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Psychological Relationship of Mechanical Ventilation Power on Mortality of Covid-19 Critically Ill Patients

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Abstract
Introduction: ARDS is a type of lung injury that causes inflammation and makes it harder for oxygen to get into the bloodstream. Some treatments have been suggested, such as using less air with each breath, increasing pressure when breathing out, and using special positions or machines to help the lungs. But it's not clear how much these treatments can improve outcomes for patients with ARDS. So far, no studies have looked specifically at whether using more or less mechanical power to help patients breathe affects their chances of survival or other important measures of health.
Objectives: This research aimed to investigate how the use of mechanical ventilation affects the likelihood of death in patients who are severely ill with COVID-19.
Methods: This study adopts a cross-sectional design and retrospective analysis, observing critically ill patients who are being treated in the Special Isolation Ward of Dr. Soetomo Hospital's intensive care unit. The population for this study consists of critically ill patients who meet the inclusion and exclusion criteria. The research sample is obtained through randomized sampling, where all eligible individuals meeting the criteria are included in the sample size.
Results: The study findings reveal a correlation between mechanical ventilation power and mortality among COVID-19 patients with ARDS. The mechanical power of ventilation is identified as a significant variable in this study, with a cut-off point of 17.4. Patients above this cut-off point are at 3.65 times higher risk of death compared to those below it. Moreover, there is evidence of a relationship between the mechanical power of ventilation variable and the P/F Ratio, as a higher mechanical power is associated with a decrease in the P/F Ratio.
Conclusions: The study has identified a correlation between the P/F Ratio variable and mortality in COVID-19 patients with ARDS. On the other hand, there is no evidence of a relationship between the compliance variable and mortality in COVID-19 patients with ARDS.

Keywords: mechanical power, ventilation, critically ill patients.

1. Introduction

Acute respiratory distress syndrome (ARDS) refers to lung injury that makes it hard to breathe. There is a lack of information on how common ARDS is and how often it causes death around the world. A study found that ARDS happens ten times more often in the US than in Europe. Some treatments for ARDS include using lower amounts of air, using a type of air pressure called PEEP, and moving the patient into different positions (Bellani et al., 2016). Mechanical ventilation, which is used to help patients breathe, transfers energy to the lungs. This energy can cause problems by making the lungs inflamed and injured (Serpa Neto et al., 2018).

Ventilator-induced lung injury (VILI) has been studied in both clinical and experimental settings, and several factors have been linked to its pathophysiology. Among these factors, tidal volume (TV) and plateau pressure have received the most attention following strain and stress. Strain refers to the proportional tidal volume in relation to the ventilated lung size, while stress corresponds to the transpulmonary pressure exerted on the lungs when an external force is applied to their structures (Cressoni et al., 2016).

Earlier research suggested that the extent of VILI was associated with the amount of energy transfer, and tidal volume (VT), plateau pressure (Pplat), respiratory rate (RR), and airflow were considered as contributing factors for VILI (Serpa Neto et al., 2018). These factors all contribute to the energy delivered to the respiratory system, which is measured in Joules per minute (J/min) and referred to as mechanical power (MP (Serpa Neto et al., 2018)). Mechanical forces directly act on the extracellular matrix of the lung framework, altering the epithelial and endothelial cells. The extent of lung parenchyma changes, from mechanical breakdown to inflammatory reactions due to the activation of macrophages, neutrophils, endothelial, and epithelial cells, depend on the magnitude of mechanical force (Cressoni et al., 2016).

Mechanical ventilation is sometimes needed to help patients with breathing difficulties, including those with severe COVID-19. However, it can sometimes cause lung injury (VILI). When a patient has VILI, their lungs can become inflamed and filled with fluid, making it even harder for them to breathe. One of the most severe lung conditions that can occur in critically ill patients is ARDS. ARDS can cause widespread inflammation in the lungs, leading to fluid accumulation in the air sacs, which makes it difficult for patients to breathe. VILI can make ARDS worse, as mechanical ventilation can cause more damage to already inflamed lungs. To prevent VILI and reduce the risk of ARDS in patients receiving mechanical ventilation, medical professionals need to be careful to use appropriate pressure and volume levels when providing respiratory support. By using lower pressure and volume levels and avoiding prolonged mechanical ventilation whenever possible, medical professionals can minimize the risk of complications and help their patients recover more quickly.

In clinical practice, Mechanical Power can be determined easily using the "power equation." According to studies conducted on healthy piglets with healthy lungs, an increase in MP during ventilation alongside an increase in RR is associated with an increase in VILI. Despite the extensive research on ventilator-induced lung injury (VILI), there has been limited investigation into the relationship between mechanical ventilation power and outcomes such as mortality in critically ill patients (Serpa Neto et al., 2018).

Therefore, we gathered data on mechanical ventilation and calculated mechanical power using the power equation for two groups of ICU patients whose data was collected retrospectively. Our aim was to investigate whether there is an independent association between ventilation mechanical power and mortality and other outcomes in critically ill patients. The main research question was to explore the relationship between mechanical power of ventilation and mortality in critically ill COVID-19 patients.

2. Literature Review

2.1. Mechanical power

2.1.1. Basic of Mechanical Power

Mechanical ventilation can cause damage to the lungs (VILI). This can be caused by various ventilator settings, including “tidal volume, driving pressure, respiratory rate, inspiratory-to-expiratory ratio, and PEEP.” Mechanical power (MP) can be calculated using several variables from a ventilator, which can help identify the amount of force applied to the lungs during ventilation. This force causes stress and strain to the lungs, which can result in VILI. The extracellular matrix of the lungs can become "fatigued" by cyclic energy loads, leading to damage to capillary walls and inflammation. This can cause extracellular edema due to changes in the extracellular matrix (Ball & Pelosi, 2017).

The volume of air in the lungs that is moved by the ventilator varies due to the cyclic energy load applied to the respiratory system. The energy load is composed of a resistive and inertial component that is generated by various factors such as pressure, surface tension force, and network resistance. The total energy expended during inspiration is calculated by multiplying the pressure applied by the volume change. However, when PEEP is activated, the system is not subjected to any cyclic energy burden because the volume remains constant. PEEP has a complex role in ventilator-provided energy as it provides sustained voltage to the extracellular matrix and accumulates potential energy. Additional energy is required to achieve a target end-inspiratory volume during cyclic tidal ventilation. Therefore, if the total volume of inspiration is the same with and without PEEP, the energy required with PEEP is lower. By calculating energy/power load, it is possible to analyze various phenomena related to VILI (Ball & Pelosi, 2017).

2.1.2. Calculation of Mechanical Power

Direct measurement of volume transfer and pressure changes in lung tissue is crucial in determining mechanical power as an indicator of VILI. However, obtaining accurate measurements of pleural pressure to estimate trans-pulmonary pressure in clinical settings is challenging. As an alternative, esophageal pressure can be used to estimate mechanical power, with values exceeding 12 J/min causing VILI in healthy pigs. Nevertheless, measuring esophageal pressure in a minimally invasive manner is not standardized and can be affected by instability and disturbances, including those caused by the activity of esophageal muscles. Additionally, frequent measurements of esophageal pressure are not commonly performed (Huhle et al., 2018).

2.1.3. Mechanical Power Components

-Respiratory system elasticity

It is generally believed that the pressure-volume relationship is linear in the initial portion of the power equation that pertains to tidal volume and driving pressure. However, if the elastance rises to the point of approaching total lung capacity due to overvoltage, the computed energy may be inadequate. If the elastance falls, for instance because of recruitment, the computed energy will be too high. The total elastance of the lungs may remain unchanged even if overdistention and recruitment occur simultaneously in separate parts of the lungs. Likewise, a change of 10-20% in elasticity (either growing or decreasing) should be reflected as a change of 5--10% in the determined mechanical power (Gattinoni et al., 2016).

-Airway resistance

It is assumed that resistance and flow remain constant during inflation. Once P peak and P plate reach their optimal level, they are sustained until the end of inspiration. As EELV rises during mechanical ventilation, airway resistance declines. In healthy individuals and those with ARDS, the calculated mechanical forces were higher than those observed when ventilation commenced at 15 cmH2O PEEP (Gattinoni et al., 2016).

-Positive end-expiratory pressure (PEEP)

When calculating the work of breathing on a ventilator, the P-V loop always starts at (0,0) regardless of PEEP and end-expiratory lung volume. PEEP does not add to the cyclic energy burden that comes with ventilation, but it does increase the energy provided to the respiratory system by a factor of PEEP multiplied by the change in volume (ΔV). This effect of PEEP is frequently overlooked, despite the fact that a 10-20% increase in PEEP increases mechanical power similarly (Gattinoni et al., 2016).

2.2. Ventilator / Ventilation-induced lung injury (VILI)

The harmful impacts of mechanical ventilation on the lungs are associated with transpulmonary pressure. Intrathoracic pressure is associated with the unfavorable effects of mechanical ventilation on hemodynamics (ie, positive or negative forces that impede or catalyze venous return through various mechanisms). Ventilator lung injury (VILI) is a side effect of mechanical ventilation caused by an excessive and unphysiological increase in transpulmonary pressure (Gattinoni et al., 2016). The injury to the lungs caused by mechanical ventilation, known as VILI (Gattinoni et al., 2016).

2.2.1. Lung Protection

The VILI concept considers the volume and pressure produced by the ventilator. Utilizing a low tidal volume strategy is the most effective method for safeguarding a patient's lungs from volutrauma. In a randomized controlled trial conducted two decades ago, it was observed that mechanical ventilation with low tidal volume decreased mortality rates in patients with ARDS, which was corroborated by a meta-analysis. Another method for safeguarding the lungs is by increasing end expiratory positive pressure to avoid atelectrauma (PEEP). An analysis of patient data revealed that higher PEEP levels decreased mortality rates in patients with mild and severe ARDS. On the other hand, latest randomized controlled studies found that greater PEEP values in patients with mild to severe ARDS were linked with higher mortality rates, increased risk of barotrauma, and prolonged durations of artificial breathing (Huhle et al., 2018).

2.2.2. Factors causing VILI

Numerous clinical and experimental studies have described vent-induced lung injury (VILI), and multiple factors have been proposed to be involved in the pathophysiology of VILI. After strain and stress, the most-studied factors are tidal volume (TV) and plateau pressure. Tidal volume, or strain, is proportional to the lung capacity. When a force is applied to the lungs, the transpulmonary pressure exerted on the lungs is equal to the stress that occurs in the pulmonary structures. Cofactors for VILI include temperature, respiration rate, and flow

(Cressoni et al., 2016). Several variables, including pressure, volume, flow, and respiratory rate, are used in conjunction with VILI to facilitate their administration by the ventilator. However, respiratory diseases such as significant pulmonary edema, which causes decreased lung dimensions, increased lung inhomogeneity, increased stress, and cyclic collapse, can hasten the start of VILI. A higher pressure burden on the ventilator is a trade-off for the benefits of PEEP in reducing lung inhomogeneity and intratidal lung collapse. While most pulmonary VILI causes are primarily the result of pulmonary edema (i.e. severe ARDS), all ventilator-associated VILI causes are components of a physical variable (i.e. mechanical force) (Gattinoni et al., 2016).

Mechanical power, or the energy supplied to the breathing system, is the sum of all these factors. Lung epithelial and endothelial cells are deformed when mechanical forces act directly on the extracellular matrix, the lungs' skeleton. The lung tissue may experience mechanical rupture or inflammatory responses due to the stimulation of macrophages, neutrophils, endothelial, and epithelial cells, depending on the severity of the impact (Cressoni et al., 2016)

2.2.3. VILI mechanism

VILI can be prevented by avoiding excessive strain and transpulmonary pressure on the lungs. In clinical practice, tidal volume per ideal body weight and plateau pressure are commonly used to measure strain and pressure, respectively. Guidelines recommend limiting tidal volumes to 6 mL/kg and plateau pressures to 30 cmH2O. However, other factors such as driving pressure, inspiratory flow, and final expiratory pressure can also contribute to VILI and should be taken into account (Giosa et al., 2019).

2.2.4. VILI and ventilator settings

Mechanical ventilation can cause VILI, which involves two mechanisms: volutrauma and atelectrauma. The amount of energy delivered by a mechanical ventilator, measured as mechanical power, is related to VILI. Lung tissue has viscoelastic properties, which means that both the static and dynamic components of the mechanical ventilator affect the lung tissue. The tidal volume (VT) delivered by the ventilator is a significant factor in VILI, and reducing VT can decrease mortality rates in patients with ARDS. The respiratory rate (RR) affects the rate of stretch on the lung tissue, and atelectrauma can be prevented by increasing positive end-expiratory pressure (PEEP) to a moderate level. A high PEEP level can reduce mortality, but an excessively high PEEP level can increase the risk of VILI (Huhle et al., 2018).

2.2.5. VILI and Mechanical Power

A study conducted retrospectively on 8,207 critically ill patients explored the relationship between Mechanical Power (MP) in the first 48 hours of mechanical ventilation and VILI. The findings of the study suggested that high MP is associated with increased mortality, shorter durations of breathing without mechanical support, and longer stays in the ICU and hospital. The two most influential factors in mortality were found to be the relative risk (RR) and driving pressure (Huhle et al., 2018).

2.2.6. Causes of ventilator-associated VILI

The settings on a ventilator, such as tidal volume, pressure, respiratory rate, and flow, can all contribute to VILI. When a patient is under general anesthesia, the standard ventilator settings may be tidal volume 420 ml, respiratory rate 12 bpm, PEEP 5 cmH2O, and a 1:2 inspiratory to expiratory time ratio. These settings can result in mechanical forces to the respiratory system that are much larger than usual, especially for patients with mild to severe ARDS. For example, the mechanical forces for severe ARDS can be up to 25 times larger than usual, which can increase the risk of VILI (Gattinoni et al., 2016).

2.2.7. Causes of VILI associated with the lungs

There are three aspects that need to be taken into consideration: the lung size, the inhomogeneity, and the recruitment. One way to ensure that mechanical power is distributed evenly across the lung tissue is by normalizing it to lung size. However, in patients with severe ARDS, lung heterogeneity can lead to a significant increase in stress on the lung tissue, which can result in a doubling of the applied pressure. In the lung parenchymal fraction, the mechanical power can be increased by a factor of two (for instance, from 50 mJ/min/ml in the airways to 100 mJ/min/ml). Atelectrauma is at the root of the pathophysiology of VILI in patients who have high recruitment but insufficient PEEP treatment (Gattinoni et al., 2016).

2.3. Acute Respiratory Distress Syndrome (ARDS)

Ashbaugh et al. suggested the classification of ARDS in 1967, and the American-European Consensus Conference (AECC) described it in 1994. The reliability and integrity of this meaning, however, have been

called into question. Rapid start of hypoxemia (PaO2/FIO2 = 200 mm Hg) and bilateral lung infiltrates on X-ray without signs of left atrial hypertension describe ARDS, as defined by the AECC. Acute lung injury (ALI) is diagnosed using the same standards, but mild hypoxemia (PaO2/FIO2 = 300 mm Hg) is allowed (Force et al., 2012).

2.4. COVID-19

The coronavirus, which has particles measuring 120-160 nm in size, is most commonly found in bats and camels. "alphacoronavirus 229E, alphacoronavirus NL63, betacoronavirus OC43, betacoronavirus HKU1, Severe Acute Respiratory Illness Coronavirus (SARS-CoV), and Middle East Respiratory Syndrome Coronavirus (MERS-CoV)" are all coronaviruses that can affect people (Riedel et al., 2019). SARS-CoV-2 mainly spreads between people through respiratory droplets, similar to how influenza is transmitted. The virus can travel up to six feet when an infected person coughs, sneezes, or talks. Studies have shown that the virus can remain viable on surfaces and in the air for several hours (Zhou et al., 2020). Symptoms usually appear within 14 days after exposure, with most cases showing up 4-5 days after being exposed to the virus (Rothan & Byrareddy, 2020)..

The severity of symptoms associated with SARS-CoV-2 can vary based on the level of invasion and inflammatory response. Respiratory distress is typically the initial symptom, as the virus readily targets lung epithelial cells expressing ACE-2 receptors through inhaled droplets.

Damaged infected cells produce cytokines like interleukin-6 (IL-6), interleukin-8 (IL-8), and tumor necrosis factor after the SARS-CoV-2 virus binds to cells abundant in angiotensin converting enzyme 2 receptors. A broad variety of cellular and tissue responses can be induced by IL-6, which is released by excited leukocytes (Pyle et al., 2017). Differentiation of B lymphocytes is aided by this factor, and it also has the ability to drive the development of some cells while suppressing the growth of others (Chen et al., 2020). Thermoregulation, bone health, and brain function are all aided by it as well. IL-6 has dual nature, with both pro- and anti-inflammatory properties (Rose-John, 2018).

The CD8+ cytotoxic T cells are responsible for eliminating virus antigens, while the CD4+ helper T cells stimulate B lymphocytes, which results in the production of antibodies. When T cells become activated and continue to eliminate infectious cells, a cytokine flood is set off. The host may experience anything from minor breathing difficulties to full-blown multi-organ failure and mortality. Vasodilation, greater vascular permeability, hypoxemia, accelerated breathing rate, and acute respiratory distress syndrome have all been linked to these cytokines. Hypercoagulation and the development of capillary microthrombi can result in widespread pulmonary embolism, which can be caused by the heightened inflammatory response that damages the endothelium. The inflammatory process impacts more than just the respiratory system, however; the circulatory system is especially vulnerable. Myocarditis is becoming increasingly common in children and young adults, according to recent reports. It is possible for the cytokine storm to induce systemic vasodilation, which in turn could cause arterial instability and poor peripheral circulation. Renal failure, liver injury manifested by increased liver tests, myocardial infarction, and other problems can all result from inadequate blood flow to the organs.

Viral infections can lead to inflammation in the subpleural region, interstitial edema, and increased vascular permeability. This can cause impaired regional vasoreactivity and vasoplegia, which counteracts hypoxic vasoconstriction, leading to an increase in the shunt fraction. The body's physiological response to hypoxemia is to increase ventilation by increasing tidal volume and respiratory rate. Inflammation, high fever, and increased oxygen consumption can further increase the metabolic boost and respiratory drive (Singer et al., 2016). However, high tidal volumes generated non-invasively can also lead to self-induced lung injury (P-SILI), similar to ventilator-induced lung damage (VILI), causing stress and pressure on the lungs (Möhlenkamp & Thiele, 2020).

High PEEP may not be beneficial for COVID-19 patients because their lungs usually have normal compliance. Mechanical ventilation is necessary if the hypoxemia intensifies and end-organ failure develops. It is advised to use personal protective equipment, prevent bag-mask ventilation, and timely intubation by a qualified operator. In order to achieve a predicted tidal body volume of roughly 8ml/kg body weight, the initial ventilator settings should have a lower beginning PEEP and a higher tidal volume than in severe ARDS. These settings should be 8 cmH2O for starting PEEP and 15 cmH2O for driving pressure. To ensure respiratory system compliance, the

tidal volume must be normalized and the PEEP can be gradually raised as necessary. In order to redistribute pulmonary perfusion and improve ventilation-perfusion, COVID-19 patients typically profit from lying flat for at least 16 hours (Möhlenkamp & Thiele, 2020).

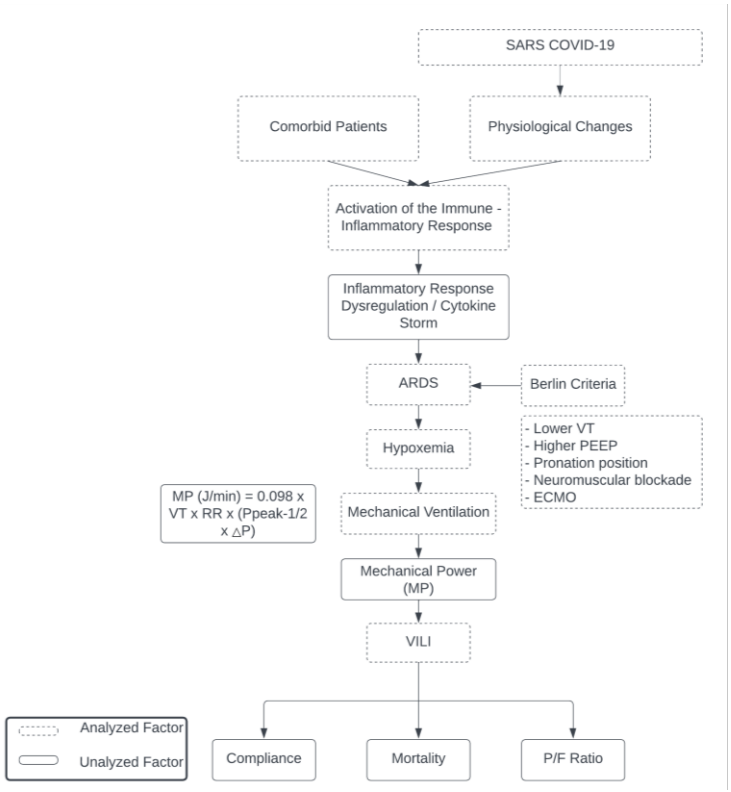

Figure 1 Conceptual Framework

ARDS is "a lung injury that is caused by inflammation and increased permeability in the lungs, leading to increased pulmonary load." The diagnosis is based on specific criteria, such as acute hypoxemic respiratory failure and bilateral chest X-ray or computed tomography. Various interventions have been suggested, including lower tidal volumes, higher PEEP, prone position, neuromuscular blockade, and extracorporeal membrane oxygenation. These interventions aim to improve lung function and oxygenation in patients with ARDS (Bellani et al., 2016).

Each time a mechanical ventilator delivers a breath, it transfers energy to the patient's respiratory system, which is used to increase the volume of the thoracic cavity and overcome airway resistance. This energy affects the extracellular matrix of the lungs, and during each respiratory cycle, a small amount of energy is stored due to the elastic recoil of the lungs. Mechanical ventilation can cause inflammation and heat due to significant energy dissipation, which can potentially result in lung tissue injury (Serpa N et al., 2018).

The two factors that have been most studied regarding strain and stress are tidal volume (TV) and plateau pressure. Strain refers to the tidal volume that corresponds to the lung's ventilated size, while stress is the pressure that develops in the lung structures due to an applied force and equals the transpulmonary pressure on the lungs. The energy generated by a mechanical ventilator is the sum of all these factors, which produce mechanical power expressed as Joules per minute (J/min) (Serpa N et al., 2018). The resulting changes in lung parenchyma can vary from mechanical rupture to inflammatory reactions, depending on the magnitude of the applied force and resulting energy (Cressoni et al., 2016).

3. Methods

This study was a cross-sectional observational analysis with a retrospective design. The primary objective of the research was to establish an association between mechanical power from ventilation and mortality in critically ill patients. This observational study aimed to observe and analyze the relationship between two variables without any intervention on the research subjects. Analytic research seeks to understand the underlying causes and mechanisms of a particular health phenomenon.

This study populations consist of critically ill patients who were receiving treatment in the intensive care unit of Dr. Soetomo Hospital Special Isolation Ward. The researchers used a randomized sampling technique to select patients who met the inclusion and exclusion criteria. To be included in the study, patients had to be over 18 years of age, receiving minimally invasive ventilation continuously for 48 hours, and have no comorbidities. Patients who were ventilated through a tracheostomy cannula during the first 48 hours of ventilation, patients who were extubated or died during the first 48 hours, and patients with incomplete medical records were excluded from the study. This study was retrospective, observational, and analytic, which means that the researchers only made observations without intervening in the patients' treatment, and they aimed to explore how and why health phenomena occur.

Table 1 Operational Definition

No	Variable	Definition	Measuring Tools and Methods	Scale
1	Mechanical power of Ventilation	"Mechanical power is variable, covering all putative causes of ventilator-induced lung injury (VILI): tidal volume, driving pressure (that is, the product of elastance and tidal volume), flow, respiratory rate, and end-expiratory positive pressure."	"In units of J/min in the second 24-hour ventilation. Mechanical Power is calculated using VT, peak pressure (Ppeak), RR, and driving pressure data (ΔP): MP (J/minutes) = 0.098 × VT × RR × (Ppeak−1/2 × ΔP)."	Ratio
2.	Mortality of critically ill patients	"A critical patient confirmed with COVID-19 who died after being admitted to the ICU of Special Isolation Ward"	-	Nominal
3.	Lung Compliance	"The elastance of the lung forces"	"VT/(Ppeak-PEEP)"	Interval
4	P/F Ratio	"Comparison of Oxygen Pressure in Arteries with Oxygen Fraction"	"PaO2/FiO2"	Interval

The study instruments used in this research were obtained from medical records, and the researcher sought ethical approval from the hospital's Health Research Ethics Committee before collecting patient data. The collected data was then recorded and tabulated, and SPSS was used for data analysis. The data were summarized using descriptive statistics such as mean and standard deviation for numerical values and total and percentage for categorical values.

The study presented data on "continuous variables as medians along with interquartile ranges, and categorical variables as totals and percentages." The comparison of proportions was done using Spearman, while t test or Wilcoxon rank sum test was used to compare continuous variables. For the primary analysis, Mechanical Power during the second 24-hour ventilation was treated as a continuous variable. To account for possible confounding factors, multivariable regression was used as the analytical technique for all the results.

Operational Framework

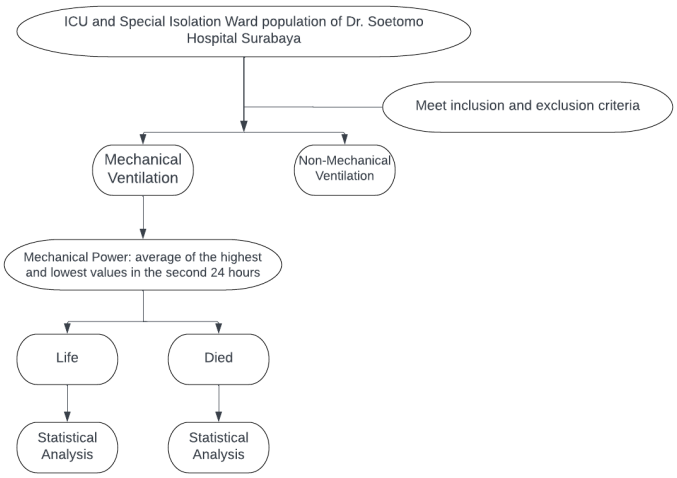

Figure 2 Operational Framework

4. Results

4.1. Characteristics of the Research Sample

Table 2 Description of Covid-19 Critically Ill Patients

Parameter	N (Min – Max)Mean ± SD
Mechanical Power of Ventilation	178 (2,8 – 35,5) 17,4 ± 5,5
P/F Ratio	178 (47 – 586,7) 164,2 ± 97,9
Lung Compliance	178 (5,6 – 123,8) 24,1 ± 14,6

The study included a sample of 178 patients, and among these patients, the average mechanical power of ventilation was found to be 17.4, with the lowest value being 2.8 and the highest being 35.5. The patients' P/F ratio had an average value of 164.2, with a standard deviation of 97.9. In terms of lung compliance, the minimum value was 5.6, and the maximum value was 123.8, with an average of 24.1 and a standard deviation of 14.6.

Table 3 Outcome Description of Covid-19 Critically Ill Patients

Outcomes	Amount	Percentage (%)
Life	65	36,5
Dead	113	63,5
Total	178	100

Out of the total sample (178 people), there were 65 patients who were still alive (36,5%) and patients who had died amounted to 113 (63,5%).

4.2. Data analysis

4.2.1. Data Normality Test

Table 4 Kolmogrof Smirnov Test Results (Data Normality)

Variable	P-Value	
	Life	Die
Mechanical Power of Ventilation	0,000	0,000
P/F Ratio	0,023	0,000
Lung Compliance	0,000	0,000

The results above reveal the P-value which is less than the significance level of α 0.05 or 5%. Therefore, it can be concluded that the tested data is not normally distributed. As a result, the data for the research variables can be utilized for the Spearman correlation test and the Wilcoxon Mann-Whitney different test.

4.2.2. Correlation Test (Spearman)

Table 5 Spearman Test Results

Variable	Correlation Coefficient	P-Value	Information
Mechanical power of ventilation	0.332	0.000	Significant
P/F Ratio	-0.337	0.000	Significant
Lung Compliance	-0.131	0.081	Not significant

From the findings presented in Table 5, the correlation test conducted between the mechanical power of ventilation and patient outcomes (mortality) revealed a p-value of 0.000, which is less than the significance level of 0.05. This result implies that there is a significant association between mechanical power of ventilation and mortality in critically ill patients with Covid-19. The correlation coefficient obtained was 0.332, indicating a positive or unidirectional relationship with a relatively strong correlation between mechanical power of ventilation and patient mortality. This suggests that if the mechanical power of ventilation in a patient is higher, their mortality risk will also increase.

According to the results presented in the analysis, the correlation test conducted between the P/F ratio and patient outcomes (mortality) showed a p-value of 0.000, which is less than the significance level of 0.05. This indicates a significant relationship between the P/F ratio and mortality in critically ill Covid-19 patients. The correlation coefficient obtained was -0.337, indicating a negative or opposite relationship and a relatively strong correlation between the P/F ratio and patient mortality. Therefore, it can be interpreted that if the P/F ratio in patients decreases, the risk of patient mortality will increase.

Based on the findings presented, the correlation test conducted between lung compliance and patient outcomes (mortality) showed a p-value of 0.081, which is greater than the significance level of 0.05. This indicates that there is no significant relationship between lung compliance and mortality in critically ill Covid-19 patients. The correlation coefficient obtained was -0.131, indicating a very weak and negative or opposite relationship between lung compliance and patient mortality. Thus, it can be interpreted that if the lung compliance in patients decreases, there may be an increased risk of patient mortality, but this relationship is very weak and may not be clinically significant.

From the Spearman correlation test above, it can be concluded that there is a relationship between mechanical power of ventilation and P/F ratio with patient mortality so that H0 in both studies is rejected. Meanwhile, there is no relationship between lung compliance and patient mortality, so H0 is accepted.

In this study, we also looking for the relationship between P/F ratio and lung compliance with mechanical power of ventilation was also carried out with the Spearman test.

Table 6 Spearman Test Results

Variable	Correlation coefficient	P-Value	Information
P/F Ratio	-0,299	0,000	Significant
Lung Compliance	0,001	0,988	Not significant

The results of the correlation test between the P/F ratio and mechanical power of ventilation obtained a p-value of 0,000 (p-value <0,05), which means that there is a relationship between the P/F ratio and mechanical power of ventilation in critically ill patients with Covid-19. It can be seen from the table, the value of the correlation coefficient between the relationship between mechanical power of ventilation and the P/F ratio in patients is -0,299 where the relationship is quite strong but has a negative/opposite value. Consequently, it can be interpreted that if the mechanical power of ventilation is higher, the P/F ratio in patients will decrease.

From table 6, the p-value is 0,988 (p-value > 0,05) then H0 is accepted, so there is no relationship between lung compliance and mechanical power of ventilation in critically ill patients with Covid-19. The correlation

coefficient value is 0,001, which means that there is no power to explain the relationship between mechanical power of ventilation and lung compliance in critically ill patients with Covid-19.

4.2.3. Difference Test Analysis (Wilcoxon Mann-Whitney)

Table 7 Wilcoxon Mann-Whitney Test Results

Variable	Z	P-Value	Information
Mechanical power of ventilation	-4,416	0,000	Significant
P/F Ratio	-4,482	0,000	Significant
Lung Compliance	-1,745	0,081	Not significant

Based on table 7, the results of the Wilcoxon Mann-Whitney test above can be seen that the value of the two p-values is 0,000 so it can be concluded that the p-value is <0,05 and H0 is rejected. If H0 is rejected, means that there is a difference between mechanical power of ventilation in Covid-19 critically ill patients with life and death outcomes and there is also a difference between the P/F ratio in Covid-19 critically ill patients with life and death outcomes. In addition, it can also be seen that the p-value of lung compliance is 0,081, so the p-value is > 0,05. In conclusion, H0 namely "there is no difference between lung compliance in critically ill Covid-19 patients with life and death outcomes" was accepted.

5. Discussion

A p-value of 0.000 was found (p-value 0,05) in the correlation test between mechanical power of ventilation and patient outcomes (mortality), indicating that there is "an association between mechanical power of ventilation and the mortality of severely ill patients using Covid-19." Patient mortality is positively and unidirectionally correlated with the use of mechanical ventilation, as measured by a correlation coefficient of 0.332. One possible interpretation of this is that a greater mechanical capacity of breathing in the patient is also associated with a higher mortality rate. There was a 63.5% mortality rate among those who relied on artificial breathing. The mortality rate for individuals diagnosed with ARDS in the same research ranged from 65.7% to 94. (Gibson et al., 2020).

This research found that "a correlation between the P/F ratio and MV in severely ill patients using the Covid-19 formula, with a p-value of 0.000 (p-value 0,05)." The correlation coefficient value between the relationship between mechanical power of ventilation and the P/F ratio in patients is -0,299 where the relationship is quite strong but has a negative/opposite value. Therefore, it is possible to assumed that when the mechanical power of ventilation is increased, the P/F ratio in patients would fall.

In this study, a correlation test was found for pulmonary compliance with patient outcome (mortality) obtained a p-value of 0,081 (p-value > 0,05), means "there is no relationship between pulmonary compliance and mortality in critically ill patients with Covid-19." The correlation coefficient value between the relationship between lung compliance and patient mortality is -0,131 where the relationship is stated to be very weak and has a negative/opposite value. Accordingly, it is possible to noted that patient mortality will increase as lung compliance diminishes. Lung Compliance is an index that can be determined by dividing Tidal Volume by the pressure control offered by a ventilator.

The use of mechanical ventilation is essential in managing critically ill COVID-19 patients with respiratory failure. However, the relationship between mechanical ventilation power and patient mortality is complex and influenced by various factors such as the patient's individual characteristics, disease severity, and ventilation timing and duration. Studies suggest that early mechanical ventilation initiation can reduce mortality in COVID-19 patients with respiratory failure. But prolonged ventilation at higher power (i.e. higher respiratory rate, higher tidal volume, and higher positive end-expiratory pressure) can cause complications such as ventilator-associated lung injury, secondary infections, and barotrauma, which may increase mortality.

Studies have shown that "early initiation of mechanical ventilation in COVID-19 patients with respiratory failure can reduce mortality." Finding the optimal mechanical ventilation strategy for each COVID-19 patient is essential to ensure the best possible outcomes. This requires close monitoring of the patient's response to mechanical ventilation, including blood gas levels, and individualized adjustments of ventilator settings based

on clinical and physiological parameters. In addition to the risk of complications from mechanical ventilation, COVID-19 patients may experience other challenges during their hospitalization, such as acute kidney injury, thromboembolic events, and sepsis. These complications can further complicate the management of mechanical ventilation and impact patient outcomes.

6. Conclusion

The analysis led to several significant findings. Firstly, a relationship between mechanical power of ventilation and Covid-19 patient mortality with ARDS was observed. Secondly, there is a cut-off point at 17.4, beyond which there is a 3.65 times higher risk of mortality. Thirdly, higher mechanical power of ventilation is inversely proportional to the P/F ratio. Fourthly, the P/F ratio is predictive of mortality in Covid-19 patients with ARDS. Lastly, there was no significant association between compliance and patient mortality.

Future studies should address some limitations of the current study. Firstly, the retrospective nature of the study may have introduced selection bias, necessitating future studies with more extensive data collection. Secondly, future studies should include biomarkers such as D-dimer, which can better predict mortality. Finally, a large-scale, prospective study with multiple centers is necessary to investigate the impact of mechanical ventilation power on Covid-19 patients with ARDS. The results of such studies can guide clinical practice and improve patient outcomes.

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