A comparative analysis of mechanical power and Its components in pressure-controlled ventilation mode and AVM-2 mode

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Abstract

Background

Mechanical ventilation is a critical therapeutic intervention in the management of patients with respiratory failure. Understanding the implications of different ventilation modes is essential in preventing ventilator-induced lung injuries (VILI). Recently, mechanical power has emerged as a critical element in the development of VILI and mortality. Previous bench work studies have suggested that new optimal (adaptive) modes, such as Adaptive Ventilation Mode 2 (AVM-2), can reduce the mechanical power in turn might reduce the rates of VILI. This study aims to compare the conventional Pressure-Controlled Ventilation (PCV) mode with an emerging design of Adaptive Ventilation Mode-2 (AVM-2), to measure the differences in mechanical power, alongside it's components of PEEP, Tidal, Elastic, Resistive, Inspiratory, Total work, tidal volume, driving pressure and Power Compliance Index.

Methods

Between January 2023 and June of 2023, we conducted a prospective crossover study on twenty-two subjects admitted to our ICU within the first day after initiation of mechanical ventilation. Subjects were initially started on PCV settings chosen by the primary treatment team, then switched to AVM-2 with comparable minute ventilation. Mechanical power and its work components (tidal, resistive, PEEP, elastic, inspiratory, total), tidal volume, driving pressure, respiratory rate, and positive end-expiratory pressure, were recorded for each patient every 15 min for the duration of 2 consecutive hours on each mode. Statistical analysis, including paired t-tests were performed to assess the significance of differences between the two ventilation modes. The data is provided in means and ± SD.

Results

There were significant differences between PCV and AVM-2 in mechanical power (J/min): 21.62 ± 7.61 vs 14.21 ± 6.41 (P < 0.001), PEEP work (J): 4.83 ± 2.71 vs 4.11 ± 2.51 (P < 0.001), Tidal work (J): 3.83 ± 1.51 vs 2.21 ± 0.89 (P < 0.001), Elastic work (J): 8.62 ± 3.13 vs 6.32 ± 3.21 (P < 0.001), Resistive work (J): 3.23 ± 1.61 vs 1.81 ± 1.31 (P 0.013), Inspiratory work (J): 6.95 ± 2.58 vs 4.05 ± 2.01 (P < 0.001), Total work (J): 11.81 ± 3.81 vs 8.11 ± 4.23 (P < 0.001). There were significant differences between PCV and AVM-2 in tidal volume (ml): 511 ± 8.22 vs 413 ± 10.21 (P < 0.001), tidal volume / IBW 7.38 ± 1.74 vs 6.49 ± 1.72 (P 0.004), driving pressure (cmH_2O): 24.45 ± 6.29 vs 20.11 ± 6.59 (P 0.012), minute ventilation (L/min): 8.96 ± 1.34 vs 7.42 ± 1.41 (P < 0.001). The respiratory rate (bpm) was not significantly different between PCV and AVM-2 19.61 ± 4.32 vs 18.32 ± 1.43 (P 0.176). There were no significant differences between PCV and AVM-2 in static compliance (ml/cmH_2O) 20.24 ± 5.16 vs 22.72 ± 6.79 (P 0.346), $PaCO_2$ (mmHg) 44.94 ± 9.62 vs 44.13 ± 10.11 (P 0.825), and $PaO_2:FiO_2$ 243.54 ± 109.85 vs 274.21 ± 125.13 (P 0.343), but significantly higher power compliance index in PCV vs AVM-2: 1.11 ± 0.41 vs 0.71 ± 0.33 (P < 0.001).

Conclusion
This study demonstrates that the choice of mechanical ventilation mode, whether PCV or AVM-2, significantly impacts mechanical power and its constituent variables. AVM-2 mode was associated with reduced mechanical power, and its' components alongside the driving pressure, and tidal volumes, indicating its potential superiority in terms of lung-protective ventilation strategies. Clinicians should consider these findings when selecting the most appropriate ventilation mode to minimize the risk of ventilator-associated complications and improve patient outcomes. Further research is warranted to explore the clinical implications of these findings and to refine best practices in mechanical ventilation.

Key words: Mechanical power, Work, PCV, AVM-2, VILI

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Introduction

The utilization of mechanical ventilators is a crucial component of treating patients with respiratory failure. It is understood that mechanical ventilation brings forth risks to the patient, like ventilator induced lung injury (VILI). With VILI, the injured lung possesses less elasticity, and as a result, a decreased ability to withstand the strain (change in lung volume relative to the resting volume) and stress (transpulmonary pressure) applied to the parenchyma during the ventilation which result in acute lung injury. [1,2] Volutrauma, barotrauma, atelectrauma, biotrauma, myotrauma, and ergotrauma constitute the fundamental factors behind VILI. [3] With regards to the energetic contribution of the ventilator to the patient's respiratory system, the respiratory rate, flow rate and shape, tidal volume, tidal pressure, and positive end expiratory pressure (PEEP) are parameters that have been managed by clinicians to minimize VILI. [4]

Mechanical power has emerged and been further explored to be a parameter contributing to the risk of VILI. Composed of pressure, volume, flow, and respiratory rate, it quantifies the work applied by the ventilator on the respiratory system over time. [4] Consideration of mechanical power allows for the consolidation of the aforementioned ventilatory parameters, unifying them to present a single variable as a function of time that represents the mechanical forces that contribute to VILI. [5] Work by Zhang, Serpa and colleagues. demonstrated that mechanical power was associated with mortality in the first few days of ventilation. [5,6]

Pressure–controlled ventilation (PCV) is a common ventilatory modality described as pressure limited, time cycled set point continuous mandatory mode utilized to control the maximum peak pressure delivered to patients through selection of inspiratory and positive end-expiratory pressures (PEEP), and inspiratory time. [7] In the acute care setting, PCV is understood to be protective against barotrauma given the option to limit peak pressures. The control of pressure leaves the tidal volume as the independent variable, giving rise to the risk of volutrauma or dead space ventilation. [8]

Adaptive ventilatory mode (AVM) is an intelligent mode designed to automate the adjustment of the ventilatory parameters through continuous measurement and response of the patient-ventilator system. These operating algorithms were driven by the Otis equation of the least work of breathing. [9]

Further innovation of this concept led to the development of AVM-2 that operates with an optimal scheme based on the inspiratory power equation as described by van der Staay and colleagues. [10] This iteration aimed to minimize the inspiratory power, acting as a more protective ventilatory mode through the reduction of tidal volume, pressure, and mechanical power.

The clinician inputs the height and gender of the patient for the calculation of IBW, chooses the % Minute Ventilation (each 1% is equivalent to 1ml/kg/min, e.g., 70 kg with 100% is equivalent to minute ventilation of 7 Lpm) usually a start between 100-130%, and the ventilator determines the optimal or target combination of the respiratory rate, tidal volume (and thus the inspiratory pressure) based on respiratory mechanics and patients' effort. As respiratory mechanics and patient effort change, the algorithm adapts its targets within a "safe zone" Figure 1.

A review of the literature reveals few studies that have compared AVM-2 with other ventilatory modes. In a small clinical study, Becher and colleagues found that AVM-2 to be more lung-protective through reduction of inspiratory power when compared to the older version AVM. [11] A bench study conducted by our group revealed that AVM-2 delivered less mechanical power as compared to Pressure Regulated Volume Control (PRVC) and Volume Controlled Ventilation (VCV) in a normal lung model. [12] A similar comparison by our group investigated this same comparison in an several severities of acute respiratory distress syndrome (ARDS) model lung with different %MV and different PEEP levels. We found a significant decrease in mechanical power, tidal volume, and inspiratory pressures. [13]

In this study, we intended to analyze the differences between the established PCV and the innovative AVM-2 modes to measure disparities in mechanical power, including its key components: PEEP work, tidal work, elastic work, resistive work, inspiratory work, total work, tidal volume, tidal volume/IBW, and driving pressure. We hypothesized that AVM-2 would deliver less work (J) and mechanical power (J/min) compared to PCV, suggestive of greater lung protection.

Figure 1: Top: animated lung showing height, gender and IBW, along with the corresponding compliance (Cstat), Inspiratory resistance (Rinsp), % Spontaneous effort, airway occlusion in 100 msec (P0.1), Expiratory trans-pulmonary pressure (PTPExp). Bottom is the AVM minute volume showing the % and target MV. Graph depicts the target respiratory rate (x-axis) vs tidal volume (y-axis) curve, blue circle and arrows indicate the optimal combination chosen by the ventilator.

Methods

A randomized crossover study was conducted at Kuakini Medical center in Honolulu, Hawaii, USA between January and June 2023. The protocol was approved by the institution review board (IRB). Consent was obtained from the subject or their next of kin before enrollment in the study. Twenty-two subjects admitted to our intensive care unit (ICU) within the first day after initiation of mechanical ventilation participated in the study. Per protocol, mechanical ventilation was initially started on PCV, settings chosen by the primary treatment team, then switched to AVM-2 with comparable minute ventilation, using two Bellavista 1000e ventilators (Vyaire Medical Inc, Chicago, USA).

Mechanical power and its components, including tidal volume, driving pressure, respiratory rate, and PEEP, were recorded for each subject every 15 minutes for a duration of 2 hours in each mode (before and after the switch). Statistical analysis, including paired t-tests were performed to assess the significance of differences between the two ventilation modes. The data is provided in means and ± SD. Paired t-test was done to compare all the variables on the PCV and the AVM-2, a P value less than 0.05 was considered statistically significant. The 95 % confidence interval was calculated using paired sample confidence interval test with negative values indicating reduction of value and positive values indicating increase in value.

Inclusion criteria was any new subject admitted to the ICU within the first 24 hours and expected to remain on the ventilator for at least 24 hours. Exclusion criteria included: 1) age younger than 18 years old, 2) pregnant patients, 3) chronic respiratory failure with ventilator dependency, and 4) mechanical ventilation more than 24 hours. Because of this, eight patients were excluded (6 were chronically ventilator dependent, 1 refused to consent, and 1 consent could not be obtained for failure to reach the surrogate). Additionally, another 15 subjects could not be enrolled as the two ventilators were already in use. Subjects' characteristics are included in Table 1.

The primary team initiated care according to standard protocol, with mechanical ventilation using the PCV with settings aimed for lung protective ventilation aiming for plateau pressures below 30 cmH_20 and tidal volume between 6-8 ml/kg IBW. After a minimum of 2 hours to a maximum of 24 hours on PCV, this was followed by switching the mode to AVM-2 with %Minute ventilation equivalent to the minute ventilation before the switch (ranged between 90-135%). PEEP and FiO_2 levels were not changed, and all the subjects had continuous pulse oximetry (SpO_2) and end tidal CO_2 ($EtCO_2$) monitoring. Values of mechanical power, tidal work, PEEP work, resistive work, driving pressure, tidal volume, tidal volume/ideal body weight (IBW), and respiratory rate were analyzed every 15 minutes for 2 hours in each mode with eight calculations each (last 2 hours immediately before the switch on PCV, and the first 2 hours after the switch on AVM-2). After the 2 hours of AVM-2, the study was concluded, and the primary team made the decision to whether continue with AVM-2 or to return to PCV. We chose those consecutive periods in the hope that the respiratory mechanics would not change significantly affecting the study results.

Sedation and hemodynamic management decisions were left up to the primary team.

Ventilator data was analyzed using iVista app (Vyaire application). Calculations were done using Matlab (Mathworks, Massachusetts, USA). Data were represented as mean and standard deviation. Paired t-test were completed to compare all variables on the PCV and the AVM-2.

Formulas used in the calculations are set out below and further explained in the discussion section. Work is expressed as force (pressure) multiplied by distance (volume) in joules, while power (joules/minute) is work multiplied by respiratory rate/minute multiplied by a conversion factor of 0.098

Total work = Peak inspiratory pressure (PIP) X Tidal volume (V_T)
PEEP work = PEEP X V_T
Tidal work = ½ V_T X Tidal pressure (PIP – $PEEP_{Total}$)
Elastic work = PEEP work + Tidal work
Resistive work = Total work - Elastic work
Inspiratory work = Tidal work + Resistive work

Table 1: Characteristics of subjects included in study

Age (years)	30 - 89 (mean 65)
Gender	14 males & 8 females
Etiology of respiratory failure	8: pneumonia (including COVID-19) 7: ARDS (including COVID-19) 3: COPD exacerbation 2: Shock state 2: GI bleeding 1: Encephalopathy
APACHE-II score	21.61 ± 8.61
A-a gradient (mmHg)	309.57 ± 147.16
$PaCO_2$ (mmHg)	44.94 ± 9.62
PaO_2:FiO_2	202.54 ± 109.85
PEEP (cmH_2O)	12.11± 5.03

Results

Results are summarized in table 2, and figures 2-4.

Nearly all variables between PCV and AVM-2 were significantly lower in AVM-2 (Table 2), including mechanical power (J/min): PCV (21.62 ± 7.61) versus (vs) AVM-2 (14.21 ± 6.41) (P < 0.001), PEEP work (J): PCV (4.83 ± 2.71) vs AVM-2 (4.11 ± 2.51) (P < 0.001), tidal work (J): PCV (3.83 ± 1.51) vs AVM-2 (2.21 ± 0.89) (P < 0.001), elastic work (J): PCV (8.62 ± 3.13) vs AVM-2 (6.32 ± 3.21) (P < 0.001), resistive work (J): PCV (3.23 ± 1.61) vs AVM-2 (1.81 ± 1.31) (P = 0.013), inspiratory work (J): PCV (6.95 ± 2.58) vs AVM-2 (4.05 ± 2.01) (P < 0.001), and total work (J): PCV (11.81 ± 3.81) vs AVM-2 (8.11 ± 4.23) (P < 0.001) (Figure 2).

The components of work included 40.7 % of PEEP, 32.2 % of tidal, and 27.1 of resistive in PCV and 50.6 % of PEEP, 27.2 % of tidal, and 22.2% of resistive in AVM-2 (Figure 3).

There were also significant differences between PCV and AVM-2 in terms of tidal volume (ml): PCV (511 ± 8.22) vs AVM-2 (413 ± 10.21) (P < 0.001), tidal volume/IBW: PCV (7.38 ± 1.74) vs AVM-2 (6.49 ± 1.72) (P 0.004), driving pressure (cmH_2O): PCV (24.45 ± 6.29) vs AVM-2 (20.11 ± 6.59) (P 0.012), and minute ventilation (L/min): PCV (8.96 ± 1.34) vs AVM-2 (7.42 ± 1.41) (P < 0.001) (Figure 4).

The respiratory rate (bpm) was not significantly different between PCV (19.61 ± 4.32) vs AVM-2 (18.32 ± 1.43) (P = 0.176). There were no significant differences between PCV and AVM-2 in static compliance (ml/cmH_2O) 20.24 ± 5.16 vs 22.72 ± 6.79 (P 0.346), $PaCO_2$ (mmHg) 44.94 ± 9.62 vs 44.13 ± 10.11 (P 0.825), and PaO_2:FiO_2 243.54 ± 109.85 vs 274.21 ± 125.13 (P 0.343), but significantly higher power compliance index (PCI) in PCV vs AVM-2 1.11 ± 0.41 vs 0.71 ± 0.33 (P < 0.001).

17 out of the 22 remained on AVM-2 till extubation, 5 subjects died during their stay in the hospital, 3 on PCV, and 2 on the AVM-2. Average length of stay on mechanical ventilator was 7.11 ± 2.4 days. Our study is not powered to evaluate mortality difference.

Table 2: Comparison of PCV and AVM-2 recorded values. Data are presented as means ± SD, 95% confidence intervals

	PCV	AVM-2	% mean difference	P Value	95% CI
Mechanical Power (J/min)	21.62 ± 7.61	14.21 ± 6.41	41.39	< 0.001	–13.37, -5.11
Total work (J)	11.81 ± 3.81	8.11 ± 4.23	37.15	< 0.001	-2.04, -6.24
PEEP work (J)	4.83 ± 2.71	4.11 ± 2.51	16.1	< 0.001	-0.68, -3.02
Tidal work (J)	3.83 ± 1.51	2.21 ± 0.89	53.64	< 0.001	-1.09, -2.26
Resistive work (J)	3.23 ± 1.61	1.81 ± 1.31	56.35	0.013	-1.29, -3.36
Elastic work (J)	8.62 ± 3.13	6.32 ± 3.21	30.8	< 0.001	-2.63, -5.59
Inspiratory work (J)	6.95 ± 2.58	4.05 ± 2.01	52.73	< 0.001	-2.89, -4.72
Tidal volume (ml)	511 ± 8.22	413 ± 10.21	21.21	< 0.001	-49.6, -145.27
Tidal volume / IBW	7.38 ± 1.74	6.49 ± 1.72	12.83	0.004	-0.12, -2.26
Respiratory rate (Bpm)	19.61 ± 4.32	18.32 ± 1.43	6.78	0.176	-5.81, -1.19
Minute ventilation (L/min)	8.96 ± 1.34	7.42 ± 1.41	18.83	< 0.001	-1.28, 2.91

Driving pressure (cmH_2O)	24.45 ± 6.29	20.11 ± 6.59	19.47	0.012	-2.78, -7.76
Static Compliance (ml/cmH_2O)	20.24 ± 5.16	22.72 ± 6.79	11.55	0.346	2.89, 8.22
Power Compliance Index	1.11 ± 0.41	0.71 ± 0.33	43.96	< 0.001	-0.41, -0.71
$PaCO_2$ (mmHg)	44.94 ± 9.62	44.13 ± 10.11	1.82	0.825	-10.53, 4.41
PaO_2: FiO_2	243.54 ± 109.85	274.21±125.13	11.85	0.343	44.36, 157.19
PEEP (cmH_2O)	12.11± 5.02	12.11± 5.02	N/A	1.0	N/A

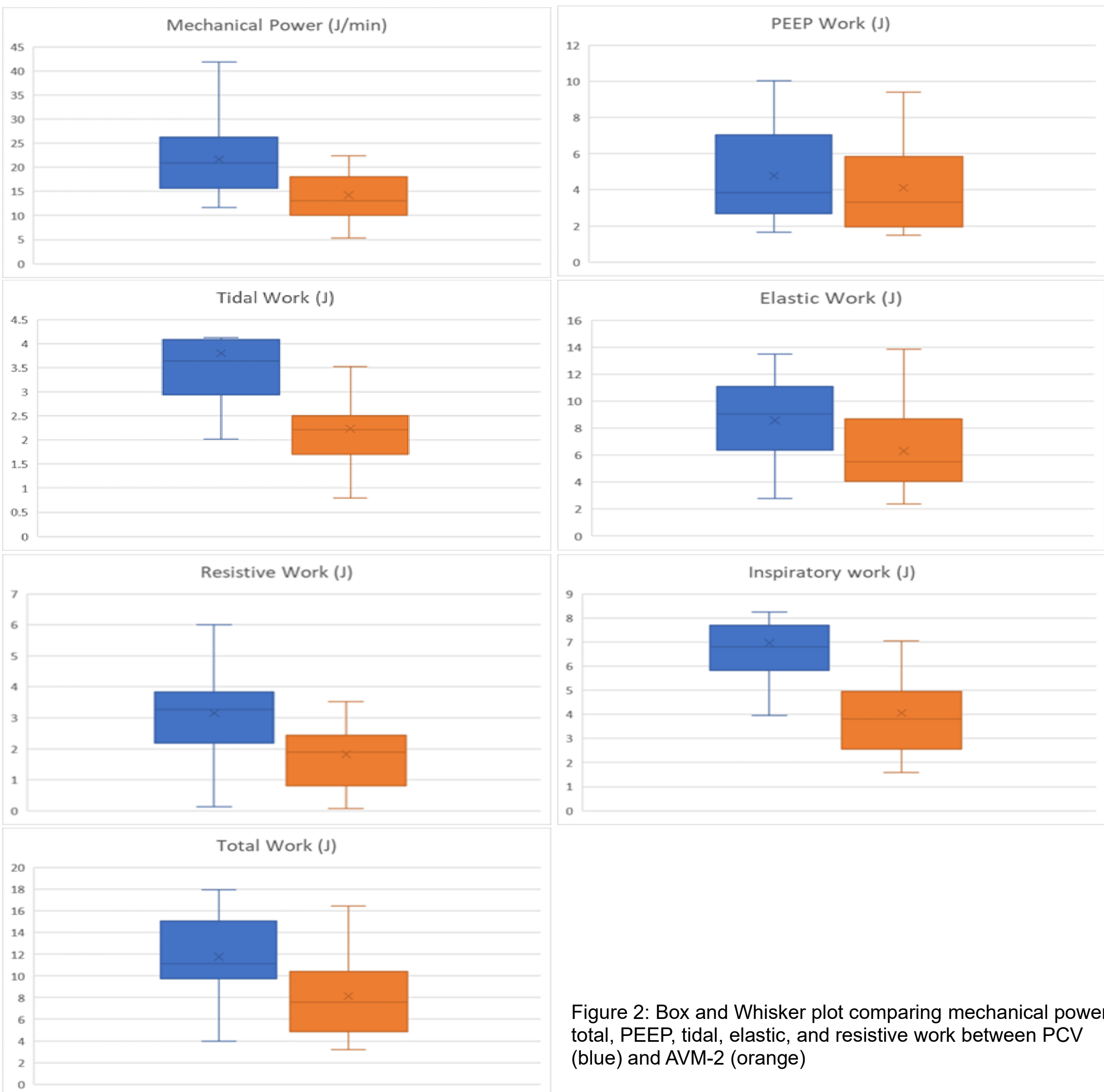

Figure 2: Box and Whisker plot comparing mechanical power, total, PEEP, tidal, elastic, and resistive work between PCV (blue) and AVM-2 (orange)

Figure 3: Pie chart showing the distribution of the components of work between PCV (left) and AVM-2 (right). PEEP (blue), tidal (orange), and resistive (blue)

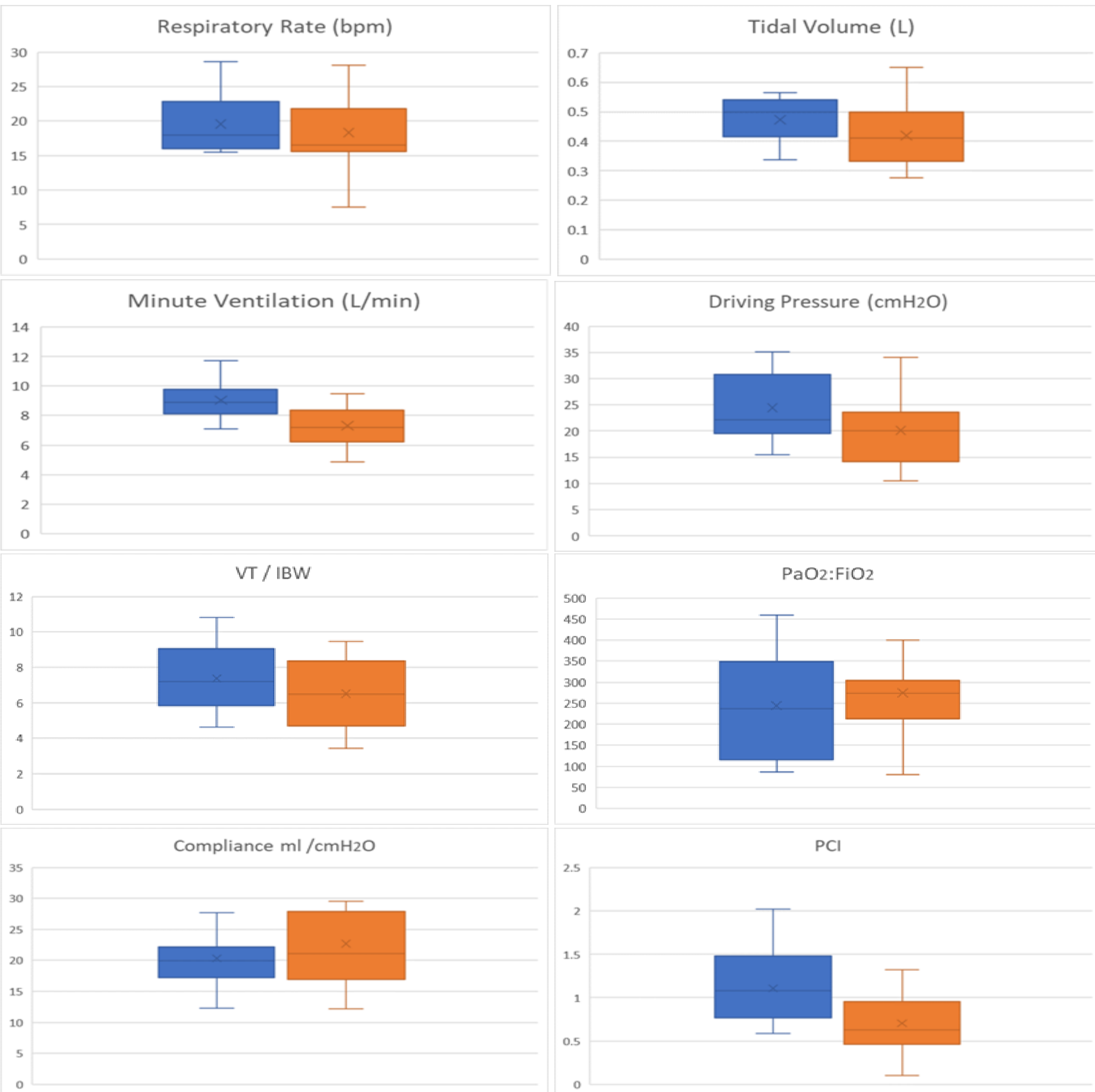

Figure 4: Box and Whisker plot comparing respiratory rate, tidal volume, minute ventilation, driving pressure, VT/IBW, PaO_2:FiO_2, compliance, and Power Compliance Index (PCI) between PCV (Blue) and AVM-2 (orange)

Discussion

Mechanical power is defined as the energy transferred to the respiratory system by the ventilator over a period of time, measured in Joules per minute. The optimal method of calculating mechanical power is geometrically integrating the area under the pressure-volume curve. [14] Comprehensive formulas have been developed but are difficult to use clinically at the bedside:

$$MP_{PCV(slope)} = 0.098 \cdot RR \cdot \left[(\Delta P_{insp} + PEEP) \cdot V_T - \Delta P_{insp}^2 \cdot C \cdot \left(0.5 - \frac{R \cdot C}{T_{slope}} + \left(\frac{R \cdot C}{T_{slope}} \right)^2 \cdot \left(1 - e^{\frac{-T_{slope}}{R \cdot C}} \right) \right) \right],$$

Where ΔP is the driving pressure, C is compliance, R is resistance, T_{Slope} is the slope of the rise time

Becher and colleagues [15] described a surrogate equation for calculating the mechanical power of pressure-controlled ventilation operating on the assumption of idealized square waveforms for airway pressure with constant resistance. In their equation, the force (P) is multiplied together with changes in volume (V), respiratory rate (RR) and a conversion factor of 0.098. [16]

$MP_{PCV} = 0.098 \times RR \times V_T (\Delta P_{insp} + PEEP)$

Of note, in pressure controlled modes, the inspiratory time might affect the tidal volume according to the time constant concept (tidal volume will increase until the inspiratory flow reaches zero flow before expiration, remains constant with further increase, and will decrease once auto-PEEP occurs). The above formula indirectly accounts for this, as the calculated tidal volume is equal to: Inspiratory Flow Rate × Inspiratory Time. [17]

Our study is the second clinical study to compare AVM-2 to other modes of ventilation. Becher and colleagues [11] studied 20 patients in a cross-sectional study to compare AVM-2 mode to AVM, which is the older version of the mode based on Otis' equation, while AVM-2 is based on the inspiratory power equation below [10]

$$f = \frac{1 + 2a \times RC \times \frac{\text{Min Vol} - (f \times V_d)}{V_d} - 1}{a \times RC}$$

Where *f* is the respiratory rate, RC is respiratory compliance, MinVol is minute ventilation, Vd is dead space, and a is (2π2)/60.

Similar to our study they found significant reductions of mechanical power, driving pressure, and tidal volume in AVM-2 compared to AVM.

Our study is the first to compare AVM-2 to the conventional mode pressure-controlled ventilation (PCV). By recording the different variables of power in 15 minutes intervals x 2 hours, we determined a statistically significant difference in mechanical power between the AVM-2 mode versus conventional PCV. There were also statistically significant differences in PEEP work, tidal work, resistive work, and subsequently elastic work, inspiratory work, and total work.

Our study is the first clinical study to calculate all the components of work. Our results concluded that all the components of work were statistically lower when AVM-2 was utilized compared to PCV.
The total work done for each breath can be further subdivided into PEEP work, tidal work, resistive work. Furthermore, the sum of the PEEP and tidal work constitute the elastic work, and the sum of the tidal and resistive work constitute the inspiratory work (Figure 5).

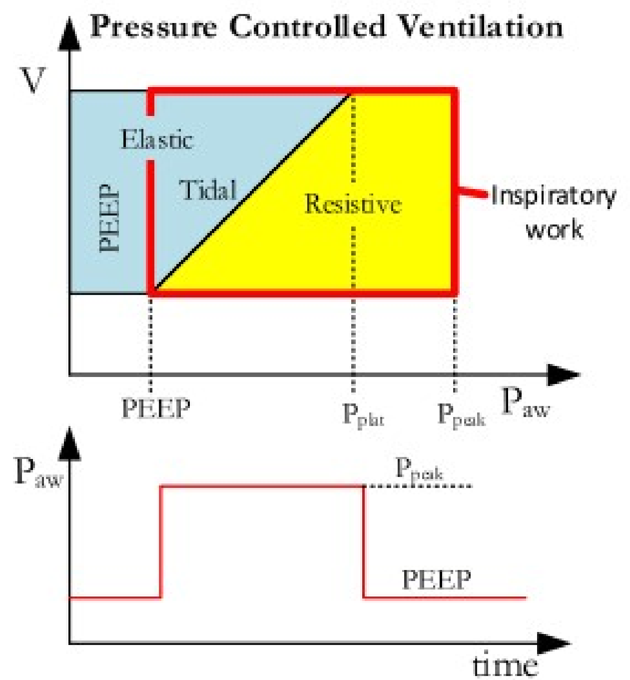

Figure 5: Components of work in a single breath in a pressure-controlled mode either PCV or AVM. Top graph is the pressure-volume curve, bottom one is airway pressure-time curve. Blue shaded area is the elastic work (PEEP and tidal), the yellow shaded is the resistive work. The red bordered area is the inspiratory work (tidal and resistive). PEEP: positive end expiratory pressure, Pplat: plateau pressure, Ppeak: peak inspiratory pressure, Paw: airway pressure, all in cmH2O. If inspiratory flow (not shown) is down to zero before exhalation, the Ppeak = Pplat. Adapted from reference 10.

The components of work were calculated per the formulas described in the method section above according to the simplified verified by Becher [15] and van der Staay. [10]

It is unclear which component or combination of components contribute to VILI. Marini and colleagues [18] argue the point that PEEP work “is temporarily stored as potential energy within the elastic tissues of the respiratory system; it later is converted to kinetic energy as the gas escapes to the atmosphere across the exhalation valve”. Similarly, the resistive energy is dissipated as heat in the airways and may not be very relevant for the development of VILI. They argue that tidal power might be the important one. Our results indicate that AVM-2 reduced tidal work by 53% and its contribution to total work by 5% from 32.2% to 27.2%.

Vassalli and colleagues [19] conducted an experimental study in porcine model with different ventilatory strategies (high tidal volume, high respiratory rate, and high PEEP) with an Iso-mechanical power, and found that different ventilatory strategies, delivered at iso-power, led to similar anatomical lung injury. Franck and colleagues [20] found correlation between mechanical power and its components in COVID-19 ARDS patients undergoing mechanical ventilation using the PCV mode.

We additionally calculated the Power Compliance Index (PCI) as the ratio of the mechanical power divided by the compliance of the respiratory system between the two modes. In a previous bench study by our group, [21] in independent lung ventilation, we coined the term Power compliance Index. Theoretically, a well aerated lung with better compliance will require less mechanical power i.e., a lower PCI, versus a non-aerated lung with poorer compliance which requires a higher mechanical power i.e., higher PCI to achieve targets of ventilation.

Our results showed significant reduction of the PCI using the AVM-2 mode. This finding confirms our previous bench work where we found lower mechanical power, and PCI between AVM-2 compared to conventional VCV and PCV in an ARDS lung model with different severities. [13]
As Marini and colleagues [18] suggested, normalizing or indexing the mechanical power to the compliance of the lung or the amount of aerated lung might be more meaningful than mechanical power alone, as it represents the amount of energy delivered to a specific injured unit. Coppola and colleagues [22] conducted a retrospective study in ARDS patients and found that the mechanical power alone did not correlate with mortality, however, mechanical power normalized to the compliance or to the amount of well-aerated tissue is independently associated with mortality.

Lower tidal volume has been linked to lower mortality in ARDS [23] and has been the standard of care in most of the guidelines. [24] AVM-2 is designed to automatically select the optimal tidal volume for each patient. In our study, we observed that the tidal volumes were lower in AVM-2 mode. This phenomenon could be attributed to the AVM-2's capability to adapt to a patient's spontaneous breathing efforts, thereby reducing mechanical support when it detects adequate spontaneous ventilation. Another explanation is that the AVM-2 mode incorporates a lung-protective strategy, deliberately utilizing the lowest effective tidal volumes per the ideal body weight.

Our findings align with those of Becher and colleagues [11] who demonstrated that tidal volumes in critically ill patients including ARDS were significantly lower under AVM-2 compared to those in AVM.
In a bench study by van der Staay and Chatburn, [10] AVM-2 was able to deliver the lowest tidal volume/IBW compared to two other optimal targeting schemes modes, adaptive support ventilation (ASV), and MID-frequency ventilation (MFV).

Our study also concluded that driving pressure (inspiratory or tidal pressure) was lower in AVM-2 mode. The driving pressure in mechanical ventilation is the difference between the plateau pressure and the total PEEP, and act as a surrogate for the inspiratory transpulmonary pressure that is linked to the stress applied to the lung [25] and has been linked to mortality in ARDS. [26] Therefore, this result could be expected because driving pressure is essentially the pressure required to deliver a set tidal volume during ventilation without considering the PEEP.

To the best of our knowledge, this is the first clinical study to directly compare tidal volumes and driving pressure between PCV and AVM-2. In previous two bench studies by our group, [12,13] in normal and ARDS lung scenarios, compared to conventional PCV and VCV, AVM-2 was able to reduce the mechanical power, driving pressures, and tidal volumes using the same minute ventilation.
However, it was noted that the driving pressures in our study was higher than the recommended by the guidelines, probably owing to the fact that the average compliance was low in our study (20.24), and the initial settings were targeting tidal volume of 6-8 ml/kg.

Though our study was not powered to investigate mortality differences using AVM-2, our observed mortality rate was 22% (5 of 22 subjects), which is lower than observed mortality of mild ARDS in the literature. [27]

Our study has some weaknesses and results should be interpreted with care. The study was done in a single tertiary care center and has a small number of subjects. Additionally, it is a crossover study not a randomized control study between two different modes. We did not record the short or long term outcomes of the subjects in the AVM-2 mode. Though we initiated the AVM-2 mode with a comparable minute ventilation to that of the PCV before the switch, our results showed that the minute ventilation was lower in the AVM-2 which could be secondary to the changes of minute ventilation on the PCV either by settings change during the 2 hours of PCV or changes of patients' effort or respiratory mechanics which would have changed the respiratory rate and tidal volume and thus the minute ventilation. We don't think that this would have changed the findings as our previous bench studies showed the same findings with the exact same minute ventilation. Additionally, the $PaCO_2$ levels were not different between both modes despite lower minute ventilation, thus a lower minute ventilation in AVM-2, might reflect lower alveolar dead-space ventilation because of less lung stretch (better compliance).

We attempted to include all patients admitted to our ICU in the study but as many as 15 had to be excluded as we only had 2 ventilators with the AVM-2 mode which could have contributed to a selection bias. Lastly, our study group included a heterogenous group of different etiologies of respiratory failure but given the mean PaO_2:FiO_2 of 202 and compliance of 20.24 ml/cmH_2O indicating that the majority of patients had restrictive or some sort of ARDS.

Conclusion

AVM-2 mode was associated with reduced mechanical power, variable work components, driving pressure, and tidal volumes, indicating its potential superiority in terms of lung-protective ventilation strategies. Clinicians should consider these findings when selecting the most appropriate ventilation mode to minimize the risk of ventilator-associated complications and improve patient outcomes. Further research is warranted to explore the clinical implications and outcomes of these findings and to refine best practices in mechanical ventilation.

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