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Comparison of volume controlled with pressure controlled ventilation during one-lung anaesthesia

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Summary

Pressure controlled ventilation (PCV) is an alternative mode of ventilation which is used widely in severe respiratory failure. In this study, PCV was used for one-lung anaesthesia and its effects on airway pressures, arterial oxygenation and haemodynamic state were compared with volume controlled ventilation (VCV). We studied 48 patients undergoing thoracotomy. After two-lung ventilation with VCV, patients were allocated randomly to one of two groups. In the first group ($n=24$), one-lung ventilation was started by VCV and the ventilation mode was then switched to PCV. Ventilation modes were performed in the opposite order in the second group ($n=24$). We observed that peak airway pressure ($P=0.000001$), plateau pressure ($P=0.01$) and pulmonary shunt ($P=0.03$) were significantly higher during VCV, whereas arterial oxygen tension ($P=0.02$) was significantly higher during PCV. Peak airway pressure (*Paw*) decreased consistently during PCV in every patient and the percentage reduction in *P*aw was 4–35% (mean 16.1 (SD 8.4) %). Arterial oxygen tension increased in 31 patients using PCV and the improvement in arterial oxygenation during PCV correlated inversely with preoperative respiratory function tests. We conclude that PCV appeared to be an alternative to VCV in patients requiring one-lung anaesthesia and may be superior to VCV in patients with respiratory disease. (*Br. J. Anaesth.* 1997; **79**: 306–310).

Key words

Ventilation, one-lung. Ventilation, volume controlled. Ventilation, pressure controlled.

During one-lung anaesthesia, arterial hypoxaemia is a major concern.[1] Volume controlled ventilation (VCV) is the traditional method of performing one-lung anaesthesia in patients undergoing thoracic surgery and an increase in airway pressure is usually observed. If the method of ventilation involves excessive amounts of airway pressure, vascular resistance of the dependent lung may be increased because of compression of intra-alveolar vessels.[2] Thus high airway pressures of the dependent lung may counteract hypoxic pulmonary vasoconstriction in the non-dependent lung by diverting blood flow away from the ventilated lung, thereby increasing pulmonary shunt fraction.[3]

One-lung ventilation implies delivering the entire tidal volume into one lung, which may result in barotrauma of the dependent lung. In order to avoid high airway pressures, anaesthetists may ventilate the dependent lung with lower tidal volumes and higher ventilatory frequencies during one-lung anaesthesia. However, lower tidal volumes have been demonstrated to predispose the dependent lung to atelectasis and worsen arterial oxygenation.[4,5]

Pressure controlled ventilation (PCV) is an alternative mode of ventilation which is used widely in severe respiratory failure. PCV has been shown to improve arterial oxygenation and decrease peak airway pressure because of its decelerating inspiratory flow.[6] Uniform distribution of inspired gas with PCV is the major cause of better arterial oxygenation in patients with respiratory failure.[7–10]

We applied PCV to one-lung anaesthesia and examined its use as a tool to improve ventilatory management of patients undergoing thoracic surgery. In our study, PCV was used for one-lung anaesthesia and its effects on airway pressure, arterial oxygenation and haemodynamic state were compared with those produced by VCV.

Patients and methods

The study was approved by the University of Istanbul Ethics Committee and all patients gave informed consent. We studied 48 patients, ASA I–III, undergoing thoracotomy requiring one-lung ventilation. On the evening before surgery, spirometry (forced vital capacity (FVC) and forced expiratory volume in 1 s (FEV_1)) was performed in the sitting position. An arterial blood-gas sample was also obtained with the patient breathing air. Patients were excluded if they had a history of cardiac, hepatic or renal disease.

All patients were premedicated with diazepam 0.15 mg kg^{-1} orally, 1 h before arrival in the operating room. On admission to the operating room, i.v.

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and intra-arterial cannulae were inserted under local anaesthesia and an ECG was attached. After pre-oxygenation, anaesthesia was induced with fentanyl 2 $\mu g\,kg^{-1}$ and propofol 2–2.5 $mg\,kg^{-1}$; tracheal intubation was facilitated with vecuronium 0.1 $mg\,kg^{-1}$. The trachea was intubated with an Univent tracheal tube (Fuji Corp, Japan). The bronchial blocker of the Univent tube was inserted into the mainstem bronchus of the operative side guided by a fibreoptic bronchoscope and separation of the lungs was demonstrated by capnography.[11] With this method, the tracing from the end-tidal carbon dioxide analyser connected to the proximal end of the bronchial blocker of the Univent shows a typical respiratory waveform. As the bronchial blocker cuff is gradually inflated, a straight line on the waveform indicates that lung isolation has occurred.

After induction of anaesthesia a 7-F pulmonary artery catheter (Abbott, Criticare Systems, USA) was inserted via the right internal jugular vein. Isoflurane 1–1.5%, fentanyl 0.05–0.1 $\mu g\,kg^{-1}\,min^{-1}$ and vecuronium 0.03 $mg\,kg^{-1}$ every 30 min were used for maintenance of anaesthesia. Fentanyl bolus doses of 50–100 μg and i.v. fluids were administered to maintain systemic arterial pressure within $\pm 20\%$ of pre-induction values. Oesophageal temperature was monitored throughout the procedure and maintained greater than 36.5 °C by a heating blanket (Biçakçilar, Turkey) placed under the patient.

Our study consisted of three steps. Two-lung ventilation (TLV) with VCV (TLV-VCV) was performed in the lateral decubitus position in all patients (Servo 900C, Siemens, Stockholm, Sweden). On initiation of one-lung ventilation, patients were allocated randomly to one of two groups. In the first group ($n=24$), one-lung ventilation was started by VCV (OLV-VCV) and the ventilation mode was then changed to PCV (OLV-PCV). The modes of ventilation were performed in the opposite order in the second group ($n=24$).

During TLV-VCV and OLV-VCV, patients' lungs were ventilated with a tidal volume of 10 $ml\,kg^{-1}$ and ventilatory frequency was adjusted to maintain arterial carbon dioxide tension (Pa_{CO_2}), at 4.5–6 kPa. PCV was initiated with a peak airway pressure that provided a tidal volume of 10 $ml\,kg^{-1}$. Ventilatory frequency was adjusted to maintain Pa_{CO_2}, at 4.5–6 kPa during OLV-PCV. With both PCV and VCV, we used a 25% inspiratory time and a 10% pause time. Inspiratory oxygen concentration ($F_{I_{O_2}}$) was adjusted to 0.5 in air during two-lung ventilation and to 1.0 during one-lung ventilation. End-tidal concentrations of carbon dioxide and isoflurane were monitored using a Criticare 1100 (Criticare Inc, USA).

In accordance with data obtained from previous studies,[12 13] all measurements were made 30 min after initiation of the ventilation mode. All operations were performed by the same surgical team and the study was completed before any pulmonary vessels were ligated. Measurements were completed during the minutes in which the surgeons stopped compressing the operative lung.

The following variables were measured and recorded at the end of each period. Arterial and venous oxygen tensions and saturations (Pa_{O_2}, Sa_{O_2}, $S\bar{v}_{O_2}$), Pa_{CO_2} and haemoglobin (Hb) were analysed within 5 min using ABL 505 and OSM3 Hemoximeter (Radiometer, Copenhagen, Denmark). Heart rate (HR), mean arterial pressure (MAP), pulmonary artery pressure (MPAP), central venous pressure (CVP) and pulmonary artery occlusion pressure (PAOP) were recorded from the Criticare 1100 (Criticare Inc, USA). Deltran II transducers (Utah Medical, USA) were used to measure pulmonary arterial and systemic pressures. The zero reference was the mid-chest level and vascular pressures were measured at end-expiration. Cardiac output (CO) was measured by a thermodilution technique using injection of 10 ml of 0.9% saline at 0 °C (Abbott Criticare Systems, USA). The mean value of three measurements was obtained. Pulmonary shunt (Q_s/Q_t) was calculated from standard formulae.[14]

Peak airway pressure (Paw) and mean airway pressure (Pmaw) were recorded during each mode of ventilation. Inspiratory plateau was established by activating the inspiratory hold on the Siemens Servo 900C ventilator. Inspiratory plateau pressure (Pplt) was measured during the last 0.3 s of each hold manoeuvre.

We studied Pa_{O_2} data in order to identify which patients would benefit from OLV-PCV. The relationship between "individual Pa_{O_2} differences between OLV-PCV and OLV-VCV" and respiratory function tests was studied by linear regression analysis.

Data are presented as mean (SD). Paired Student's t test, ANOVA and chi-square test were used to test the significance of the differences between groups. Statistical significance was assumed at $P=0.05$.

Results

Patient data are shown in table 1. Mean values for ventilation, gas exchange and haemodynamic variables at each time are presented in tables 2 and 3. During one-lung ventilation, arterial hypoxaemia (Sa_{O_2} <90%) requiring reinflation of the collapsed lung did not occur in any patient.

Pa_{CO_2}, Sa_{O_2}, $S\bar{v}_{O_2}$, Hb, MAP, MPAP, PAOP, CVP and CO did not differ significantly between any of the ventilation modes.

Comparison of the OLV-VCV and OLV-PCV modes revealed significant differences in Paw, Pplt,

Table 1 Patient data. FVC = Forced vital capacity, FEV_1 = forced expiratory volume in 1 s, Pa_{O_2} = arterial oxygen tension, Pa_{CO_2} = arterial carbon dioxide tension (mean (SD) [range] or number) ($n=48$)

Age (yr)	56.4 [30–78]
Weight (kg)	70.3 (11.5) [46–95]
Sex (M/F)	38/10
Right-left-sided thoracotomy	28/20
Lobectomy/pneumonectomy	29/19
Preoperative FVC (% predicted)	77.1 (15) [46–117]
Preoperative FEV_1 (% predicted)	76.8 (14) [43–112]
Preoperative Pa_{CO_2} (kPa)	5 (0.63) [3.95–6.7]
Preoperative Pa_{O_2} (kPa)	10.2 (1.12) [7.9–12.8]

Table 2 Ventilation and gas exchange data (mean (SD)). *P*aw = Peak airway pressure, *P*plt = plateau pressure, *P*maw = mean airway pressure, Pa_{O_2} = arterial oxygen tension, Sa_{O_2} = arterial oxygen saturation, Pa_{CO_2} = arterial carbon dioxide tension, SV_{O_2} = mixed venous oxygen saturation, *Q*s/*Q*t = intrapulmonary shunt, TLV = two-lung ventilation, OLV = one-lung ventilation, PCV = pressure controlled ventilation, VCV = volume controlled ventilation. $^*P<0.05$ compared with OLV-VCV, $^{***}P<0.001$ compared with OLV-VCV

	TLV-VCV	OLV-VCV	OLV-PCV
Tidal volume (ml)	708 (112)	714 (111)	710 (114)
*P*aw (cm H_2O)	21.4 (4.7)	28.3 (5.1)	23.65 (3.85)***
*P*plot (cm H_2O)	15.7 (3.8)	18.5 (4.2)	17.8 (3.7)*
*P*maw (cm H_2O)	6.7 (1.4)	7.8 (2)	7.95 (1.55)
Pa_{O_2} (kPa)	27.5 (7.9)	28.4 (15.6)	32.3 (14.4)*
Sa_{O_2} (%)	99.1 (0.98)	98.3 (3.1)	98.7 (2.1)
Pa_{CO_2} (kPa)	4.8 (0.77)	5.06 (0.87)	5.05 (0.9)
SV_{O_2} (%)	80.7 (6.8)	81.2 (8.3)	81.4 (7.23)
*Q*s/*Q*t (%)	16.7 (6.2)	40.2 (10.1)	36.2 (10)*

Table 3 Haemodynamic data (mean (SD)). HR = Heart rate, MAP = mean arterial pressure, MPAP = mean pulmonary artery pressure, PAOP = pulmonary artery occlusion pressure, CVP = central venous pressure, CO = cardiac output, TLV = two-lung ventilation, OLV = one-lung ventilation, PCV = pressure controlled ventilation, VCV = volume controlled ventilation

	TLV-VCV	OLV-VCV	OLV-PCV
HR (beat min^{-1})	77.5 (11)	79.4 (9.8)	78.8 (10)
MAP (mm Hg)	94 (14)	92 (11)	92.7 (12)
MPAP (mm Hg)	18 (3.2)	18.6 (3)	19.1 (4.6)
PAOP (mm Hg)	15.1 (2.3)	15.5 (3.7)	15.7 (3.7)
CVP (mm Hg)	10.8 (3.7)	11.4 (3.6)	11.6 (3.6)
CO (litre min^{-1})	4.4 (0.9)	4.55 (0.8)	4.6 (0.87)

Pa_{O_2} and *Q*s/*Q*t. *P*aw was significantly lower during OLV-PCV than during OLV-VCV (23.65 (3.85) cm H_2O *vs* 28.3 (5.1) cm H_2O) ($P=0.000001$). Compared with OLV-VCV, *P*aw decreased consistently during OLV-PCV in every patient, ranging from 1 to 13.5 cm H_2O. The percentage *P*aw difference was 4–35% (mean 16.1 (8.4) %). *P*plt was also significantly lower during OLV-PCV than during OLV-VCV (17.8 (3.7) *vs* 18.5 (4.2) cm H_2O) ($P=0.01$).

Mean Pa_{O_2} was significantly higher during OLV-PCV (32.3 (14.4) kPa *vs* 28.4 (15.6) kPa) ($P=0.03$) than during OLV-VCV. Arterial oxygenation improved in 31 patients with OLV-PCV and in 17 patients with OLV-VCV ($P = 0.008$). Twenty-three of 31 patients (74%) who had an improved Pa_{O_2} value with OLV-PCV had an FVC lower than 77%, whereas only six of 17 (35%) patients who had an improved Pa_{O_2} with VCV had an FVC lower than 77% ($P=0.02$). Also, 19 of 31 patients (61%) whose Pa_{O_2} improved with OLV-PCV had an FEV_1 lower than 77%, whereas five of 17 (30%) patients whose Pa_{O_2} improved with OLV-VCV had an FVC lower than 77% ($P=0.07$). On the basis of linear regression analysis "individual Pa_{O_2} difference between OLV-PCV and OLV-VCV" showed a significant correlation with FVC ($r=-0.30$, $P=0.02$) (fig. 1), but no significant correlation with FEV_1 ($r=-0.24$, $P=0.09$).

Mean *Q*s/*Q*t was significantly higher during OLV-VCV (40.2 (10.1) *vs* 36.2 (10) %, $P=0.02$) than during OLV-PCV. *Q*s/*Q*t was found to be higher during OLV-VCV in 32 patients, whereas OLV-PCV resulted in higher *Q*s/*Q*t values in only 16 patients ($P=0.002$).

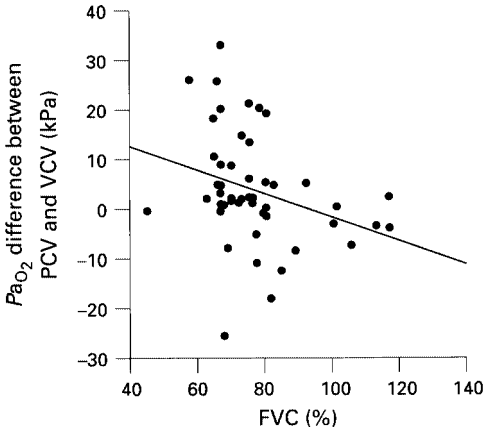

Figure 1 Individual Pa_{O_2} difference between PCV and VCV *vs* FVC (forced vital capacity) ($r=-0.30$, $P=0.02$) ($y=168.6-1.8x$).

Discussion

VCV is the common mode of ventilation used in patients undergoing one-lung anaesthesia. In our study, we performed pressure controlled ventilation to overcome some of the problems observed with one-lung anaesthesia. We planned to use almost the same tidal volumes to examine the effects of each mode on airway pressures, pulmonary shunt and arterial oxygenation. As tidal volumes greater than 10 ml kg^{-1} were reported to increase airway pressure and pulmonary vascular resistance in the dependent lung,[5 15 16] we chose a tidal volume of 10 ml kg^{-1} for the dependent lung which was reported to be least detrimental to arterial oxygenation.[17] As it has been documented previously that Pa_{O_2} tends to increase over 1 h after the onset of one-lung anesthesia,[18] we randomized the order of PCV and VCV.

High airway pressures may lead to barotrauma of the dependent lung.[19] It has been reported that high peak inspiratory pressure has some influence on the incidence of barotrauma[20] and avoiding high peak inspiratory pressures seems to be a safe form of ventilation.[21] In our study, peak inspiratory pressures were consistently lower during PCV. A reduction in peak inspiratory pressure of 4–35% induced by PCV is an advantage for one-lung anaesthesia.

It has been demonstrated previously that PCV is associated with sustained reductions in peak inspiratory pressure because of its decelerating flow profile.[7 8] In studies performed in patients with respiratory failure, peak inspiratory pressure was shown to decline from 38 to 33 cm H_2O[9] and from 43 to 40 cm H_2O[10] by PCV. The beneficial effects of reduced peak inspiratory pressures during PCV in terms of minimizing lung injury have been attributed to concomitant reductions in peak alveolar pressure and consequent decreases in alveolar distension.[22] However, there are no studies comparing the

incidence of iatrogenic lung injury using PCV or VCV. It was postulated that the very rapid initial flows with PCV may cause different effects on filling patterns, and thus the risk of barotrauma from shearing may be less using pressure *vs* volume breaths.[23]

There is a growing tendency to define end-inspiratory plateau pressure as a more important determinant of barotrauma than peak airway pressure.[24] In our study, plateau pressure was found to be significantly lower during PCV. Furthermore, it is possible during VCV for some alveoli to receive higher pressures during inspiration than the end-inspiratory plateau pressure. In contrast, in no parts of the lung can the pressure be higher than the preset pressure during PCV.[22]

Because of the high peak inspiratory pressures usually observed with the initiation of one-lung anaesthesia, most anaesthetists tend to ventilate the dependent lung with lower tidal volumes and higher respiratory frequencies aiming to protect the dependent lung from hyperinflation. However, lower tidal volumes have been demonstrated to predispose the dependent lung to develop atelectasis, thereby worsening arterial oxygenation.[4 5] We assume that lower airway pressures obtained with PCV allow the use of higher tidal volumes during one-lung anaesthesia.

Factors that play a role in the hypoxaemia frequently seen during one-lung anaesthesia include reduction in dependent lung volume caused by induction of anaesthesia, abdominal and mediastinal compression, suboptimal positioning, low ventilation: perfusion ratios and atelectasis because of a high $F_{I_{O_2}}$, and problems in removal of secretions.[25] Hypoxic pulmonary vasoconstriction is an auto-regulatory mechanism that diverts blood flow away from hypoxic, atelectatic lung towards the remaining normoxic, ventilated lung and it is very effective in reducing hypoxic lung blood flow when the percentage hypoxic lung is intermediate (30–70%), that is the amount of lung that is hypoxic during one-lung anaesthesia.[26]

Compression of small intra-alveolar vessels during inflation of alveoli produces increased resistance to pulmonary blood flow in the dependent lung. This increase in vascular resistance may divert blood flow to the non-ventilated alveoli of the non-dependent lung. Further increases in tidal volume and airway pressure in the dependent lung are then unlikely to improve oxygenation and may make arterial oxygenation worse by diverting blood flow to the non-dependent lung.[4 16 17] Although not necessarily reflecting intra-alveolar pressure, the high peak and plateau pressures observed with VCV in our study may play a role in diverting blood flow to the non-dependent lung and produce a higher pulmonary shunt.

One of the aims of this study was to investigate the effects of PCV on arterial oxygenation during one-lung anaesthesia. It was demonstrated earlier that improvement in arterial oxygenation by PCV was not related to variations in mean airway pressure but resulted mostly from the beneficial effects of the decelerating inspiratory flow pattern produced by PCV on the distribution of gas within the lung.[7] In diseased lungs with different time constants, the decelerating flow of PCV sustains alveolar pressure longer than the constant flow of VCV, thereby aiding recruitment and improving homogenous distribution of inspired gas. In our study, $P_{a_{O_2}}$ improved in most patients during PCV and mean $P_{a_{O_2}}$ was significantly higher than that during VCV. The improvement in arterial oxygenation during PCV may be explained by the flow profile of this mode and also the lower pulmonary shunt obtained with PCV.

To answer the question "which patients benefited more from PCV" we investigated the correlation between individual $P_{a_{O_2}}$ differences between PCV and VCV, and respiratory function tests. We found that there was a significant correlation between individual $P_{a_{O_2}}$ difference between PCV and VCV, and FVC. Patients whose $P_{a_{O_2}}$ improved with PCV generally had a poor FVC (fig. 1). Many of the studies demonstrating better arterial oxygenation with PCV compared with VCV were performed in patients with severe respiratory failure.[7–9] As may be expected, those patients who had dependent lung pathology of varying severity, especially patients with restrictive disease, benefited from the decelerating flow pattern of PCV which improved distribution of alveolar gas and arterial oxygenation.

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