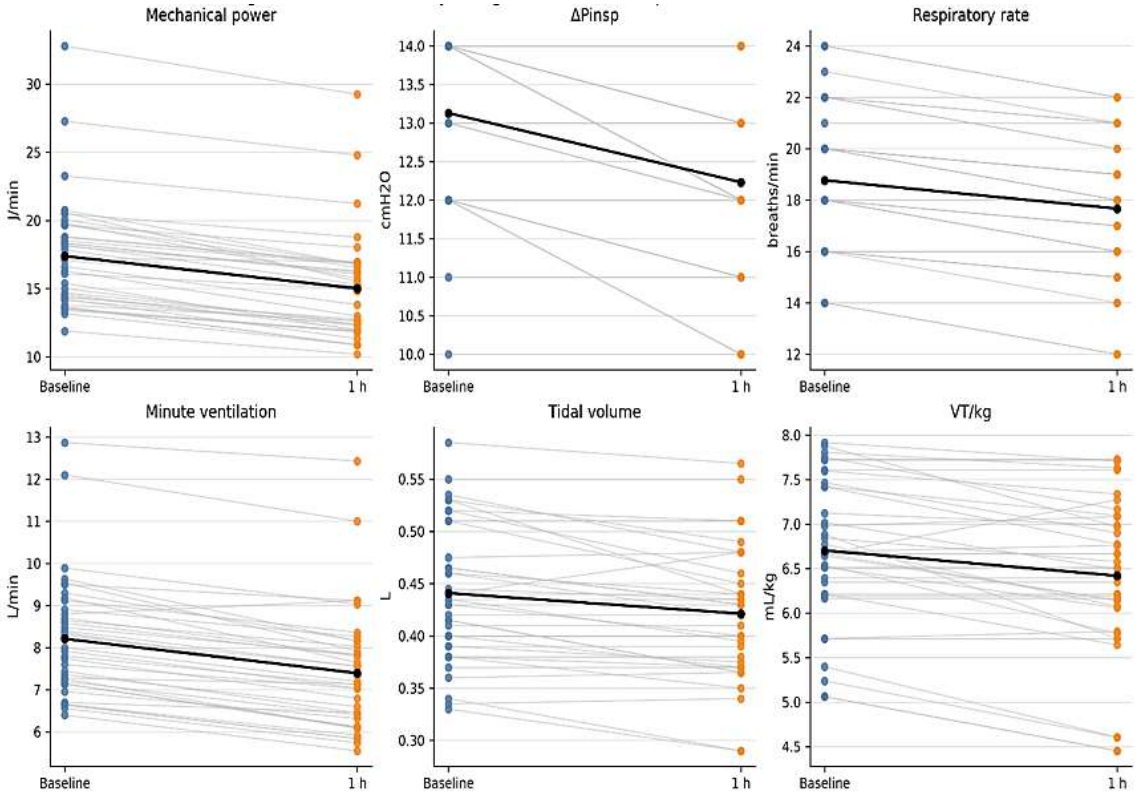


Figure 2: Paired changes in ventilatory variables and mechanical power from baseline to 1 h after reductive ventilatory adjustments in protective ventilatory observations. Panels show MP, ΔPinsp, RR, minute ventilation, VT, and VT/kg.

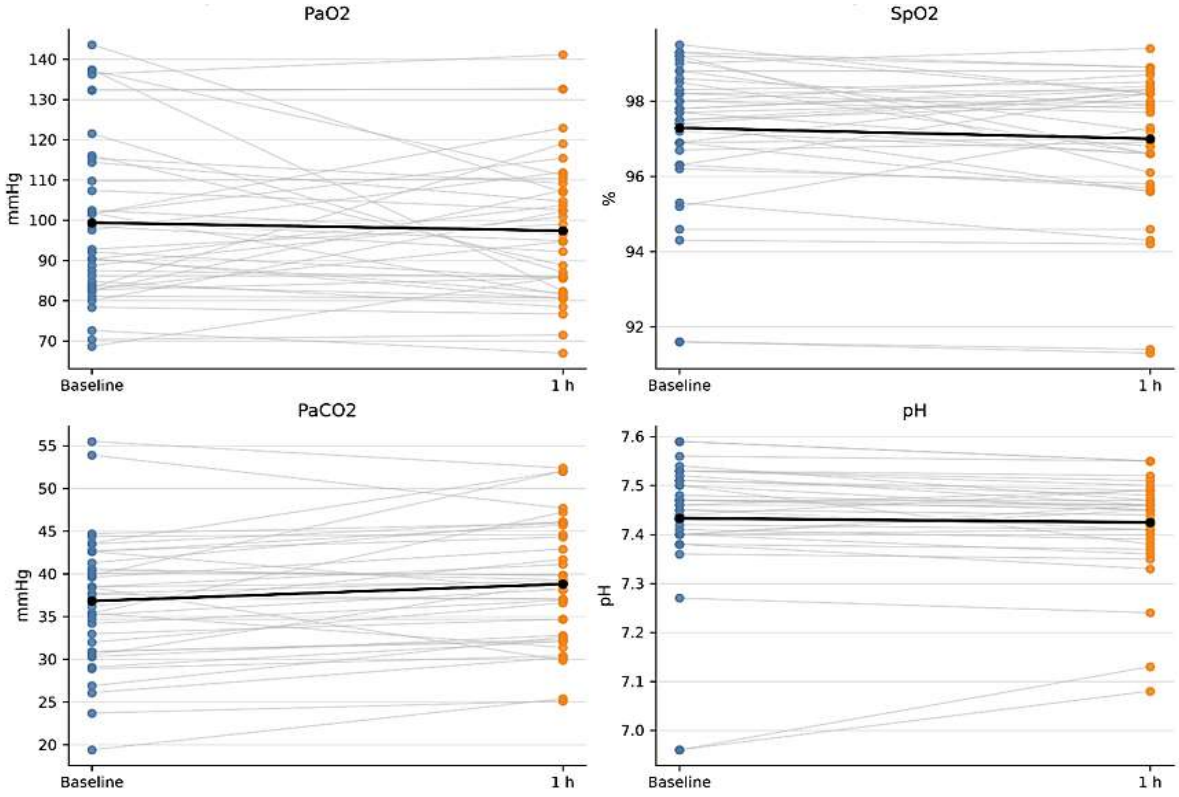


Figure 3: Arterial blood gas variables before and one hour after reductive ventilatory adjustments in protective ventilatory observations. PaO_2, SpO_2, and PaO_2/FiO_2 remained stable, whereas $PaCO_2$ increased modestly and pH decreased slightly while remaining within the physiological range.

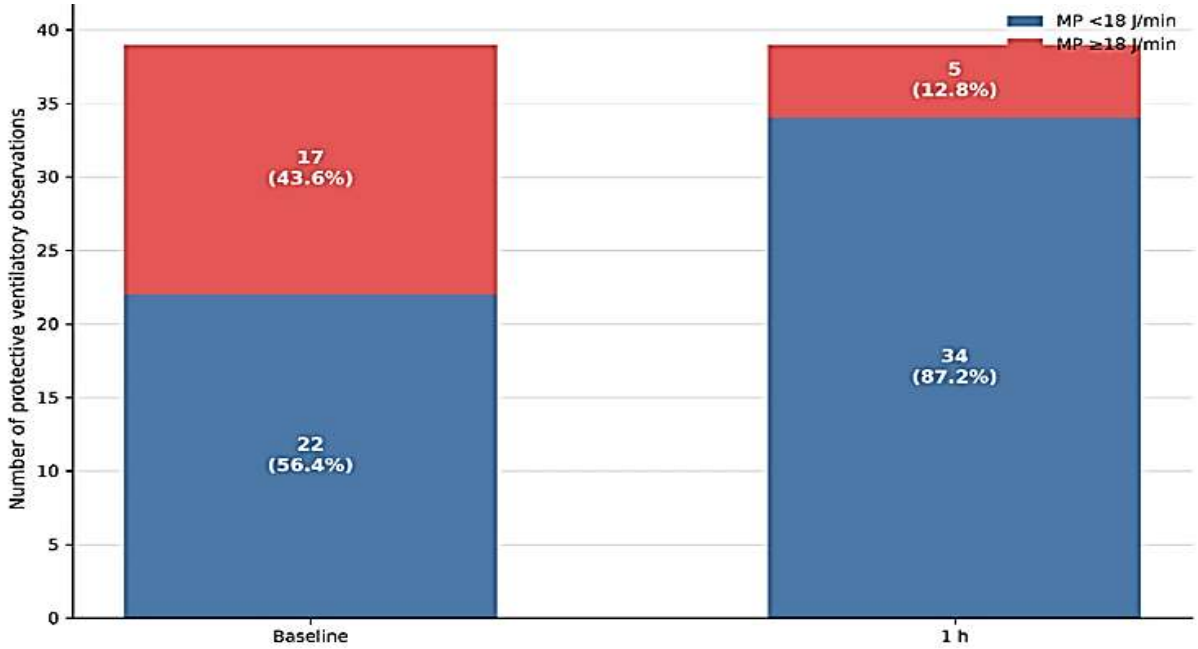


Figure 4: Distribution of protective ventilatory observations according to mechanical power category at baseline and after 1 h. The proportion of observations with MP < 18 J/min increased after the ventilatory adjustments, whereas the proportion with MP ≥ 18 J/min decreased.

Discussion

Elevated mechanical power (MP) has been associated with higher SpO_2 and PaO_2, and lower $PaCO_2$. [17] A recent trial showed that targeting SpO_2 at 90%, 94%, or 98% did not affect duration of mechanical ventilation or adverse events such as stroke or pneumothorax. [18] Current evidence recommends maintaining PaO_2 between 70 and 110 mmHg [19] and $PaCO_2$ between 35 and 50 mmHg. [20] In our study, mean values remained within these ranges throughout the observation period, with SpO_2 ranging from 96.9% to 97.3%, PaO_2 from 94.9 to 102.8 mmHg, and $PaCO_2$ from 35.9 to 39.6 mmHg.

Because the simplified Becher equation incorporates respiratory rate, tidal volume, PEEP, and inspiratory pressure variation as direct components of mechanical power, reductions in respiratory rate or inspiratory pressure variation are mathematically expected to reduce calculated MP. Therefore, the reduction in MP itself should not be interpreted as an independent physiological effect of the interventions. Rather, the clinically relevant finding of the present study is that small ventilatory adjustments were feasible and were accompanied by preserved oxygenation and arterial pH, despite a modest increase in $PaCO_2$ and without observed clinical instability. Accordingly, our findings should be interpreted as demonstrating the feasibility and short-term physiological tolerance of modest ventilatory adjustments that lower calculated mechanical power, rather than as evidence that reducing calculated mechanical power per se improves clinical outcomes.

We calculated MP using the simplified formula proposed by Becher et al. [16] which has been validated in PCV and applied in different clinical contexts. [21] Protective ventilation may be understood as the strategy that achieves the lowest MP compatible with adequate gas exchange. [6] However, the relative contribution of its individual components: tidal volume, pressures, and respiratory rate remain incompletely defined. [14] In the present study, reductions in ΔPinsp, RR, and RR–ΔPinsp were tested, each exerting distinct effects on stress, strain, and frequency, thereby influencing MP.

The Becher formula estimates MP from respiratory rate, tidal volume, PEEP, and inspiratory pressure variation, scaled by 0.098 to yield joules per minute. [16] Under the decelerating flow pattern of PCV, energy delivery is greatest early in inspiration and decreases as flow approaches zero, and peak inspiratory pressure may approximate plateau pressure under passive conditions. [16] Accordingly, no inspiratory pause was applied because ventilation was pressure-controlled rather than volume-controlled. In addition, no neuromuscular blocking agents were administered. Given the close correspondence between pressures in passive PCV, ΔPinsp was operationally considered the lung-inflating pressure responsible for generating tidal volume.

Burn patients may exhibit particularly high MP values, with reported means around 22.83 J/min. [7] MP values above 17 J/min have also been associated with increased mortality, suggesting that reducing MP may represent a relevant therapeutic target. [8] In the present study, despite fulfilling conventional protective ventilation criteria, 17 of the 39 ventilatory observations (43.6%) exhibited MP values ≥18 J/min at baseline. This finding indicates that compliance with accepted tidal-volume and pressure thresholds does not necessarily ensure a low mechanical power profile.

Following the reductive interventions, only 5 of the 39 observations (12.8%) remained above this threshold, whereas 12 of the 17 observations (70.6%) with initially elevated MP migrated to values below 18 J/min. These findings suggest that even modest reductions in ventilatory load may substantially modify the distribution of mechanical power and facilitate migration toward MP values that have previously been associated with more favorable prognostic profiles. Levels between 24.42 and 30.01 J/min have been associated with mortality, whereas levels between 17.42 and 22.31 J/min have been associated with survival in burn patients. [7] Thresholds differentiating survivors and non-survivors in ARDS have been reported at 24.7 versus 18.5 J/min in mild, 25.7 versus 21.3 J/min in moderate, and 28.7 versus 23.5 J/min in severe ARDS. [22]

In burn patients receiving PCV, higher ΔPinsp and RR and lower VT were associated with mortality, emphasizing the importance of both driving pressure and frequency of stress application. [7] Respiratory rates above 20 cycles/min have been linked to high-energy MP (> 17 J/min), compared with values around 17 cycles/min. [5] These observations informed our analysis of ΔPinsp, RR, and their combination. An exploratory analysis of ventilatory observations meeting conventional protective criteria suggested higher respiratory rates among observations with elevated mechanical power. Ventilations with MP < 18 J/min showed a mean respiratory rate of approximately 17.5 breaths/min, whereas those with MP ≥ 18 J/min showed a mean respiratory rate of approximately 20.4 breaths/min. Although the present study was not designed to define a respiratory-rate threshold for protective ventilation, this finding is consistent with the concept that the number of high-energy respiratory cycles delivered over time may contribute to ventilator-induced lung injury. [5] It also supports the physiological rationale underlying mechanical power, in which respiratory frequency directly influences cumulative energy transfer to the respiratory system. Pressures and respiratory rate have been identified as major determinants of MP, and RR reduction has been proposed as a key strategy to reduce MP. [14] However, in ARDS, maintaining low MP may require combining low tidal volume with higher RR. [23] Others have argued that only the elastic component of MP should be minimized, independently of elastance or resistance. [13]

The pressure and volume components of MP exhibit nonlinear behavior, whereas frequency contributes linearly. [16] Consistent with this concept, reducing ΔPinsp produced the greatest decrease in MP in our study, followed by the combined RR–ΔPinsp strategy and RR reduction alone. Although the RR group showed the smallest MP reduction, the decrease remained statistically significant. Comparisons among intervention strategies should be interpreted cautiously because some patients contributed more than one ventilatory observation to the analytical sample. Overall, our findings reinforce that compliance with conventional protective ventilatory parameters does not necessarily ensure a low mechanical power profile and support further investigation into energetic targets during protective ventilation.

Overall, our findings reinforce that compliance with conventional protective ventilatory parameters does not necessarily ensure a low mechanical power profile and support further investigation into energetic targets during protective ventilation. Finally, this was a single center study with randomization of the sequence of ventilatory interventions.

Among its limitations are the small sample size resulting from the selection of PCV observations that met protective criteria and the short interval between intervention and outcome assessment (1 hour). Consequently, the durability of MP reduction over time could not be determined. Another limitation is that the study evaluated a physiological endpoint rather than patient-centered clinical outcomes. Although MP values ≥18 J/min have been associated with worse prognosis in previous studies, the present investigation was not designed to determine whether migration below this threshold translates into reductions in ventilator-induced lung injury, duration of mechanical ventilation, ICU length of stay, or mortality. In addition, the threshold of 18 J/min was used as a prognostically relevant reference based on previous literature, but the optimal MP target for severely burned patients remains uncertain and may differ across clinical populations.

No similar prospective interventional studies were identified in the literature, as most available evidence derives from retrospective analyses or experimental models. Future multicenter, randomized studies including longer observation periods and clinical outcome measures are needed to further elucidate the role of small MP reductive ventilatory adjustments in burn patients.

Conclusion

A substantial proportion of ventilatory observations meeting conventional protective tidal-volume and pressure criteria still exhibited elevated mechanical power, indicating that compliance with accepted protective ventilatory parameters does not necessarily ensure a low mechanical power profile. In severely burned patients undergoing protective pressure-controlled ventilation, small ventilatory adjustments were associated with significant reductions in calculated mechanical power and promoted migration toward MP values below 18 J/min without causing asynchrony, desaturation, or clinically relevant acidosis during the observation period. All three reductive strategies were effective, although the greatest reduction in calculated mechanical power was observed after decreases in inspiratory pressure variation (ΔPinsp). These findings support the feasibility and short-term physiological tolerance of modest ventilatory adjustments designed to reduce calculated mechanical power while preserving arterial blood gas and clinical stability.

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