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ORIGINAL ARTICLE

Intra-operative ventilator mechanical power as a predictor of postoperative pulmonary complications in surgical patients

A secondary analysis of a randomised clinical trial

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BACKGROUND Studies in critically ill patients suggest a relationship between mechanical power (an index of the energy delivered by the ventilator, which includes driving pressure, respiratory rate, tidal volume and inspiratory pressure) and complications.

OBJECTIVE We aimed to assess the association between intra-operative mechanical power and postoperative pulmonary complications (PPCs).

DESIGN Post hoc analysis of a large randomised clinical trial.

SETTING University-affiliated academic tertiary hospital in Melbourne, Australia, from February 2015 to February 2019.

PATIENTS Adult patients undergoing major noncardiothoracic, nonintracranial surgery.

INTERVENTION Dynamic mechanical power was calculated using the power equation adjusted by the respiratory system compliance (C_{RS}). Multivariable models were used to assess the independent association between mechanical power and outcomes.

MAIN OUTCOME MEASURES The primary outcome was the incidence of PPCs within the first seven postoperative days. The secondary outcome was the incidence of acute respiratory failure.

RESULTS We studied 1156 patients (median age [IQR]: 64 [55 to 72] years, 59.5% men). Median mechanical power adjusted by C_{RS} was 0.32 [0.22 to 0.51] ($J\,min^{-1}$)/($ml\,cmH_2O^{-1}$). A higher mechanical power was also independently associated with increased risk of PPCs [odds ratio (OR 1.34, 95% CI, 1.17 to 1.52); $P < 0.001$) and acute respiratory failure (OR 1.40, 95% CI, 1.21 to 1.61; $P < 0.001$).

CONCLUSION In patients receiving ventilation during major noncardiothoracic, nonintracranial surgery, exposure to a higher mechanical power was independently associated with an increased risk of PPCs and acute respiratory failure.

TRIAL REGISTRATION Australia and New Zealand Clinical Trials Registry no: 12614000790640.

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Introduction

Approximately 300 million surgical procedures are performed globally each year.[1] Postoperative pulmonary complications (PPCs) are estimated to occur in more than 30% of patients after major surgery and are associated with increased morbidity, mortality and healthcare costs.[2–5] Therefore, it is desirable to identify potentially

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modifiable factors that may be associated with an increased risk of PPCs. Previous studies in critically ill patients with acute respiratory distress syndrome (ARDS) have reported an association between high driving pressure [ΔP, the difference between the plateau pressure (P_{plat}) and the level of positive end expiratory pressure (PEEP)] and adverse outcome.[6–8] Furthermore, previous studies have also suggested an association between high ΔP and adverse outcomes in surgical patients.[9,10]

Recently, it has been proposed that the extent of the ventilator-induced lung injury (VILI) may relate to the amount of energy transferred from the ventilator to the lungs, a concept referred to as 'mechanical power'.[11–15] Measurement of mechanical power is determined by a combination of factors including tidal volume (V_T), inspiratory pressure, respiratory rate and inspiratory flow rate, all of which determine the amount of energy generated during mechanical ventilation.[12] The amount of energy per unit of time, expressed in joules per minute ($J\,min^{-1}$), is then referred to as the 'mechanical power'. Previous studies have demonstrated that mechanical power is associated with increased mortality in ICU patients with and without ARDS.[15–18] To date, no studies have assessed the association of mechanical power with PPCs in patients undergoing major surgery.

Recently, an RCT evaluating the impact of V_T on the incidence of PPCs was published and showed no impact of lower or higher V_T on this outcome.[19] However, as pointed out in an accompanying letter,[20] the study failed to consider the association between mechanical power and the risk of PPCs. Accordingly, we performed a post hoc analysis of a large randomised clinical trial to assess the association of mechanical power with clinical outcomes in patients receiving mechanical ventilation during major noncardiothoracic, nonintracranial surgery. The aim of this study was to assess the association between mechanical power and the development of PPCs within the first seven postoperative days in adult patients receiving mechanical ventilation during major surgery. We hypothesised that mechanical power would be associated with increased risk of PPCs.

Methods

Design

This was a post hoc analysis of an investigator-initiated, assessor-blinded, single-centre, randomised clinical trial conducted in a tertiary hospital in Melbourne, Australia. The protocol and statistical analysis plan,[21] and the primary trial have been published.[19] The trial was registered in ANZCTR: ACTRN12614000790640.

Ethics

Ethical approval for this study was granted by the Austin Hospital Human Research Ethics Committee, Austin Hospital, Heidelberg, Victoria, Australia on the 2 July, 2014 (HREC/14/Austin/260).

Patients

Patients were included in the primary trial if they were older than 40 years of age, scheduled to have major noncardiothoracic, nonintracranial surgery with an expected duration more than 2 h and invasive arterial pressure monitoring was planned to be part of their routine care. Patients were excluded if they were pregnant, scheduled to have cardiac, thoracic or intracranial neurological surgery, or if they had been previously enrolled in the trial. For the present study, we further excluded patients without the data needed for the calculation of mechanical power, and those with missing data with respect to PPCs.

Intervention

All patients received volume-controlled ventilation with an applied PEEP of 5 cmH_2O. Immediately after randomisation, patients were assigned to receive lung ventilation with either a low V_T ($6\,ml\,kg^{-1}$ predicted body weight, PBW) or a conventional V_T ($10\,ml\,kg^{-1}$ PBW). Predicted body weight was calculated as $50 + 0.91 \times$ [height (cm) – 152.4] for male individuals and $45.5 + 0.91 \times$ (height (cm) – 152.4) for female individuals. The V_T and PEEP were fixed and maintained for the duration of the surgical procedure. All other aspects of intra-operative care, including the inspired fraction of oxygen (FiO_2), respiratory rate, anaesthesia technique (including type of sedative used), fluid management, use of vasoactive drugs, analgesia plan, use of prophylactic antibiotics and antiemetics agents, were administered at the discretion of the treating anaesthesiologist. Neuromuscular blocking agents were used in all patients according to local protocol.

Data collection and definitions

A standardised case report form was used for data collection. The research staff collected all data directly from the clinical chart. All patients were assessed daily by the trial research team for the first seven postoperative days or until hospital discharge (whichever came first). Research staff, blinded to the intra-operative intervention, collected information regarding the clinical outcomes. After the first 7 days (if the patient was still in hospital), additional data were retrieved from the electronic medical record. Intra-operatively, all ventilatory data and vital signs were collected prospectively as the lowest and/or highest values during the procedure. In this analysis, the highest peak inspiratory pressure (P_{peak}) and highest respiratory rate and the fixed protocolised PEEP and V_T in the intra-operative period were considered for the calculations.

Dynamic ΔP and mechanical power

All patients were ventilated with a volume-controlled ventilation mode and did not have spontaneous breathing during assessment. Dynamic mechanical power was calculated using the power equation: mechanical power

$(J\,min^{-1}) = 0.098 \times V_T \times$ respiratory rate $\times$ $[(P_{peak} - (0.5 \times \Delta P)]$.[14] The dynamic mechanical power $(\Delta P) = P_{peak} -$ PEEP. Respiratory dynamic system compliance (C_{RS}) was calculated as V_T/dynamic ΔP. Mechanical power was normalised to the C_{RS} as a correlate of lung size, and calculated as mechanical power/C_{RS}.[14,18,22,23]

Outcomes

The list of outcomes for the original trial is described in the supplement (eMethods, http://links.lww.com/EJA/A625). The primary outcome for this post hoc analysis was the same as that of the original trial, the incidence of a composite of PPCs, defined as positive if any component developed within the first 7 days after surgery (see eMethods in the supplement for the definition). The secondary clinical outcome was acute respiratory failure within the first 7 days.

Statistical analysis

A convenience sample size was considered, and all patients included in the original trial were considered in this secondary analysis. Continuous variables were reported as median [interquartile range, IQR] and compared with Wilcoxon rank-sum tests, and categorical variables as number (%) and compared with Fisher exact tests. For better convergence of the models, the distribution of mechanical power was transformed to a mean of 2.50 with a standard deviation of 1. Transformation to a mean of 2.50 rather than 0 ensured that all values were positive. The following variables were considered for adjustment in all models described below: age, sex, baseline SpO_2, baseline bicarbonate, randomisation group and the Assess Respiratory Risk in Surgical Patients in Catalonia (ARISCAT) score. To facilitate comparisons between variables, all continuous variables were standardised to interpret their effect on outcome in standard deviation units.

First, a multivariable generalised linear model with binomial distribution considering mechanical power as the predictor of interest was constructed: this included the variables described above. For all models and outcomes, ORs with 95% CI were reported: the OR represents the increase in 1 standard deviation for continuous variables. To further assess the impact of mechanical power, eight quantiles of increasing mechanical power were created and the estimates for each quantile derived from the model above were plotted.

As a sensitivity analysis, the models described above were re-assessed considering the absolute mechanical power. In addition, the effect of mechanical power was assessed in each of the allocation groups (low or conventional V_T). The amount of missing data was low, and is reported in the supplement (eTable 1, http://links.lww.com/EJA/A625). All analyses were performed using R version 4.0.2 (R Foundation for Statistical Computing), and a two-sided P less than 0.05 was considered significant.

Results

Patients

From February 2015 to February 2019, we randomised 1236 patients. Of these, 627 were assigned to receive a low tidal volume and 609 patients to receive a conventional tidal volume ventilation. Thirty patients were excluded as either the surgery did not proceed, or the anaesthesiologist did not use the trial protocol ventilation, or there was no arterial line. The data from the remaining 1206 patients were used in the primary analysis. After further exclusions, 1156 patients were eligible for the final analysis, with 583 in the low tidal volume and 566 in the conventional tidal volume group (Fig. 1).

Baseline characteristics and clinical outcomes of the patients are shown in Table 1. The median [IQR] age was 64 [55 to 72] years, 59.5% were men and the median [IQR] ARISCAT score was 26 [19 to 38]. Within the cohort, 56.9% were classified as at moderate risk of PPCs, and 47.6% of the patients were classified as ASA 3. The most common comorbidities were hypertension and obesity, which were present in 52 and 37.4% of the patients, respectively. Abdominal surgery was the most common surgical procedure type (57.4%) of which 48% were laparoscopic. PPCs within the first 7 days occurred in 39.6% of the patients, and acute respiratory failure in 18.2%. Hospital mortality rate was 1%. All characteristics and clinical outcomes were well balanced between the randomisation groups (Table 1).

Mechanical ventilation

Ventilatory and surgical variables are shown in Table 2. Median P_{peak} for all the patients was 23 [20 to 28] cmH_2O and it was lower in the low tidal volume group: 22 [18 to 26] vs. 24 [21 to 29] cmH_2O, P less than 0.001. Median ΔP was 18 [15 to 23] cmH_2O and it was lower in the low tidal volume group: 17 [13 to 21] vs. 19 [16 to 24] cmH_2O, P less than 0.001. Median mechanical power adjusted by compliance was 0.32 [0.22 to 0.51] $(J\,min^{-1})/(ml\,cmH_2O^{-1})$ and was higher in the low tidal volume group: 0.35 [0.23 to 0.57] vs. 0.30 [0.20 to 0.47] $(J\,min^{-1})/(ml\,cmH_2O^{-1}$, $P = 0.001$).

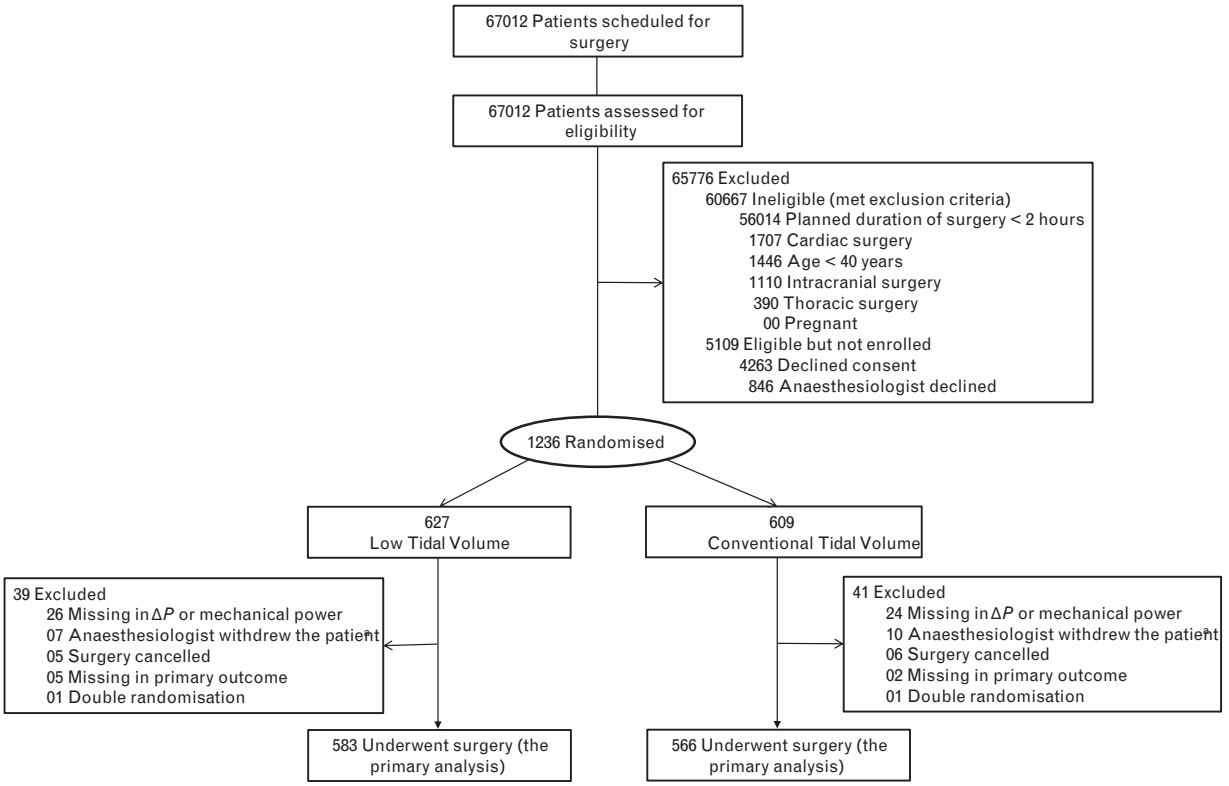
Association of mechanical power and outcomes

On the univariable analysis, higher mechanical power was associated with increased risk of PPCs and acute respiratory failure (Supplement, eTable 2, http://links.lww.com/EJA/A625). On multivariable analysis, mechanical power was significantly associated with increased risk of PPCs [OR, 1.34 (95% CI, 1.17 to 1.52), $P < 0.001$] and acute respiratory failure [OR, 1.40 (95% CI, 1.21 to 1.61), $P < 0.001$] after adjusting for other confounders (Table 3 and Fig. 2).

Sensitivity analysis

In all models, higher absolute mechanical power was associated with increased risk of PPCs and acute

Fig. 1 Flow chart.



respiratory failure (Supplement, eTable 3 and eFigure 1, http://links.lww.com/EJA/A625). In addition, the effect found was persistent in both allocation groups (Supplement, eFigures 2 and 3, http://links.lww.com/EJA/A625).

Discussion

Key findings

In this post hoc analysis of a large randomised controlled trial of adult patients receiving mechanical ventilation during noncardiothoracic, nonintracranial major surgery, exposure to higher mechanical ventilation intensities, as measured by mechanical power, was associated with an increased risk of PPCs and acute respiratory failure in the first seven postoperative days. The effect was consistent after adjustment for several important factors known to be associated with clinical outcomes in this population and stronger than that of many other key variables.

Relationship with previous studies

In the original trial, there was no impact of low V_T on clinical outcomes.[19] This was despite significantly lower peak pressure and significantly higher respiratory rates in the low tidal volume group. Thus, logically, the impact of other intra-operative ventilatory variables should be assessed. Previous studies in critically ill patients have suggested that whilst lower inspiratory pressures are beneficial, higher respiratory rates may be harmful.[20] This led to an interest in how such opposing factors may interact. Mechanical power, a measure that aims to integrate static and dynamic parameters of ventilation, could be a more important variable in the relationship between ventilation and lung injury.[20] This is based on the rationale that VILI is not only a function of static parameters, such as strain from V_T or stress from inspiratory pressures but also on the complex interplay between static and dynamic variables, including the rate of lung deformation (strain rate) and the cycling frequency, or respiratory rate.[14,20] Mechanical power has been suggested as an index of the overall energy applied to the lungs, which encompasses the impact of both driving pressure and respiratory rate.

Several previous studies have identified an association between a higher ΔP and mechanical power with adverse outcomes in critically ill patients receiving mechanical ventilation in the ICU. An individual patient data meta-analysis published in 2015 indicated the potential association of a higher ΔP with increased mortality in patients with ARDS.[6] More recently, a study performed in patients with acute respiratory failure showed that cumulative exposure to higher intensities of mechanical ventilation, assessed through daily measurements of ΔP and mechanical power, was associated with harm, even for short durations of exposure.[24] In patients with ARDS,

Table 1 Baseline characteristics and clinical outcomes of the included patients

	Overall ($n = 1149$)	Low Tidal Volume ($n = 583$)	Conventional Tidal Volume ($n = 566$)	*P* value
Age (years)	64.0 [55.0 to 72.0]	65.0 [54.0 to 72.0]	64.0 [55.0 to 73.0]	0.572
Male gender to [no. (%)]	684 (59.5)	352 (60.4)	332 (58.7)	0.589
Weight (kg)	81.0 [69.4 to 95.0]	80.2 [68.8 to 95.0]	81.0 [70.7 to 94.0]	0.604
BMI (kg m^{-2})	28.1 [24.8 to 32.2]	28.0 [24.5 to 32.5]	28.1 [25.1 to 31.9]	0.629
ARISCAT score[a]	26.0 [19.0 to 37.8]	26.0 [19.0 to 37.2]	26.0 [19.0 to 37.8]	0.313
Low risk	373 (35.8)	183 (34.1)	190 (37.5)	
Moderate risk	593 (56.9)	317 (59.1)	276 (54.5)	0.310
High risk	76 (7.3)	36 (6.7)	40 (7.9)	
ASA physical status				0.621
1, healthy	112 (9.9)	62 (10.8)	50 (8.9)	
2, mild systemic disease	424 (37.4)	214 (37.2)	210 (37.6)	
3, severe systemic disease	540 (47.6)	267 (46.4)	273 (48.8)	
4, Constant threat to life	58 (5.1)	32 (5.6)	26 (4.7)	
Baseline SpO_2 (%)	97.0 [96.0 to 98.0]	97.0 [96.0 to 98.0]	97.0 [96.0 to 98.0]	0.721
Baseline HCO_3 (mmol l^{-1})	26.0 [24.0 to 28.0]	26.0 [24.0 to 27.5]	26.0 [24.0 to 28.0]	0.666
Baseline haemoglobin (g dl^{-1})	13.8 [12.5 to 14.9]	13.8 [12.7 to 14.9]	13.8 [12.4 to 14.9]	0.196
Baseline creatinine (mg dl^{-1})	0.9 [0.7 to 1.1]	0.9 [0.7 to 1.1]	0.9 [0.7 to 1.1]	0.693
Comorbidities				
Diabetes	232 (20.2)	112 (19.2)	120 (21.2)	0.419
Hypertension	597 (52.0)	285 (48.9)	312 (55.1)	0.039
Obesity[b]	415 (37.4)	216 (38.1)	199 (36.6)	0.620
Coronary artery disease	184 (16.0)	88 (15.1)	96 (17.0)	0.421
Chronic kidney disease[c]	121 (10.5)	55 (9.4)	66 (11.7)	0.249
Chronic liver disease	98 (8.5)	46 (7.9)	52 (9.2)	0.461
Smoking	193 (16.8)	90 (15.4)	103 (18.2)	0.236
Chronic obstructive pulmonary disease	119 (10.4)	57 (9.8)	62 (11.0)	0.561
Asthma	125 (10.9)	64 (11.0)	61 (10.8)	0.925
Interstitial lung disease	9 (0.8)	7 (1.2)	2 (0.4)	0.178
Bronchiectasis	2 (0.2)	1 (0.2)	1 (0.2)	0.999
Obstructive sleep apnoea	119 (10.4)	57 (9.8)	62 (11.0)	0.561
Recent respiratory infection	15 (1.3)	8 (1.4)	7 (1.2)	0.999
Type of surgery				0.961
Abdominal	660 (57.4)	335 (57.5)	325 (57.4)	
Laparoscopic	317/660 (48.0)	153/335 (45.7)	164/325 (50.5)	0.243
General	7 (0.6)	5 (0.9)	2 (0.4)	
Ear, nose and throat	29 (2.5)	16 (2.7)	13 (2.3)	
Orthopaedic	84 (7.3)	42 (7.2)	42 (7.4)	
Plastic	63 (5.5)	29 (5.0)	34 (6.0)	
Spinal	228 (19.8)	116 (19.9)	112 (19.8)	
Vascular	50 (4.4)	25 (4.3)	25 (4.4)	
Others	28 (2.4)	15 (2.6)	13 (2.3)	
Clinical outcomes				
Postoperative pulmonary complications	455 (39.6)	227 (38.9)	228 (40.3)	0.673
Acute respiratory failure	209 (18.2)	103 (17.7)	106 (18.7)	0.647
Hospital length of stay (days)	5.0 [3.0 to 9.0]	6.0 [3.0 to 10.0]	5.0 [3.0 to 8.0]	0.069
Hospital mortality	12 (1.0)	6 (1.0)	6 (1.1)	0.999

Data are median [IQR] or *n* (%). ARISCAT, Assess Respiratory Risk in Surgical Patients in Catalonia; COPD, chronic obstructive pulmonary disease; HCO_3, bicarbonate; SpO_2, oxygen saturation from pulse oximetry. [a] Score range is from 0 to 123; higher scores indicate a higher risk of postoperative pulmonary complications. Patients with scores of 26 or greater are considered at intermediate risk; those with a score greater than 44 are considered at high risk. [b] Defined as BMI greater than 30 kg m^{-2}. [c] Defined as KDIGO CKD stage 2 or greater.

higher ΔP was also associated with increased hospital and 3-year mortality.[17]

In patients receiving mechanical ventilation during major surgery, a higher ΔP was found to be significantly associated with the development of PPCs.[9] In addition, ΔP was a relevant potential mediator on the effect of ventilation on outcomes in these patients.[9] In a different cohort from a different country, a higher ΔP was again found to be significantly associated with increased risk of PPCs in surgical patients.[10] However, no study assessed the impact of mechanical power on clinical outcomes of surgical patients.

Mechanical power is a potentially unifying variable incorporating most of the factors associated with development of VILI, and a higher mechanical power is associated with worse outcomes in patients receiving mechanical ventilation.[14] In critically ill patients, a higher mechanical power during ventilation was associated with higher risk of in-hospital mortality.[15] This was confirmed in a cohort of patients with acute respiratory failure,[24] and the adjustment of mechanical power by predicted body weight increased its predictive ability in patients with ARDS.[16] In the long-term, mechanical power was also associated with 3-year mortality, as was ΔP.[17] A recent study has shown that in ARDS patients, mechanical power captures

Table 2 Ventilatory and surgical variables in the included patients

	Overall (n=1149)	Low tidal volume (n=583)	Conventional tidal volume (n=566)	P value
Tidal volume				
Absolute (ml)	475.0 [385.0 to 630.0]	395.0 [340.0 to 450.0]	620.0 [526.2 to 700.0]	<0.001
Adjusted (ml kg^{-1}) PBW[a]	7.7 [6.0 to 10.0]	6.0 [6.0 to 6.1]	10.0 [9.9 to 10.0]	<0.001
PEEP (cmH_2O)	5 [5 to 5]	5 [5 to 5]	5 [5 to 5]	0.999
Peak pressure (cmH_2O)	23.0 [20.0 to 28.0]	22.0 [18.0 to 26.0]	24.0 [21.0 to 29.0]	<0.001
Driving pressure (cmH_2O)	18.0 [15.0 to 23.0]	17.0 [13.0 to 21.0]	19.0 [16.0 to 24.0]	<0.001
Respiratory rate (breaths min^{-1})	14.0 [10.0 to 16.0]	16.0 [14.0 to 18.0]	12.0 [10.0 to 12.0]	<0.001
Respiratory system compliance (ml cmH_2O^{-1})	27.1 [20.5 to 35.6]	22.7 [17.9 to 30.0]	31.2 [25.0 to 39.4]	<0.001
Mechanical power				
Absolute (J min^{-1})	9.0 [7.0 to 11.4]	8.0 [6.4 to 10.6]	9.7 [7.9 to 12.2]	<0.001
Adjusted by compliance (J min^{-1}/ml cmH_2O^{-1})	0.32 [0.22 to 0.51]	0.35 [0.23 to 0.57]	0.30 [0.20 to 0.47]	0.001
SpO_2 (%)	97.0 [96.0 to 98.0]	97.0 [95.0 to 98.0]	97.0 [96.0 to 98.0]	0.005
FiO_2 (%)	70.0 [50.0 to 95.0]	70.0 [50.0 to 94.8]	70.0 [50.0 to 95.0]	0.660
$etCO_2$ (%)	39.0 [36.0 to 42.0]	41.0 [38.0 to 44.0]	37.0 [34.0 to 40.0]	<0.001
Arterial blood gas after induction				
pH	7.40 [7.36 to 7.43]	7.37 [7.34 to 7.41]	7.42 [7.39 to 7.44]	<0.001
P_aO_2 (mmHg)	222.5 [167.0 to 286.0]	216.0 [161.2 to 285.0]	229.5 [172.2 to 286.0]	0.130
P_aCO_2 (mmHg)	41.0 [37.7 to 45.1]	43.8 [40.3 to 47.6]	39.0 [36.0 to 42.0]	<0.001
HCO_3 (mmol l^{-1})	25.0 [23.7 to 26.0]	25.0 [24.0 to 26.1]	24.6 [23.4 to 25.9]	<0.001
PaO_2/FiO_2 (mmHg)	418.3 [333.3 to 491.7]	412.2 [321.5 to 491.2]	429.2 [348.0 to 491.9]	0.065
Haemoglobin (g dl^{-1})	12.6 [11.3 to 13.7]	12.6 [11.4 to 13.7]	12.5 [11.3 to 13.7]	0.546
Base excess (mEq l^{-1})	0.3 [−1.0 to 1.8]	0.1 [−1.0 to 1.7]	0.5 [−0.7 to 2.0]	0.031
Lactate (mmol l^{-1})	1.1 [0.8 to 1.5]	1.1 [0.8 to 1.4]	1.1 [0.8 to 1.5]	0.013
Arterial blood gas prior to closure				
pH	7.37 [7.32 to 7.41]	7.34 [7.30 to 7.38]	7.39 [7.35 to 7.42]	<0.001
P_aO_2 (mmHg)	184.0 [145.0 to 232.0]	181.0 [143.0 to 227.0]	189.0 [146.0 to 241.0]	0.129
P_aCO_2 (mmHg)	42.0 [38.0 to 47.0]	44.8 [41.6 to 49.4]	39.3 [36.3 to 43.6]	<0.001
HCO_3 (mmol l^{-1})	24.0 [22.6 to 25.0]	24.0 [23.0 to 25.4]	23.8 [22.2 to 25.0]	<0.001
PaO_2/FiO_2 (mmHg)	397.4 [310.0 to 464.0]	385.5 [302.0 to 457.5]	402.2 [317.3 to 471.0]	0.044
Haemoglobin (g dl^{-1})	122.0 [108.0 to 133.0]	123.0 [108.0 to 133.0]	120.0 [108.0 to 133.0]	0.483
Base excess (mEq l^{-1})	−1.0 [−2.2 to 0.4]	−1.0 [−2.5 to 0.3]	−0.7 [−2.0 to 0.6]	0.020
Lactate (mmol l^{-1})	1.2 [0.9 to 1.8]	1.2 [0.9 to 1.7]	1.3 [0.9 to 1.8]	0.002
Duration of surgery (min)	188.0 [139.0 to 257.2]	190.0 [137.0 to 270.0]	185.0 [140.5 to 250.0]	0.236

Data are median [IQR] or n (%). ABG, arterial blood gas; $etCO_2$, end-tidal carbon dioxide; FiO_2, inspired fraction of oxygen; HCO_3, bicarbonate; P_aCO_2, partial pressure of carbon dioxide; P_aO_2, partial pressure of oxygen; PBW, predicted body weight; PEEP, positive end-expiratory pressure; SpO_2, oxygen saturation from pulse oximetry.
[a] PBW was calculated as 50 + 0.91 x [height (cm) − 152.4] for men and 45.5 + 0.91 x [height (cm) − 152.4] for women.

the applied energy in a way that ΔP does not.[18] However, the additional clinical importance of a more complex variable like mechanical power is still controversial. In addition, no previous studies have assessed the impact of mechanical power measured specifically in patients receiving mechanical ventilation during major surgery and its potential impact on postoperative outcomes.

Given the parameters included in the calculation of mechanical power, there are mainly two ways of reducing mechanical power in clinical practice: reducing driving pressure and/or respiratory rate. As yet, this balance has not been assessed in surgical patients but it appears from the evaluation of critically ill patients that the primary approach may be to reduce the driving pressure first.[24]

Study implications

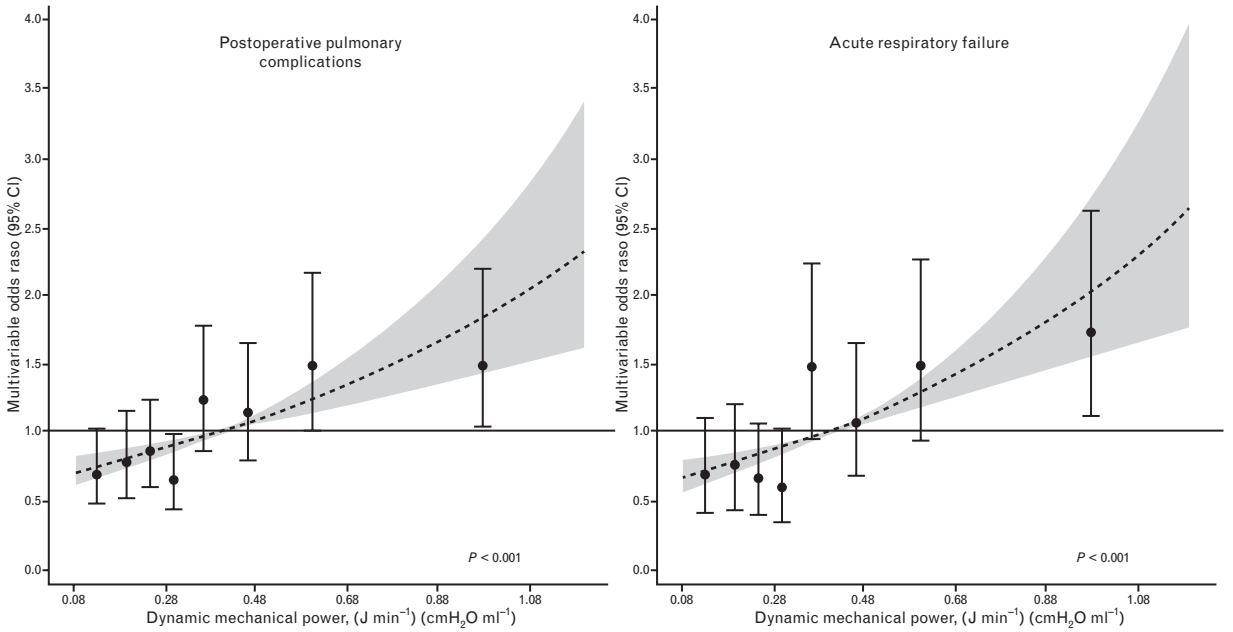
Our findings imply that there is an association between mechanical power and PPCs in adult patients receiving mechanical ventilation during general anaesthesia for major surgery. As mechanical power was normalised to C_{RS}, as recently suggested,[22,23] its combination of flow and respiratory rate provided an additional component to

Table 3 Independent impact of mechanical power on outcomes after adjustment for confounders

	Postoperative pulmonary complications		Acute respiratory failure	
	Odds ratio (95% CI)	P value	Odds ratio (95% CI)	P value
Age	1.11 (0.97 to 1.26)	0.126	1.12 (0.95 to 1.31)	0.184
Male gender	1.15 (0.89 to 1.48)	0.296	1.20 (0.88 to 1.66)	0.251
Baseline SpO_2	0.86 (0.67 to 1.10)	0.237	0.85 (0.62 to 1.16)	0.306
Baseline HCO_3	0.91 (0.80 to 1.04)	0.162	0.79 (0.68 to 0.92)	0.003
Low tidal volume group	0.85 (0.75 to 0.97)	0.016	0.88 (0.75 to 1.03)	0.114
ARISCAT score	1.82 (1.58 to 2.11)	<0.001	1.07 (0.91 to 1.27)	0.403
Mechanical power adjusted by compliance	1.34 (1.17 to 1.52)	<0.001	1.40 (1.21 to 1.61)	<0.001

All continuous variables were standardised before inclusion and odds ratio represents the increase in one standard deviation of the variable. ARISCAT, Assess Respiratory Risk in Surgical Patients in Catalonia; CI, confidence interval; HCO_3, bicarbonate; SpO_2, oxygen saturation from pulse oximetry.

Fig. 2 Odds ratio for postoperative pulmonary complications and acute respiratory failure.



Dashed lines and grey areas represent odds ratio and 95% confidence interval for increasing values of dynamic mechanical power analysed as a continuous variable and centralised in the mean of each variable. Circles and error bars are odds ratio and 95% confidence interval for eight quantiles of increasing dynamic mechanical power. The equation for dynamic mechanical power is the mechanical power divided by the respiratory system compliance. All models adjusted for age, sex, baseline SpO_2, baseline bicarbonate, randomisation group and the Assess Respiratory Risk in Surgical Patients in Catalonia (ARISCAT) score.

quantify repetitive and dynamic energy.[12,14,18,22] The elements of mechanical power may thus represent modifiable risk factors of PPC.

Strengths and limitations

This is the first study to assess the impact of mechanical power in surgical patients during anaesthesia. The analysis was derived from a large randomised clinical trial. The assessment of outcomes in the original trial was blinded to treatment allocation, attenuating ascertainment bias. Also, patients who underwent surgery expected to last more than 2 h were selected to increase the putative adverse effect of mechanical ventilation. Furthermore, different types of surgery were included, which increased the generalisability of the findings.

The study has important limitations. This is a post hoc analysis of a clinical trial. Thus, no causal relationship can be inferred or determined. Also, harmful stress and subsequent VILI are caused by transpulmonary ΔP, but we only had measurements of dynamic airway ΔP. Similar to other cohorts of mechanically ventilated patients,[15,25,26] including surgical patients,[27] static measurements of P_{plat} were available in only a minority of patients. Airway ΔP does correlate with transpulmonary ΔP but rather it represents a surrogate, which might be affected by numerous factors (e.g. resistive pressures, chest wall compliance and spontaneous breathing).[28,29] However, in a real-life clinical scenario, P_{plat} is rarely measured during surgery,[27] and the dynamic measurements reported in this study could be considered. We considered only one measurement of peak pressure in the present study, and no longitudinal sequential measurement was considered. In addition, no information was available on blood loss, surgical manipulation or intraoperative positioning. Also, a high mechanical power may reflect the degree of lung injury, and to assess the causal relationship of each of these variables with PPCs, a randomised clinical trial is needed.

Conclusion

In this study of adult patients receiving mechanical ventilation during major surgery, exposure to higher mechanical ventilation intensity, as measured by higher mechanical power, was associated with an increased risk of PPCs and acute respiratory failure within the first 7 days of the postoperative period. Our findings provide a rationale for the conduct of controlled studies aimed at decreasing the mechanical power applied to the lungs during intra-operative mechanical ventilation in patients undergoing major surgery.

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