

Effects of the Beach Chair Position, Positive End-expiratory Pressure, and Pneumoperitoneum on Respiratory Function in Morbidly Obese Patients during Anesthesia and Paralysis

Franco Valenza, M.D.,* Federica Vaggini, M.D.,† Alberto Tiby, M.D.,† Silvia Francesconi, M.D.,† Giulio Ronzoni, M.D.,† Massimiliano Guglielmi, M.D.,† Marco Zappa, M.D.,‡ Ezio Lattuada, M.D.,‡ Luciano Gattinoni, M.D., F.R.C.P.§

Background: The authors studied the effects of the beach chair (BC) position, 10 cm H₂O positive end-expiratory pressure (PEEP), and pneumoperitoneum on respiratory function in morbidly obese patients undergoing laparoscopic gastric banding.

Methods: The authors studied 20 patients (body mass index 42 ± 5 kg/m²) during the supine and BC positions, before and after pneumoperitoneum was instituted (13.6 ± 1.2 mmHg). PEEP was applied during each combination of position and pneumoperitoneum. The authors measured elastance (E_{rs}) of the respiratory system, end-expiratory lung volume (helium technique), and arterial oxygen tension. Pressure-volume curves were also taken (occlusion technique). Patients were paralyzed during total intravenous anesthesia. Tidal volume (10.5 ± 1 ml/kg ideal body weight) and respiratory rate (11 ± 1 breaths/min) were kept constant throughout.

Results: In the supine position, respiratory function was abnormal: E_{rs} was 21.71 ± 5.26 cm H₂O/l, and end-expiratory lung volume was 0.46 ± 0.1 l. Both the BC position and PEEP improved E_{rs} ($P < 0.01$). End-expiratory lung volume almost doubled (0.83 ± 0.3 and 0.85 ± 0.3 l, BC and PEEP, respectively; $P < 0.01$ vs. supine zero end-expiratory pressure), with no evidence of lung recruitment (0.04 ± 0.1 l in the supine and 0.07 ± 0.2 in the BC position). PEEP was associated with higher airway pressures than the BC position (22.1 ± 2.01 vs. 13.8 ± 1.8 cm H₂O; $P < 0.01$). Pneumoperitoneum further worsened E_{rs} (31.59 ± 6.73 ; $P < 0.01$) and end-expiratory lung volume (0.35 ± 0.1 l; $P < 0.01$). Changes of lung volume correlated with changes of oxygenation (linear regression, $R^2 = 0.524$, $P < 0.001$) so that during pneumoperitoneum, only the combination of the BC position and PEEP improved oxygenation.

Conclusions: The BC position and PEEP counteracted the major derangements of respiratory function produced by anesthesia and paralysis. During pneumoperitoneum, only the combination of the two maneuvers improved oxygenation.

A NUMBER of authors have shown that during anesthesia and paralysis, respiratory function in obese patients is greatly impaired,¹⁻⁴ and further worsened when laparoscopic procedures are performed.⁵⁻⁸

* Assistant Professor, Staff Anesthesiologist, † Staff Anesthesiologist, § Full Professor, Head of the Department, Istituto di Anestesia e Rianimazione, ‡ Staff Surgeon, Istituto di Chirurgia Generale.

Received from the Fondazione Ospedale Maggiore, Mangiagalli e Regina Elena-IRCCS, Milan, Italy. Submitted for publication January 11, 2007. Accepted for publication July 19, 2007. Support was provided solely from institutional and/or departmental sources. Presented at the annual meetings of the European Society of Anaesthesiology, Gothenburg, Sweden, April 7-10, 2001, and Nice, France, April 6-9, 2002.

Address correspondence to Dr. Valenza: Università degli Studi di Milano, Fondazione Ospedale Maggiore, Mangiagalli e Regina Elena-IRCCS, Via F. Sforza 35, 20122 Milano, Italy. franco.valenza@unimi.it. Information on purchasing reprints may be found at www.anesthesiology.org or on the masthead page at the beginning of this issue. ANESTHESIOLOGY's articles are made freely accessible to all readers, for personal use only, 6 months from the cover date of the issue.

Several strategies have been tested to improve oxygenation. Positive end-expiratory pressure (PEEP) has been shown to improve respiratory function⁹⁻¹⁸; however, it may cause relative hypotension.^{15,18-20} Large tidal volumes or a high respiratory rate have been tested with negative results such that the authors concluded that the only way to safely improve oxygenation is to increase the fraction of inspired oxygen (F_{IO₂}).⁸ The effects of body positioning on respiratory mechanics have also been investigated: The head-up position has been shown to be beneficial as opposed to the head-down position,^{7,21} even if this was not always the case.⁸

Apart from the investigation of Perilli *et al.*,¹⁵ most of the aforementioned studies investigated either position or PEEP, leaving the interested reader with an incomplete picture, particularly if considering the comparison between the two maneuvers and the effects of pneumoperitoneum on these maneuvers.

Moreover, the main endpoints of most studies were respiratory system compliance and oxygenation. However, Pelosi *et al.*²² clearly showed that end-expiratory lung volume decreases exponentially with the increase of body mass index, and that oxygenation correlates with lung volume.

Based on these considerations, we decided to investigate the effects of the beach chair position and PEEP on respiratory function in obese patients during anesthesia and paralysis, before and after intraabdominal pressure was increased with pneumoperitoneum, giving peculiar reference to lung volume, considered as the main outcome variable, and its correlation to oxygenation.

Materials and Methods

The ethics committee of our institution (Fondazione Ospedale Maggiore, Mangiagalli e Regina Elena-IRCCS, Milan, Italy) approved the study. Patients were fully informed that the protocol would imply an extension of the anesthesia time and were aware of the potential implications of the additional time of anesthesia and paralysis. Written informed consent was obtained from each patient.

Study Population

Twenty consecutive morbidly obese patients undergoing laparoscopic gastric banding were included. Patients

with heart failure (defined as New York Heart Association classification >2) or coronary disease and/or documented obstructive disease currently being treated were excluded from the study.

Anesthesia

On the morning of investigation, patients received midazolam (0.05 mg/kg, per os), atropine (1 mg, per os), metoclopramide (10 mg, endovenous), and nizatidine (100 mg, endovenous). Upon arrival in the theater, each patient was given 100% oxygen. After topical anesthesia was applied to the oropharynx (5% lidocaine), a continuous infusion of remifentanyl (0.1 μ g/kg ideal weight/min) was commenced, and tracheal intubation (cuffed tube 7-7.5; Portex, London, England, United Kingdom) was performed during spontaneous breathing and fiberoptic vision. Once the correct position of the tube was verified, anesthesia was induced with intravenous midazolam (0.154 \pm 0.036 mg/kg ideal weight), and muscle paralysis was induced with 0.1 mg/kg vecuronium bromide and maintained with subsequent boluses when needed. Thereafter, remifentanyl and midazolam were administered as a continuous infusion (0.2-0.4 μ g/kg ideal weight/min and 0.2-0.3 mg/kg ideal weight/h, respectively). Depth of anesthesia was assessed by clinical signs, and paralysis was assessed by ulnar nerve stimulation (train-of-four). At the end of the protocol, general anesthesia was maintained with sevoflurane.

Standard monitoring was used throughout the procedure, and the radial artery was cannulated for invasive blood pressure and blood gas monitoring.

Protocol Procedure

A schema of the protocol is summarized in figure 1. As shown, it included four different steps:

Supine position, before pneumoperitoneum was induced (step 1)

Beach chair position, before pneumoperitoneum was induced (step 2)

Supine position, after pneumoperitoneum was induced (step 3)

Beach chair position, after pneumoperitoneum was induced (step 4)

The beach chair position was obtained as a reverse Trendelenburg position (30° head-up) with the legs lifted to the abdomen.

At each step, zero end-expiratory pressure (ZEEP) or 10 cm H₂O PEEP (PEEP) was applied in a random fashion (closed envelopes).

Throughout the protocol, ventilator settings (Servo 900C; Siemens-Elema, Solna, Sweden) were maintained unchanged as follows: volume control mode with constant inspiratory flow (0.700 \pm 0.104 l/s), tidal volume of 10.5 \pm 1 ml/kg ideal body weight, respiratory rate of 11 \pm 1 breaths/min, inspiratory time of 33% with no pause, and 60% inspiratory oxygen fraction.

After each combination of position (supine, beach chair position), pneumoperitoneum (without, with), and PEEP (0, 10 cm H₂O) was set, a recruitment maneuver was performed (three consecutive inspiratory holds of 5 s at 45 cm H₂O airway pressure). Thereafter, 15 min of mechanical ventilation was allowed before the following measurements were taken:

Physiologic Measurements

Intraabdominal Pressure. Intraabdominal pressure was measured using a transurethral bladder catheter. Using a sterile technique, 100 ml normal saline was infused through the urinary catheter into the bladder. Intraabdominal pressure was recorded as mean pressure at end-expiration. The zero was set at the level of the pubis.

Pneumoperitoneum was generated by insufflating carbon dioxide into the abdomen (40-l High flow insufflator; Stryker Endoscopy, San Jose, CA). Displayed pressures were recorded.

End-expiratory Lung Volume. The end-expiratory lung volume was measured using the closed-circuit helium dilution method.

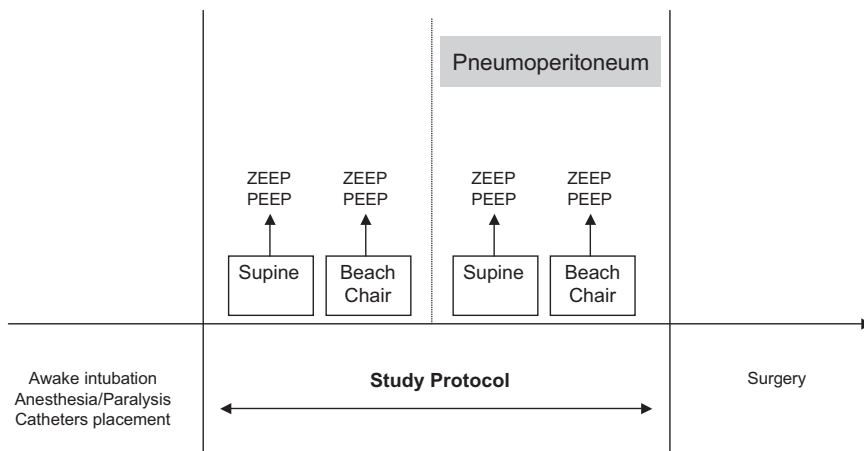


Fig. 1. A scheme of the protocol procedure. PEEP = 10 cm H₂O positive end-expiratory pressure; ZEEP = zero end-expiratory pressure.

Respiratory Mechanics. Airway pressure (P_{aw}) and gas flow were measured at the endotracheal tube opening, while esophageal pressure (P_{es}) was measured from an esophageal balloon (Bicore CP-100; Irvine, CA) inflated with 0.5–1 ml of air, positioned at the lower third of the esophagus. The validity of P_{es} was verified according to Baydur *et al.*²³ Because the patients were paralyzed, the airway and esophageal pressure were altered by compressing the thorax with the airways occluded at end-expiration. This method was previously used to partition respiratory mechanics in obese patients.²⁴ Both flow and pressure signals were recorded on a personal computer *via* an analog-to-digital converter (Colligo; Elektor, Milan, Italy) at a sample rate of 200 Hz and stored for subsequent computer analysis.

We recorded P_{aw} and P_{es} during 4–5 s of airway occlusions at end-expiration followed by an occlusion at end-inspiration. Static elastance of the total respiratory system (E_{rs}) was computed as $\Delta P_{aw}/V_T$, where ΔP_{aw} is the difference between plateau end-inspiratory and end-expiratory airway pressure (corrected for intrinsic PEEP) and V_T is the tidal volume. Static elastance of the chest wall (E_{cw}) was computed as $\Delta P_{es}/V_T$, where ΔP_{es} is the difference between plateau end-inspiratory and end-expiratory esophageal pressure. Static lung elastance (E_l) was calculated as $E_l = E_{rs} - E_{cw}$.

Resistances of the Respiratory System, Chest Wall, and Lung. Maximum ($R_{max,rs}$) and minimum ($R_{min,rs}$) resistances of the respiratory system were computed as $(P_{aw,max} - P_2)/\text{Flow}$ and $(P_{aw,max} - P_1)/\text{Flow}$, where $P_{aw,max}$ is the maximal pressure at end-inspiration after the occlusion maneuver, P_1 is the pressure of the airway immediately after the sudden decrease from $P_{aw,max}$, and P_2 is the plateau pressure measured at the end of the occlusion. The minimum resistance of the respiratory system represents the so-called ohmic component, whereas the maximum includes ohmic and resistances caused by stress relaxation or resistances caused by time constant inequalities. These are called “additional” resistances (D_{rs}) and are calculated as $R_{max,rs} - R_{min,rs}$.

There was no appreciable decrease of esophageal pressure after the occlusion (P_1), suggesting that minimum chest wall resistances were negligible, so that chest wall resistances may be considered entirely due to viscoelastic properties of the chest wall (DR_{cw}). Finally, maximum (R_{L}), minimum ($R_{min,L}$), and additional (DR_{L}) resistances of the lung were obtained by using transpulmonary pressures, *i.e.*, airway minus esophageal pressures.

Static Inflation Pressure–Volume Curves and Alveolar Recruitment

Static pressure–volume (P–V) curves were measured at 0 and 10 cm H₂O PEEP by performing intermittently a series of end-inspiratory airway occlusions at different inflation volumes that ranged between 0.1 and 1.0 l, in

100-ml steps, and recording the P_{aw} and P_{es} at end-expiration and after each end-inspiration. End-expiratory volume corresponded to the elastic equilibrium volume in each patient, as evidenced by zero flow during expiratory pause and absence of changes in P_{aw} after airway occlusion.

To estimate alveolar recruitment, we first computed the difference of gas volume measured with helium at 10 and 0 cm H₂O pressure (actual Δ gas volume). We then computed on the P–V curve, starting at 0 cm H₂O PEEP, the volume expected at 10 cm H₂O (expected Δ gas volume), *i.e.*, $V = 10/a^{(1/b)}$. We defined alveolar recruitment as the difference between the actual Δ gas volume and the expected Δ gas volume. Any positive difference implies recruitment (*i.e.*, upward shift of the P–V curve).

Gas Exchange

Arterial blood samples were taken from the radial artery and analyzed for arterial partial pressure of oxygen (P_{aO_2}) and arterial partial pressure of carbon dioxide (P_{aCO_2}) (IL 1312 BGM; Instrumentation Laboratory Company, Lexington, MA). Alveolar–arterial difference was then calculated with standard formulas to correct for hypercapnia.

The physiologic dead space fraction (V_D/V_T) was also computed according to the following formula: $V_D/V_T = (P_{aCO_2} - P_{ECO_2})/P_{aCO_2}$, where P_{ECO_2} is the mixed expired carbon dioxide partial pressure. This was obtained by means of continuous expiratory air sampling (CO₂SMO PLUS 8100; Novamatrix Medical System Inc., Wallingford, CT).

Hemodynamics

Blood pressure was measured invasively through a radial catheter. Heart rate was derived from the electrocardiogram.

Statistics

The study was designed to enroll 20 patients, providing a power of 0.8 with a minimum detectable difference of the means of end-expiratory lung volume of 0.2 l, an expected SD of 0.1 l, and an α value of 0.01. The mean value of three breaths was used for pressure and flow variables at each experimental condition. We used analysis of variance for repeated measures to test the effects of the beach chair position and PEEP with or without pneumoperitoneum, considering steps (see Materials and Methods, Protocol Procedure) and PEEP as entry into the model. The Bonferroni correction was applied for multiple comparisons. Multiple linear regression analysis was conducted to correlate changes of oxygenation (P_{aO_2}) to changes of end-expiratory lung volume.^{25,26} Statistical significance was accepted as $P < 0.05$. Analysis was performed with the SAS System for Windows version 9.1 (Cary, NC).

Table 1. Effect of Beach Chair Position and PEEP without Pneumoperitoneum

| | Supine | | Beach Chair | |
|--|--------------|---------------|---------------|----------------|
| | ZEEP | PEEP | ZEEP | PEEP |
| IAP, cm H ₂ O | 17.87 ± 5.45 | 19.23 ± 5.22 | 23.92 ± 4.35* | 25.63 ± 4.25*† |
| P _{aw} , cm H ₂ O | 19.01 ± 3.08 | 22.11 ± 2.01* | 13.80 ± 1.8*† | 21.65 ± 1.69*† |
| PEEPi, cm H ₂ O | 3.38 ± 3.18 | 0.51 ± 0.36* | 0.62 ± 0.46* | 0.44 ± 0.23* |
| E _{rs} , cm H ₂ O/l | 21.71 ± 5.26 | 16.28 ± 3.48* | 18.05 ± 3.60* | 15.64 ± 3.10* |
| E _l , cm H ₂ O/l | 13.84 ± 4.83 | 9.75 ± 3.21 | 11.63 ± 4.01 | 9.94 ± 3.18 |
| E _{cw} , cm H ₂ O/l | 7.87 ± 2.42 | 6.53 ± 1.92 | 6.42 ± 2.50 | 5.70 ± 2.11 |
| R _{max,rs} , ml · cm H ₂ O ⁻¹ · l ⁻¹ · s ⁻¹ | 15.6 ± 1.1 | 14.8 ± 2.0 | 15.1 ± 1.8 | 14.0 ± 2.3 |
| R _{min,rs} , ml · cm H ₂ O ⁻¹ · l ⁻¹ · s ⁻¹ | 10.6 ± 1.4 | 10.2 ± 2.0 | 10.2 ± 1.3 | 9.5 ± 1.1 |
| DR _{rs} , ml · cm H ₂ O ⁻¹ · l ⁻¹ · s ⁻¹ | 5.0 ± 1.0 | 4.5 ± 1.1 | 4.8 ± 1.3 | 4.5 ± 1.8 |
| PaO ₂ , mmHg | 177 ± 50 | 201 ± 50 | 203 ± 51* | 243 ± 45*†‡ |
| Paco ₂ , mmHg | 38.3 ± 5.2 | 38.6 ± 5.0 | 38.9 ± 5.2 | 37.8 ± 5.0 |
| VD/VT | 0.40 ± 0.08 | 0.39 ± 0.07 | 0.37 ± 0.07 | 0.40 ± 0.09 |
| MAP, mmHg | 78 ± 9 | 75 ± 11 | 72 ± 10* | 71 ± 9 |
| HR, beats/min | 69 ± 12 | 66 ± 12 | 69 ± 13 | 67 ± 12 |

* $P < 0.01$ vs. supine ZEEP. † $P < 0.01$ vs. supine PEEP. ‡ $P < 0.01$ vs. beach chair ZEEP.

DR_{rs} = additional airway resistance; E_{cw} = chest wall elastance; E_l = lung elastance; E_{rs} = respiratory system elastance; HR = heart rate; IAP = intraabdominal pressure; MAP = mean arterial blood pressure; Paco₂ = arterial partial pressure of carbon dioxide; PaO₂ = arterial partial pressure of oxygen; P_{aw} = plateau airway pressure; PEEP = 10 cm H₂O positive end-expiratory pressure; PEEPi = intrinsic PEEP; R_{max,rs} = maximum airway resistance; R_{min,rs} = minimum airway resistance; V_D/V_T = physiologic dead space; ZEEP = zero end-expiratory pressure.

Results

Study Population

Twenty of 21 patients approached for consent for this study accepted. The 20 consented patients (5 men, 15 women) were aged 37 ± 10 yr, and their body mass index was 42 ± 5 kg/m². The forced vital capacity and forced expired volume in 1 s before surgery were $92 \pm 16.5\%$ and $91 \pm 14.6\%$, respectively. Gas exchange parameters were within normal ranges.

Protocol Procedure

The protocol lasted approximately 160 min. No problems occurred during this time or during the following time needed for surgery. None of the patients experienced delayed awakening at the end of the surgical procedure or had to be reintubated postoperatively. The postoperative course was unremarkable.

Respiratory Mechanics in the Supine Position

As shown in table 1 (column supine, ZEEP) and figure 2, during sedation and paralysis in the supine position, the obese patients we investigated were characterized by increased intraabdominal pressure, low end-expiratory lung volumes (0.46 ± 0.1 l), and increased elastance of both lung and chest wall. Maximum and additional resistances of the respiratory system were high, the increase being mainly due to lung resistances.

Overall respiratory mechanics worsened after pneumoperitoneum was induced, as shown in table 2. Lung volume was as low as 0.35 ± 0.1 l; respiratory system elastance further worsened compared with baseline ($P < 0.001$), mainly because of chest wall elastance impairment ($P < 0.001$). Airway resistance worsened, even if not significantly ($P = 0.0745$), and intrinsic PEEP

was higher ($P = 0.0075$). Oxygenation improved ($P = 0.0057$). Carbon dioxide tension ($P < 0.001$) and mean arterial pressure ($P < 0.001$) were significantly higher during pneumoperitoneum. The P-V curve of the respiratory system was shifted rightward during pneumoperitoneum as compared with baseline (fig. 3A).

Effect of the Beach Chair Position and PEEP before Pneumoperitoneum Was Induced

As shown in table 1 and figure 2, the beach chair position compared with the supine position improved lung volume (0.85 ± 0.3 l; $P < 0.001$) and respiratory system elastance ($P = 0.0122$). Airway resistances were not affected ($P = 0.1079$). Gas exchange improved during the beach chair position ($P = 0.0468$). Mean arterial pressure decreased during the beach chair position ($P = 0.0134$), whereas heart rate ($P = 1.00$) was unaffected.

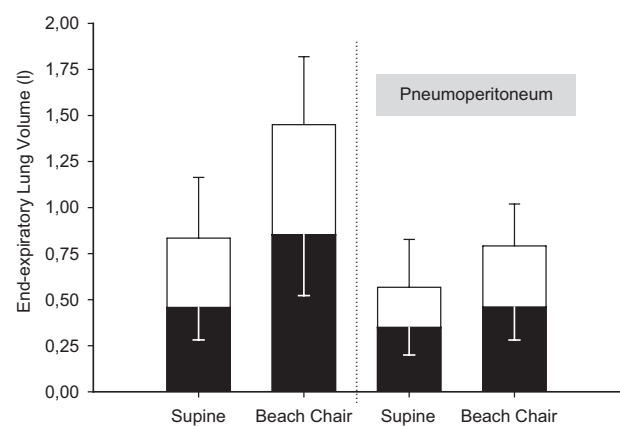


Fig. 2. The effects of the beach chair position and positive end-expiratory pressure (PEEP) on end-expiratory lung volumes. **Black columns** represent values at zero end-expiratory pressure; **white columns** represent values at 10 cm H₂O PEEP.

Table 2. Effect of Beach Chair Position and PEEP after Pneumoperitoneum Was Induced

| | Supine | | Beach Chair | |
|--|---------------|---------------|----------------|-----------------|
| | ZEEP | PEEP | ZEEP | PEEP |
| IAP, cm H ₂ O | 26.28 ± 3.92§ | 26.95 ± 7.20 | 30.00 ± 5.80* | 30.79 ± 5.29* |
| P _{aw} , cm H ₂ O | 27.52 ± 4.00§ | 29.01 ± 2.61 | 20.70 ± 2.66*† | 24.64 ± 2.37†‡ |
| PEEPi, cm H ₂ O | 5.03 ± 3.98§ | 0.99 ± 0.65* | 2.47 ± 2.24*† | 0.82 ± 0.40* |
| E _{rs} , cm H ₂ O/l | 31.59 ± 6.73§ | 25.52 ± 5.79* | 24.97 ± 5.10* | 19.22 ± 4.38*†‡ |
| E _l , cm H ₂ O/l | 16.11 ± 5.87 | 11.69 ± 4.08 | 13.91 ± 5.78 | 9.92 ± 4.18 |
| E _{cw} , cm H ₂ O/l | 15.49 ± 4.34§ | 13.83 ± 4.42 | 11.06 ± 3.82* | 9.29 ± 3.04*† |
| R _{max,rs} , ml · cm H ₂ O ⁻¹ · l ⁻¹ · s ⁻¹ | 18.4 ± 4.2 | 17.1 ± 3.2 | 18.1 ± 3.3 | 16.1 ± 1.9 |
| R _{min,rs} , ml · cm H ₂ O ⁻¹ · l ⁻¹ · s ⁻¹ | 12.5 ± 3.3 | 11.4 ± 3.5 | 11.8 ± 1.8 | 11.2 ± 1.5 |
| DR _{rs} , ml · cm H ₂ O ⁻¹ · l ⁻¹ · s ⁻¹ | 5.9 ± 3.0 | 5.7 ± 2.4 | 6.3 ± 2.2 | 4.9 ± 1.6 |
| PaO ₂ , mmHg | 203 ± 51§ | 205 ± 55 | 206 ± 51 | 223 ± 51§ |
| Paco ₂ , mmHg | 44.2 ± 7.0§ | 45.2 ± 7.8 | 47.9 ± 8.8*† | 48.0 ± 9.7*† |
| V _D /V _T | 0.41 ± 0.08 | 0.42 ± 0.07 | 0.40 ± 0.07 | 0.41 ± 0.07 |
| MAP, mmHg | 109 ± 14§ | 99 ± 17 | 110 ± 13 | 103 ± 12 |
| HR, beats/min | 73 ± 16 | 70 ± 14 | 82 ± 14 | 80 ± 15 |

* $P < 0.01$ vs. supine ZEEP. † $P < 0.01$ vs. supine PEEP. ‡ $P < 0.01$ vs. beach chair ZEEP. § $P < 0.01$, before vs. after pneumoperitoneum within supine ZEEP.

DR_{rs} = additional airway resistance; E_{cw} = chest wall elastance; E_l = lung elastance; E_{rs} = respiratory system elastance; HR = heart rate; IAP = intraabdominal pressure; MAP = mean arterial blood pressure; Paco₂ = arterial partial pressure of carbon dioxide; PaO₂ = arterial partial pressure of oxygen; P_{aw} = plateau airway pressure; PEEP = 10 cm H₂O positive end-expiratory pressure; PEEPi = intrinsic PEEP; R_{max,rs} = maximum airway resistance; R_{min,rs} = minimum airway resistance; V_D/V_T = physiologic dead space; ZEEP = zero end-expiratory pressure.

The use of 10 cm H₂O PEEP improved lung volume (0.83 ± 0.3 l, $P = 0.0014$) and respiratory system elastance ($P < 0.001$). There was no significant effect on airway resistance ($P = 0.1133$), oxygenation ($P = 1.000$), mean arterial pressure ($P = 0.3596$), or heart rate ($P = 1.00$).

Between the beach chair position and PEEP, there were no differences of end-expiratory lung volume ($P = 1.00$), oxygenation (PaO₂; $P = 1.00$), respiratory system elastance ($P = 1.00$), or mean arterial pressure ($P = 0.2359$). Airway pressure was significantly higher during PEEP application ($P < 0.001$).

The P-V curves of the respiratory system before pneumoperitoneum was induced are shown in figure 3. The curves at ZEEP and PEEP in both positions were almost superimposed. The alveolar recruitment was 0.04 ± 0.1 l in the supine and 0.07 ± 0.2 l in the beach chair position; values are not significantly different (supine vs. beach chair position, $P = 0.47$).

Effect of the Beach Chair Position and PEEP after Pneumoperitoneum Was Induced

As shown in table 2 and figure 2, during pneumoperitoneum, the beach chair position improved lung volume (0.45 ± 0.2 l; $P = 0.0177$) and respiratory system elastance ($P < 0.001$), mainly because of changes in chest wall elastance ($P = 0.0002$). Mean arterial pressure was unaffected ($P = 0.7852$). Similarly, PEEP improved lung volume (0.55 ± 0.1 l; $P = 0.003$) and respiratory system elastance ($P = 0.0132$), with no effect on mean arterial pressure ($P = 1.00$). Respiratory system resistances were unaffected by both the beach chair position ($P = 1.00$) and PEEP ($P = 0.6805$). During pneumoperitoneum,

neither the beach chair position ($P = 1.00$) nor PEEP ($P = 1.00$) led to significant changes of oxygenation.

The P-V curves of the respiratory system after pneumoperitoneum was induced are shown in figure 3. As described above, the slopes of the curves were lower than before pneumoperitoneum was induced; however, the behavior was similar. In fact, curves at ZEEP and PEEP in both positions were almost superimposed. The alveolar recruitment was -0.014 ± 0.111 l in the supine and 0.04 ± 0.127 l in the beach chair position; values were not significantly different (supine vs. beach chair position, $P = 0.273$).

End-expiratory Lung Volume and Oxygenation

Changes of oxygenation (calculated with supine ZEEP values as a reference) correlated with changes of end-expiratory lung volume (fig. 4; $P < 0.001$, $R^2 = 0.524$).

Discussion

The aim of this study was to assess the effects of the beach chair position, PEEP, and pneumoperitoneum on respiratory mechanics in morbidly obese patients during anesthesia and paralysis. The main results were that both the beach chair position and PEEP significantly improved lung volumes and respiratory mechanics. Each maneuver individually improved oxygenation at baseline, whereas the combination of the two was needed during pneumoperitoneum.

Respiratory Mechanics in the Supine Position

Our results confirm previous observations that obese patients sustain a major derangement of respiratory func-

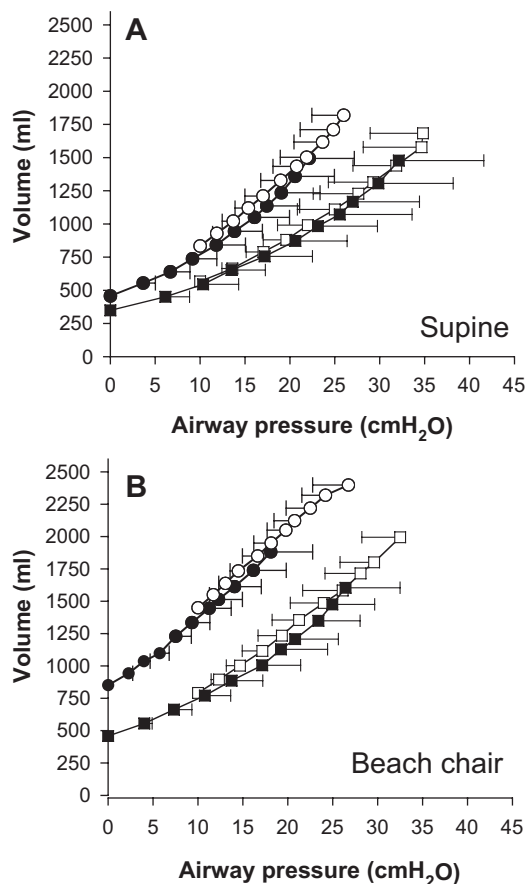


Fig. 3. Pressure–volume curves taken in the supine position (A) and in the beach chair position (B). In each panel, values taken at baseline are shown as *circles*, and those obtained during pneumoperitoneum are shown as *squares*. *Closed symbols* indicate values at zero end-expiratory pressure, and *open symbols* indicate those at 10 cm H₂O positive end-expiratory pressure. To compare curves, each one starts on the y-axis from the volume corresponding to the mean value of end-expiratory lung volume measured by helium dilution technique.

tion during anesthesia and paralysis.^{1–4} In fact, end-expiratory lung volume measured in supine position was as low as 0.5 l, in accordance with the body mass index and end-expiratory lung volume relation described by Pelosi *et al.*²² Elastance and airway resistance were greater than normal, and there was hypoxemia relative to inspiratory fraction of oxygen.

As suggested by the P-V curve analysis (fig. 3A), we did not find evidence of recruitable lung tissue. This is in contrast to the previous report by Pelosi *et al.*¹³ However, the values of body mass index of our patients were lower than those in the study of Pelosi *et al.* (42 ± 5 as opposed to 51 ± 8 kg/m²). We took our measurements soon after induction of anesthesia, whereas Pelosi *et al.* investigated patients at the end of surgery and after transfer from the operating room to the intensive care unit. Moreover, we performed frequent recruitment maneuvers to normalize lung volumetric history. This likely contributed to our results. Whalen *et al.*¹⁸ recently showed that recruitment maneuvers may be an effective

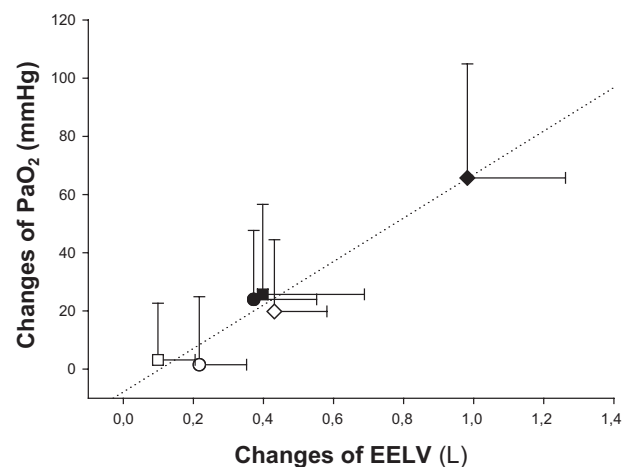


Fig. 4. Correlation between changes of end-expiratory lung volume (EELV) and changes of oxygenation (arterial partial pressure of oxygen [PaO₂]). Changes were calculated considering supine zero end-expiratory pressure values as a reference. *Open symbols* refer to values taken during pneumoperitoneum, and *closed symbols* refer to those at baseline. *Squares* represent values obtained during the beach chair position, *circles* represent values at 10 cm H₂O positive end-expiratory pressure, and *diamonds* represent values obtained with the combination of the beach chair position and 10 cm H₂O positive end-expiratory pressure. Multiple regression analysis was conducted (see Materials and Methods, Statistics).

mode of improving intraoperative respiratory mechanics and oxygenation in obese patients.

Therefore, the decrease of lung volume and increase of lung elastance in the supine position may be explained by a prevalent decrease of the size of the alveoli rather than atelectasis. Of note is the fact that in the supine position, some grade of intrinsic PEEP developed. This may be due to flow limitation, as suggested by Pankov *et al.*²⁷

Pneumoperitoneum further worsened respiratory mechanics, as previously shown in both normal-weight^{28–35} and obese^{5–8} patients.

Interestingly, oxygenation improved during pneumoperitoneum, possibly because of the effects of pneumoperitoneum on hemodynamics. Pneumoperitoneum, in fact, activates sympathetic tone and contributes to increase arterial blood pressure.⁵ The fact that pneumoperitoneum has been shown to greatly affect venous return, particularly in volume-depleted subjects, and the overall hemodynamic stability of our patients may suggest that they were not hypovolemic. This notion could not be substantiated in the current investigation.

Effects of the Beach Chair Position and PEEP on Respiratory Mechanics

Several investigations have considered the effects of head-up position on either normal-weight³⁶ or obese^{6–8,21} patients. We measured lung volumes and found that these almost doubled during the beach chair position. The effect of bowels sliding under gravity and relieving the diaphragm is possibly relevant, as suggested by the increase of bladder

pressure in the beach chair as compared with the supine position. The results of the effects of PEEP were consistent with previous reports.⁹⁻¹⁸

A positive feature of this study was the investigation of the relative effects of the beach chair position and PEEP: It was intriguing to find that the two maneuvers produced similar effects on lung volume, respiratory system elastance, or oxygenation. However, similarly to laparotomy surgery,¹⁵ airway pressures were much lower during the beach chair position than during PEEP application. Therefore, under the perspective of lung-protective ventilation strategies,³⁷ the beach chair position would be preferred.

One must consider, however, that recruitable atelectasis, if any, was negligible (fig. 3): The picture might be different with lung collapse, hence with sicker patients. In this light, the recruitment maneuvers we frequently performed must be strongly considered. Whalen *et al.*¹⁸ have in fact shown that recruitment maneuvers improve oxygenation and respiratory mechanics as long as lungs are ventilated and PEEP is applied.

The combination of the beach chair position and PEEP improved lung volume before pneumoperitoneum was instituted. However, at high lung volumes, the P-V curve began to flatten, suggesting overdistention of the alveoli. Although there was no hemodynamic compromise in that state, we would suggest that this condition is not desirable *per se*.

On the contrary, during pneumoperitoneum, this did not occur, possibly because of the low starting volumes in this condition. Interestingly, whereas PEEP induced some de-recruitment in the supine position (as evidenced from a negative value of calculated lung recruitment and from P-V curves in fig. 3), if anything, there was some recruitment during the beach chair position.

Of note is the fact that during pneumoperitoneum, only the combination of the beach chair position and PEEP led to a significant lung volume increase and oxygenation improvement, whereas the single maneuvers did not (fig. 4). This is partly in contrast to the results of Sprung *et al.*,⁸ who found that arterial oxygenation was not affected by body position, pneumoperitoneum, or mode of ventilation. However, the same group of authors recently showed that recruitment maneuvers and PEEP are effective on lung mechanics and oxygenation, even if short-lived.¹⁸ Therefore, it is possible that, given the high abdominal pressure during pneumoperitoneum application, only the combination of the beach chair position and the recruitment maneuver followed by PEEP was able to counteract the detrimental effects of pneumoperitoneum. Accordingly, low PEEP may be insufficient to counteract pneumoperitoneum. The optimum PEEP is ideally achieved by titrating the "best" PEEP, recognizing that our data and others¹⁵ suggest that 10 cm H₂O may be reasonable.

Of note is the fact that changes of lung volume correlated with changes of oxygenation, further underlying

the crucial role of end-expiratory lung volume in these patients.

Limitations of the Study

The sequence of measurements, because of technical difficulties, was randomized only for PEEP application within each step of the protocol (fig. 1). This is a major limitation. However, we do not think that the protocol sequence influenced the results of lung volume, respiratory mechanics, or resistances. Carbon dioxide tension was of course affected by the protocol design provided respiratory rate was kept constant and pneumoperitoneum generated insufflating carbon dioxide into the abdomen. However, we corrected oxygenation for this possible bias by calculating the alveolar to arterial difference and did not find significant differences with Pao₂.

The rather short time between steps (approximately 15 min) is another limitation. This time was chosen because of the many measurements to be taken. However, we believe that 15 min was enough to reset respiratory mechanics at each step of the protocol.

The technique we used to estimate end-expiratory lung volume might have underestimated lung volumes because of air trapping at low lung volumes. However, the same technique has been used in the past.¹³ Moreover, in 10 patients, we also measured end-expiratory lung volume by the release technique (measuring flow while releasing PEEP, then calculating volume as integral of flow tracing): The values obtained with the two techniques were correlated ($R^2 = 0.793$, $P < 0.001$), suggesting that gas trapping (that would have appeared with the release technique) was negligible (intercept 0.098 l).

Conclusion

We have shown that the beach chair position and PEEP may be used to counteract the major derangements produced by anesthesia and paralysis in morbidly obese patients. The beach chair position and PEEP each similarly improved lung volume, oxygenation, and respiratory mechanics at baseline. However, during pneumoperitoneum, only the combination of the two improved oxygenation.

References

1. Sharp J, Henry J, Sweany S, Meadows W, Pietras R: Effects of mass loading the respiratory system in man. *J Appl Physiol* 1964; 19:959-65
2. Waltemath CL, Bergman NA: Respiratory compliance in obese patients. *ANESTHESIOLOGY* 1974; 41:84-5
3. Luce JM: Respiratory complications of obesity. *Chest* 1980; 78:626-31
4. Damia G, Mascheroni D, Croci M, Tarenzi L: Perioperative changes in functional residual capacity in morbidly obese patients. *Br J Anaesth* 1988; 60:574-8
5. Dumont L: Changes in pulmonary mechanics during laparoscopic gastroplasty in morbidly obese patients. *Acta Anaesthesiol Scand* 1997; 41:408-13
6. Casati G, Comotti L, Tommasino C, Leggieri E, Bignami F, Tarantino F, Torri G: Effects of pneumoperitoneum and reverse Trendelenburg position on cardio-

- pulmonary function in morbidly obese patients receiving laparoscopic gastric banding. *Eur J Anaesth* 2000; 17:300-5
7. Perilli V, Sollazzi L, Bozza P, Modesti C, Chierichini A, Tacchino RM, Ranieri R: The effects of the reverse Trendelenburg position on respiratory mechanics and blood gases in morbidly obese patients during bariatric surgery. *Anesth Analg* 2000; 91:1520-5
 8. Sprung J, Whalley D, Falcone T, Wilks W, Navratil J, Bourke D: The effects of tidal volume and respiratory rate on oxygenation and respiratory mechanics during laparoscopy in morbidly obese patients. *Anesth Analg* 2003; 97:268-74
 9. Hedenstierna G, Santesson J: Breathing mechanics, dead space and gas exchange in the extremely obese, breathing spontaneously and during anaesthesia with intermittent positive pressure ventilation. *Acta Anaesthesiol Scand* 1976; 20:248-54
 10. Santesson J: Oxygen transport and venous admixture in extremely obese: Influence of anaesthesia and artificial ventilation with and without positive end-expiratory pressure. *Acta Anaesthesiol Scand* 1976; 20:387-94
 11. Eriksen J, Andersen J, Rasmussen J, Sorensen B: Effects of ventilation with large tidal volumes or positive end-expiratory pressure on cardiorespiratory function in anesthetized obese patients. *Acta Anaesthesiol Scand* 1978; 22:241-8
 12. Tokics L, Hedenstierna G, Strandberg A, Brismar B, Lundquist H: Lung collapse and gas exchange during general anesthesia: Effects of spontaneous breathing, muscle paralysis, and positive end-expiratory pressure. *ANESTHESIOLOGY* 1987; 66:157-67
 13. Pelosi P, Ravagnan I, Giurati Panigada M, Bottino N: Positive end-expiratory pressure improves respiratory function in obese patients but not in normal subjects during anaesthesia and paralysis. *ANESTHESIOLOGY* 1999; 91:1221-31
 14. Yoshino J, Akata T, Takahashi S: Intraoperative changes in arterial oxygenation during volume-controlled mechanical ventilation in modestly obese patients undergoing laparotomies with general anesthesia. *Acta Anaesthesiol Scand* 2003; 47:742-50
 15. Perilli V, Sollazzi L, Modesti C, Annetta M, Sacco T, Bocci M, Tacchino R, Proietti R: Comparison of positive end-expiratory pressure with reverse Trendelenburg position in morbidly obese patients undergoing bariatric surgery: Effects on hemodynamics and pulmonary gas exchange. *Obes Surg* 2003; 13: 605-9
 16. Chalhoub V, Yazigi A, Sleilaty G, Haddad F, Noun R, Medi-Jebara S, Yazebeck P: Effect of vital capacity manoeuvres on arterial oxygenation in morbidly obese patients undergoing bariatric surgery. *Eur J Anaesth* 2006; 7:1-6
 17. Erlandsson K, Odenstedt H, Lundin S, Stenquist O: Positive end-expiratory pressure optimization using electric impedance tomography in morbidly obese patients during laparoscopic gastric bypass surgery. *Acta Anaesthesiol Scand* 2006; 50:833-9
 18. Whalen FX, Gajic O, Thomson GB, Kendrick ML, Que FL, Williams BA, Joyner MJ, Hubmayr RD, Warner DO, Sprung J: The effects of the alveolar recruitment maneuver and positive end-expiratory pressure on arterial oxygenation during laparoscopic bariatric surgery. *Anesth Analg* 2006; 102:298-305
 19. Moffa S, Quinn J, Slotman G: Hemodynamic effects of carbon dioxide pneumoperitoneum during mechanical ventilation and positive end-expiratory pressure. *J Trauma* 1993; 35:613-7
 20. Kraut EJ, Amira S, Barbosa R, Wolf BM: Impairment of cardiac performance by laparoscopy in patients receiving positive end-expiratory pressure. *Arch Surg* 1999; 134:76-80
 21. Boyce JR, Ness T, Castroman P, Gleysteen JJ: A preliminary study of the optimal anesthesia positioning for the morbidly obese patient. *Obes Surg* 2003; 13:4-9
 22. Pelosi P, Ravagnan I, Croci M, Tredici S, Pedoto A, Lissoni A, Gattinoni L: The effects of body mass on lung volumes, respiratory mechanics, and gas exchange during general anesthesia. *Anesth Analg* 1998; 87:654-60
 23. Baydur A, Behrakis PK, Zin WA, Jaeger M, Milic-Emili L: A simple method for assessing the validity of the esophageal balloon technique. *Am Rev Respir Dis* 1983; 126:788-91
 24. Pelosi P, Croci M, Ravagnan I, Vicardi P, Gattinoni L: Total respiratory system, lung, and chest wall mechanics in sedated-paralyzed postoperative morbidly obese patients. *Chest* 1996; 109:144-51
 25. Bland J, Altman D: Statistics notes: Correlation, regression and repeated data. *BMJ* 1994; 308:896
 26. Bland J, Altman D: Statistics notes: Calculating correlation coefficients within repeated observations: 1. Correlation within subjects. *BMJ* 1995; 310:446
 27. Pankow W, Podszus T, Gutheil T, Penzel T, Peter J, Von Wichert P: Expiratory flow limitation and intrinsic positive end-expiratory pressure in obesity. *J Appl Physiol* 1998; 85:1236-43
 28. Drummond M: Pressure-volume relationships in the lung during laparoscopy. *Br J Anaesth* 1978; 50:261-70
 29. Erice F, Fox GS, Salib YM, Romano E, Meakins JL, Magder SA: Diaphragmatic function before and after laparoscopic cholecystectomy. *ANESTHESIOLOGY* 1993; 79:966-75
 30. Fahy BG, Barnas GM, Flowers JL, Nagle SE, Njoku MJ: The effects of increased abdominal pressure on lung and chest wall mechanics during laparoscopic surgery. *Anesth Analg* 1995; 81:744-50
 31. Oikkonen M, Tallgren M: Changes in respiratory compliance at laparoscopy: Measurements using side stream spirometry. *Can J Anaesth* 1995; 42:495-7
 32. Fahy BG, Barnas GM, Nagle SE, Flowers JL, Njoku MJ, Agarwal M: Changes in lung and chest wall properties with abdominal insufflation of carbon dioxide are immediately reversible. *Anesth Analg* 1996; 82:501-5
 33. Chassard D, Berrada KR, Tournadre JP, Bouletreau P: The effect of increase in end-tidal carbon dioxide on lower esophageal sphincter tone. *Anesth Analg* 1996; 82:374-6
 34. Pelosi P, Foti G, Cereda M, Vicardi P, Gattinoni L: Effects of carbon dioxide insufflation for laparoscopic cholecystectomy on the respiratory system. *Anaesthesia* 1996; 51:744-9
 35. Carry PY, Gallet D, Francois Y, Perdrix JP, Sayag A, Gilly F, Eberhard A, Banssillon V, Baconnier P: Respiratory mechanics during laparoscopic cholecystectomy: The effects of the abdominal wall lift. *Anesth Analg* 1998; 87:1393-7
 36. Odeberg S, Ljungqvist O, Svenberg T, Gannedahl P, Backdahl M, von Rosen A, Sollevi A: Haemodynamic effects of pneumoperitoneum and the influence of posture during anaesthesia for laparoscopic surgery. *Acta Anaesthesiol Scand* 1994; 38:276-83
 37. Gattinoni L, Carlesso E, Cadringer P, Valenza F, Vaginelli F, Chiumello D: Physical and biological triggers of ventilator-induced lung injury and its prevention. *Eur Respir J Suppl* 2003; 43:158-25