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Understanding ventilator-induced lung injury: The role of mechanical power

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ABSTRACT

Mechanical ventilation stands as a life-saving intervention in the management of respiratory failure. However, it carries the risk of ventilator-induced lung injury. Despite the adoption of lung-protective ventilation strategies, including lower tidal volumes and pressure limitations, mortality rates remain high, leaving room for innovative approaches. The concept of mechanical power has emerged as a comprehensive metric encompassing key ventilator parameters associated with the genesis of ventilator-induced lung injury, including volume, pressure, flow, resistance, and respiratory rate. While numerous animal and human studies have linked mechanical power and ventilator-induced lung injury, its practical implementation at the bedside is hindered by calculation challenges, lack of equation consensus, and the absence of an optimal threshold. To overcome the constraints of measuring static respiratory parameters, dynamic mechanical power is proposed for all patients, regardless of their ventilation mode. However, establishing a causal relationship is crucial for its potential implementation, and requires further research. The objective of this review is to explore the role of mechanical power in ventilator-induced lung injury, its association with patient outcomes, and the challenges and potential benefits of implementing a ventilation strategy based on mechanical power.

Abbreviations: ARDS, acute respiratory distress syndrome; C_{RS}, respiratory system compliance; ΔP, driving pressure (P_{plat} - PEEP); ΔP_{dyn}, dynamic driving pressure (P_{peak} - PEEP); EEAP, end-expiratory airway pressure; EELV, end-expiratory lung volume; E_{RS}, elastance of the respiratory system; FRC, functional residual capacity; HR, hazard ratio; J/min, Joules per minute; MP, mechanical power; OR, odds ratio; P_{aw}, pressure in the airways; PBW, predicted body weight; PCV, pressure-controlled ventilation; PEEP, positive end-expiratory pressure; P_{peak}, peak pressure; P_{plat}, plateau pressure; RCTs, randomized controlled trials; RR, respiratory rate; R_{RS}, resistance of the respiratory system; V, air flow; VCV, volume-controlled ventilation; VILI, ventilator-induced lung injury; V_T, tidal volume..

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1. Introduction

Mechanical ventilation is a life-saving intervention in the management of acute respiratory failure. However, it can lead to ventilator-induced lung injury (VILI), which can cause or worsen pre-existing lung injury [1]. Currently, four main mechanisms are thought to be responsible for the pathogenesis of VILI: alveolar over-distention (volutrauma), trans-alveolar over-pressurization (barotrauma), high shear forces from cyclic opening and collapse of atelectatic but recruitable alveoli (atelectotrauma), and inflammation (biotrauma) [1-3]. These mechanisms increase alveolar permeability, edema, hemorrhage, and ultimately alveolar collapse, further impairing gas exchange. Patients with VILI may face prolonged mechanical ventilation, increased risk of multi-organ failure, and higher mortality, emphasizing the need to prevent or mitigate VILI [4,5].

Over the past decades, extensive research has attempted to identify mechanical ventilation parameters that are associated with an increased risk of VILI in patients with acute respiratory distress syndrome (ARDS). Despite the implementation of contemporary lung-protective ventilation strategies, focusing on limiting tidal volume (V_T) and inspiratory pressures [6,7], mortality in patients with ARDS remains high [5], highlighting the need for novel strategies. Recently, mechanical power (MP) has emerged as a comprehensive measure to estimate the risk of VILI [8]. MP represents the total amount of energy transferred by the ventilator to the respiratory system over time and encompasses all parameters thought to be associated with the genesis of VILI, including V_T, pressure, air flow (V), resistance, and respiratory rate (RR) [8]. Studies have demonstrated that high MP is associated with worse patient outcomes in ARDS [9-15]. Therefore, limiting MP has gained attention as a promising approach for lung-protective ventilation. However, the practical implementation of MP in clinical practice has encountered challenges due to calculation complexities and the lack of evidence from randomized controlled trials (RCTs) supporting its role in improving patient outcomes.

This review aims to synthesize the existing literature on MP, its role in VILI and its association with patient outcomes. Additionally, we discuss the challenges faced in implementing MP at the bedside and explore different approaches.

2. Current lung protective ventilation strategies

To minimize VILI, lung-protective ventilation strategies aim to reduce the mechanical stress and strain applied to the lungs during mechanical ventilation. The landmark ARDS Network's lower V_T RCT demonstrated the importance of limiting V_T and plateau pressure (P_{plat}) in mitigating VILI, resulting in decreased mortality (39.8 vs. 31.0 %) and more ventilator-free days (12 ± 11 days vs. 10 ± 11 days) [16]. These results were further supported in a meta-analysis [17], and benefit patients both with and without ARDS [18-20], establishing ventilation with low V_T and P_{plat} as integral components of lung-protective ventilation [6,7]. Injured lungs often undergo atelectasis and lose lung volume, commonly referred to as the "baby lung" [21]. When adjusting V_T to predicted body weight (PBW), it is crucial to consider changes in the fraction of aerated lung tissue (i.e. functional residual capacity (FRC)) that maintains normal inflation, reflecting the respiratory system compliance (C_{RS}).

3. Mechanical power: definition and quantification

In 2016, the innovative concept of MP emerged as a new strategy to further reduce VILI and improve patient outcome [8]. This approach leveraged fundamental thermodynamic principles governing energy transfer to model the energy exchanged between the ventilator and the respiratory system on each breath. In the context of mechanical ventilation, inspiratory tidal energy, representing the work transferred from the ventilator to the respiratory system during each breath, can be quantified by numerically integrating the area between the inspiratory limb of the airway pressure and the volume axis [8,22]. (Fig. 1) Referred to as the geometric method, it is essentially used in research. This total energy can be divided into three components [8,23,24]:

1. The *elastic static* component, corresponds to energy delivered just once when positive end expiratory pressure (PEEP) is applied. Using a spring analogy, it represents the stretch from the resting point of the lung's parenchyma and the chest wall, also known as the FRC, to the end-expiratory lung volume (EELV), at the set PEEP. This component is not included in either the mathematical models or the geometric method used to calculate MP.
2. The *elastic dynamic* component, corresponds to the energy expended during each breath to counteract the recoil of the respiratory system throughout the dynamic phase of inspiration. This component is subdivided into the recoil due to PEEP, representing the baseline stretch of the fibers, and the energy needed to overcome the elasticity of the respiratory system due to volume variation (i.e. V_T). Analogous to a spring, it illustrates the stretch from PEEP to P_{plat}.
3. The *resistive* component, corresponds to the work required to overcome the resistive forces associated with V, comprised of the airways as well as the tubes connecting the patient to the ventilator.

MP, expressed in Joules per minute (J/min), is determined by summing the elastic dynamic and resistive components, then multiplying the total by the RR.

The equation of motion dictates the overall pressure in the airways (P_{aw}), at any given time (t):

$$P_{aw}(t) = (\dot{V}(t) \times R_{RS}) + (V_T(t) \times E_{RS}) + EEAP$$

V(t): air flow, L/s. R_{RS}*: resistance of the respiratory system,* $cmH_2O/L/s$*.* V_T*(t): tidal volume, L.* E_{RS}*: elastance of the respiratory system,* cmH_2O/L*. EEAP: end-expiratory airway pressure,* cmH_2O*.*

Integrating P_{aw} during inspiration gives P_{insp}. Multiplying P_{insp} by V_T provides tidal energy. Further multiplying tidal energy by RR provides mechanical power.

$$Mechanical\ Power \;=\; V_T \;\times\; P_{insp} \;\times\; RR$$

V_T: tidal volume, L. P_{insp}*: cumulative resistive and elastic pressures exerted on the respiratory system during inspiration,* cmH_2O*. RR: respiratory rate, /min.*

Limiting MP has emerged as a promising strategy for lung-protective ventilation, aiming to mitigate the disruptive deformation of cells and the extracellular matrix caused by a high energy load over time, thereby reducing the risk of VILI [25]. (Fig. 2) To apply this concept at the bedside without relying on ventilator waveform analysis, multiple mathematical formulas have been proposed [8,23,26].

The incorporation of PEEP into MP remains a topic of ongoing discussion, primarily due to the following two main points: (1) PEEP is a static pressure and does not contribute to dynamic volume changes, (2) current mathematical models assume a positive and linear relationship between PEEP and MP (when keeping all other variables constant), failing to consider a U-shaped relationship between PEEP and VILI [27]. By raising the FRC to EELV, PEEP serves to counteract the effects of cyclic opening and closing of small airways (shear stress), and prevents alveolar collapse (atelectrauma). This process contributes to improved aerated lung homogeneity, which leads to increased C_{RS}. As changes in C_{RS} induce pressure or volume changes, they consequently affect MP values. In volume-controlled ventilation (VCV), PEEP affects P_{plat}, while in pressure-controlled ventilation (PCV), PEEP influences V_T, further impacting MP. This underscores the interdependence between PEEP and MP, highlighting the complexities surrounding its integration into the understanding of VILI. To address this issue, one approach is to conceptualize the respiratory system as a spring, with its resting point at the equilibrium of opposing elastic recoil forces between the lung's

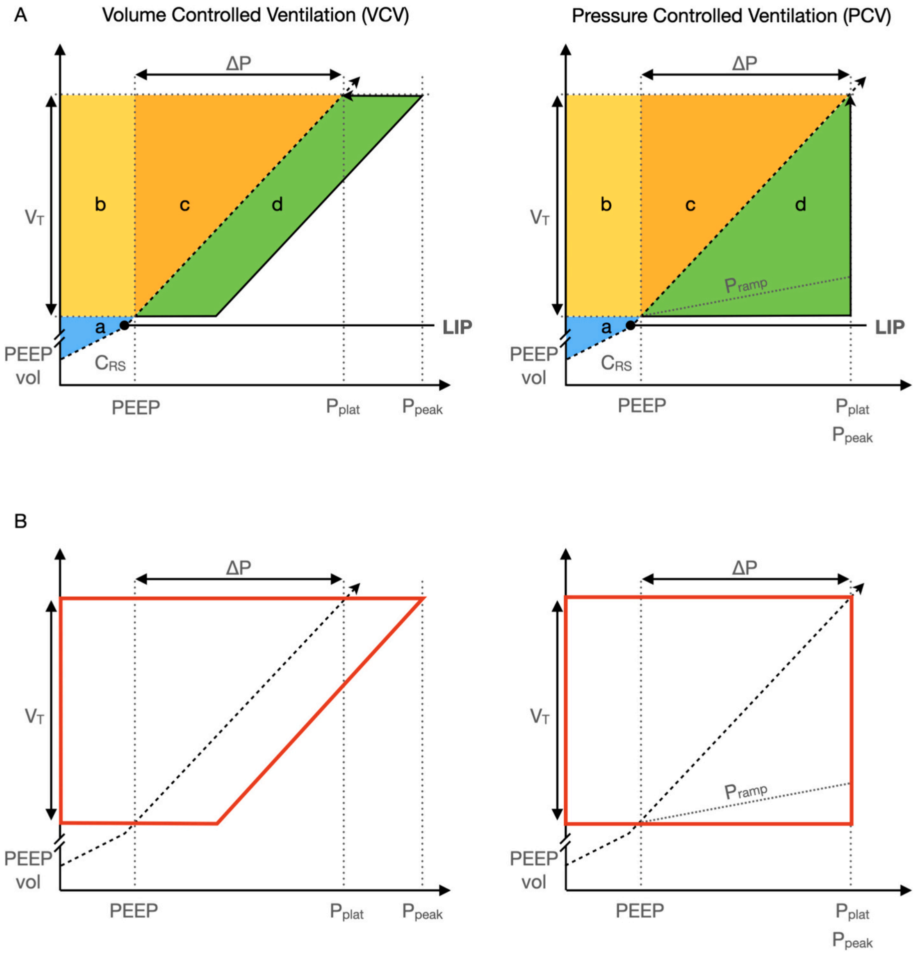

Fig. 1. Tidal energy during volume- and pressure-controlled ventilation.

Panel **A** Illustrates the different components of tidal energy using the geometrical view: (a) the elastic static pressure at baseline set by PEEP, (b) the elastic dynamic pressure (PEEP), (c) the elastic dynamic pressure (ΔP), and (d) the flow resistive pressure. Components (b) and (c) are often unified as the dynamic elastic pressure. The sum of these pressure components, when multiplied by the respiratory rate, results in mechanical power. Panel **B** illustrates the tidal power, represented as the red contour, as calculated by the "power equation". The elastic static pressure at baseline set by PEEP is not integrated in the mathematical model. In VCV, the tidal energy is uniformly distributed throughout the inspiratory phase. In PCV, tidal energy is primarily concentrated at the beginning of inspiration, however can be overestimated with a slower pressure ramp (P_{ramp}) is set. ΔP: driving pressure, V_T: tidal volume, PEEP vol: lung volume at PEEP, C_{RS}: compliance of the respiratory system, PEEP: positive end-expiratory pressure, P_{plat}: plateau pressure, P_{peak}: peak inspiratory pressure, LIP: lower inflection point. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

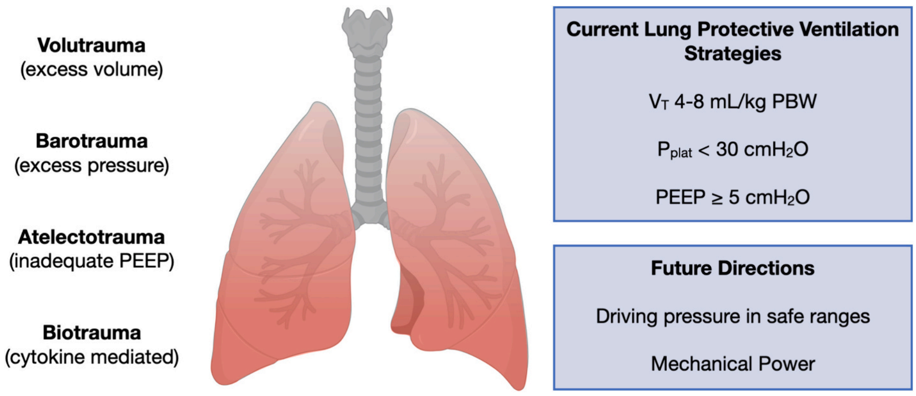

Fig. 2. Mechanisms for the pathogenesis of VILI and targets for VILI mitigation.

Illustration depicting the multifaceted pathogenesis of ventilator-induced lung injury (VILI) and targeted mitigation strategies. PEEP: positive end-expiratory pressure. V_T: tidal volume. PBW: predicted body weight. P_{plat}: Plateau pressure.

parenchyma and chest wall (i.e., at FRC). Any additional force that offset this equilibrium, represented as work exerted by the spring, should be integrated into the MP concept, meaning that it is the absolute pressure introduced into the respiratory system, rather than the change in pressure, that determines VILI [3,28].

Other factors that have not typically been considered in traditional approaches to lung protective ventilation, include peak pressure (P_{peak}) and RR, are incorporated in the MP concept and may also play a role in VILI. Although the energy from P_{peak} is primarily dissipated throughout the proximal airways as heat, studies suggest that high P_{peak}, measured over 45–50 cmH_2O, can lead to alterations in microvascular permeability and are associated with worse patient outcome [29-31]. The impact of RR on VILI is also an area of ongoing research. As minute ventilation depends on RR and V_T, the latter is often limited to follow lung protective recommendations at the expense of high RR. Experimental studies have indicated that the higher the RR, the more susceptible the lung is to VILI [32-34]. Previous work has identified that lower RR in ventilated rabbit lungs decreased edema formation and perivascular hemorrhage, leading to improved lung histopathology and reduced VILI [35]. In the LUNG SAFE study, RR was identified as a modifiable factor independently associated with hospital mortality, and current ARDS Network recommendations suggest keeping RR $\leq$ 35 breaths per minute [5].

3.1. *MP during Volume-Controlled Ventilation (VCV)*

The original "power equation" was validated in VCV using the geometric method in patients with both normal lungs and ARDS [8]. (Table 1) Limitations of this equation, besides its complexity, are its reliance on an inspiratory and expiratory hold to distinguish between the resistive and elastic airway pressure components, and the calculation of C_{RS} and resistance of the respiratory system (R_{RS}), hindering its real-time applicability at the bedside. To enhance its clinical utility, two equations have been developed to calculate MP in VCV under constant inspiratory V. (1) The "simplified power equation" was initially devised for the a priori calculation of MP based on specific lung characteristics available at the bedside through the ventilator. However, this method still requires both flow holds, and assumes a constant C_{RS} and R_{RS} [8,36]. (2) Recently, a practical alternative equation, based on V measurements instead of P_{plat}, enables the calculation of MP on a breath-by-breath basis [23]. However, it assumes that R_{RS} is constant at 10 $cmH_2O \times$ *sec*/L. These two equations were found to be highly correlated with the reference-standard geometric method, with the limits of agreement falling within 2 J/min [36].

3.2. *MP during Pressure-Controlled Ventilation (PCV)*

Due to the decelerating flow pattern in PCV, the highest energy delivery rate occurs at the beginning of inspiration. When V reaches zero, P_{peak} is equivalent to P_{plat}. Several extended equations model this decelerating flow pattern [26,37]. A simplified surrogate equation that can be calculated at each breath without inspiratory holds [26] has shown similar accuracy to more complex equations, referencing the geometric method [36,37]. However, the accuracy of this equation is contingent on the rate of pressurization. When the pressurization from PEEP to P_{peak} is instantaneous (square-wave), the equation aligns with the value obtained through numerical integration of tidal energy. Conversely, the presence of a slower pressure ramp results in an overestimation of tidal energy and, consequently, MP.

4. Mechanical power: experimental and clinical findings

Numerous animal [34,38,39] and human [9-11,40-47] studies have linked higher MP to VILI. In a porcine study assessing the association

Table 1

Key bedside equations for calculating mechanical power in volume- and pressure-controlled ventilation.

Equation name		Mathematical equation	Advantages	Potential pitfalls
Volume-controlled ventilation	Mechanical power equation	$MP = \{V_T^2 \times (1/2 \times E_{RS} + RR \times (1 + I/E)/(60 \times I/E) \times R_{RS}) + V_T \times PEEP\} \times RR \times 0.098$ [8]	• Has been validated in patients with both normal lungs and ARDS.	• Complex equation • For volume-controlled ventilation • Requires an inspiratory and expiratory hold • Assumes linear C_{RS} throughout inspiration • Assumes constant V and R_{RS} during inflation
	Simplified mechanical power equation	$MP = V_T \times (P_{peak} - (\Delta P)/2) \times RR \times 0.098$ [8]	• Has been validated in patients with both normal lungs and ARDS.	• Requires an inspiratory and expiratory hold • Assumes linear C_{RS} throughout inspiration • Assumes constant V and R_{RS} during inflation
		$MP = (V_T \times (P_{peak} + PEEP + V/6) \times RR)/20$ [23]	• Approximated the conversion factor for L/cmH_2O to Joules, from 0.098 to 0.1 • No need for inspiratory hold • Allows for the calculation of MP on a breath-by-breath basis	• Assumes consistent inspiratory V • Assumes a constant R_{RS} of 10 $cmH_2O \times$ sec/L • Assumes linear C_{RS} throughout inspiration
Pressure-controlled ventilation		$MP = V_T \times (\Delta P_{dyn} + PEEP) \times RR \times 0.098$ [26]	• No need for inspiratory hold • Allows for calculation of MP on a breath-by-breath basis	• Excludes inspiratory pressure rise time • Assumes constant V and R_{RS} through a squared pressure delivery during inflation
Dynamic mechanical power equation		$MP_{dyn} = V_T \times (P_{peak} - (\Delta P_{dyn})/2) \times RR \times 0.098$ [40]	• No need for inspiratory hold • Allows for calculation of MP on a breath-by-breath basis	• Assumes linear C_{RS} throughout inspiration • Assumes constant V and R_{RS} during inflation

Four equations for calculating mechanical power are designed for bedside use. The most user-friendly and robust equation is the "simplified power equation". By utilizing this equation, and further substituting ΔP with ΔP_{dyn}, it enables a breath-by-breath assessment of mechanical power, effectively overcoming the challenges associated with manual flow holds. V_T: tidal volume, E_{RS}: elastance of the respiratory system, RR: respiratory rate, I/E: inspiratory-to-expiratory time ratio, R_{RS}: resistance of the respiratory system, PEEP: positive end expiratory pressure, P_{peak}: peak inspiratory pressure, ΔP: driving pressure (P_{plat} - PEEP), V: air flow, ΔP_{dyn}: dynamic driving pressure (P_{peak} - PEEP), C_{RS}: respiratory system compliance.

between near-apneic ventilation and VILI in subjects on extracorporeal life support with ARDS, higher MP was associated with increased histologic lung damage and fibroproliferation scores [38]. In a secondary analysis of an RCT, higher MP was associated with increased risk for developing ARDS (odds ratio [OR] of 1.03 per 1 J/min MP (95 % confidence interval [CI]: 1.00, 1.06)) [44]. A recent retrospective study involving COVID-19 patients revealed that for every 1 standard deviation increase (equivalent to 7.1 J/min) in median MP during the initial 24 h of mechanical ventilation, was associated an increase in 30-day mortality (OR 1.26 [95 % CI: 1.09, 1.46]) [45]. This association held true irrespective of the COVID-19 diagnosis. Finally, retrospective data has found an association between intraoperative MP and postoperative pulmonary complications, even in low-risk surgical patients [42,46,47]. These studies suggest an association between MP, VILI, and mortality, across diverse patient populations.

4.1. Dynamic mechanical power

In an attempt to circumvent the constraints of measuring static respiratory parameters, dynamic ΔP (ΔP_{dyn}), represented as P_{peak} - PEEP, has been proposed to assess the risk of VILI [40,48,49]. Dynamic parameters are advantageous as they are automatically displayed by the ventilator during each breath, allowing continuous monitoring of physiological changes over time. Under VCV, static and dynamic measures of ΔP may vary due to airway resistances (primarily due to the endotracheal tube). In contrast, under PCV, such differences are minimal. Static and dynamic ΔP seem to provide similar information [50]. By employing a modified “simplified power equation”, dynamic MP can be calculated as:

$$dynamic\ MP = V_T \times \left(P_{peak} - \frac{(\Delta P_{dyn})}{2}\right) \times RR \times 0.098$$

ΔP_{dyn}: change in airway pressure during inspiration (P_{peak}-PEEP), cmH_2O. *0.098: conversion factor from L/cmH_2O to Joules.*

The use of dynamic MP is attractive for clinicians as it simplifies its bedside calculation for all mechanically ventilated patients, irrespective of their ventilation mode [40-43]. One study reported an increased 30-day mortality risk with each daily increment in dynamic MP (hazard ratio [HR] 1.06 [95 % credible interval: 1.053, 1.066]) in patients with acute respiratory failure, irrespective of their mode of ventilation [40], while another study found a significant hazard of death associated with each 1 J/min dynamic MP increment (HR 1.12 [95 % CI: 1.01, 1.36]) [41]. In the intraoperative setting, dynamic MP demonstrated an association with postoperative pulmonary complications in patients undergoing abdominal surgery, with the dynamic MP increase being attributed to factors beyond ΔP_{dyn} alone [42]. These findings support the potentially important role of dynamic MP in understanding VILI risk, suggesting that early monitoring and limiting prolonged exposure to high dynamic MP could decrease mortality by mitigating cumulative adverse effects.

5. Mechanical power: limitations and open questions

The presence of a specific MP threshold responsible for VILI development remains uncertain. In an experimental study involving healthy piglets, a transpulmonary MP > 12 J/min was associated with VILI development (assessed by computed tomography-based identification of edema), regardless of set V_T or RR [34]. Levels > 25 J/min resulted in potentially lethal lung damage. In a pig model exploring the relationship between MP and vascular congestion as a marker of VILI, results showed that even at 3 J/min there was elevated lung weight, lung wet-to-dry ratio, and worsening histological lung damage parameters [39]. In a secondary analysis of two RCTs of ARDS patients, those with a MP > 12 J/min after 24 h of mechanical ventilation had higher 90-day mortality [10]. A retrospective analysis of ARDS patients from two cohorts reported increased hospital length-of-stay and mortality with a MP > 17 J/min [9]. Importantly, both cohorts noted a decrease in MP from the first to the second day, suggesting potential improvement in lung injury or adjustments in ventilator settings. Finally, a prospective ARDS screening program found that MP > 22 J/min in patients on invasive mechanical ventilation for > 24 h was associated with fewer ventilator-free days and higher 28-day and 3-year mortality [11]. From these studies, a definitive and consistent MP threshold causing VILI has not been established. Additionally, the absence of a universally accepted MP calculation formula further complicates threshold determination.

Traditional lung-protective management relies on static ventilator parameters obtained via manual flow holds to assess the risk of VILI [6,7]. These parameters allow for respiratory system relaxation and redistribution of volume and distending pressures, providing measurements under stable conditions considered as the standard. However, static measurements of C_{RS} and ΔP using P_{plat} may underestimate the maximum pressure experienced by vulnerable lung units during dynamic inflation in heterogeneous lungs [51]. Alternative methods for more accurate estimates of alveolar pressure and stress exist but require manual intervention and are not commonly utilized in the intensive care unit (ICU). Consequently, P_{plat} measurements are infrequent, mainly for severely ill patients with significant respiratory support needs, restricting MP's application to a specific subset of patients already at increased risk for VILI [5,52]. Additionally, current mathematical equations for MP calculation rely on the absence of spontaneous breathing efforts.

Despite its advantages, dynamic MP has a number of potential limitations. ΔP_{dyn} depends on the ventilation mode; in VCV, increased R_{RS} raises P_{peak}, leading to a larger gap between ΔP_{dyn} and ΔP, resulting in underestimating MP values. In PCV, this equation provides an estimate of the elastic dynamic component alone. In pressure support, diaphragmatic contraction lowers P_{peak} compared to P_{plat}, which reduces ΔP_{dyn}, leading to an overestimation of MP values. Despite these limitations, dynamic MP may be the most accurate single equation for estimating VILI risk across all ventilation modes, irrespective of patient effort. Future validation studies are essential, employing the geometric method in both controlled and spontaneously breathing models.

The current concept of MP quantifies the energy delivery during lung inflation, neglecting the energy expenditure during lung deflation. To address this, researchers have proposed the implementation of expiratory-flow control as an additional strategy to further reduce stresses during a respiratory cycle and ultimately reduce the risk of VILI [53].

As can be seen from the MP equations, many of the included variables are strongly related to each other and it is difficult to determine the individual impact of each variable on MP, and therefore VILI. When examining the association between MP, ΔP, and 60-day hospital mortality in three ARDS RCTs, results found significant associations for ΔP (HR 1.44 [95 % CI: 1.28, 1.62]) and MP (HR 1.39 [95 % CI: 1.28, 1.52]) [54]. However, when both variables were included in the same model, the HR decreased (1.2 for ΔP [95 % CI: 1.03, 1.40]; 1.26 for MP [95 % CI: 1.11, 1.43]), suggesting potential risk information overlap due to their interrelationship, as ΔP is a component of the MP equation. Interestingly, recent data has shown that ΔP is four times more important than RR in predicting mortality, and (4 × ΔP) + RR equation has exhibited comparable predictive performance to MP in patients with and without ARDS [15,55].

The current MP concept has faced criticism for its omission of standard lung volume (normalized to patient height), as MP is influenced by the quantity of aerated lung tissue available for tidal ventilation (normalized to C_{RS} or FRC) [21]. In patients with ARDS, a low C_{RS} is associated with increased mortality [56]. One study found that MP, measured 20 min after a recruitment maneuver, was initially not associated with patient outcomes. However, it became associated when normalized to well-inflated tissue (assessed by chest imaging) or when normalized to C_{RS} [57]. However, a study demonstrated that MP, whether normalized to PBW or to C_{RS}, showed similar predictive discrimination when compared to absolute MP in ARDS patients [58]. Currently, there is no consensus on whether MP should be normalized.

6. Clinical implications and future research

While the association between higher MP and its cumulative effect on ICU mortality has been established [40,43], there are no RCTs demonstrating efficacy of a ventilatory strategy focused on limiting MP. The absence of a clear VILI mitigation threshold, coupled with variations in calculation methods, introduces additional complexity for designing a simple strategy to be implemented in such an RCT. Therefore, specific clinical recommendations on the routine use of MP remain premature.

As we wait for RCTs, next steps involve further exploring the relationship between MP and patient-centered outcomes, and exploring the individual contributions of each variable within the MP calculation. An area of particular interest is exploring the association between dynamic MP and patient outcomes as the necessary parameters are readily available for all mechanically ventilated patients. Furthermore, future research should consider exploring MP in a broader group of patients with acute hypoxemic respiratory failure, who similar to patients with ARDS, are also at a high risk for both VILI and mortality. These investigations have the potential to both simplify and extend the applicability of MP to a larger group of critically ill patients.

7. Conclusion

Despite advances in lung-protective ventilation strategies, patients with respiratory failure remain at risk for poor outcomes. The concept of MP serves as a comprehensive metric, quantifying the amount of energy transferred from the ventilator to the respiratory system over time. While promising as a target to further mitigate VILI, its clinical application is limited by the lack of a consensus on calculation methods, relevant thresholds, and RCTs demonstrating efficacy of such an approach. Until ventilators integrate MP measurements using the geometric method, dynamic MP offers a practical and continuous alternative for assessing VILI risk. While MP holds potential to enhance patient outcomes, further research is needed.

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CRediT authorship contribution statement

Stephan von Düring: Writing – review & editing, Writing – original draft, Visualization, Validation, Project administration, Investigation, Conceptualization. **Ken Kuljit S. Parhar:** Writing – review & editing, Validation, Investigation. **Neill K.J. Adhikari:** Writing – review & editing, Validation, Investigation. **Martin Urner:** Writing – review & editing, Validation, Investigation. **S. Joseph Kim:** Writing – review & editing, Validation. **Laveena Munshi:** Writing – review & editing, Validation. **Kuan Liu:** Writing – review & editing, Validation. **Eddy Fan:** Writing – review & editing, Writing – original draft, Validation, Supervision, Investigation, Conceptualization.

Declaration of competing interest

SvD has received speaking honoraria from Löwenstein Medical.

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