

Body Position Alters Mechanical Power and Respiratory Mechanics During Thoracic Surgery

Davide Chiumello, MD,* Paolo Formenti, MD,† Luca Bolgiaghi, MD,†
Giovanni Mistraletti, MD,†‡ Miriam Gotti, MD,† Francesco Vetrone, MD,§
Alessandro Baisi, MD,|| Luciano Gattinoni, MD,¶ and Michele Umbrello, MD†

BACKGROUND: During thoracic surgery, patients are usually positioned in lateral decubitus and only the dependent lung ventilated. The ventilated lung is thus exposed to the weight of the contralateral hemithorax and restriction of the dependent chest wall. We hypothesized that mechanical power would increase during one-lung ventilation in the lateral position.

METHODS: We performed a prospective, observational, single-center study from December 2016 to May 2017. Thirty consecutive patients undergoing general anesthesia with mechanical ventilation (mean age, 68 ± 11 years; body mass index, 25 ± 5 kg·m⁻²) for thoracic surgery were enrolled. Total and partitioned mechanical power, lung and chest wall elastance, and esophageal pressure were compared in supine and lateral position with double- and one-lung ventilation and with closed and open chest both before and after surgery. Mixed factorial ANOVA for repeated measurements was performed, with both step and the period before or after surgery as 2 within-subject factors, and left or right body position during surgery as a fixed, between-subject factor. Appropriate interaction terms were included.

RESULTS: The mechanical power was higher in lateral one-lung ventilation compared to both supine and lateral position double-lung ventilation (11.1 ± 3.0 vs 8.2 ± 2.7 vs 8.7 ± 2.6 ; mean difference, 2.9 J·minute⁻¹ [95% CI, 1.4 – 4.4 J·minute⁻¹] and 2.4 J·minute⁻¹ [95% CI, 0.9 – 3.9 J·minute⁻¹]; $P < .001$ and $P = .002$, respectively). Lung elastance was higher during lateral position one-lung ventilation compared to both lateral and supine double-lung ventilation (24.3 ± 8.7 vs 9.5 ± 3.8 vs 10.0 ± 3.8 ; mean difference, 14.7 cm H₂O·L⁻¹ [95% CI, 11.2 – 18.2 cm H₂O·L⁻¹] and 14.2 cm H₂O·L⁻¹ [95% CI, 10.8 – 17.7 cm H₂O·L⁻¹], respectively) and was higher compared to predicted values (20.1 ± 7.5 cm H₂O·L⁻¹). Chest wall elastance increased in lateral position double-lung ventilation compared to supine (11.1 ± 3.8 vs 6.6 ± 3.4 ; mean difference, 4.5 cm H₂O·L⁻¹ [95% CI, 2.6 – 6.3 cm H₂O·L⁻¹]) and was lower in lateral position one-lung ventilation with open chest than with a closed chest (3.5 ± 1.9 vs 7.1 ± 2.8 ; mean difference, 3.6 cm H₂O·L⁻¹ [95% CI, 2.4 – 4.8 cm H₂O·L⁻¹]). The end-expiratory esophageal pressure decreased moving from supine position to lateral position one-lung ventilation while increased with the opening of the chest wall.

CONCLUSIONS: Mechanical power and lung elastance are increased in the lateral position with one-lung ventilation. Esophageal pressure monitoring may be used to follow these changes. (Anesth Analg 2020;130:391–401)

KEY POINTS

- **Question:** What is the power delivered by the ventilator and how do the mechanical properties of the lung and chest wall change during the different phases of thoracic surgery?
- **Findings:** The mechanical power delivered by the ventilator increased during lateral position and with one-lung ventilation despite the reduction in tidal ventilation. Lung elastance was higher than expected.
- **Meaning:** The power delivered by the ventilator is higher in lateral position than in the supine position irrespective of the reduction in the tidal volume and the mechanical characteristics of the dependent lung deteriorate during anesthesia.

From the *Struttura Complessa (SC) Anestesia e Rianimazione, Ospedale San Paolo – Polo Universitario, Azienda Socio-Sanitaria Territoriale (ASST) Santi Paolo e Carlo, and Dipartimento di Scienze della Salute, Università degli Studi di Milano, Milan, Italy; †SC Anestesia e Rianimazione, ASST Santi Paolo e Carlo, Ospedale San Paolo – Polo Universitario, Milan, Italy; ‡Dipartimento di Fisiopatologia Medico-Chirurgica e dei Trapianti, Università degli Studi di Milano, Milan, Italy; §Dipartimento di Scienze della Salute, Università degli Studi di Milano Milan, Italy; ||Unità Operativa (UO) Chirurgia Toracica, Ospedale San Paolo – Polo Universitario, ASST Santi Paolo e Carlo, and Dipartimento di Scienze della Salute, Università degli Studi di Milano, Milan, Italy; and ¶Department of Anesthesiology, Emergency, and Intensive Care Medicine, Georg-August-University of Göttingen, Göttingen, Germany.

Accepted for publication March 18, 2019.

Copyright © 2019 International Anesthesia Research Society
DOI: 10.1213/ANE.00000000000004192

Funding: None.

The authors declare no conflicts of interest.

Supplemental digital content is available for this article. Direct URL citations appear in the printed text and are provided in the HTML and PDF versions of this article on the journal's website (www.anesthesia-analgesia.org).

Preliminary data for this study were presented as a poster presentation at the European Respiratory Society 2017 International Congress, September 9–13, 2017, Milan, Italy.

Reprints will not be available from the authors.

Address correspondence to Davide Chiumello, MD, SC Anestesia e Rianimazione, ASST Santi Paolo e Carlo, Ospedale San Paolo – Polo Universitario, Via Di Rudini, 8, 20142 Milano, Italy. Address e-mail to davide.chiumello@unimi.it

Mechanical ventilation in patients undergoing thoracic surgery is often complex^{1,2} because patients are usually positioned in lateral decubitus and the operative lung is intermittently deflated to facilitate surgical exposure. One potential strategy to avoid excessive tidal volume (V_T) delivery to the dependent lung when the other is deflated is to decrease the delivered V_T when ventilation is switched to 1 lung. However, the effect of thoracic surgery and lateral position on single-lung elastance is not well characterized. The dependent lung is submitted to higher compressive forces generated by the abdominal content, restriction of the dependent chest wall, gravitational shift of the mediastinum content, and weight of the contralateral hemithorax.³ The overall effect in the dependent lung in lateral position is a further reduction in functional residual capacity (FRC) and an increase in lung elastance.^{4–6} On the contrary, after the pleura is opened, the operative lung is electively deflated to facilitate surgical exposure. Thus, the dependent lung may receive a higher minute ventilation than during 2-lung ventilation, potentially increasing its stress (transpulmonary pressure) and strain despite the reduced V_T .

Although esophageal pressure may not represent the overall pleural pressure, it effectively measures changes in transpulmonary pressure due to body position.⁷ Because stress and strain are primary determinants of ventilator-induced lung injury,⁸ smaller V_T s and increased positive end-expiratory pressure (PEEP) have been proposed for dependent lung ventilation.² Current data suggest that, besides V_T and PEEP, other parameters such as respiratory rate (RR) and flow may affect ventilator-induced lung injury.^{9,10}

Taken together, it has been hypothesized that the extent of lung injury depends on the total amount of “mechanical power” delivered by the ventilator per unit of time.¹¹ Mechanical power is a single variable which combines volume, pressures, flow, and RR. In animals, the mechanical power delivered by the ventilator correlated with the development of ventilator-induced lung injury.¹² In humans, a 2018 trial found an association between higher mechanical power and worse outcomes in mechanically ventilated, critically ill patients.¹³

Acute lung injury may develop in up to 4% of patients undergoing one-lung ventilation for thoracic surgery, despite the use of protective ventilation strategies.¹⁴ Moreover, this lung injury seems to be related to the stress caused by mechanical ventilation during one-lung ventilation.¹⁵ We hypothesized that the mechanical power required to ventilate patients during thoracic surgery would increase with lateral decubitus position and 1 (nondependent) lung ventilation. Our primary outcome was thus the change in mechanical power while switching from supine to lateral position, and from double- to one-lung ventilation, during thoracic

surgery. Secondary outcomes were postoperative pulmonary complications, changes in lung and chest wall elastance, and changes in airway and transpulmonary driving pressure and in end-expiratory esophageal pressure in the supine and lateral positions. In addition, we used our findings to estimate the contributions of PEEP, elastance, body position, and resistance-related components to mechanical power.

METHODS

Patients

Ethical approval for this study (no. 42960/2016) was provided by the Comitato Etico Interaziendale Milano Area A, Milano, Italy (chairperson: Prof. A. M. Di Giulio). Written informed consent was obtained according to the Italian regulations. This manuscript adheres to the applicable STrengthening the Reporting of OBServational studies in Epidemiology (STROBE) guidelines.

Thirty consecutive patients undergoing general anesthesia for thoracic surgery between December 2016 and May 2017 at Azienda Socio-Sanitaria Territoriale (ASST) Santi Paolo e Carlo Hospital were enrolled. Exclusion criteria were patients <18 years of age, esophageal varices grade 2 or higher, severe coagulopathy, esophageal or gastric surgery performed in the last 6 months, or previous thoracic surgery.

After arrival in the operating theater, standard monitoring, including pulse oximetry, electrocardiography, and noninvasive blood pressure, was established. An arterial catheter was placed at the discretion of the attending anesthesiologist. Before surgery, patients were premedicated with midazolam 0.15 mg·kg⁻¹ IV. Induction of general anesthesia included propofol 1.5–2 mg·kg⁻¹, fentanyl 1.5 µg·kg⁻¹, and cisatracurium 0.15 mg·kg⁻¹ was then administered. Anesthesia was maintained with sevoflurane titrated to a minimal alveolar concentration of 1.8%, and a remifentanyl infusion (0.1 µg·kg⁻¹·minute⁻¹). Paralysis was achieved with additional doses of cisatracurium to maintain 0–1 train-of-four responses of the adductor pollicis muscle after the stimulation of the ulnar nerve throughout anesthesia. The trachea was intubated with a left-sided double-lumen endobronchial tube (Rusch; Teleflex Medical, Seattle, WA) of the appropriate size (37–41 French). The correct position of the double-lumen endobronchial tube was verified by bronchoscopy both with the patient supine and in the lateral position. Subsequently, the endobronchial and tracheal cuffs were inflated, and an inspiratory pause was performed to exclude any possible air leaks. All surgical procedures were performed by a single surgeon. Intraoperative fluid was limited to 2–4 mL·kg⁻¹·hour⁻¹ of crystalloid for the entire duration unless hemodynamic instability. At the end of the surgery, a chest tube was positioned without air suction. All patients were extubated in the

operating theater and transferred to a postanesthesia care unit or the surgical ward.

Ventilation

A Flow-i (Maquet, Rastatt, Germany) mechanical ventilator was used for all the procedures. All patients were ventilated in volume control mode with a V_T of $8 \text{ mL}\cdot\text{kg}^{-1}$ of predicted body weight during double-lung ventilation and $5 \text{ mL}\cdot\text{kg}^{-1}$ during one-lung ventilation. A constant inspiratory flow mode with 33% inspiratory time was used. RR was initially set at 12 breaths/min and then adjusted to target an end-tidal carbon dioxide concentration of 35 mmHg. During one-lung ventilation, RR was increased to maintain the same minute volume as in double-lung ventilation. A PEEP of 8 cm H_2O was applied in all cases, and the oxygen fraction was titrated to ensure an arterial oxygen saturation between 96% and 99%.

Measurements

Measurements were performed during anesthesia and paralysis in (1) supine position during double-lung ventilation; (2) lateral position double-lung ventilation; (3) lateral position one-lung ventilation closed chest; and (4) lateral position one-lung ventilation open chest before surgery. These same measurements were then repeated after surgery. Lateral and supine positions were standardized between patients to allow comparison. The study protocol is summarized in Supplemental Digital Content, Figure S1, <http://links.lww.com/AA/C796>.

Airway pressure was measured proximal to the endotracheal tube with a dedicated pressure transducer (MPX 2010 DP; Motorola, Solna, Sweden). To account for the different resistive properties of double- and single-lumen endotracheal tubes, the pressure gradient across the tube for every patient in every ventilator condition was calculated.¹⁶ The measured peak airway pressure (P_{MAX}) was then corrected by the pressure gradient across the tube. Further details about the calculation are presented in Supplemental Digital Content, Methods M1, <http://links.lww.com/AA/C796>. The corrected P_{MAX} value is presented throughout the manuscript.

As a surrogate for pleural pressure, esophageal pressure was measured using a standard balloon catheter (Smart Cath; Viasys, Palm Springs, CA) consisting of a tube 103-cm long with an external diameter of 3 mm and a thin-walled balloon 10-cm long. The esophageal catheter was emptied of air and introduced transorally into the esophagus to reach the stomach at a depth of 50–55 cm from the mouth. Subsequently, the balloon was inflated with 1.5 mL of air. The intragastric position of the catheter was confirmed by a positive pressure deflection of

intra-abdominal pressure during an external manual epigastric pressure. Subsequently, the catheter was retracted and positioned in the low esophageal position. Esophageal balloon could be placed in all patients. The amount of gas in the balloon was periodically checked, ensuring a constant inflation volume of 1.5 mL during the whole study. All traces were sampled at 100 Hz and processed on a dedicated data acquisition system (Colligo and Computo; Elekton, Milan, Italy; www.elekton.it).

Mechanical Power

We defined mechanical power as the energy delivered from the ventilator to the respiratory system per breath times the RR. Mechanical power is expressed in $\text{J}\cdot\text{minute}^{-1}$ and is calculated for our study according to Gattinoni et al.¹¹ Briefly, the energy delivered to the respiratory system is composed of a static component, due to PEEP and PEEP volume, and a dynamic cyclic component, due to driving pressure and V_T above PEEP, plus the additional, resistive component generated by the pressure required to cause gas flow. Because energy is equal to the pressure applied times the change in volume, an equation¹¹ can be defined as follows:

$$\text{Power} = 0.098 \times \text{RR} \times \text{TV} \times \left(P_{\text{peak}} - \frac{1}{2} \times [P_{\text{plat}} - \text{PEEP}] \right)$$

where TV the tidal volume, P_{peak} the P_{MAX} and P_{plat} the end-inspiratory plateau pressure. In our study, to isolate the power delivered to the lung (versus the lung and chest wall), we calculated mechanical power delivered using transpulmonary pressure instead of airway pressure. See Supplemental Digital Content, Methods M2, <http://links.lww.com/AA/C796>, for a more detailed explanation of the equation of mechanical power and its PEEP-, elastance-, and resistance-related components and for the determination of the partitioned mechanical power applied to the lungs.

Respiratory Mechanics

The static airway and esophageal pressures were measured during an end-inspiratory and end-expiratory pause. End-expiratory esophageal pressure was measured during an expiratory pause. The airway and transpulmonary driving pressure were computed as the difference between the airway and transpulmonary pressure between the end-inspiratory and the end-expiratory pause. Respiratory system, lung, and chest wall elastances were computed according to the following standard formulae¹⁷:

$$\text{Ers}(\text{cm H}_2\text{O}\cdot\text{L}^{-1}) = \frac{\left(\begin{array}{l} \text{Airway pressure at end inspiration} \\ - \text{Airway pressure at PEEP} \end{array} \right)}{\text{Tidal volume}}$$

$$E_l(\text{cm H}_2\text{O}\cdot\text{L}^{-1}) = \frac{\left(\begin{array}{l} \text{Transpulmonary pressure at end inspiration} \\ - \text{Transpulmonary pressure at PEEP} \end{array} \right)}{\text{Tidal volume}}$$

$$E_{cw}(\text{cm H}_2\text{O}\cdot\text{L}^{-1}) = \frac{\left(\begin{array}{l} \text{Esophageal pressure at end inspiration} \\ - \text{Esophageal pressure at PEEP} \end{array} \right)}{\text{Tidal volume}}$$

where E_{rs} is the Respiratory system elastance; E_l the lung elastance; and E_{cw} the chest wall elastance. Because the 2 lungs are in parallel, assuming that in supine position the elastance of both lungs is similar, the predicted elastance of the ventilated lung in lateral position should be equal to twice the lung elastance in supine position.¹⁸

Postoperative Pulmonary Complications

As a post hoc analysis, we assessed the incidence of postoperative pulmonary complications. These were defined as pulmonary abnormalities occurring during the postoperative period and resulting in clinically significant disease or dysfunction¹⁹ and included respiratory infection, respiratory failure, pleural effusion, atelectasis, pneumothorax, bronchospasm, and aspiration pneumonitis. Definitions of pulmonary complications are presented in Supplemental Digital Content, Methods M3, <http://links.lww.com/AA/C796>.

Statistical Analysis

Data were analyzed using Stata 11 (StataCorp LP, College Station, TX). Normality was assessed by the Shapiro–Francia test. Results are reported as mean (SD) if normally distributed or median (25–75th percentiles) otherwise. The analysis on the variables recorded over the 4 different steps (supine double-lung ventilation, lateral double-lung ventilation, lateral one-lung ventilation with closed chest, and lateral one-lung ventilation with open chest) was performed by mixed factorial ANOVA for repeated measurements, with both step and the period before or after surgery as 2 within-subject factors, and left or right body position during surgery as a fixed, between-subject factor. The interaction effects between (1) step on the period before or after surgery, (2) left or right body position on the step, and (3) left or right body position on the time before or after surgery as well as (4) between left or right position, step, and the period before or after surgery were included in the model. The statistical significance of the within-subject factors was corrected with the Greenhouse–Geisser method. Pairwise post hoc multiple comparisons between the marginal means were performed, when appropriate, according to Tukey honestly significant difference method. In the case of statistically significant interactions, pairwise post hoc multiple interaction comparisons have been performed using the same Tukey method for

Table 1. Patient Characteristics

Patient number	30
Age (y)	72 (12)
Male sex, n (%)	18/30 (60)
Actual body weight (kg)	74 (15)
Ideal body weight (kg)	64 (6)
Body mass index (kg·m ⁻²)	25 (5)
ASA physical status, n (%)	
Class I–II	16/30 (53.3)
Class III–IV	14/30 (46.7)
Underlying diseases, n (%)	
Hypertension	18/30 (60)
Coronary artery disease	1/30 (3.3)
Diabetes	5/30 (16.7)
Active smoker, n (%)	9/30 (30)
Pulmonary function (% of predicted)	
Forced vital capacity	99.2 (17.7)
FEV ₁	91.4 (17.5)
FEV ₁ /forced vital capacity	96.3 (12.5)
CO diffusion capacity	71.3 (18.5)
Chronic lung disease, n (%)	
Obstructive	6/30 (20)
Restrictive	1/30 (3.3)
Previous lung surgery, n (%)	5/30 (16.7)
Lung surgery, n (%)	
Wedge resection	14/30 (47)
Lobectomy	16/30 (53)
Decubitus, n (%)	
Left-side dependent	19/30 (63)
Right-side dependent	11/30 (37)
Intraoperative timing (min)	
Anesthesia length	201 (52)
Surgery length	138 (39)
One-lung ventilation length	137 (35)
Infused fluid amount (mL)	2150 (2000–2500)

Variables are expressed as mean (SD) or median (interquartile range).

Abbreviations: ASA, American Society for Anesthesiologists; CO, carbon monoxide; FEV₁, forced expiratory volume in 1 s.

multiple comparison. Adjusted P values and 95% CIs are reported for pairwise comparisons. The comparison of mechanical power during lateral position and one-lung ventilation with the predicted value was performed with a Student t test for paired data. Two-tailed P values $<.05$ were considered statistically significant.

Based on previous data on mechanical power in patients undergoing double-lung mechanical ventilation in supine position and with otherwise healthy lungs,¹¹ we calculated that a sample size of 30 patients would allow us to demonstrate a Cohen f effect size of 0.25 if a correlation among the 4 repeated measurements of 0.6 is conservatively assumed and a correction for nonsphericity of 0.7 is applied at a significance level of .05 and a power of 80%. Sample size calculation was performed with G*Power 3 statistical software (Heinrich-Heine-Universität Düsseldorf, Düsseldorf, DE).

RESULTS

All 30 patients successfully underwent the protocol. The main characteristics are reported in Tables 1 and 2 and Supplemental Digital Content, Tables S1–S2, <http://links.lww.com/AA/C796>. Fifty-three percent of patients ($N = 16$) were American Society for Anesthesiologists

	Supine Double-Lung Ventilation	Lateral Double-Lung Ventilation	Lateral One-Lung Ventilation	Lateral One-Lung Ventilation Open Chest	P
V _T (mL)	474 (65)	476 (65)	349 (51) ^{a,b}	349 (51) ^b	<.001
V _T (mL·kg ⁻¹ ideal body weight)	7.5 (0.8)	7.5 (0.9)	5.5 (0.7) ^{a,b}	5.5 (0.7) ^b	<.001
RR (breaths/min)	14 (2)	14 (2)	19 (2) ^{a,b}	18 (2) ^b	<.001
VE (L·minute ⁻¹)	6.5 (1.2)	6.5 (1.2)	6.5 (1.0)	6.5 (1.1)	.373
F _{IO₂}	0.65 (0.08)	0.66 (0.08)	0.65 (0.08)	0.67 (0.07)	.093
PEEP (cm H ₂ O)	7.9 (0.8)	7.9 (0.8)	7.9 (0.8)	8.1 (0.4)	.326
P _{max} (cm H ₂ O)	21.8 (4.6)	23.4 (3.6) ^a	29.5 (5.1) ^{a,b}	28.4 (4.4) ^{a,b}	<.001
Corrected P _{max} (cm H ₂ O)	17.9 (4.3)	19.6 (3.9) ^a	26.4 (5.0) ^{a,b}	25.1 (4.3) ^{a,b}	<.001
Plateau airway pressure (cm H ₂ O)	15.2 (2.1)	16.8 (1.9) ^{a,b}	18.0 (2.7) ^{a,b}	16.8 (2.9) ^{a,b}	<.001
Plateau esophageal pressure (cm H ₂ O)	13.6 (3.5)	9.9 (3.1) ^{a,b}	4.8 (2.5) ^{a,b}	5.4 (2.2) ^{a,b}	<.001
End-expiratory esophageal pressure (cm H ₂ O)	10.6 (3.7)	4.8 (2.4) ^{a,b}	2.4 (1.7) ^{a,b}	4.2 (2.3) ^{a,b}	<.001
Airway driving pressure (cm H ₂ O)	7.7 (2.1)	9.3 (1.7) ^{a,b}	10.4 (2.6) ^{a,b}	9.2 (2.7) ^{a,b}	<.001
Transpulmonary driving pressure (cm H ₂ O)	4.6 (1.7)	4.2 (1.8)	8.3 (3.1) ^{a,b}	8.3 (3.3) ^b	<.001
Respiratory system elastance (cm H ₂ O·L ⁻¹)	16.7 (4.8)	20.1 (4.2) ^{a,b}	30.6 (8.3) ^{a,b}	26.7 (8.1) ^{a,b}	<.001
Chest wall elastance (cm H ₂ O·L ⁻¹)	6.6 (3.4)	11.1 (3.8) ^{a,b}	7.1 (2.8) ^a	3.5 (1.9) ^{a,b}	<.001
Lung elastance (cm H ₂ O·L ⁻¹)	10.0 (3.8)	9.5 (3.8)	24.3 (8.7) ^{a,b}	24.0 (8.4) ^{a,b}	<.001

N = 30 patients. Variables are expressed as mean (SD). The analysis on the variables recorded over the 4 different steps (supine double-lung ventilation, lateral double-lung ventilation, lateral one-lung ventilation with closed chest, and lateral one-lung ventilation with open chest) was performed by mixed factorial ANOVA for repeated measurements, with both step and the period before or after surgery as 2 within-subject factors, and left or right body position during surgery as a fixed, between-subject factor. Appropriate interaction terms were included in the model. The significance of the within-subject factors was corrected with the Greenhouse–Geisser method. Pairwise post hoc multiple comparisons were performed according to Tukey honestly significant difference method when appropriate. Adjusted P values and 95% CIs are reported for pairwise comparisons, and 2-tailed P values <.05 were considered statistically significant.

Abbreviations: F_{IO₂}, fraction of inspired oxygen; PEEP, positive end-expiratory pressure; P_{max}, peak airway pressure; RR, respiratory rate; VE, minute ventilation; V_T, tidal volume.

^aP < .05 versus previous step.

^bP < .05 versus supine double-lung ventilation.

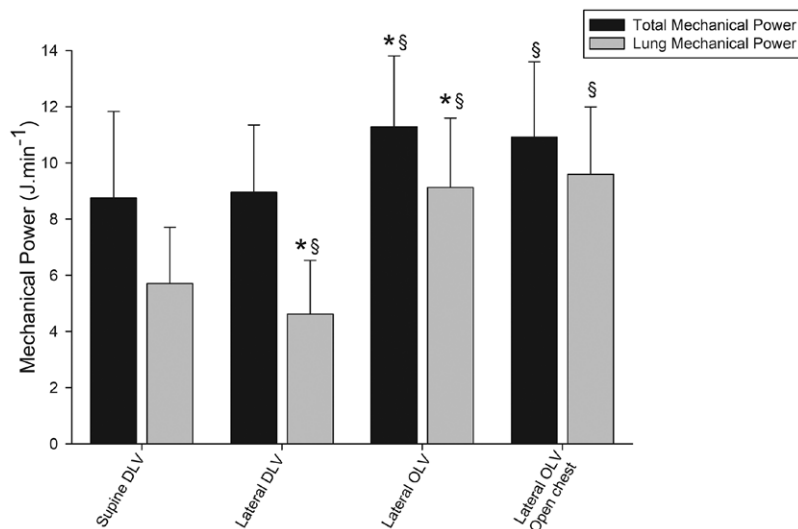


Figure 1. Total and lung mechanical power during the different study steps before surgery. The analysis on the variables recorded over the 4 different steps (supine DLV, lateral DLV, lateral OLV with closed chest, and lateral OLV with open chest) was performed by mixed factorial ANOVA for repeated measurements, with both step and the period before or after surgery as 2 within-subject factors, and left or right body position during surgery as a fixed, between-subject factor. Appropriate interaction terms were included in the model. The significance of the within-subject factors was corrected with the Greenhouse–Geisser method. Pairwise post hoc multiple comparisons were performed according to Tukey honestly significant difference method when appropriate. Adjusted P values and 95% CIs are reported for pairwise comparisons, and 2-tailed P values <.05 were considered statistically significant. Data are shown as mean and SD. *P < .05 versus previous step; §P < .05 versus supine DLV. DLV indicates double-lung ventilation; OLV, one-lung ventilation.

class I or II, and the remainder (N = 14) were American Society for Anesthesiologists class III or IV. Nineteen and 11 patients underwent left and right lateral side, respectively. As dictated by the protocol, the applied V_T was lower and RR was higher in lateral position compared to supine position (Table 2). Intrinsic PEEP was not detected during any of the study steps.

Mechanical Power

Both total and lung mechanical power were higher in lateral position during one-lung ventilation with open or closed chest when compared to power in the supine position. Power was also greater in lateral position during one-lung ventilation than with double-lung ventilation (Figure 1). Supplemental Digital Content, Figure S2,

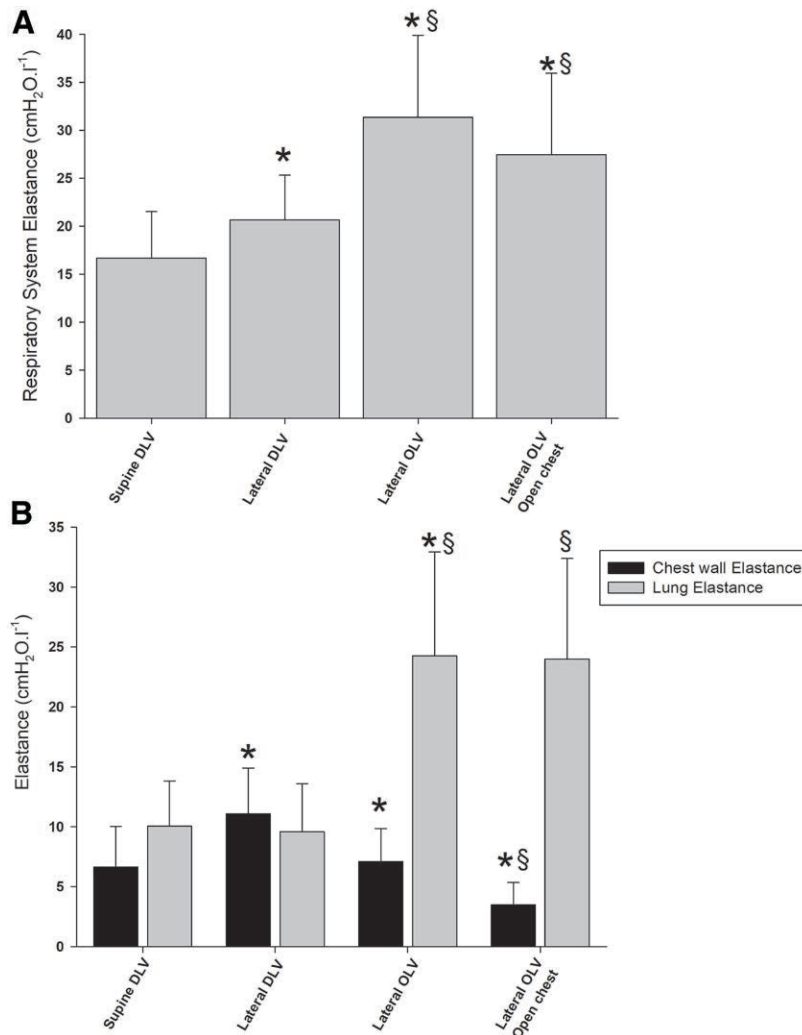


Figure 2. Respiratory system, lung and chest wall elastance during the different study steps before surgery. The analysis on the variables recorded over the 4 different steps (supine DLV, lateral DLV, lateral OLV with closed chest, and lateral OLV with open chest) was performed by mixed factorial ANOVA for repeated measurements, with both step and the period before or after surgery as 2 within-subject factors, and left or right body position during surgery as a fixed, between-subject factor. Appropriate interaction terms were included in the model. The significance of the within-subject factors was corrected with the Greenhouse–Geisser method. Pairwise post hoc multiple comparisons were performed according to Tukey honestly significant difference method when appropriate. Adjusted *P* values and 95% CIs are reported for pairwise comparisons, and 2-tailed *P* values < .05 were considered statistically significant. Data are shown as mean and SD. **P* < .05 versus previous step; §*P* < .05 versus supine DLV. DLV indicates double-lung ventilation; OLV, one-lung ventilation.

<http://links.lww.com/AA/C796>, shows the behavior of the PEEP-, elastance-, and resistance-related components of total and lung mechanical power in the different study steps. The comparison of total and lung mechanical power in patients who did and did not develop postoperative pulmonary complications is reported in Supplemental Digital Content, Table S3, <http://links.lww.com/AA/C796>. In the period after surgery, both total and lung mechanical power were increased as compared to the period before surgery (*P* < .001). No statistically significant interaction was found between side and step for either total or lung mechanical power.

Respiratory Mechanics

Respiratory system elastance increased in lateral position with double-lung ventilation compared to supine

position (Figure 2A). During lateral position and one-lung ventilation with closed chest, respiratory system elastance was higher compared to lateral double-lung ventilation and lateral one-lung ventilation open chest (30.6 [8.3] vs 20.1 [4.2] and 26.7 [8.1]; mean difference, 10.7 cm H₂O·L⁻¹ [95% CI, 7.1–14.3 cm H₂O·L⁻¹] and 3.9 cm H₂O·L⁻¹ [95% CI, 0.5–7.3 cm H₂O·L⁻¹], respectively) (Figure 2A; Table 2). Respiratory system elastance did not differ at the end of surgery when compared to that before surgery (*P* = .304).

Lung elastance was higher during lateral position and one-lung ventilation compared to both lateral position with double-lung ventilation and supine position (24.3 [8.7] vs 9.5 [3.8] vs 10.0 [3.8]; mean difference, 14.7 cm H₂O·L⁻¹ [95% CI, 11.2–18.2 cm H₂O·L⁻¹] and 14.2 cm H₂O·L⁻¹ [95% CI, 10.8–17.7 cm H₂O·L⁻¹],

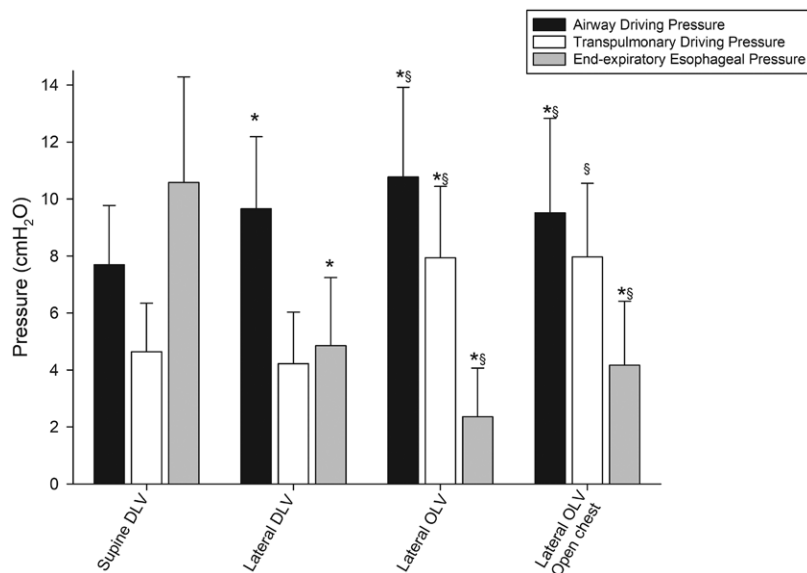


Figure 3. Driving pressure and end-expiratory oesophageal pressure during the different study steps before surgery. The analysis on the variables recorded over the 4 different steps (supine DLV, lateral DLV, lateral OLV with closed chest, and lateral OLV with open chest) was performed by mixed factorial ANOVA for repeated measurements, with both step and the period before or after surgery as 2 within-subject factors, and left or right body position during surgery as a fixed, between-subject factor. Appropriate interaction terms were included in the model. The significance of the within-subject factors was corrected with the Greenhouse–Geisser method. Pairwise post hoc multiple comparisons were performed according to Tukey honestly significant difference method when appropriate. Adjusted *P* values and 95% CIs are reported for pairwise comparisons, and 2-tailed *P* values < .05 were considered statistically significant. Data are shown as mean and SD. **P* < .05 versus previous step; §*P* < .05 versus supine DLV. DLV indicates double-lung ventilation; OLV, one-lung ventilation.

respectively) (Figure 2B). The computed lung elastance during lateral position and one-lung ventilation was higher compared to the predicted (24.3 cm H₂O·L⁻¹ [8.7 cm H₂O·L⁻¹] vs 20.1 cm H₂O·L⁻¹ [7.5 cm H₂O·L⁻¹]; *P* < .005).

Chest wall elastance was higher in lateral position with double-lung ventilation compared to supine position. Chest wall elastance was also lower in lateral position and one-lung ventilation with open chest compared to closed chest and lateral position (Figure 2B).

Driving Pressure and End-Expiratory Esophageal Pressure

After reducing V_T s from 8 to 5 mL·kg⁻¹, airway and transpulmonary driving pressure were higher in lateral position with one-lung ventilation compared to supine and lateral position with double-lung ventilation (10.4 cm H₂O [2.6 cm H₂O] vs 9.3 cm H₂O [1.7 cm H₂O] vs 7.7 cm H₂O [2.1 cm H₂O] and 8.3 cm H₂O [3.1 cm H₂O] vs 4.2 cm H₂O [1.8 cm H₂O] vs 4.6 cm H₂O [1.7 cm H₂O], respectively) (Figure 3; Table 2).

The end-expiratory esophageal pressure decreased when transitioning from supine to lateral position with one-lung ventilation and increased with the opening of the chest wall (Figure 3).

Right Compared to Left Lateral Position

No differences in respiratory system, lung, and chest wall elastance were observed in patients managed in right versus left lateral decubitus position. Similarly,

in lateral position during double-lung ventilation, no differences in airway or transpulmonary driving pressure, or respiratory system, lung, and chest wall elastance, or total or partitioned mechanical power were found between right and left lateral decubitus position (Supplemental Digital Content, Figures S3–S6, <http://links.lww.com/AA/C796>).

End-expiratory esophageal pressures were higher in right lateral position during one-lung ventilation when compared to left lateral position (5.2 cm H₂O [2.7 cm H₂O] vs 3.5 cm H₂O [1.7 cm H₂O] and 3.3 cm H₂O [1.5 cm H₂O] vs 1.8 cm H₂O [1.6 cm H₂O], respectively) (Figure 4; Supplemental Digital Content, Table S2, Figure S4, <http://links.lww.com/AA/C796>).

Postoperative Pulmonary Complications

Seven patients (23.3%) developed ≥1 postoperative pulmonary complication: 2 developed respiratory infections, 2 developed respiratory failure, 1 developed both atelectasis and bronchospasm, and 2 had pleural effusion. The total energy delivered to the lungs during one-lung ventilation (ie, the product of total or lung mechanical power times the duration of one-lung ventilation) was higher in patients who later during their stay developed postoperative pulmonary complications. Supplemental Digital Content, Table S3, <http://links.lww.com/AA/C796>, shows the comparison of total and lung mechanical power in the different study steps in patients who did and did not develop postoperative pulmonary complications.

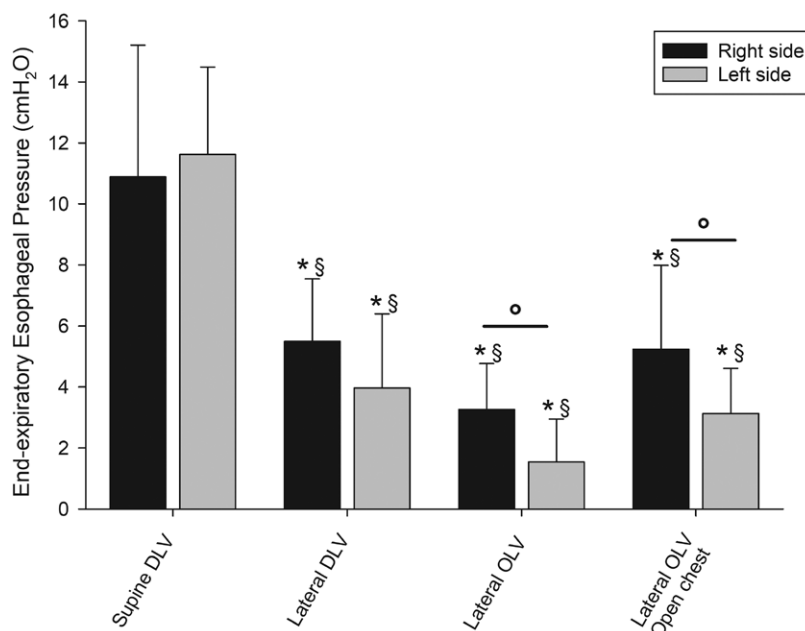


Figure 4. End-expiratory oesophageal pressure in patients positioned on the left and right side during the different study steps before surgery. The analysis on the variables recorded over the 4 different steps (supine DLV, lateral DLV, lateral OLV with closed chest, and lateral OLV with open chest) was performed by mixed factorial ANOVA for repeated measurements, with both step and the period before or after surgery as 2 within-subject factors, and left or right body position during surgery as a fixed, between-subject factor. Appropriate interaction terms were included in the model. The significance of the within-subject factors was corrected with the Greenhouse–Geisser method. Pairwise post hoc multiple comparisons were performed according to Tukey honestly significant difference method when appropriate. Adjusted *P* values and 95% CIs are reported for pairwise comparisons, and 2-tailed *P* values <.05 were considered statistically significant. Data are shown as mean and SD. **P* < .05 versus previous step; §*P* < .05 versus supine DLV; °*P* < .05 right versus left side. DLV indicates double-lung ventilation; OLV, one-lung ventilation.

DISCUSSION

In our prospective study of patients undergoing thoracic surgery under general anesthesia with one-lung ventilation, we found that (1) the mechanical power delivered to the dependent lung increased in lateral position during one-lung ventilation compared to supine position; (2) respiratory system elastance and lung elastance also increased during one-lung ventilation in lateral position; (3) chest wall elastance increased in lateral position with double-lung ventilation as compared to supine; (4) the opening of the chest was associated with a decrease in the respiratory system and chest wall elastance; and (5) end-expiratory esophageal pressure decreased in lateral position and increased with opening of the chest and was significantly higher in patients managed in right compared to left lateral position.

Mechanical Power and Driving Pressure

When compared to previously suggested markers of ventilator-induced lung injury (eg, stress and strain, driving pressure, and elastance), mechanical power incorporates flow, RR, and PEEP and allows a single value to represent the effects of mechanical ventilation on the lung. A 2016 study reported a relationship between the mechanical power and ventilator-induced lung injury, supporting the potential value of mechanical power.¹² In our study, we found

that mechanical power (total and lung) did not differ between supine and lateral position with double-lung ventilation even though driving pressure was higher. However, in lateral position and in one-lung ventilation, lung mechanical power was higher than in supine position and double-lung ventilation despite a decrease in V_T due to an increase in both its elastance and resistive component.² Although we cannot suggest any clinical mechanical power threshold for possible injury, our data suggest that the only decrease of V_T from 8 to 5 mL·kg⁻¹ of ideal body weight did not reduce the mechanical power. Thus, the dependent lung might undergo an underappreciated amount of injury.

Respiratory System, Lung, and Chest Wall Elastance

The main effects of general anesthesia on the respiratory system are a reduction in FRC with alveolar collapse and a higher movement of the nondependent regions of the diaphragm compared to the dependent ones during normal tidal ventilation.^{4,20–22} When positioned in lateral decubitus for thoracic surgery, the dependent lung is always ventilated while the nondependent can be ventilated or collapsed depending on surgical need. Thus, the presence or absence of ventilation significantly affects the distribution of ventilation and FRC. In healthy subjects, respiratory system

elastance is increased in the lateral position with double-lung ventilation compared to supine position.⁵ Thomas et al⁶ reported a similar finding in patients with lung disease. Our study adds further support to these findings. We observed greater respiratory system elastance in lateral position with double ventilation compared to supine, mostly due to an increase in the chest wall elastance. Furthermore, during one-lung ventilation, the elastance was also higher compared to supine position and double-lung ventilation. Consistent with our study, Larsson et al²³ observed that when moving from supine to lateral position during anesthesia, the static elastance of the respiratory of dependent side increased and that of nondependent decreased.

Respiratory system elastance did not change from the beginning to the end of surgery. This finding suggests that the duration of anesthesia did not affect the intrinsic mechanical characteristics of the respiratory system.

To better understand the mechanical behavior of the respiratory system, we separately evaluated lung and chest wall components. However, distinguishing between lung and chest wall is difficult because changes in esophageal pressure may represent artifact. The esophageal pressure only approximates pleural pressure.^{7,24,25} However, changes in esophageal pressure are related to the changes in pleural pressure,²⁶ suggesting that the computation of lung and chest wall elastance reasonably approximates mechanical properties among the different body position.

During one-lung ventilation, the V_T previously delivered to the 2 lungs is now delivered only to the dependent lung. Because right and left lung elastance are similar,⁴ the expected lung elastance of 1 lung should then equal the twice the elastance of the 2 lungs in supine position.¹⁸ Thus, when ventilating 1 lung compared to 2 lungs, an increase in lung elastance should be expected.¹⁸ However, in our study, the measured lung elastance of the dependent lung in lateral position during 1 ventilation was higher than expected, suggesting a deterioration in intrinsic lung characteristics. Although the mechanism underlying this deterioration is unclear, possible mechanisms include an increase in the compressive forces of the mediastinum and abdomen, an alteration in the surface tension, a reduction in lung gas volume, and a higher amount of compression atelectasis and transdiaphragmatic pressure due to a higher abdominal pressure gradient.²⁷

Induction of general anesthesia has a minimal effect on chest wall elastance.²¹ In our study, the lateral position was associated with an increase in chest wall elastance compared to supine position, possibly due to an increase in the dependent rib cage elastance due to the contact with bed. We found, however, that the chest wall elastance decreased after the institution of

one-lung ventilation. Although our data do not allow us to determine the cause of this effect, one possibility is that collapse of the nondependent lung may allow mediastinal structures to move more freely, limiting the restrictive effect of the dependent chest wall rib cage. A further decrease in chest wall elastance was found with the opening of the chest wall in lateral position, supporting this possibility.²⁸

End-Expiratory Esophageal Pressure

Esophageal pressure is an established approach to estimating the average pleural pressure surrounding the lung. However, it can overestimate the pleural pressure of the nondependent zone and underestimate that of dependent regions.²⁹ Furthermore, the pleural pressure varies due to the gravitational gradient.²⁶ In addition, end-expiratory esophageal pressure can be artifactually changed by the weight of the heart and great vessels in different body positions. To counterbalance these positional artifacts, a reduction of 3 cm H₂O in end-expiratory esophageal pressure has been suggested.³⁰ In this study, we did not correct the end-expiratory esophageal pressure values.

Previous studies found in spontaneously breathing healthy subjects that end-expiratory esophageal pressure significantly decreased with changes in position from supine to lateral or prone.³⁰⁻³² We found that end-expiratory esophageal pressure was lower in lateral position with double-lung ventilation. In addition, we did not observe any difference between right and left lateral position with double-lung ventilation. These changes may be due to mediastinal structures (heart and vessels) pressing on the esophagus in supine but not in lateral position.^{31,33}

During the transition from double- to one-lung ventilation, end-expiratory esophageal pressure decreased due to the reduction in pleural pressure by the collapse of the nondependent lung. When the chest wall was opened, end-expiratory esophageal pressure increased. Mechanisms for this effect include a change in pleural pressure from negative to atmospheric and a downward shift of the unsupported mediastinum.²³

We observed a higher end-expiratory esophageal pressure in right compared to left position during one-lung ventilation. Mechanisms for this effect include the lower volume of the left lung,³⁴ thus leading to a smaller decrease in pleural pressure during right decubitus compared to left decubitus due to less traction of the smaller left lung on the pleural surface.

Although our study was limited to the mechanical properties of the lung and respiratory system, it is possible that the increase in mechanical power may partly explain the development of acute lung injury after one-lung ventilation.³⁵ We found that patients who developed postoperative pulmonary

complications received a higher amount of mechanical energy during one-lung ventilation with the open chest (Supplemental Digital Content, Table S3, <http://links.lww.com/AA/C796>). Further studies are warranted to better elucidate whether a strategy-limiting mechanical power may decrease adverse respiratory outcomes.

Because results of the exploratory analyses we performed were not adjusted for multiple comparisons and for multiple secondary outcomes, given their exploratory nature, a potential limitation of this approach is an increased risk of type I error. As such, those results should be considered as “hypothesis generating” only.

In conclusion, during lateral position and one-lung ventilation, mechanical power and lung elastance are increased compared to supine position and double-lung ventilation despite the reduction in tidal ventilation. Further reduction of V_T in such settings may be considered. Further work is needed to better understand the effect of one-lung ventilation and mechanical power on the development of ventilator-associated lung injury. ■■

DISCLOSURES

Name: Davide Chiumello, MD.

Contribution: This author helped with substantial contribution to the design of the study, analyze and interpret the data, and draft the first version of the manuscript.

Name: Paolo Formenti, MD.

Contribution: This author helped analyze the data, enroll patients, and revise the draft critically for important intellectual content.

Name: Luca Bolgiaghi, MD.

Contribution: This author helped perform the literature search, enroll patients, record clinical data, and revise the draft critically for important intellectual content.

Name: Giovanni Mistraletti, MD.

Contribution: This author helped perform the literature search, enroll patients, record clinical data, and revise the draft critically for important intellectual content.

Name: Miriam Gotti, MD.

Contribution: This author helped analyze the data, enroll patients, record clinical data, and revise the draft critically for important intellectual content.

Name: Francesco Vetrone, MD.

Contribution: This author helped record clinical data and revise the draft critically for important intellectual content.

Name: Alessandro Baisi, MD.

Contribution: This author helped enroll patients, perform the surgical procedures, and revise the draft critically for important intellectual content.

Name: Luciano Gattinoni, MD.

Contribution: This author helped with substantial contribution to the design of the study, analyze and interpret the data, and draft the first version of the manuscript.

Name: Michele Umbrello, MD.

Contribution: This author helped with substantial contribution to the design of the study, analyze and interpret the data, and draft the first version of the manuscript.

This manuscript was handled by: Avery Tung, MD, FCCM.

REFERENCES

1. Bignami E, Saglietti F, Di Lullo A. Mechanical ventilation management during cardiothoracic surgery: an open challenge. *Ann Transl Med.* 2018;6:380.
2. Sentürk M. New concepts of the management of one-lung ventilation. *Curr Opin Anaesthesiol.* 2006;19:1–4.
3. Grichnik KP, Clark JA. Pathophysiology and management of one-lung ventilation. *Thorac Surg Clin.* 2005;15:85–103.
4. Rehder K, Hatch DJ, Sessler AD, Fowler WS. The function of each lung of anesthetized and paralyzed man during mechanical ventilation. *Anesthesiology.* 1972;37:16–26.
5. Tanskanen P, Kyttä J, Randell T. The effect of patient positioning on dynamic lung compliance. *Acta Anaesthesiol Scand.* 1997;41:602–606.
6. Thomas PJ, Paratz JD, Lipman J, Stanton WR. Lateral positioning of ventilated intensive care patients: a study of oxygenation, respiratory mechanics, hemodynamics, and adverse events. *Heart Lung.* 2007;36:277–286.
7. Cherniack RM, Farhi LE, Armstrong BW, Proctor DF. A comparison of esophageal and intrapleural pressure in man. *J Appl Physiol.* 1955;8:203–211.
8. Chiumello D, Carlesso E, Cadringer P, et al. Lung stress and strain during mechanical ventilation for acute respiratory distress syndrome. *Am J Respir Crit Care Med.* 2008;178:346–355.
9. Hotchkiss JR Jr, Blanch L, Murias G, et al. Effects of decreased respiratory frequency on ventilator-induced lung injury. *Am J Respir Crit Care Med.* 2000;161:463–468.
10. Simonson DA, Adams AB, Wright LA, Dries DJ, Hotchkiss JR, Marini JJ. Effects of ventilatory pattern on experimental lung injury caused by high airway pressure. *Crit Care Med.* 2004;32:781–786.
11. Gattinoni L, Tonetti T, Cressoni M, et al. Ventilator-related causes of lung injury: the mechanical power. *Intensive Care Med.* 2016;42:1567–1575.
12. Cressoni M, Gotti M, Chiurazzi C, et al. Mechanical power and development of ventilator-induced lung injury. *Anesthesiology.* 2016;124:1100–1108.
13. Serpa Neto A, Deliberato RO, Johnson AEW, et al; PROVE Network Investigators. Mechanical power of ventilation is associated with mortality in critically ill patients: an analysis of patients in two observational cohorts. *Intensive Care Med.* 2018;44:1914–1922.
14. Licker M, de Perrot M, Spiliopoulos A, et al. Risk factors for acute lung injury after thoracic surgery for lung cancer. *Anesth Analg.* 2003;97:1558–1565.
15. Gothard J. Lung injury after thoracic surgery and one-lung ventilation. *Curr Opin Anaesthesiol.* 2006;19:5–10.
16. Guttman J, Eberhard L, Fabry B, Bertschmann W, Wolff G. Continuous calculation of intratracheal pressure in tracheally intubated patients. *Anesthesiology.* 1993;79:503–513.
17. Formenti P, Umbrello M, Piva IR, et al. Drainage of pleural effusion in mechanically ventilated patients: time to measure chest wall compliance? *J Crit Care.* 2014;29:808–813.
18. Barnas GM, Sprung J, Choi D, Kahn R. Lung mechanical behavior during one-lung ventilation. *J Cardiothorac Vasc Anesth.* 1997;11:604–607.
19. Miskovic A, Lumb AB. Postoperative pulmonary complications. *Br J Anaesth.* 2017;118:317–334.
20. Nunn JF. Effects of anaesthesia on respiration. *Br J Anaesth.* 1990;65:54–62.
21. Rehder K, Mallow JE, Fibuch EE, Krabill DR, Sessler AD. Effects of isoflurane anesthesia and muscle paralysis on respiratory mechanics in normal man. *Anesthesiology.* 1974;41:477–485.

22. Brismar B, Hedenstierna G, Lundquist H, Strandberg A, Svensson L, Tokics L. Pulmonary densities during anesthesia with muscular relaxation—a proposal of atelectasis. *Anesthesiology*. 1985;62:422–428.
23. Larsson A, Malmkvist G, Werner O. Variations in lung volume and compliance during pulmonary surgery. *Br J Anaesth*. 1987;59:585–591.
24. Akoumianaki E, Maggiore SM, Valenza F, et al; PLUG Working Group (Acute Respiratory Failure Section of the European Society of Intensive Care Medicine). The application of esophageal pressure measurement in patients with respiratory failure. *Am J Respir Crit Care Med*. 2014;189:520–531.
25. Mauri T, Yoshida T, Bellani G, et al; PLeUral pressure working Group (PLUG—Acute Respiratory Failure section of the European Society of Intensive Care Medicine). Esophageal and transpulmonary pressure in the clinical setting: meaning, usefulness and perspectives. *Intensive Care Med*. 2016;42:1360–1373.
26. Pelosi P, Goldner M, McKibben A, et al. Recruitment and derecruitment during acute respiratory failure: an experimental study. *Am J Respir Crit Care Med*. 2001;164:122–130.
27. Wulff KE, Aulin I. The regional lung function in the lateral decubitus position during anesthesia and operation. *Acta Anaesthesiol Scand*. 1972;16:195–205.
28. Grimby G, Hedenstierna G, Löfström B. Chest wall mechanics during artificial ventilation. *J Appl Physiol*. 1975;38:576–580.
29. Hubmayr RD, Walters BJ, Chevalier PA, Rodarte JR, Olson LE. Topographical distribution of regional lung volume in anesthetized dogs. *J Appl Physiol Respir Environ Exerc Physiol*. 1983;54:1048–1056.
30. Washko GR, O'Donnell CR, Loring SH. Volume-related and volume-independent effects of posture on esophageal and transpulmonary pressures in healthy subjects. *J Appl Physiol (1985)*. 2006;100:753–758.
31. Ferris BG Jr, Mead J, Frank NR. Effect of body position on esophageal pressure and measurement of pulmonary compliance. *J Appl Physiol*. 1959;14:521–524.
32. Milic-Emili J, Mead J, Turner JM. Topography of esophageal pressure as a function of posture in man. *J Appl Physiol*. 1964;19:212–216.
33. Westbrook PR, Stubbs SE, Sessler AD, Rehder K, Hyatt RE. Effects of anesthesia and muscle paralysis on respiratory mechanics in normal man. *J Appl Physiol*. 1973;34:81–86.
34. Cressoni M, Gallazzi E, Chiurazzi C, et al. Limits of normality of quantitative thoracic CT analysis. *Crit Care*. 2013;17:R93.
35. Della Rocca G, Coccia C. Acute lung injury in thoracic surgery. *Curr Opin Anaesthesiol*. 2013;26:40–46.