Turk J Intensive Care 2026;24(1):77-82
DOI: 10.4274/TJIC.2026.689

ORIGINAL RESEARCH

The impact of the inspiratory-to-expiratory ratio on mechanical power in ARDS patients

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ABSTRACT

Objective: Mechanical power (MP) is a promising parameter that may guide lung-protective ventilation strategies. The MP equation encompasses all variables involved in the pathogenesis of ventilator-induced lung injury (VILI), as well as the inspiratory-to-expiratory (I:E) ratio. Shortening the inspiratory time—by altering the I:E ratio—increases gas flow and may lead to lung injury. The optimal I:E ratio for preventing VILI remains uncertain. The present study aimed to investigate the effects of different I:E ratios on inspiratory gas flow and MP in patients with acute respiratory distress syndrome (ARDS).

Materials and Methods: This study was conducted on 19 adult patients diagnosed with moderate ARDS who were admitted to the intensive care unit and received mechanical ventilation. Patients were ventilated at a PEEP level of 10 cmH_2O, a tidal volume of 6 mL/kg, and three sequentially applied I:E ratios (1:2, 1:1, and 1.5:1), each maintained for 10 minutes. MP and respiratory parameters across the three I:E groups were compared using repeated-measures ANOVA.

Results: Compared with the I:E ratio of 1:2, ventilation with an I:E ratio of 1:1 resulted in slower inspiratory gas flow, lower inspiratory resistance and peak inspiratory pressure, higher dynamic compliance, and lower MP values. Plateau pressure and driving pressure did not differ significantly. When comparing I:E 1:1 with I:E 1.5:1, a persistent slowing of inspiratory gas flow was observed, but no significant difference in MP was detected.

Conclusion: In patients with ARDS, transitioning the I:E ratio from the conventional 1:2 to 1:1 or 1.5:1 resulted in progressively slower inspiratory gas flow and lower resistive and total mechanical power, suggesting that reducing inspiratory flow may enhance lung-protective ventilation.

Keywords: ARDS, ventilator-induced lung injury, mechanical power, inspiratory-to-expiratory ratio

Introduction

Mechanical ventilation is widely used in the management of respiratory failure and remains one of the cornerstones of intensive care medicine. Ventilator-induced lung injury (VILI) refers to the damage caused by positive-pressure ventilation (1). The development of VILI is influenced by several factors, including tidal volume, transpulmonary pressure, respiratory rate, flow rate, and positive end-expiratory pressure (PEEP) (2-4). These parameters represent the energy applied to the respiratory system, and the total energy delivered per minute is termed mechanical power (MP) (5,6). Mechanical power has been identified as an independent risk factor associated with mortality in critically ill patients (7).

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Received: 15.01.2024 **Accepted:** 05.02.2026 **Published:** 26.03.2026

The comprehensive formula developed by Gattinoni et al. incorporates all parameters associated with VILI, as well as the inspiratory-to-expiratory (I:E) ratio (6):

$$MPrs = RR \times \{ \Delta V^2 \times [(1/2 \times ELrs) + RR \times (1 + I:E) / (60 \times I:E) \times Raw] + (\Delta V \times PEEP) \} \times 0.098$$

The viscoelastic structure of the lung causes the inspiratory phase of positive-pressure ventilation to require more energy than expiration (8). This energy dissipation corresponds to the area of hysteresis on the pressure–volume (P–V) curve (8). The energy expended within the lung parenchyma is used both for expansion and recoil during breathing and to overcome viscoelastic resistance during rapid inspiration and early expiration (9). This energy is also consumed when direct structural damage occurs at the microelement level (9). Increasing strain rates applied to the viscoelastic polymer structure of the lung elevate the risk of injury (10). Studies have shown that shortening the inspiratory time and altering the I:E ratio to increase gas flow can result in higher strain rates and promote the development of ventilator-induced pulmonary edema (10).

Studies have demonstrated that high driving pressure (DP) and high tidal volume (TV) are the main mediators of ventilator-induced lung injury (VILI) (11-13). In mechanical ventilators, the conventional I:E ratio, in which the inspiratory time is shorter than or equal to the expiratory time, is typically set to 1:2 or 1:1 (9). Although traditional ventilation strategies employing high pressures, high volumes (12 mL/kg), and high gas flow rates have largely been abandoned, the standard I:E ratio of 1:2 remains unchanged. The I:E ratio, similar to respiratory rate and tidal volume, is a key mechanical ventilation parameter that determines gas flow rate. The relationship between the I:E ratio and VILI is thought to depend on gas flow dynamics (14). Slow inspiratory gas flow is considered a critical factor in preventing VILI (9,14).

In intensive care units, different inverse ratio ventilation (IRV) strategies—such as I:E ratios of 1.5:1 and 2:1—are often employed to enhance oxygenation, serving as an essential intervention for patients with ARDS (15). However, the optimal I:E ratio for preventing VILI in this patient population remains uncertain.

This study aimed to determine the impact of varying I:E ratios on inspiratory flow and MP in ARDS patients.

Materials and Methods

Patients

Ethical approval for the study was obtained from the Clinical Research Ethics Committee of the University of Health Sciences, Bakırköy Dr. Sadi Konuk Training and Research Hospital (Decision No: 2019-19-15). Informed consent was obtained from all patients or their legal representatives upon admission to the intensive care unit.

This observational study was conducted during a consecutive three-month period in the Intensive Care Unit of the Department of Anesthesiology and Reanimation at Bakırköy Dr. Sadi Konuk Training and Research Hospital. Nineteen adult patients diagnosed with moderate ARDS according to the Berlin Criteria were included in the study (16).

All patients were ventilated using Maquet Servo-i ventilators (Sweden). According to the Berlin Criteria, the enrolled patients were classified as having moderate ARDS (16). Patient characteristics are presented in Table 1.

Patients were ventilated in volume-controlled mode under deep sedation with propofol and remifentanil, and neuromuscular blockade was achieved using rocuronium. Invasive mechanical ventilation was applied with a PEEP of 10 cmH_2O, a tidal volume of 6 mL/kg, and a respiratory rate of 12 breaths/min. Each patient was ventilated sequentially with three different inspiratory-to-expiratory ratios (1:2, 1:1, and 1.5:1), each maintained for 10 minutes.

All respiratory parameters were recorded every minute using the ImdSoft Metavision/QlinICU Clinical Decision Support Software (Canada), including peak

inspiratory pressure (Ppeak), positive end-expiratory pressure (PEEP), respiratory rate (RR), inspiratory gas flow, inspiratory resistance (Ri), expiratory resistance (Re), end-expiratory velocity (Vee), static compliance (Cstat), and dynamic compliance (Cdyn).

Plateau pressure (Pplat) was automatically measured by the ventilator with a 5% end-inspiratory pause (Tpause). Driving pressure (DP) was calculated using a pre-defined formula on the server (DP = Pplat – PEEP). MP and its components (resistive and elastic) were automatically calculated based on formulas previously defined on the server, as described by Gattinoni et al. (6).

Calculation of total, elastic, and resistive mechanical power:

$$MP\ total = RR \times \{\Delta V^2 \times [\ (1/2 \times ELrs) + RR \times (1 + I:E) / (60 \times I:E) \times Raw\ ] + (\Delta V \times PEEP)\} \times 0.098$$

$$MP\ elastic = RR \times \{\ \Delta V^2 \times [\ (1/2 \times ELrs)\ \}\ \times 0.098$$

$$MP\ resistive = RR \times \{\ \Delta V^2 \times [\ RR \times (1 + I:E) / (60 \times I:E) \times Raw\ ] \times 0.098$$

Statistical methods

Statistical analyses were performed using GraphPad Prism software (version 5.01). Data obtained at different inspiratory-to-expiratory (I:E) ratios (1:2, 1:1, and 1.5:1) were compared using repeated-measures ANOVA. Bonferroni post hoc tests were applied to identify significant pairwise differences between groups, with $p < 0.05$ considered statistically significant.

The primary outcome of the study was defined as the difference in resistive mechanical power between the I:E 1:2 and I:E 1:1 ratios. Based on a preliminary pilot study including five patients, the mean ± SD values for resistive mechanical power were calculated as 0.9 ± 0.3 J/min for I:E 1:2 and 0.65 ± 0.3 J/min for I:E 1:1. According to a power analysis based on these differences, with an alpha error of <0.05 and a power of 80%, the required sample size was estimated to be 18 patients.

The presence of auto-PEEP was assessed by performing an expiratory hold maneuver after each adjustment to measure total PEEP (17). If the end-expiratory flow (Flowee) was close to zero, the absence of intrinsic PEEP was assumed (17). In this study, Flowee values obtained at all I:E ratios were measured close to zero (0.008 ± 0.005; 0.015 ± 0.005; 0.022 ± 0.013), indicating the absence of intrinsic PEEP in the study population.

Results

This study was conducted on 19 patients diagnosed with moderate ARDS. Of these, 7 were female and 12 were male. The characteristics of the patients included in the study are presented in Table 1.

The difference between the mean mechanical power values of I:E 1:2 and I:E 1:1 was calculated as 0.33 J/min (4.3%) and found to be statistically significant ($p < 0.0001$). No statistically significant difference was observed between the mean and standard deviation values of I:E 1:2 vs. I:E 1.5:1 or I:E 1:1 vs. I:E 1.5:1 ($p > 0.05$). There was no statistically significant difference in the mean and standard deviation values of elastic

Table 1. Patient characteristics

Parameter	Mean ± SD
Female, n (%)	7 (%37)
Age, years	65 ± 11
PBW, kg	71 ± 2
BMI, kg/m²	28 ± 1.3
APACHE II score	25 ± 9
Expected mortality, %	59 ± 29
pH	7.43 ± 0.2
PaO_2, mmHg	62 ± 7
SO_2, %	91 ± 2
PCO_2, mmHg	44 ± 16
PaO_2/FiO_2, mmHg	119 ± 14
Duration of IMV, hours	272 ± 118
Length of ICU stay, hours	442 ± 224

APACHE II: Acute Physiology and Chronic Health Evaluation II, ARDS: Acute Respiratory Distress Syndrome, BMI: Body Mass Index, FiO_2: Fraction of Inspired Oxygen, IMV: Invasive Mechanical Ventilation, ICU: Intensive Care Unit, PaO_2: Arterial Oxygen Partial Pressure, PBW: Predicted Body Weight, PCO_2: Arterial Carbon Dioxide Partial Pressure, SO_2: Arterial Oxygen Saturation.

mechanical power among the three I:E ratios (1:2, 1:1, and 1.5:1) ($p > 0.05$). The difference between the mean resistive mechanical power values of I:E 1:2 vs. I:E 1:1 and I:E 1:2 vs. I:E 1.5:1 was calculated as 0.3 J/min (4.0%) and was statistically significant ($p < 0.0001$). No statistically significant difference was detected between the mean and standard deviation values of resistive mechanical power for I:E 1:1 vs. I:E 1.5:1 ($p > 0.05$) (see Table 2).

The differences between the mean and standard deviation values of Ppeak, WOBv, and Ri for I:E 1:2 vs. I:E 1:1 and I:E 1:2 vs. I:E 1.5:1 were statistically significant ($p < 0.05$). However, no statistically significant differences were observed for these parameters between I:E 1:1 and I:E 1.5:1 ($p > 0.05$). Statistically significant differences were also found in the mean and standard deviation values of inspiratory gas flow, Vee, and Cdyn across all three I:E ratios ($p < 0.05$), whereas Pplat, DP, and Cstat showed no statistically significant differences ($p > 0.05$) (see Table 2).

Discussion

MP, encompassing all parameters linked to VILI, serves as a key guide in its prevention. In this study, while all parameters in the MP equation developed by Gattinoni et al. were kept constant, the effect of changes in the I:E ratio on total MP and its components was investigated.

The inspiratory gas flow, Ri, and Ppeak values at an I:E ratio of 1:1 were found to be lower compared with those at an I:E ratio of 1:2. Consequently, the I:E 1:1 setting was associated with higher dynamic compliance and lower MP values. When I:E 1:1 was compared with I:E 1.5:1, the inspiratory gas flow remained slower; however, no clinically significant differences were observed in other respiratory parameters. The significant difference in MP between I:E 1:2 and I:E 1:1 was attributed to the reduction in the MPresistive, resulting from slower inspiratory gas flow and decreased Ri. Changing the I:E ratio from 1:2 to 1:1 reduced the amount of MP applied to the

Table 2. Mechanical power and its components at different I:E ratios (Mean ± SD)

	I:E 1:2 vs I:E 1:1	I:E 1:2 vs I:E 1.5:1	I:E 1:1 vs I:E 1.5:1
MP_{tot}, J/min	7.6±1.0 vs 7.3±0.8*	7.6±1.0 vs 7.3±0.9 ns	7.3±0.8 vs 7.3±0.9 ns
$MP_{elastic}$, J/min	2.4±0.6 vs 2.4±0.6ns	2.4±0.6 vs 2.5±0.6ns	2.4±0.6 vs 2.5±0.6ns
$MP_{resistive}$,J/min	0.9±0.3 vs 0.6±0.2***	0.9±0.3 vs 0.6±0.3***	0.6±0.2 vs 0.6±0.3ns
WOB_v, J	0.86±0.16 vs 0.70±0.15***	0.86±0.16 vs 0.68±0.14***	0.70±0.15 vs 0.68±0.14ns
P_{peak}, cmH_2O	24.5±2.6 vs 22.9±2.7***	24.5±2.6 vs 23.0±2.8***	22.9±2.7 vs 23.0±2.8ns
P_{plat}, cmH_2O	21±2.5 vs 21.1±2.5ns	21±2.5 vs 21.2±2.5ns	21.1±2.5 vs 21.2±2.5ns
DP, cmH_2O	11.1±2.6 vs 11.2±2.7ns	11.1±2.6 vs 11.2±2.6ns	11.2±2.7 vs 11.2±2.6ns
$Flow_{ins}$, L/s	0.37±0.04 vs 0.20±0.02***	0.37±0.04 vs 0.16±0.02***	0.20±0.02 vs 0.16±0.02***
Ri, cmH_2O. s/L	9.6±3.3 vs 8.5±2.9*	9.6±3.3 vs 8.7±3.6*	8.5±2.9 vs 8.7±3ns
Re, cmH_2O. s/L	17.2±3.8 vs 17.5±3.8ns	17.2±3.8 vs 18.0±4.1*	17.5±3.8 vs 18.0±4.1ns
Vee, L / s	0.008±0.005 vs 0.015±0.005*	0.008±0.005 vs 0.022±0.013***	0.015±0.005 vs 0.022±0.013*
C_{stat}, ml/cmH_2O	34.5±9.6 vs 34.7±9.6ns	34.5±9.6 vs 34.1±9.2ns	34.7±9.6 vs 34.1±9.2ns
C_{dyn}, ml/cmH_2O	35.8±9.4 vs 36.3±9.4**	35.8±9.4 vs 34.4±9.0**	36.3±9.4 vs 34.4±9.0**

MPtot: total mechanical power, MPelastic: Elastic mechanical power, MPresistive: Resistive mechanical power, Cst: Static compliance, Cdyn: Dynamic compliance, Ppeak: Peak inspiratory pressure, Pplat: Plateau pressure, DP: Driving Pressure, $Flow_{ins}$: inspiratory gas flow, Ri: inspiratory resistance, Re: expiratory resistance, Vee= end expiratory velocity.

ns: not significant ($p \geq 0.05$); * $p < 0.05$; ** $p < 0.01$; *** $p < 0.0001$.

conductive components of the respiratory system that deliver gas to the alveoli. Since the elastic component of MP, which represents the energy applied to the alveoli, did not change, this reduction was considered clinically insignificant in terms of VILI risk.

Minimizing gas flow velocity during the inspiratory phase of the respiratory cycle is recommended to reduce the energy that contributes to lung injury (5,9,14). Therefore, the use of high airway pressures, high tidal volumes, or high respiratory frequencies should be avoided (5,9). Excessive energy levels can severely damage the alveolocapillary barrier, while even small amounts may cause localized injury in already weakened lung regions (9). The reduction of ventilated areas—the so-called "baby lung" phenomenon—can lead to the rupture of microstructural elements and increase stress on the remaining lung tissue. Once a certain threshold is exceeded, this may result in an exponential progression of injury. Thus, gas flow plays a critical role under such conditions (9). Adopting a higher I:E ratio, such as 1:1 instead of the conventional 1:2, may promote a slower and more homogeneous inspiratory flow, thereby reducing the MP applied to the lungs.

In an experimental study, the energy accumulated in the airway tissue during slow, constant inspiratory flow was found to be approximately half of that measured in the group ventilated with an exponentially increasing, high inspiratory flow pattern (18). Another study conducted under operating room conditions emphasized that an I:E ratio of 1:1 provides the minimum energy dissipation (19). To optimize mechanical power and prevent ventilator-induced lung injury (VILI), it has been recommended that the I:E ratio be adjusted to 1:1 (20).

An experimental study in mice investigated the effect of prolonging inspiratory time on ventilator-induced lung injury (VILI) (21). In this study, the use of low tidal volume with I:E ratios of 1:1 or 1:2 did not result in the development of VILI. In the high tidal volume group, however, the I:E ratio of 1:2 produced fewer histological signs of lung injury and better compliance values compared with I:E 1:1. In contrast, the group ventilated with high tidal volume and an I:E ratio of 1:1 showed worsened oxygenation and compliance, as well as a significantly increased mortality rate. The authors concluded that prolongation of inspiratory time and an increased I:E ratio exacerbated VILI in mice. However, in that study, the increased VILI and mortality associated with an I:E ratio of 1:1 were observed only when high tidal volumes were applied, which are already a well-known risk factor for VILI.

Limitations

This study has several limitations. First, it was conducted in a single center with a relatively small sample size, which may limit the generalizability of the findings. Second, the study was performed under controlled ventilation settings with fixed tidal volume and PEEP levels; therefore, the results may not be directly applicable to patients under spontaneous or assisted ventilation. Third, the analysis was limited to short-term physiological effects, and no long-term clinical outcomes such as mortality or duration of ventilation were evaluated.

Conclusion

In patients with ARDS, transitioning the I:E ratio from the conventional 1:2 to 1:1 or 1.5:1 resulted in progressively slower inspiratory gas flow and lower resistive and total mechanical power, suggesting that reducing inspiratory flow may enhance lung-protective ventilation.

Ethical approval

This study has been approved by the Bakırköy Dr. Sadi Konuk Training and Research Hospital Clinical Research Ethics Committee (approval date: 30.09.2019, number: 2019-19-15). Written informed consent was obtained from the participants or their legal representatives.

Author contribution

Study conception and design: SA, FT; data collection: SA, FT, ÖA; analysis and interpretation of results: SA, FT, ÖA; draft manuscript preparation: SA, FT, ZÇ, GOH, NÇ. The author(s) reviewed the results and approved the final version of the article.

Source of funding

The authors declare the study received no funding.

Conflict of interest

The authors declare that there is no conflict of interest.

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