Pressure-controlled ventilation improves oxygenation during laparoscopic obesity surgery compared with volume-controlled ventilation

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Background. We compared pressure and volume-controlled ventilation (PCV and VCV) in morbidly obese patients undergoing laparoscopic gastric banding surgery.

Methods. Thirty-six patients, BMI \( \geq 35 \text{ kg m}^{-2} \), no major obstructive or restrictive respiratory disorder, and \( P_{aCO_2} < 6.0 \text{ kPa} \), were randomized to receive either VCV or PCV during the surgery. Ventilation settings followed two distinct algorithms aiming to maintain end-tidal CO\(_2\) \((E_{CO_2})\) between 4.40 and 4.66 kPa and plateau pressure \((P_{plateau})\) as low as possible. Primary outcome variable was peroperative \( P_{plateau} \). Secondary outcomes were \( P_{aO_2} \) \((FIO_2\) at 0.6 in each group) and \( P_{aCO_2} \) during surgery and 2 h after extubation. Pressure, flow, and volume time curves were recorded.

Results. There were no significant differences in patient characteristics and co-morbidity in the two groups. Mean pH, \( P_{aO_2} \), \( S_{aO_2} \), and the \( P_{aO_2}/FIO_2 \) ratio were higher in the PCV group, whereas \( P_{aCO_2} \) and the \( E_{CO_2} - P_{aCO_2} \) gradient were lower (all \( P < 0.05 \)). Ventilation variables, including plateau and mean airway pressures, anaesthesia-related variables, and postoperative cardiovascular variables, blood gases, and morphine requirements after the operation were similar.

Conclusions. The changes in oxygenation can only be explained by an improvement in the lungs ventilation/perfusion ratio. The decelerating inspiratory flow used in PCV generates higher instantaneous flow peaks and may allow a better alveolar recruitment. PCV improves oxygenation without any side-effects.

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From January to June 2005, 36 obese patients undergoing laparoscopic bariatric surgery (gastric banding) were included. The inclusion criteria were BMI > 35 kg m$^{-2}$, age 18 yr or above, no major obstructive or restrictive pulmonary disease (defined as < 70% of predicted values for pulmonary function test variables of volume and flow), and $P_{aCO_2} <$ 6 kPa. Preoperative exclusion criteria were patient refusal, anticipated inability to perform early postoperative extubation, no signed informed consent form, and lack of understanding by the patient of the purpose of the study. Intraoperative exclusion criteria were inability to perform tracheal intubation in conditions of usual practice, inability to maintain stable mechanical ventilation settings for 30 min, inability to maintain an appropriate $V_{E}CO_2$, inability to remove the tracheal tube in the operating room, and conversion to laparotomy.

The preoperative evaluation included Epworth Sleepiness Scale and a physical examination. Patients underwent pulmonary function tests and blood gases, cardiac evaluation (and echocardiography if ordered by the cardiologist), and cardiorespiratory polygraphy if sleep apnoea syndrome was suspected. All patients had to attend five physiotherapy sessions before surgery.

The primary outcome variable was plateau pressure after 45 min of pneumoperitoneum. The null hypothesis was that plateau pressures with VCV and PCV modes were equivalent and the alternative hypothesis that they were different. Secondary outcomes were $P_{aO_2}$ and $P_{aCO_2}$ after 45 min of ventilation during pneumoperitoneum and 2 h after extubation.

Members of a team of two anaesthetists and three surgeons provided care to all the patients. Patients were randomized into two groups to receive mechanical ventilation using either VCV or PCV mode. The randomization was done using a software developed by our statistical department (Unité de Recherche Clinique, AP–HP, Hôpital Européen Georges Pompidou, Paris, France). A standardized protocol was used for anaesthesia. It included standard monitoring (ECG, non-invasive arterial pressure, pulse oximetry, anaesthetic gas, and CO2 analyser), preoxygenation, and induction and maintenance of total i.v. anaesthesia using a target-controlled infusion pump (Primea®, Fresenius Vial SA, Grenoble, France) delivering propofol and sufentanil to maintain a constant cerebral concentration of 4 $\mu$g ml$^{-1}$ of propofol and 0.3 $\mu$g ml$^{-1}$ of sufentanil. An atracurium infusion was started to maintain muscle relaxation at < 2 twitches (train-of-four ratio) of the orbicular muscle of the eye. Tracheal intubation was performed. Bispectral Index® was used to monitor level of consciousness (BIS® technology, Aspect Medical Systems, Meern, The Netherlands). The anaesthesiologist in charge was free to adapt targets and drug doses during surgery according to the individual needs of the patient. Patients were placed in a 25° head-up position.

An Evita 2 ventilator (Dräger, Antony, France) was used for ventilation with either VC or PC modes. The $\dot{V}_{E}CO_2$ was maintained between 4.4 and 4.6 kPa and plateau pressure was kept as low as possible with an upper limit of 40 cm.

The aim of this study was to compare the effects of PCV with VCV on airway pressures, blood gases, and haemodynamic variables in obese patients undergoing laparoscopic gastric banding.

**Methods**

This prospective randomized controlled study was approved by the Institutional Review Board (Comité Consultatif de Protection des Personnes) of the Necker University Hospital (Paris, France). All patients were asked for their signed and informed consent.
H$_2$O, according to a distinct algorithm for each ventilation mode (Fig. 3). As in obese patients, plateau pressure may be a poor indication of transpulmonary pressure, and because of impaired chest compliance in these patients, a limit of 30 cm H$_2$O was considered too low for these patients.

Tidal volume was initially set at 8 ml kg$^{-1}$ of ideal weight [i.e. $50+0.91 \times ($height in cm$-152.4$) for men and $45.5+0.91 \times ($height in cm$-152.4$) for women]. The ratio of inspiratory-to-expiratory time ($I:E$) was 1:2, and the $F_{I_{O_{2}}}$, 0.6. The inspiratory flow rate in the VCV mode was set so that plateau time was 20% of inspiratory time ($T_{i}$), allowing the ventilator to measure plateau pressure. In the PCV mode, a drop to zero inspiratory flow was checked on the flow-time curve to maximize the tidal volume generated for a given level of inspiratory pressure and to allow a comparison of plateau pressures between the two modes. A PEEP of 5 cm H$_2$O was applied to all the patients. Absence of auto-PEEP was ensured by a drop to zero expiratory flow on the flow-time curve in both modes. A heat and moisture exchanger was used for every patient.

After 45 min of laparoscopy during CO$_2$ pneumoperitoneum, and $\varepsilon_{CO_2}$ and MV at a steady state for the last 10 min, blood gas analysis was performed. Cardiovascular variables (heart rate and arterial pressure) were recorded. In addition, $Sp_{O_{2}}$, $v_{CO_{2}}$, Bispectral index, respiratory rate, tidal volume, MV, peak airway pressure, plateau pressure, PEEP, peak inspiratory flow, pneumoperitoneum pressure, dynamic total compliance and total airway resistance, dead-space, and carbon dioxide output ($VCO_{2}$) were recorded. Pressure and flow time curves were recorded over 1 min using VentView graphic software (Dräger, Antony, France). Mean airway pressure was calculated from the area under the pressure vs time curve over three ventilation cycles for each patient. Assuming intraoperative VO$_2$ at 130 ml min$^{-1}$ m$^{-2}$, and given that body surface areas were similar in both groups, we calculated the difference between theoretical alveolar oxygen partial pressure ($P_{A_{O_{2}}}$) as given by the alveolar gas equation, and measured $P_{A_{O_{2}}}$ from the following equation:

$$P_{A_{O_{2}}} - P_{A_{O_{2}}} = 713 \times 0.6 - \frac{P_{A_{CO_{2}}} \times \text{meas.}}{(VCO_{2}\text{meas.}/VO_{2}\text{estim.})} - P_{A_{O_{2}}} \text{meas.}$$

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**Fig 3** Algorithm for VCV and PCV settings. Vt, tidal volume; RR, respiratory rate; PEEP, positive end-expiratory pressure; $P_{\text{plateau}}$, plateau pressure; VCV, volume-controlled ventilation; PCV, pressure-controlled ventilation; $I:E$, inspiratory to expiratory time ratio; $F_{I_{O_{2}}}$, inspired fraction of oxygen; $E_{CO_{2}}$, end-tidal CO$_2$. 

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Ideal body weight calculation

**VCV:** $V_{t}=8$ ml kg$^{-1}$

**PCV:** $P_{\text{plateau}}$, set so that $V_{t}=8$ ml kg$^{-1}$

$I:E=1:2$

$F_{I_{O_{2}}}=0.6$ PEEP=$5$ cm H$_2$O

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$\varepsilon_{CO_{2}}>4.66$ kPa

Increase RR by 2 min$^{-1}$ every 5 min until 18 min$^{-1}$

Failure?

**PCV:** increase $P_{\text{plateau}}$ by 2 cm H$_2$O

**VCV:** increase $V_{t}$ by 1 ml kg$^{-1}$ every 5 min until $V_{t}=12$ ml kg$^{-1}$

$P_{\text{plateau}}$ limited to 40 cm H$_2$O

Failure?

**VCV:** Decrease $V_{t}$ by 1 ml kg$^{-1}$

**PCV:** Decrease $P_{\text{plateau}}$ by 2 cm H$_2$O every 5 min until $V_{t}=6$ ml kg$^{-1}$

$\varepsilon_{CO_{2}}<4.4$ kPa

Decrease RR by 2 min$^{-1}$ every 5 min until 10 min$^{-1}$

Failure?
No recruitment manoeuvres were performed after tracheal intubation. After surgery, prostigmine 2.5 mg and atropine 1.5 mg were given if the train-of-four ratio was above or equal to two twitches. Before extubation, $F_{I_{CO_2}}$ was increased to 1.0 with the patients breathing spontaneously. After 2 h of monitoring in the recovery room, arterial blood gases were sampled and heart rate, arterial pressure, $SP_{o_2}$, and total postoperative morphine doses were recorded. Nasal oxygen was given, if necessary, providing an $SP_{o_2}$ above 95%.

The statistical analysis was performed using NCSS software (NCSS, Kaysville, UT, USA). After a pilot study in 10 patients, we hypothesized a mean difference of 3.0 cm H$_2$O in plateau pressure between the two groups, and the standard deviation of plateau pressure values to be 3.1 cm H$_2$O. The sample size was calculated with alpha risk set at 5% and the power of the study at 80%; at least 18 patients were required in each group to detect a difference. Continuous variables were analysed by parametric or non-parametric tests depending upon their distribution as given by the Shapiro–Wilk and Anderson–Darling tests. Values are expressed as mean (SD).

This study was registered in the Protocols Registration System of the National Institutes of Health (http://ClinicalTrials.gov) under the title ‘Comparison Between Volume Controlled Ventilation and Pressure Controlled Ventilation for Laparoscopic Bariatric Surgery in Obese Patients’ (Identifier NCT00224653).

**Results**

The study included 36 patients randomized into two groups of 18 patients according to ventilation mode (VCV or PCV). No patient was excluded or withdrawn from the study. One surgical procedure was not completed because of gastric injury, but data were recorded before the incident occurred, under standard conditions of ventilation and laparoscopy.

There were no significant differences between the two groups in patient characteristics (Table 1), co-morbidity (Table 2), and preoperative test results (Table 3).

Intraoperative ventilation variables were not significantly different 45 min after initiating laparoscopy (Table 4). Peak inspiratory flow was higher in PCV group than in VCV (52 vs. 41 litre s$^{-1}$, $P<0.01$), as was the proportion of tidal volume delivered at half effective inspiratory time (67% in PCV vs. 53% in VCV, $P<0.01$). In one of the patients in the PCV group, $tv_{CO_2}$ value reached 4.9 kPa, but this was not due to any difficulty in ventilation: the respiratory rate (16 bpm) and plateau pressure (23 cm H$_2$O) were relatively low. One patient in VCV group reached a respiratory rate of 22 bpm but, for each patient, the absence of auto-PEEP was checked by a drop to zero expiratory flow of the flow-time curve.

### Table 1 Patient characteristics. Data are given as mean (SD) (range). There were no differences between the groups. PCV, pressure-controlled ventilation; VCV, volume-controlled ventilation.

<table>
<thead>
<tr>
<th></th>
<th>PCV (n=18)</th>
<th>VCV (n=18)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>40 (9) (27–61)</td>
<td>40 (12) (23–62)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>121 (21) (85–180)</td>
<td>119 (17) (96–160)</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.65 (0.09) (1.52–1.79)</td>
<td>1.64 (0.09) (1.50–1.83)</td>
</tr>
<tr>
<td>BMI (kg m$^{-2}$)</td>
<td>44 (5) (36–56)</td>
<td>44 (5) (38–55)</td>
</tr>
<tr>
<td>Ideal weight (kg)</td>
<td>57 (8) (45.1–73)</td>
<td>57 (9) (43–78)</td>
</tr>
<tr>
<td>Body surface area (m$^2$)</td>
<td>2.35 (0.25) (1.90–2.99)</td>
<td>2.32 (0.20) (2.06–2.75)</td>
</tr>
<tr>
<td>Gender (M/F)</td>
<td>3/15</td>
<td>4/14</td>
</tr>
<tr>
<td>ASA (I/II/III)</td>
<td>1/16/1</td>
<td>1/15/2</td>
</tr>
<tr>
<td>Systolic arterial pressure (mm Hg)</td>
<td>141 (14) (110–170)</td>
<td>136 (19) (100–170)</td>
</tr>
<tr>
<td>Diastolic arterial pressure (mm Hg)</td>
<td>82 (10) (60–100)</td>
<td>79 (7) (62–90)</td>
</tr>
<tr>
<td>Heart rate (beats min$^{-1}$)</td>
<td>79 (13) (60–109)</td>
<td>76 (8) (64–96)</td>
</tr>
<tr>
<td>Epworth Sleepiness Scale</td>
<td>5 (3) (1–13) (n=13)</td>
<td>4 (2) (0–8) (n=14)</td>
</tr>
</tbody>
</table>

### Table 2 Incidence of co-morbidity. PCV, pressure-controlled ventilation; VCV, volume-controlled ventilation. There were no significant differences between the groups.

<table>
<thead>
<tr>
<th>Number of patients affected</th>
<th>PCV</th>
<th>VCV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep apnoea syndrome</td>
<td>4/18</td>
<td>4/18</td>
</tr>
<tr>
<td>Hypertension</td>
<td>8/18</td>
<td>8/18</td>
</tr>
<tr>
<td>Coronaropathy</td>
<td>0/18</td>
<td>1/18</td>
</tr>
<tr>
<td>Lower limb ischaemia</td>
<td>0/18</td>
<td>2/18</td>
</tr>
<tr>
<td>Diabetes</td>
<td>5/18</td>
<td>7/18</td>
</tr>
<tr>
<td>Asthma</td>
<td>5/18</td>
<td>1/18</td>
</tr>
<tr>
<td>Dyslipidaemia</td>
<td>9/18</td>
<td>7/18</td>
</tr>
<tr>
<td>Arthritis</td>
<td>9/18</td>
<td>3/18</td>
</tr>
<tr>
<td>Hyperuricaemia</td>
<td>0/16</td>
<td>1/14</td>
</tr>
</tbody>
</table>

### Table 3 Preoperative tests. Mean (SD) (range). PCV, pressure-controlled ventilation; VCV, volume-controlled ventilation; FEV1, forced expiratory volume in 1 s; FVC, forced vital capacity; TLC, total lung capacity; $Pa_{Ar}$, arterial oxygen/carbon dioxide partial pressure; $Sa_{Ar}$, arterial oxygen saturation; $TCO_2$, total carbon dioxide; $LVEF$, left ventricular ejection fraction.

<table>
<thead>
<tr>
<th></th>
<th>PCV</th>
<th>VCV</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV1 (litre s$^{-1}$)</td>
<td>3 (0.8) (1.7–5)</td>
<td>3 (0.8) (1.9–5.3)</td>
</tr>
<tr>
<td>FVC (litre)</td>
<td>3.5 (0.9) (2.1–5.1)</td>
<td>3.4 (0.95) (2.1–5.8)</td>
</tr>
<tr>
<td>FEV1/FVC</td>
<td>84 (3) (78–89)</td>
<td>85 (5) (77–93)</td>
</tr>
<tr>
<td>TLC (litre)</td>
<td>5 (1.2) (3.7–7.5)</td>
<td>5 (1.1) (3.3–6.6)</td>
</tr>
<tr>
<td>Bronchial hyper-reactivity</td>
<td>3$^3$</td>
<td>4$^4$</td>
</tr>
<tr>
<td>pH</td>
<td>7.42 (0.02) (7.40–7.5)</td>
<td>7.41 (0.02) (7.40–7.44)</td>
</tr>
<tr>
<td>$Pa_{O_2}$ (kPa)</td>
<td>11.7 (1.3) (9.2–14.9)</td>
<td>11.7 (1.8) (9.4–16.0)</td>
</tr>
<tr>
<td>$Pa_{CO_2}$ (kPa)</td>
<td>5.2 (0.5) (4.1–6.0)</td>
<td>5.3 (0.43) (4.5–6.2)</td>
</tr>
<tr>
<td>$Sa_{O_2}$ (%)</td>
<td>98 (1)$^*$ (96–99)</td>
<td>98 (1)$^*$ (95–100)</td>
</tr>
<tr>
<td>$TCO_2$ (mmol litre$^{-1}$)</td>
<td>25 (2) (20–27)</td>
<td>26 (2)$^*$ (23–28)</td>
</tr>
<tr>
<td>$LVEF$ (%)</td>
<td>67 (5)$^*$ (58–70)</td>
<td>66 (8)$^*$ (60–79)</td>
</tr>
</tbody>
</table>

Values of haemodynamic variables were similar in both groups intraoperatively and after operation (Table 5).

Values of intraoperative blood gases were different between the two groups (Table 6); mean pH, $Pa_{Ar}$, $Sa_{Ar}$, and $TCO_2$.
the \( P_{aO2}/FIO2 \) ratio were higher in the PCV group \( (P<0.05) \). \( P_{aCO2} \) and \( \kappa_{aCO2} - P_{aCO2} \) gradient were lower in PCV group (Table 4). There was no difference in dynamic compliance and airway resistance, dead-space, dead-space-to-tidal volume ratio, and CO2 output. The alveolar-to-arterial oxygenation gradient (Table 4) was lower in the PCV group than in the VCV group (28.5 vs 34.9 kPa, \( P<0.05 \)).

There was no difference in anaesthesia-related variables between the two groups (Table 7).

After operation, there were no significant differences between the two groups. One patient suffered from respiratory acidosis with a pH value of 7.20 and \( P_{aO2} \) 10 kPa; this was considered to be due to morphine.

After 2 h in the recovery room, nasal oxygen requirements were similar [mean (sd) (range) 2 (2) (0–4) litre min\(^{-1}\) for PCV and 3 (2) (0–9) litre min\(^{-1}\) for VCV], as were total morphine doses [8 (7) (0–20) mg for PCV and 7 (6) (0–20) mg for VCV].

### Discussion

This study comparing VCV and PCV using two different algorithms to set mechanical ventilation during laparoscopic gastric banding in obese patients has shown differences in arterial blood oxygenation (\( P_{aO2} \) and \( S_{aO2} \)) and
ventilation variables (pH and $P_{a\text{CO}_2}$) in favour of the PCV mode. These differences emerged although plateau pressures were similar in the two groups. These pressures reached 26 cm H$_2$O with both VCV and PCV when providing sufficient MV for CO$_2$ removal in all of our patients, substantiating the existence of ventilation problems in obese patients as previously reported. The mean $P_{a\text{CO}_2}$ (in mm Hg)/$F_{I\text{CO}_2}$ ratio in the VCV group was under 200 in these patients, with normal preoperative pulmonary function tests and blood gases, further signifying the perioperative impairment in respiratory function in these patients. Different ventilation strategies, including PEEP, pneumoperitoneum, and plateau pressure values. Furthermore, metabolic acidosis was not suspected in any patient and there was no difference in haemodynamics between the two groups as the haemodynamic variables, VCO$_2$, dead-space, and $V'/Q'$ were similar. Balick-Weber and colleagues recently demonstrated the absence of transoesophageal echocardiographic changes when switching from VCV to PCV during laparoscopic urological procedures. Oxygenation was not modified in this study but only 21 non-randomized non-obese patients were studied for 20 min of PCV, after VCV. In anaesthetized dogs, Baker and colleagues had already found that, while keeping $Vt$ and respiratory frequency constant, dead space to tidal volume ratio, $P_{a\text{CO}_2}$, and alveolar-to-arterial oxygenation gradient decreased whereas $P_{a\text{CO}_2}$, mean airway pressure, total dynamic compliance, and chest wall compliance increased using the decelerating flow when compared with constant flow. The increase that they found in the mean airway pressure [from 3.87 (1.86) to 5.03 (2.27) cm H$_2$O] in part explained the improvement in gas exchange but, because their decelerating insufflation mode did not have a pressure limit, the pressure–time curve was very different from the one observed in the PCV mode and it reached higher pressure values. However, we did not find any difference in mean airway pressure in this study. Al Saady and Bennett, comparing a decelerating flow with a constant flow during inspiration in 14 ventilated patients for respiratory failure, found a significant increase in $P_{a\text{CO}_2}$ and a reduction in the dead space to tidal volume ratio and in the alveolar-to-arterial oxygenation gradient, while $Vt$, Ti, respiratory rate, and $I/E$ ratio were kept unchanged. Their results are similar to ours except for the small changes in $P_{a\text{CO}_2}$ in our study. Unzueta and colleagues recently compared PCV with VCV during one-lung ventilation for thoracic surgery using a cross-over design and, similar to our study, found no differences in $Vt$ and plateau pressures, but also in arterial oxygenation unlike the findings in our study. Nevertheless, their study patients were not obese and the time periods allocated to each mode were limited to 30 min during one-lung ventilation.

The PCV mode uses a decelerating inspiratory flow and provides the highest possible flow value. This option is available on all recent anaesthesia ventilators, even though only the models fitted with a piston or a turbine work in the same way as an intensive care ventilator. With the earlier ventilators, instantaneous flow often could not be set, or it did not exceed 50 litre min$^{-1}$ but was high enough to reach the chosen plateau pressure rapidly in nearly all situations commonly encountered during anaesthesia. However, an insufficient flow in the PCV mode can lead to a decrease in tidal volume. An intensive care ventilator able to generate a high enough flow ($>150$ litre min$^{-1}$) to reach plateau pressure with a steep slope was therefore chosen for our obese patients.

The three key determinants of $P_{a\text{CO}_2}$ are inspired oxygen pressure, alveolar ventilation, and ventilation/perfusion ratio. Since we set $F_{I\text{CO}_2}$ at 0.6 for all patients, the reason for the difference in oxygenation between VCV and PCV would be a change in the lung ventilation/perfusion ratio. For a given tidal volume, inspiratory flow reaches much higher values with the PCV than with the VCV mode. In our study, it was 52 litre min$^{-1}$ in PCV group and 41 litre min$^{-1}$ in VCV group. Consequently, 67% of the $Vt$ was delivered at half inspiratory time (excluding plateau time) in PCV and 53% in VCV group. Thus, we hypothesize that for the highest flow in the PCV mode, mean airway and plateau pressures measured at the end of inspiration grossly underestimate the instantaneous regional pressures reached in the lungs at the beginning of insufflation. Alveoli with short time constant may be initially over inflated, but then a more homogeneous distribution of the $Vt$ in all the ventilated alveoli could follow, reducing the amount of atelectasis by an improved alveolar recruitment. Furthermore, even if inspiratory flow is very low at the end of inspiration in PCV, only in VCV it drops to zero during the whole plateau time. The better preserved ventilation/perfusion ratio during PCV mode is marked by
a difference in the alveolar-to-arterial oxygenation gradient, in our study, this was 28.5 kPa in the PCV group and 34.9 kPa in the VCV group. Also, differences in the $V_{\text{A}} - V_{\text{a}}$ gradient, pH and $P_{\text{a}}$CO$_2$ in the two groups, despite the similar values for MV, support the hypothesis of a better ventilation/perfusion ratio in the PCV group.

Extending sufficiently the plateau time in VCV, and thus increasing inspiratory flow, might provide the same of a better ventilation/perfusion ratio in the PCV group.

Each patient had high intraoperative $P_{\text{a}}$O$_2$ values in our study but the supplemental amount of oxygen given by PCV gives the anaesthesiologist some more security in the obese patient whose non-hypoxic apnoea duration is very short.

The absence of difference in the postoperative $P_{\text{a}}$O$_2$ measurements can be explained by postoperative atelectasis due to ventilation with an $I_{\text{E}}$, set to 1.0 before extubation and to general anaesthesia which generates persistent atelectasis in the morbidly obese patients.

Preoperative pulmonary function tests did not show any difference between the two groups. Only 13 of our patients underwent a metacholine test and sensitive patients did not receive preoperative treatment as a matter of routine. It is currently not clear whether obesity, bronchial hyper reactivity, and asthma are related.

The limitations of this study are that it is single-blinded, it lacks invasive haemodynamic monitoring and that it could lack the power to detect a slight difference between MV and VT between the two groups. Such a difference would not be important clinically. In addition, using PCV routinely requires a good knowledge of its operating principles and a careful setting of the alarm limits, particularly the MV and the VT alarms; a sudden change in the patient’s compliance could increase or lower those two variables.

In conclusion, PCV compared with VCV during anaesthesia for laparoscopic bariatric surgery improves gas exchanges without increasing ventilation pressures or causing any haemodynamic side-effects.

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