



Comprehensive study of mechanical power in controlled mechanical ventilation: Prevalence of elevated mechanical power and component analysis

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KEYWORDS

Mechanical ventilation;
Mechanical power;
Strain;
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Abstract

Objective: To determine the prevalence of elevated mechanical power (MP) values (>17 J/min) used in routine clinical practice.

Design: Observational, descriptive, cross-sectional, analytical, multicenter, international study conducted on November 21, 2019, from 8:00 AM to 3:00 PM. NCT03936231.

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◇ The names of the Mechanical Power Day collaborators is provided in [Appendix](#).

Setting: One hundred thirty-three Critical Care Units.

Patients: Patients receiving invasive mechanical ventilation for any cause.

Interventions: None.

Main variables of interest: Mechanical power.

Results: A population of 372 patients was analyzed. PM was significantly higher in patients under pressure-controlled ventilation (PC) compared to volume-controlled ventilation (VC) (19.20 ± 8.44 J/min vs. 16.01 ± 6.88 J/min; $p < 0.001$), but the percentage of patients with $PM > 17$ J/min was not different (41% vs. 35%, respectively; $p = 0.382$). The best models according to AIC_{cw} expressing PM for patients in VC are described as follows: Surrogate Strain (Driving Pressure) + PEEP + Surrogate Strain Rate (PEEP/Flow Ratio) + Respiratory Rate. For patients in PC, it is defined as: Surrogate Strain (Expiratory Tidal Volume/PEEP) + PEEP + Surrogate Strain Rate (Surrogate Strain/Ti) + Respiratory Rate + Expiratory Tidal Volume + Ti.

Conclusions: A substantial proportion of mechanically ventilated patients may be at risk of experiencing elevated levels of mechanical power. Despite observed differences in mechanical power values between VC and PC ventilation, they did not result in a significant disparity in the prevalence of high mechanical power values.

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PALABRAS CLAVE

Ventilación mecánica;
Mechanical power;
Strain;
Strain rate

Estudio Integral de la Potencia Mecánica en la Ventilación Mecánica Controlada: Prevalencia de Potencia Mecánica Elevada y Análisis de Componentes

Resumen

Objetivo: Determinar la prevalencia de valores elevados de potencia mecánica (PM) (>17 J/min) utilizados en la práctica clínica habitual.

Diseño: estudio observacional, descriptivo de corte transversal, analítico, multicéntrico e internacional, realizado el 21 de noviembre de 2019 en horario de 8 a 15 horas. NCT03936231.

Ámbito: Ciento treinta y tres Unidad de Cuidados Críticos.

Pacientes: pacientes que recibirán ventilación mecánica por cualquier causa.

Intervenciones: ninguna

VARIABLES DE INTERÉS PRINCIPALES: Potencia mecánica.

Resultados: se analizaron 372 enfermos. La PM fue significativamente mayor en pacientes en ventilación controlada por presión (PC) que en ventilación controlada por volumen (VC) ($19,20 + 8,44$ J/min frente a $16,01 + 6,88$ J/min; $p < 0,001$), pero el porcentaje de pacientes con $PM > 17$ J/min no fue diferente (41% frente a 35% respectivamente; $p = 0,382$). Los mejores modelos según AIC_{cw} que expresan la PM para los enfermos en VC se describe como: Strain subrogante (Presión de conducción) + PEEP + Strain Rate subrogante (PEEP/cociente de flujo) + Frecuencia respiratoria. Para los enfermos en PC se define como: Strain subrogante (Volumen tidal expiratorio/PEEP) + PEEP + Strain Rate subrogante (Strain subrogante/Ti) + Frecuencia respiratoria + Expiratory Tidal Volumen + Ti.

Conclusiones: Gran parte de los pacientes en ventilación mecánica en condiciones de práctica clínica habitual reciben niveles de potencia mecánica peligrosos. A pesar de las diferencias observadas en los valores de potencia mecánica entre la ventilación VC y PC, este porcentaje de riesgo fue similar en PC y VC.

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Introduction

In terms of materials science, mechanical power refers to the ability of a material or component to withstand loads and transmit energy without undergoing deformation.¹ In mechanical ventilation, the term Mechanical Power (MP) defines the amount of energy delivered to the respiratory system (rather than the lung) in a minute (rather than 1 cycle).² Gattinoni proposed a formula for calculating MP obtained by geometric transformation of the pressure-

volume loop and based on the main ventilator parameters in volume-controlled (VC) mode: tidal volume (V_t), driving pressure (DP), flow, respiratory rate (RR), and positive end-expiratory pressure (PEEP).³ Several simplified versions of the same formula have been developed.⁴⁻⁶ Becher and van der Meijden have also proposed formulas for calculating MP in pressure control (PC).^{7,8}

Several prospective studies have linked MP to developing ventilator-induced lung injury⁹ (VILI), and even death¹⁰⁻¹² of mechanically ventilated patients.¹³ Recent clinical studies

have suggested that MP levels above 18–20 J/min are associated with an increased risk of death in patients undergoing mechanical ventilation.^{14,15}

The primary objective of this study is to determine the prevalence of elevated MP values (>17 J/min) utilized in routine clinical practice. This will be achieved through the application of established formulas described in existing literature, specifically focusing on patients undergoing controlled ventilation. As a secondary objective, the study aims to investigate the weight of distinct components of mechanical power in both volume-controlled (VCV) and pressure-controlled (PCV) ventilation modes.

Methods

This observational, descriptive, cross-sectional, analytical, multi-centre, and international study was conducted on November 21st, 2019, from 8 a.m. to 3 pm (Supplementary material 1). Eligible patients were all those who on November 21, 2019, during the specified time, met the inclusion criteria: being admitted to a critical care area and receiving mechanical ventilation for any cause.

Exclusion criteria included patients admitted to critical care areas who were not receiving invasive mechanical ventilation at the time during the time specified, receiving invasive mechanical ventilation for less than 6 h, tracheotomized patients, patients ventilated with APRV (Airway Pressure Release Ventilation) y BiLEVEL (BiLevel Positive Airway Pressure) modes, non-invasive ventilation, selective ventilation, ventilation through nasotracheal intubation, and patients undergoing extracorporeal circulatory and respiratory support (ECMO-VV, ECMO-VA, and external carbon dioxide removal devices).

Clinical-demographic patient variables, variables related to mechanical ventilation, and analytical variables were recorded (Supplementary material 1).

The formula used to calculate MP in VC modes was the simplified formula proposed by Gattinoni et al.: $MP = 0.098 \times Vt \times RR \times PIP - [(Pplat - PEEP)/2]$. In the PC mode, the simplified formula proposed by Becher et al. was: $MP = 0.098 \times Vt \times RR \times (\Delta P_{ins} + PEEP)$. Based on recent publications, a threshold of high mechanical power was established at 17 J/min.^{14–16}

The study received approval from the Clinical Research Ethics Committee of Marqués de Valdecilla University Hospital, Santander, Spain. Informed consent was required. Subsequently, the necessary documentation was provided to each participating centre to obtain approval from their respective Clinical Research Ethics Committees, ensuring that all participating centres had the necessary approvals before starting the study.

The study was registered in the ClinicalTrials.gov database: NCT03936231.

Statistical analysis

A descriptive analysis of the sample was conducted. Results are presented as percentages for categorical variables, and as mean \pm standard deviation for continuous quantitative variables that followed a normal distribution (Kolmogorov-Smirnov).

Chi-square tests (Z test) were used for bivariate comparisons of categorical variables. For continuous variables with normal distribution, parametric tests (Welch) and their non-parametric equivalents were used for those that did not present a normal distribution.

Multivariable regression models were adjusted using the general linear model. The fit of each model was evaluated with the analysis of its studentized residuals and with normal QQ plots (supplementary material 2).¹⁷ The variables were selected for each model using the Akaike Information Criterion (AIC).

Theoretical concepts of material science indicate that the total stress which supports a viscoelastic body when deformed comes from the combination of the stress induced by the elastic behaviour and the stress induced by the viscous behaviour. To explore the respiratory variables most possibly related to the energy transmitted to the lungs, we carried out a process of algorithmic selection of regression models. For the adjustment of each model, the MP was used as the dependent variable and other different variables, depending on the ventilatory mode, were used as independent variables. In the selection of variables to develop the different models, we considered those that we believe best represented the viscoelastic component of the lung: Thus, in VC modes, in which the ventilator administers a fixed Vt, the independent variable that represented the elastic behaviour (surrogate of strain) was the DP and the one that represented the viscous behavior (surrogate of the strain rate) was the quotient Flow/PEEP. In PC modes, in which the ventilator tries to maintain the peak pressure (and subsequently the DP), the elastic behaviour of the parenchyma was represented with VTe/PEEP (as a surrogate of the strain; where VTe = volume tidal expiratory and PEEP: positive end-expiratory pressure), and the viscous behaviour of the parenchyma (surrogate of the strain rate) was represented with the surrogate of the strain divided by the inspiratory time ((VTe/PEEP)/Ti).¹⁸ For both modes PEEP was used as a surrogate for FRC.

For model comparison, information entropy measures of predictive accuracy were used, removing the cases with missing values at the start to fit the models to the same observations. This procedure reflects our interest in the models' relative performance, not their absolute information values. The Akaike's Information Criterion with the finite sample correction (AICc) was used to decide what the best model (of the candidates set, and in the sense of the expected Kullback-Leibler discrepancy) was. Then, for each model, the Akaike Weights^{19–21} and the Turing-Good Weights of Evidence (WOE) against every model relative to best model were computed.²²

For every model we computed the Likelihood of the Model (relative to the best one) given the data. Using every model AICc, the model with the minimum AICc was selected as the best one (in the expected Kullback-Leibler discrepancy sense). Then, the relative model likelihoods are normalized (i.e., divided by the sum of the likelihoods of all models) to obtain Akaike weights (the probability of every model in this set to be the best one). And in the last step, the WOE against (in decibans), relative to the best one, of all models were computed. Weight of Evidence in deciBans (WOE_{EgaBM}) refers to a numerical measure that utilizes deciBans to express the weight of evidence or the strength

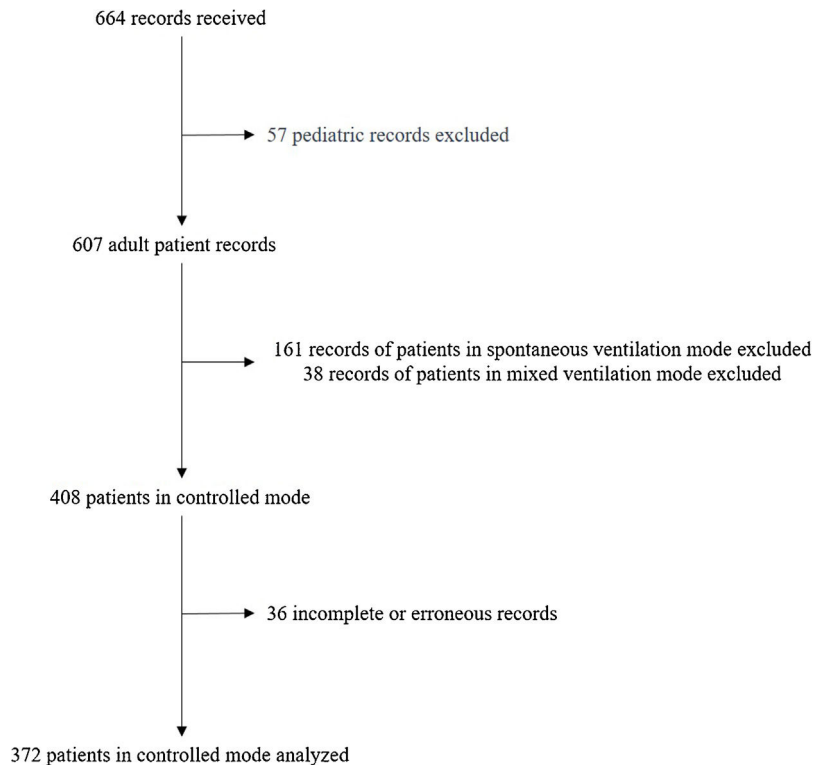


Figure 1 Flowchart of the study patients.

of an argument or set of data in a specific context. DeciBans are a relative unit used in various disciplines to quantify the relationship between two quantities, typically in terms of a difference in intensity or proportion.

All the additional analysis was done in R 3.6.3.

Results

A total of 664 critically ill patients from 133 intensive care units in 15 countries (Argentina, Brazil, Chile, China, Colombia, Ecuador, Spain, Honduras, Italy, Morocco, Mexico, Peru, Poland, Portugal, and Uruguay) were registered. Fifty-seven records were in pediatric patients, 196 in non-ventilated patients in controlled mode. Out of the 411 patients enrolled in controlled mode, 36 were excluded due to errors in the record or having incomplete records (Flowchart in Fig. 1).

Spain led the participation with 188 patients, followed by Ecuador with 54 and Argentina with 34. Colombia and Mexico also made significant contributions with 18 and 30 patients, respectively. Regarding the collaboration of ICUs, Spain led once again with 56 participating ICUs. Ecuador contributed 8 ICUs, while Argentina and Colombia provided 10 and 4 ICUs, respectively. Other countries like Italy, Chile, and Uruguay also participated in the study, standing out for the number of patients and ICUs involved (Fig. 2).

Of the 372 patients who were ventilated in controlled the main cause of orotracheal intubation was respiratory failure (33%; 95%CI: 12–63), followed by neurological impairment (31%; 95%CI: 11–60). Table 1 shows the main clinical-demographic, gasometric, and ventilatory variables analyzed in the total sample and the main differences between the compared groups are shown in Table 2.

The MP value in the patients under VC ventilation sample was 16 (7) J/min. For patients under PC ventilation, it was 19 (8) J/min. The difference in MP values was 3 J/min (95%CI: 1–5; $p < 0.001$) higher in the pressure-controlled group. In 38% of the analyzed subjects, the mechanical power value was greater than 17 J/min. The prevalence of high MP (number of patients with MP > 17 J/min / number of ventilated patients) was 38%. The values of the different variables of the MP formula in VC and PC are shown in Table 3; no significant difference was found in the prevalence of mechanical power exceeding 17 J/min between the two groups ($p = 0.382$).

The Table 4 presents the information for nine different models of patients under VC, numbered from 1 to 9. The best model is Model 7 (DP + PEEP + StrainRate.Subr + RR), as it has the lowest AICc value. The other models are compared to this best model. The Delta.AICc column shows the difference between the AICc value of each model and the AICc value of Model 7. The data less well support models with larger Delta.AICc values. The Akaike weight of evidence (AICcWt) column shows the weight of evidence in favor of each model, with higher values indicating stronger support. The weight of evidence in deciBans, calculated as $10 \cdot \log_{10}(\text{AICcWt}/\max(\text{AICcWt}))$ (WOEagaBM) column presents the weight of evidence in deciBans, with positive values indicating support for a given model and negative values indicating evidence against it.

Based on Table 5, the model with the highest WOEagaBM in patients under pressure control ventilation is Model 16 (Strain.subr.PC + eVT + PEEP + StrainRate.subr.PC + RR), with a WOEagaBM value of 0. Model 16 has a K value of 7, an AICc value of 1652, a Delta.AICc of 0, a ModelLik value of

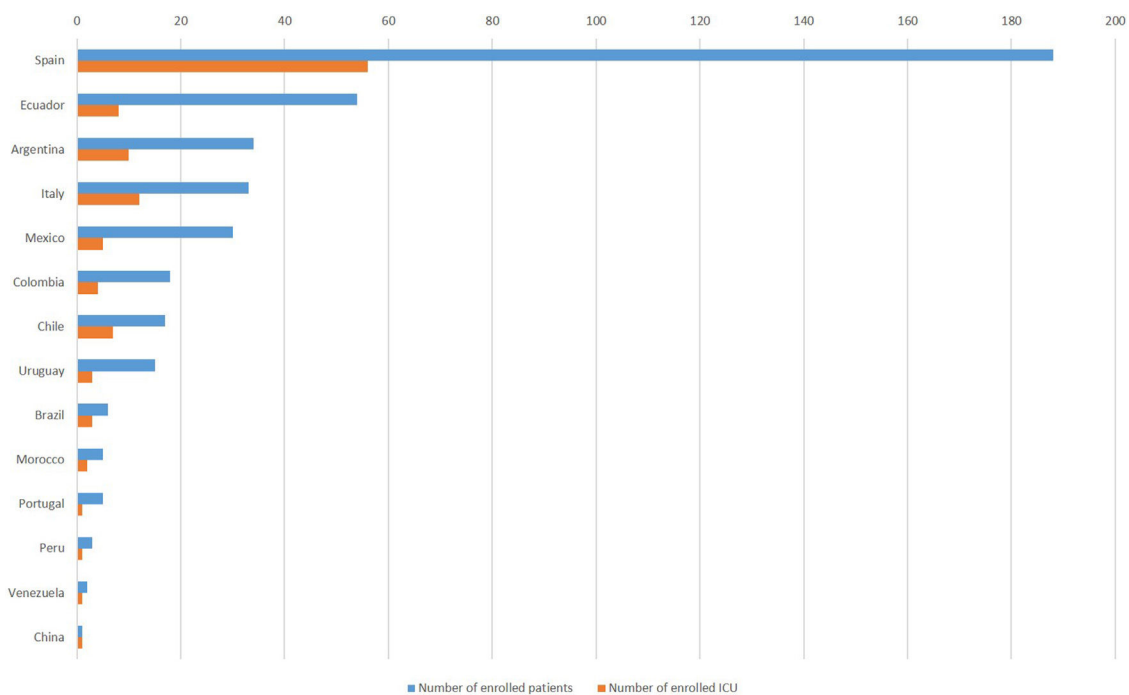


Figure 2 Distribution of patients and participation of ICUs in the study by country.

1e+0, an AICcWt value of 4.55e−1, an LL value of −819, and a Cum.Wt value of 0.455. The WOEgaBM values for the other models range from −975 to 0, with the models having higher values being less supported by the evidence. Therefore, Model 16 is the best supported model according to the weight of evidence analysis provided.

The algorithmic selection of models for each ventilatory mode is detailed in Supplementary Material 3 and 4.

Discussion

In the studied population, nearly two-fifths of subjects undergoing controlled mechanical ventilation exceeded the 17 J/min mechanical power threshold. Despite variations in mechanical power values between patients undergoing volume-controlled (VC) and pressure-controlled (PC) ventilation, no significant differences were observed in the prevalence of elevated mechanical power values.

The calculation of MP in mechanical ventilation is a complex topic that is still evolving. There are various formulas and methods for calculating MP, and often a large amount of detailed information and precise measurements are required for its application.^{23,24} There are numerous formulas and approaches to calculate MP in mechanical ventilation, and MP values can vary significantly. Simplified formulas – validated in previous studies – such as those proposed by Gattinoni et al.²⁵ and Becher et al.²⁶ are simple and practical approaches for MP calculation in VC and PC modes, respectively.

In this context, the specific MP values described in the literature for VC and PC are heterogeneous. Protti et al.²⁷ found that MP values in VC varied between 4 and 43 J/min, depending on the formula used for MP calculation. In a study by Amato et al.²⁸ MP values in VC varied between 10.6 and 16.4 J/min, depending on the formula used. Regarding MP

values in PC, a study by Costa et al.²⁹ found that MP values varied between 8.8 and 17.3 J/min, depending on the formula used. In another study by Bellani et al.,³⁰ MP values in PC varied between 8.7 and 13.2 J/min, depending on the formula used.

Our results reveal a significant difference in MP values between patients ventilated in VC and PC modes. This finding is consistent with the data obtained by Rietveld et al.,³¹ who found that in 46 mechanically ventilated patients without spontaneous breathing, the mean MP in VC was significantly lower than in PC (absolute difference of 1.26 J/min; SD 0.14 J/min; $p < 0.00001$). These results contradict the hypothesis that PC modes produce less MP than VC, based on the idea that the maximum peak pressure is equivalent to Pplat.³²

In general, the use of surrogates is a commonly employed strategy in modelling complex systems, including the human respiratory system. Surrogates represent lung tissue’s response to different ventilation modes, allowing for the evaluation of how mechanical ventilation affects lung tissue. In the literature, several studies have utilized surrogates for respiratory system evaluation. For example, in a 2016 study, Bellani et al.³³ used strain and strain rate surrogates to evaluate the relationship between MP and clinical outcomes in acute respiratory distress syndrome (ARDS) patients treated with mechanical ventilation. In another 2018 study, Sinderby et al.³⁴ used volume and pressure surrogates to evaluate different ventilation modes in patients with acute respiratory failure.

The correlation between PEEP and FRC in the context of mechanical ventilation is grounded in the principles of alveolar structure and lung elastic properties. The application of PEEP in mechanical ventilation involves maintaining a constant positive pressure in the airways, with effects on maintaining alveolar patency. Consequently, this resem-

Table 1 Characteristics of the cohort of 372 patients ventilated in controlled mode.

	Values	
Demographic variables		
Age (years), mean (SD)	58.18	(17.96)
Height (cm), mean (SD)	166.51	(8.94)
Weight (Kg), mean (SD)	75.71	(16.75)
Sex	237	(64.1%)
Men, <i>n</i> (%)		
Personal history		
Smoker, <i>n</i> (%)	98	(26.3%)
COPD, <i>n</i> (%)	46	(12.4%)
COPD GOLD c–d, <i>n</i> (%)	11	(3%)
Asthma, <i>n</i> (%)	8	(2.2%)
History of lung neoplasia, <i>n</i> (%)	6	(1.6%)
Previous radiotherapy, <i>n</i> (%)	5	(1.3%)
Lobectomy, <i>n</i> (%)	8	(2.2%)
Reason for respiratory admission	112	(30.1%)
Cause of intubation		
Weaning failure, <i>n</i> (%)	1	(0.3%)
Hemodynamic instability, <i>n</i> (%)	51	(13.7%)
Respiratory failure, <i>n</i> (%)	126	(33.9%)
Neurological disorder, <i>n</i> (%)	127	(34.1%)
Other, <i>n</i> (%)	1	(0.3%)
Presence of ARDS (The Berlin classification) ^a		
No ARDS, <i>n</i> (%)	301	(80.9%)
Mild ARDS, <i>n</i> (%)	21	(5.6%)
Moderate ARDS, <i>n</i> (%)	23	(6.2%)
Severe ARDS, <i>n</i> (%)	22	(5.9%)
Continuous iv sedation, <i>n</i> (%)	296	(79.6%)
Continuous iv analgesia, <i>n</i> (%)	322	(86.6%)
Continuous iv muscle relaxation, <i>n</i> (%)	36	(9.7%)
Gasometric data		
pH, mean (SD)	7.39	(0.95)
PO ₂ (mmHg), mean (SD)	98.68	(51.46)
PCO ₂ (mmHg), mean (SD)	39.6	(8.42)
HCO ₃ (mmol/L), mean (SD)	23.82	(5.28)
EB (mEq/L), mean (SD)	–0.63	(6.58)
Lactate (mmol/L), mean (SD)	2.98	(4.94)

COPD: Chronic Obstructive Pulmonary Disease; ARDS: Acute Respiratory Distress Syndrome.

^a The Berlin classification: Clinical criteria of acute lung injury within one week of a known clinical insult or new or worsening respiratory symptoms and bilateral opacities on chest imaging. Mild ARDS: PaO₂/FiO₂ (arterial oxygen tension/fraction of inspired oxygen) ratio between 200 and 300 mm Hg on PEEP ≥ 5 cm H₂O or CPAP; Moderate ARDS: PaO₂/FiO₂ ratio between 100 and 200 mm Hg on PEEP ≥ 5 cm H₂O or CPAP; Severe ARDS: PaO₂/FiO₂ ratio of ≤ 100 mm Hg on PEEP ≥ 5 cm H₂O or CPAP.

bles the condition of the FRC (the amount of air that remains in the lungs after a normal expiration, preventing collapse of smaller alveoli and providing a foundation of air for the next inspiration). Since applying PEEP during mechanical ventilation has a similar effect to maintaining the FRC, it can be postulated that PEEP could serve as an indirect indicator of FRC in patients under ventilatory support. However, it is important to recognize that PEEP and FRC are not equivalent, and there are physiological and clinical differences between them. PEEP can influence alveolar distension and transpulmonary pressures, whereas FRC is related to functional reserve and preventing alveolar collapse.³⁵

The presence of strain and strain rate surrogates in the AIC-established models of MP in our results may be related to

the viscoelastic behaviour of the lung.³⁶ Lung tissue has viscoelastic behaviour, which means it can deform and recover its original shape in response to applied forces. Still, the deformation and recovery may have different speeds and characteristics depending on the viscoelastic properties of the tissue.³⁷ In a study by Gattinoni et al., it was demonstrated that using Driving Pressure (as a surrogate for strain) and Flow/PEEP (as a surrogate for strain rate) in patients on controlled ventilation was associated with a reduction in mortality compared to using plateau pressure as an adjustment parameter.³⁸ A study by Talmor et al. found that using strain and strain rate surrogates for adjusting mechanical ventilation parameters in patients with ALI was associated with improved arterial oxygenation and reduced mechanical ventilation time.³⁹

Table 2 Main differences between patients ventilated under pressure control and patients ventilated under volume control.

	Pressure Control <i>n</i> = 174	Volumen Control <i>n</i> = 198	<i>p</i>
Demographic variables			
Age (years), mean (SD)	58 (18)	58 (17)	0.879
Height (cm), mean (SD)	165 (9)	167 (9)	0.022
Weight (Kg), mean (SD)	74 (17)	76 (16)	0.186
Sex	103 (59%)	134 (68%)	0.06
Men, <i>n</i> (%)			
Personal history			
Smoker, <i>n</i> (%)	35 (20%)	63 (32%)	0.007
COPD, <i>n</i> (%)	14 (8%)	32 (16%)	0.021
COPD GOLD c-d, <i>n</i> (%)	3 (1.8%)	8 (4.1%)	0.219
Asthma, <i>n</i> (%)	6 (3.5%)	2 (1%)	0.110
History of lung neoplasia, <i>n</i> (%)	2 (1.1%)	4 (2%)	0.509
Previous radiotherapy, <i>n</i> (%)	3 (1.78%)	2 (1%)	0.532
Lobectomy, <i>n</i> (%)	3 (1.8%)	5 (2.5%)	0.639
Reason for respiratory admission	49 (29%)	63 (32%)	0.449
Cause of intubation	0 (0%)	1 (0.5%)	0.431
Weaning failure, <i>n</i> (%)	23 (13%)	28 (14%)	
Hemodynamic instability, <i>n</i> (%)	56 (32%)	70 (35%)	
Respiratory failure, <i>n</i> (%)	67 (39%)	60 (30%)	
Neurological disorder, <i>n</i> (%)	26 (15%)	38 (19%)	
Other, <i>n</i> (%)	0 (0%)	1 (0.5%)	
Presence of ARDS (The Berlin classification)*			0.684
No ARDS, <i>n</i> (%)	141 (82%)	160 (82%)	
Mild ARDS, <i>n</i> (%)	11 (6.3%)	10 (5%)	
Moderate ARDS, <i>n</i> (%)	12 (7%)	11 (5.5%)	
Severe ARDS, <i>n</i> (%)	8 (4.5%)	14 (7%)	
Continuous iv sedation, <i>n</i> (%)	126 (72%)	170 (86%)	0.001
Continuous iv analgesia, <i>n</i> (%)	143 (82%)	179 (90%)	0.064
Continuous iv muscle relaxation, <i>n</i> (%)	8 (4.5%)	28 (14%)	0.006
Gasometric data			
pH, mean (SD)	7.39 (0.08)	7.38 (0.09)	0.384
PO ₂ (mmHg), mean (SD)	107 (48)	109 (46)	0.619
PCO ₂ (mmHg), mean (SD)	36 (11)	37 (14)	0.433
HCO ₃ (mmol/L), mean (SD)	22 (7)	22 (8)	0.950
EB (mEq/L), mean (SD)	-0.8 (5.5)	-0.14 (6.3)	0.276
Lactate (mmol/L), mean (SD)	2.6 (5)	2.3 (4)	0.537

COPD: Chronic Obstructive Pulmonary Disease; ARDS: Acute Respiratory Distress Syndrome. *The Berlin classification: Clinical criteria of acute lung injury within one week of a known clinical insult or new or worsening respiratory symptoms and bilateral opacities on chest imaging. Mild ARDS: PaO₂/FiO₂ (arterial oxygen tension/fraction of inspired oxygen) ratio between 200 and 300 mm Hg on PEEP ≥ 5 cm H₂O or CPAP; Moderate ARDS: PaO₂/FiO₂ ratio between 100 and 200 mm Hg on PEEP ≥ 5 cm H₂O or CPAP; Severe ARDS: PaO₂/FiO₂ ratio of ≤100 mm Hg on PEEP ≥ 5 cm H₂O or CPAP.

We consider that the main strengths of the study encompass its multicenter and international design, the inclusion of a diverse sample of critically ill patients, the utilization of rigorous statistical methodologies, and the incorporation of sophisticated analyses and measures for model selection and comparison. Furthermore, the study’s transparency and detailed presentation of the employed procedures contribute to its robustness.

As for the weaknesses of the study, first: the analysis of variables with significance which are included in the calculi for the mechanical power has potential statistical collinearity, as a circular argument. Second: it is an observational study, which means that a controlled and randomized intervention was not performed. Consequently,

causal relationships between mechanical ventilation modes and measured outcomes cannot be established. Additionally, although multivariable regression models were adjusted using the AIC, and the fit of each model was evaluated with the analysis of their standardized residuals and with QQ plots for normality, it is possible that other fitting models could have been used, which could affect the results. In other hand, the patient sample per hospital and the single-day period may raise concerns regarding the representativeness and robustness of the results.

With the presented data, we can conclude that a substantial portion of patients undergoing mechanical ventilation may be at risk of experiencing elevated mechanical power levels, potentially contributing to complications associated

Table 3 Main differences in mechanical power values and ventilatory parameters between patients undergoing pressure-controlled ventilation and patients undergoing volume-controlled ventilation.

Variables	Pressure control n = 174		Volumen control n = 198		p
Mechanical power, J/min	19.20	(8.44)	16.01	(6.88)	<0.001
Mechanical power >17 J/min, n (%)	72	(41%)	69	(35%)	0.382
PEEP, cmH2O	7.03	(2.07)	7.29	(2.68)	0.279
Plateau pressure, cmH2O	18.94	(4.84)	18.65	(4.77)	0.589
PIP, cmH2O	23.53	(6.19)	24.98	(6.69)	0.034
Compliance, mL/cmH2O	49.52	(36.91)	44.64	(21.52)	0.157
Raw, cmH2O/(L/seg)	18.09	(47.61)	11.85	(5.83)	0.162
Tidal Volumen set, mL	464.95	(74.52)	458.55 ^a	(64.78) ^a	0.395
Vte, mL	464.69	(91.18)	454.26	(83.19)	0.294
Respiratory rate	17.23	(3.38)	17.86	(4.37)	0.116
FiO2	41.90	(15.84)	44.84	(16.87)	0.085
ETCO2, mmHg	36.32	(12.39)	34.32	(8.53)	0.381
P/F	282.56	(164.01)	267.02	(38.57)	0.325

Variable values are reported as mean and standard deviation (in parentheses).

PEEP: positive end-expiratory pressure; PIP: Peak inspiratory pressure; Raw: Airway resistance; Vte: Tidal Volumen expiratory measured; FiO2: Fraction of Inspired Oxygen; ETCO2: End-tidal CO2; P/F: PaO2/FiO2 ratio.

^a Obtained, no set.

Table 4 Model weights of evidence against the best one (in deciBans), for patients under volume control.

Modnames	K	AICc	Delta_AICc	ModelLik	AICcWt	LL	Cum.Wt	WOEagaBM
Model 7	6	1083	0	1e + 0	6.67e - 1	-535	0.667	0
Model 9	7	1085	2.16	3.40e - 1	2.27e - 1	-535	0.894	-4.69
Model 5	6	1086	3.68	1.59e - 1	1.06e - 1	-537	1	-7.99
Model 3	5	1122	39.5	2.65e - 9	1.77e - 9	-556	1	-85.8
Model 6	5	1153	70.5	5e - 16	3.34e - 16	-571	1	-153
Model 8	6	1155	72	2.28e - 16	1.52e - 16	-571	1	-156
Model 4	5	1179	96.2	1.28e - 21	8.56e - 22	-584	1	-209
Model 2	4	1222	139	6.06e - 31	4.04e - 31	-607	1	-302
Model 1	3	1232	149	3.69e - 33	2.46e - 33	-613	1	-324

Modnames: Model names; K: Number of parameters in the model; AICc: Akaike Information Criterion corrected for small sample sizes; Delta_AICc: Difference between the AICc value of each model and the AICc value of the best model; ModelLik: Model likelihood; AICcWt: Akaike weight of evidence; LL: Log-likelihood; Cum.Wt: Cumulative weight of evidence; WOEagaBM: Weight of evidence in deciBans, calculated as $10 \cdot \log_{10}(AICcWt/\max(AICcWt))$.

with ventilator-induced lung injury. Despite the observed differences in mechanical power values between VC and PC ventilation, no statistically significant difference was found in the prevalence of mechanical power exceeding 17 J/min

between the two groups. This indicates that while mechanical power levels differed, they did not result in a significant disparity in the prevalence of elevated mechanical power values.

Table 5 Model weights of evidence against the best one (in deciBans), for patients under pressure control.

Modnames	K	AICc	Delta_AICc	ModelLik	AICcWt	LL	Cum.Wt	WOEagaBM
Model 16	7	1652	0	1e + 0	4.55e - 1	-819	0.455	0
Model 18	8	1653	1.16	5.59e - 1	2.54e - 1	-818	0.709	-2.53
Model 13	6	1654	1.78	4.10e - 1	1.86e - 1	-821	0.896	-3.87
Model 15	7	1655	2.95	2.29e - 1	1.04e - 1	-820	1	-6.40
Model 9	6	1694	42.2	6.94e - 10	3.16e - 10	-841	1	-91.6
Model 11	7	1695	43.3	4.05e - 10	1.84e - 10	-840	1	-93.9
Model 17	7	1766	114	1.47e - 25	6.69e - 26	-876	1	-248
Model 14	6	1777	125	7.94e - 28	3.61e - 28	-882	1	-271
Model 5	5	1780	128	1.67e - 28	7.60e - 29	-885	1	-278
Model 7	6	1781	129	1.00e-28	4.56e - 29	-884	1	-280

Table 5 (Continued)

Modnames	K	AICc	Delta_AICc	ModelLik	AICcWt	LL	Cum.Wt	WOEagaBM
Model 10	6	1781	129	9.98e – 29	4.54e – 29	–884	1	–280
Model 12	6	1787	135	4.40e – 30	2.00e – 30	–887	1	–294
Model 8	5	1799	147	1.24e – 32	5.65e – 33	–894	1	–319
Model 6	5	1861	209	3.73e – 46	1.70e – 46	–925	1	–454
Model 4	4	1879	227	4.75e – 50	2.16e – 50	–935	1	–493
Model 2	3	2033	381	2.16e – 83	9.84e – 84	–1013	1	–827
Model 3	3	2046	394	2.63e – 86	1.20e – 86	–1020	1	–856
Model 1	3	2101	449	3.29e – 98	1.50e – 98	–1047	1	–975

Modnames: Model names; K: Number of parameters in the model; AICc: Akaike Information Criterion corrected for small sample sizes; Delta_AICc: Difference between the AICc value of each model and the AICc value of the best model; ModelLik: Model likelihood; AICcWt: Akaike weight of evidence; LL: Log-likelihood; Cum.Wt: Cumulative weight of evidence; WOEagaBM: Weight of evidence in deciBans, calculated as $10^{\log_{10}(AICcWt/\max(AICcWt))}$.

Contribución de los autores

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Patricia Escudero-Acha: Initial study design, study promotion, data refinement.

Alberto Medina Villanueva: Initial study design, manuscript writing and translation correction.

Aurio Fajardo Campoverdi: Corrección de la versión final del manuscrito, Promoción del estudio, Análisis estadístico.

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Mechanical Power Day Group*: Variable collection.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:<https://doi.org/10.1016/j.medine.2023.11.004>.

References

- Callister WD, Rethwisch DG. Fundamentos de la Ciencia e Ingeniería de Materiales. sexta edición; 2014, <http://dx.doi.org/10.1177/0267659115587416>. Editorial Reverte.
- Cressoni M, Gotti M, Chiurazzi C, Massari D, Algieri I, Amini M, et al. Mechanical power and development of ventilator-induced lung injury. *Anesthesiology*. 2016;124:1100–8, <http://dx.doi.org/10.1097/ALN.0000000000001070>.

- Gattinoni L, Tonetti T, Cressoni M, Cadringer P, Herrmann P, Moerer O, et al. Ventilator-related causes of lung injury: the mechanical power. *Intensive Care Med*. 2016;42:1567–75, <http://dx.doi.org/10.1007/s00134-016-4505-2>.
- Giosa L, Busana M, Bonifazi M, Boscolo A, Pasetto A, Zanatta P, et al. Mechanical power at a glance: a simple surrogate for volume-controlled ventilation. *Intensive Care Med*. 2019;7:61, <http://dx.doi.org/10.1186/s40635-019-0276-8>.
- Aşar S, Acicbe Ö, Çukurova Z, Hergünel GO, Canan E, Çakar N. Bedside dynamic calculation of mechanical power: a validation study. *J Crit Care*. 2020;56:167–70, <http://dx.doi.org/10.1016/j.jcrrc.2019.11.020>.
- Chi Y, He H, Long Y. A simple method of mechanical power calculation: using mean airway pressure to replace plateau pressure. *J Clin Monit Comput*. 2021;35:1139–47, <http://dx.doi.org/10.1007/s10877-020-00575-y>.
- Becher T, van der Staay M, Schädler D, Frerichs I, Weiler N. Calculation of mechanical power for pressure-controlled ventilation. *Intensive Care Med*. 2019;45:1321–3, <http://dx.doi.org/10.1007/s00134-019-05685-9>.
- van der Meijden S, Molenaar M, Somhorst P, Schoe A. Pulmonary mechanics and mortality in mechanically ventilated patients without acute respiratory distress syndrome: a cohort study. *Shock*. 2018;49:311–6, <http://dx.doi.org/10.1097/SHK.0000000000000961>.
- Fuller BM, Page D, Stephens RJ, Roberts BW, Drewry AM, Ablordeppey E, et al. Pulmonary mechanics and mortality in mechanically ventilated patients without acute respiratory distress syndrome: a cohort study. *Shock*. 2018;49:311–6, <http://dx.doi.org/10.1097/SHK.0000000000000928>.
- Parhar KK, Zjadewicz K, Soo A, Sutton A, Zjadewicz M, Doig L, et al. Epidemiology, mechanical power, and 3-year outcomes in acute respiratory distress syndrome patients using standardized screening. An observational cohort study. *Ann Am Thorac Soc*. 2019;16:1263–72, <http://dx.doi.org/10.1513/AnnalsATS.201903-2300C>.
- Zhang Z, Zheng B, Liu N, Ge H, Hong Y. Mechanical power normalized to predicted body weight as a predictor of mortality in patients with acute respiratory distress syndrome. *Intensive Care Med*. 2019;45:856–64, <http://dx.doi.org/10.1007/s00134-019-05624-5>.
- Coppola S, Caccioppola A, Froio S, Formenti P, De Giorgis V, Galanti V, et al. Effect of mechanical power on intensive care mortality in ARDS patients. *Crit Care*. 2020;24:246, <http://dx.doi.org/10.1186/s13054-020-02963-2>.
- Martin U, Peter J, Bettina H, Wettstein M, Ferguson D, Fan E. Time-varying intensity of mechanical ventilation and mortality in patients with acute respiratory failure: a registry-based,

- prospective cohort study. *Lancet Respir Med.* 2020;8:905–13, [http://dx.doi.org/10.1016/S2213-2600\(20\)30325-8](http://dx.doi.org/10.1016/S2213-2600(20)30325-8).
14. Serpa N, Deliberato RO, Johnson AEW, Bos LD, Amorim P, Pereira SM, et al. Mechanical power of ventilation is associated with mortality in critically ill patients: an analysis of patients in two observational cohorts. *Intensive Care Med.* 2018;44:1914–22, <http://dx.doi.org/10.1007/s00134-018-5424-2>.
 15. Guérin C, Papazian L, Reignier J, Ayzac L, Loundou A, Forel JM. Investigators of the Acurasys and Proseva trials. Effect of driving pressure on mortality in ARDS patients during lung protective mechanical ventilation in two randomized controlled trials. *Crit Care.* 2016;20:384, <http://dx.doi.org/10.1186/s13054-016-1513-8>.
 16. González-Castro A, Modesto I Alapont V, Cuenca Fito E, Peñasco Y, Escudero Acha P, Huertas Martín C, et al. The Bayes factor in the analysis of mechanical power in patients with severe respiratory failure due to SARS-CoV-2. *Med Intensiva.* 2023;22, <http://dx.doi.org/10.1016/j.medin.2023.03.005>.
 17. Modesto I Alapont V, Medina-Villanueva A. Decibans: it is time to weigh the evidence about diagnostic accuracy. *Med Intensiva.* 2022;46:173–4, <http://dx.doi.org/10.1016/j.medin.2021.05.002>.
 18. Modesto I Alapont V, Aguar Carrascosa M, Medina Villanueva A. Stress, strain and mechanical power: is material science the answer to prevent ventilator induced lung injury? *Med Intensiva (Engl Ed).* 2019;43:165–75, <http://dx.doi.org/10.1016/j.medine.2018.06.004>.
 19. Wagenmakers EJ, Farrell S. AIC model selection using Akaike weights. *Psychon Bull Rev.* 2004;11:192–6.
 20. McElreath R. Overfitting, regularization and information criteria. In: McElreath, editor. *Bayesian rethinking: a Bayesian course with examples in R and Stan.* Chapman&Hall/CRC texts in statistical science series; 122. 1st ed. Boca Raton (FL). USA: Taylor&Francis Group; 2016.
 21. Claeskens G, Lid Hjort N. *Model selection and model averaging.* Cambridge Univ. Press; 2008.
 22. Gillies D. The turing-good weight of evidence function and popper's measure of the severity of a test. *Br J Philos Sci.* 1990;41:143–6 <http://www.jstor.org/stable/688010>
 23. 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;42:1360–73, <http://dx.doi.org/10.1007/s00134-016-4420-y>.
 24. Bellani G, Grasselli G, Teggie-Droghi M, Mauri T, Coppadoro A, Brochard L, et al. Do spontaneous and mechanical breathing have similar effects on average transpulmonary and alveolar pressure? A clinical crossover study. *Crit Care.* 2016;20:142, <http://dx.doi.org/10.1186/s13054-016-1325-5>.
 25. Gattinoni L, Tonetti T, Cressoni M. Ventilator-related causes of lung injury: the mechanical power. *Intensive Care Med.* 2016;42:1567–75, <http://dx.doi.org/10.1007/s00134-016-4505-2>.
 26. Chi Y, He HW, Long Y. Progress of mechanical power in the intensive care unit. *Chin Med J (Engl).* 2020;133:2197–204, <http://dx.doi.org/10.1097/CM9.0000000000001018>.
 27. Protti A, Andreis DT, Monti M, Santini A, Sparacino CC, Langer T, et al. Lung stress and strain during mechanical ventilation: any difference between statics and dynamics? *Crit Care Med.* 2013;41:1046–55, <http://dx.doi.org/10.1097/CCM.0b013e318274161c>.
 28. 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;372:747–55, <http://dx.doi.org/10.1056/NEJMs1410639>.
 29. Costa EL, Borges JB, Melo A, Suarez-Sipmann F, Toufen C Jr, Bohm SH, et al. Bedside estimation of recruitable alveolar collapse and hyperdistension by electrical impedance tomography. *Intensive Care Med.* 2009;35:1132–7, <http://dx.doi.org/10.1007/s00134-009-1447-8>.
 30. Betensley AD, Khalid I, Crawford J, Pensler RA, DiGiovine B. Patient comfort during pressure support and volume controlled-continuous mandatory ventilation. *Respir Care.* 2008;53:897–902.
 31. Rietveld PJ, Snoep JWM, Lamping M, van der Velde F, de Jonge E, van Westerloo DW, et al. Mechanical power differs between pressure-controlled ventilation and different volume-controlled ventilation modes. *Crit Care Explor.* 2022;4:e0741, <http://dx.doi.org/10.1097/CCE.0000000000000741>.
 32. Silva PL, Cruz FF, Fujisaki LC, Oliveira GP, Samary CS, Ornelas DS, et al. Hypervolemia induces and potentiates lung damage after recruitment maneuver in a model of sepsis-induced acute lung injury. *Intensive Care Med Exp.* 2019;7:38, <http://dx.doi.org/10.1186/s40635-019-0246-8>.
 33. Gama de Abreu M, Cuevas M, Spieth PM, Carvalho AR, Hietschold V, Stroszczyński C, et al. Regional lung aeration and ventilation during pressure support and biphasic positive airway pressure ventilation in experimental lung injury. *Crit Care.* 2010;14:R34, <http://dx.doi.org/10.1186/cc8912>.
 34. Sinderby C, Navalesi P, Beck J, Skrobik Y, Comtois N, Friberg S, et al. Neural control of mechanical ventilation in respiratory failure. *Nat Med.* 1999;5:1433–6, <http://dx.doi.org/10.1038/71012>.
 35. Kallet RH. Should PEEP titration be based on chest mechanics in patients with ARDS? *Respir Care.* 2016;61:876–90, <http://dx.doi.org/10.4187/respcare.04657>.
 36. Xie Y, Qian Y, Liu K, Liu S, Zheng H, Cao L, et al. Correlation analysis between mechanical power and lung ultrasound score and their evaluation of prognosis in patients with acute respiratory distress syndrome. *Zhonghua Wei Zhong Bing Ji Jiu Yi Xue.* 2019;31:704–8, <http://dx.doi.org/10.3760/cma.j.issn.2095-4352.2019.06.009>.
 37. Rausch SM, Haberthür D, Stampanoni M, Schittny JC, Wall WA. Local strain distribution in real three-dimensional alveolar geometries. *Ann Biomed Eng.* 2011;39:835–43, <http://dx.doi.org/10.1007/s10439-011-0355-5>.
 38. Gattinoni L, Marini JJ, Pesenti A, Quintel M, Mancebo J, Brochard L. The "baby lung" became an adult. *Intensive Care Med.* 2016;42:663–73, <http://dx.doi.org/10.1007/s00134-015-4200-7>.
 39. Talmor D, Sarge T, O'Donnell CR, Ritz R, Malhotra A, Lisbon A, et al. Esophageal and transpulmonary pressures in acute respiratory failure. *Crit Care Med.* 2006;34:1389–94, <http://dx.doi.org/10.1097/01.CCM.0000215515.16739.1E>.