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RESPIRATION AND THE AIRWAY

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>35 kg m^{-2}, no major obstructive or restrictive respiratory disorder, and Pa_{CO_2}<6.0 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 Pa_{O_2} (FI_{O_2} at 0.6 in each group) and Pa_{CO_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, Pa_{O_2}, Sa_{O_2}, and the Pa_{O_2}/FI_{O_2} ratio were higher in the PCV group, whereas Pa_{CO_2} and the $E'_{CO_2}-Pa_{CO_2}$ 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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Difficulties in ventilation are frequently encountered during anaesthesia in morbidly obese patients. These difficulties are related to difficult intubation[1–3] and mask ventilation,[3–5] restrictive and obstructive syndromes,[6 7] and abnormal ventilatory mechanics during laparoscopic surgery.[3 8–10] The use of volume-controlled ventilation (VCV) is common, as this has been the only available mode on ventilators for a long time. This mode utilizes a constant flow (Fig. 1) to deliver a target tidal volume (Vt) and thus insures a satisfactory minute ventilation (MV), despite frequently seen high-pressure levels in obese patients.[8] The mechanical consequences of reduced lung compliance and chest wall compliance, added to the reduction of functional residual capacity induced by the surgical pneumoperitoneum, explain impaired alveolar ventilation and the high pressures.[3 8 11]

Pressure-controlled ventilation (PCV) has been proposed as an alternative to VCV in ICU patients with adult respiratory distress syndrome,[12 13] and in obese patients to achieve adequate oxygenation and normocapnia.[9] The two differences between VCV and PCV are the flow pattern and the chosen target: PCV uses a decelerating flow

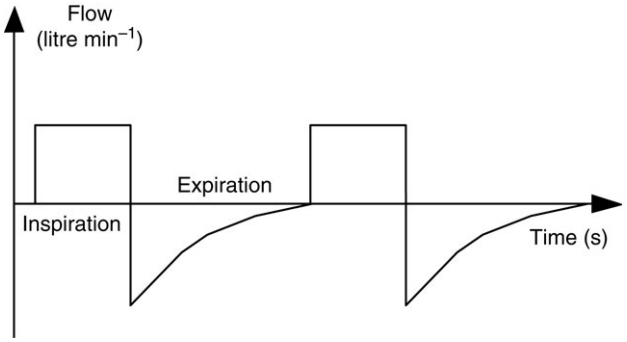

Fig 1 VCV flow pattern, constant flow insufflation.

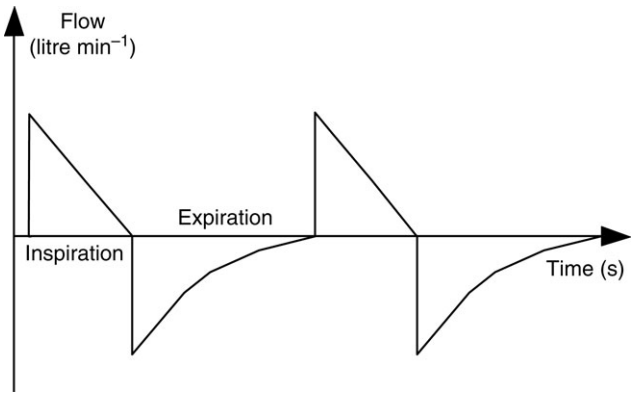

Fig 2 PCV flow pattern, decelerating flow insufflation.

(Fig. 2), which reaches the highest possible value at the beginning of inspiration, while having a preset pressure limitation but no minimum Vt. Flow diminishes throughout inspiration according to the pressure target, and the resulting Vt depends on the pressure limitation and on the chest compliance. These characteristics of PCV (faster tidal volume delivery, different gas distribution, and high and decelerating inspiratory flow) tend to compensate for any potential reduction in ventilation caused by pressure limitation.[14] Furthermore, the limitation of pressure levels has a positive effect on the patient's haemodynamics and might even reduce the risk of barotrauma.[12] Owing to these theoretical advantages, and our clinical experience, we suggested that, in obese patients undergoing laparoscopy, PCV could provide sufficient MV, ensuring adequate CO_2 removal and improved oxygenation, while using a lower plateau pressure than VCV.

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.

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 Pa_{CO_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 E'_{CO_2}, inability to remove the tracheal tube in the operating room, and conversion to laparotomy.

The preoperative evaluation included Epworth Sleepiness Scale[15] 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 Pa_{O_2} and Pa_{CO_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 CO_2 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 μg ml^{-1} of propofol[16] and 0.3 ng ml^{-1} of sufentanil.[17] 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 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

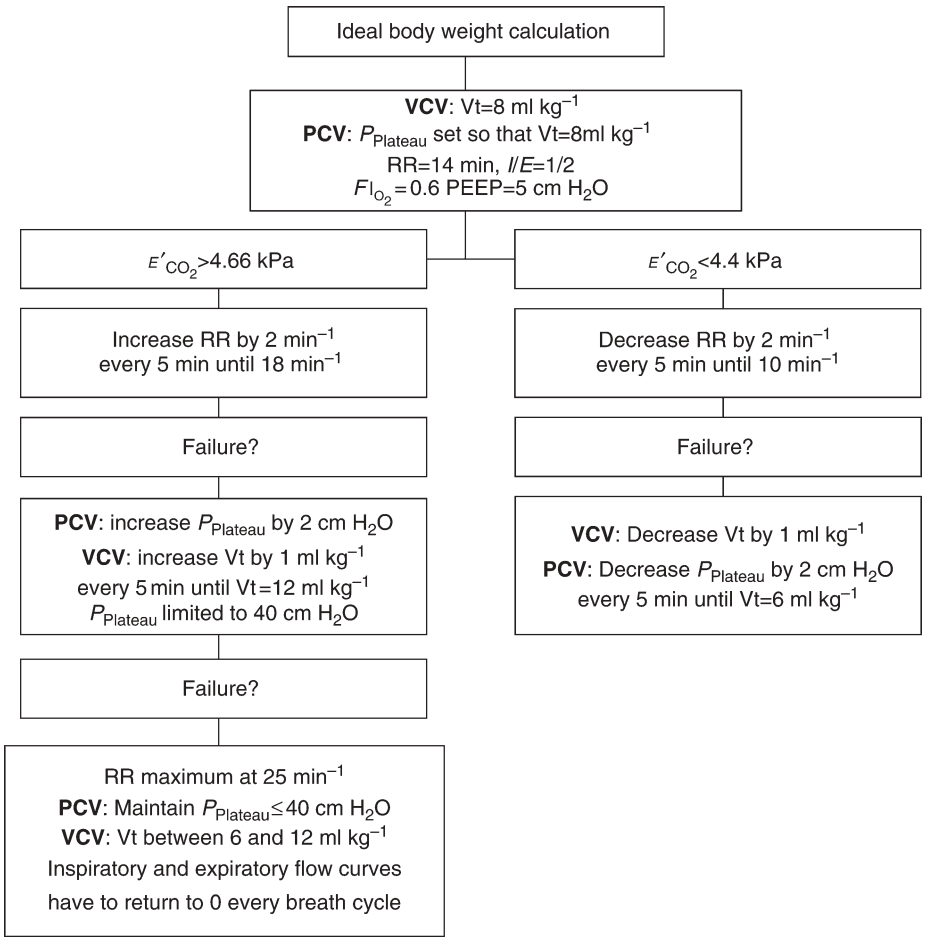

Fig 3 Algorithm for VCV and PCV settings. Vt, tidal volume; RR, respiratory rate; PEEP, positive end-expiratory pressure; $P_{plateau}$, plateau pressure; VCV, volume-controlled ventilation; PCV, pressure-controlled ventilation; *I*/*E*, inspiratory to expiratory time ratio; $F_{I_{O_2}}$, inspired fraction of oxygen; E'_{CO_2}, end-tidal CO_2.

H_2O, 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_2O was considered too low for these patients.[6 8] Tidal volume was initially set at 8 ml kg^{-1} of ideal weight [i.e. 50+0.91×(height in cm−152.4) for men and 45.5+0.91×(height in cm−152.4) for women].[18] 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 (Ti), 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_2O 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 E'_{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}, E'_{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 Pa_{O_2} from the following equation:

$$P_{A_{O_2}} - Pa_{O_2} = 713 \times 0.6 - \frac{Pa_{CO_2}\,\text{meas.}}{(VCO_2\,\text{meas.}/VO_2\,\text{estim.})} - Pa_{O_2}\,\text{meas.}$$

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, $F_{I_{O_2}}$ 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, $S_{p_{O_2}}$, and total postoperative morphine doses were recorded. Nasal oxygen was given, if necessary, providing an $S_{p_{O_2}}$ 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_2O in plateau pressure between the two groups, and the standard deviation of plateau pressure values to be 3.1 cm H_2O. 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). *P*-value of <0.05 is considered significant.

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, E'_{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_2O) were relatively low. One patient in VCV group reached a respiratory rate of 22 bpm but, as 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

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

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

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

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_{O_2}/Pa_{CO_2}, arterial oxygen/carbon dioxide partial pressure; Sa_{O_2}, arterial oxygen saturation; TCO_2, total carbon dioxide; LVEF, left ventricular ejection fraction. **n*=17, †*n*=16, ‡*n*=7, ¶*n*=6, §*n*=15. Arterial blood gases were performed in room air. There was no significant difference between the two groups

	PCV	VCV
FEV1 (litre s^{-1})	3 (0.8) (1.7–5)	3 (0.8) (1.9–5.3)
FVC (litre)	3.5 (0.9) (2.1–5.1)	3.4 (0.95) (2.1–5.8)
FEV1/FVC	84 (3) (78–89)	85 (5) (77–93)
TLC (litre)	5 (1.2)* (3–7.5)	5 (1.1)† (3.3–6.6)
Bronchial hyper-reactivity	3‡	4¶
pH	7.42 (0.02) (7.40–7.5)	7.41 (0.02)† (7.40–7.44)
Pa_{O_2} (kPa)	11.7 (1.3)† (9.2–14.9)	11.7 (1.8)§ (9.4–16.0)
Pa_{CO_2} (kPa)	5.2 (0.5) (4.1–6.0)	5.3 (0.4)† (4.5–6.2)
Sa_{O_2} (%)	98 (1)* (96–99)	98 (1)† (95–100)
TCO_2 (mmol $litre^{-1}$)	25 (2) (20–27)	26 (2)§ (23–28)
LVEF (%)	67 (5)¶ (58–70)	66 (8)‡ (60–79)

Values for 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_{O_2}, Sa_{O_2}, and

Table 4 Intraoperative ventilation variables. Mean (SD) (range). PCV, pressure-controlled ventilation; VCV, volume-controlled ventilation; Ti, inspiratory time; E'_{CO_2}, end-tidal CO_2; VCO_2, carbon dioxide output; VO_2, oxygen delivery; $P_{A_{O_2}}$, partial pressure of oxygen in alveoli. *Mann–Whitney test; †*t*-test; ‡*n*=17

	PCV (*n*=18)	VCV (*n*=18)	*P*-value
Respiratory rate (bpm)	18 (0.5) (16–18)	18 (1.0) (18–22)	NS
Tidal volume (Vt) (ml)	613 (91) (481–858)	573 (81) (430–700)	NS
Vt ml kg^{-1} ideal wt	11 (1.4) (8.8–13.2)	10.2 (1.2) (8.13–12.43)	NS
Vt ml kg^{-1} true wt	5.1 (1.0) (3.7–6.9)	4.8 (0.6) (3.9–5.8)	NS
%Vt at Ti/2	67 (5) (58–78)	53 (5) (46–71)	<0.01*
Minute volume (litre min^{-1})	10.9 (1.8) (7.2–15.5)	10.6 (1.8) (8.3–14.2)	NS
Peak pressure (cm H_2O)	–	33 (4) (25–41)	–
Plateau pressure (cm H_2O)	26 (4) (20–33)	27 (3) (20–30)	NS
Mean airway pressure (cm H_2O)	12 (1) (10–15)	12 (2) (10–15)	NS
Peak inspiratory flow (litre s^{-1})	52 (7) (39–63)	41 (7) (32–57)	<0.01†
Sp_{O_2} (%)	99 (1) (97–100)	98 (3) (93–100)	NS
E'_{CO_2} (kPa)	4.5 (0.13) (4.3–4.9)	4.5 (0.13) (4.3–4.7)	NS
E'_{CO_2}–Pa_{CO_2} gradient (kPa)	0.67 (0.27) (0–1.33)	0.93 (0.27) (0.67–1.33)	<0.01†
Dynamic compliance (ml cm H_2O^{-1})	30 (4) (20–36)	30 (6) (18–41)	NS
Resistance (cm H_2O $litre^{-1}$ s^{-1})	17 (5) (11–27)	17 (5) (12–28)	NS
Dead space (ml)	189 (27) (114–244)	176 (26) (132–217)	NS
Dead space/Vt	0.31 (0.04) (0.19–0.37)	0.31 (0.05) (0.20–0.39)	NS
VCO_2 (ml min^{-1})	276 (51) (191–360)	275 (57) (195–366)	NS
VO_2 (ml min^{-1})=130×body surface area	306 (32) (248–389)	302 (26) (268–358)	NS
Estimated $P_{A_{O_2}}$–measured Pa_{O_2} (kPa)	28.5 (8.2) (12.4–37.0)‡	34.9 (8.0) (21.4–43.2)	0.01*

Table 5 Haemodynamic variables in the two groups. Mean (SD) (range). PCV, pressure-controlled ventilation; VCV, volume-controlled ventilation. There were no differences between the groups

	Intraoperative		Postoperative	
	PCV	VCV	PCV	VCV
Systolic arterial pressure (mm Hg)	131 (21) (100–180)	119 (22) (88–159)	129 (16) (105–160)	127 (21) (95–187)
Diastolic arterial pressure (mm Hg)	81 (16) (49–104)	76 (15) (54–102)	76 (12) (52–94)	79 (15) (53–109)
Mean arterial pressure (mm Hg)	96 (17) (67–125)	89 (16) (66–115)	98 (16) (71–138)	94 (19) (66–136)
Heart rate (beats min^{-1})	84 (15) (57–117)	77 (14) (46–101)	76 (16) (50–111)	76 (18) (40–111)

Table 6 Arterial blood gases in the two groups; mean (SD) (range). PCV, pressure-controlled ventilation; VCV, volume-controlled ventilation; $F_{I_{O_2}}$, fraction of inspired oxygen; TCO_2, total carbon dioxide. *n =17. Fisher's *t*-test for all variables except for Sa_{O_2} (Mann–Whitney test); NS, not significant

	Intraoperative			Postoperative		
	PCV	VCV	*P*-value	PCV	VCV	*P*-value
pH	7.40 (0.03)[a] (7.34–7.46)	7.38 (0.02) (7.33–7.43)	0.041	7.36 (0.02) (7.32–7.40)	7.36 (0.05) (7.20–7.40)	NS
Pa_{O_2} (kPa)	22.5 (8.5)* (13.1–40.2)	15.9 (5.9) (8.4–28.9)	0.011	16.1 (5.2) (9.5–30.1)	14.8 (4.5) (8.3–24.8)	NS
Pa_{CO_2} (kPa)	5.2 (0.4) (4.4–6.0)	5.4 (0.3) (5.2–6.0)	0.014	5.9 (0.4) (5.0–6.7)	6.0 (1.2) (5.2–10.0)	NS
Sa_{O_2} (%)	99 (1)* (98–100)	98 (2) (92–100)	0.010	98 (2) (94–100)	98 (2) (92–100)	NS
TCO_2 (mmol $litre^{-1}$)	25 (1) (22–28)	25 (2) (22–28)	NS	26 (1) (24–28)	26 (2) (23–30)	NS
Pa_{O_2} (in mm Hg)/$F_{I_{O_2}}$	281 (107) (163–503)	199 (74) (105–362)	0.011			

the $Pa_{O_2}/F_{I_{O_2}}$ ratio were higher in the PCV group ($P<0.05$). Pa_{CO_2} and E'_{CO_2}–Pa_{CO_2} 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 CO_2 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 Pa_{CO_2} 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 (Pa_{O_2} and Sa_{O_2}) and

Table 7 Anaesthesia-related variables; mean (SD) (range). There were no significant differences between the two groups

	PCV (n=18)	VCV (n=18)
Bispectral index	32 (11) (10–50)	31 (8) (14–45)
Propofol (g)	1.78 (0.46) (1.22–2.85)	1.81 (0.72) (1.05–3.45)
Sufentanil (μg)	61 (24) (37–135)	56 (10) (40–78)
Atracurium (mg)	61 (14) (50–100)	61 (14) (40–90)
Operative time (min)	73 (24) (44–125)	83 (40) (45–193)
Pneumoperitoneum (mm Hg)	13 (1) (11–14)	13 (1) (11–15)

ventilation variables (pH and Pa_{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_2O 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.[6] The mean Pa_{O_2} (in mm Hg)/$F_{I_{O_2}}$ 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[19] or high Vt,[9 20] with variable effects have been proposed to improve oxygenation during laparoscopic surgery. Recruitment manoeuvres have been shown to be effective intraoperatively.[21]

To explain the higher Pa_{O_2} values for the same Vt using the PCV mode, higher plateau pressures could have been expected, but this was not the case in our study. We calculated the mean airway pressure from our data recordings which is a key variable in alveolar gas exchange. It is related to mean alveolar pressure and is used to assess total lung volume.[22] We found no difference in mean airway pressure between VCV and PCV for identical MV, 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 E'_{CO_2} were similar. Balick-Weber and colleagues[23] 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[24 25] had already found that, while keeping Vt and respiratory frequency constant, dead space to tidal volume ratio, Pa_{CO_2}, and alveolar-to-arterial oxygenation gradient decreased whereas Pa_{O_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_2O] 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 Pa_{O_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 Pa_{CO_2} in our study.[26] Unzueta and colleagues[27] 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 Pa_{O_2} are inspired oxygen pressure, alveolar ventilation, and ventilation/perfusion ratio. Since we set $F_{I_{O_2}}$ 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.[26] 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;[28] in our study, this was 28.5 kPa in the PCV group and 34.9 kPa in the VCV group. Also, differences in the $E'_{CO_2}-Pa_{CO_2}$ gradient, pH and Pa_{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 effects on gas exchange as those observed in PCV.

Each patient had high intraoperative Pa_{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.[29]

The absence of difference in the postoperative Pa_{O_2} measurements can be explained by postoperative atelectasis due to ventilation with an FI_{O_2} set to 1.0 before extubation and to general anaesthesia which generates persistent atelectasis in the morbidly obese patients.[9 10]

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.[30]

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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References

1 Voyagis GS, Kyriakis KP, Dimitriou V, Vrettou I. Value of oropharyngeal Mallampati classification in predicting difficult laryngoscopy among obese patients. *Eur J Anaesthesiol* 1998; **15**: 330–4

2 el-Ganzouri AR, McCarthy RJ, Tuman KJ, Tanck EN, Ivankovich AD. Preoperative airway assessment: predictive value of a multivariate risk index. *Anesth Analg* 1996; **82**: 1197–204

3 Adams JP, Murphy PG. Obesity in anaesthesia and intensive care. *Br J Anaesth* 2000; **85**: 91–108

4 Langeron O, Masso E, Huraux C, *et al*. Prediction of difficult mask ventilation. *Anesthesiology* 2000; **92**: 1229–36

5 Kheterpal S, Han R, Tremper KK, *et al*. Incidence and predictors of difficult and impossible mask ventilation. *Anesthesiology* 2006; **105**: 885–91

6 Pelosi P, Croci M, Ravagnan I, *et al*. Respiratory system mechanics in sedated, paralyzed, morbidly obese patients. *J Appl Physiol* 1997; **82**: 811–8

7 Biring MS, Lewis MI, Liu JT, Mohsenifar Z. Pulmonary physiologic changes of morbid obesity. *Am J Med Sci* 1999; **318**: 293–7

8 Sprung J, Whalley DG, Falcone T, Warner DO, Hubmayr RD, Hammel J. The impact of morbid obesity, pneumoperitoneum, and posture on respiratory system mechanics and oxygenation during laparoscopy. *Anesth Analg* 2002; **94**: 1345–50

9 Ogunnaike BO, Jones SB, Jones DB, Provost D, Whitten CW. Anesthetic considerations for bariatric surgery. *Anesth Analg* 2002; **95**: 1793–805

10 Eichenberger A, Proietti S, Wicky S, *et al*. Morbid obesity and postoperative pulmonary atelectasis: an underestimated problem. *Anesth Analg* 2002; **95**: 1788–92, table of contents

11 Amato MB, Barbas CS, Medeiros DM, *et al*. Beneficial effects of the 'open lung approach' with low distending pressures in acute respiratory distress syndrome. A prospective randomized study on mechanical ventilation. *Am J Respir Crit Care Med* 1995; **152**: 1835–46

12 Mercat A, Graini L, Teboul JL, Lenique F, Richard C. Cardiorespiratory effects of pressure-controlled ventilation with and without inverse ratio in the adult respiratory distress syndrome. *Chest* 1993; **104**: 871–5

13 Prella M, Feihl F, Domenighetti G. Effects of short-term pressure-controlled ventilation on gas exchange, airway pressures, and gas distribution in patients with acute lung injury/ARDS: comparison with volume-controlled ventilation. *Chest* 2002; **122**: 1382–8

14 Davis K Jr, Branson RD, Campbell RS, Porembka DT. Comparison of volume control and pressure control ventilation: is flow waveform the difference? *J Trauma* 1996; **41**: 808–14

15 Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep* 1991; **14**: 540–5

16 Schnider TW, Minto CF, Gambus PL, *et al*. The influence of method of administration and covariates on the pharmacokinetics of propofol in adult volunteers. *Anesthesiology* 1998; **88**: 1170–82

17 Gepts E, Shafer SL, Camu F, *et al*. Linearity of pharmacokinetics and model estimation of sufentanil. *Anesthesiology* 1995; **83**: 1194–204

18 The ARDS Network. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. The Acute Respiratory Distress Syndrome Network. *N Engl J Med* 2000; **342**: 1301–8

19 Pelosi P, Ravagnan I, Giurati G, *et al*. Positive end-expiratory pressure improves respiratory function in obese but not in normal subjects during anesthesia and paralysis. *Anesthesiology* 1999; **91**: 1221–31

20 Sprung J, Whalley DG, Falcone T, Wilks W, Navratil JE, Bourke DL. 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, table of contents

21 Whalen FX, Gajic O, Thompson GB, *et al*. 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

22 Marini JJ, Ravenscraft SA. Mean airway pressure: physiologic determinants and clinical importance—Part 2: clinical implications. *Crit Care Med* 1992; **20**: 1604–16

23 Balick-Weber CC, Nicolas P, Hedreville-Montout M, Blanchet P, Stephan F. Respiratory and haemodynamic effects of volume-controlled vs pressure-controlled ventilation during laparoscopy: a cross-over study with echocardiographic assessment. *Br J Anaesth* 2007; **99**: 429–35

24 Baker AB, Babington PC, Colliss JE, Cowie RW. Effects of varying inspiratory flow waveform and time in intermittent positive pressure ventilation. I: introduction and methods. *Br J Anaesth* 1977; **49**: 1207–20

25 Baker AB, Colliss JE, Cowie RW. Effects of varying inspiratory flow waveform and time in intermittent positive pressure ventilation. II: various physiological variables. *Br J Anaesth* 1977; **49**: 1221–34

26 Al-Saady N, Bennett ED. Decelerating inspiratory flow waveform improves lung mechanics and gas exchange in patients on intermittent positive-pressure ventilation. *Intensive Care Med* 1985; **11**: 68–75

27 Unzueta MC, Casas JI, Moral MV. Pressure-controlled versus volume-controlled ventilation during one-lung ventilation for thoracic surgery. *Anesth Analg* 2007; **104**: 1029–33, tables of contents

28 Guyton AC, Hall JE. Transport of oxygen and carbon dioxide in the blood and body fluids. In: Saunders W, ed. *Textbook of Medical Physiology*, 9th Edn. Philadelphia: Elsevier, 1996; 513–23

29 Gander S, Frascarolo P, Suter M, Spahn DR, Magnusson L. Positive end-expiratory pressure during induction of general anesthesia increases duration of nonhypoxic apnea in morbidly obese patients. *Anesth Analg* 2005; **100**: 580–4

30 Chinn S. Concurrent trends in asthma and obesity. *Thorax* 2005; **60**: 3–4