Influence of age, mechanical power, its fragments and components on the mortality rate in SARS-CoV-2 patients undergoing mechanical ventilation

Claudio Luciano Franck [1], Gustavo Maysonnave Franck [2], Raquel Galvão Feronato [3]

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Abstract

Introduction
The Acute Respiratory Distress Syndrome caused by the Coronavirus 2019 (SARS-CoV-2) may be associated with the Acute Respiratory Distress Syndrome (ARDS) and Ventilation Induced Lung Injury (VILI). However, there are still doubts about the potential damage generators and their influences on patient outcome

Objective
To analyze the mechanical ventilation factors that influence the mortality in SARS-CoV-2. Assess the outcomes based on age, on parameters of the mechanical ventilator, on Mechanical Power and on its fragments through univariate and multivariate analysis of age, PEEP, Driving Pressure, elastance

Method
Observational, longitudinal, prospective, analytical, and quantitative study of age and of the parameters of the mechanical ventilator, alongside the calculous of the Mechanical Power and its components of patients with SARS-CoV-2

Results
We identified significant impact on the outcome in the univariate analysis of age ($p<0.001$), respiratory rate ($p=0.047$), elastance ($p<0.001$), compliance ($p<0.001$), driving pressure ($p<0.001$), inspiratory pressure variation ($p<0.001$), peak airway pressure ($p=0.009$), plateau pressure ($p<0.013$), PEEP ($p<0.001$), dynamic elastic power ($p<0.001$) and static elastic power ($p=0.005$). In the multivariate analysis the increase in age ($p<0.001$), in elastance ($p=0.0029$) and in Mechanical Power ($p=0.023$), and the reduction in PEEP ($p=0.044$) showed significant impact on the death risk

Conclusion
The increase in age and in mechanical power with increased dynamic elastic power and decreased static elastic power influenced the mortality rate of patients with SARS-CoV-2 undergoing mechanical ventilation, i.e. it is related to the increase in driving pressure to overcome a high elastance and low capacity to recruit for PEEP

Keywords: SARS-CoV-2 infection, Mortality, Mechanical Power, Ventilation Induced Lung Injury, Acute Respiratory Distress Syndrome.

Authors:
1-MD, PhD. Faculdade Evangélica Mackenzie do Paraná, Hospital da Clínicas da Universidade Federal do Paraná, Brazil
2-Academic, Universidade Tecnológica Federal do Paraná, Brazil
3-Academic, Universidade Federal do Paraná, Brazil

Corresponding author: claudiolfranck@hotmail.com
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Introduction

It is proposed to diagnose the SARS-CoV-2 infection through the detection of radiologic consolidation and bilateral air bronchogram with peripheral ground-glass opacity, simultaneously with a Partial Arterial Pressure of O_2 over Fraction of Inspired O_2 ratio (PaO_2/FiO_2) under 300, hypoxemia and respiratory insufficiency with pulmonary inflammation, which can evolve to fibrosis when associated with mechanical ventilation. [1]

In 1990, Ventilation induced lung injury (VILI) [2] was defined as the lesions derived from the application of mechanical powers and from the transfer of energy to the lung tissue that causes inflammation. ARDS and VILI intertwine in a unique and complex manner, in such a way that the contribution of each to the lethality of patients is indistinguishable. [3]

As they occur concomitantly, it is impossible to separate VILI from the underlying process that causes ARDS, but VILI is predicted by the progressive reduction in ventilated lung volume without response to recruitment maneuvers. [4] It is established that the injury produced is determined by the pressure gradient and by the resulting alveolar deformity, Stress and Strain, respectively. [5]

However, the understanding of the biophysical causes of VILI has redirected the attention to the exposition of the alveolar-capillary membrane to damaging levels of energy and power. [4] In addition, logically, VILI also depends on the vulnerability of the lung tissue, [6] but it is important to recognize the quality and quantity of lung injury that each component of mechanical ventilation can determine.[7]

Controversies persist regarding the best oxygenation and ventilation strategies for patients with ARDS caused by SARS-CoV-2 that avoid VILI. The protective ventilation strategy is applied when tidal volume is kept between 4 and 8 ml/Kg of predicted body weight, plateau pressure lower than 30 cmH_2O, driving pressure lower than 15cmH_2O [8] and PEEP is maintained between 8 and 14 cmH_2O. [9]

The modern concept of protective mechanical ventilation allows a parametric excess only during the initial critical care period, requiring the minimization of ventilation parameters as soon as the clinical status of the patient stabilizes.[10] This procedure is justified based on the understanding that the inadequate application of the mechanical force with elevation of its components respiratory rate, tidal volume, airway pressures and flow increase the levels of mechanical power, compromising the purpose of resting the lungs promoted by the mechanical ventilation, and worsening outcomes by increasing the risk of VILI. [11-12]

Comprised of plateau pressure, driving pressure, PEEP, tidal volume, flow, resistance, elastance and respiratory rate, [13-14] mechanical power depicts the energy transferred to the respiratory system by the mechanical ventilator [13] during a certain period in Joule per minute (J/min). [11-15]

Even though 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 generator of VILI, its predictive precision is still alleged, [16-17] given there are patients submitted to potential VILI generating tidal volume and pressures that survive, making the correlation between ventilatory strategy and clinical status questionable. [18] However, lower levels of mechanical power are desired, [19] as it unifies the components of the mechanical ventilation in an attempt to build a unity of indicators that reflect VILI. [11] The protective mechanical ventilation strategies in ARDS intend to reduce the chances of VILI, however it is still unclear the contribution of each component [20] of the mechanical ventilation to the higher risk of VILI.

The quantitative fragmentation of the energy required to inflate the lungs may point to protective ventilation strategies effective to the reduction of the risk of VILI, [16] and may clarify the contribution of each component of the power to the generation of such injury.

The product of the tidal volume per plateau pressure defines the total elastic power, [17] which is

subdivided into dynamic and static elastic powers. The dynamic one is equal to the energy necessary to

inflate the lungs, whereas the static one is the energy required to balance out the potential energy stored in the respiratory system by the PEEP. [21] The total inflation energy, that is, the mechanical power is equal to the total elastic power plus the total kinetic power, [17] which is the energy spent on overcoming the airway and tissue resistance to the flow. [21] The product of these equations by the respiratory rate and by the conversion constant 0.098 results in Joules per minute.

This study aim to analyze the interference of the mechanical ventilation on the outcomes of patients with moderate ARDS caused by SARS-CoV-2 undergoing mechanical ventilation, to find the average value of energy applied by the ventilator through the mechanical power, to compare the outcomes of survival and death with the components of the mechanical ventilation, and to determine the mechanical power and its subdivisions in SARS-CoV-2 through the implementation of univariate and multivariate analysis of age, PEEP, driving pressure, elastance and mechanical power.

Methods

Longitudinal, prospective, observational, analytical, and quantitative study of the information collected from the ventilation parameters of patients with moderate ARDS caused by SARS-CoV-2 admitted to an intensive care unit in a university hospital between May 2021 and September 2021.This research was approved by the Research Ethics Committee of the Federal University of Paraná, in the process numbered 4.571.036.

As an inclusive criterion for the data collection of subjects, it was required a PaO_2:FiO_2 ratio between 100 and 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 147 subjects with SARS-CoV-2 and moderate ARDS that remained intubated in the volume-controlled ventilation in a Puritan BennetTM840® (Minneapolis, Minnesota, USA) under deep sedation, analgesia and neuromuscular blocking agents (NMBA). A total of 1029 components of mechanical ventilation were documented, and we chose to collect the parameters every 3 days to establish the average mechanical power required throughout the subjects stay.

On the other hand, in the analysis for comparison of outcomes, we documented the last parameters of the mechanical ventilation and calculated the mechanical power and its subdivisions just before the suspension of the NMBA or the death of the subject.

We transcribed the respiratory rate (RR), tidal volume (Vt), inspiratory flow (Finsp), inspiratory time (Tinsp), expiratory time (Texp), average pressure (Pmedia), peak pressure (Ppeak), plateau pressure (Pplateau) through the inspiratory pause and intrinsic PEEP (PEEPI) through the expiratory pause. From these data, we generated minute volume (Ve), driving pressure, resistive pressure (Presist), static compliance (C), elastance (E), inspiratory resistance (Rinsp) expiratory resistance (Rexp), expiratory flow (Fexp) and mechanical power. [23]

- *Mechanical Power* = $0.098 \times RR \times Vt \left[P_{Peak} - 0.5 \left(P_{Plateau} - PEEP\right)\right]$

  Mechanical power was subdivided [22] and each part was multiplied by the respiratory rate and the conversion constant 0.098, so that it would be possible to determine its values in Joules per minute (J/min):

- Dynamic elastic power = $0.098 \times RR \times Vt \left(0.5 \times E \times Vt^2\right)$

- Static elastic power = $0.098 \times RR \left(Vt \times PEEP\right)$

- Inspiratory flow resistive power = $0.098 \times RR \left(Vt \times R / T_{insp}\right)$

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

The 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, which was subdivided in its components dynamic elastic power, static elastic power and inspiratory flow resistive power.

The multivariate analysis of the physiological variable age alongside the same analysis for the static and elastic components of the mechanical ventilation represented by PEEP and driving pressure, respectively, the later been determined by the resistance from the elastance and by the energy required to inflate the lungs, thus, Mechanical Power.

For the description of the quantitative variables, statistics of average, median, minimal, and maximal values and standard deviation were included. For the comparison of the two possible outcomes regarding their relation to the quantitative variables, a Student's t-test was performed for independent samples. The estimation of Pearson 's correlation coefficient was considered for the analysis of association between the quantitative variables. To evaluate the impact of the collective association of variables on the outcome, a model of Logistic Regression was adjusted, estimating the Odds Ratio (OR) 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 indicated statistical significance.

Results

1029 calculus of Mechanical Power were made from the data of 147 subjects with SARS-CoV-2 for the time they were intubated for the maintenance of mechanical ventilation in volume-controlled ventilation under deep sedation, analgesia and neuromuscular blocking agents. The average value of Mechanical Power was 26.71*J*/min, which is close to the average value of 26.60*J*/min found in the 147 samples collected just before the withdrawal of NMBA or death of each subject that met the criteria for inclusion in this study.

The average length of stay in the ICU was 21 days, with minimal of 7 days and maximum of 56 days. Among the 147 subjects enrolled in the study, 81 are males (55.10%) and 66 are females (44.90%) with a minimum age of 20 years, maximum of 79 years and mean age of 52.2 years, the estimated mortality was 63.9%, with 94 deaths and 53 survivors. Of the survivors, 25 were females (37.90%) and 28 were males (34.50%). In the univariate analysis of age, a mean age of 45.7 ± 11.4 years was observed in those who survived and 55.9 ± 13.6 years in those who died ($p<0.001$), as shown in Figure 1.

In the analysis of each component of the mechanical ventilation, 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, respiratory rate ($P=0.047$), the variables derived from the pulmonary conditions: elastance ($P<0.001$) and compliance ($P<0.001$), as well as the pressure variables driving pressure ($P<0.001$), inspiration pressure variation ($P<0.001$), peak pressure ($P=0.009$), plateau pressure ($P<0.013$) and PEEP ($P<0.001$) showed statistical significance.

A box diagram depicting the interference of the static elastic and dynamic elastic components, PEEP and driving pressure, respectively and the need of Stress to promote Strain, represented by the elastance, are highlighted in Figure 2.

After analyzing the components of mechanical ventilation separately, the set of them unified in the form of energy by mechanical power was analyzed. In Table 2, the univariate analysis of the influence of mechanical power on the outcomes shows no statistical significance ($P=0.864$).

However, to better understand the features of the total inspiratory energy, mechanical power was subdivided in its three factors: 1) inspiratory flow resistive power: resulting in dissipation of heat, 2) dynamic elastic power: propulsion energy of the driving pressure required to overcome the elastance, and 3) static elastic power: applied by PEEP in order to avoid the collapse of the alveoli.

In the analysis of each variable, the null hypothesis of equal averages between survival and death versus the alternative hypothesis of different averages were tested.

Table 2 shows the descriptive statistics for each variable considering its results obtained in this study. When separate, both the dynamic elastic power (P<0.001) and the static elastic power (P=0.005) varied according to the outcome. Patients that died presented higher values of dynamic elastic power (P<0.001) and lower static elastic power (P=0.005) when compared to these energetic variables of the group of patients that survived, as shown in Figure 3.

The impact of age, as well as of PEEP, driving pressure and elastance all related to static and dynamic energies on the outcome in the univariate analyses, simultaneously with the irrelevance of changes in the unifying concept of Mechanical Power to the outcome moved the execution of a multivariable analysis of these variables. Hence, a model of logistic regression was built so that it would be possible to study the group of the selected variables as a whole and its effect on the outcome.

To evaluate the quality of the adjustment, the null hypothesis of adequate explanation of the outcome by the model versus the alternative hypothesis of inadequacy were tested. The result of the statistical test did not indicate rejection of the null hypothesis (p=0.899). The results of this quality analysis are shown in Table 3.

As displayed in Table 3, when studied alongside the other variables, increases in age (p<0.001) increase the death risk. In regard to PEEP (p=0.044), alongside the other variables, there is evidence that lower values of PEEP increase the risk of death. For elastance (p=0.029), in the presence of the other variables, greater values increase the risk of death.

The values of driving pressure in the analysis of each variable showed an impact on the outcome (p<0.001), however in the presence of the other factors there was no evidence of association between this variable and the outcome (p=0.245). In contrast, the values of Mechanical Power showed no influence on the outcomes (p=0.864) in the univariate analysis, whereas in the multivariate analysis there is evidence that higher values of Mechanical Power increase the risk of death (p=0.023)

Variable	Outcome	N	Mean	Median	Min.	Max.	Standard Deviation	P value
F_{Insp} (L/min)	**Survival**	53	53.3	54.0	35	74	8.9	0.903
	Death	94	53.1	53.5	24	82	8.9	
Compliance (L/cmH_2O)	**Survival**	53	0.034	0.034	0.016	0.060	0.010	< 0.001
	Death	94	0.027	0.023	0.009	0.077	0.013	
Vt (L)	**Survival**	53	0.35	0.36	0.25	0.50	0.06	0.367
	Death	94	0.34	0.34	0.23	0.54	0.06	
Ve (L/min)	**Survival**	53	11.32	11.20	7.56	17.00	2.12	0.890
	Death	94	11.37	10.85	7.68	18.20	2.13	
RR/min	**Survival**	53	32.0	32.0	25	36	3.1	0.047
	Death	94	33.1	35.0	22	37	3.0	
P_{Peak} (cmH_20)	**Survival**	53	29.4	29.0	22	38	3.7	0.009
	Death	94	31.3	31.0	20	48	4.9	

Δ_{Insp}(cmH_20)	**Survival**	53	15.0	15.0	7.0	24.5	3.1	< 0.001
	Death	94	19.5	18.3	8.0	42.0	5.7	
Plateau (cmH_20)	**Survival**	53	25.4	25.0	19	31	2.9	0.013
	Death	94	27.0	27.0	17	44	4.6	
Resistance (cmH_20)	**Survival**	53	4.0	4.0	1	10	2.1	0.343
	Death	94	4.3	4.0	2	10	1.8	
ΔP (cmH_20)	**Survival**	53	11.0	11.0	5.0	22.5	3.1	< 0.001
	Death	94	15.2	14.3	5.0	38.0	5.7	
PEEP (cmH_20)	**Survival**	53	14.4	15.0	6.3	20.0	3.3	< 0.001
	Death	94	11.8	11.0	5.0	21.0	4.0	
PEEP I (cmH_20)	**Survival**	53	0.8	0.7	0	2.7	0.5	0.504
	Death	94	0.7	0.6	0.2	2.4	0.4	
T_{Insp} (s)	**Survival**	53	0.76	0.74	0.66	1.00	0.06	0.237
	Death	94	0.75	0.74	0.60	1.00	0.07	
T_{Exp} (s)	**Survival**	53	1.13	1.06	0.95	1.58	0.17	0.087
	Death	94	1.08	1.01	0.90	1.73	0.16	
Elastance **(cmH_2O/L)**	**Survival**	53	31.84	29.27	1667	62.50	10.27	< 0.001
	Death	94	45.97	44.28	12.96	106.67	19.78	
F_{Exp} (l/min)	**Survival**	53	19.19	18.31	11.83	31.25	4.09	0.689
	Death	94	19.47	19.18	11.85	30.89	3.98	
R_{Insp} ($cmH_2O/L/min$)	**Survival**	53	0.142	0.130	0.030	0.361	0.069	0.163
	Death	94	0.159	0.138	0.056	0.444	0.072	
R_{Exp} ($cmH_2O/L/min$)	**Survival**	53	0.040	0.039	-	0.096	0.025	0.347
	Death	94	0.037	0.031	0.008	0.148	0.022	
P_{Median} (cmH_20)	**Survival**	48	19.4	19.0	14.0	25.0	2.8	0.472
	Death	88	19.0	18.0	12.0	28.0	3.5	

Table 1: Comparison between each variable and their effect on outcomes.
Δ_{Insp}: Delta inspiratory pressure, ΔP: Delta pressure, F_{Insp}: Inspiratory flow, F_{Exp}: Expiratory flow, P_{Peak}: Peak inspiratory flow, P_{Median}: Median pressure, RR: Respiratory rate, R_{Insp}: Inspiratory resistance, R_{Exp}: Expiratory flow, T_{Insp}: Inspiratory time, T_{Exp}: Expiratory time, Ve: Minute ventilation

Variable (*J*/min)	Outcome	N	Media	Median	Min.	Max.	Standard Deviation	*P* value
Mechanical Power or Total Inspiratory Energy	**Survival**	53	26.80	25.24	15.83	44.98	7.59	0.864
	Death	94	26.58	24.96	13.72	50.01	50.01	
Elastic Energy Dynamic Inflation	**Survival**	53	6.17	5.93	2.21	13.89	2.25	< 0.001
	Death	94	8.41	7.76	1.96	24.11	3.51	
Inspiratory Flux Resistance Energy	**Survival**	53	4.56	3.81	0.83	11.66	2.73	0.562
	Death	94	4.81	4.51	1.51	11.11	2.18	
Static Elastic Energy	**Survival**	53	16.07	15.61	6.27	27.59	5.07	0.005
	Death	94	13.36	11.71	5.35	33.34	5.84	

Table 2: Fragmentation of Mechanical Power and their effect on outcomes

Variable	P value		Risk Ranking	Odds Ratio	95% Confidence Interval
	Univariate	Multivariate			
Age (Years)	< 0.001	< 0.001	Older ages	1.063	(1.029 - 1.099)
Driving pressure (cmH_20)	< 0.001	0.245	---	1.245	(0.859 - 1.806)
PEEP (cmH_20)	< 0.001	0.044	Lower values	1.217	(1.006 - 1.473)
Elastance (cmH_2O/L)	< 0.001	0.029	Higher values	1.118	(1.012 - 1.237)
Mechanical Power (*J*/min)	0.864	0.023	Higher values	1.134	(1.018 - 1.263)

Table 3: Multivariate analysis of age, PEEP, driving pressure, elastance and mechanical power

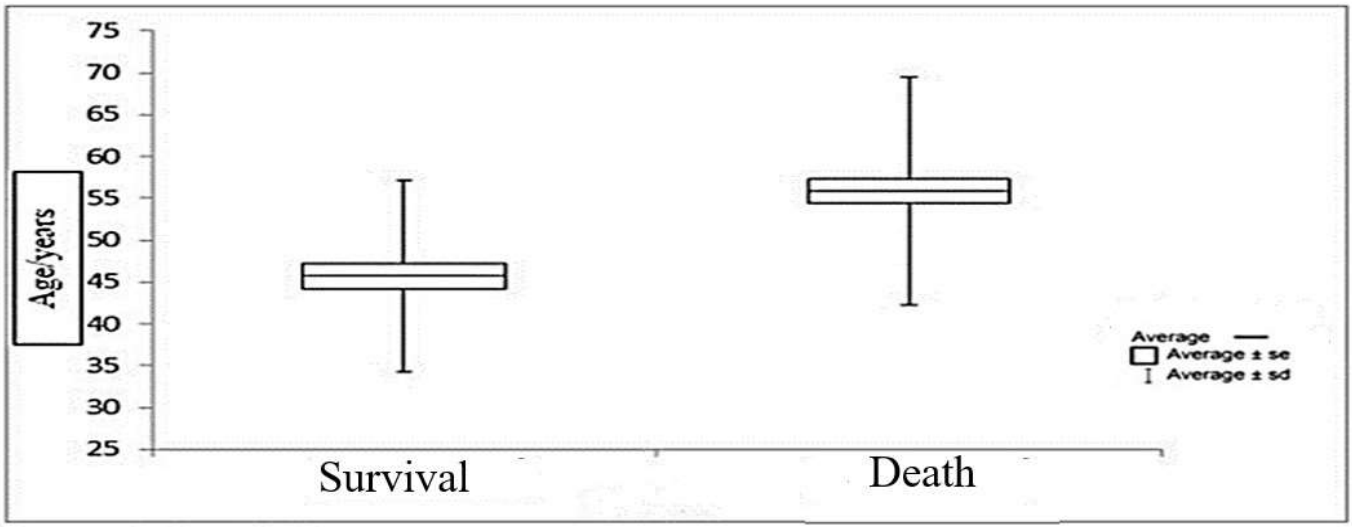

Figure 1: Effects of age on outcomes

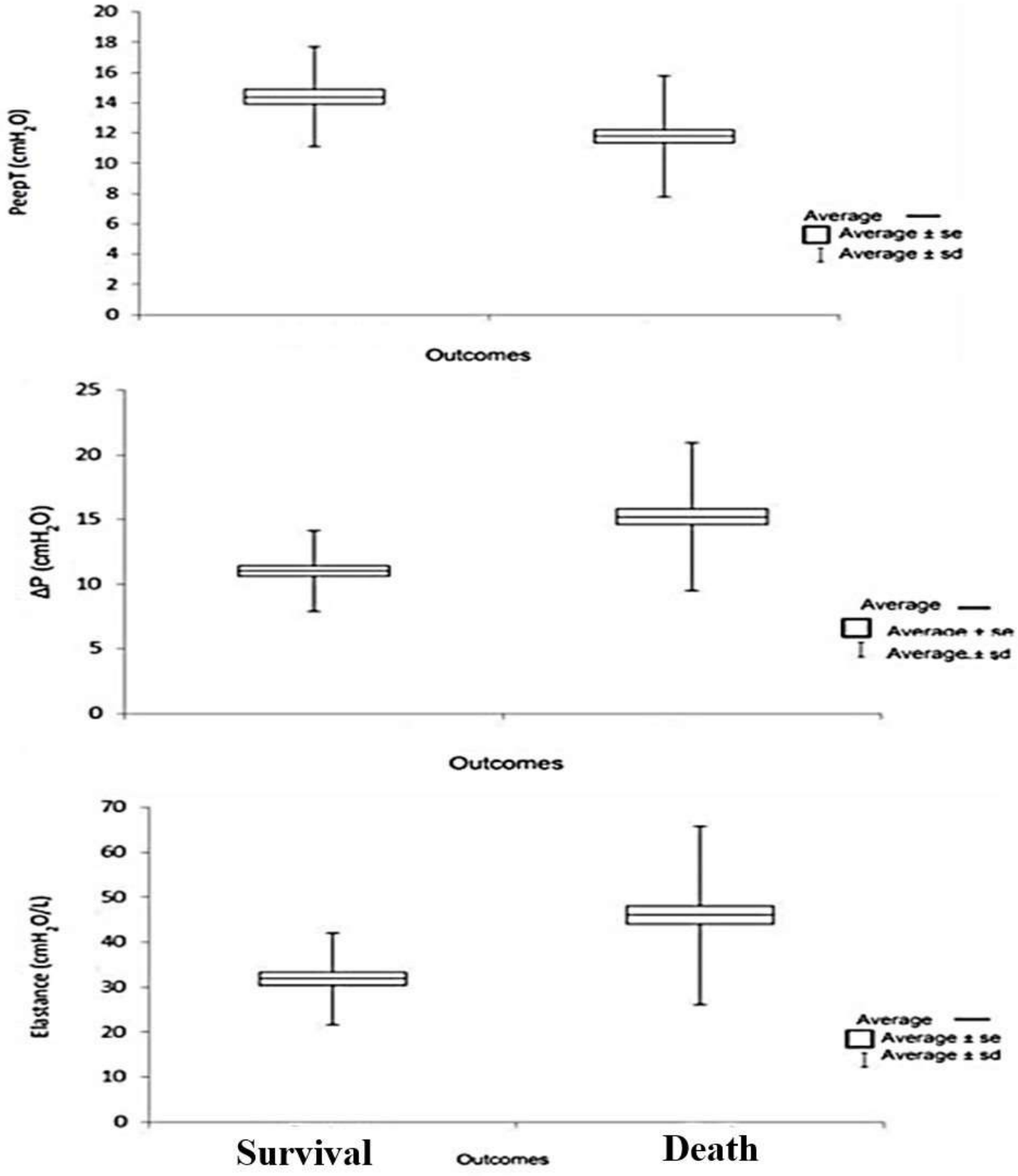

Figure 2: Effect of PEEP, driving pressure and elastance values on outcomes

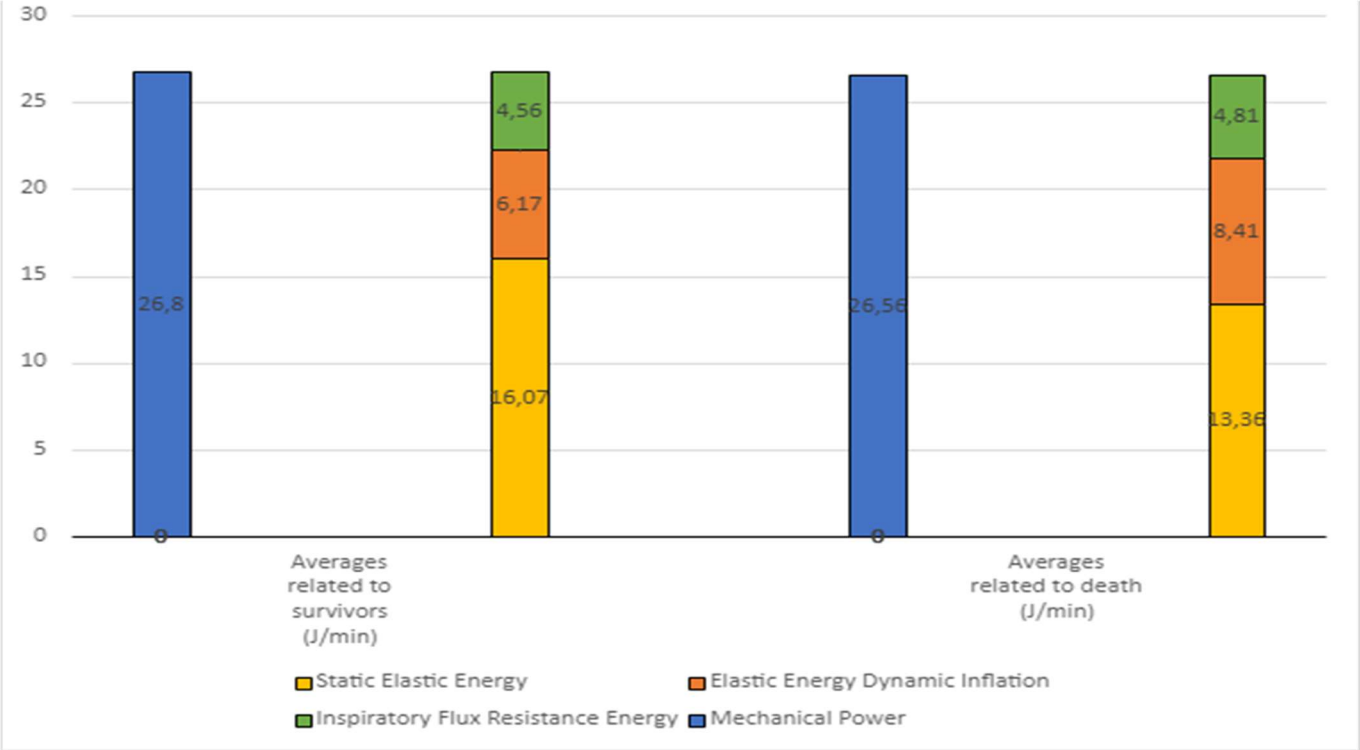

Figure 3: Fragmentation of mechanical power and its effect on outcomes
Blue: Mechanical power, Yellow: Static elastic energy, Orange: Elastic energy dynamic inflation, Green: Inspiratory flux resistance energy

Discussion

A more advanced age seems to consolidate as a factor related to *SARS-CoV-2* death. We noticed a significant statistical difference in the outcome between the average ages of 49.5 and 53.1 years, [23] a similar fact found in this work except for a larger interval in the difference between the average ages, a divergence seemingly related both to an association between survival and average age of 45.7 years as well as between the evolution to death and average age of 55.9 years. In the multivariate analysis it is estimated that each additional year increases the risk of death.

We ranked SARS-CoV-*2* patients' lungs in two phenotypes: high compliance, low elastance and low recruitability to PEEP named *L*; and those with low compliance, high elastance and good recruitability to PEEP named *H* [25-26]. Infection progression can lead to change in phenotype *L* to *H* with high lung weight and extensive pulmonary consolidations. [26]

However, this work found that the model parameters of mechanical ventilation close to the time of death diverged from both categories, presenting high elastance and low recruitability to PEEP possibly related to the non-recoverability vortex of VILI. One can see there was a stronger relation between the risk of death and higher values of elastance and lower values of PEEP in both in the univariate and multivariate analyses.

The normalization of Mechanical Power value as a safety threshold for the prevention of VILI would depend on the knowledge of lung volumes and its distribution, as well as the *Stress* and *Strain* on the pulmonary heterogeneity. [14-27] An elevated Mechanical Power regardless of the combination of its components can lead to *VILI* [13] especially when it exceeds 12*J*/min, whereas over 17*J*/min it is associated with a higher mortality rate [19-28] and between 19 to 24*J*/min denotes the severity of ARDS. [29] There seems to be no influence on mortality between mechanical power and

transpulmonary mechanical power, however the accuracy of values and outcome prediction are more related to the transpulmonary measurement. [19]

This work identified intermediate values of Mechanical Power of 26.71*J*/min, regardless of the evolution time, and 26.60*J*/min when obtained from the last measurements close to the moment of death or withdrawal of NMBA. Despite the high values, the univariate analysis did not show differences in outcome, whereas the multivariate analysis suggests interference in the outcome, further estimating that for every additional unit in mechanical power there is an increase of 13.4% on the mortality risk. The results of this study are limited by being observational and analytical, in which there was no comparison of ventilation strategies and their subsequent results.

The safety threshold for mechanical power values beyond which VILI is inevitable [11-14] and whether a Mechanical Power-based mechanical ventilation strategy can improve patients' clinical status [14] are still unknown as it is unclear the contribution of each ventilation parameter for the generation of such pulmonary injury.

The total inspiratory power, represented by Mechanical Power, was divided into inspiratory flow resistive power, dynamic elastic power and static elastic power. The collected data indicates that the mean dynamic elastic power values was superior, and the mean static elastic power was inferior in the group of patients who died in comparison to the same variables of the group that survived. Hence, the more critical patients needed a greater propulsion force generated by driving pressure to overcome the elastance of the pulmonary tissue, exhibiting low recruitability through PEEP.

However, to set apart a single parameter of mechanical ventilation as the only one responsible for the generation of VILI can be a mistake, as it depends on a combination of factors that bring about the mechanical power [7] and depends on the interaction between these components. [17]

Driving pressure depicts the tidal volume contained by the compliance of the respiratory system in each moment, [5] while elastance refers to the tendency of a material to resist distension or distortion. In this research, age that represents a physiological variable, elastance that depicts the pulmonary condition of strain because of stress also showed significant impact on the mortality of patients with SARS-CoV-2 both in the univariate and in the multivariate analysis.

Conclusion

The increase in age and in Mechanical Power with increased dynamic elastic power and decreased static elastic power influenced the mortality rate of patients with SARS-CoV-2 undergoing mechanical ventilation, i.e. it is related to the increase in driving pressure to overcome a high elastance and low capacity to recruit for PEEP.

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