Correlations of mechanical power and its components with age and its interference in the outcome of SARS-CoV-2 in subjects undergoing pressure-controlled ventilation

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Abstract

Introduction
SARS-CoV-2 may be associated with ARDS and the VILI. However, there are still doubts about the correlations and the interference of tidal energy in the outcomes. The objective of this study was to verify the correlations and interference of mechanical power and its components with age in the outcome in SARS-CoV-2 of subjects undergoing pressure-controlled ventilation (PCV).
Method
Longitudinal, prospective, observational, analytical, and quantitative study of the information collected on two parameters of the mechanical ventilator, to calculate the mechanical power by Becher formula in 163 subjects with SARS-CoV-2 and moderate ARDS between May 2021 to September 2021.
Results
Correlations were found between mechanical power and its components, except for compliance (P 0.234), elastance (P 0.515), resistance (P 0.570) and age (P 0.180). There was a significant impact on the outcome in the univariate analysis of age, as well as of mechanical power and its components, except for positive end expiratory pressure (PEEP) (P 0.874), minute ventilation (Ve) (P 0.437), resistive pressure (P_{Resist}) (P 0.410) and resistance (P 0.071). The multivariate analysis of mechanical power, plateau pressure ($P_{Plateau}$), tidal volume (VT), driving pressure (ΔP) and elastance, showed that only mechanical power correlated to death (P 0.04) and for each additional unit in J/minute there is a 6.2% increase in the odds of death (95% IC 0.3%; 12.4%).
Conclusion
There are correlations between mechanical power and its components, except for compliance, elastance, resistance, and age. There is interference in the outcome in the univariate analysis of age, as well as of mechanical power and its components, except PEEP, Ve, P_{Resist} and resistance, but the multivariate analysis showed that only mechanical power correlates with the outcome in SARS-CoV-2 undergoing PCV.

Keywords: SARS-CoV-2 infection; Mortality; Ventilation Induced Lung Injury; Acute Respiratory Distress Syndrome

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Introduction

The diagnosis of The Acute Respiratory Distress Syndrome (ARDS) caused by the Coronavirus 2019 (SARS-CoV-2) infection is made by the finding of radiological consolidations and bilateral air bronchogram with peripheral ground-glass opacity, associated with hypoxemia with a relationship between Partial Pressure of O_2 over inspired O_2 Fraction (P_aO_2/F_iO_2) below 300 mmHg and respiratory failure with pulmonary inflammation, which can progress to fibrosis when associated with mechanical ventilation. [1]

Ventilation-Induced Lung Injury (VILI) is defined as the injury generated by the application of mechanical forces and by the transfer of energy to the lung tissue with ARDS. [2] The complex conjunction of VILI and ARDS makes them indistinguishable and makes it impossible to determine the individual contribution to lethality. [3] Therefore, it is not possible to clinically disassociate VILI from the underlying process that causes ARDS. [4]

VILI depends on the vulnerability of lung tissue, [5] but its risks increase with the reduction of the ventilated lung area. [4] The injury produced by the energy flow is determined by the pressure gradient and the resulting alveolar deformity, Stress and Strain, respectively. [6] The understanding of the biophysical causes of VILI has redirected attention from the pattern of inflation related to tidal volume and pressures to exposing the alveolar-capillary membrane to harmful levels of energy and potency. [4]

Protective ventilation is considered when tidal volume is maintained between 4-8 ml/kg of predicted weight, plateau pressure is less than 30 cmH_2O, driving pressure is less than 15 cmH_2O [7] and PEEP is maintained between 8 and 14 cmH_2O. [8] However, doubts persist about the safer adjustments of mechanical ventilation components and parameters in ARDS to attenuate VILI. [4,5]

Thus, mechanical power emerges as a physiological concept that aims to simplify the analysis of mechanical ventilation, portraying it through the set of mechanical ventilation components, [2-9] as well as the injury produced by the energy originated in the pressure gradient that promotes alveolar deformation. [6] Although there is a tendency to replace the concepts based on tidal volume and pressures in the tidal cycle for the understanding of mechanical power as a generator of VILI, its predictive accuracy is still questionable. [10-11]

Mechanical power can be calculated using the geometric method, measuring the dynamic inspiratory area of airway pressure and volume curve during the respiratory cycle, or using equations. [12]

In volume-controlled ventilation (VCV) there is a linear increase in airway pressure during inspiration [13] and the inspiratory flow remains constant, while in pressure-controlled ventilation (PCV) the flow decelerates while the pressure in the airways remains constant [12] generating the variation of the inspiratory pressure (ΔP_{insp}) together with the resistance and the compliance of the respiratory system. [14] The different shapes of the pressure-volume curve under VCV and PCV imply that a single formula for calculating mechanical power could not accurately predict the energy transferred to the respiratory system in both ventilation models. [15]

Becker and colleagues [13] and Van der Meijden and colleagues [14] reported two formulas for measuring mechanical power in the PCV modality. However, the need for an inspiratory pause, resistances and the presence of an exponential flow decay, make these formulas impractical in practice, so Becher and colleagues proposed a simplified formula that is easy to use and with satisfactory accuracy to be used at the bedside, [12] even disregarding the time of increase in inspiratory pressure (T_{slope}), resistance and compliance, the simplified formula was restricted to tidal volume, respiratory rate, PEEP and ΔP_{insp} sufficient for most clinical situations. [13] (Figure 1) In essence, the formulas for calculating the mechanical power for PCV or VCV consider the tidal volume multiplied by the pressures, that is, the result of the inspiratory work by the set of static elastic, dynamic elastic and resistive components in $cmH_2O.L$, which when multiplied by the respiratory rate determines the value of the inspiratory energy in $cmH_2O.L/minute$. This value multiplied by the 0.098 converter factor shows the mechanical power in joules per minute. [15]

Comprehensive formula

$$MP = 0.098 \cdot RR \cdot V_t \cdot \left[PEEP + \Delta P_{insp} \cdot \left(1 - e^{\frac{-T_{insp}}{RC}} \right) \right]$$

Simplified formula

$$MP = 0.098 \cdot RR \cdot V_t \cdot \left[PEEP + \Delta P_{insp} \right]$$

Figure 1: ΔPinsp is the pressure (cmH2O) above PEEP during pressure-controlled ventilation, Tinsp is the inspiratory time (s), and C and R are the respiratory system compliance (ml/cmH2O) and resistances (cmH2Osl−1) respectively, 0.098 is a conversion factor from cmH2O l min−1 in J/min, RR is the respiratory rate, and V_T is the tidal volume in liters

Mechanical power corresponds to the amount of energy transferred from the ventilator to the respiratory system [3] and it is a physical quantity that brings together the components of mechanical

ventilation, which contribute to the genesis of VILI. [16]

Low Mechanical power values do not prevent VILI, as lung size and heterogeneity must be considered. [9] VILI takes place from a strain sufficient to disrupt the extracellular matrix of the support structure for the alveolar network, which is dependent on the influences of amplifying forces concentrating on geometric stress and parenchymal expansion rate and a cumulative number of cycles. [5] VILI hinges on the relationship between the total energy and the components of mechanical ventilation, which highlights the importance of refining the parametric adjustments of the ventilator. [11] However, doubts persist about which are the safest adjustments of mechanical ventilation components in a lung with ARDS. There is a tendency to reduce them to mitigate VILI. [4] In addition, factors such as: heterogeneity, reduction of ventilated lung areas, respiratory effort, changes in pH, vascular pressure, disease stage and predisposition itself are implicated. [17]

This study aims to verify the correlations and interference of mechanical power and its components with age in the outcome in SARS-CoV-2 of subjects undergoing PCV.

Methods

Longitudinal, prospective, observational, analytical, and quantitative study of the information collected from the ventilation parameters of adult patients age above 18 with moderate ARDS caused by SARS-CoV-2 admitted to an intensive care unit in a university hospital between May 2021 and September 2021. The research was approved by the Research Ethics Committee of the Federal University of Paraná numbered 4.571.036, Hospital das Clínicas da Universidade Federal do Paraná, Brazil.

Inclusion criteria of subjects enrolled: PaO_2:FiO_2 ratio between 150-200 on mechanical ventilation, thorax radiography or computed tomography showing bilateral opacities and with RT-PCR confirming the SARS-CoV-2 infection. There was a total of 163 subjects with SARS-CoV-2 and moderate ARDS mechanically ventilated with pressure-controlled ventilation using the Puritan Bennet TM840® (Minneapolis, Minnesota, USA) under deep sedation and analgesia.

Daily collections of respiratory parameters were selected on three consecutive days prior to the definitive withdrawal of sedation. Thus, 489 sets of mechanical ventilation parameters were collected, three for each of the individuals, which were tabulated individually to obtain their means and used for mechanical power calculations.

The results were used to verify their relationships with survival or death and the correlations of mechanical ventilation components with mechanical power in the statistical analyses. We transcribed the respiratory rate (RR), tidal volume (V_T), inspiratory flow (F_{Insp}), inspiratory time (T_{Insp}), expiratory time (T_{Exp}), positive end expiratory pressure (PEEP), median pressure (P_{Median}), peak pressure (P_{Peak}), plateau pressure ($P_{Plateau}$) using a three-second inspiratory pause. From these data, we generated minute volume (Ve), delta inspiratory pressure (ΔInsp), driving pressure (ΔP), resistive pressure (P_{resist}), static compliance (C), elastance (E), inspiratory resistance (R) and mechanical power.

Mechanical Power Becher [13]

$$0.098 \times RR \times V_T \times (PEEP + \Delta P_{Insp})$$

These results were transcribed to an Excel® spreadsheet and the subsequent statistical analysis were performed in a data processing system by IBM SPSS Statistics v.20.0. Armonk, NY: IBM Corp, Stata/SE 14.1, (Stata Corp LP, USA).

Statistical analysis

For the description of the quantitative variables, statistics of average, median, minimal, and maximal values and standard deviation were considered. To assess the association between the components of mechanical ventilation and mechanical power (J/min), the Pearson correlation coefficient was estimated for each one, measuring the linear association between the component and mechanical power. Additionally, the null hypothesis of the absence of a linear association between the mechanical ventilation component and mechanical power (correlation coefficient equal to zero) was tested versus the alternative hypothesis of the existence of a linear association (correlation coefficient different from zero). For the comparison of the two possible outcomes regarding their relation to the quantitative variables, a t Student Test was performed for independent samples. A univariate analysis of the physiological variable age was applied alongside the same analysis of each parameter of the mechanical ventilator and of its unity represented in Mechanical Power.

A multivariate analysis of the physiological variable age alongside the same analysis for the components of the mechanical ventilation represented by PEEP, driving pressure, elastance and the mechanical power. To evaluate the impact of the collective association of variables on the outcome, a model of Logistic Regression was adjusted, estimating the Odds Ratio of each variable while maintaining constant the remaining ones included in the model. The model adjustment was conducted based on the Hosmer and Lemeshow ‘s test. Finally, the P values under 0.05 was considered statistically significant.

Results

The average length of stay in the ICU was 21 days, with minimal of 9 days and maximum of 59 days,

the mean ventilator length of stay was 11 days. The total data was composed of 91 male patients (56%) and 72 female patients (44%) with minimal age of 22, maximum age of 89, and mean age of 53.7 years. Amongst the 163 patients included in this study, the estimated mortality was of 48.47%, with 79 deaths and 84 survived. Of those who died, 41 were females (51.89%) and 38 were males (48.11%).

The univariate analysis of the comparison between those who died and survived in relation to age, it was observed that the mean age of 58.7 years is more related to deaths and that the mean age of 48.87 years is more related to survival (P <0.001), that is, the result indicates that older ages may be more related to death. In this same comparison model, it was observed that the mean value of mechanical power of 33.34 J/minute is more related to mortality and that the mean value of 28.06 J/minute is more related to survival (P 0.009), that is, the result indicates that higher values of mechanical power may be more related to death, as shown in figure 2.

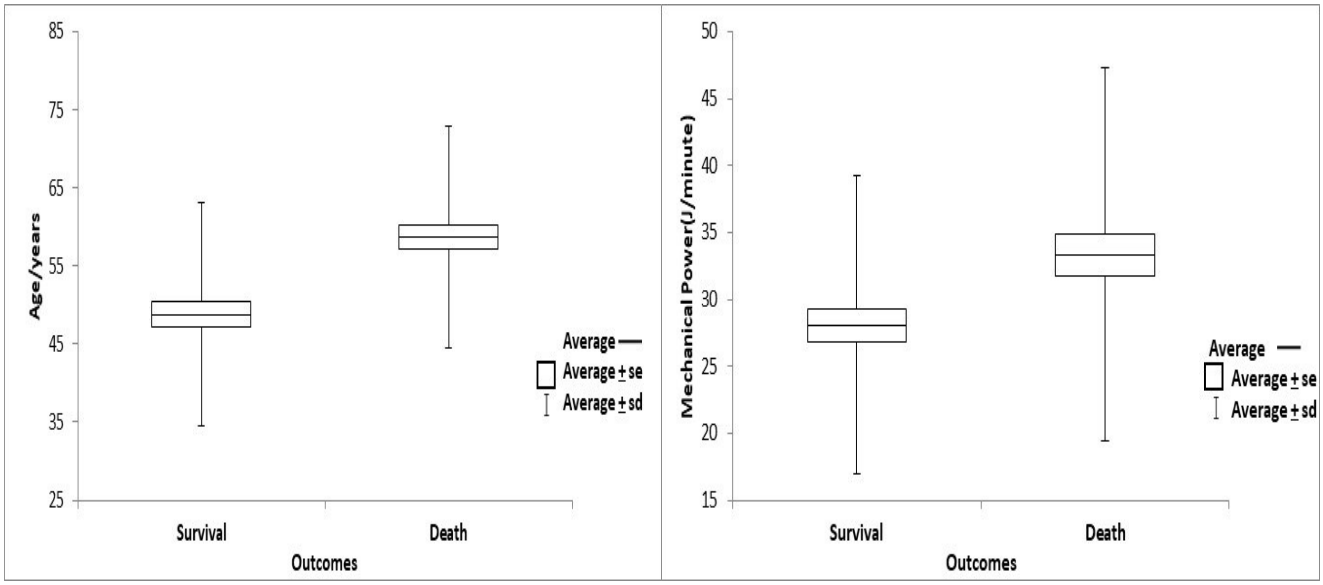

Figure 2: Effects of age and effects of mechanical power on outcomes

The analysis of Pearson's correlation between age and mechanical power, a weak negative correlation (R -0.11) was found but without statistical significance (P 0.18), as shown in Figure 3.

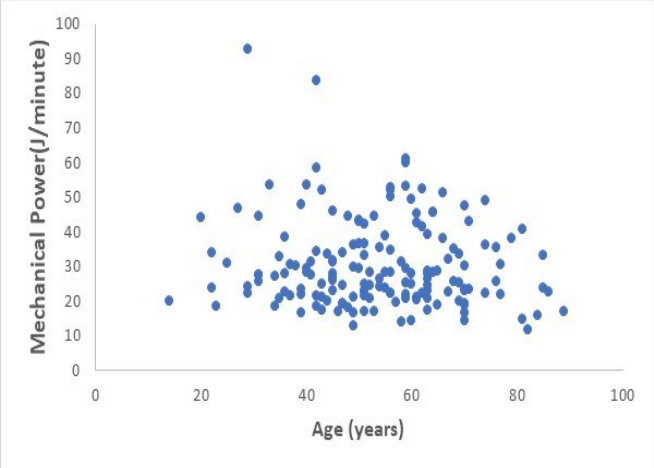

Figure 3: Dispersion diagram between mechanical power and age.

489 calculations of Mechanical Power were made with the mean value of was 30.70 J/min in the three days of data collection in SARS-Cov-2 patients with moderate ARDS on pressure-controlled ventilation of each subject that met the criteria for inclusion in this study. In the analysis of each component of the mechanical ventilation and the mechanical power, the null hypothesis of equal averages of survival and death versus the alternative hypothesis of different averages were tested. The descriptive statistics are outlined considering the results of each variable obtained in this study in Table 1.

Regarding the comparison between the outcomes of survival and death, inspiratory flow (P 0.011), tidal volume (P <0.001), respiratory rate (P <0.001), inspiratory time (P <0.001), expiratory time (P <0.001), the variables derived from the pulmonary conditions: elastance (P <0.001) and compliance (P <0.001), as well as the pressure variables peak pressure (P <0.001), delta inspiratory pressure: PIP - PEEP (P <0.001), driving pressure: Plateau pressure - PEEP (P <0.001), median pressure (P 0.001), plateau pressure (P <0.001) showed statistical significance. Except for PEEP, Ve, P_{Resist} and resistance, the mean values of all other components showed significance in relation to the outcome in the isolated assessment (Table 1).

To understand the influence of the components on the mechanical power result, the mechanical ventilation components were separately correlated with the results obtained from the formula. Table 2 below shows the estimated correlations as well as the P values of the statistical tests.

Variable	Outcome	N	Mean	Median	Mín.	Max.	Standard Deviation	P value
Mechanical power (J/min)	**Survival**	84	28.06	25.39	11.92	83.85	11.14	0.009
	Death	79	33.34	28.79	14.49	92.88	13.92	
F_{Insp} (L/min)	**Survival**	84	33.16	31.20	21.82	73.60	8.70	0.011
	Death	79	29.78	29.60	12.60	56.25	7.95	
Compliance (L/cmH_20)	**Survival**	84	0.040	0.037	0.021	0.074	0.010	< 0.001
	Death	79	0.027	0.027	0.010	0.050	0.009	
Elastance (cmH_20/L)	**Survival**	84	26.72	26.93	13.51	48.72	6.64	< 0.001
	Death	79	42.41	37.50	20.00	100.00	16.21	
Resistence ($cmH_2O/L/min$)	**Survival**	84	0.09	0.08	0.02	0.23	0.05	0.071
	Death	79	0.11	0.09	0.06	0.41	0.07	
V_T (L)	**Survival**	84	0.49	0.48	0.30	0.92	0.10	< 0.001
	Death	79	0.41	0.40	0.21	0.75	0.10	
Ve (L/min)	**Survival**	84	10.78	10.40	6.56	27.60	3.31	0.437
	Death	79	11.19	10.88	5.60	22.04	3.37	
RR /min	**Survival**	84	22.1	22	14	36	4,9	< 0.001
	Death	79	27.8	27	14	40	6.1	
P_{Peak} (cmH_20)	**Survival**	84	26.2	26	16	35	3.8	< 0.001
	Death	79	29.8	29	20	43	5.0	
ΔP_{Insp} (cmH_20)	**Survival**	84	15.4	15	10	23	2.6	< 0.001
	Death	79	19.1	18	12	29	4.2	
$P_{Plateau}$(cmH_20)	**Survival**	84	23.5	23	14	33	3.6	< 0.001
	Death	79	26.9	26	17	41	4.6	
P_{Resist} (cmH_20)	**Survival**	84	2.7	3	1	6	1.2	0.410
	Death	79	2.9	3	2	9	1.6	
ΔP (cmH_20)	**Survival**	84	12.7	12	8	20	2.5	< 0.001
	Death	79	16.2	15	9	25	3.8	
P_{Median} (cmH_20)	**Survival**	84	16.0	16	10	25	3.2	0.001
	Death	79	18.1	17	11	28	4.3	
PEEP (cmH_20)	**Survival**	84	10.8	10	6	18	2.6	0.874
	Death	79	10.7	10	6	20	3.0	
T_{Insp} (s)	**Survival**	84	0.90	0.90	0.60	1.20	0.13	< 0.001
	Death	79	0.83	0.80	0.62	1.10	0.11	
T_{Exp} (s)	**Survival**	84	1.94	1.86	0.87	3.43	0.56	< 0.001
	Death	79	1.44	1.40	0.65	3.39	0.53	

Table 1: Comparison between each variable, mechanical power and their effect on outcomes. ΔInsp: Delta inspiratory pressure, ΔP: Driving pressure, F_{Insp}: Inspiratory flow, P_{Peak}: Peak inspiratory flow, P_{Median}: Median pressure, RR: Respiratory rate, R_{Insp}: Inspiratory resistance, T_{Insp}: Inspiratory time, T_{Exp}: Expiratory time, Ve: Minute ventilation, VT: tidal volume, VT/PBW: tidal volume per predicted body weight.

The F_{Insp}, RR, Vt, P_{Peak}, $P_{Plateau}$, P_{Media}, PEEP, ΔP_{Insp} and ΔP demonstrated a moderate positive correlation, while Ve had a strong positive correlation and P_{Resist} had a weak positive correlation. In contrast, the elastance, compliance, resistance didn`t show significant correlations with the result of mechanical power. Figure 4 shows the correlations between mechanical power and elastance and with its components (tidal volume, plateau pressure, ΔP) that define the concept of protective ventilation.

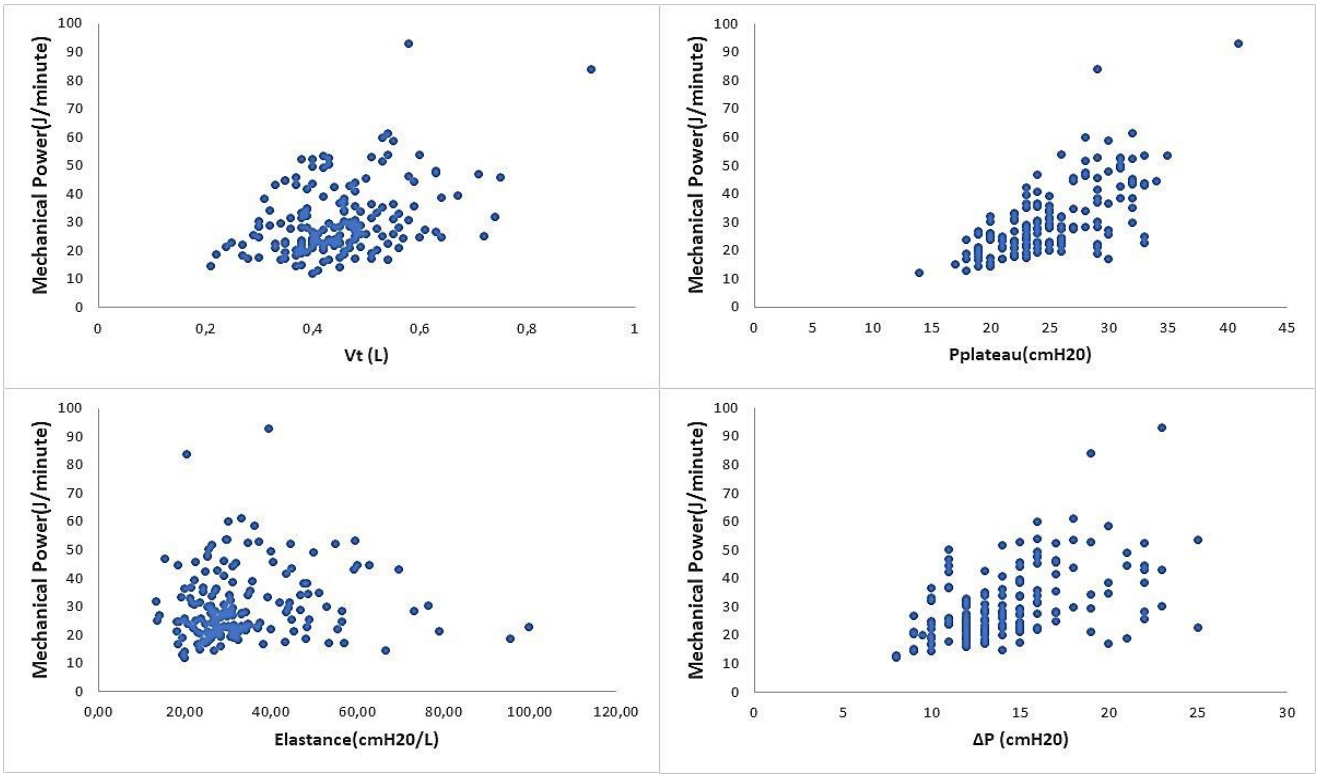

Figure 4: Mechanical power dispersion diagram with its components of protective ventilation. V_T – tidal volume; $P_{Plateau}$ - plateau pressure. ΔP: driving pressure, V_T; tidal volume, $P_{plateau}$: plateau pressure.

For the joint evaluation of selected variables and their effect on the outcome, a logistic regression model was adjusted. Mechanical power was selected, which represents the unified set of mechanical ventilation components, the components that currently determine the values of protective ventilation, which are $P_{Plateau}$, V_T and ΔP, as well as the elastance that is derived from the pulmonary condition. To assess the goodness of fit, the null hypothesis of model adequacy to explain the outcome was tested versus the alternative hypothesis of non-adequacy. The result of the statistical test did not indicate the rejection of the null hypothesis (P 0.116). Depending on the adequacy of the model, the results of this analysis are presented in the table 3.

Variable	correlation coefficient	P value
F_{Insp} (L/min)	0.61	< 0.001
Compliance (L/cmH_2O)	- 0.09	0.234
Elastance (cmH_2O/L)	0.05	0.515
Resistence (cmH_2O/L/min)	- 0.04	0.570
V_T (L)	0.41	< 0.001
Ve (L/min)	0.92	< 0.001
Rr /min	0.66	< 0.001
P_{Peak} (cmH_2O)	0.69	< 0.001
ΔP_{Insp} (cmH_2O)	0.52	< 0.001
$P_{Plateau}$ (cmH_2O)	0.68	< 0.001
P_{Resist} (cmH_2O)	0.17	0.028
ΔP (cmH_2O)	0.50	< 0.001
P_{Media} (cmH_2O)	0.64	< 0.001
PEEP (cmH_2O)	0.43	< 0.001
T_{Insp} (s)	- 0.37	< 0.001
T_{Exp} (s)	- 0.58	< 0.001

Table 2: Correlations between mechanical power and parameters of mechanical ventilation

Variables	P value		Risk Ranking	Odds Ratio	95% Confidence Interval
	Univariate	Multivariate			
V_T (L)	< 0.001	0.236	---	0.911	(0.780 - 1.064)
Elastance(cmH_20/L)	< 0.001	0.580	---	1.060	(0.862 - 1.303)
ΔP (cmH_20)	< 0.001	0.450	---	1.239	(0.708 - 2.166)
$P_{Plateau}$(cmH_20)	< 0.001	0.586	---	0.953	(0.802 - 1.133)
Mechanical power (J/minute)	0.009	0.040	Higher values	1.062	(1.003 - 1.124)

Table 3: Multivariate analysis between mechanical power and its components of protective ventilation and elastance. V_T: tidal volume, ΔP: Delta pressure, Pplateau - plateau pressure.

As shown in Table 3, in the joint presence of the stipulated variables there is no evidence of association of V_T (L), elastance (cmH_20/L), ΔP (cmH_2O) and $P_{plateau}$ (cmH_2O) with the outcome. However, mechanical power (J/minute) was the only variable that, in the presence of the others, demonstrated that it can interfere with the outcome with statistically significant evidence (P 0.04) and that the higher its values, the higher the risk of death. It is estimated that, controlling for the other variables included in the model, for each additional unit in J/minute of mechanical power, there is an increase of 6.2% in the odds of death (95% CI 0.3%-12.4%)

Discussion

A more advanced age seems to consolidate itself as a major factor related to death by SARS-CoV-2, as already shown a statistically significant difference between the mean ages of 49.5 and 53.1 years, [18] or even with a greater interval of difference with the mean age of 45.7 years for the survivors and 55.9 years for the non survivors, [19] our data corroborate those findings, in which it is observed the average age of 58.7 years was more related to deaths and the average age of 48.87 years is more related to survival (P <0.001).

On the other hand, it is observed that the average value of mechanical power of 33.34 J/minute is more related to deaths and that the average value of 28.06 J/minute is more related to survival (P 0.009), this fact was not found in the previous study in VCV, with a mean of 26.80 J/minute in survivors and 26.58 J/minute in non survivors (P 0.864). [19] In this study, it was observed that older ages and higher mean values of mechanical power are more related to death, however, in the analysis of Pearson's correlation between age and mechanical power, there was no correlation between age and the values of mechanical power (P 0.18), that is, the relationship of the results of age and mechanical power with the death and survival do not depend on the conjunction between them.

The mean mechanical power values in the PCV modality reported by van der Meijden was 24.31 J/minute in patients with ARDS, 11.49 J/min in the postoperative period and 14.54 J/minute in other clinical circumstances. [14] These data demonstrate that ARDS is the clinical situation that requires the most tidal energy. In our current study, carried out in patients with moderate ARDS in SARS-CoV-2, an even higher mean mechanical power values of 30.70 J/minute were observed, which also exceeded the mean values of 26.71 J/minute found in the VCV modality in SARS-CoV-2 with moderate ARDS. [19] In both cases, the energy dissipated to the patient per breath cycle is proportional to the hysteresis area of the PV loop. For the same PEEP, Pplat and respiratory rate, the energy dissipated during VCV is lower than during PCV. The energy during the inspiration phase is minimized if the flow is kept constant during inspiration. [20] Similar findings in a bench study that showed that showed the AVM-2 mode delivered significantly less mechanical power than VCV, which in turn was less than PCV in various ARDS severity. [21]

The impact of mechanical power is consistent and independent of the presence of ARDS and the use of neuromuscular blockers, and even with the maintenance of tidal volume and driving pressure at low values, it was associated with worse outcomes, which suggests that mechanical power adds information that go beyond just the tidal volume and driving pressure. [22]

Regarding the comparison between survival and death outcomes, except for PEEP, Ve, $P_{Rresist}$ and resistance, the mean values of all the other components showed significance in relation to the outcome in the isolated assessment. However, these components are interrelated during mechanical ventilation, so evaluating their behavior in the outcome together with other variables is essential.

Mechanical power was selected, which represents the unified set of mechanical ventilation components, with the other components that currently determine the values of protective ventilation, which are $P_{Plateau}$, VT, ΔP, as well as the elastance that are derived from the pulmonary conditions. It was demonstrated that only the mechanical power may interfere with the outcome of SARS-CoV-2 with moderate ARDS and that for

each unit in J/minute of mechanical power there is a 6.2% increase in the chances of death.

Considering the parameters of protective ventilation, the mean $P_{Plateau}$ values related to death was higher than the mean of those who survived, however the mean value of 26.9 cmH_2O related to death is below 30 cmH_2O, [7] which is the value considered to be protective. Perhaps the mechanical power rating the distending pressure built into the equation should not be the $P_{plateau}$, but by the transpulmonary pressure, similarly, the transpulmonary mechanical power may be a more accurate measure as the actual stress in the lung units can be up to 4 times greater than that measured in the upper airways. [15] Likewise, the mean value of ΔP related to death was higher than the mean of those who survived, but for this component the mean value of 16.2 cmH_20 related to death is above 15 cmH_2O, [7] which is the value considered to be protective.

The application of high V_T increases the stress and strain, proportional to the level of mechanical power. Alterations of lung structure and inflammatory reaction accounts for the increase in extravascular lung water and decreased FRC. Interestingly, the specific lung elastance remained constant, as found in patients with ARDS, where the “baby lung” maintains relatively normal intrinsic elastic properties. [23]

In PCV, a safe limiting pressure is defined as a target generating the tidal volume, which is related to lung compliance and airway resistance. [24] Unlike the $P_{Plateau}$ and the ΔP, the mean V_T values in those who died (6.61 ml/kg IBW) was lower than the mean of those who survived (7.9 ml/kg IBW). Two possibilities may be acceptable, the first would be the hypothesis that lower V_T are related to more severe ARDS and that they were imposed by the operator on ventilator adjustments to prioritize lung protection with lower volumes, the second is, the higher V_T resulting from a greater compliance with the milder ARDS, that is, in both, it seems that the severity of the ARDS suppressed protective ventilation.

Mechanical power delivered to the lung is the product of the determinants of mechanical ventilation, in this way the approach to the adjustments made in V_T and ΔP have an impact on the values of mechanical power, [2] however the search for lower values should be prioritized, especially in ARDS, when there is heterogeneity and the functional lung volume is reduced, imprecise, unpredictable, and not proportional to the predicted weight. [25] In this study, it was observed that V_T and ΔP have a moderate positive correlation with mechanical power, while elastance and compliance showed no correlation. The impression given by these correlations is that the severity of ARDS and the high momentary values of mechanical power do not suffer reciprocal interference from high elastance and low compliance, but these results are incompatible with the pathophysiology. Probably, the result of these correlations is due to manipulations in the adjustments of ΔP and PEEP on the ventilator to obtain the desired V_T. Thus, in order to recognize if the mechanical power is increased by iatrogenesis in the ventilation adjustments, or if it is increased by the severity of ARDS, or even if it is related to incurability by VILI, we would possibly have to somehow index the mechanical power with the compliance or elastance. [21]

Theoretically, a well aerated lung with better compliance will require less mechanical power i.e. lower Power Compliance Index (higher Power Elastance Index), versus a non-aerated lung with poorer compliance will require higher mechanical power i.e. higher Power Compliance Index (lower Power Elastance index) to achieve targets of ventilation. Some authors suggested that well aerated compliant lung might be less vulnerable to develop VILI by the intensity of the mechanical power, [25] on the other hand some suggested that a healthy lung might actually be more vulnerable to injury versus an already injured lung. [26]

In our study, the mean Power Compliance and Elastance Index for survivors were 0.701 and 1.05 compared to 1.23 and 0.78 respectively for the non survivors which emphasizes the concept of indexing or normalizing the mechanical power to other variables of mechanical ventilation. [27]

Our study has some limitations that should be taken in context while interpreting the results of our study. The study was a retrospective study with no specific protocol for ventilation though lung protective strategies like low tidal volume, lowest possible ΔP, the data and calculations were done during thre consecutive days but not every day during mechanical ventilation, effects of other therapies like prone position, inhaled vasodilators, bronchodilators were not accounted for.

Conclusion

There are correlations between mechanical power and its components, except for compliance, elastance, resistance and age. The outcome is influenced in the univariate analysis by age, as well as of mechanical power and its components, except for PEEP, Ve, P_{Resist} and resistance, but the multivariate analysis showed that only mechanical power interferes with the outcome in SARS-CoV-2 undergoing PCV. Further studies with other causes of ARDS and other modes need to be done to confirm our findings.

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