

Mechanical Power Normalized to Predicted Body Weight is Associated with Mortality in Critically Ill Patients: A Cohort Study

Yanhong Zhu

The First People's Hospital of Pinghu

Wenyong Peng

Jinhua Municipal Central Hospital

Shuai Zhen

Jinhua Municipal Central Hospital

Xiaofeng Jiang (✉ 719085915@qq.com)

Jinhua Municipal Central Hospital

Research Article

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Abstract

Background: Mechanical power (MP), defined as the amount of energy produced by mechanical ventilation and released into the respiratory system, has long been considered a critical factor in the pathogenesis of ventilator-induced lung injury. However, our knowledge suggests that the effects of MP may be proportional to their involvement in total lung function size. Therefore, MP normalized to predicted body weight (norMP) should be greater than absolute MP value. The objective of this research is to determine the connection between norMP and mortality in critically ill patients who have been on invasive ventilation for at least 48 hours.

Methods: This is a study of data stored in the databases of the MIMIC-III, which contains data of critically ill patients for over 50,000. The study involved critically ill patients who had been on invasive ventilation for at least 48 hours. norMP was the relevant exposure. The major endpoint was ICU mortality, the secondary endpoints were 30-day, 90-day mortality; ICU length of stay, the number of ventilator-free days at day 28.

Result: The study involved a total of 1301 critically ill patients. This study revealed that norMP was correlated with ICU mortality [OR per quartile increase 1.20 (95% CI 1.07–1.35), $p = 0.009$]. Similarly, norMP was correlated with ventilator-free days at day 28, ICU length of stay. In the subgroup analysis, high norMP was associated with ICU mortality whether low or high Vt (OR 1.22, 95% CI 1.01–1.47, $p = 0.040$; OR 1.28, 95% CI 1.06–1.54, $p = 0.011$, respectively). But high norMP was associated with ICU mortality only in low PIP (OR 1.18, 95% CI 1.01–1.37, $p = 0.034$)

Conclusion: Our findings indicate that higher norMP is independently linked with elevated ICU mortality and various other clinical findings in critically ill patients with a minimum of 48 hours of invasive ventilation.

Background

In surgical operations and critically ill patients, mechanical ventilation is a vital component of supportive treatment used to preserve respiratory function and minimize respiratory work (1–3). During mechanical ventilation, however, the mechanical force produced by the interaction of the ventilator and the respiratory tract may cause damage to the lungs. This condition is known as ventilator-induced lung injury (VILI) (4–6). A complex combination of various mechanical forces acting on the pulmonary structure during mechanical ventilation such as macrophages, endothelial cells, type I and II epithelial cells, peripheral airways, and extracellular matrix (ECM) appears to result of the pathogenesis of VILI (1, 3).

The severity of VILI is determined by the settings on the ventilator (7). Certain variables, such as tidal volume (Vt), respiratory rate (RR), driving pressure (ΔP) and positive end-expiratory pressure (PEEP), are set directly on the ventilator by the clinician (1). Others, such as peak (PIP) and plateau pressures, rely on the patient's respiratory system or on his interaction with the ventilator. Up to now so far, all these factors have been assessed separately (8), while mechanical power (MP), defined as the amount of energy

produced by mechanical ventilation and released into the respiratory system, has been considered to be the determinant factor in the pathogenesis of VILI (7, 9, 10). Given that MP incorporates numerous aspects of mechanical ventilation, it should outperform individual ventilator setting components in terms of modulating the final effect on the VILI (7, 9).

The applicability of MP in current clinical practice is increasing by accurately calculated through the "power equation" (7). Increases in MP measured on the second day after ICU admission were correlated with increased hospital mortality in a recent study of 8207 critically ill mechanically ventilated patients (11). In addition, our knowledge suggests that the effects of MP may be proportional to their involvement in total lung function size. Therefore, MP normalized to predicted body weight (norMP) should outperform absolute MP value (8). Just like normalizing tidal volume to predicted weight (PBW) (12). However, research to date has not yet determined whether norMP is associated with outcomes of critically ill mechanically ventilated patients.

The objective of this study was to explore the prognostic role of mechanical power normalized to predicted body weight in the clinical outcomes of intensive care patients.

Methods

Data source

The Massachusetts Institute of Technology's Laboratory for Computational Physiology maintains the Multiparameter Intelligent Monitoring in Intensive Care III (MIMIC III, V.1.4) database, which includes data on over 50,000 patients admitted to the intensive care unit at Beth Israel Deaconess Medical Center between 2001 and 2012 (13). We attended a training course on 'protecting human subjects' in order to apply for access to the database.

The establishment of the database was approved by the institutional review boards of the Massachusetts Institute of Technology (Cambridge, MA) and Beth Israel Deaconess Medical Center (Boston, MA). The author Jiang extracted the data for this study after passing the National Institutes of Health's online training course (certification number: 9322422).

Population selection criteria

In total, 58976 intensive care unit (ICU) patients were recorded in the MIMICIII database, of these, we included in our study patients who were older than 16 at the time of their initial admission and who underwent invasive ventilation for a minimum of 48 consecutive hours. Patients were excluded if they met the criteria: had incomplete ventilatory variables to calculate MP and norMP, had > 1% missing data, and/or were died or extubated during the first 48 h. We used only data from the patient's initial ICU admission or initial hospitalization.

Data extraction

The structured query language (SQL) was used to extract data from the database, and included tidal volume (V_t), positive end–expiratory pressure (PEEP), peak pressure (PIP), RR, and the inspired fraction of oxygen (FiO_2). The following equation was used to calculate mechanical power (7, 11):

$MP(J/min) = 0.098 \times V_t \times RR \times (PIP - \Delta P \times 0.5)$, where the driving pressure (ΔP) = PIP – PEEP (14).

$norMP (\times 10^{-3} J/min/kg) = MP/PBW$ (15), where PBW was the predicted body weight calculated by using the equation as used in previous studies of ventilation (16):

$PBW = 50.0 + 0.91(\text{height [cm]} - 152.4)$ in males,

$PBW = 45.5 + 0.91(\text{height [cm]} - 152.4)$ in females.

Due to the fact that the patients provided multiple measurements, the mean of the highest and lowest values obtained during the second 24 hours was used. The norMP in the second day of ventilation was chosen because during the first 24 hours usually mechanical ventilation is subjected to several changes and may result in more noise.

The following demographic data (using first 24 hours of admission data) were collected: age, gender, ethnicity (white, black, or other), height, weight, comorbidities, and disease severity scores (Acute Physiology and Chronic Health Evaluation [APACHE] III) (17, 18). Vital signs and laboratory measurements were captured as lowest and the highest values in the first day of ventilation.

Clinical outcome

To gather information about ICU patients' status, the follow-up followed from ICU admission and ended at death. The major endpoint was ICU mortality, the secondary endpoints included 30-day, 90-day mortality; ICU length of stay (ICU_LOS), the number of ventilator-free days at day 28 (VFD_28, specified as the days from effective weaning to day 28; patients who died prior to weaning were considered to have no ventilator-free days).

Statistical analyses

Continuous variables are presented in the tables as the median with interquartile ranges. The required Wilcoxon test, or Kruskal– Wallis test, was applied. Chisquared test or Fisher's exact test was used for categorical variables, which are presented as a percentage. Patients were categorized into groups according to ICU mortality.

The median and interquartile range of norMP was used to classify all patients. For all outcomes, univariate and multivariate regression were used to account for potential confounding variables. Additionally, subgroup analyses were conducted to determine the relationship between norMP and the primary outcome according to the V_t and PIP levels.

Statistical significance was described as a two-sided $p < 0.05$. SPSS software was used for all statistical analysis (SPSS-22.0; IBM Corp., Armonk, NY, USA).

Results

Finally, 1301 patients fulfilled the requirements for the study. Table 1 summarizes the demographic characteristics of survivors and non-survivors. norMP was significantly lower for survivors (265.9 (200.7 – 345.6)) than non-survivors (282.8 (218.2 – 381.6)) ($p = 0.004$), but the MP has no significantly difference between survivors and non-survivors.

norMP had a median of 269.9 and an interquartile range of 203.5–355.9, respectively. All patients were divided into quartile according to their norMP as follows: less than 203.5, quartile 1, ($n = 326$); from 203.5 to 269.8, quartile 2 ($n = 326$); from 269.9 to 355.8, quartile 3, ($n = 324$); greater than 355.8, quartile 4 ($n = 325$). The clinical outcomes of patients in various groups were summarized in Table 2. ICU mortality, ICU_LOS, VFD_28 showed statistically significant difference ($p < 0.05$). However, there was no evidence that 30-day mortality and 90-day mortality between the groups was statistically different ($p > 0.05$).

Fig. 1 illustrates the results of the univariate and multivariate analysis of the primary outcome. Crude outcome shows that High norMP was associated with increased ICU mortality (OR= 1.16, 95% CI 1.04–1.29, $p < 0.001$). In addition, norMP in the second 24h still had a strong correlation with increased ICU mortality even after adjustment for covariates (OR= 1.20, 95% CI 1.07–1.35, $p = 0.001$; OR= 1.27, 95% CI 1.12–1.45, $p = 0.009$, respectively).

Fig. 2 illustrates the results of the multivariate analysis of the secondary outcome. norMP in the second 24h of ventilation was also associated with ICU length of stay and the number of ventilator-free days (Fig. 2b). But there was no association between norMP and 30-day mortality or 90-day mortality (Fig. 2a).

According to the concepts of protective ventilation (19), we define low Vt as Vt less than 8ml/PBW and low PIP as PIP less than 30cmH₂O. In the subgroup analysis (Fig. 3), high norMP was associated with ICU mortality whether low or high Vt (OR= 1.22, 95% CI 1.01–1.47, $p = 0.040$; OR= 1.28, 95% CI 1.06–1.54, $p = 0.011$, respectively). But high norMP was associated with ICU mortality only in low PIP (OR= 1.18, 95% CI 1.01–1.37, $p = 0.034$).

Discussion

The essential findings of this research can be summarized in the following points: (1) norMP in the second 24h of ventilation is independently correlated with increased ICU mortality of critically ill patients who receive invasive ventilation for more than 48 h; (2) increased norMP is independently correlated with longer stay in ICU and a lower number of ventilator-free days and alive at day 28; (3) high norMP was associated with ICU mortality whether low or high Vt, but high norMP was associated with ICU mortality only in low PIP.

During invasive ventilation, a lung protection ventilation strategy that provides adequate gas exchange while minimizing VILI should be used (20, 21). VILI is thought to be mainly associated with excessive pressure, excessive volume and atelectasis (8, 22). Therefore, mechanical ventilation strategies to reduce VILI were focus on reducing every potential determinant, such as respiratory rate, tidal volume, and PEEP (16, 23–27). And while individual ventilator parameters have been extensively studied in previous research, few studies have considered these factors comprehensively. MP is a composite of these variables and therefore could have a better predictive value for clinical outcomes such as mortality (11). As described above, norMP is superior to MP cause the effects of MP may be proportional to their involvement in total lung function size. In fact, for similar mechanical power values, according to different ventilated lung surfaces, different energy would be delivered. Previous study (15) normalizing the mechanical power to the predicted body weight as a proxy for lung size, demonstrated that the norMP had the highest AUROC among all ventilator parameters and better predicted in-hospital mortality. Similar to previous study, the results of this analysis provided evidence that norMP may be a predictor of poor outcomes for ICU patients undergoing invasive ventilation.

Since the two important factors of ventilator parameters are tidal volume and airway pressure, we evaluated the effect of norMP on the prognosis of different tidal volumes and airway pressure levels. Recently, the use of low V_t is supported by protection ventilation. And in line with our hypothesis, we discovered that high MP was correlated with ICU mortality even when V_t was low. It suggested that norMP adds additional information beyond volume. In addition, our research also demonstrated that high norMP was associated with ICU mortality only in low PIP. We suppose that among excessive pressure, excessive volume, and atelectasis, excessive pressure has the greatest impact on lung damage. The mechanical damage and inflammatory damage caused by excessive pressure can no longer be remedied by other protective measures. This suggests that in protective ventilation, it is important to maintain a low airway pressure while maintaining a low norMP.

These findings suggest that using norMP to set up ventilator may be attractive because it combines the effects of different ventilator parameters. If modifying a single parameter has little effect on the amount of energy transmitted to the lung tissue, it may not always protect the lungs (28). For example, according to the concepts of protective ventilation, a decrease in volume necessitates an increase in respiratory rate to offset the loss of minute volume. Higher respiratory rate may lead to higher norMP, so volume reduction may not result in profit according to our current study and previous studies (29, 30). It will be useful to protect lung when the ventilators directly display norMP applied to the respiratory system in the future, so that the caregiver can titrate ventilation to reduce the amount of energy supplied to the lung tissue.

Certainly, the current analysis has some limitations. Firstly, in order to determine patients with more serious illness and patients with ample exposure time, we only selected patients who underwent invasive ventilation for at least 48 hours. The current findings, however, cannot be generalized to patients who were extubated or died within the first 48 hours. Secondly, norMP was calculated only one time, not during the ICU stay. Therefore, it did not accurately represent the temporal changes in the norMP

administered to the patient. Thirdly, since the datasets used in this study were from publicly-available data, it is questionable if the airway pressure was collected under consistent standard conditions, such as without spontaneous breathing efforts. Finally, it is difficult to quantify functional lung size. In present study, we indirectly described the size of functional lungs through the PBW. However, other conditions lead to decrease functional lungs size, such as ARDS, was not considered.

Conclusions

High norMP is independently correlated with increased ICU mortality and many other clinical outcomes in critically ill patients who undergo invasive ventilation for at least 48 hours.

Abbreviations

BMI: body mass index; PBW: predicted body weight; CHF: congestive heart failure; bpm: beats per minute; SpO₂: pulse oximetry; MAP: mean arterial blood pressure; FiO₂: inspired fraction of oxygen; PEEP: positive end-expiratory pressure; PIP: peak inspiratory pressure; RR: respiratory rate; V_t: tidal volume; MP: mechanical power; norMP: mechanical power normalized to predicted body weight; ICU: intensive care unit; LOS: length of stay; VFD₂₈: Ventilator-free days at day 28.

Declarations

Ethics approval and consent to participate

The datasets used for the current study come from [[MIMIC-III ver.1.4](#)] repository. To access the database, we completed the National Institutes of Health's web-based course Protecting Human Research Participants.

Consent for publication

Not applicable

Availability of data and materials

The datasets of the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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No funding was obtained for this study.

Authors' contributions

Yanhong Zhu and Xiaofeng Jiang designed this study. Shuai Zhen extracted the Data. Yanhong Zhu analyzed the data and drafted the manuscript. Xiaofeng Jiang and Wenyong Peng critically revised the manuscript. All authors read and approved the final manuscript.

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Tables

Table1. Comparisons of demographics between survivors and non-survivors

Baseline characteristics	Survivors (<i>n</i> = 936)	Non-survivors (<i>n</i> = 365)	<i>p</i> value
Age, years	61.3 (48.5 – 74.0)	68.3 (56.0 – 79.0)	< 0.001
Male gender	508 / 936 (54.3)	191 / 365 (52.3)	0.527
Weight, kg	82.7 (69.0 – 99.0)	78.0 (65.0 – 92.0)	< 0.001
Height, cm	170 (163 – 178)	168 (160 – 176)	0.015
BMI, kg/m ²	28.4 (24.3 – 33.9)	26.8 (23.2 – 32.1)	0.001
PBW, kg	63.9 (54.7 – 73.1)	61.7 (52.5 – 71.8)	0.026
Admission type			0.001
Selective	82 / 936 (8.8)	11 / 365 (3.0)	
Emergency	836 / 936 (89.3)	349 / 365 (95.6)	
Urgent	18 / 936 (1.9)	5 / 365 (1.4)	
Ethnicity			0.005
White	647 / 936 (69.1)	223 / 365 (61.1)	
Black	85 / 936 (9.1)	31 / 365 (8.5)	
Other	204 / 936 (21.8)	111 / 365 (30.4)	
Comorbidities			
CHF	170 / 936 (18.1)	78 / 365 (21.4)	0.186
Cardiac arrhythmias	212 / 936 (22.6)	110 / 365 (30.1)	0.005
Valvular disease	53 / 936 (5.7)	30 / 365 (8.2)	0.090
Hypertension	143 / 936 (15.3)	56 / 365 (15.3)	0.977
Diabetes	260 / 936 (27.8)	119 / 365 (32.6)	0.085
Neurological condition	139 / 936 (14.9)	46 / 365 (12.6)	0.297
Chronic pulmonary condition	215 / 936 (23.0)	88 / 365 (24.1)	0.662
Renal failure	160 / 936 (17.1)	75 / 365 (20.5)	0.146
Liver condition	95 / 936 (10.1)	50 / 365 (13.7)	0.068
Severity of illness			
APACHE III	51 (38 – 69)	65 (50 – 85)	< 0.001
Vital signs in the beginning of ventilation			

Heart rate, bpm	89 (77 – 103)	89 (75 – 103)	0.496
MAP, mmHg	82 (76 – 91)	83 (76 – 91)	0.465
SpO ₂ , %	98 (96 – 99)	98 (96 – 99)	0.016
Temperature, °C	37.0 (36.4 – 37.5)	36.7 (36.1 – 37.3)	< 0.001
Laboratory in the beginning of ventilation			
pH	7.36 (7.28 – 7.42)	7.34 (7.26 – 7.41)	0.002
PaO ₂ / FiO ₂ , mmHg	238 (152 – 351)	213 (132 – 334)	0.047
PaCO ₂ , mmHg	41 (37 – 49)	40 (34 – 49)	0.059
Second day of ventilation parameters			
Tidal volume, ml/kg PBW	7.8 (6.8 – 8.8)	7.9 (6.8 – 8.8)	0.880
PEEP, cmH ₂ O	7 (5 – 10)	7 (5 – 10)	0.016
PIP, cmH ₂ O	24 (20 – 28)	26 (22 – 30)	< 0.001
Respiratory rate, bpm	20 (17 – 23)	21 (18 – 24)	< 0.001
Minute ventilation, L/min	9.2 (7.7 – 11.1)	9.6 (8.6 – 11.7)	0.009
MP, J/min	16.6 (12.6 – 22.1)	17.2 (13.1 – 23.3)	0.082
norMP, 10 ⁻³ J/min/kg	265.9 (200.7 – 345.6)	282.8 (218.2 – 381.6)	0.004

Data are median (interquartile range) or No / Total (%)

BMI: body mass index; PBW: predicted body weight; CHF: congestive heart failure; bpm: beats per minute; SpO₂: pulse oximetry; MAP: mean arterial blood pressure; FiO₂: inspired fraction of oxygen; PEEP: positive end-expiratory pressure; PIP: peak inspiratory pressure; MP: mechanical power; norMP: mechanical power normalized to predicted body weight

Table 2. Clinical outcomes of subjects by the quartile of the norMP

	norMP				<i>p</i> value
	quartile 1 < 203.5	quartile 2 203.5–269.8	quartile 3 269.9–355.8	quartile 4 ≥355.9	
ICU mortality	79 (24.2)	87 (26.7)	90 (27.8)	109 (33.5)	0.05
30-day mortality	102 (31.3)	107 (32.8)	109 (33.6)	116 (35.7)	0.687
90-day mortality	118 (36.2)	127 (39.0)	125 (38.6)	132 (40.6)	0.713
ICU_LOS, day	7.9 (5.2 – 12.1)	8.2 (5.7 – 13.1)	9.1 (5.6 – 14.1)	9.3 (6.0 – 15.6)	0.002
VFD_28, day	21.3 (0 – 24.5)	20.2 (0 – 24.1)	20 (0 – 23.8)	16.3 (0 – 22.5)	< 0.001

Data are median (interquartile range) or No / Total (%)

norMP: mechanical power normalized to predicted body weight; ICU: intensive care unit; LOS: length of stay; VFD_28: Ventilator-free days at day 28

Figures

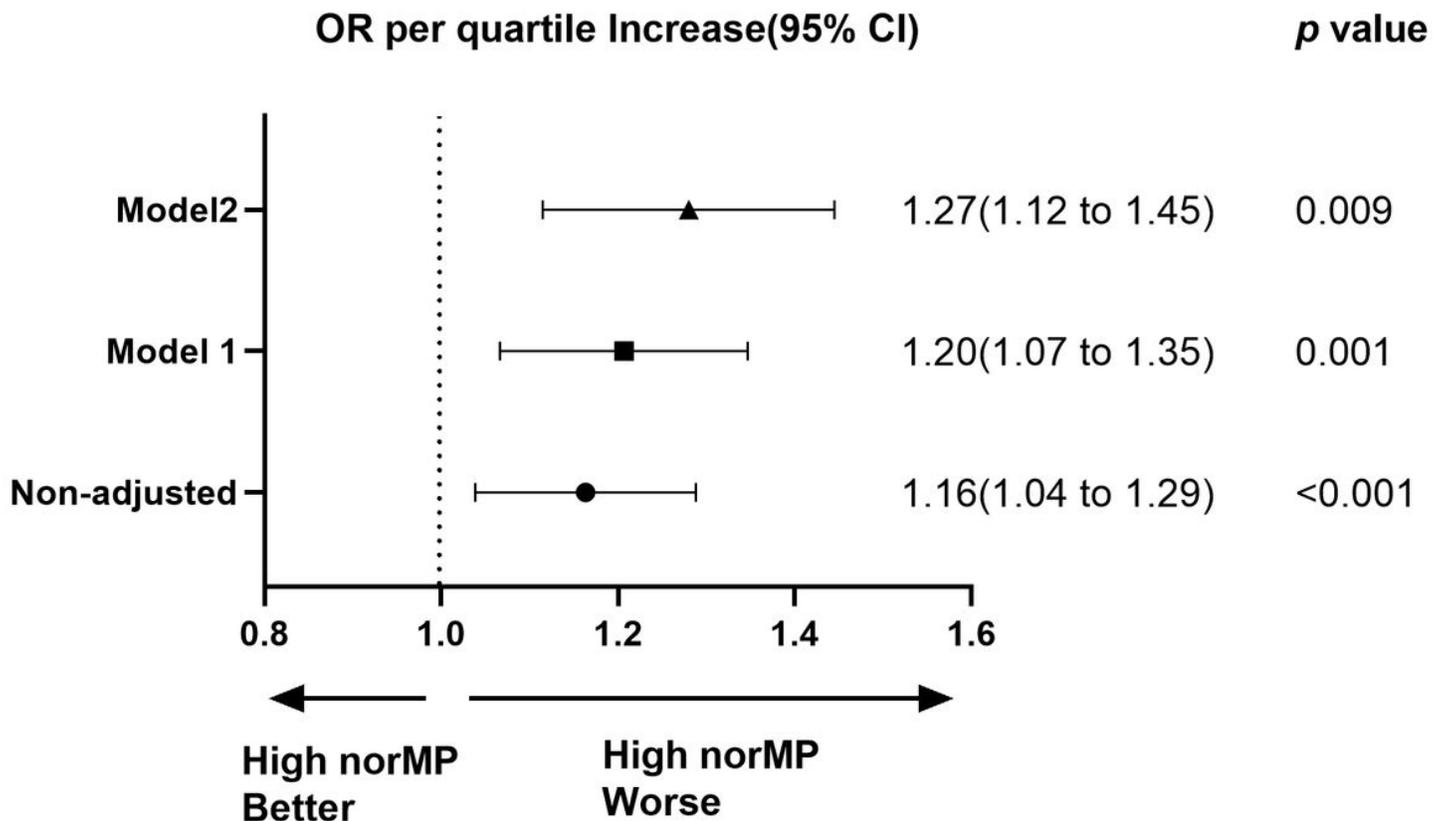


Figure 1

norMP in the second 24h of ventilation and ICU mortality. Model 1 was adjusted for the confounders age, sex and ethnicity. Model 2 was adjusted for the confounders, including age, sex, ethnicity, BMI, admission type, comorbidities, APACHE, heart rate, MAP, SpO2, temperature, pH, PaO2 / FiO2, PaCO2. The odds ratio represents the odds of death per quartile increase in norMP. norMP: mechanical power normalized to predicted body weight.

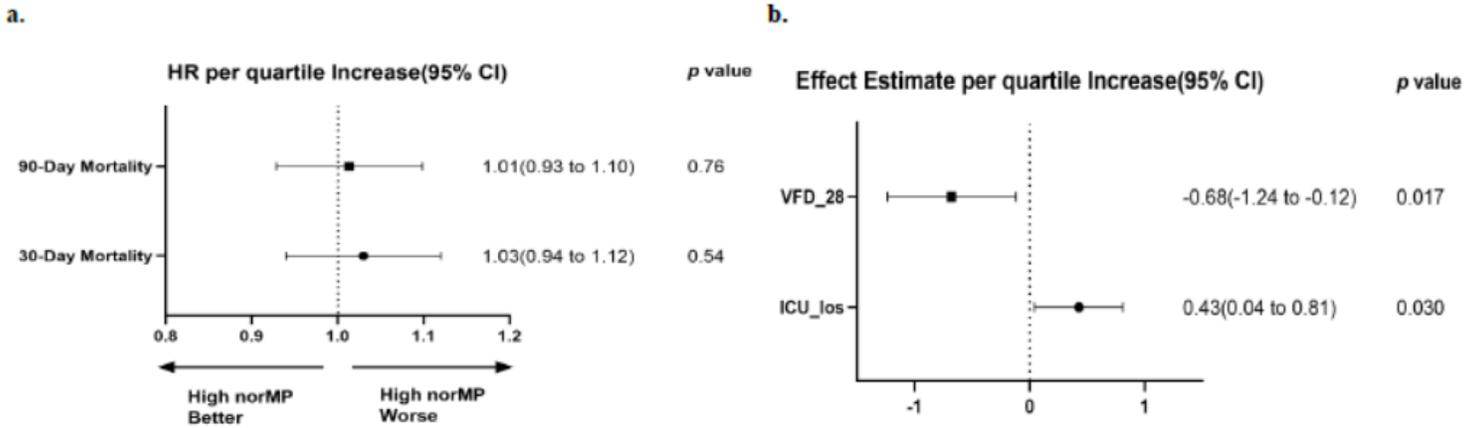


Figure 2

norMP in the second day of ventilation and secondary outcomes. a Odds ratio represents the odds of death per quartile increase in norMP. b Effect estimates and 95% confidence interval from the multivariable linear regression for VFD_28 and ICU_los. Effect estimate refers to the change in the outcome variable per quartile increase in norMP. norMP: mechanical power normalized to predicted body weight; VFD_28: Ventilator-free days at day 28; ICU: intensive care unit; LOS: length of stay.

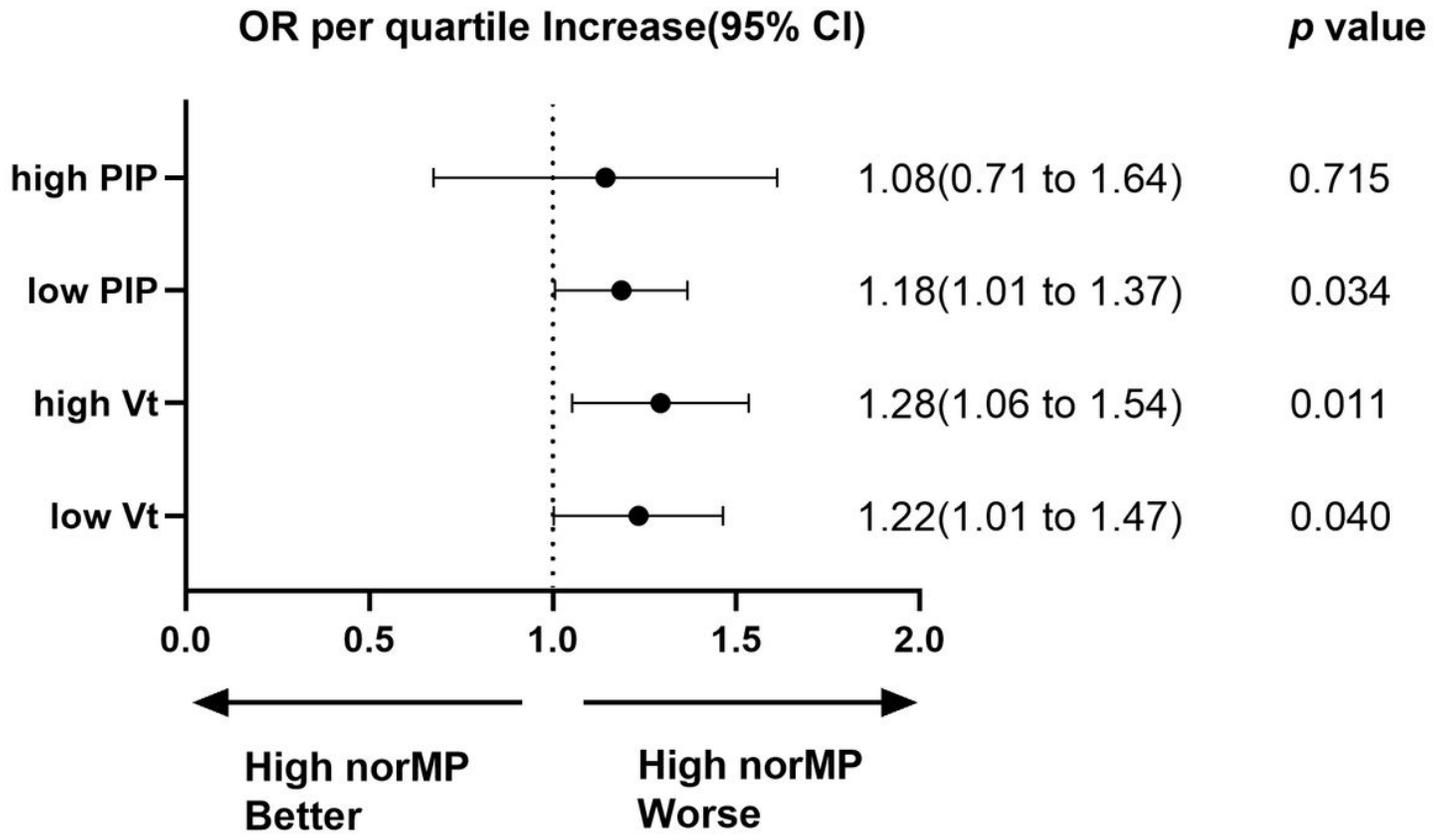


Figure 3

Subgroup analysis of the association between norMP and ICU mortality according to different tidal volumes and airway pressure levels. The odds ratio represents the odds of death per quartile increase in norMP. norMP: mechanical power normalized to predicted body weight; Vt: tidal volume; PIP: peak inspiratory pressure.