Progress of mechanical power in the intensive care unit

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Abstract

Mechanical power of ventilation, currently defined as the energy delivered from the ventilator to the respiratory system over a period of time, has been recognized as a promising indicator to evaluate ventilator-induced lung injury and predict the prognosis of ventilated critically ill patients. Mechanical power can be accurately measured by the geometric method, while simplified equations allow an easy estimation of mechanical power at the bedside. There may exist a safety threshold of mechanical power above which lung injury is inevitable, and the assessment of mechanical power might be helpful to determine whether the extracorporeal respiratory support is needed in patients with acute respiratory distress syndrome. It should be noted that relatively low mechanical power does not exclude the possibility of lung injury. Lung size and inhomogeneity should also be taken into consideration. Problems regarding the safety limits of mechanical power and contribution of each component to lung injury have not been determined yet. Whether mechanical power-directed lung-protective ventilation strategy could improve clinical outcomes also needs further investigation. Therefore, this review discusses the algorithms, clinical relevance, optimization, and future directions of mechanical power in critically ill patients.

Keywords: Mechanical power; Mechanical energy; Ventilator-induced lung injury; Lung-protective ventilation; Acute respiratory distress syndrome

Introduction

Mechanical ventilation acts as a double-edged sword. It can rest the lungs and improve oxygenation, but it can also cause lung injury when inappropriately used. A series of pulmonary complications caused by mechanical ventilation are known as ventilator-induced lung injury (VILI).[1] The growing understanding of the injury mechanism helps researchers to identify risk factors of lung injury, including tidal volume,[2] respiratory rate,[3] pressures,[4,5] and flow.[6]

It has been proposed in recent years that the process of gas delivery by a ventilator can actually be regarded as that of power transference.[7] Hence, mechanical power of ventilation, which combines tidal volume, respiratory rate, and airway pressure, was proposed as a possible factor leading to VILI. The greater the power, the more likely a lung injury will occur.

What is Mechanical Energy/Power?

The concept of work of breathing may be more familiar to us. It refers to the total expenditure of energy developed by the respiratory muscles to overcome the resistance of ventilation during spontaneous breathing and is equal to the integral of the trans-pulmonary pressure and tidal volume.[8]

In physics, energy is defined as the capacity to do work, and work is the energy transmitted by a force. Doing work is accompanied by the transfer of energy. It is also applied in respiratory physiology. Therefore, the concept of mechanical energy of ventilation is essentially derived from that of work of breathing and thus can be defined as the energy delivered to the respiratory system or lungs by a ventilator. Mechanical power is the total energy expended over a period of time and is usually expressed as joules per minute (J/min).

How can Mechanical Energy/Power be Calculated?

The "gold standard": the geometric method

Since there is little difference between mechanical energy and work of breathing, it is not hard to conclude that mechanical energy equals the integral of airway pressure and tidal volume. For every cycle of controlled ventilation, mechanical energy is defined as the area between the inspiratory limb of the pressure and the volume axis.[9] Volume here refers to the tidal volume, while pressure

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refers to either airway pressure or trans-pulmonary pressure, corresponding to mechanical energy applied to the respiratory system or lungs, respectively. The geometric method, which measures the area under the pressure-volume curve, is the "gold standard" for calculating the mechanical energy. However, this method is not practical for clinicians without the help of a ventilator that automatically measures the mechanical energy.

Mechanical power during volume-controlled mode

To understand the calculation of mechanical power, the classic equation of motion must be reviewed as:

$$P_{\text{peak}} = \Delta P + P_R + \text{PEEP},$$

where P_{peak} is the peak airway pressure, ΔP is the elastic pressure or driving pressure that overcomes the elastic recoil of alveoli, P_{R} is the resistive pressure that overcomes airway resistance and PEEP is positive end-expiratory pressure.

Under volume-controlled mode, the area under pressure-volume curve that measures the mechanical energy can be calculated by integrating each component of peak airway pressure with tidal volume. As Figure 1 shows, provided that the inspiratory flow is constant where the elastic pressure is linearly related to tidal volume, the integral of elastic pressure with tidal volume will be half of their product, namely, the area of a triangle. The integral of either resistive pressure or PEEP with tidal volume is their simple product. Hence, the calculation of mechanical energy can be simplified as the area of a trapezoid, with the peak pressure as the long side, PEEP plus resistance pressure as the short side and tidal volume as the height. Finally, mechanical energy can be expressed as follows:

$$\begin{aligned}\text{Mechanical energy} &= 0.098 \times 1/2 \times V_{\text{T}} \times (P_{\text{peak}} + \text{PEEP} + P_{\text{R}}) \\ &= 0.098 \times V_{\text{T}} \times (P_{\text{peak}} - 1/2 \times \Delta P),\end{aligned}$$

where V_{T} represents for tidal volume and ΔP is equal to plateau pressure minus PEEP. About 0.098 is a conversion factor to obtain the result from liter·cmH_2O to J/min. Mechanical power is the product of mechanical energy and respiratory rate. For example, under volume-controlled mode and constant inspiratory flow, tidal volume = 400 mL, respiratory rate = 15/min, peak pressure = 20 cmH_2O, plateau pressure = 15 cmH_2O, and PEEP = 5 cmH_2O, the mechanical energy will approximately be 0.6 J, and mechanical power will be 9 J/min.

Mechanical power during pressure-controlled mode

It has been proven that the mechanical power calculated by the equation above has good consistency with that calculated by the geometric method, either in acute respiratory distress syndrome (ARDS) or non-ARDS patients,[7] for either the supine or prone position.[10] However, it should be kept in mind that this formula is correct only under the condition of volume-controlled mode and constant inspiratory flow. It is not suitable for pressure-controlled mode,[11] in which the airway resistance is not constant and the pressure-volume relationship is not linear. Nevertheless, Becher *et al*[12] show that under the assumption of an ideal "square wave" of airway pressure in pressure-controlled mode, mechanical energy could be calculated according to another simplified formula as follows:

$$\text{Mechanical energy} = 0.098 \times V_{\text{T}} \times P_{\text{peak}}$$

The value calculated by this formula undoubtedly overestimates the true value, but the two values are highly correlated with a small bias caused by disregarding the inspiratory pressure rise time, which seems to be acceptable for clinicians.[12]

Mechanical power during pressure support mode

Spontaneous breathing complicates the situation by inducing the opposite change on airway pressure in

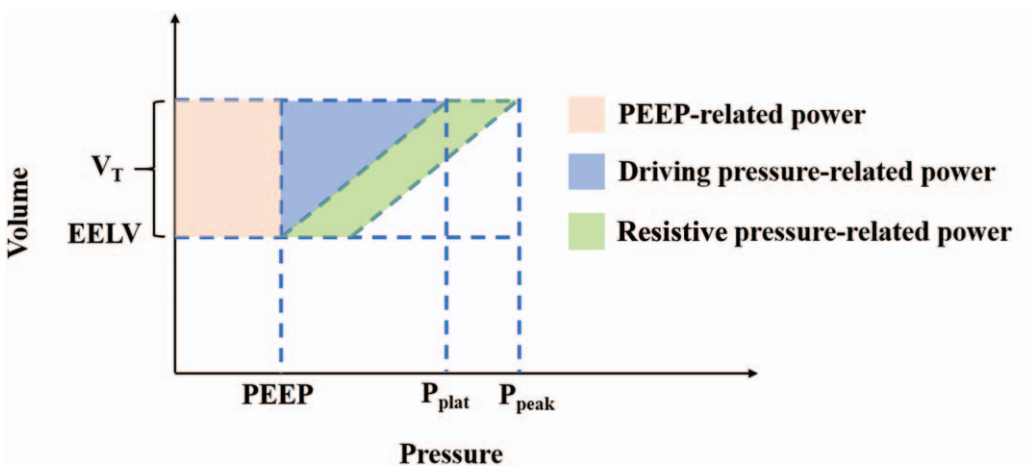

Figure 1: Schematic diagram that shows the calculation of mechanical energy. Under volume-controlled mode with constant inspiratory flow, mechanical energy can be divided into three components: PEEP-related (yellow region), driving pressure-related (blue region; to overcome the elastic recoil of alveoli), and resistive pressure-related (green region; to overcome airway resistance) energy. V_T: Tidal volume; EELV: End-expiratory lung volume; PEEP: Positive end-expiratory pressure; P_{plat}: Plateau airway pressure; P_{peak}: Peak airway pressure.

contrast to the ventilator.[13] Therefore, the peak pressure and power will be underestimated. The only way to solve this problem seems to be inserting an esophageal balloon that measures the trans-pulmonary pressure and using the geometric method. During pressure-support ventilation, the actual "peak pressure" is the plateau pressure assessed by end-inspiratory airway occlusion.[14,15] Although the pressure is not linearly related to the volume in this case, the product of plateau pressure and tidal volume as the simplified mechanical energy may still provide similar information to the actual value.

However, patient-ventilator asynchrony, not uncommon in moderate-to-severe ARDS, is another factor that contributes to VILI. Accompanied by spontaneous breathing, it allows the tidal volume, flow, and airway pressure to fluctuate, which confuses clinicians when calculating the mechanical power. In addition, the distribution of tidal ventilation is different with or without spontaneous inspiratory effort.[16] It is currently unknown whether the distribution of power is the same as that of tidal volume and whether it has an impact on VILI. More studies are eagerly awaited.

Summary and special considerations in mechanical power calculation

Mechanical power can be calculated using different methods [Table 1]. For better clinical use, the unit conversion coefficient 0.098 is usually replaced by 0.1 with only minor bias. It should be also noted that the parameter PEEP in these equations refers to the total PEEP (set PEEP + intrinsic PEEP).

Mechanical Power as a Promising Indicator of VILI

As mentioned earlier, mechanical power includes three components – respiratory rate, tidal volume, and airway pressure. Each of them contributes to VILI. Therefore, mechanical power is not a new concept but a collection of traditional indicators reflecting VILI. However, the overall power, rather than any single factor, seems to better predict VILI.

In an experiment by Cressoni *et al*,[9] healthy piglets were ventilated with high trans-pulmonary pressure and tidal volume for 54 h, and the respiratory rate was the only difference between the groups. They found that 12 J/min was the threshold power that induced VILI. To confirm this finding, they applied a low tidal volume with a high respiratory rate to reach a power greater than 12 J/min, and all the piglets in this group developed VILI. In the same series of their research,[19] the inspiratory-to-expiratory ratio was adjusted to change the inspiratory flow. A higher flow was associated with VILI, which may be explained by the fact that under the same tidal volume, a shorter inspiratory time means a higher airway pressure corresponding to a higher power. In another study, a correlation between power and serum fibrosis biomarkers has been found in ARDS patients.[20]

Mechanical power also has a good performance in predicting patient outcomes. Several large retrospective studies have revealed a close relationship between mechanical power and mortality in intensive care unit patients[17,21-23] [Table 2]. In a randomized trial involving 1010 ARDS patients, the use of higher PEEP was associated with a higher risk of 28-day mortality, despite similar driving pressure between groups.[24] Another study found that increasing PEEP without significant reduction of driving pressure was associated with a significant increase in mechanical power, which suggested mechanical power may be the link between driving pressure and morality.[25]

VILI is not only dependent on ventilator settings, but also influenced by lung size and inhomogeneity, which explains why ARDS patients are more vulnerable to VILI than other patients.[26,27] A "baby lung" of ARDS bears more power on per ventilated lung unit. Therefore, the concept of "intensity," which is mechanical power normalized by the aerated lung tissue volume, was proposed.[28] When

Table 1: Different methods for mechanical power calculation.

Year	First author	Calculation	Note
2016	Cressoni M[9]	$Power_{rs} = RR \cdot \int_{EELV}^{EELV+V_T} f(V)dV$ f(V) = airway pressure at given volume	Known as the "gold standard" method for both controlled and assisted ventilation modes
2016	Gattinoni L[7]	$Power_{rs} = RR \cdot \left\{ V_T^2 \cdot \left[\frac{1}{2} \cdot EL_{rs} + RR \cdot \frac{(1+I:E)}{60 \cdot I:E} \cdot R_{aw} \right] + V_T \cdot PEEP \right\}$ $Power_{rs} = 0.098 \times V_T \times RR \times (P_{peak} - 1/2 \times \Delta P)$	Applicable for volume-controlled mode with constant inspiratory flow
2016	Guerin C[17]	$Power_{rs} = 0.098 \times V_T \times RR \times \Delta P$	Simple but considers only the driving pressure-related power
2019	Becher T[12]	$Power_{rs} = 0.098 \times V_T \times RR \times (\Delta P + PEEP)$	Applicable for pressure-controlled mode Overestimates but has good consistency with the actual power
2019	Giosa L[18]	$Power_{rs} = \frac{V_T \times RR \times (P_{peak} + PEEP + F/6)}{20}$	Another simple equation to estimate mechanical power without any intervention under volume-controlled ventilation

$Power_{rs}$: Mechanical power of respiratory system; RR: Respiratory rate; EELV: End-expiratory lung volume; V_T: Tidal volume; EL_{rs}: Elastance of respiratory system; *I*:*E*: Inspiratory-to-expiratory ratio; R_{aw}: Airway resistance; PEEP: Positive end-expiratory pressure; P_{peak}: Peak airway pressure; ΔP: Driving pressure; *F*: Inspiratory flow.

Table 2: MP and clinical outcomes.

Year	Authors	Population	Outcomes	Key point
2016	Guérin *et al*[17]	787 ARDS patients enrolled in two independent randomized controlled trials, namely, ACURASYS and PROSEVA	MP was related to survival: an increase in the hazard ratio of 1.03 per unit of power	No safe dose of mechanical ventilation
2018	Serpa Neto *et al*[21]	8207 patients from the databases of the MIMIC-III and eICU studies	Even at a low V_T and a low ΔP condition, a high MP was associated with in-hospital mortality [OR: 1.70 (1.32–2.18)]	MP adds additional information beyond volume and pressure
2019	Zhang *et al*[22]	5159 ARDS patients from eight randomized controlled trials conducted by the ARDS-Net	The discrimination of MP normalized to predicted body weight in predicting mortality was significantly better than the absolute MP ($P = 0.011$ for DeLong test)	The effect of MP on VILI was dependent on the functional lung size

MP: Mechanical power; ARDS: Acute respiratory distress syndrome; ACURASYS: ARDS et Curarisation Systematique; PROSEVA: Effect of Prone Positioning on Mortality in Patients With Severe and Persistent Acute Respiratory Distress Syndrome; MIMIC: Medical Information Mart for Intensive Care; V_T: Tidal volume; ΔP: Driving pressure; OR: Odds ratio; VILI: Ventilated-induced lung injury.

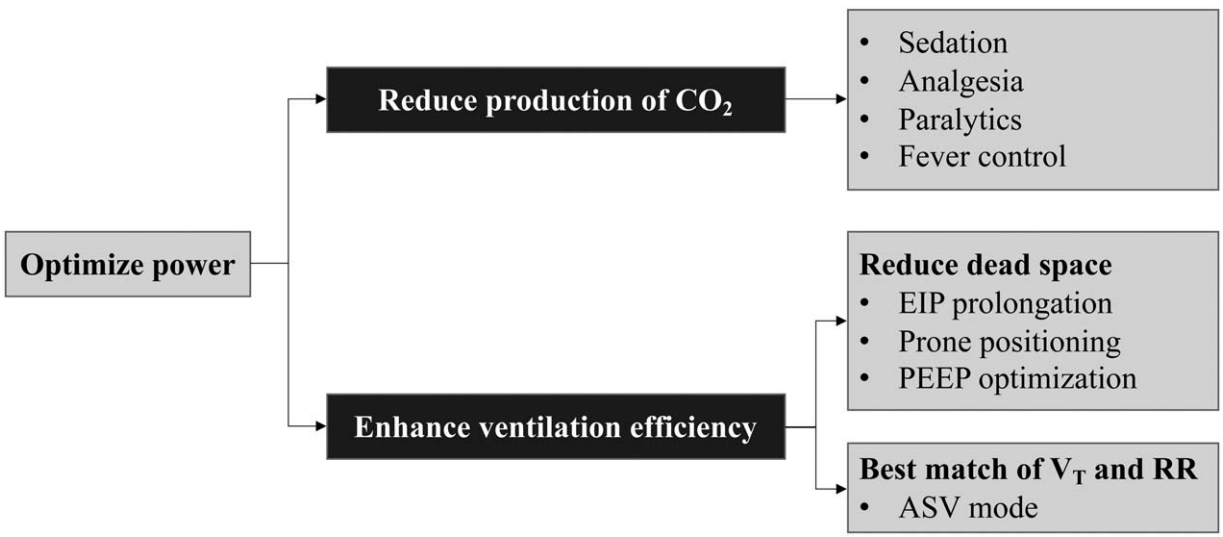

Figure 2: Strategies to optimize mechanical power. CO_2: Carbon dioxide; EIP: End-inspiratory pause; PEEP: Positive end-expiratory pressure; V_T: Tidal volume; RR: Respiratory rate; ASV: Adaptive support ventilation.

normalized by the compliance of respiratory system, a surrogate for lung size, mechanical power seems to better predict mortality outcome in patients with ARDS than the mechanical power alone.[22] On the contrary, compared with an intermediate tidal volume strategy, a low tidal volume ventilation strategy (6 mL/kg *vs.* 10 mL/kg predicted body weight) did not result in more ventilator-free days in patients without ARDS,[29] who have a normal functional residual capacity so that the "intensity" imposed on them will not reach a critical value that injuries the lungs.

Strategies to Optimize Mechanical Power

Since mechanical power is closely related to VILI, it is crucial to keep the power as low as possible. According to its equation, tidal volume and respiratory rate can be limited to control power. However, inadequate minute ventilation and thus carbon dioxide retention may be the price that has to be paid when we try to minimize power by decreasing minute ventilation. While mild hypercapnia is probably acceptable, severe hypercapnia could raise the risk of acute cor pulmonale.[30] To optimize mechanical power without excessively elevated partial pressure of arterial carbon dioxide ($PaCO_2$), there are two main ways: to reduce the production of carbon dioxide or to enhance ventilation efficiency[31] [Figure 2]. Ventilation efficiency is the ability of carbon dioxide (CO_2) clearance at the expense of mechanical power needed. To enhance ventilation efficiency means more CO_2 clearance without an increase of mechanical power or less mechanical power without decrease of CO_2 clearance.

Reduce the production of CO_2

Several factors including fever, pain, and respiratory distress will increase oxygen consumption and production

of CO_2 in critically ill patients. More ventilation demand is needed to excrete extra CO_2, which may contribute to VILI. Production of CO_2 can be reduced by fever control, sedation, analgesia, and paralytics if necessary.

Reduce dead space to promote CO_2 clearance

Some authors have reported that end-inspiratory pause prolongation is a feasible maneuver to decrease dead space-to-tidal volume ratio and $PaCO_2$ without change of minute ventilation.[32,33] Prone positioning improved gas distribution and thus reducing dead space for some patients with ARDS, and those with decreased $PaCO_2$ after prone positioning had better outcomes.[34] Moreover, higher PEEP level is associated with more over-distension that contributes to dead space. An optimized PEEP may reduce both dead space and mechanical power.

Reach the best match of tidal volume and respiratory rate

Another way to enhance ventilation efficiency gains its inspiration from the minimal work of breathing principle described by Otis *et al*.[35] Based on Otis' equation, tidal volume and respiratory rate can be optimized under a fixed minute ventilation to reach the lowest mechanical power by a ventilator mode named adaptive support ventilation.[36] In addition, a refined adaptive support ventilation mode was even more efficient in minimizing total mechanical power (16.8 ± 3.9 J/min *vs*. 18.6 ± 4.6 J/min) with the same level of $PaCO_2$ (39.1 ± 5.8 mmHg *vs*. 38.4 ± 4.2 mmHg) according to Becher *et al*'s pilot study.[37]

"Safety Threshold" of Mechanical Power

If VILI could be expressed as a function of mechanical power, there may exist a critical threshold value above which VILI is very likely to develop. Finding the "safety threshold" for mechanical power would undoubtedly guide the assessment and prevention of VILI. We learned from a study that VILI developed in healthy piglets if a mechanical power threshold of 12 J/min was exceeded.[9] Lung size and inhomogeneity that would increase vulnerability to VILI were well controlled and comparable between groups. However, the properties of the lung vary in critically ill patients and the influence of species on the power threshold is unknown. Therefore, the direct translation of this threshold to the clinical setting would not be feasible. A large retrospective study found a consistent increase in the risk of death with mechanical power higher than 17.0 J/min.[21] Yet a specific threshold was not available. To find a safety threshold of mechanical power, it should at least be normalized for the amount of aerated lung volume.[38]

Duration of hazardous power should also be taken into account since lung damage usually develops over time rather than happening at a specific time point. If the value of mechanical power maintains at a hazardous level or even increases progressively over time, suggesting the deterioration of lung condition, alternative methods such as extracorporeal respiratory support should be considered.[39] The duration of mechanical ventilation before extracorporeal membrane oxygenation (ECMO) was found an independent risk factor associated with a poor prognosis.[40] A retrospective study compared the baseline ventilation settings in patients with severe ARDS who received ECMO and those who did not.[41] Baseline mechanical power tended to be higher in patients who finally received ECMO than those who did not (23 ± 35 mJ·min^{-1}·L^{-1} *vs*. 13 ± 9 mJ·min^{-1}·L^{-1}). Another multi-center prospective cohort study of patients undergoing ECMO for ARDS has shown a markedly reduced mechanical power (26.1 ± 12.7 J/min *vs*. 6.6 ± 4.8 J/min) after ECMO initiation and the application of ultra-protective ventilation.[42] Nevertheless, it remains to be explored that above what level of mechanical power the initiation of extracorporeal respiratory support would improve patients' outcomes.

Is It Safe Enough for Low or Lower Mechanical Power?

According to what we learned from Cressoni *et al*'s study,[9] it may be assumed that as long as mechanical power is kept below a safety threshold, higher tidal volume should not be injurious. However, the assumption was soon questioned. Under the control of relatively low and comparable mechanical power between groups, VILI still developed in the high tidal volume group after invasive ventilation for 2 h in rats with ARDS induced by endotoxin.[43,44] Two studies above suggested that tidal volume can independently contribute to VILI regardless of the degree of mechanical power. Clinicians would be misled and accept a higher tidal volume if they were to take mechanical power as the sole determinant of VILI.

Another consideration comes with PEEP. While "the lower PEEP, the lower power" may be true,[45] clinicians should decrease PEEP with caution because the lungs also tend to collapse with decreasing PEEP especially in ARDS patients, which results in (1) increased shunt that depresses oxygenation; (2) decreased compliance and a higher driving pressure-related power; (3) more atelectasis and tidal recruitment; and (4) a smaller lung size and even higher "intensity" [Figure 3].

In an animal experiment that tested the influence of PEEP on power and lung histology in healthy piglets ventilated for 50 h, mechanical power was similar at 0 (8.8 ± 3.8 J/min), 4 (8.9 ± 4.4 J/min), and 7 (9.6 ± 4.3 J/min) cmH_2O, while parameters related to lung atelectasis and inflammation decreased at PEEP of 7cmH_2O compared with 0 cmH_2O, which suggested the protective effect of PEEP from atelectrauma at a certain range.[46] In an experimental model of acute lung injury, Yoshida *et al*[47] found that spontaneous breathing at low PEEP level not only generated greater tidal recruitment, but also caused a larger degree of pendelluft (shift of air within the lung from non-dependent to dependent regions without a change in tidal volume), which resulted in local overstretch of the dependent lung. Compared with lower PEEP, an optimized PEEP set after lung recruitment reduced both tidal recruitment and pendelluft.[48,49] Hence, it should be noted that the mechanical power itself does not reflect atelectrauma.

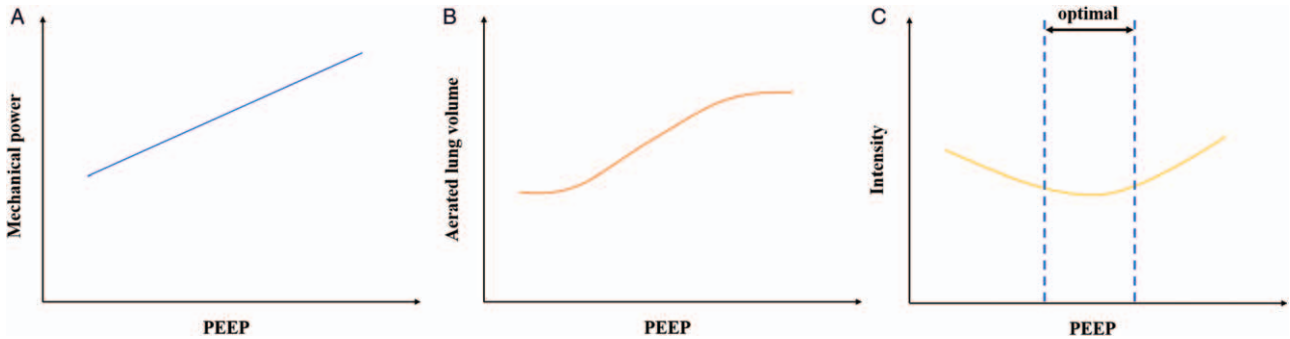

Figure 3: Relationships between mechanical power (A), aerated lung volume (B), intensity (C) and PEEP. Usually, mechanical power increases with increasing PEEP. In a certain range, the aerated lung volume expands the most with per unit increase in PEEP. As a result, the power distributed over the aerated lung volume, that is, the intensity, would reach its lowest value in that "optimal range". PEEP: Positive end-expiratory pressure.

Another pilot study showed that in some ARDS patients, an increased regional mechanical power at independent lung regions was found using electrical impedance tomography during a decremental PEEP trial,[50] indicating a potential risk for increased regional volutrauma with lower PEEP in these subjects. Therefore, the factors above should be considered when trying to decrease PEEP to minimize power.

Future Directions for Mechanical Power

Current progress and limitations in mechanical power have identified potential fields for further exploration. Firstly, an accurate and convenient algorithm for mechanical power calculation during assisted ventilation is required since current equations will fail when spontaneous breathing exists. Secondly, the safety thresholds of mechanical power probably vary for different lung conditions. The cutoff value for severely injured lungs is supposed to be much lower than that for normal or mildly injured lungs. Mechanical power normalized to lung size and inhomogeneity may have better performance. Thirdly, the components of mechanical power contribute unequally to its value as shown by Gattinoni *et al*,[7] which creates a question that which component is most important to VILI. To answer this question may require a deeper understanding in the molecular mechanism of VILI.[51] The one most related may be adjusted preferentially when the safety threshold of mechanical power is exceeded. In all, more data and investigations are required to reveal the mystery of mechanical power.

Conclusions

Mechanical power is an attractive concept that combines various components contributing to VILI. It may provide better insights for lung-protective ventilation. Simplified equations have enabled the easy estimation of mechanical power at the bedside. While higher power is associated with VILI and worse outcomes, low power does not exclude the possibility of lung injury. Other factors associated with VILI including lung size, inhomogeneity, and patient-ventilator asynchrony should also be considered. Problems regarding improved algorithm, safety limits, and most important component of mechanical power have not been determined yet. Further research is required to evaluate whether mechanical power-directed lung-protective ventilation strategy could improve clinical outcomes.

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Conflicts of interest

None.

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