



Ten Reasons to Use Mechanical Power to Guide Ventilator Settings in Patients Without ARDS

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3.1 Introduction

Mechanical ventilation is frequently used in patients without acute respiratory distress syndrome (ARDS): during surgery, in the intensive care unit (ICU; e.g., to support breathing in respiratory and neurological failure), and for artificial respiration in cardiac arrest. Even though mechanical ventilation is a life-saving strategy, it may cause ventilator-induced lung injury (VILI). Analyses of the main factors involved in VILI have focused on separate evaluation of static parameters—such as tidal volume; peak (P_{peak}), plateau (P_{plat}), and driving (ΔP) pressures; and positive end-expiratory pressure (PEEP)—and of dynamic ones—airflow, inspiratory time, and respiratory rate. However, in the real-world setting, these factors interact with each other. Thus, combining these factors into a single parameter—the “mechanical power” imparted to the lung by the ventilator—may be a more suitable strategy for both research and clinical purposes.

Although early implementation of protective ventilation in patients without ARDS has been associated with better prognosis [1], optimization of mechanical ventilation settings is considered less important in daily clinical practice. In this chapter, we will discuss ten questions regarding the importance of the sole single ventilator parameter that has been associated with mortality in patients without ARDS: mechanical power. This parameter will then be discussed, focusing on the use of different formulas for its calculation and on the evidence of its impact on lung damage from preclinical and clinical studies.

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3.2 Is Tidal Volume Associated with Mortality in Patients Without ARDS? No

The tidal volumes that are used in mechanically ventilated patients without ARDS have progressively been decreasing; however, the evidence for benefits of low tidal volumes remains scarce. A systematic review evaluated the change in tidal volume during a 39-year period (from 1975 to 2014) [2] and showed that tidal volumes have decreased significantly in the ICU (annual decrease of 0.16 ml/kg) and in the operating room (annual decrease of 0.09 ml/kg). In the PRoVENT study [1], tidal volume was around 8 ml/kg predicted body weight (PBW) and did not differ between surviving patients and those who died. In a secondary analysis of the PRoVENT, tidal volume was not identified as a potential parameter associated with survival in patients without ARDS [3], which could be attributed to the fact that most patients were protectively ventilated. In the PRoVENT trial, low tidal volume and intermediate tidal volume strategies were compared in patients without ARDS, focusing on the number of ventilator-free days and mortality rate at 28 days [1]. The low-tidal-volume group started at 6 ml/kg PBW, with decrements of 1 ml/kg PBW every hour to a minimum of 4 ml/kg PBW, whereas the intermediate-tidal-volume group started at 10 ml/kg PBW. P_{plat} was maintained at less than 25 cmH₂O; if it exceeded this threshold, tidal volume was progressively reduced by 1 ml/kg PBW. Ventilator-free days and mortality rate at 28 days did not differ between the low-tidal-volume (mean = 7.3 ml/kg PBW) and intermediate-tidal-volume (mean = 9.1 ml/kg PBW) groups. The intermediate-tidal-volume group showed higher P_{plat} and ΔP in the first 3 days of the study, but still within a protective range for patients without ARDS. Additionally, use of the low-tidal-volume strategy was associated with CO₂ retention and respiratory acidosis, perhaps related to less efficient alveolar ventilation. Furthermore, the use of oversedation to maintain low tidal volume may be associated with increased delirium, ventilator asynchrony, and the possibility of effort-induced lung injury. To date, there are no clinical trial data showing benefit or harm of low-tidal-volume ventilation in patients without ARDS. Since ARDS is only very rarely recognized at its onset, the safest approach is to use protective ventilation with tidal volume <8 ml/kg PBW.

3.3 Is Driving Pressure Associated with Mortality in Patients Without ARDS? No

The driving pressure of the respiratory system (ΔP_{RS}) can be easily calculated at the bedside as P_{plat} minus pressure at end-expiratory occlusion (PEEP + intrinsic PEEP). The ΔP_{RS} represents the tidal volume normalized by the respiratory system compliance. Since the compliance of the respiratory system is proportional to the volume of aerated lung, ΔP_{RS} is the tidal volume corrected for the end-expiratory lung volume, thus estimating the strain during tidal breath. In other words, if tidal volume remains constant, if the compliance of the respiratory system is low, the ΔP_{RS} will proportionally increase; conversely, if the compliance of the respiratory system is high, the ΔP_{RS} will proportionally decrease. Furthermore, under constant

respiratory system compliance, ΔP_{RS} will increase at higher tidal volumes. Finally, ΔP_{RS} might be affected by the effects of PEEP on non-aerated lung areas. At a constant tidal volume, if PEEP increases lung volume by recruitment of previously collapsed alveoli, ΔP_{RS} will decrease; if PEEP increases lung volume by overdistension of previously aerated alveoli, ΔP_{RS} will increase. In patients without ARDS, the lung volume at end-expiration and the compliance of the respiratory system are either within normal ranges or moderately reduced, and low PEEP levels are usually applied, thus avoiding alveolar overdistension. In a retrospective study, ΔP_{RS} on day 1 was not associated with hospital mortality in patients without ARDS [4]. A secondary analysis of the PRoVENT trial [3] showed no association between ΔP_{RS} (≤ 12 vs. >12 cmH₂O) and hospital mortality. However, caution is warranted, given the relatively small sample size of the study. Therefore, to date there is no indication to guide ventilatory strategy on the basis of ΔP_{RS} alone in patients without ARDS.

3.4 Is P_{peak} Associated with Mortality in Patients Without ARDS? Yes

P_{peak} is determined by the inspiratory flow and the position where pressure is measured along the respiratory circuit. In fact, it represents the total pressure gradient, at a fixed flow, to overcome the elastic, resistive, and viscoelastic properties of the respiratory system, the artificial airways, and the ventilator circuit. P_{peak} is one of the easiest respiratory variables to monitor at the bedside during mechanical ventilation. In patients with increased airway resistance, under volume-controlled ventilation, after occluding the airways at end-inspiration, the difference between P_{peak} and P_{plat} represents the resistive properties (patient and ventilator circuit), whereas during pressure-controlled ventilation, P_{peak} is close to P_{plat} , since flow at expiration is almost zero. In patients without increased airway resistance and/or ventilator circuit obstruction, P_{peak} is approximately equal to P_{plat} . A secondary analysis of PRoVENT reported a significant association between $P_{\text{peak}} > 18$ cmH₂O and inhospital mortality in patients without ARDS [3]. In conclusion, we suggest using a P_{peak} pressure < 30 cmH₂O and a $P_{\text{plat}} < 25$ cmH₂O in mechanically ventilated patients without ARDS in volume- and pressure-controlled ventilation.

3.5 Is PEEP Associated with Mortality in Patients Without ARDS? Yes

PEEP has been proposed to prevent or at least minimize atelectrauma during controlled mechanical ventilation. However, PEEP can also lead to lung injury due to overdistension (so-called volutrauma) and impair right ventricular function and hemodynamics. In a systematic review [5] of preclinical studies in small and large mammals, “high PEEP” (versus lower PEEP) or “PEEP” (versus no PEEP) was associated with improved respiratory system compliance and better oxygenation. However, “high PEEP” and “PEEP” were also associated with occurrence of hypotension, a reduction in cardiac output or development of hyperlactatemia, with no

differences in mortality. Based on the findings of this systematic review in experimental settings, it is uncertain whether PEEP at any level truly prevents lung injury, and most trials suggested potential harmful effects on the systemic circulation. Patients without ARDS who have less atelectasis and better compliance of the respiratory system seem to have a better balance between benefit and harm in favor of lower PEEP levels. Nevertheless, whether higher PEEP levels can be associated with clinical improvement over time is unknown. A meta-analysis of clinical trials [6] compared lower (2.0 ± 2.8 cmH₂O) vs. higher (9.7 ± 4.0 cmH₂O) levels of PEEP and found no benefits in terms of mortality and duration of invasive ventilation in surgical or medical ICU patients. In addition, pooling all data in a recent cohort [3] of patients without ARDS, high PEEP levels (>5 cmH₂O) were associated with higher hospital mortality. A prospective randomized controlled trial in patients without ARDS to test the effects of low PEEP (lowest possible PEEP between 0 and 5 cmH₂O) and high PEEP (PEEP of 8 cmH₂O) on the number of ventilator-free days and survival at day 28 is ongoing [7]. To date, the evidence suggests that PEEP should not exceed 5 cmH₂O in patients without ARDS.

3.6 Is Respiratory Rate Associated with Mortality in Patients Without ARDS? No

Respiratory rate is one of the easiest variables to measure at the bedside. It may be controlled by the ventilator, by the patient, or both. Usually, respiratory rate is set to keep an adequate level of arterial partial pressure of carbon dioxide (PaCO₂) and arterial pH. Experimental studies have shown that increased respiratory rate may be associated with VILI [8, 9]. From a physiological standpoint, the respiratory rate can reflect the number of cycles of injured stress and strain in the lung parenchyma. In the PRVENT trial [1], respiratory rate was higher in volume-controlled compared to pressure-support ventilation groups, regardless of tidal volume. Conversely, PaCO₂ was higher in the low-tidal-volume compared to intermediate-tidal-volume group during volume-controlled ventilation, but not in pressure-support ventilation, suggesting that the increased spontaneous breathing decreased the dead space, thus improving gas exchange. In conclusion, we suggest using assisted mechanical ventilation modes in patients without ARDS, thus allowing selection of an appropriate respiratory rate for the patient.

3.7 Mechanical Energy and Power Calculations: Should We Abandon More Complex Formulas in Favor of Simplified Ones? Yes

The contribution of the energy and power applied to the lung parenchyma during mechanical ventilation, resulting in VILI, is known as ergotrauma. Work is force \times distance and, in the case of the lungs, since pressure is force/area and volume is length \times area, the product of pressure and volume is force \times length, which is the work done during lung inflation. Work of breathing and energy are expressed in the same unit (J). Energy has been calculated as tidal volume (ml) \times ΔP (cmH₂O),

which gives a unit of ml/cmH₂O. (To convert from ml/cmH₂O to joules, all variables should be transformed to SI units.) Power is the rate of energy expenditure (Energy × Respiratory rate) [10, 11].

Different equations are available to calculate mechanical power. Since resistive properties are included, it should be mentioned that mechanical power calculation depends on the site of airway pressure measurement, i.e., whether at the end of the tracheal tube or at a transducer within the mechanical ventilator. In the first condition, only the resistance of the endotracheal tube is added to the airway resistance of the patient, while in the second condition, the total resistance encompasses the whole of the mechanical ventilator circuit, including the endotracheal tube. To correctly calculate mechanical power, the resistances of both the tracheal tube and mechanical ventilator should be subtracted or otherwise accounted for.

The complex equation initially described by Gattinoni et al. [12] is:

$$\text{Mechanical power (J / min)} = 0.098 \times \text{Respiratory rate} \\ \times \left\{ \begin{aligned} & \left(\text{Tidal volume} \right)^2 \times \left[\frac{1}{2} E_{rs} + \text{Respiratory rate} \times \left[\frac{(1 + I : E)}{60} \times I : E \right] \times R_{aw} \right] \\ & + \text{Tidal volume} \times \text{PEEP} \end{aligned} \right\}$$

where 0.098 is the conversion factor for units, E_{rs} is the respiratory system elastance, $I:E$ is the ratio between inspiratory and expiratory time, and R_{aw} is the airway resistance.

One important benefit of using this more complex formula is that it enables quantification of the relative contribution of each component (tidal volume, respiratory rate, ΔP_{RS} , PEEP, $I:E$, and airflow). This has been done previously by changing one parameter at a time and observing the overall effect on mechanical power [12]. Therefore, this formula takes into account the resistive properties and PEEP (and, consequently, lung volume changes caused by PEEP). The main disadvantages of this formula are that it is laborious to calculate and may be conditioned to perfect calibration of the mechanical ventilator, since more variables are used to calculate the mechanical power.

Furthermore, there are controversies regarding the computation of mechanical energy in a static condition, since there is no cyclic movement of the respiratory system after sole application of PEEP [10]. Therefore, when PEEP is included and lung volume is changed by PEEP application, both the static and dynamic components of mechanical energy should be calculated.

The static component of mechanical energy, which takes into account elastic mechanical power (MP_{ELAST}), can be calculated using the following formula:

$$MP_{ELAST} = 0.098 \times \text{Respiratory rate} \times \left[\text{Tidal volume} \times (P_{plat} - \text{PEEP}) \right] / 2$$

The dynamic component of mechanical energy, which takes into account resistive mechanical power (MP_{RES}), can be calculated using the following formula:

$$MP_{RES} = 0.098 \times \text{Tidal volume} \times \text{Respiratory rate} \\ \times \left(P_{peak} \times \Delta P_{RS} - 2 \times \text{PEEP} - \Delta P_{RS} \right) / 2$$

The simplest formula, which includes both elastic and resistive properties and is intended for use in critically ill patients [13], is composed of tidal volume, P_{peak} , respiratory rate, and ΔP [12]:

$$\text{Mechanical power (J / min)} = 0.098 \times \text{Tidal volume} \times \text{Respiratory rate} \\ \times \left(P_{\text{peak}} - \frac{1}{2} \times \Delta P_{\text{RS}} \right)$$

Serpa Neto et al. [13] tested the hypothesis that mechanical power would be independently associated with patient-centered outcomes in mechanically ventilated critically ill patients for 48 h. This study was performed in two large cohorts of ICU patients whose data were prospectively collected in two databases. Mechanical power was independently associated with higher in-hospital mortality, higher ICU mortality, fewer ventilator-free days, and longer ICU and hospital stays. One of the most important findings of this post hoc analysis was that, even at low tidal volume and low ΔP_{RS} , high mechanical power was associated with worse outcomes in critically ill patients. The combination of potential respiratory variables related to lung injury into a single parameter, instead of using only one respiratory variable, may have high predictive value for worse outcomes in the critically ill.

The concept of intensity, which is the distribution of mechanical power per unit of lung surface area [11, 14] or static compliance of the respiratory system ($C_{\text{RS}} = \text{Tidal volume} / \Delta P_{\text{RS}}$), has also been proposed.

For the aforementioned formula, intensity (J/min) is given as $= 0.098 \times \text{Tidal volume} \times \text{Respiratory rate} \times (P_{\text{peak}} - \frac{1}{2} \times \Delta P_{\text{RS}}) / \text{Tidal volume} / \Delta P_{\text{RS}}$.

$$\text{After simplification, Intensity (J / min)} = \text{Respiratory rate} \times \Delta P_{\text{RS}}^2 / 2$$

If PEEP and resistive properties are added to the mechanical power formula, the intensity would be stated as:

$$\text{Intensity (J / min)} = 0.098 \times \text{Respiratory rate} \times \left[P_{\text{peak}} - (\Delta P_{\text{RS}} / 2) \right] \times \Delta P_{\text{RS}}$$

A recent study investigated whether mechanical power normalized for predicted body weight (norMP) could be a better marker of mortality compared to other ventilator variables [15]. NorMP (AUC = 0.751) showed good predictive value for mortality compared to non-normalized mechanical power (AUC = 0.747). Mechanical power normalized by respiratory system compliance (a surrogate of intensity) was an even better predictor of mortality (AUC = 0.753).

3.8 Does Use of a Simple Formula Enable Calculation of Mechanical Power in Volume-Controlled Ventilation? Yes: Simply Change the Variables and Observe the Consequences

Usually, changing a single respiratory variable may not promote lung protection if this change is not followed by significant changes in lung mechanics [16]. This phenomenon can be observed in different clinical scenarios. However, calculation

of mechanical power using the simplest formula, which takes into account tidal volume, respiratory rate, P_{peak} , and ΔP_{RS} , can help understand why lung protection may not occur after changing only one respiratory variable.

The effects of different tidal volumes on mechanical power during volume-controlled ventilation have been simulated in a hypothetical patient with a PBW equal to 64 kg (the average body weight reported in patients without ARDS) [3]. The inspiratory:expiratory ratio was kept constant at 1:2. Three different respiratory system compliance values—0.02 l/cmH₂O (low), 0.04 l/cmH₂O (intermediate), and 0.06 l/cmH₂O (high)—were tested, with tidal volume reduced from 10 to 6 ml/kg PBW, total minute ventilation kept constant (9.6 l/min), and PEEP = 5 cmH₂O. To maintain minute ventilation constant, respiratory rate was increased from 15 to 25 bpm. As shown in Fig. 3.1, together with the increment in

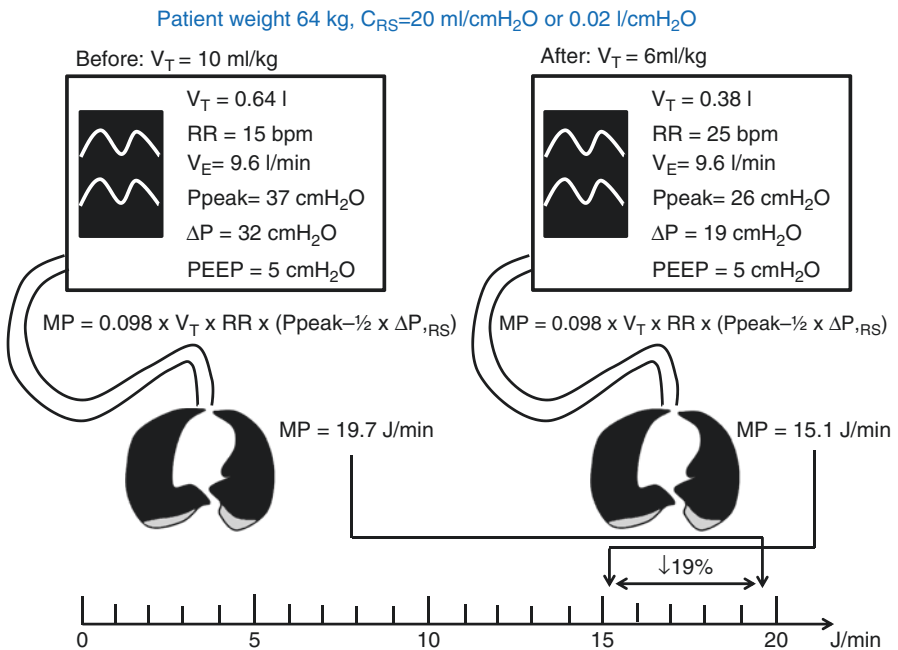
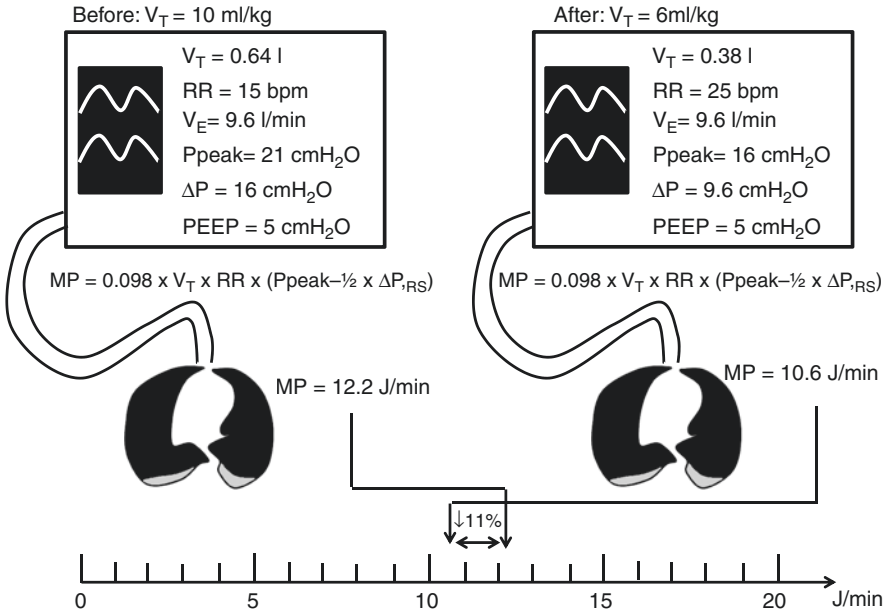


Fig. 3.1 Simulation of the effects of mechanical power (MP) during volume-controlled mechanical ventilation in a patient with a predicted body weight of 64 kg (the average body weight of patients without ARDS). Inspiratory:expiratory ratio kept constant at 1:2. Three different respiratory system compliance (C_{RS}) values—0.02 l/cmH₂O (low), 0.04 l/cmH₂O (intermediate), and 0.06 l/cmH₂O (high)—were tested, with tidal volume (V_T) reductions from 10 ml/kg predicted body weight (PBW) to 6 ml/kg PBW, while keeping total minute ventilation (V_E) constant (9.6 l/min) at positive end-expiratory pressure (PEEP) = 5 cmH₂O. To maintain constant minute ventilation, respiratory rate (RR) was increased from 15 to 25 bpm. A decrease in inspiratory time (from 1.3 to 0.8 s) and concomitant increase in airflow (from 0.36 to 0.59 l/s) are expected to follow, increasing the resistive pressure. Assuming a patient with constant airway resistance (4 cmH₂O/l/s), the increase in airflow would increase the resistive pressure by about 1.5 cmH₂O, with an impact in P_{peak} (one of the components of the mechanical power calculation). Reductions in V_T were associated with lower mechanical power in all conditions. Furthermore, the percent decrease in mechanical power after the reduction in V_T was greater at low compared to intermediate or higher respiratory system compliances (19% vs. 11% vs. 9%, respectively)

Patient weight 64 kg, $C_{RS}=40 \text{ ml/cmH}_2\text{O}$ or $0.04 \text{ l/cmH}_2\text{O}$



Patient weight 64 kg, $C_{RS}=60 \text{ ml/cmH}_2\text{O}$ or $0.06 \text{ l/cmH}_2\text{O}$

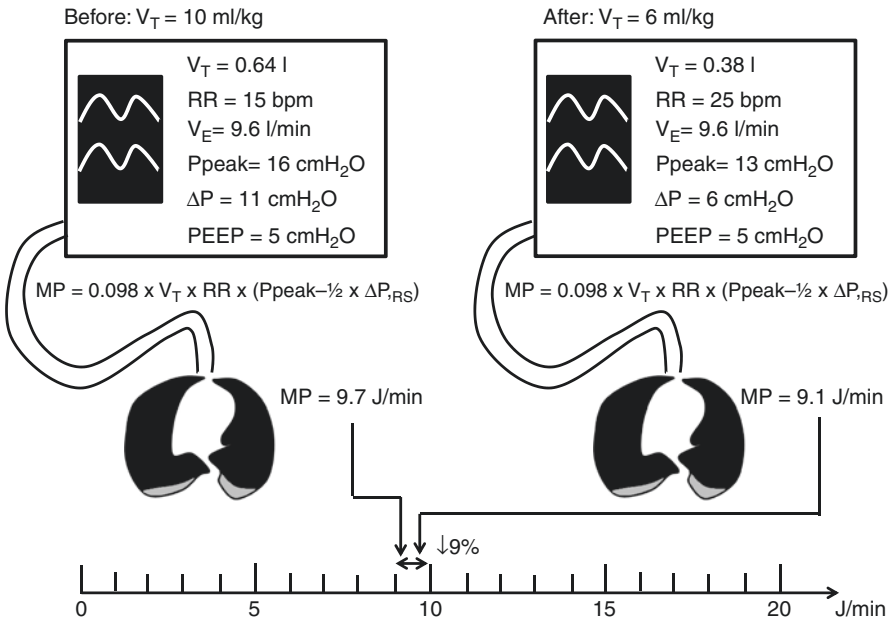


Fig. 3.1 (continued)

respiratory rate from 15 to 25 bpm, a decrease in inspiratory time (from 1.3 to 0.8 s) is expected, as well as a concomitant increase in airflow (from 0.36 to 0.59 l/s), which would increase the resistive pressure. Considering a patient with constant airway resistance (4 cmH₂O/l/s), the increase in airflow would increase the resistive pressure to approximately 1.5 cmH₂O, which would have an impact on P_{peak} . Reduction in tidal volume was associated with lower mechanical power in all conditions. Furthermore, the percentage of decrease in mechanical power after the reduction in tidal volume was greater at low compared to intermediate or higher compliances of the respiratory system (19% vs. 11% vs. 9%, respectively). This suggests that the effects of tidal volume reduction on mechanical power are greater at low respiratory system compliance.

Becher et al. [17], in a retrospective analysis of two previous studies, proposed two power equations for pressure-controlled ventilation (MP_{PCV}): a simplified formula,

$$\text{MP}_{\text{PCV}} = 0.098 \times \text{Respiratory rate} \times \text{Tidal volume} \times (\Delta P_{\text{insp}} + \text{PEEP})$$

and a comprehensive equation,

$$\begin{aligned} & \text{MP}_{\text{PCV(slope)}} \\ &= 0.098 \times \text{Respiratory rate} \times \left[\left((\Delta P_{\text{insp}} + \text{PEEP}) \times \text{Tidal volume} - \Delta P_{\text{insp}}^2 \times C \right) \right. \\ & \quad \left. \times \left(0.5 - R \times C / T_{\text{slope}} + \left(R \times C / T_{\text{slope}} \right)^2 \times \left(1 - e^{-T_{\text{slope}}/R \times C} \right) \right) \right] \end{aligned}$$

where R is resistance and C is compliance. Both formulas were compared to reference values obtained through integration of pressure-volume loops from the mechanical ventilator. The authors found that the simplified equation estimated mechanical power for pressure-controlled ventilation with a small bias, which resulted from not taking T_{slope} into account. On the other hand, the comprehensive equation was able to correct this bias, but requires knowledge of T_{slope} , resistance, and compliance. The formula proposed by Becher et al. was tested in a recent small validation study by van der Meijden et al. [18], in which the authors proposed a different equation:

$$\text{MP}_{\text{PCV}} = 0.098 \times \text{Respiratory rate} \times \text{Tidal volume} \times [\text{PEEP} + \Delta P_{\text{insp}} \times (1 - e^{-T_{\text{insp}}/R \times C})]$$

These authors found that, for higher mechanical power values, the method proposed by Becher et al. [17] was somewhat inaccurate, although testing in larger samples was still warranted. Meanwhile, Becher et al. tested the equation proposed by van der Meijden et al. using a dataset of 301 pressure-volume loops obtained from 42 patients [19]. The authors found that on average, the equation proposed by van der Meijden et al. led to underestimation of mechanical power, with a bias of -0.56 J/min and a correlation coefficient of $r^2 = 0.937$, which is

inferior to both the “simplified” and “comprehensive” equations originally proposed by Becher et al. ($r^2 = 0.981$ and $r^2 = 0.985$, respectively). In addition, the difference could be related to the assumption that, during pressure-controlled ventilation, end-inspiratory flow and pressure rise time are zero. In those respiratory cycles with end-inspiratory flow above zero, the formula will thus lead to underestimation of mechanical power, while pressure rise times above zero will lead to overestimation of mechanical power. The exchange of letters between these two groups of authors, with equation proposals and reanalysis of a larger dataset, is a step towards better understanding of mechanical power calculation during pressure-controlled ventilation [20].

3.9 Can Mechanical Power Be Computed During Assisted Ventilation? Yes

Mechanical energy and power, by definition, represent the total energy transferred from the mechanical ventilator in association with the respiratory muscles. To date, clinical studies have focused on relaxed respiratory muscles; therefore, only the mechanical power provided by the ventilator is computed [12, 21]. Nevertheless, the mechanical power generated by the respiratory muscles was calculated using an esophageal catheter in two pediatric studies [22, 23]. In an experimental study, mechanical power was calculated during pressure-support ventilation in rats. However, the authors chose to calculate only the portion attributable to the mechanical ventilator, which was reduced in pressure-support ventilation compared to pressure-controlled ventilation [24]. The calculation of lung mechanical power was done using the trapezoidal rule, as the integral of the inspiratory transpulmonary pressure versus the inspired tidal volume curve by numeric integration. A similar calculation was done in controlled mechanical ventilation [12].

There are further challenges to be overcome regarding calculation of respiratory system mechanical power during spontaneous breathing: (1) adjustment for chest wall movement; (2) distribution of mechanical power across the lung surface, which may not follow the same distribution determined by lung inhomogeneity [10]; and (3) the proper contribution of respiratory muscle activity to overall mechanical power under assisted mechanical ventilation.

3.10 Is Mechanical Power Associated with Lung Injury in Experimental Models? Yes

Recent studies have described the impact of mechanical power on VILI [25, 26, 29, 30]. In one such investigation, lung edema was observed when transpulmonary mechanical power was higher than 12.1 J/min [25]. Several analyses have been

done to evaluate the relative contribution of tidal volume and respiratory rate to mechanical power [12]. An experimental study [26] investigated the impact of high and low mechanical power, obtained as combinations of low and high tidal volume and respiratory rate, on VILI. Mechanical power was calculated using different formulas [12, 27, 28]. Even when combined with high tidal volume, low mechanical power resulted in greater lung damage, thus suggesting the importance of using protective low tidal volume in ARDS [26]. PEEP is also an important parameter to calculate mechanical power. In this line, Collino et al. investigated the effects of increasing PEEP values on mechanical power in healthy lungs [30]. For this purpose, piglets were ventilated by a tidal volume (14.9 ml/kg) similar to functional residual capacity (FRC), which gives an overall strain close to 1, and increasing levels of PEEP were tested (0, 4, 7, 11, 14, and 18 cmH₂O) for 50 h. Mechanical power was calculated by the formula proposed by Gattinoni et al. [12], which takes into account the $PEEP \times \Delta V$ related to PEEP.

The authors showed that mechanical power was constant from 0 up to 7 cmH₂O, which could be reflected by improvement of lung elastance within this range. On the other hand, increasing PEEP from 7 up to 18 cmH₂O was associated with greater damage and relevant hemodynamic instability. This experimental study emphasizes that PEEP may be a major determining factor of damage in certain settings, while in others, the driving pressure, respiratory rate, and tidal volume may be more important. More recently, Felix et al. [31] tested the hypothesis that the impact of an abrupt increase in tidal volume would be attenuated if tidal volume were increased slowly enough to reduce alveolar mechanical heterogeneity and VILI. Although the primary focus was not related to mechanical power itself, the authors showed that extending the adaptation period increased cumulative power and induced VILI, since animals were exposed to injurious strain earlier and for a longer time [32]. Therefore, the rapid recognition of high mechanical power mitigates lung damage.

3.11 Is Mechanical Power Associated with Mortality in Patients Without ARDS? Yes

In their dual-dataset study, Serpa Neto et al. [13] observed an association between increased risk of death and mechanical power higher than 17.0 J/min (Fig. 3.2). The most important result of this post hoc analysis was that even at low tidal volume and low ΔP , high mechanical power was associated with fewer ventilator-free days, longer ICU stays, and higher inhospital mortality. Similar findings were found in a secondary analysis of 1705 mechanically ventilated patients without ARDS [33]. By using the same formula described by Serpa Neto et al. [13], the authors found that ΔP , P_{plat} , and mechanical power were associated with inhospital mortality.

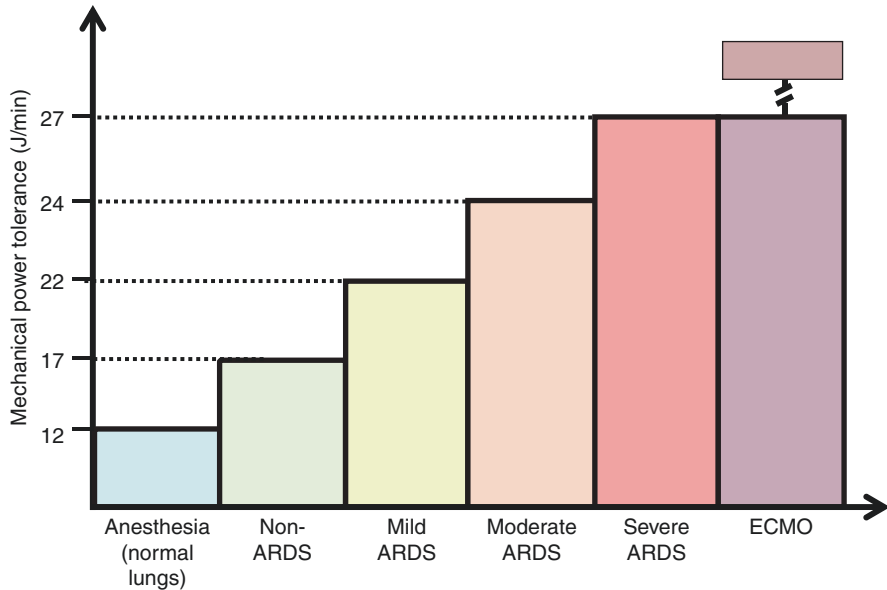


Fig. 3.2 Mechanical power tolerance. For anesthesia in a patient with normal lungs, the reported values are within very low limits, around 12 J/min. In critically ill patients without acute respiratory distress syndrome (non-ARDS), the threshold value of mechanical power to prevent ARDS is <17 J/min. In ARDS, regardless of severity, values are higher than in non-ARDS. For mild, moderate, and severe ARDS, the tolerated values are 22, 24, and 27 J/min. If mechanical power exceeds 27 J/min, extracorporeal membrane oxygenation (ECMO) should be considered

3.12 Conclusion

In patients without ARDS, ventilatory management has been associated with mortality. Commonly targeted ventilator parameters, such as tidal volume and ΔP , are not associated with mortality in patients without ARDS, while respiratory rate—which has been comparatively neglected in clinical trials—is gaining attention. Mechanical power, which pools these variables to reflect the amount of energy transferred from the mechanical ventilator to the lung parenchyma over time, can be easily calculated at the bedside. Experimental and clinical data show it is associated with VILI and outcomes. In short, mechanical power might be considered as a potential tool to optimize ventilation settings in patients without ARDS and should be validated for this purpose in prospective observational and interventional studies.

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