Mechanical power in AVM-2 versus conventional ventilation modes in a normal lung model: A bench study

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Abstract

Introduction

Recent studies suggested that the energy delivered by the mechanical ventilator to the lungs termed the mechanical power can induce and increase the risks of ventilator induced lung injury. The components of the mechanical power include the variables delivered by the ventilator: tidal volume, respiratory rate, inspiratory flow, airway pressure. Adaptive Ventilator Mode-2 (AVM-2) is a pressure-controlled mode with an optimal targeting scheme based on the inspiratory power equation that adjusts the respiratory rate and tidal volume to achieve a target minute ventilation. This mode conceptually should reduce the mechanical power delivered to the patients and thus reduce the incidence of ventilator induced lung injury.

Methodology

A bench study using a lung simulator (TTL, Michigan Instruments, Michigan, USA) was conducted. We constructed a passive single compartment normal respiratory mechanics model with compliance of 50 ml/cmH_2O, and resistance of 10 cmH_2O/L/s, with IBW 70 kg. We compared three different ventilator modes: Adaptive Ventilation Mode-2 (AVM-2), Pressure Regulated Volume Control (PRVC), and Volume Controlled Ventilation (VCV) in four different scenarios: 2 levels of minute ventilation 7 and 10.5 Lit/min (Experiment 1 and 2 respectively), each with 2 different PEEP levels 5 and 10 cmH_2O (Experiment A and B respectively) termed Experiments 1A, 1B, 2A, and 2B respectively.

The AVM-2 mode automatically selects the optimal tidal volume, and respiratory rate per the dialed percent minute ventilation with an I:E ratio of 1:1. In the PRVC, VCV we selected target tidal volume 6ml/kg/IBW (420 ml), and respiratory rate adjusted to match the minute ventilation for the AVM-2 mode. I:E ratio was kept 1:2 to avoid intrinsic PEEP. The study was conducted using a bellavista™ 1000 e Ventilator (Vyaire Medical, Illinois, USA).

The mechanical power delivered by the ventilator for each mode was computed and compared between the three modes in each experiment. Statistical analysis was done using Kruskal-Wallis test to analyze the difference between the three modes, post HOC Tukey test was used to analyze the difference between each mode with the confidence intervals, $P < 0.05$ was considered statistically significant.

Results

There were statistically significant differences between all the three modes regarding the ventilator delivered mechanical power. The AVM-2 mode delivered significantly less mechanical power than VCV which in turn was less than PRVC. Experiment 1A: AVM-2 8.76 ± 0.05, VCV 9.78 ± 0.04, PRVC 10.82 ± 0.08, $P < 0.001$ Experiment 1B: AVM-2 11.27 ± 0.09 VCV 12.81 ± 0.05, PRVC 13.88 ± 0.06, $P < 0.001$. Experiment 2A: AVM-2 14.76 ± 0.05, VCV 15.79 ± 0.05, PRVC 18.29 ± 0.07, $P < 0.001$, Experiment 2B: AVM-2 18.76 ± 0.04, VCV 20.56 ± 0.04, PRVC 21.17 ± 0.03, $P < 0.001$.

Discussion

AVM2 mode delivered less mechanical power compared to two conventional modes using low tidal volume in a normal lung model. This might reduce the incidence of ventilator induced lung injury. Results need to be validated in more clinical studies.

Keywords: AVM-2, Mechanical power, VILI

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Introduction

Ventilator-induced lung injury (VILI) is an acute lung injury of the lung parenchyma as a consequence of mechanical ventilation. Ventilator parameters that can contribute to acute lung injury include pressure, [1] volume, [2] flow, [3] and respiratory rate. [4] The principal mechanisms of ventilator-induced lung injury (VILI) are volutrauma, barotrauma, atelectrauma, biotrauma, and ergotrauma. [5]

Barotrauma or alveolar overdistension results from increased transpulmonary pressure and is the main determinant of VILI. [6] The landmark ARDS Network trial demonstrated that limiting tidal volume (6 vs.12 mL/kg predicted body weight and plateau airway pressure ($\leq$ 30 vs. $\leq$ 50 cmH_2O) improved survival in patients with ARDS. [7] Later studies in patients ventilated without ARDS showed that alveolar overdistension from high tidal volumes may increase risk for VILI. [8] Additionally, the repetitive alveolar opening and collapsing create shear forces causing mechanical injury to the adjacent alveoli and small airways termed atelectrauma. [9] Biotrauma caused by mechanical ventilator triggering an extensive biological response, including activation of a proinflammatory and pro-injurious cytokine cascade. [10,11] Recently the term Ergotrauma has been coined to describe the energy and power applied to the lung and to their potential contribution to VILI. [12,13]

In the diseased lung, transpulmonary pressure for a given airway pressure may vary greatly based on the elastance of the respiratory system. [6] VILI therefore is the result of interactions between the patient and the ventilator. While factors related to the patient's respiratory system are more difficult to control, the ventilator parameters including respiratory rate, flow rate and shape, tidal volume, tidal pressure (also known as driving pressure or inspiratory pressure), and positive end expiratory pressure that can contribute to VILI are more easily adjusted by clinicians. [5]

Highlights

AVM-2 delivered less mechanical power for the same minute ventilation compared to conventional modes of ventilation in a normal lung model

As stated above, tidal volume and plateau pressures have been the targets for reducing the risk of VILI for two decades. More recently mechanical power has been proposed as a possible target. Mechanical power unifies various ventilator parameters that contribute to VILI into a single variable that represents the mechanical forces delivered to the lungs per unit time. [14]

Since the publication of the mechanical power equation, several retrospective studies have shown an association between mechanical power and mortality. [15,16] The PRoVENT-COVID study showed that the mechanical power was independently associated with mortality with greater risk in higher quartiles of mechanical power. [15] Another retrospective analysis [16] showed an increased risk of mortality associated with a difference in optimal mechanical power greater than 5 J/min, particularly in those with refractory respiratory failure.

Given the association between mechanical power and mortality, adaptive ventilation modes were designed to ensure optimization of the patient's work of breathing. Adaptive ventilation modes are modes that are closed loop modes that automatically adjusts based on an optimum targeting scheme which targets the lowest "cost" to the patient. [17]

Adaptive ventilation mode-2 (AVM-2) is a pressure-controlled ventilator mode developed in 2017 that uses the mean inspiratory power delivered by the ventilator as the basis for its optimum targeting scheme to reduce mechanical power. In AVM-2, the inspiratory power (equation below) is calculated by summation of *resistive power*, which describes the work to deliver the flow through the natural and artificial airways, and *tidal power*, which describes the work needed to expand the lungs and the chest wall. [17] Inspiratory power differs from total power, as it does not account for PEEP power, which together with tidal power are components of elastic power. [17]

However, because PEEP does not displace air in the lungs, its theoretical contribution to mechanical power is debatable. [18]

$$\dot{w}_{Insp} = \frac{1}{2 \cdot C} \cdot f \cdot \left(\frac{\dot{v}_A}{f} + v_D\right)^2 \cdot \left(1 + \coth\left(\frac{T_I}{2 \cdot R \cdot C}\right)\right)$$

Inspiratory work equation. Coth: cotangens hyperbolicus function, C: compliance, f: respiratory frequency, R: resistance, and TI is the set inspiratory time, V_D: dead space, V_A: minute ventilation. Adapted from reference 17

We hypothesized that when compared to traditional modes of mechanical ventilation, AVM-2 would deliver ventilation with less mechanical power and thus be more lung protective.

Material and Methods

A bench study using a lung simulator (TTL, Michigan Instruments, Michigan, USA), we constructed a single compartment normal respiratory mechanics according to Arnal's parameters of simulation [19] in a passive model with compliance of 50 ml/cmH_2O, and resistance of 10 cmH_2O/L/s, with IBW 70 kg.

We compared three different ventilator modes that guarantee minimal minute ventilation with different targeting schemes. [20] Pressure controlled mode with an *optimal* targeting scheme: Adaptive Ventilation Mode-2 (AVM-2), Pressure controlled mode with *adaptive* targeting scheme: Pressure Regulated Volume Control (PRVC), and Volume controlled *set point* targeting scheme: Volume Controlled Ventilation (VCV) in four different scenarios. The scenarios constituted of 2 levels of minute ventilation 7 and 10.5 Lit/min (Experiment 1 and 2 respectively), each scenario with 2 different PEEP levels 5 and 10 cmH_2O (Experiment A and B respectively) termed Experiments 1A, 1B, 2A, and 2B respectively. Settings and parameters are summarized in table 1.

We chose two common conventional modes of ventilation utilized by clinicians worldwide that guarantee a minimum minute ventilation, [21] one that uses a volume-controlled mode (VCV) and the other is a pressure controlled adaptive mode (PRVC). We chose a low tidal volume strategy of 6 ml/kg IBW. Though it remains debatable what the optimal target tidal volume in normal lung conditions, it is generally advised to target between 6-8 ml/kg.

The AVM-2 mode automatically selects the optimal tidal volume, and respiratory rate combination per the dialed percent minute ventilation, while in the PRVC, VCV we selected target tidal volume 6ml/kg/IBW (420 ml), and respiratory rate adjusted to match the minute ventilation from the AVM-2 mode.

Inspiratory: Expiratory ratio was kept 1:2 in VCV and PRVC to avoid intrinsic PEEP which can alter the equations, as the equations assume no intrinsic PEEP, while AVM-2 uses 1:1 ratio. Both AVM-2 and PRVC utilize the decelerating inspiratory flow, while in VCV we elected to use the ramp flow rather than the square continuous flow to avoid high peak inspiratory pressures.

The study was done using a bellavista™ 1000 e Ventilator (Vyaire Medical, Illinois, USA).

The mechanical power delivered by the ventilator for each mode was obtained by two methods. First, the data obtained from the simulator was computed and calculated. Second, the mechanical power was obtained directly from the ventilator software. Both data results correlated to each other. The mechanical power results were compared between the three modes in each experiment. Fifty breaths (5 breath/min for 10 minutes) were analyzed in each mode in each experiment.

Statistical analysis was done using Kruskal-Wallis to analyze the difference between the three modes, post HOC Tukey test was used to analyze the difference between each mode with the confidence intervals, $P < 0.05$ was considered statistically significant.

Results

Results are presented as mean ± SD and summarized in tables 2 & 3 and figure 1. In each of the 4 experiments, there were statistically significant differences between all the three modes regarding the ventilator delivered mechanical power. The post HOC analysis confirmed that the AVM-2 mode delivered significantly less mechanical power than VCV, which in turn was less than PRCV in all four experiments.

Experiment 1A: AVM 8.76 ± 0.05, VCV 9.78 ± 0.04, PRVC 10.82 ± 0.08, $P < 0.001$ Experiment 1B: AVM 11.27 ± 0.09 VCV 12.81 ± 0.05, PRVC 13.88 ± 0.06, $P < 0.001$. Experiment 2A: AVM 14.76 ± 0.05, VCV 15.79 ± 0.05, PRVC 18.29 ± 0.07, $P < 0.001$, Experiment 2B: AVM 18.76 ± 0.04, VCV 20.56 ± 0.04, PRVC 21.17 ± 0.03, $P < 0.001$.

	AVM-2			VCV			PRVC		
	VT VT/KG	Δ P	RR	VT VT/KG	Δ P	RR	VT VT/KG	Δ P	RR
Experiment 1 A	445 ± 10 6.4	8.5 ± 0.3	14	420 ± 9 6.1	9.5 ± 0.5	17	420 ± 14 6.1	10 ± 0.4	17
Experiment 1 B	450 ± 7 6.4	8.5 ± 0.5	14	420 ± 11 6.1	9.5 ± 0.5	17	420 ± 12 6.1	10 ± 0.5	17
Experiment 2 A	544 ± 9 7.7	10 ± 04	18	420 ± 13 6.1	10 ± 0.1	25	420 ± 15 6.1	11 ± 0.3	25
Experiment 2 B	547 ± 10 7.7	11 ± 02	18	420 ± 15 6.1	10.1 ± 0.2	25	420 ± 11 6.1	11 ± 0.4	25

Table 1: Characteristics of each experiment. Δ P is the tidal pressure (inspiratory pressure above PEEP) in cmH_2O, VT and VT/KG in ml. RR: respiratory rate per minute. AVM: Adaptive ventilation mode, PRVC: Pressure regulated Volume Control, VCV: Volume Control Ventilation

	AVM-2	VCV	PRVC	P value
Experiment 1 A	8.76 ± 0.05	9.78 ± 0.04	10.82 ± 0.08	< 0.001
Experiment 1 B	11.27 ± 0.09	12.81 ± 0.05	13.88 ± 0.06	< 0.001
Experiment 2 A	14.76 ± 0.05	15.79 ± 0.05	18.29 ± 0.07	< 0.001
Experiment 2 B	18.76 ± 0.04	20.56 ± 0.04	21.17 ± 0.03	< 0.001

Table 2: Mechanical Power in each mode. AVM: Adaptive ventilation mode, PRVC: Pressure regulated Volume Control, VCV: Volume Control Ventilation

	AVM-2	PRVC
VCV		
- Experiment 1 A	- P < 0.01 95% CI 0.975 - 1.064	- P < 0.01 95% CI 0.995 - 1.084
- Experiment 1 B	- P < 0.01 95% CI 1.497 - 1.602	- P < 0.01 95% CI 1.007 - 1.112
- Experiment 2 A	- P < 0.01 95% CI 0.9763 - 1.063	- P < 0.01 95% CI 2.456 - 2.543
- Experiment 2 B	- P < 0.01 95% CI 1.771 - 1.828	- P < 0.01 95% CI 0 .171 - 0.228
PRVC		
- Experiment 1 A	- P < 0.01 95% CI 2.015 to 2.104	
- Experiment 1 B	- P < 0.01 95% CI 2.557 - 2.662	
- Experiment 2 A	- P < 0.01 95% CI 3.476 - 3.563	
- Experiment 2 B	- P < 0.01 95% CI 1.971 to 2.028	

Table 3: Post HOC Tukey test with 95% Confidence Intervals

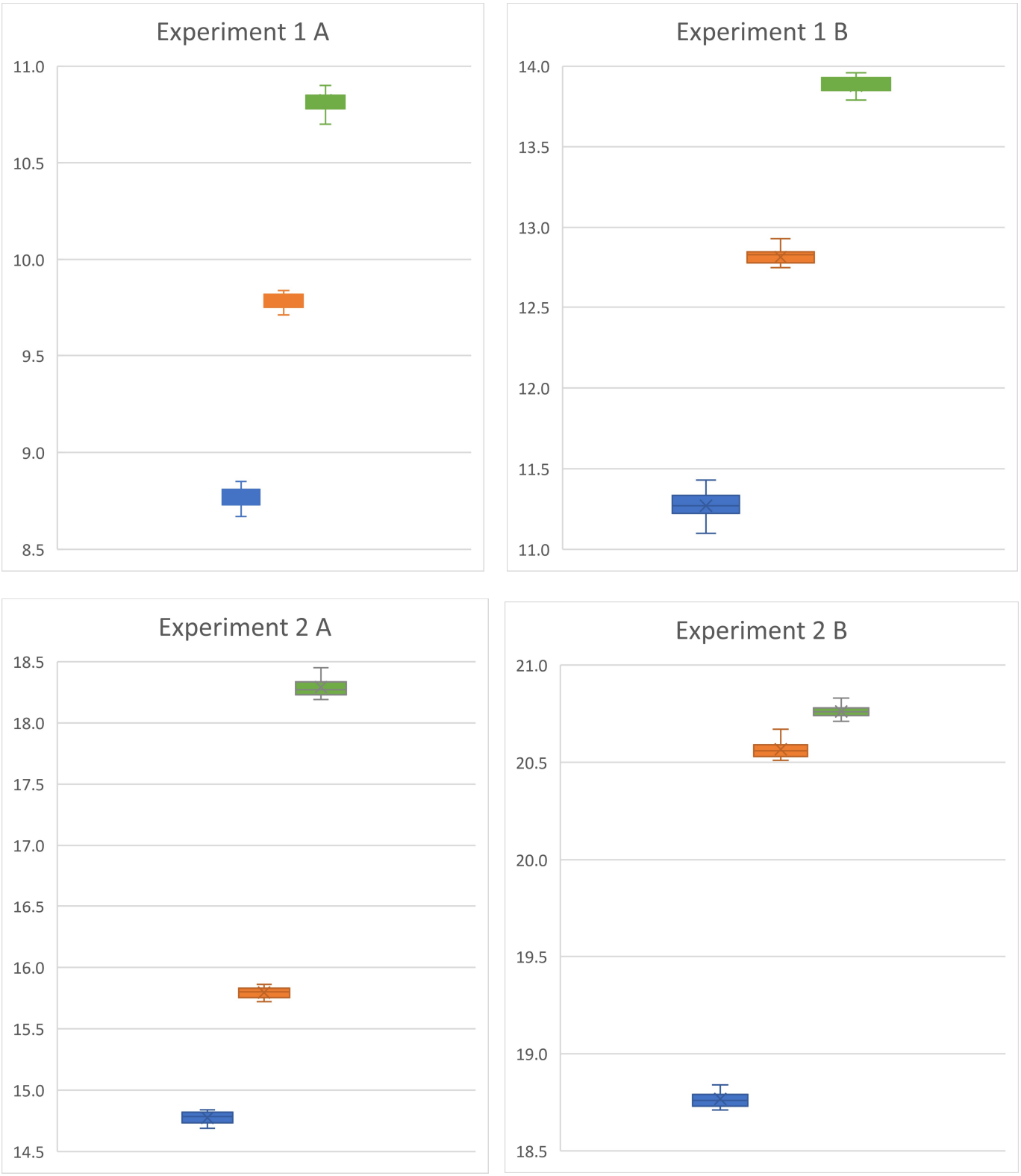

Figure 1: Histogram for each experiment with means and SD. X Axis: Mechanical power in J/min, Y axis: Mode. Blue: AVM-2, Orange: VCV, Green: PRVC

Discussion

This is the first study to compare the performance of AVM-2 mode with the conventional modes of mechanical ventilator using a lung simulator. In this observational study, AVM-2 was found to deliver a lower mechanical power compared with traditional modes of ventilation like Pressure Regulated Volume Control (PRVC), and Volume Controlled Ventilation (VCV).

Different ventilator settings have been shown to reduce ventilation induced lung injury. Previous studies have shown that lower tidal volume ventilation in patients with and without ARDS is lung protective [7,22–24]. Another study found that decreased driving pressure was associated with increased survival in ARDS patients. [25] However, these two variables are not the only contributors to VILI. Recent studies have found that mechanical power, which combines multiple ventilatory parameters including tidal volume, inspiratory flow, tidal (driving) pressure, RR (respiratory rate), and possibly PEEP to be an important parameter for VILI. [26,27] Higher mechanical power is associated with higher mortality and increased duration of mechanical ventilation. These outcomes were seen even in patients with low tidal volumes and driving pressures. [28]

In our study we found that ventilation with AVM-2 resulted in lower mechanical power compared to two different conventional modes of ventilation using a lung protective ventilation strategy. When using the 100% Minute Ventilation in AVM-2, the average tidal volume (tidal volume/Kg) was in the range of low tidal volume ventilation (6.4-6.5 ml/kg) while the tidal pressure (inspiratory pressure above PEEP) was lower (8.8-9 cmH_2O) than in the other two modes. When using the 150% Minute ventilation, the tidal volume and V_T/Kg was higher (7.7-7.8 ml/kg) than the conventional modes but was still in the range of 6-8 ml/kg which is considered within an acceptable range, [29] and the tidal pressure was similar to the conventional modes.

Our resultant V_T/kg are in agreement to those by van der Staay and Remus in their description of AVM-2 compared to AVM-1, [30] and close to the mathematical model developed by van der Stay and Chatburn [17] in their evaluation of three different optimal ventilator modes, though their model was based on the predicted dead space. [17,30] Albeit the differences in mechanical power in our study with normal respiratory mechanics, we hypothesize that those results will be more pronounced in a more severe restrictive mechanics like ARDS. Currently, such a study is in progress.

Our findings are in line with those from previous studies which compared AVM-2 with other ventilator modes. A study done by Becher et al, [31] which compared AVM-2 with AVM-1, which is another adaptive mode which utilizes the Otis' equation of least work of breathing as its target scheme in ARDS patients and found that ventilation with AVM-2 led to less mechanical power compared to AVM-1. To our knowledge there are no studies comparing AVM-2 with conventional modes of ventilation. Further large-scale studies on patients are needed to prove the lung protective benefits of AVM-2.

It is still unclear which component of the mechanical power equation is the most significant or injurious to the lung, and whether the work and power equations should be indexed to the respiratory system compliance or the amount of aerated lung. [32] One possible explanation is that if mechanical power is not normalized to lung size, it does not reflect the actual energy dissipated into the lung (i.e., amount of the generated VILI). Smaller lungs require lower mechanical power to minimize VILI. [12] Thus, it has been proposed to normalize the mechanical power according to the predicted body weight, the compliance, or the amount of well-inflated tissue.

In addition, it remains unclear, how the mechanical power correlates in passive or active patients, or if the transpulmonary mechanical power could be more indicative of VILI. [33] Our hypothesis is that the respiratory system compliance and the amount of well-inflated tissue should better reflect the amount of lung-aerated tissue exposed to the energy load during the mechanical ventilation. Furthermore, the transpulmonary mechanical power normalized to well-inflated tissue might better predicts mortality, suggesting that in addition to the amount of resistive capacity of the lung, assessed as compliance or amount of well-aerated tissue, the partitioned lung mechanic characteristics computed by esophageal pressure, would have a determining role on the effect of mechanical power. [34,35]

The mechanical power corresponds to the sum of pressure necessary to overcome elastic load (*Elastic work*), resistance (*Resistive work*) and possibly PEEP. [36] (*Tidal work = Elastic work – PEEP*).

The strategies to prevent VILI require a definition of excessive *Stress* and *Strain* with a reduction in normalized inspiratory mechanical power by lung volume. [37] Excessive V_T, RR and PEEP can generate VILI, but the effect of each of these variables in the context of mechanical power remains controversial. The question remains in determining characteristic subtypes of VILI that each variable can cause. [12,36] However it was found that with the same mechanical power varying the V_T, RR and PEEP, the lung damage was similar and an independent component. [38]

The high RR can cause VILI even with V_T lower than 6 ml/kg, because the RR is intrinsic to the energy provided by the ventilator. [39] In this research, in the comparison between the ventilatory modalities, it can be seen that the doubling of PEEP determined a substantial increase in mechanical power in all modalities, while the increase in RR in the VCV and PRCV modality had a more intense repercussion in their mechanical power than the elevation of the VT in the AVM-2, as well as the variation of its peak pressures, which were insufficient to reach the values of the mechanical power of the VCV and PRVC.

More studies are needed in the future to determine the exact effects of mechanical power in lung protective ventilation, and the relationship between the mechanical power and the stress and strain applied on the lung units.

There are some limitations in the present bench study. The study was conducted using a lung simulator, not in real patients, with the inherent limitations of lung simulation regarding oxygenation, ventilation, and hemodynamics.

We studied only passive conditions due to the difficulty of calculating the patient's muscle work and thus total work in active breathing conditions. Theoretically, the higher patient muscle work, will reduce the ventilator work in some modes (e.g. VCV and PRVC) with same total work, or might be additive to ventilator work thus increasing total work in other modes like pressure-controlled ventilation.

We chose only one set of compliance and resistance to model normal lung mechanics, and only two different levels of minute ventilation 100% and 150% which are considered within the accepted range in normal conditions, [35] and two levels of PEEP 5 and 10 cmH_2O which are also considered within the range used for normal respiratory mechanics. Regardless, our mathematical calculation shows that those factors would not change our findings. We used a low tidal volume of 6ml/kg in the conventional modes that resulted in higher respiratory rate compared to AVM-2 especially in Experiment 2A and 2B, however, according to our calculations, the mechanical power would have been higher in both the VCV and the PRVC if we used 7 or 8 ml/kg with lower respiratory rate.

In conclusion, AVM-2 mode delivered less mechanical power compared to two common conventional modes using low tidal volume in a normal lung model. This may reduce the incidence of ventilator induced lung injury. More studies are needed to confirm our findings.

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