Intraoperative Recruitment Maneuver Reverses Detrimental Pneumoperitoneum-induced Respiratory Effects in Healthy Weight and Obese Patients Undergoing Laparoscopy

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ABSTRACT

Background: Pulmonary function is impaired during pneumoperitoneum mainly as a result of atelectasis formation. We studied the effects of 10 cm H2O of positive end-expiratory pressure (PEEP) and PEEP followed by a recruitment maneuver (PEEP+RM) on end-expiratory lung volume (EELV), oxygenation and respiratory mechanics in patients undergoing laparoscopic surgery.

Methods: Sixty consecutive adult patients (30 obese, 30 healthy weight) in reverse Trendelenburg position were prospectively studied. EELV, static elastance of the respiratory system, dead space, and gas exchange were measured before and after pneumoperitoneum insufflation with zero end-expiratory pressure, with PEEP alone, and with PEEP+RM. Results are presented as mean ± SD.

Results: Pneumoperitoneum reduced EELV (healthy weight, 1195 ± 405 vs. 1724 ± 774 ml; obese, 751 ± 258 vs. 886 ± 284 ml) and worsened static elastance and dead space in both groups (in all P < 0.01 vs. zero-end expiratory pressure before pneumoperitoneum) whereas oxygenation was unaffected. PEEP increased EELV (healthy weight, 570 ml, P < 0.01; obese, 364 ml, P < 0.01) with no effect on oxygenation. Compared with PEEP alone, EELV and static elastance were further improved after RM in both groups (P < 0.05), as was oxygenation (P < 0.01). In all patients, RM-induced change in EELV was 16% (P = 0.04). These improvements were maintained 30 min after RM. RM-induced changes in EELV correlated with change in oxygenation (r = 0.42, P < 0.01).

Conclusion: RM combined with 10 cm H2O of PEEP improved EELV, respiratory mechanics, and oxygenation during pneumoperitoneum whereas PEEP alone did not.

What We Already Know about This Topic

❖ Pulmonary function is impaired during laparoscopy as a result of diaphragm shift, which causes airway closure and collapse of dependent lung regions.

What This Article Tells Us That Is New

❖ In patients undergoing laparoscopy, positive end-expiratory pressure increased respiratory elastance but did not improve oxygenation. Addition of a recruitment maneuver increased respiratory elastance and oxygenation in normal weight and obese patients.

GENERAL anesthesia, even in patients with healthy lungs, causes impairment in gas-exchange and respiratory mechanics.1 Such effects are mainly the result of the formation of atelectasis.2,3 Laparoscopic surgery worsens respiratory mechanics in healthy weight4–6 and obese patients.7–11 The increase in intraabdominal pressure during pneumoperitoneum causes cranial shift of the end-expiratory position of the diaphragm, further reducing end-expiratory lung volume (EELV), and predisposes patients to airway closure and collapse of dependent lung regions.12–15 Several strategies have been investigated to improve oxygenation and respiratory mechanics during laparoscopic surgery. The use of high positive end-expiratory pressure...
(PEEP) and positioning (e.g., beach chair position) has effectively improved respiratory function and oxygenation in obese patients.\textsuperscript{7–11} whereas PEEP alone improved oxygenation only slightly.\textsuperscript{16,17} PEEP also efficiently counteracted the upward shift of the diaphragm during laparoscopy in healthy weight patients, limited surgical effects on respiratory mechanics, and improved oxygenation.\textsuperscript{18–20}

The use of a recruitment maneuver (RM) effectively re-expended atelectasis after anesthesia induction, increased EELV, and improved oxygenation in healthy weight\textsuperscript{21,22} and obese patients.\textsuperscript{23} RMs also improved oxygenation in obese patients undergoing laparoscopic procedures.\textsuperscript{17,24,25} However, a single RM may not be sufficient,\textsuperscript{23,24} and PEEP is required to prevent rapid reoccurrence of atelectasis—especially when a high-inspired oxygen fraction is used.\textsuperscript{26} Conversely, although pneumoperitoneum led to increased atelectasis and altered respiratory mechanics in healthy weight patients,\textsuperscript{2,22} the value of RMs in such patients has been little studied during laparoscopy.\textsuperscript{28}

Most of the aforementioned studies investigated the effects of PEEP and RM on oxygenation. However, alveolar recruitment—an anatomical phenomenon that exhibits as restored aeration on computed tomography—often fails to coincide with functional recruitment as defined by improved gas exchange.\textsuperscript{29} In addition, Strang et al.\textsuperscript{27} recently showed that oxygenation did not correlate with atelectasis formation during pneumoperitoneum, indicating that oxygenation may be a poor indicator of the extent of lung collapse.\textsuperscript{2,27} Oxygenation may, therefore, be an unreliable marker of recruitment effects\textsuperscript{28} and inadequately sensitive to detect lung overdistension.\textsuperscript{30,31} Despite the potential clinical relevance thereof, intraoperative measurement of functional residual capacity is uncommon in routine practice.\textsuperscript{32,33} A functional residual capacity value—defined as the relaxed equilibrium volume of the lungs when there is no pressure difference between the alveoli and the atmosphere—is normally obtained in a spontaneously breathing patient at the end of a normal expiration. In this context, EELV is used to denote functional residual capacity during mechanical ventilation. We previously demonstrated that, after induction of anesthesia, PEEP improves efficiently both EELV and respiratory mechanics, with no major effect on oxygenation.\textsuperscript{34} Ventilation at low EELV may instigate or worsen lung injury, possibly as a result of repeated airway closure.\textsuperscript{35} In addition, EELV was found to be a more sensitive indicator of PEEP-induced reaeration and alveolar recruitment than was oxygenation.\textsuperscript{36} Therefore, we investigated the effects of RM after application of PEEP, on EELV modifications, respiratory mechanics, and oxygenation in healthy weight and obese patients undergoing laparoscopic surgery. We hypothesized that RM would be useful to counteract the detrimental effects of pneumoperitoneum, especially after EELV reduction.

Materials and Methods

After obtaining the approval of our institutional review board (Clermont-Ferrand, France), written informed consent was obtained from all patients. Sixty adult patients (30 obese [body mass index higher than 35 kg/m\textsuperscript{2}], 30 healthy weight [body mass index less than 25 kg/m\textsuperscript{2}]) with American Society of Anesthesiologists Physical Status Classification scores of 1–3, scheduled for laparoscopic procedures of at least 1 h, were prospectively included in the current study. Exclusion criteria were age younger than 18 yr, pregnancy, emergency surgery, heart failure (defined as New York Heart Association classification more than 3), coronary disease, and chronic obstructive pulmonary disease.

Anesthetic management was standardized as follows. General anesthesia was induced using propofol (2 mg/kg) and remifentanil (0.25 μg · kg\textsuperscript{-1} · min\textsuperscript{-1}) and was maintained, to a target bispectral index between 40 and 50, with continuous infusion of propofol (using a target-controlled infusion) and remifentanil. Anesthetic concentrations were based on ideal body weight. Muscle paralysis was induced with succinylcholine (1 mg/kg) to facilitate tracheal intubation (cuffed tube 7–7.5; Portex, Inc., London, England), and was maintained with subsequent bolus doses of cisatracurium as indicated by orbicular nerve stimulation (train-of-four ratio). No patient required fiber-optic intubation. The duration of anesthesia induction (defined as the time between the end of preoxygenation and tracheal intubation) and occurrence of hypoxic apnea (defined as peripheral oxygen saturation levels less than 92%)\textsuperscript{37} after anesthesia was induced were recorded for all patients. Intraoperative fluid intake was maintained using 8 ml · kg\textsuperscript{-1} · h\textsuperscript{-1} of normal saline solution. Standard monitoring included continuous electrocardiograph, heart rate, peripheral oxygen saturation, and end-tidal carbon dioxide concentration (ET\textsubscript{CO}\textsubscript{2}) recording. The radial artery was cannulated before induction of anesthesia, in line with the standard practice of our institution, for invasive blood pressure and blood gas monitoring. Arterial pulse pressure variation (ΔPP) was monitored throughout the surgical procedure, as previously described.\textsuperscript{38} Bolus doses of hydroxyethylstarch (HES 130/0.4, Voluven; Fresenius Kabi, Bad Hamburg, Germany) were given, as necessary, up to 50 ml/kg, to maintain ΔPP at less than 13%, especially before RM.

Study Protocol

A schema of the protocol is shown in figure 1. In both weight groups, anesthesia induction and the study procedure were performed in the beach chair position, as previously described.\textsuperscript{7} Before induction of anesthesia, preoxygenation (spontaneous breathing of 100% oxygen via facemask) was conducted for 5 min before tracheal intubation. Immediately after intubation, patients were mechanically ventilated (Engström Carestation; Datex-Ohmeda, General Electric, Helsinki, Finland) with the ventilator in the volume-controlled mode and tidal volume at 8 ml/kg\textsuperscript{-1} ideal body weight, a respiratory rate adjusted to maintain an arterial carbon dioxide tension of 35–42 mmHg, and an inspiratory/expiratory ratio of 1/2. The inspiratory oxygen fraction (F\textsubscript{IO}\textsubscript{2}) was 0.5
and zero end-expiratory pressure (ZEEP) was applied before the onset of pneumoperitoneum (step 1).

Pneumoperitoneum was generated by insufflating carbon dioxide into the abdomen with the intraabdominal pressure maintained at 12 cm H$_2$O (40-l High-Flow Insufflator; Stryker Endoscopy, San Jose, CA). After an equilibrium time of 10 min in ZEEP (step 2), PEEP was increased to 10 cm H$_2$O for 10 min (step 3). Thereafter, RM was performed, consisting of the application of continuous positive airway pressure (40 cm H$_2$O/40 s). In the event of a drop in systolic blood pressure by more than 20%, RM would have been terminated.

To analyze the specific effects of RM, 10 patients in each group were subjected to PEEP of 10 cm H$_2$O alone. For the others, immediately after RM was applied, PEEP was adjusted to 10 cm H$_2$O. In all patients, a second set of measurements was obtained at 5 min (step 4) and 30 min (step 5) postintervention (RM or no RM). Apart from application of recruitment maneuver and adjustment of PEEP level, basal ventilatory settings were kept constant throughout.

**Physiologic Measurements**

In both groups, preoperative lung function tests and EELV measurements were obtained using the helium dilution method (Spirodyn'R; Dyn'R, Muret, France), in a 30° head-up position at end-expiration, to obtain reference EELV values when awake. Patients were asked to breathe normally (i.e., at their usual tidal volume).

At each step of the experiment (before and after induction of pneumoperitoneum in ZEEP, PEEP 10 cm H$_2$O before intervention, 5 min and 30 min after intervention, and PEEP 10 cm H$_2$O after the pneumoperitoneum was exsufflated), peak ($P_{aw,max}$) and plateau end-inspiratory ($P_{plat}$) airway pressures were recorded using the end-inspiratory and end-expiratory occlusion technique. Intrinsic PEEP was evaluated by means of end-expiratory occlusion. The quasistatic elastance of the total respiratory system (E$_{rs}$) was calculated as $\Delta P_{aw}/V_T$, where $\Delta P_{aw}$ is the difference between plateau end-inspiratory and end-expiratory airway pressure at a period of no-flow (corrected for intrinsic PEEP), and $V_T$ is the tidal volume. EELV was measured twice (wash-out/wash-in method) using an automated procedure available on the ventilator (COVX module; GE Healthcare, Helsinki, Finland). EELV measurements, which reflect the amount of gas in the lungs, require an inspired oxygen fraction step change of 0.1, without interruption of mechanical ventilation or any need for supplementary tracer gases, as previously described. Previous studies have specifically evaluated the reproducibility, accuracy, and precision of EELV measurements provided by the ventilator.

To avoid any influence of the step change in FIO$_2$, arterial blood samples were taken from the radial artery before preoxygenation and at each step of the protocol just before EELV measurement. Arterial partial pressure of oxygen (PaO$_2$), arterial partial pressure of carbon dioxide (PaCO$_2$), and arterial pH were measured using a blood-gas analyzer (IL Synthesis; Instrumentation Laboratory®, Lexington, MA).

The physiologic dead space to VT ratio ($V_D/V_T$) was computed according to the following formula: $V_D/V_T = (PaO_2 - PECO_2)/PaCO_2$, where PECO$_2$ is the mixed expired carbon dioxide partial pressure. Data were obtained by means of continuous expiratory air sampling, using a mainstream sensor placed in-line between the endotracheal tube and the Y-piece (CO$_2$ SMO PLUS 8100; Novametrix Medical Sys-
dent group differences were determined using the unpaired Stu-

... variance or the Kruskal-Wallis H-test, as appropriate.

Categorical and quantitative data were correlated using a

... exchange, and respiratory mechanics between two points in

... time were performed using a paired Student t test. Inter-
group differences were determined using the unpaired Student t test or the Kruskal-Wallis H-test otherwise. A two-way analysis of variance with different size groups was performed to test the effect of categorical parameters on longitudinal data. Comparisons of EELV, gas exchange, and respiratory mechanics between two points in time were performed using a paired Student t test. Inter-group differences were determined using the unpaired Student t test or the Kruskal-Wallis H-test, as appropriate. Post hoc analyses for pairwise comparisons were performed with the Bonferroni test. When applicable, correlations between variables were analyzed using Pearson coefficient correlation when the variables were normally distributed and Spearman $p$ coefficient otherwise. Statistical testing was two-tailed with significance assumed at $P < 0.05$.

### Results

The 60 patients approached for consent to participate in this study accepted. Data from all 60 patients are included in the analysis. Patient baseline characteristics are summarized in table 1. Except for weight and body mass index, baseline characteristics were similar between the study groups. Surgical procedures performed were laparoscopic gastric resection $(n = 16)$, splenectomy $(n = 8)$, and hepatectomy $(n = 6)$ in the healthy weight group; laparoscopic sleeve gastrectomy $(n = 18)$ and Roux-en-Y gastric bypass $(n = 12)$ in the obese group. No relevant clinical problems occurred during any procedure or during surgery. No patients needed mechanical ventilation after the operation. Preoperative reference values of EELV were $2860 \text{ ml}$ in healthy weight patients and $2170 \text{ ml}$ in obese patients $(P < 0.01)$.

During PEEP changes or RM, no significant differences in hemodynamic data were observed (tables 2 and 3).

### Effects of Anesthesia Induction and Pneumoperitoneum Insufflation

There was no difference in the duration of anesthesia induction between the two groups $(166 \pm 13 \text{ vs. } 159 \pm 14 \text{ s}$, in the healthy weight and obese group, respectively; $P = 0.13$). Although no healthy weight patients developed hypoxic apnea, one obese patient did $(P = 0.31)$. Compared with pre-induction values, anesthesia induction and mechanical ventilation with ZEEP significantly reduced $\text{PaO}_2/\text{FiO}_2$ ratios (healthy weight, $448 \pm 72 \text{ vs. } 341 \pm 90 \text{ mmHg}$, $P < 0.01$; obese, $394 \pm 75 \text{ mmHg} \text{ vs. } 214 \pm 90 \text{ mmHg}$, $P < 0.01$) and EELV ($-40$ and $-59\%$, in healthy weight and obese patients, respectively; both $P$ values less than $0.01 \text{ vs. reference EELV values when awake}$).

Pneumoperitoneum further reduced EELV in the two groups (table 4, both $P$ values less than $0.01 \text{ vs. EELV before pneumoperitoneum}$). Pneumoperitoneum increased $\text{PaCO}_2$ in the two study groups, whereas oxygenation was unaffected (tables 2 and 3). Pneumoperitoneum also increased Pa-

### Table 1. Patients Characteristics ($N = 60$)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Healthy Weight Study Group</th>
<th>Obese Study Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PEEP $\pm$ RM</td>
<td>PEEP</td>
</tr>
<tr>
<td></td>
<td>$(n = 20)$</td>
<td>$(n = 10)$</td>
</tr>
<tr>
<td>Age, yr</td>
<td>$48 \pm 11$</td>
<td>$49 \pm 7$</td>
</tr>
<tr>
<td>Sex, m/f, no.</td>
<td>$10/10$</td>
<td>$6/4$</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>$65 \pm 13$</td>
<td>$66 \pm 11$</td>
</tr>
<tr>
<td>Height, cm</td>
<td>$170 \pm 8$</td>
<td>$172 \pm 7$</td>
</tr>
<tr>
<td>Ideal body weight, kg</td>
<td>$58 \pm 11$</td>
<td>$60 \pm 10$</td>
</tr>
<tr>
<td>BMI, kg/m$^2$</td>
<td>$22 \pm 3$</td>
<td>$23 \pm 1$</td>
</tr>
<tr>
<td>ASA Classification, 1/2/3, no.</td>
<td>$3/11/6$</td>
<td>$2/5/3$</td>
</tr>
</tbody>
</table>

All data are presented as mean $\pm$ SD unless otherwise specified. $^* P < 0.05$ vs. healthy weight group.

ASA Classification = American Society of Anesthesiologists Physical Status Classification; BMI = body mass index; f = female; m = male; PEEP = 10 cm H$_2$O positive end-expiratory pressure; RM = recruitment maneuver.
For all variables measured in healthy weight patients (N = 30), n = 10 for positive end-expiratory pressure (PEEP); n = 20, PEEP + recruitment maneuver (RM). All data are presented as mean ± SD unless otherwise specified.

* P < 0.01, vs. pneumoperitoneum after ZEEP. † P < 0.01, vs. pneumoperitoneum before ZEEP. ‡ P < 0.01, vs. PEEP 10 cm H₂O. § P < 0.01, PEEP + RM vs. PEEP.

ETCO₂ and the V₃/V₄ ratio (both P values less than 0.01). Overall respiratory mechanics worsened after pneumoperitoneum was induced (tables 2 and 3).

No intrinsic PEEP was detected in healthy weight or obese patients.

**Effects of PEEP**

Compared with ZEEP after pneumoperitoneum was induced, PEEP 10 cm H₂O significantly increased EELV in both study groups (healthy weight, 570 ml, P < 0.001; obese, 364 ml, P < 0.001). There was a significant difference between the two study groups (P = 0.003). In all patients, PEEP-induced changes for EELV were 46% (P < 0.001, compared with ZEEP after pneumoperitoneum) with no significant difference between PEEP+RM and PEEP alone (table 4). PEEP also lowered PaCO₂ and Pa-ETCO₂ in the two groups with no significant effect on oxygenation (tables 2 and 3). Overall respiratory mechanics improved after application of PEEP.

**Effects of the Recruitment Maneuver**

After RM, EELV further increased in both study groups (healthy weight, 154 ml; obese, 233 ml, fig. 2). Compared to PEEP alone, RM-induced changes of EELV were 10% in healthy weight and 20% in obese patients, with a statistically significant difference between the two groups (P = 0.026). In all patients, RM-induced change in EELV was 16% (P = 0.04). After RM, gas exchange also improved in the two study groups (tables 2 and 3). In contrast, PEEP alone did not cause any significant change in oxygenation. Compared
Changes in EELV with RM during Pneumoperitoneum

Table 3. Respiratory Mechanics, Gas Exchange, and Hemodynamic Data in Obese Patients (N = 30)

<table>
<thead>
<tr>
<th>Variable</th>
<th>ZEEP after Pneumoperitoneum</th>
<th>ZEEP before Pneumoperitoneum</th>
<th>PEEP 10 cm H2O</th>
<th>5 min</th>
<th>30 min</th>
<th>End of Surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak airway pressure, cm H2O</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>PEEP</td>
<td>28 ± 5</td>
<td>34 ± 5*</td>
<td>37 ± 3†</td>
<td>37 ± 2</td>
<td>36 ± 4</td>
<td>33 ± 5</td>
</tr>
<tr>
<td>PEEP + RM</td>
<td>26 ± 4</td>
<td>31 ± 5*</td>
<td>33 ± 4†</td>
<td>31 ± 4§</td>
<td>31 ± 5§</td>
<td>30 ± 5§</td>
</tr>
<tr>
<td>End-inspiratory plateau pressure, cm H2O</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>PEEP</td>
<td>17 ± 3</td>
<td>20 ± 3*</td>
<td>23 ± 3†</td>
<td>23 ± 4†</td>
<td>24 ± 5†</td>
<td>22 ± 4</td>
</tr>
<tr>
<td>PEEP + RM</td>
<td>16 ± 3</td>
<td>19 ± 3*</td>
<td>22 ± 3†</td>
<td>19 ± 2§</td>
<td>20 ± 3§</td>
<td>19 ± 2§</td>
</tr>
<tr>
<td>Intrinsic PEEP, cm H2O</td>
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<td>—</td>
<td>—</td>
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<td>—</td>
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<tr>
<td>PEEP</td>
<td>0.5 ± 0.5</td>
<td>0.5 ± 0.3</td>
<td>0.4 ± 0.6</td>
<td>0.4 ± 0.5</td>
<td>0.5 ± 0.3</td>
<td>0.5 ± 0.2</td>
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<tr>
<td>PEEP + RM</td>
<td>0.6 ± 1</td>
<td>0.5 ± 0.4</td>
<td>0.5 ± 0.5</td>
<td>0.7 ± 0.4</td>
<td>0.4 ± 0.5</td>
<td>0.4 ± 0.6</td>
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<tr>
<td>Ers, cm H2O/ml</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>PEEP</td>
<td>34 ± 4</td>
<td>40 ± 6*</td>
<td>28 ± 8†</td>
<td>28 ± 6</td>
<td>30 ± 7</td>
<td>27 ± 5†</td>
</tr>
<tr>
<td>PEEP + RM</td>
<td>31 ± 6</td>
<td>39 ± 10*</td>
<td>25 ± 7†</td>
<td>20 ± 5†‡§</td>
<td>22 ± 6†§</td>
<td>18 ± 5†‡§</td>
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<td>Arterial pH</td>
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<tr>
<td>PEEP</td>
<td>7.42 ± 0.04</td>
<td>7.41 ± 0.03</td>
<td>7.39 ± 0.02†</td>
<td>7.39 ± 0.04†</td>
<td>7.38 ± 0.02‡</td>
<td>7.39 ± 0.03†</td>
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<tr>
<td>PEEP + RM</td>
<td>7.44 ± 0.03</td>
<td>7.40 ± 0.02</td>
<td>7.38 ± 0.03†</td>
<td>7.39 ± 0.03‡</td>
<td>7.37 ± 0.03†</td>
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<tr>
<td>PaO2, mmHg</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
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<td>—</td>
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<tr>
<td>PEEP</td>
<td>102 ± 39</td>
<td>94 ± 28</td>
<td>104 ± 28</td>
<td>104 ± 20</td>
<td>98 ± 21</td>
<td>106 ± 15</td>
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<tr>
<td>PEEP + RM</td>
<td>107 ± 46</td>
<td>105 ± 43</td>
<td>111 ± 40</td>
<td>146 ± 37†‡§</td>
<td>149 ± 40†‡§</td>
<td>169 ± 42†‡§</td>
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<tr>
<td>PaCO2, mmHg</td>
<td>—</td>
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<tr>
<td>PEEP</td>
<td>42 ± 4</td>
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<td>41 ± 4</td>
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<tr>
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<td>41 ± 2†</td>
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<td>40 ± 3</td>
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<tr>
<td>PEEP</td>
<td>9 ± 1</td>
<td>14 ± 3*</td>
<td>10 ± 4†</td>
<td>10 ± 3†</td>
<td>11 ± 2†</td>
<td>9 ± 3†</td>
</tr>
<tr>
<td>PEEP + RM</td>
<td>10 ± 2</td>
<td>14 ± 2*</td>
<td>9 ± 3†</td>
<td>5 ± 1§ ‡§</td>
<td>6 ± 2‡§</td>
<td>5 ± 2‡§</td>
</tr>
<tr>
<td>Vr/Vt</td>
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<td>—</td>
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<tr>
<td>PEEP</td>
<td>0.30 ± 0.02</td>
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<td>0.42 ± 0.04</td>
<td>0.44 ± 0.02</td>
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<tr>
<td>PEEP + RM</td>
<td>0.35 ± 0.04</td>
<td>0.45 ± 0.6*</td>
<td>0.40 ± 0.06</td>
<td>0.24 ± 0.06†‡§</td>
<td>0.32 ± 0.05†‡§</td>
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</tr>
<tr>
<td>Mean arterial pressure, mmHg</td>
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<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
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<tr>
<td>PEEP</td>
<td>83 ± 24</td>
<td>87 ± 24</td>
<td>82 ± 10</td>
<td>82 ± 14</td>
<td>86 ± 10</td>
<td>80 ± 9</td>
</tr>
<tr>
<td>PEEP + RM</td>
<td>88 ± 17</td>
<td>85 ± 15</td>
<td>80 ± 20</td>
<td>82 ± 16</td>
<td>82 ± 10</td>
<td>84 ± 12</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>PEEP</td>
<td>70 ± 15</td>
<td>72 ± 25</td>
<td>73 ± 12</td>
<td>74 ± 24</td>
<td>77 ± 15</td>
<td>79 ± 20</td>
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<tr>
<td>PEEP + RM</td>
<td>75 ± 16</td>
<td>74 ± 15</td>
<td>77 ± 18</td>
<td>80 ± 15</td>
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<td>74 ± 12</td>
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For all variables measured in obese patients (N = 30), n = 10 for positive end-expiratory pressure (PEEP); n = 20, PEEP + recruitment maneuver (RM). All data are presented as mean ± SD unless otherwise specified.

* P < 0.01, † P < 0.01, ‡ P < 0.01, § P < 0.05, PEEP + RM vs. PEEP.

Ers = elastance of the respiratory system; PaCO2 = arterial partial pressure of carbon dioxide; Pa-ETCO2 = difference between arterial and end-tidal partial pressure of carbon dioxide; Vr/Vt = physiological dead space; ZEEP = zero end-expiratory pressure.

to PEEP alone, overall respiratory mechanics further improved after RM was performed (tables 2 and 3).

Thirty minutes after intervention, EELV was lower but still significant in the two groups compared to ZEEP after pneumoperitoneum was induced (table 4). There was no significant difference compared to PEEP 10 cm H2O preintervention. The difference between the PEEP only and the PEEP+RM groups was significant only in healthy weight patients. Furthermore, in contrast to PEEP alone, 30 min after intervention, improvements in PaO2, Pa-ETCO2, and the Vr/Vt ratio were all sustained with PEEP+RM (tables 2 and 3).

End-expiratory Lung Volume, Static Elastance, Oxygenation, and Dead Space

A significant correlation was found between changes in EELV with pneumoperitoneum and changes in respiratory system elastance (r = 0.49, P < 0.01) and Pa-ETCO2 (r = 0.39, P = 0.012), as well as between changes in elastance and dead space after pneumoperitoneum (r = 0.46, P < 0.01). No correlation was found between change of EELV with pneumoperitoneum and changes of oxygenation (P = 0.18).

A significant but weak correlation was found between PEEP-induced change in EELV (calculated using ZEEP during pneumoperitoneum as the reference) and change in respiratory system elastance (r = 0.33, P = 0.03) whereas no correlation was found between change in EELV with application of PEEP and oxygenation (P = 0.90). RM-induced changes in EELV correlated with changes in respiratory system elastance (r = 0.51, P < 0.001) and oxygenation (r = 0.34, P = 0.03). A significant correlation was also found between RM-induced change of EELV (with PEEP 10 cm H2O before RM as the reference) and change of oxygenation (r = 0.42, P < 0.01).

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Discussion

During pneumoperitoneum, in the beach chair position, 10 cm H2O of PEEP improved EELV and respiratory elastance with no major change in oxygenation, whereas RM further improved EELV, respiratory elastance, and oxygenation in healthy weight and obese patients.

Pneumoperitoneum worsens respiratory mechanics in healthy weight and obese patients.7–10 Such effects may be related to reduced lung volumes and atelectasis formation, as previously confirmed by CT.12 After induction of anesthesia, we found an average EELV of approximately 900 ml in obese patients and 1,700 ml in healthy weight patients, a result that is in line with measurements obtained by spiral CT and helium dilution technique in the absence of PEEP.16,23,47 Likewise, these results are in agreement with previous findings that pneumoperitoneum further reduced EELV after anesthesia induction in healthy weight and obese patients.7,48 Although morbid obesity per se may cause significant changes in respiratory system function and oxygenation,49 the effects of pneumoperitoneum on oxygenation have been described as variable. Valenza et al.7 found that, despite severe impairment in respiratory mechanics and reduced EELV, oxygenation was improved during pneumoperitoneum. We confirm data indicating that oxygenation was not affected by pneumoperitoneum.6,17 Similar results were obtained in healthy weight patients. This finding is partly in agreement with the results of Sprung et al.,6 who reported that alterations in respiratory mechanics induced by pneumoperitoneum in healthy weight patients were greater than those seen in obese patients.

Ventilation at low EELV may cause or worsen lung injury, most likely as a result of the opening and closing of atelectatic lung regions and peripheral airways, whereas PEEP attenuates such injury.35 Both PEEP and positioning (e.g., reverse Trendelenburg position) have been found to attenuate the effects of pneumoperitoneum on respiratory mechanics and lung volumes.7,18,19 Among patients with a pneumoperitoneum-induced decrease in lung volume, PEEP increased EELV above the closing capacity.19 We found that PEEP 10 cm H2O only partly counteracted the detrimental effects of pneumoperitoneum on respiratory system elastance. No major effect on oxygenation was evident in either of our two study groups. Recent studies have also found that PEEP alone was insufficient to improve oxygenation during an increase in intraabdominal pressure.24,50 Indeed, PEEP may increase the normally aerated lung fraction in parallel with a reduction in the proportion of poorly aerated lung tissue although the extent of atelectasis may remain unchanged.25

An RM has been proposed as a valuable device during pneumoperitoneum in obese patients.9,17,24 In contrast, few data are available on the use of RM in healthy weight patients. It has been shown in normal-weight patients that a single insufflation of 40 cm H2O for 8 s was sufficient to open atelectatic areas after induction of anesthesia.51 In a recent study, Maisch et al.58 demonstrated that, in normal-weight patients, RM in conjunction with PEEP 10 cm H2O provided significant improvements in EELV, respiratory mechanics, and oxygenation during operation without any derangement of the lung and diaphragm position. We measured EELV, a sensitive indicator of airway collapse and PEEP-induced re-aeration,36 and found that, compared to PEEP alone, the RM of 40 cm H2O for 40 s was associated with average increases in EELV of 150 and 230 ml, respectively, for healthy weight and obese patients. We also observed a marked increase in oxygenation associated with improved respiratory system elastance, dead space, and Pa-ETCO2. These observations are in agreement with previous results where RM decreased Pa-ETCO2, a useful indicator of lung collapse and reopening after open-lung PEEP,35 which in turn reduced dead space.45 Nevertheless, high insufflation pressures during RM may expose patients to hemodynamic instability, especially among those who are hypovolemic.53 Therefore, we have taken special care that patients were normovolemic before performing RM. In addition, as an increase in EELV may result from alveolar recruitment and/or overdistension,33,39 we measured respiratory system elastance and dead space to distinguish between these possibili-

Table 4. End-expiratory Lung Volume, ml (N = 60)

<table>
<thead>
<tr>
<th>Measure</th>
<th>Healthy Weight Study Group</th>
<th>Obese Study Group</th>
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<tbody>
<tr>
<td></td>
<td>PEEP + RM (n = 20)</td>
<td>PEEP (n = 10)</td>
</tr>
<tr>
<td>After ZEEP</td>
<td>1,724 ± 774</td>
<td>1,802 ± 511</td>
</tr>
<tr>
<td>Before ZEEP</td>
<td>1,194 ± 405</td>
<td>1,225 ± 266</td>
</tr>
<tr>
<td>PEEP</td>
<td>1,750 ± 472*</td>
<td>1,690 ± 275*</td>
</tr>
<tr>
<td>Postintervention</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>5 min</td>
<td>1,958 ± 461†‡</td>
<td>1,664 ± 341*</td>
</tr>
<tr>
<td>30 min</td>
<td>1,928 ± 546*†‡</td>
<td>1,541 ± 248*</td>
</tr>
</tbody>
</table>

All data are presented as mean ± SD unless otherwise specified. *P < 0.001 vs. pneumoperitoneum before ZEEP. †P < 0.001 vs. PEEP 10 cm H2O. ‡P < 0.001 PEEP + RM vs. PEEP only. PEEP = positive end-expiratory pressure (10 cm H2O); RM = recruitment maneuver; ZEEP = zero end-expiratory pressure.
ties. However, we did not measure lung elastance, which may have been more relevant. During both pneumoperitoneum and beach chair position, PEEP-induced improvement in respiratory system elastance with PEEP was mainly related to changes in chest wall elastance. After pneumoperitoneum was induced, intraabdominal pressure and chest wall elastance both remained constant. Thus, it may be assumed that changes in elastance after RM mostly affected the lungs. Although PEEP 10 cm H₂O was beneficial during pneumoperitoneum in healthy weight and obese patients, it may be argued that, when searching for safer ventilation, applying lower levels of PEEP to an open lung could be effective in healthy weight patients. It has been shown that application of PEEP 5 cm H₂O alone was effective in reducing atelectasis during pneumoperitoneum and improving oxygenation in normal-weight patients. Our results are in contrast to previous findings for normal-weight patients where it was reported that RM followed by ZEEP significantly reduced atelectasis for at least 20 min when using reduced FIO₂. In addition, we found that, in contrast to PEEP alone, most healthy weight patients still improved in oxygenation and EELV after RM was performed without evidence for overdistension with PEEP 10 cm H₂O. Our results are consistent with those of Maisch et al. who reported that, in normal-weight patients without further EELV reduction by pneumoperitoneum insufflation, RM followed by high levels of PEEP is required to increase EELV effectively with reduced dead space.

Our study had several limitations. First, we did not measure the real extent of alveolar recruitment after application of PEEP and RM. Nevertheless, EELV measurements using the modified nitrogen wash-out/wash-in method correlate well with EELV as measured by CT, as well as with changes in lung aeration and consolidation gathered through CT. Moreover, we excluded patients with airway disease (especially those with chronic obstructive pulmonary disease) for whom differences between CT measurement and ventilation-based assessment techniques may be relevant. Second, we did not conduct a detailed evaluation of hemodynamics during our investigation. However, a recent study confirmed the hemodynamic safety of RM and application of PEEP in intravascular volume-loaded patients. Third, our procedural steps were not randomized. Owing to the specific procedures required by our study protocol and difficulties in collecting these measurements, it was difficult to design a randomized study. Fourth, the rather short interval between procedural steps is an additional limitation. However, the equilibration time of 10 min allowed readings to be within the accuracy limits issued by the instrument manufacturers. Moreover, as the sequence was the same in both groups, we believe our comparisons are valid. Fifth, although oxygenation and respiratory mechanics were sustainably improved at 30 min, we cannot exclude later variations in these parameters. Indeed, repeated RM have been shown to improve both compliance and oxygenation, compared with a single RM. Therefore, repeated RM in conjunction with PEEP may represent an “optimal” open-lung approach.

**Conclusion**

Pneumoperitoneum worsens reduction in EELV and respiratory mechanics produced by anesthesia induction among normal-weight and obese patients, with no major effect on oxygenation. In contrast to PEEP alone, a PEEP of 10 cm H₂O combined with RM induces sustained improvements in EELV, gas exchange, and respiratory mechanics, and may be useful in counteracting the detrimental effects of pneumoperitoneum—especially on lung volume reduction in healthy weight and obese patients.

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**References**


Changes in EELV with RM during Pneumoperitoneum

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