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Mechanical power in mechanical ventilation: Physiologic basis, evidence, and clinical implications

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Abstract

Invasive mechanical ventilation is a cornerstone of supportive care for patients with acute respiratory failure and acute respiratory distress syndrome (ARDS), yet ventilator-induced lung injury (VILI) remains a major contributor to morbidity and mortality. Conventional lung-protective strategies have focused on limiting individual ventilatory variables such as tidal volume, driving pressure, and plateau pressure, but these parameters incompletely capture the cumulative mechanical burden imposed on the lung. Mechanical power has emerged as an integrative concept that quantifies the total energy transferred from the ventilator to the respiratory system per unit time, incorporating tidal volume, airway pressures, inspiratory flow, respiratory rate, and positive end-expiratory pressure.

This narrative review summarizes the physiologic basis of mechanical power, methods for bedside calculation, and current experimental and clinical evidence linking mechanical power to lung injury and patient outcomes. Preclinical studies consistently demonstrate that increasing mechanical power exacerbates structural lung damage and inflammation, even when individual ventilatory variables remain within conventionally accepted ranges. Observational clinical studies across diverse populations including ARDS, acute hypoxemic respiratory failure, and general ICU cohorts have shown robust associations between higher mechanical power and increased mortality, prolonged mechanical ventilation, and longer ICU stay. Reported risk ranges commonly fall between 14 and 18 J/min, although no universal safe threshold has been established.
Important limitations remain. Mechanical power is typically calculated at the level of the respiratory system and does not fully account for heterogeneity in lung size, regional stress distribution, or the fraction of energy dissipated within vulnerable lung units. Normalization to predicted body weight, respiratory system compliance, or aerated lung volume appears to improve prognostic discrimination, supporting a personalized interpretation aligned with the “baby lung” concept. Moreover, emerging data suggest that time-varying and cumulative exposure to high mechanical power may be more relevant than single time-point measurements.

In conclusion, mechanical power provides a coherent, energy-based framework for interpreting ventilatory intensity and VILI risk. At present, it should be viewed as a complementary biomarker rather than a validated therapeutic target. Future research should focus on injury-relevant energy metrics, optimal normalization strategies, and randomized trials testing protocolized mechanical power–guided ventilation strategies.

Keywords: Mechanical power, VILI, ARDS

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Introduction

Invasive mechanical ventilation is an essential life-sustaining therapy for patients with acute respiratory failure and acute respiratory distress syndrome (ARDS). [1] Despite decades of research and the widespread adoption of lung-protective ventilation strategies, mortality among mechanically ventilated patients remains substantial. [2] Since the landmark ARDS Network trial demonstrated improved survival with low tidal volume ventilation, clinical practice has largely focused on limiting static variables such as tidal volume and plateau pressure. [3] However, these parameters alone do not fully capture the complexity of ventilator-induced lung injury (VILI), nor have they led to consistent further reductions in mortality. [4]

VILI is now understood as a multifactorial process resulting from excessive mechanical stress and strain applied to a heterogeneous and vulnerable lung. [5] Traditional concepts such as volutrauma, barotrauma, atelectrauma, rheotrauma, and biotrauma describe distinct but interrelated injury mechanisms. [6] While each has been investigated individually, clinical attempts to mitigate VILI by targeting single ventilatory variables have yielded limited success. [7] This has prompted growing interest in integrative approaches that better reflect the total mechanical burden imposed by the ventilator.

Mechanical power has emerged as one such integrative concept. [8] Defined as the amount of energy transferred from the ventilator to the respiratory system per unit time, mechanical power incorporates tidal volume, airway pressures, inspiratory flow, respiratory rate, and positive end-expiratory pressure (PEEP) into a single metric. [8] By accounting for both static and dynamic components of ventilation, mechanical power provides a unifying physical framework that links ventilator settings to the cumulative energy load delivered to the lung. [8]

Since the original formulation of mechanical power as a ventilator-related determinant of lung injury, a substantial body of experimental and clinical research has accumulated. [9] Animal studies have demonstrated that increasing mechanical power leads to structural lung damage, supporting a causal relationship between energy load and VILI (Ergotrauma). [10] In parallel, observational studies across diverse patient populations, including ARDS, acute hypoxemic respiratory failure, and general ICU cohorts, have consistently reported associations between higher mechanical power and adverse clinical outcomes, particularly mortality. [11] Several studies have suggested risk ranges for mechanical power, most commonly between 14 and 18 J/min, although these thresholds appear to vary according to patient characteristics and ventilatory context. [12,13]

Despite these consistent associations, important uncertainties remain. Mechanical power does not account for heterogeneity in lung size, regional stress distribution, or the fraction of energy that is actually dissipated within vulnerable lung units. [8] Moreover, evidence from randomized clinical trials targeting personalized ventilation strategies, including those based on driving pressure, transpulmonary pressure, imaging, or mechanical power, has not demonstrated clear improvements in mortality. [14,15] These findings highlight the distinction between mechanical power as a severity or prognostic marker and its role as a modifiable therapeutic target.

Accordingly, the clinical relevance of mechanical power continues to be debated. [16] Key questions include whether mechanical power provides information beyond established parameters such as driving pressure, whether normalization to lung size or compliance improves its predictive value, and whether cumulative exposure over time is more important than single-point measurements. Addressing these questions is essential before mechanical power can be incorporated into routine lung-protective ventilation strategies.

In this review, we summarize the physiologic basis of mechanical power, describe methods for its calculation, and synthesize current experimental and clinical evidence linking mechanical power to VILI and patient outcomes. We also discuss the limitations of existing data, the role of normalization and time-varying exposure, and the implications for future research aimed at translating this concept into safe and effective clinical practice.

Conceptual and physiologic basis of mechanical power

Work, Energy, and Power in Respiratory Mechanics

In respiratory mechanics, work refers to the force (pressure) applied to move a volume of gas, whereas energy (Joules) represents the cumulative work performed during a single breath. Power is the rate at which this energy is delivered over time (Joules/minute). [4] In the context of mechanical ventilation, mechanical power quantifies the energy transferred from the ventilator to the respiratory system per unit time and thus incorporates both the magnitude of each breath and the frequency at which breaths are delivered. [7]

From a physical perspective, the energy delivered during a tidal breath corresponds to the area enclosed by the inspiratory limb of the airway pressure–volume curve. [1] Mechanical power is obtained by multiplying this energy per breath by the respiratory rate. [4] This formulation emphasizes that lung injury risk is not determined solely by the size of individual breaths or peak pressures, but also by how often the lung is exposed to these mechanical loads. [4]

This energy-based framework provides a unifying interpretation of VILI. [17] Parameters traditionally considered independently such as tidal volume, airway pressure, inspiratory flow, respiratory rate, and PEEP, which are all contributors to the total mechanical energy applied to the respiratory system. [7] Mechanical power therefore represents a summary measure of ventilatory intensity rather than a novel injurious mechanism. [4]

Respiratory system versus lung: Where the energy is spent

Mechanical power is typically calculated at the level of the respiratory system, which includes both the lung parenchyma and the chest wall. [18] The total energy delivered by the ventilator is partitioned into elastic and resistive components. [7] The elastic component reflects energy required to overcome the elastance of the respiratory system and to increase lung volume above its resting state. This energy is stored transiently as elastic potential energy and is, in principle, recoverable during expiration. The resistive component reflects energy dissipated as gas flows through the airways and is largely lost as heat.

The elastic component can be further subdivided into elastic static and elastic dynamic components. Elastic static work corresponds to the energy required to raise the respiratory system from zero end-expiratory pressure to the set PEEP level. This energy is stored in the respiratory system at end-expiration and is present even before tidal inflation begins. Elastic dynamic work, in contrast, represents the additional energy required to inflate the lung from PEEP to the end-inspiratory volume (i.e., tidal inflation). This portion corresponds to the driving pressure component of the pressure–volume relationship and is closely linked to tidal volume and compliance. In geometric terms, elastic static work is represented by the rectangular area related to PEEP on the pressure–volume curve, whereas elastic dynamic work corresponds to the triangular area associated with tidal expansion above PEEP. Consequently, combining the resistive and elastic dynamic (tidal) work, we get the term: inspiratory work (Figure 1). [19,20]

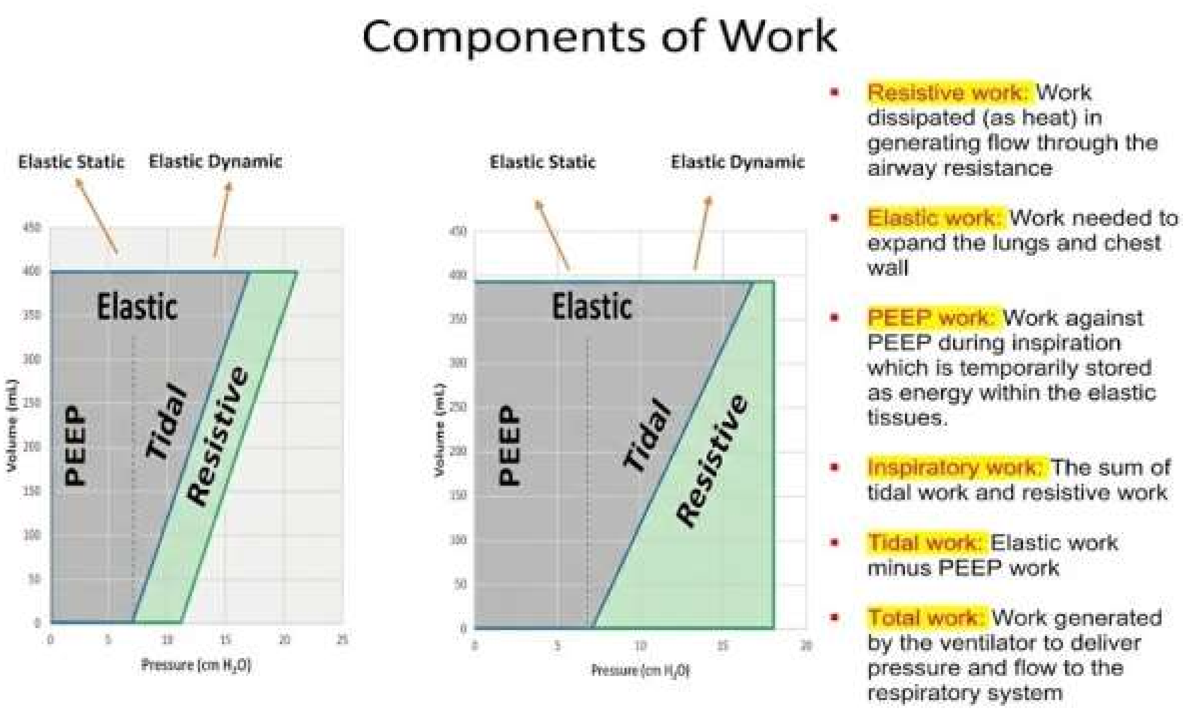

Figure 1: Total work consists of elastic work (elastic static [PEEP work] + elastic dynamic [tidal work]) and resistive work. Elastic static work represents the energy required to maintain PEEP, elastic dynamic work corresponds to tidal inflation above PEEP, and resistive work reflects energy dissipated through airway resistance.

Importantly, only a fraction of the total mechanical power delivered to the respiratory system is directly relevant to lung injury. [21] Energy spent inflating the chest wall or dissipated through airway resistance does not necessarily translate into parenchymal damage. This distinction is particularly relevant in patients with altered chest wall mechanics, such as obesity, abdominal hypertension, or increased pleural pressure, where airway pressures may substantially overestimate the energy applied to the lung itself. [22]

Furthermore, lung injury does not depend on global energy delivery alone, but on how that energy is distributed within a heterogeneous lung. In conditions such as ARDS, functional lung size is markedly reduced, and energy delivered by the ventilator is concentrated within a smaller fraction of aerated tissue. [23] As a result, identical levels of respiratory system mechanical power may impose very different mechanical burdens at the level of the lung parenchyma. [24]

Energy dissipation and the concept of power intensity

A critical refinement of the mechanical power concept lies in distinguishing between energy that is merely delivered to the respiratory system and energy that is dissipated within lung tissue.[24] Tissue injury is associated with irreversible energy dissipation, particularly from dynamic deformation and repetitive opening–closing phenomena, rather than from elastic static energy (PEEP-related energy) that is stored and largely recovered during expiration.

Several mechanisms contribute to energy dissipation in the lung, including viscoelastic deformation of tissue, friction between structural elements, and repetitive opening and closing of unstable alveolar units. [25] While some degree of energy dissipation occurs even in healthy lungs, the rate and localization of dissipation appear to be key determinants of injury. This has led to the concept of power intensity, which describes the amount of dissipated energy per unit time delivered to a given volume of lung tissue.

From this perspective, global mechanical power may be a necessary but insufficient descriptor of VILI risk. Injury is more likely when energy is dissipated rapidly and locally within vulnerable regions, such as areas adjacent to collapsed or edematous lung units. This framework helps explain why mechanical power thresholds vary across studies and patient populations, and why normalization of mechanical power to lung size or compliance often improves its association with clinical outcomes. [9]

Taken together, these physiologic considerations highlight that mechanical power should be interpreted as a surrogate for the intensity of mechanical stress applied to the lung, rather than as a direct measure of tissue injury. [24] Understanding where energy is spent and how it is dissipated provides essential context for interpreting associations between mechanical power and outcomes and underscores the need for cautious translation of this concept into clinical practice.

Calculation of mechanical power at the bedside

Standard equations

In volume-controlled ventilation with constant inspiratory flow, mechanical power can be derived from the equation of motion of the respiratory system as the energy delivered per breath multiplied by respiratory rate. This formulation integrates elastic work (related to tidal volume and driving pressure), resistive work (related to inspiratory flow and airway resistance), and the baseline energy associated with PEEP all of which are known to contribute to VILI risk. Mechanical power therefore provides the most complete estimate of ventilatory energy load received by the respiratory system.

The first, most comprehensive, equation for mechanical power was proposed and validated against the geometric method in both healthy and diseased patients by Gattinoni et al. [8] This is calculated in Joules per minute (J/min) as follows:

$$0.098 \cdot RR \cdot \left\{\Delta V^2 \cdot \left[0.5 \cdot ELrs + RR \cdot \frac{(1 + I:E)}{(60 \cdot I:E)} \cdot Raw\right] + \Delta V \cdot PEEP\right\}$$

where ΔV is tidal volume, ELrs is the elastance of the respiratory system, I:E is the inspiratory to expiratory ratio, and Raw is airway resistance.

For practical bedside application in volume-controlled ventilation (VCV) with constant flow, a simplified linear equation derived from the equation of motion can be used:

$$0.098 \text{ x RR x VT x } (Ppeak - (Pplat - PEEP)\ 2)$$

This formulation assumes constant inspiratory flow and incorporates both elastic and resistive components without requiring full geometric integration. The constant 0.098 converts cmH_2O·L/min into Joules/min.

For pressure-controlled ventilation (PCV), Becher et al. proposed a simplified equation:

$$0.098 \text{ x RR x VT x } (\Delta Pinsp + PEEP)$$

where ΔPinsp represents the set inspiratory pressure above PEEP.

This formulation approximates the square pressure waveform typical of PCV and allows estimation of mechanical power without inspiratory hold maneuvers. [26]

Approximations and simplified formulas

While comprehensive in accounting for both resistive and elastic components of mechanical power, the "standard" equation proposed by Gattinoni et al. requires an inspiratory hold to distinguish between these elements. Therefore, its utility in both clinical practice and research may be limited. Various groups have proposed simplified, bedside calculations with readily available parameters - such as tidal volume, peak or plateau pressure, and respiratory rate.

The primary example comes from Giosa et al. [27] which omitted direct measurement of airway resistance and elastance by assuming a mean total respiratory system resistance in mechanically ventilated patients of 10 cmH_20/L/sec. Termed the "Surrogate" equation, this calculation does not require an inspiratory hold and only uses variables that are readily displayed on modern ventilators (i.e. minute ventilation, peak pressure, and PEEP). It can therefore be calculated on a breath-to-breath basis. Follow-up validation studies have found this to correlate strongly with the geometric measurement of mechanical power with limits of agreement within 2 J/min. [27,28] The surrogate equation is displayed as follows:

$$\frac{VE \bullet (Peak\ Pressure\ +\ PEEP\ +\ \frac{F}{6})}{20}$$

where VE is minute ventilation (L/min) and F is inspiratory flow rate (L/min). Peak Pressure and PEEP are both measured in centimeters of water (cmH_2O).

More recently, a universal surrogate equation termed the mean airway pressure–minute ventilation product (mM) has been proposed as a mode-independent estimate of mechanical power.

$$mM = \bar{P}aw \times VE$$

where $\bar{P}aw$ is mean airway pressure and VE is minute ventilation.

In a large simulation study including 2,000 data points per mode, the mM equation demonstrated strong correlation with geometrically derived mechanical power in both VCV and PCV ($R^2 \approx 0.93$). [29,30] Subsequent evaluation during spontaneous breathing conditions showed preserved correlation trends, supporting its applicability beyond passive ventilation. [29]

This approach offers the advantage of simplicity, universality across modes, and the ability to be calculated directly from ventilator-displayed parameters without inspiratory hold or measurement of resistance.

Practical Measurement Issues

Even when simplified, determining mechanical power requires additional calculations that may limit its practicality, applicability, and generalizability. Firstly, the use of mechanical power as a predictive tool remains of unclear benefit compared to more conventional approaches. Secondly, all calculations are based on a number of assumptions including controlled ventilation settings with the absence of spontaneous breathing, constant flow patterns, and linear respiratory system mechanics. This only includes a specific subset of patients who meet these criteria and may affect its clinical accuracy in others. Third, airway pressures reflect the combined properties of the lung and chest wall, which may overestimate lung-specific energy delivery in patients with altered chest wall mechanics. Finally, current equations provide only single time-point measurements, whereas cumulative and time-varying mechanical power appears more relevant to development of lung injury and worsening clinical outcomes.

Recently, studies have shown that MP can be calculated automatically in real time. [31] New generation ventilators can integrate software that directly measures the MP using the gold standard geometrical method in real time.

Determinants of mechanical power

Mechanical power is determined by the integrated effects of tidal volume, driving pressure, inspiratory flow, respiratory rate, and PEEP. [8] Each variable contributes through distinct elastic and resistive mechanisms, and their interaction defines the overall intensity of mechanical ventilation rather than any single setting alone. [4]

Tidal volume and driving pressure are dominant determinants of mechanical power because they govern the elastic work of ventilation. [4] Importantly, their contribution is nonlinear: incremental increases in tidal volume or driving pressure result in disproportionately larger increases in delivered energy. [8] This effect is magnified in conditions such as ARDS, where ventilation is confined to a reduced fraction of normally aerated lung (the so-called "baby lung") leading to higher regional stress and strain for a given global tidal volume. [8,23] Consistent with this concept, clinical outcomes are more closely associated with driving pressure than with tidal volume alone, underscoring the central role of lung size adjusted deformation in ventilator-induced lung injury. [32]

Respiratory rate influences mechanical power by determining how frequently energy is delivered to the lung. Even when per-breath energy is modest, high respiratory rates can substantially increase cumulative energy exposure over time. [4] This highlights that the risk of ventilator-induced lung injury depends not only on the magnitude of stress and strain per breath, but also on their repetition, integrating both intensity and duration of ventilation into a single conceptual framework.

Inspiratory flow and flow waveform primarily affect the resistive component of mechanical power. Higher inspiratory flow rates increase resistive pressure losses across the airways and endotracheal tube, thereby increasing the energy dissipated within the respiratory system. [33] Moreover, unfavorable flow profiles may augment dynamic lung stress during inflation, even in the absence of changes in static airway pressures, suggesting that flow-related factors can modulate lung injury risk independently of tidal volume and plateau pressure (Rheotrauma).

PEEP is incorporated into most mechanical power equations as a linear additive component, reflecting the higher baseline pressure at which tidal ventilation occurs. [8] However, whether the static elastic component associated with PEEP should be included in mechanical power calculations remains debated.

From a physical perspective, PEEP represents stored elastic energy that does not involve additional dynamic volume displacement during tidal inflation. Accordingly, some authors have argued that the component of elastic work mathematically represented as PEEP × tidal volume (PEEP × VT) reflects baseline elastic energy rather than additional dynamic work during tidal inflation and may lead to an overestimation of injurious energy exposure. [34] Furthermore, inclusion of PEEP as a linear contributor to mechanical power is inconsistent with clinical observations suggesting a U-shaped relationship between PEEP and VILI risk.

Nevertheless, PEEP modifies lung mechanics by influencing recruitment, stress distribution, and functional lung size. Thus, although the static elastic component may not directly represent dissipated energy, PEEP indirectly affects the dynamic stresses that contribute to lung injury. Mechanical power therefore captures exposure at the respiratory system level but may not fully reflect the biological consequences of static versus dynamic energy application. [35,36]

Collectively, these determinants emphasize that mechanical power reflects the global burden of ventilatory energy applied to the lungs. Similar levels of mechanical power can arise from different combinations of ventilatory variables, potentially leading to different patterns of regional stress, strain, and injury. Understanding the individual and interactive contributions of these determinants is therefore essential for interpreting mechanical power in both experimental and clinical contexts.

Preclinical Evidence: Mechanical Power and Ventilator-Induced Lung Injury

Experimental models and bench studies provide the strongest support for the use of mechanical power as a predictor of VILI to date. In animal models, incrementally increasing mechanical power has consistently resulted in lung injury based on biochemical (eg. CXCR3/CXCL10, MMP-2) and radiologic evidence of inflammation, histologic findings of lung damage, and impaired gas exchange. [37,38] Of note, lung damage occurs even when individual ventilatory variables remain within conventionally "acceptable" ranges, which suggests that the use of traditional, static parameters alone may not provide a complete assessment of the pressures experienced by the lungs. Thus, by integrating multiple contributing factors, these studies suggest that mechanical power may provide greater utility for clinicians to predict or even prevent onset of VILI.

These findings are further validated by studies in human patients who are mechanically ventilated with ARDS, which provides evidence of both biochemical and histologic lung damage associated with mechanical power. Xie et al. conducted a prospective observational study in 95 patients with ARDS, which found a statistically significant, positive correlation between mechanical power and serum TGF-ß and CTGF. [37] Mechanical power was also found to be significantly higher in the group who developed pulmonary fibrosis. In a study of 68 ARDS patients undergoing open lung biopsy, Li et al. similarly noted that increasing mechanical power was associated with increased risk for histologic fibrosis (OR 1.493, 95% CI 1.014-2.200, P = 0.042). [39] However, it is likely that more experimental studies are required to further elucidate any physical and biochemical mechanism by which mechanical power may precipitate lung injury.

Many studies have additionally attempted to identify a specific mechanical power "threshold", above which lung injury becomes more likely. This threshold is often set in the range of approximately 12-13 J/min in healthy or mildly injured lungs, as evidenced by previous animal models. [10,40] Specifically, a study of 36 healthy piglets found that a mechanical power threshold of 13±1 J/min statistically discriminated between less vs. more severe lung injury and hemodynamic impairment.[40] However, another study in healthy pigs found evidence of VILI regardless of mechanical power, even at the lowest 3 J/min for 48 hours. [41]

Preclinical and clinical evidence is therefore insufficient to support the use of a specific power threshold. In addition, measurements of mechanical power vary substantially across models, experimental conditions, and methodology. All these factors, combined with differences in severity of lung injury, duration of ventilation, flow patterns, and species ultimately limit the generalizability of any single, safe threshold. Further studies will be required to normalize actual mechanical power to "expected" mechanical power based on variables such as body mass, functional lung size, lung condition, etc.

Overall, experiments in animal models provide invaluable and well-controlled information, which are often unattainable and unethical through other methods. They are, however, intrinsically limited by anatomic and physiologic differences in lung structure, breathing mechanics, and inflammatory responses to injury. Many studies are further conducted in healthy animal models or with acute lung injury induced via lavage, which are both imperfect reflections of the lung conditions in patients with ARDS. Therefore, their findings are primarily useful to guide further research regarding the importance of mechanical power in determining or preventing VILI.

Clinical evidence: Associations with outcomes

General evidence linking mechanical power to clinical outcomes

The largest systematic review to date, enrolling 46 studies and 314,823 patients with ARDS, demonstrated that 87% of included studies reported a significant association between higher mechanical power and worse clinical outcomes. These outcomes included increased mortality, prolonged duration of mechanical ventilation, and longer ICU length of stay. Importantly, the review consistently identified threshold effects between 14 and 18 J/min, suggesting a clinically relevant range above which the risk of ventilator-induced lung injury (VILI) increases substantially. [9]

Earlier evidence from large observational cohorts further supports these observations. An analysis of 8,207 mechanically ventilated patients from the MIMIC-III and eICU databases demonstrated a progressive increase in mortality risk when mechanical power exceeded 17 J/min. Each 5 J/min increase in mechanical power was independently associated with higher in-hospital mortality (OR 1.06, 95% CI 1.01–1.11, $P = 0.021$; OR 1.10, 95% CI 1.02–1.18, $P = 0.010$, respectively). Notably, this association remained significant even among patients receiving low tidal volume ventilation, underscoring that mechanical power captures injurious mechanisms not fully reflected by individual ventilator parameters alone. [11]

In non-ARDS ICU patients, an individual patient data meta-analysis of three randomized controlled trials including 1,962 patients confirmed that mechanical power was independently associated with 28-day mortality (HR 1.13, 95% CI 1.05–1.22, $P < 0.001$), with hazard ratios increasing progressively across rising mechanical power levels. These associations persisted even after stratification by individual mechanical power components (tidal volume, airway pressure, and respiratory rate), suggesting that mechanical power provides incremental prognostic information beyond its individual components. [42]

Importance of normalizing mechanical power

Emerging evidence indicates that normalization of mechanical power to the lung size or respiratory mechanics may improve its prognostic performance compared with absolute mechanical power alone. Large contemporary cohorts of patients with ARDS, including analyses focused on pressure-controlled ventilation, consistently support mechanical power as an independent prognostic marker. In a study by Zhang et all, normalized mechanical power showed superior discrimination for mortality compared with absolute mechanical power, and was independently associated with increased mortality in patients with moderate to severe ARDS (OR 1.11, 95% CI 1.02–1.23, $P = 0.021$; OR 1.13, 95% CI 1.03–1.24, $P < 0.008$, respectively), but not in mild ARDS (OR 0.99, 95% CI 0.91–1.07, $P = 0.862$). However, the magnitude of association and the optimal formulation of mechanical power appear to vary depending on patient population and ventilatory mode. [43]

Coppola et al. also provided pivotal evidence in a cohort of 222 patients with ARDS, demonstrating that mechanical power normalized to respiratory system compliance and mechanical power normalized to well-inflated lung tissue were independently associated with ICU mortality (RR 2.69, 95% CI 1.10–6.56, $P = 0.029$; RR 1.79, 95% CI 1.16–2.76, $P = 0.008$, respectively), whereas absolute mechanical power was not. These findings highlight the importance of accounting for the functional lung size receiving the applied mechanical energy. [14]

More recently, Khorasanee et al. validated these concepts in a large cohort of 3,578 mechanically ventilated patients. In this study, mechanical power normalized to compliance and the mechanical power ratio both demonstrated superior discrimination for ICU mortality (AUROC 0.71 for both) compared with absolute mechanical power (AUROC 0.69). These results reinforce the notion that normalization enhances the clinical utility of mechanical power for risk stratification. [44]

Time-varying and cumulative mechanical power

Beyond static measurements, several studies have highlighted the importance of time-varying and cumulative exposure to mechanical power. A retrospective cohort study in patients with ARDS employed Bayesian joint models to assess time-varying intensity of mechanical ventilation and demonstrated that both time-varying static driving pressure (HR 1.03, 95% CI 1.01–1.05, P < 0.001) and mechanical power (HR 1.01, 95% CI 1.002–1.02, P = 0.01) were significantly associated with 28-day mortality in patients with early negative fluid balance. [45]

Similarly, a retrospective observational study in patients with COVID-19–related ARDS evaluated mechanical power over the first four calendar days of ventilation and demonstrated that each increment in mechanical power was associated with an increased hazard of death (HR 1.12, 95% CI 1.01–1.36, P = 0.018). Importantly, sensitivity analyses showed that greater cumulative exposure to high mechanical power was associated with higher 28-day mortality. This finding supports the concept that total mechanical energy delivered over time, rather than a single time-point measurement, contributes to ventilator-associated lung injury. [46] The summary of the current evidence is shown in Table 1.

Normalization and personalization

The same absolute mechanical power does not imply the same biological risk because energy delivery is distributed across lungs that differ markedly in size, recruitability, and mechanical properties. [43,47] In injured lungs with reduced functional volume ("baby lung"), a given mechanical power is concentrated within a smaller amount of aerated tissue, increasing local stress and strain. [23,48] Chest wall mechanics further modulate how airway-level mechanical power translates to lung-level loading, contributing to interpatient heterogeneity. [49,50]

To address this variability, several normalization approaches have been proposed. Normalizing mechanical power to predicted body weight (PBW) provides a simple surrogate for lung size and has strengthened associations with outcomes in some cohorts. [43] Adjustment to respiratory system compliance or aerated lung volume (from imaging or surrogates) more directly reflects functional lung size and often improves risk discrimination. [44,48] Transpulmonary pressure–based formulations aim to isolate lung specific loading from chest wall effects, but their routine use is limited by measurement complexity. [50,51]

Whether normalized mechanical power offers incremental predictive value beyond established metrics remains an open question. [16] Several analyses show that mechanical power correlates closely with driving pressure and respiratory rate, and in some datasets simplified models perform similarly. [44] However, normalization, particularly to lung size or compliance, appears to recover prognostic information not captured by plateau or driving pressure alone. In a retrospective study, it found that mechanical power normalized to compliance and MP_{ratio} had better discrimination for ICU mortality than standard mechanical power. [44] This supports a personalized, context-dependent interpretation of ventilatory risk rather than reliance on a single global cutoff.

Clinical application: Toward mechanical power–guided ventilation

As an integrated measure of ventilatory intensity over time, mechanical power–guided ventilation has been proposed as a more nuanced extension of current lung-protective ventilation strategies. For bedside implementation, however, a standardized framework defining which ventilator parameters are actively adjusted versus held constant is required. Conceptually, mechanical power may be reduced by lowering tidal volume and driving pressure (elastic load), decreasing respiratory rate (exposure frequency), moderating inspiratory flow or waveform (resistive and dynamic components), and avoiding unnecessary escalation of PEEP (baseline pressure–related energy).

Among these variables, respiratory rate warrants particular attention. Because mechanical power is fundamentally a time-dependent construct, respiratory rate directly governs the repetition of energy transfer to lung tissue. Emerging data suggest that respiratory rate may represent the most clinically relevant dynamic determinant of cumulative energy exposure. In a retrospective observational study employing Bayesian model averaging in patients with severe ARDS, respiratory rate was the only variable consistently associated with

Table1. Summary and characteristics of latest studies for treatment

Author / Year	Designs	Population	Key findings	Statistical estimates
Urabankowski et al. [9]	Systematic review	ARDS	40 studies reported that higher MP was associated with (1) increased mortality, (2) longer duration of mechanical ventilation, and (3) prolonged ICU stay	NA
Serpa Neto et al. [11]	Retrospective cohort	mechanically ventilated patients	Mortality risk increased progressively when MP exceeded 17 J/min and OR per 5 J/min increased.	OR 1.06, 95% CI 1.01–1.11, P = 0.021 (MIMIC-III) OR 1.10, 95% CI 1.02–1.18, P = 0.010 (eICU)
Van Meene et al. [42]	Meta-analysis	Non-ARDS	MP was independently associated with 28-day mortality. Other individual ventilatory components stratified for mechanical power were not associated with mortality.	HR 1.13, 95% CI 1.05–1.22, P < 0.001
Zhang et al, [43]	IPD meta-analysis	ARDS	Normalized MP showed superior discrimination for mortality compared with absolute MP. Normalized MP was independently associated with increased mortality in moderate to severe ARDS	OR 1.11, 95% CI 1.02–1.23, P = 0.021 (moderate ARDS) OR 1.13, 95% CI 1.03–1.24, P < 0.008 (severe ARDS)
Coppola et al. [14]	Prospective cohort	ARDS	MP normalized to well-inflated tissue and to respiratory system compliance were independently associated with ICU mortality, whereas absolute MP was not.	RR 2.69, 95% CI 1.10–6.56, P = 0.029 (well-inflated tissue) RR 1.79, 95% CI 1.16–2.76, P = 0.008 (respiratory system compliance)
Khorasanee et al. [44]	Retrospective cohort	mechanically ventilated patients	MP normalized to compliance and the MP ratio showed superior discrimination for ICU mortality compared with absolute MP.	AUROC 0.71, 95% CI 0.69-0.73, P = 0.007 (MP normalized to compliance) AUROC 0.71, 95% CI 0.68-0.73, P = 0.0014 (MP ratio) AUROC 0.69, 95% CI 0.66-0.71 (MP alone)
Hu et al. [45]	Retrospective cohort	ARDS	Time-varying MP was independently associated with 28-day mortality.	HR 1.03, 95% CI 1.01–1.05, P <0.001
Schuijt et al. [46]	Retrospective cohort	COVID-19 ARDS	Higher cumulative MP exposure was associated with increased 28-day mortality.	HR 1.12, 95% CI 1.01–1.36, P =0.018

multiple mechanical power formulations across time points.[52] These findings support the concept of "cyclic energy," whereby repetitive stress application may contribute to tissue injury even when per-breath stress remains within conventionally acceptable limits.

In practice, early reduction in ΔP through tidal volume adjustment or treatment of low-compliance states, together with avoidance of excessive respiratory rates, appears to provide the most consistent mitigation of cumulative ventilatory exposure. This perspective aligns with principles of material fatigue, in which repetition of submaximal stress may ultimately determine structural failure.

Nevertheless, reduction of mechanical power should not be viewed as a singular therapeutic objective, given important physiologic tradeoffs across disease states. Though associated with higher mechanical power, hyperventilation may be required in the setting of hypercapnia or metabolic acidosis. Current lung-protective strategies already prioritize low tidal volumes with acceptance of permissive hypercapnia, reflecting the same tradeoff between gas exchange and lung protection central to mechanical power-guided approaches.[3,53] Similarly, PEEP titration should balance alveolar recruitment against overdistension rather than be guided by mechanical power alone. Although increases in PEEP contribute to mechanical power, they may simultaneously reduce atelectrauma by stabilizing recruited lung units. Hemodynamic compromise, particularly at higher mean airway pressures, may ultimately limit how aggressively mechanical power can be reduced.

Patient effort introduces additional complexity. Standard mechanical power formulations assume passive conditions, limiting applicability in assisted ventilation modes. Vigorous spontaneous effort may substantially increase transpulmonary pressures and regional stress despite apparently acceptable ventilator-derived mechanical power, raising concern for patient self-inflicted lung injury (P-SILI). Consequently, mechanical power-guided ventilation is most straightforward in controlled modes and may, in select cases, require deep sedation or neuromuscular blockade.

Nevertheless, emerging data suggest that estimation of mechanical power during spontaneous effort is feasible using simplified surrogate equations. In a recent simulation-based evaluation, the mean airway pressure–minute ventilation (mM) equation demonstrated very strong correlation with total and ventilator mechanical power in pressure-controlled ventilation, even in the presence of spontaneous respiratory effort, though correlation with patient-generated power was weaker in volume-controlled modes. [29] These findings indicate that mechanical power can be estimated under assisted conditions, but accuracy may depend on ventilatory mode and the magnitude of patient effort.

Extension of mechanical power-guided strategies into assisted ventilation therefore necessitates careful titration of support based on assessments of patient effort, including esophageal pressure monitoring, occlusion maneuvers, or detailed ventilator waveform analysis. Further clinical validation is required before mechanical power can be reliably used as a therapeutic target in spontaneously breathing patients.

Additional implementation barriers include heterogeneity in mechanical power calculation across ventilatory modes, dependence on assumptions regarding flow profiles and resistance, and the limitation that airway-derived mechanical power reflects the respiratory system rather than lung parenchyma alone. These constraints highlight the potential need for adjunctive physiologic monitoring modalities to guide ventilator adjustments. In a recent randomized crossover pilot trial by Jimenez et al., electrical impedance tomography (EIT) guided PEEP titration consistently reduced mechanical power in ventilated patients with moderate-severe ARDS compared with the use of a standard high-PEEP/FiO_2 table. [54]

Knowledge gaps and future research

What RCTs Should Test

Most of the current evidence linking mechanical power to adverse outcomes is observational, and therefore cannot establish whether mechanical power is a causal, modifiable driver of harm or primarily a marker of disease severity and ventilatory requirements. [12,24] Large cohorts consistently show an association between higher mechanical power and mortality, but they also highlight key uncertainties: proposed “thresholds” vary, and at least one large registry-based analysis did not identify a consistent safe lower boundary across a broad acute hypoxemic respiratory failure (AHRF) population. RCTs should therefore move beyond asking whether “lower is better” in general, and instead test specific mechanical power-reduction strategies, in well-defined eligible populations, with protocolized co-interventions to avoid confounding by oxygenation/CO_2/hemodynamic trade-offs.

An important and underexplored avenue is the use of closed-loop or automated ventilation modes specifically designed to optimize ventilatory mechanics in real time. Emerging data suggest that newer “intelligent” modes, such as Adaptive Support Ventilation (ASV) and Adaptive Ventilation Mode (AVM-2), may reduce mechanical power compared with conventional pressure-controlled ventilation. In a prospective pilot study comparing ASV with nonautomated pressure-controlled ventilation in passive critically ill patients, ASV was associated with significantly lower mechanical power (median 15.1 vs 22.9 J/min), driven primarily by reductions in applied pressures and respiratory rate rather than tidal volume. [55] Similarly, comparative analyses of AVM-2 have demonstrated lower mechanical power and improved distribution of its elastic and resistive components relative to conventional modes. [56] These findings suggest that algorithm-driven breath-by-breath adjustment of respiratory rate and pressure targets may represent a feasible strategy for mechanical power minimization.

Another pitfall of MP based ventilation is that all the focus has been on the inspiratory mechanical power while ignoring the expiratory portion of the breath cycle. Expiration exerts its own mechanical work in a direction opposite to that of inspiration. The concept of Mechanical is the complete tidal ventilation cycle, including both inspiratory and expiratory parts, is graphically represented by the hysteresis area surrounded by a pressure–volume (PV) loop. [57]

However, these studies remain small and observational, and whether automated reductions in mechanical power translate into meaningful improvements in clinical outcomes remains unknown. Randomized controlled trials comparing intelligent ventilation modes with protocolized lung-protective ventilation are therefore warranted.

1) Interventions to test (pragmatic, bedside-feasible)
A useful design principle is to randomize patients to a mechanical power–guided strategy vs standard lung-protective ventilation, with an algorithm that prioritizes which ventilator variables to adjust. Candidate interventions include:

- RR-first mechanical power minimization within acceptable $PaCO_2$/pH bounds (especially relevant because time-varying exposure suggests cumulative intensity matters).
- Flow/Inspiratory time modulation (e.g., avoiding unnecessarily high inspiratory flow and unfavorable waveforms), acknowledging that mechanical power integrates flow-dependent work and that the clinical relevance of dynamic inflation is an active debate.
- PEEP strategies anchored to "net injury-relevant load" rather than oxygenation alone, because PEEP increases the baseline energy component but may reduce atelectrauma by stabilizing recruitment.
- Normalization-guided targets (e.g., mechanical power per predicted body weight, per compliance, or per aerated lung estimate) because multiple observational datasets suggest normalized mechanical power may better track risk than raw mechanical power.

2) Targets to test (absolute vs individualized) Observational syntheses frequently identify risk ranges around 14–18 J/min, and a recent PCV only cohort also noted increased mortality around 16–18 J/min, but the CHEST registry analysis argues against a universal "safe" threshold in AHRF.
Accordingly, RCTs could compare:

- Fixed target arms (e.g., mechanical power <15 J/min) vs standard care, *and/or*
- Personalized targets based on lung size surrogates (compliance, imaging-derived aerated volume, or physiologic phenotype), which may be more consistent with the "baby lung" concept and with the physics-based critique that global mechanical power may misrepresent regional risk.

3) Eligible populations (where signal is strongest and intervention is feasible). Eligibility should match both biology and feasibility of measurement/implementation:

- Early AHRF/ARDS within 24 hours of intubation, because early exposure appears meaningful and is a practical window for protocolized intervention.
- Controlled ventilation strata (deep sedation ± neuromuscular blockade) vs assisted/spontaneous breathing strata, since standard mechanical power formulas can be invalid with patient effort; alternatively, use dynamic-pressure–based estimates applicable across modes, with careful validation.
- Phenotype-enriched subgroups (moderate–severe hypoxemia, high recruitability, marked inhomogeneity), where regional stress concentration is more likely and where a reduction in "injury-relevant" load might produce a detectable effect.

A key caution is that prior RCT syntheses of "personalized ventilation" approaches (including DP-, transpulmonary pressure-, imaging-, and mechanical power-related strategies) have not shown clear mortality benefit, emphasizing that future trials need better-defined targets, more standardized algorithms, and potentially better patient selection.

Candidate Endpoints
Because mortality effects may be modest and require large sample sizes, future trials can consider a hierarchy of endpoints, combining mechanistic outcomes with patient-centered outcomes.

1) Patient-centered clinical endpoints

- Ventilator-free days (VFDs) and successful extubation rate, which are sensitive to both pulmonary recovery and competing risks.
  ICU/hospital mortality, ideally with pre-specified time horizons and adjustment for competing risks.
  Duration of ventilation / ICU length of stay, frequently reported in observational mechanical power studies and systematic syntheses.

2) Mechanistic endpoints (to demonstrate biological plausibility)

- Biomarkers of epithelial/endothelial injury and inflammation (e.g., sRAGE, SP-D, IL-6/IL-8), aligned with the concept that injurious energy delivery manifests as tissue injury and biotrauma.
- Physiologic trajectory (compliance, dead space indices, oxygenation and ventilatory ratio), to capture whether mechanical power reduction changes lung mechanics rather than simply trading one impairment for another.

3) Imaging endpoints (regional injury and heterogeneity)

- Electrical impedance tomography (EIT) or CT-based measures of regional ventilation, recruitability, and overdistension/collapse balance—particularly relevant because power intensity and regional stress concentration are likely drivers of injury not reflected by global mechanical power.

A practical approach is a composite primary endpoint with key secondary mechanistic endpoints demonstrating that the intervention actually reduces injury-relevant load.

From mechanical power to injury-relevant power

A major conceptual gap is that mechanical power is usually computed at the airway opening and summarized as a global respiratory system value. The physics-based critique is that injury depends less on total delivered energy and more on where energy is dissipated, how quickly, and within how small a tissue volume.

Measured tidal volume is considered globally, without accounting for physiologic and alveolar dead space. As ventilator-induced lung injury occurs at the alveolar level, airway-derived mechanical power may not accurately reflect the true energy applied to functional alveolar units.

Recent theoretical work has proposed the assessment of trans-alveolar pressure and alveolar tidal volume derived from esophageal manometry and volumetric capnometry as a means to estimate alveolar compliance, resistance, and trans-alveolar mechanical power. In this framework, mechanical power could be expressed using alveolar tidal volume and trans-alveolar driving pressure rather than global variables. While this concept remains investigational and requires clinical validation, it highlights an important knowledge gap between airway-based mechanical power and the mechanical load experienced by alveoli. [58]

Subsequently, recent conceptual refinements argue that total mechanical power should not be considered the most VILI-relevant construct. Mechanical power can be partitioned into flow-resistive, dynamic elastic (driving), and static elastic (PEEP-related) components. Because flow-resistive energy is largely dissipated in proximal airways before reaching the alveoli, the elastic components of power may be more directly implicated in parenchymal deformation. [59]

Building on this framework, Marini and colleagues have proposed the concept of "hazardous elastic power" defined as the fraction of elastic power that exceeds a regional alveolar stress threshold (Pt). In this model, only elastic energy above Pt contributes to injurious overstrain, whereas elastic energy below that threshold may be biologically tolerated. [59] This approach integrates three determinants of VILI: stress amplitude (elastic pressure), cumulative energy delivery (elastic power), and intrinsic tissue vulnerability.

Complementing this theoretical refinement, a recent Bayesian post-hoc analysis of the mechanical power day dataset demonstrated that elastic static power the PEEP-related component of elastic power—was more strongly associated with ARDS severity than total mechanical power, dynamic elastic power, or resistive power. [60] Elastic static power showed the highest posterior inclusion probability and independent correlation with both mild and moderate/severe ARDS, suggesting that baseline strain (PEEP-related load) may represent a clinically relevant contributor to disease severity.

These findings challenge the prevailing assumption that dynamic components alone drive injury and suggest that the distribution and threshold-exceeding fraction of elastic power may be more informative than global mechanical power.
Future work should therefore focus on bridging mechanical power to "injury-relevant power" through three directions:

1) Dissipation-focused metrics
- Separate stored (recoverable) elastic energy from dissipated (irreversible) energy, since only the latter is mechanistically linked to tissue damage.
- Develop clinically feasible proxies of dissipated energy (e.g., PV-loop hysteresis components) and test whether they outperform mechanical power for predicting injury and response.

2) Normalization to functional lung size
- Quantify "power per available lung" using compliance, predicted body weight, or imaging-derived aerated volume. Observational work suggests normalized mechanical power strengthens associations with outcomes, consistent with the idea that the same global energy may be concentrated into a smaller "baby lung."

3) Regional stress and heterogeneity
- Incorporate measures of inhomogeneity and regional strain concentration, because global mechanical power cannot capture stress risers adjacent to collapsed units. This motivates trial enrichment by phenotype and incorporation of EIT/CT endpoints.

Overall, the next generation of studies should treat mechanical power as a useful starting metric while explicitly testing whether strategies that reduce mechanical power (or its normalized/dissipated/regional analogues) can improve clinically meaningful outcomes without unacceptable trade-offs.

Conclusion

Mechanical power provides an integrative framework for understanding how ventilatory variables jointly contribute to the mechanical load imposed on the respiratory system during invasive mechanical ventilation. This energy based construct offers a coherent physiologic perspective that extends beyond traditional static parameters.

Experimental and observational studies consistently demonstrate an association between higher mechanical power and adverse outcomes, including ventilator-induced lung injury and mortality, across a range of patient populations. However, current evidence does not establish mechanical power as a validated therapeutic target. Proposed thresholds vary across studies, and randomized trials of personalized ventilation strategies including those conceptually aligned with mechanical power have not shown clear improvements in mortality. These limitations reflect fundamental challenges inherent to the concept. As a result, global mechanical power may incompletely represent the biological processes that lead to lung injury.

At present, mechanical power should be viewed as a useful summary of ventilatory intensity and a complementary lens through which to interpret ventilator settings, rather than as a standalone clinical target. Future research should focus on refining energy-based metrics to better capture injury-relevant mechanisms, identifying patient populations most likely to benefit from intervention, and testing protocolized strategies that reduce injurious mechanical load without compromising gas exchange or hemodynamic stability.

In this context, mechanical power may ultimately contribute to a more nuanced approach to lung-protective ventilation while recognizing the current limits of evidence and the need for carefully designed prospective trials.

References

1. Paudel R, Trinkle CA, Waters CM, et al. Mechanical power: A New Concept in Mechanical Ventilation. Am J Méd Sci 2021;362(6):537-545.

2. Düring S von, Liu K, Munshi L, et al. The association between mechanical power within the first 24 hours and icu mortality in mechanically ventilated adult patients with acute hypoxemic respiratory failure: A registry-based cohort study. Chest 2025;168(4):901-911.

3. Network ARDS, Brower RG, Matthay MA, et al. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. N Engl J Med 2000; 342(18):1301-1308.

4. Marini JJ, Rocco PRM, Gattinoni L. Static and dynamic contributors to ventilator-induced lung injury in clinical practice. Pressure, energy, and power. Am J Respir Crit Care Med 2019; 201(7):767-774.

5. Slutsky AS, Ranieri VM. Ventilator-induced lung injury. N Engl J Med 2013; 369(22):2126-2136.

6. Tremblay LN, Slutsky AS. Ventilator-induced injury: from barotrauma to biotrauma. *Proc Assoc Am Physicians*. 1998; 110(6):482-488.

7. Gattinoni L, Collino F, Camporota L. Mechanical power: meaning, uses and limitations. Intensiv Care Med 2023; 49(4):465-467.

8. Gattinoni L, Tonetti T, Cressoni M, et al. Ventilator-related causes of lung injury: the mechanical power. Intensiv Care Med 2016; 42(10):1567-1575.

9. Urbankowski T, Pasledni R, Darowski M. Mechanical power in mechanical ventilation and its association with ventilator-induced lung injury: A systematic review. Respir Med 2025; 250:108525.

10. Cressoni M, Gotti M, Chiurazzi C, et al. Mechanical power and development of ventilator-induced lung injury. Anesthesiology 2016; 124(5):1100-1108.

11. Neto AS, Deliberato RO, Johnson AEW, et al. Mechanical power of ventilation is associated with mortality in critically ill patients: an analysis of patients in two observational cohorts. Intensive Care Medicine 2018; 44(11):1914-1922.

12. Goedegebuur J, Smits FE, Snoep JWM, et al. Mechanical power is associated with mortality in pressure-controlled ventilated patients: A Dutch, single-center cohort study. Crit Care Explor 2024;6(12):e1190.

13. Manrique S, Ruiz-Botella M, Murillo N, et al. Impact of mechanical power on ICU mortality in ventilated critically ill patients: a retrospective study with continuous real-life data. Eur J Med Res. 2024; 29(1):491.

14. Coppola S, Caccioppola A, Froio S, et al. Effect of mechanical power on intensive care mortality in ARDS patients. Critical Care 2020; 24(1):246.

15. Urner M, Jüni P, Hansen B, et al. Time-varying intensity of mechanical ventilation and mortality in patients with acute respiratory failure: a registry-based, prospective cohort study. Lancet Respir Med 2020; 8(9):905-913.

16. Costa ELV, Slutsky AS, Brochard LJ, et al. Ventilatory variables and mechanical power in patients with acute respiratory distress syndrome. Am J Respir Crit Care Med. 2021; 204(3):303-311.

17. Silva PL, Ball L, Rocco PRM, et al. Power to mechanical power to minimize ventilator-induced lung injury? Intensiv Care Med Exp 2019; 7(Suppl 1):38.

18. Talmor D, Sarge T, O'Donnell CR, et al. Esophageal and transpulmonary pressures in acute respiratory failure. Crit Care Med 2006; 34(5):1389-1394.

19. Staay M van der, Chatburn RL. Advanced modes of mechanical ventilation and optimal targeting schemes. Intensiv Care Med Exp 2018; 6(1):30.

20. Keitoku K, Yeo J, Cabbat R, Daoud EG. Mechanical power and Power Compliance Index in independent lung ventilation. New insight. J Mech Vent 2022; 3(2):124-131.

21. Grieco DL, Chen L, Brochard L. Transpulmonary pressure: importance and limits. Ann Transl Med 2017; 5(14):285-285.

22. Loring SH, O'Donnell CR, Behazin N, et al. Esophageal pressures in acute lung injury: do they represent artifact or useful information about transpulmonary pressure, chest wall mechanics, and lung stress? J Appl Physiol 2010; 108(3):515-522.

23. Gattinoni L, Pesenti A. The concept of "baby lung." Intensive Care Med 2005; 31(6):776-784.

24. Bates JHT, Kaczka DW, Kollisch-Singule M, et al. Mechanical power and ventilator-induced lung injury: What does physics have to say? Am J Respir Crit Care Med 2024; 209(7):787-788.

25. Busana M, Zinnato C, Romitti F, et al. Energy dissipation during expiration and ventilator-induced lung injury: an experimental animal study. J Appl Physiol 2022; 133(5):1212-1219.

26. Becher T, Staay M van der, Schädler D, et al. Calculation of mechanical power for pressure-controlled ventilation. Intensive Care Med 2019; 45(9):1321-1323.

27. Giosa L, Busana M, Pasticci I, et al. Mechanical power at a glance: a simple surrogate for volume-controlled ventilation. Intensive Care Med Exp 2019; 7(1):61.

28. Chiumello D, Gotti M, Guanziroli M, et al. Bedside calculation of mechanical power during volume- and pressure-controlled mechanical ventilation. Crit Care 2020; 24(1):417.

29. Lee P, Morikawa K, Daoud E. Evaluation of the mean airway pressure - Minute ventilation (mM) Equation for mechanical power during spontaneous breathing. J Mech Vent 2025; 6(2):72-78.

30. Daoud E, Lee P, Toma S, Franck C. Mean airway pressure - Minute ventilation product (mM): A simple and universal surrogate equation to calculate mechanical power in both volume and pressure controlled ventilation. J Mech Vent 2024; 5(2):46-55.

31. Chiumello D, Coppola S, Formenti P, et al. A validation study of a continuous automatic measurement of the mechanical power in ARDS patients. J Crit Care 2022; 67:21-25.

32. Amato MBP, Meade MO, Slutsky AS, et al. Driving pressure and survival in the acute respiratory distress syndrome. N Engl J Med 2015; 372(8):747-755.

33. Santini A, Mauri T, Corte FD, et al. Effects of inspiratory flow on lung stress, pendelluft, and ventilation heterogeneity in ARDS: a physiological study. Crit Care 2019; 23(1):369.

34. Huhle R, Neto A, Schultz M, et al. Is mechanical power the final word on ventilator-induced lung injury? No. Ann Transl Med 2018; 6(19):394.

35. Silva AL da, Magalhaes RF, Conceicao PHL, et al. Effects of similar mechanical power resulting from different combinations of respiratory variables on lung damage in experimental acute respiratory distress syndrome. Crit Care Med 2025; 53(6):e1303-e1313.

36. Tonetti T, Vasques F, Rapetti F, et al. Driving pressure and mechanical power: new targets for VILI prevention. Ann Transl Med 2017; 5(14):286-286.

37. Xie Y, Zheng H, Mou Z, et al. High expression of CXCL10/CXCR3 in ventilator-induced lung injury caused by high mechanical power. BioMed Res Int 2022; 2022(1):6803154.

38. Scharffenberg M, Wittenstein J, Ran X, et al. Mechanical power correlates with lung inflammation assessed by positron-emission tomography in experimental acute lung injury in pigs. Front Physiol 2021; 12:717266.

39. Li HH, Wang CW, Chang CH, et al. Relationship between mechanical ventilation and histological fibrosis in patients with acute respiratory distress syndrome undergoing open lung biopsy. J Pers Med. 2022; 12(3):474.

40. Collino F, Rapetti F, Vasques F, et al. Positive end-expiratory pressure and mechanical power. Anesthesiology 2019; 130(1):119-130.

41. Romitti F, Busana M, Palumbo MM, et al. Mechanical power thresholds during mechanical ventilation: An experimental study. Physiol Rep 2022; 10(6):e15225.

42. Meenen DMP van, Algera AG, Schuijt MTU, et al. Effect of mechanical power on mortality in invasively ventilated ICU patients without the acute respiratory distress syndrome: An analysis of three randomized clinical trials. Eur J Anaesthesiol 2023; 40(1):21-28.

43. Zhang Z, Zheng B, Liu N, et al. Mechanical power normalized to predicted body weight as a predictor of mortality in patients with acute respiratory distress syndrome. Intensive Care Med 2019; 45(6):856-864.

44. Khorasanee R, Sanderson B, Tomarchio E, et al. Mechanical power normalisation methods to predict ICU mortality: a retrospective cohort study. Ann Intensiv Care 2025;15(1):149.

45. Hu W, Zhang S, He Z, et al. Impact of time-varying intensity of mechanical ventilation on 28-day mortality depends on fluid balance in patients with acute respiratory distress syndrome: A retrospective cohort study. Front Med 2022; 9:906903.

46. Schuijt MTU, Meenen DMP van, Martin–Loeches I, et al. Association of time–varying intensity of ventilation with mortality in patients with COVID−19 ARDS: Secondary analysis of the PRoVENT–COVID study. Front Med 2021; 8:725265.

47. Caironi P, Carlesso E, Cressoni M, et al. Lung recruitability is better estimated according to the berlin definition of acute respiratory distress syndrome at standard 5 cmH2o rather than higher positive end-expiratory pressure. Crit Care Med 2015; 43(4):781-790.

48. Pistillo N, Castelluccio P, Suzuki I, et al. Mechanical power correlates with stress, strain, and atelectrauma only when normalized to aerated lung size in patients with acute respiratory distress syndrome. Crit Care Explor 2023;5(10):e0982.

49. Galizia M, Ghidoni V, Catozzi G, et al. Predictors of VILI risk: driving pressure, 4DPRR and mechanical power ratio—an experimental study. Intensiv Care Med Exp 2024;12(1):116.

50. Williams EC, Motta-Ribeiro GC, Melo MFV. Driving pressure and transpulmonary pressure. Anesthesiology 2019; 131(1):155-163.

51. Arnal JM, Saoli M, Garnero A. Airway and transpulmonary driving pressures and mechanical powers selected by INTELLiVENT-ASV in passive, mechanically ventilated ICU patients. Heart Lung 2020; 49(4):427-434.

52. Fajardo-Campoverdi A, Mamani-Cruz L, Ibarra-Estrada M, et al. Cyclic energy: the transcendental relevance of respiratory rate. A retrospective observational study with Bayesian analysis. J Mech Vent 2024; 5(1):1-10.

53. Hickling KG, Walsh J, Henderson S, et al. Low mortality rate in adult respiratory distress syndrome using low-volume, pressure-limited ventilation with permissive hypercapnia: A prospective study. Crit Care Med 1994; 22(10):1568-1578.

54. Jimenez JV, Munroe E, Weirauch AJ, et al. Electric impedance tomography-guided PEEP titration reduces mechanical power in ARDS: a randomized crossover pilot trial. Crit Care 2023; 27(1):21.

55. Buiteman-Kruizinga LA, Mkadmi HE, Schultz MJ, et al. Comparison of mechanical power during adaptive support ventilation versus nonautomated pressure-controlled ventilation—a pilot study. Crit Care Explor 2021; 3(2):e0335.

56. Takaoka K, Toma S, Lee P, et al. A comparative analysis of mechanical power and Its components in pressure-controlled ventilation mode and AVM-2 mode. J Mech Vent 2023; 4(4):130-140.

57. Wu SH, Kor CT, Mao IC, et al. Accuracy of calculating mechanical power of ventilation by one commonly used equation. J Clin Monit Comput 2022; 36(6):1753-1759.

58. Daoud E, Franck C. Alveolar mechanics: A new concept in respiratory monitoring. J Mech Vent 2022; 3(4):178-188.

59. Marini JJ, Crooke PS, Rocco PRM. Mechanical power of ventilation: tracking the damaging component. Crit Care 2026; 30(1):29.

60. Fajardo-Campoverdi A, González-Castro A, Alapont VM i, et al. Elastic static power, its correlation with acute respiratory distress syndrome severity: A Bayesian post-hoc analysis of the Mechanical Power Day cross-sectional trial. Med Intensiva 2025; 49(5):502128.