EVALUATION OF SIMPLIFIED MECHANICAL POWER AND DISSIPATED ENERGY CALCULATIONS IN PHYSICAL RESPIRATORY MODELS WITH TISSUE AND AIRWAY RESISTANCE

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Abstract

Mechanical power (MP) calculation is a promising predictor of ventilator-induced lung injury, yet simplified bedside equations rely on airway opening pressure, potentially missing key information about tissue-level stresses, and involve unclear contributions of PEEP and airway flow resistance. This study compared simplified MP equations in physical models of the respiratory system with either tissue viscoelastic (R_t) or airway flow (R_{aw}) resistance, evaluating how pressure measurement location affects delivered and dissipated energy estimates. Six physical models (No-resistance, Tissue resistance, Flow resistance, and three combinations of R_t with different R_{aw}) were ventilated with the same volume-controlled parameters. Pressure was measured at the airway opening and at an artificial lung level with vital signs monitor, sampled at 100 Hz. Mechanical energy was calculated using both simplified equations and a geometric method based on the pressure-volume loops. Simplified MP equations produced similar mechanical energy estimates for Tissue resistance model and Flow resistance model (R_{aw} = 5 $cmH_2O{\cdot}s{\cdot}L^{-1}$) when pressure was measured at the airway opening. However, measurements at the artificial lung level revealed marked differences in delivered and dissipated energy. Simplified MP equations may misrepresent tissue-level energy, particularly when R_{aw} dominates. Future studies should focus on refining energy estimation methods, considering driving transpulmonary pressures, inspiratory hold duration, and tissue versus flow resistance.

Keywords

viscoelasticity, dissipated energy, respiratory system model, mechanical ventilation, mechanical power

Introduction

Although mechanical ventilation is a sophisticated method of artificial respiratory support, it remains a non-physiological intervention associated with high mortality, especially in the management of acute respiratory failure. In fact, the use of conventional positive pressure ventilation with repeated inflation of the lungs may further exacerbate existing lung injury (barotrauma, volutrauma, biotrauma, ergotrauma) and thus cause ventilator induced lung injury (VILI) [1]. There are several recommendations to provide the lung protective ventilation, such as: low tidal volumes (*VT*), low driving and plateau pressures, appropriately chosen positive end-expiratory pressure (*PEEP*), pronation, alveolar recruitment (opening maneuvers), reasoned (based on careful monitoring of target physiological parameters), or minimization of mechanical power delivery to the lungs [2].

Recent studies suggest that mechanical power (MP), representing the rate of energy delivery from the ventilator to the lungs, may be a significant predictor of VILI risk [3–6]. The gold standard for calculating mechanical energy (E) is the geometric method, which consists in calculating the area under the inspiratory curve of the pressure-volume (PV) loop for each respiratory cycle [7]. This requires numerically integrating airway pressure (P_{aw}) with respect to changes in inspired tidal volume (VT_i):

$$E = \int_0^{VT_i} P_{aw} \cdot dVT. \quad (1)$$

MP is then derived by calculating E per breath and scaling it by the respiratory rate to determine the energy per minute, usually expressed in joules per minute [8]. The use of the geometric method, however, is computationally challenging and requires special equipment with a high sampling rate to record pressure and volume waveforms. Various simplifications of MP

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calculations for volume-controlled ventilation have been proposed in the last years to facilitate bedside calculations in clinical environments [6, 7, 9, 10], with all pressure-related terms in the equations expressed in cmH_2O. Gattinoni et al. [6] proposed a simple method to quantify E delivered to the respiratory system with each breath during mechanical ventilation:

$$E = 0.098 \cdot \left[VT^2 \cdot \left(\frac{1}{2 \cdot C_{rs}} + \frac{R_{aw}}{T_i}\right) + VT \cdot PEEP\right], \quad (2)$$

where E (J) corresponds to the delivered mechanical energy during the inspiratory phase of the respiratory cycle, VT (L) represents the delivered tidal volume, C_{rs} ($L \cdot cmH_2O^{-1}$) represents compliance of the respiratory system, R_{aw} ($cmH_2O \cdot s \cdot L^{-1}$) represents airway flow resistance, T_i (s) is the inspiratory time. Conversion factor 0.098 was used to recalculate cmH_2O to Pa. The equation is derived from the equation of motion and describes total airway pressure as the sum of three components: the elastic recoil needed to inflate the lungs, the flow-resistive pressure needed to move air through the airways, and the baseline tension ($PEEP$). Each of these pressures, when related to the change in lung volume, contributes to a distinct portion of the total mechanical energy. The benefit is that it clearly separates the elastic work of inflation, the energy dissipated in overcoming airway resistance, and the static elastic baseline due to $PEEP$, while linking all of them directly to measurable ventilator parameters.

Another approach that simplifies the integration is based on Gattinoni equation (2) but reduces significantly the computational effort. The disadvantage, however, is the persistent need for an inspiratory hold. This method is usually referred to as Simplified or Comprehensive [6, 7] (hereinafter referred to as Comprehensive):

$$E = 0.098 \cdot VT \cdot \left[P_{\text{peak}} - \frac{1}{2} \cdot \left(P_{\text{plat}} - PEEP\right)\right], \quad (3)$$

where P_{peak} is the peak pressure and P_{plat} is the plateau pressure. The following methods of calculating E do not require any intervention during mechanical ventilation and can therefore be calculated from the ventilation parameters still shown on the ventilator display. By modifying the Comprehensive equation (3) by substituting P_{plat} for P_{peak}, we obtain a Dynamic equation [9]:

$$E = 0.098 \cdot VT \cdot \left[P_{\text{peak}} - \frac{1}{2} \cdot \left(P_{\text{peak}} - PEEP\right)\right]. \quad (4)$$

Giosa et al. [10] introduced a Surrogate equation that assumes a constant airway flow resistance ($10\ cmH_2O \cdot s \cdot L^{-1}$), making it easier to calculate but potentially less accurate:

$$E = VT \cdot \left(\frac{P_{\text{peak}} + PEEP + \left(\frac{Q}{6}\right)}{20}\right), \quad (5)$$

where the flow rate (Q) divided by six is obtained by multiplying the flow rate by the flow resistance and converting from seconds to minutes. The equation of Chi et al. [11] assumes that the pressure fractions in the Gattinoni equation (2) can be replaced by the mean airway pressure (P_{mean}) over the entire respiratory cycle, weighted by the ratio of expiratory to inspiratory pressure due to the inclusion of the entire respiratory cycle. As there is no plateau pressure in the equation, no inspiratory hold is required for the calculation:

$$E = 0.098 \cdot VT \cdot \left[P_{\text{mean}} + \frac{T_e}{T_i}\left(P_{\text{mean}} - PEEP\right)\right], \quad (6)$$

where T_e is the expiratory time and T_i is the inspiratory time. The mathematical derivation of the simplified MP equations is accessible in Supplementary material A.

All the equations presented used only the measured parameters at the airway opening to determine the delivered E. However, the evaluation of MP as a measure of VILI development would be most relevant if volume and related pressure changes were measured directly at the lung parenchyma [12]. Furthermore, the inclusion of $PEEP$ in simplified MP calculations despite no mechanical movement is controversial. $PEEP$ sets a baseline level of lung inflation, affecting total mechanical load and potentially underestimating MP if ignored [13]. On the other hand, reducing $PEEP$ to lower MP may be misleading because $PEEP$ has both protective and adverse effects depending on its relationship to pleural pressure [14]. $PEEP$ affects lung recruitability by altering stress distribution—if it reduces driving pressure and increases compliance, MP decreases, and vice versa [4]. Finally, it is questionable whether resistive MP is related to the development of VILI [12]. Amato et al. [15] found that driving pressure is the strongest predictor of survival in acute respiratory distress syndrome (ARDS) patients, and a retrospective study found that high MP was associated with higher mortality, with respiratory rate and driving pressure being the key factors associated with mortality [16].

Silva et al. [4] proposed the use of transpulmonary driving pressure to determine delivered E. This equation estimates E without considering R_{aw} and $PEEP$. In another study [14], the authors investigated different methods of calculating MP in mechanically ventilated patients, comparing MP calculated from P_{aw} and including $PEEP$, MP calculated from driving pressure (P_D) and driving transpulmonary pressure (P_L), and both excluding $PEEP$. MP calculated from P_{aw} and including $PEEP$ produced significantly higher MP delivery estimates than the other two, but only calculations including P_D and P_L predicted 28-day mortality.

Recently, dissipated energy has started to be considered as a potential contributor to VILI [6, 17, 18]. In physical terms, dissipated energy reflects the irreversible loss of mechanical energy in the respiratory system during a respiratory cycle, while mechanically it is the portion of inspiratory energy not recovered during expiration. Mathematically, it corresponds to the area enclosed by the inspiratory and expiratory limbs of the PV loop. During the inspiratory phase, part of the energy

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is stored as elastic energy and part is dissipated by various mechanisms in the airways and lung tissue. The remaining energy is then recovered during expiration. Gotti et al. [19, 20] suggested that static dissipated energy, not involving R_{aw}, is associated with VILI and decreases with lower tidal volumes at constant minute ventilation. Massari et al. [21] associated higher dissipated energy in the lung parenchyma with high tidal volumes, lung recruitability, strain and lung inhomogeneity. Several studies have shown that maintaining a constant inspiratory and expiratory flow with an *I*:*E* ratio close to 1:1 during ventilation can minimize energy dissipation [17, 22]. Spraider et al. [23] found that compliance-guided flow-controlled ventilation (FCV) reduced dissipated energy and improved gas exchange and more homogeneous gas distribution without evidence of overinflation compared to pressure-controlled ventilation, whereas Busana et al. [24] reported no significant effect on VILI with FCV. Thus, how to quantify injurious dissipated energy in the lung from bedside *PV* loops remains unclear [25].

An assumption that can also complicate the accuracy of *E* calculations is the viscoelasticity of the parenchymal tissue. Resistance of the respiratory system is caused not only by the resistance of the airways, but also by the resistance of the lung tissue [26]. When inspiratory flow stops, the pressure quickly drops from its peak (P_{peak}) to alveolar pressure (P_{alv}) as a result of artificial and anatomical flow resistance. During the subsequent inspiratory hold, there may be a slower decrease from P_{alv} to P_{plat} associated with parenchymal stress relaxation due to viscoelasticity [18, 27, 28]. The pressure difference between P_{alv} and P_{plat}, which likely corresponds to viscoelastic losses [5, 29, 30], is typically encompassed within what caregivers attribute to the difference between P_{peak} and P_{plat}, which is used to estimate R_{aw}. However, this pressure difference represents unmeasured energy dissipated through viscoelastic losses and it possibly contributes to direct damage caused by microfractures in extracellular matrix elements [5]. A limitation of using some simplified equations may also be that there is no defined length of the inspiratory hold. Therefore, the P_{plat} used in the equation may not accurately quantify the forces and injurious energy causing damage. Also, higher pressures resulting from higher flow rates have been found to be associated with the development of VILI, indicating viscoelasticity [18].

In our previous study [31], we have designed a physical viscoelastic respiratory system model and investigated the effect of tissue viscoelasticity on delivered mechanical energy with the possibility of distinguishing tissue resistance (R_t) from R_{aw} using proximal pressure measured at the airway opening. No difference was found in the peak airway pressures and the delivered *E*, calculated by the geometrical method, between the viscoelastic model with R_t and the model incorporating R_{aw}, despite the different origins of the resistances situated at different locations.

The aim of this study was to evaluate how simplified mechanical power equations differ in their estimates of delivered mechanical energy in physical respiratory models with either tissue viscoelastic or airway flow resistance. The second objective was to assess how increasing airway flow resistance affects the calculated mechanical energy and whether this corresponds to the actual energy measured at the lung level, and to analyze the contribution of tissue and flow resistance to energy dissipation.

Methods

Physical models of the respiratory system were formed from combinations of tissue viscoelastic resistance (R_t) and different airway flow resistances (R_{aw}). The linear compliance (C_L) of the artificial lung of Adult Lung Simulator (Michigan Instruments, Kentwood, MI, USA), set at 30 mL·cmH_2O^{-1}, was utilized for each model of the respiratory system. R_{aw} was represented by linear resistances of 5, 10 and 15 cmH_2O·s·L^{-1} (Hans Rudolph Inc., Shawnee, KS, USA).

R_t was represented by a Maxwell body, comprising a compliance and a dashpot resistance, to simulate viscoelastic properties. This was achieved by a glass syringe (Socorex, Ecublens, Switzerland) with a throttle valve, acting as a mechanical damper and the airflow resistance of the throttle valve was adjusted to achieve the same maximum airway pressure measured at the airway opening as in model with the linear airway flow resistance of 5 cmH_2O·s·L^{-1} [31]. The system models a single homogenous compartment with two degrees of freedom, where pressure depends on the gas volume in the artificial lung and the flow to or from the syringe.

Six different physical models of the respiratory system were used: No-resistance (without R_t and without R_{aw}), Flow resistance 5 (without R_t and with R_{aw} of 5 cmH_2O·s·L^{-1}), Tissue resistance (with R_t and without R_{aw}), Tissue + Flow resistance 5 (with R_t and R_{aw} of 5 cmH_2O·s·L^{-1}), Tissue + Flow resistance 10 (with R_t and R_{aw} of 10 cmH_2O·s·L^{-1}) and Tissue + Flow resistance 15 (with R_t and R_{aw} of 15 cmH_2O·s·L^{-1}). Mechanical ventilation was performed using a Veolar-FT ventilator (Hamilton Medical, Bonaduz, Switzerland) set to volume-controlled mode with tidal volume *VT* = 1000 mL, *PEEP* = 5 cmH_2O, inspiratory to expiratory time ratio (*I*:*E*) = 1:1, inspiratory hold = 30%, and respiratory rate (*RR*) of 10 min^{-1}, corresponding to constant inspiratory flow rates of approximately 51 L·min^{-1}. P_{aw} and *VT* were measured at the airway opening using a D-Lite spirometry sensor connected to the E-CAiOVX anesthesia and spirometry module of the Datex-Ohmeda S/5 vital signs monitor (Datex-Ohmeda, Madison, WI, USA). Pressure at the lung level (P_L) was recorded in mmHg via the invasive blood pressure port of the E-PSMP hemodynamic module and converted to cmH_2O. All signals were

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collected using S/5 Collect software on a laptop computer and sampled at 100 Hz.

Data processing and statistical analysis

The averaged waveforms of P_{aw}, P_L and V over the entire respiratory cycle from five representative respiratory cycles for each respiratory system model were plotted in Fig. 1. Standard deviations were calculated but were too small to be shown in the graphs. The values of P_{peak}, P_{plat}, compliance C, airway flow resistance R_{aw}, and P_{mean}, calculated from the measured waveforms of five representative respiratory cycles, were listed in Table S1 in Supplementary Material B.

Next, the dependence of P_{aw} and P_L on VT were plotted in the form of PV loops for each respiratory system model in Fig. 2A and 2B. Using the PV loops, the mechanical energy (E) delivered during the inspiratory phase of the respiratory cycle was calculated for each respiratory system model using the geometric method, i.e., by numerically integrating the area under the respective curve. Dissipated energy was calculated as the hysteresis area of the PV loop, obtained from the difference between inspiratory and expiratory pressures over the corresponding volume changes. The calculation was performed using the pressure measured at the airway opening with $PEEP$ (E_{aw}), pressure at the airway opening without $PEEP$ (E_D), and pressure measured at the artificial lung level without $PEEP$ (E_L). The equations used for these calculations are provided in Supplementary Material A. The measured pressure at the airway opening with $PEEP$ (P_{aw}) and without $PEEP$ (P_D), as well as the measured pressure at the artificial lung level without $PEEP$ (P_L), were used. Finally, E was calculated according to simplified mechanical energy equations (2–6) for each model of the respiratory system, and the average values of E were included in Table S2 of Supplementary Material B.

The calculated inspiratory E_{aw}, E_D, E_L by the geometric method and the E obtained from the simplified equations (2–6) for Tissue resistance and Flow resistance 5 models were plotted on a graph for comparison (Fig. 3A). A two-tailed paired t-test was used to evaluate the statistical difference between Tissue resistance and Flow resistance 5 models for each simplified E equation. A p-value less than 0.05 was considered statistically significant. In addition, E_L, which represents the driving energy acting directly at the lung level, was used as a reference. The ratio of each calculated E to the reference E_L was then determined (Fig. 3B).

Lastly, models of the respiratory system consisting of only R_t or R_t with R_{aw} of 5, 10, and 15 $cmH_2O \cdot s \cdot L^{-1}$ were used to investigate the effect of increasing flow resistance on the calculated E in simplified equations (Fig. 4).

Results

The time course of the measured pressure P_{aw} at the airway opening (Fig. 1A) at $RR = 10\ min^{-1}$ showed that the highest peak pressure was measured in Tissue + Flow resistance model 15 (approx. 50 cmH_2O) and the lowest in No-resistance model (appx. 36 cmH_2O). The curves for Tissue resistance and Flow resistance 5 models overlapped during the inspiratory phase and reached the same P_{peak} and P_{plat}, as intended. However, the decrease in pressure during inspiratory hold was different for these two models. Similar P_{plat} was measured for all models. Under the selected flow settings, in models incorporating both flow and tissue resistance, the rapid pressure drop from P_{peak} to P_{alv} lasted approximately 0.1 s, reflecting flow resistance. This was followed by a slow exponential decline of about 1 s from P_{alv} to P_{plat}, corresponding to viscoelasticity.

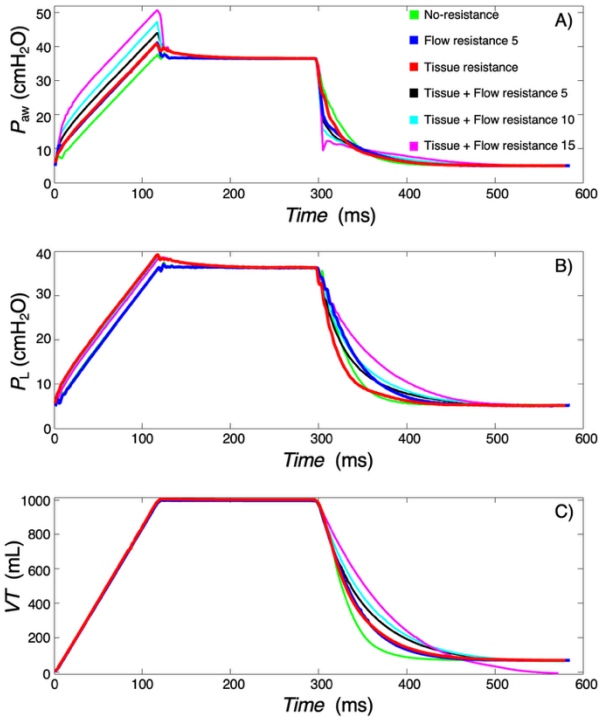

Fig. 1: Time dependence of P_{aw} (A), P_L (B) and VT (C) during the whole respiratory cycle for each respiratory system model. No-resistance (green), Flow resistance 5 (blue), Tissue resistance (red), Tissue + Flow resistance 5 (black), Tissue + Flow resistance 10 (cyan), Tissue + Flow resistance 15 (magenta).

Looking at Fig. 1B, where the pressure was measured at the artificial lung level, the results were different. The lowest P_{peak} was again measured in No-resistance model, but now also in Flow resistance 5 model (appx.

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36 cmH_2O). The curves for the other models (containing R_t) overlapped throughout the inspiratory phase, reached similar P_{peak} (appx. 39 cmH_2O) and P_{plat} during the inspiratory hold.

Fig. 1C showed that the inspiratory *VT* did not differ between the models. However, the rate of expired tidal volume varied between the models, with the slowest rate in Tissue + Flow resistance 15 model and the fastest in No-resistance model.

The *PV* loops in Fig. 2A showed the lowest inspiratory *E* (simply put, the product of pressure and volume) and the narrowest hysteresis area in No-resistance model. By adding R_t or R_{aw}, the inspiratory curves shifted from No-resistance model, resulting in an increase in inspiratory *E* and a larger hysteresis area (increase in dissipated energy). In Tissue resistance and Flow resistance 5 models, the inspiratory *E* were the same and the hysteresis areas were similar.

The resulting *PV* loops in Fig. 2B for the pressure measured at the artificial lung level (P_L) did not follow the same trend. The hysteresis area was the largest for Tissue resistance model (0.87 ± 0.07 J), whereas the hysteresis areas for No-resistance and Flow resistance 5 models were very similar and narrow (appx. 0.20 J). There was no difference in all models with R_t in the inspiratory phase of the *PV* loop, where the curves mostly overlapped, indicating nearly identical pressure–volume behavior across models in this part of the cycle, but in the expiratory phase, increasing R_{aw} narrowed the hysteresis area.

Table 1, which presents the inspiratory and dissipated E_{aw}, E_D, and E_L calculated from the *PV* loops using the geometric method, showed that the inspiratory E_{aw} and E_D differed by approximately 0.5 J for all models. The difference is due to the omission of *PEEP* from the calculation of E_D. However, the dissipated E_{aw} and E_D did not differ between the models. Next, for Tissue resistance and Flow resistance 5 models, the results were very similar for inspiratory E_{aw} (2.49 ± 0.04 J versus 2.51 ± 0.03 J), inspiratory E_D (2.00 ± 0.04 J versus 2.01 ± 0.03 J), dissipated E_{aw} (1.25 ± 0.12 J versus 1.15 ± 0.10 J), and dissipated E_D (1.21 ± 0.12 J versus 1.11 ± 0.10 J), as briefly described in Fig. 2A. But the results were again clearly different for E_L. The inspiratory E_L is about 20% higher for Tissue resistance model than for Flow resistance 5 model (1.88 J versus 1.57 J). The dissipated E_L was about 4 times higher for Tissue resistance model than for Flow resistance 5 model (0.87 J versus 0.18 J). And lastly, increasing flow resistance resulted in such a significant increase in dissipated E_{aw} and E_D that both were even higher than inspiratory E_L for Tissue + Flow resistance 10 model and Tissue + Flow resistance 15 model, despite dissipated E_L decreasing with the increasing flow resistance.

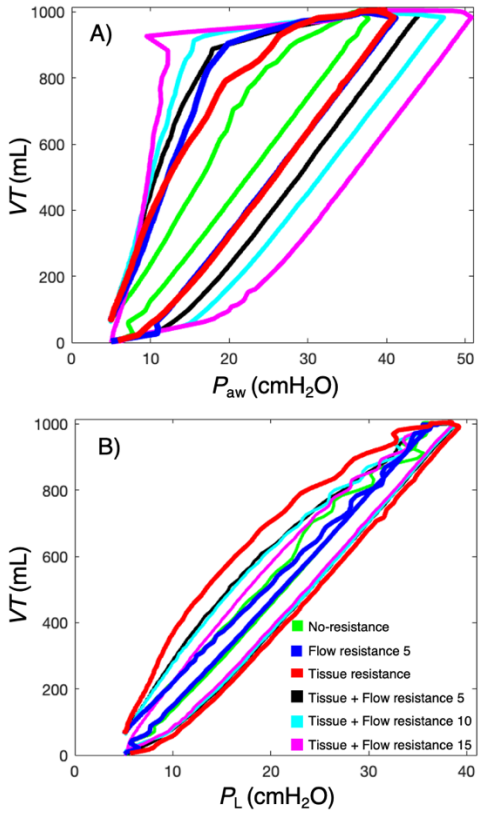

Fig. 2: PV loops for P_{aw} (A) and P_L (B) during the whole respiratory cycle for each respiratory system model. No-resistance (green), Flow resistance 5 (blue), Tissue resistance (red), Tissue + Flow resistance 5 (black), Tissue + Flow resistance 10 (cyan), Tissue + Flow resistance 15 (magenta).

Table 1: The inspiratory and dissipated E_{aw}, E_D and E_L for all models of the respiratory system calculated using the geometric method (1).

Model of the respiratory system	Inspiratory E_{aw} (J)	Dissipated E_{aw} (J)	Inspiratory E_D (J)	Dissipated E_D (J)	Inspiratory E_L (J)	Dissipated E_L (J)
No-resistance	2.20 ± 0.08	0.56 ± 0.12	1.70 ± 0.08	0.53 ± 0.12	1.61 ± 0.04	0.21 ± 0.01
Flow resistance 5	2.49 ± 0.04	1.25 ± 0.12	2.00 ± 0.04	1.21 ± 0.12	1.57 ± 0.03	0.18 ± 0.06
Tissue resistance	2.51 ± 0.03	1.15 ± 0.10	2.01 ± 0.03	1.11 ± 0.10	1.88 ± 0.02	0.87 ± 0.07
Tissue + Flow resistance 5	2.77 ± 0.07	1.60 ± 0.13	2.28 ± 0.07	1.57 ± 0.13	1.83 ± 0.06	0.62 ± 0.10
Tissue + Flow resistance 10	3.03 ± 0.08	1.98 ± 0.15	2.55 ± 0.08	1.94 ± 0.15	1.82 ± 0.07	0.59 ± 0.14
Tissue + Flow resistance 15	3.40 ± 0.05	2.36 ± 0.06	2.91 ± 0.05	2.36 ± 0.06	1.81 ± 0.02	0.47 ± 0.03

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A statistically significant difference between Tissue resistance and Flow resistance 5 models was found only for inspiratory E_L and the simplified Chi equation (6), as shown in Fig. 3A. At the same time, E_L was the lowest for both models compared to other equations. It is also evident that there was no difference between E_{aw} and Gattinoni equation (2) and Comprehensive equation (3) for either Tissue resistance or Flow resistance 5 models, where E is equal to 2.5 J. Inspiratory E_D was lower than inspiratory E_{aw} for both models by the aforementioned 0.5 J, while Dynamic equation (4) and Surrogate equation (5) showed values approximately 10% lower and 10% higher than E_{aw}, respectively.

The calculated E from all equations exhibited a higher value in comparison to E_L, which was used as a reference in this case, as demonstrated in Fig. 3B. Of the simplified equations, Dynamic (4) overestimated the least by 20% for Tissue resistance model and 45% for Flow resistance 5 model, and Chi (6) overestimated the most by 85% for Tissue resistance model and 120% for Flow resistance 5 model. However, E_D values calculated by the geometric method were closest to the E_L for both models.

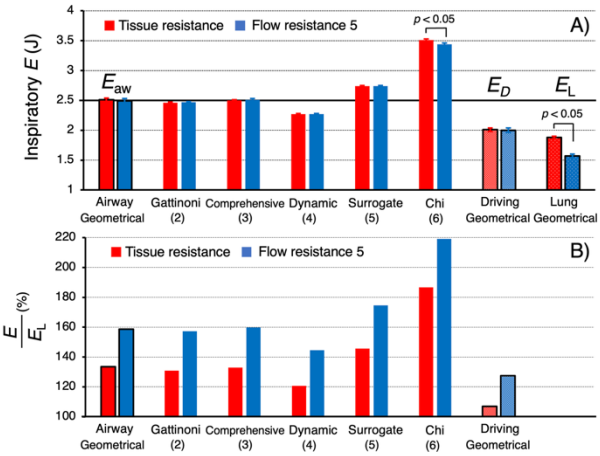

Fig. 3: The calculated inspiratory E_{aw}, E_D, E_L by the geometric method and the mechanical energy obtained from the simplified equations (2–6) for Tissue resistance and Flow resistance 5 (A); The ratio of each calculated E to the reference E_L (B).

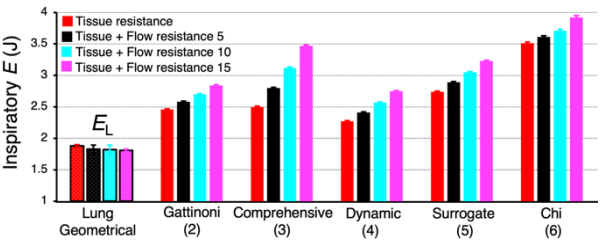

Fig. 4: The effect of increasing flow resistance on E in simplified equations in models of the respiratory system consisting of only tissue resistance or tissue resistance with flow resistances of 5, 10, and 15 $cmH_2O{\cdot}s{\cdot}L^{-1}$.

Although there was no increase in E_L due to increasing R_{aw}, the increase in E was significant for all simplified equations (Fig. 4). The highest increase in E due to increasing R_{aw} occurred for the Comprehensive equation (3), where the difference between the lowest and highest E was equal to 1 J. In contrast, the smallest difference was observed for the Gattinoni equation (2), where the difference was only 0.4 J.

Discussion

The main findings of this study are that simplified mechanical power (MP) calculations provide similar estimates of delivered mechanical energy (E) in physical respiratory models with either tissue viscoelastic (R_t) or airway flow (R_{aw}) resistance, despite significant differences observed at the lung level and different origins of the resistances situated at distinct locations. Furthermore, the simplified MP calculations overestimate E when R_{aw} dominates compared to the E measured at the lung level. Increasing R_t or R_{aw} elevated dissipated energy measured at the airway opening, whereas only R_t increased the dissipated energy at the lung level. In contrast, increased R_{aw} paradoxically decreased energy dissipation at the lung level.

The unique setup of the respiratory model with R_t allowed the evaluation of E associated with the viscoelastic behavior of the lung parenchyma, a known [5, 29, 30] but overlooked characteristic. Rapid inflation of the artificial lung of the simulator induces a transient negative pressure inside the syringe due to the high flow resistance of the throttle valve, temporarily reducing model compliance. This effect is time-dependent and is influenced by the resistance of the throttle valve and the volume increase of the syringe. When an inspiratory hold is applied, the pressure inside the syringe equilibrates to atmospheric pressure while the pressure at the artificial lung level decreases exponentially [31]. This results in a plateau pressure similar to that of models with no resistance or R_{aw} only (Fig. 1). This unique feature was achieved by the low coefficient of friction of the borosilicate glass of the syringe [31]. Baseline E dissipation was quantified in No-resistance model. Table 1 showed that the minimum achievable hysteresis using the artificial lung of the Adult Lung Simulator was approximately 0.56 ± 0.12 J for E_{aw} and 0.53 ± 0.12 J for E_D, while the artificial lung alone exhibited a hysteresis of 0.21 ± 0.01 J. This suggests that roughly 0.3 J originates from the intrinsic flow resistance of the test apparatus, with the remaining E dissipation likely attributable to the hysteresis of the rubber of the artificial lung of the simulator and the use of a not fully thermocompensated model. Thus, the small difference between the inspiratory E_D and E_L in Tissue resistance model (with no added R_{aw}) was due only to the limitations of the apparatus.

The simplified MP equations analyzed here rely on several assumptions—linear compliance and airway resistance, constant inspiratory flow during volume-

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controlled ventilation, or homogeneous mechanical properties in each lung compartment. Although newer simplified MP equations for spontaneous breathing or MP normalization to lung size, compliance, or functional residual capacity have emerged [30, 32, 33], none account for the viscoelastic properties of lung tissue or the duration of inspiratory holds, both of which can significantly affect the calculation of delivered *E*.

In our study, E_L served as a reference for potentially harmful *E* as it reflects the *E* acting directly at the lung level [12, 14]. However, in clinical practice, E_L is difficult to measure and requires measurement using an esophageal catheter [34]. For Tissue resistance model, the so-called Dynamic equation (4), proposed in the study by Urner et al. [9], overestimated the least among the simplified equations compared to the geometrically calculated E_L (Fig. 3B). This is due to the fact that only P_{peak} was used for the calculation, which showed almost no difference between the pressure measured at the airway opening and at the artificial lung level (the small difference was due to the limitations of the test apparatus). Furthermore, if the energy component for *PEEP* (static elastic energy) of approximately 0.5 J is removed from the calculation (4), *E* would be essentially the same.

Although Chi equation (6) does not require an inspiratory hold, its application significantly increased the calculated *E*. Notably, the inspiratory hold revealed a difference between Tissue resistance model and Flow resistance 5 (Fig. 3A), due to the use of P_{mean} in the equation and the presence of the exponential pressure decrease during the hold in Tissue resistance model. However, when the results were analyzed without the inclusion of the inspiratory hold, as assumed by Chi equation (6), the *E* between Tissue resistance model and Flow resistance 5 model was identical, as shown in the Supplementary Material B.

Our results are consistent with those of Chiumello et al. [7], who found strong correlations between simplified MP equations (Comprehensive and Surrogate) and a reference geometric method based on *PV* loops in 40 sedated and paralyzed ventilated patients. However, our data show that simplified equations may misrepresent the *E* acting at the lung level. Increasing R_{aw} caused an increase in *E* for all simplified equations, despite no change at the lung level (Fig. 4). The effect of increasing R_{aw} was least pronounced with the Gatinnoni equation (2) and most significant with the Comprehensive equation (3). This discrepancy may hinder the setting of appropriate ventilation parameters in patients with high R_{aw} if MP thresholds are strictly followed. However, this does not negate the potential harm of elevated airway pressures, which can contribute to distal lung injury [35].

While dissipated energy has been suggested as a possible contributor to VILI [6, 17, 18], it is unclear how to quantify the harmfulness of this energy. Our findings emphasize that the location of the measurement significantly affects the interpretation. *PV* loops recorded at the airway opening showed increasing hysteresis with higher R_{aw} in both inspiratory and expiratory phases (Fig. 2). However, the energy dissipation in the inspiratory phase of the *PV* loop is largely driven by R_{aw}, the effect of which on VILI is not straightforward [12]. Measured at the lung level, high R_{aw} decreased the energy dissipated in the expiratory phase of the *PV* loop, similar to mechanical control of expiratory flow [22–24]. R_t increased energy dissipation across both phases at the lung level but behaved similarly to R_{aw} when measured at the airway opening. In addition, the duration of the inspiratory hold may significantly affect energy dissipation. During the hold, parenchymal stress relaxation induces a pressure drop that may alter the shape and area of the expiratory *PV* loop. This mechanism further underscores the role of tissue viscoelasticity in energy dissipation. Taken together, R_t appears to be a dominant contributor to energy dissipation at the lung level, while R_{aw} predominantly affects measurements at the airway opening—highlighting the risk of misinterpretation if only proximal measurements are considered.

It is also important to note that the calculation of *E* using simplified equations that rely on values provided on the ventilator display may be affected by proprietary algorithms of the ventilator. For example, the length of the inspiratory hold may or may not affect the calculated values of P_{plat}, compliance, and R_{aw} [36], thereby affecting the values calculated using the simplified equations. Additionally, since the effect of *PEEP* on *E* is definitely not linear [12] and the contribution of airway resistance to VILI remains unclear, driving transpulmonary pressure may be a more appropriate parameter for estimating energy delivery and dissipation. A possible surrogate for this difficult-to-measure value could be the pressure measured at the airway opening immediately after the start of the inspiratory hold, as it may reflect alveolar pressure acting on the lung tissue. A longer inspiratory hold could help monitor viscoelastic relaxation of the parenchyma; however, leaks in the breathing circuit may confound these measurements and caregiver interpretation. Ultimately, large clinical studies are needed to determine whether different methods of calculating delivered or dissipated energy correlate with outcomes such as mortality, development of VILI, or pulmonary edema. Potential approaches include randomized trials, which could be appropriate if ventilator settings were prospectively adjusted to minimize mechanical energy delivery to the respiratory system according to a selected equation, or large observational cohorts with high-resolution ventilator data, which would probably be more feasible and would allow both comparison of simplified equations with a geometric reference and direct comparison among the simplified equations themselves to assess their prognostic accuracy.

This study has several limitations. First, although physical models offer controlled conditions to isolate and analyze specific variables, they are simplifications

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and cannot fully replicate the complexity of human lung mechanics. Despite this, the effects described here are consistent with phenomena observed in clinical practice. Second, pressure and volume curves during the expiratory phase may be influenced by the design of the expiratory valve and ventilator control algorithms. However, this influence is unlikely to have significantly affected the main findings. A possible limitation of this study is the use of relatively high tidal volumes compared with typical clinical practice. This setting was chosen because smaller tidal volumes produced only minimal displacement of the bellow-based lung simulator from its resting position. Such small displacement would have amplified the effect of mechanical imperfections of the simulator, making it difficult to reliably separate the elastic, resistive, and viscoelastic components. The physical model with tissue resistance could, however, be further optimized in future work, for example by using a syringe of different size or employing a custom-built simulator designed to achieve greater displacement at lower tidal volumes. Finally, this study did not evaluate energy dissipation due to recruitment during inspiration, which has been recently suggested as a possible contributor to VILI [37].

Conclusion

We demonstrated that simplified mechanical power (MP) equations provide similar estimates of delivered energy in physical models with either tissue viscoelastic (R_t) or airway flow (R_{aw}) resistance, despite differences in the nature and location of these resistances. Although these equations are practical for bedside use, they tend to overestimate the actual mechanical energy delivered to the lung when R_{aw} is dominant. Our findings further show that R_t, rather than R_{aw}, is the primary contributor to energy dissipation at the lung level, indicating that the calculations based solely on airway pressures may not accurately reflect the mechanical stresses responsible for lung injury. These results underscore the importance of considering R_t and direct lung-level measurements when evaluating mechanical energy delivery and its potential for ventilator-induced lung injury. Future studies should focus on refining energy estimation methods, considering driving transpulmonary pressures and inspiratory hold duration, and to validate these approaches in clinical practice.

Ethics approval and consent to participate

Not applicable.

Competing interests

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Availability of data and materials

The datasets generated and analyzed during the current study are available in the repository at https://ventilation.fbmi.cvut.cz/data/ (accessed on 19th May 2025).

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