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


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 (Paw=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 Paw was 4–35% (mean 16.1 (so 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

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 (FEV₁)) 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.

Accepted for publication: April 25, 1997.


MEHMET TUĞRUL*, MD, EMRE ÇAMCI, MD, HASAN KARADENİZ, MD, MİRT ŞENTÜRK, MD, KAMIL PEMBEÇ, MD, KUTAY AKİR, MD, Department of Anaesthesiology and Intensive Care, University of Istanbul, Istanbul Medical Faculty, Türkiye.
and intra-arterial cannulae were inserted under local anaesthesia and an ECG was attached. After pre-oxygenation, anaesthesia was induced with fentanyl 2 μg 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 fibroptic bronchoscope and separation of the lungs was demonstrated by capnography. 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 μg 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 ±20% of pre-induction values. Oesophageal temperature was monitored throughout the procedure and maintained greater than 36.5 °C by a heating blanket (Bıçakcılar, 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 (PaCO₂ (arterial)) 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 PaCO₂ 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 (FiO₂) 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, 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 (PaO₂, SaO₂, SvO₂, PaO₂, 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 (Qs/Qt) was calculated from standard formulae.

Peak airway pressure (Paw) and mean airway pressure (Pmean) 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 (Pplat) was measured during the last 0.3 s of each hold manoeuvre.

We studied PaO₂ data in order to identify which patients would benefit from OLV-PCV. The relationship between “individual PaO₂ 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 (SaO₂ <90%) requiring reinflation of the collapsed lung did not occur in any patient.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Patient data. FVC = Forced vital capacity, FEV₁ = forced expiratory volume in 1 s, PaO₂ = arterial oxygen tension, PaCO₂ = arterial carbon dioxide tension (mean (sd) [range] or number) (n=48)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>56.4 [30–78]</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>70.3 (11.5) [46–95]</td>
</tr>
<tr>
<td>Sex (M/F)</td>
<td>38/10</td>
</tr>
<tr>
<td>Right-left-sided thoracotomy</td>
<td>28/20</td>
</tr>
<tr>
<td>Lobectomy/pneumonectomy</td>
<td>29/19</td>
</tr>
<tr>
<td>Preoperative FVC (% predicted)</td>
<td>77.1 (15) [46–117]</td>
</tr>
<tr>
<td>Preoperative FEV₁ (% predicted)</td>
<td>76.8 (14) [43–112]</td>
</tr>
<tr>
<td>Preoperative PaO₂ (kPa)</td>
<td>5 (0.63) [3.95–6.7]</td>
</tr>
<tr>
<td>Preoperative PaCO₂ (kPa)</td>
<td>10.2 (1.12) [7.9–12.8]</td>
</tr>
</tbody>
</table>
Table 2 Ventilation and gas exchange data (mean (SD)).

<table>
<thead>
<tr>
<th>Ventilation mode</th>
<th>Tidal volume (ml)</th>
<th>Paw (cm H2O)</th>
<th>Pplat (cm H2O)</th>
<th>Pmaw (cm H2O)</th>
<th>PAOP (mm Hg)</th>
<th>MAP (mm Hg)</th>
<th>HR (beat min⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TTV-VCV</td>
<td>708 (112)</td>
<td>21.4 (4.7)</td>
<td>15.7 (3.8)</td>
<td>6.7 (1.4)</td>
<td>27.5 (7.9)</td>
<td>99.1 (0.98)</td>
<td>167.2 (6.2)</td>
</tr>
<tr>
<td>OLV-VCV</td>
<td>714 (111)</td>
<td>28.3 (5.1)</td>
<td>18.5 (4.2)</td>
<td>7.8 (2)</td>
<td>28.4 (15.6)</td>
<td>98.3 (3.1)</td>
<td>162.0 (6.2)</td>
</tr>
<tr>
<td>OLV-PCV</td>
<td>710 (114)</td>
<td>23.65 (3.85)</td>
<td>17.8 (3.7)</td>
<td>7.95 (1.55)</td>
<td>32.3 (14.4)</td>
<td>98.7 (2.1)</td>
<td>161.0 (6.2)</td>
</tr>
</tbody>
</table>

PaO₂ and Qs/Qt. Paw was significantly lower during OLV-PCV than during OLV-VCV (23.65 (3.85) cm H₂O vs 28.3 (5.1) cm H₂O) (P = 0.000001). Compared with OLV-VCV, Paw decreased consistently during OLV-PCV in every patient, ranging from 1 to 13.5 cm H₂O. The percentage Paw difference was 4–35% (mean 16.1 (8.4) %). Pplat was also significantly lower during OLV-PCV than during OLV-VCV (17.8 (3.7) vs 18.5 (4.2) cm H₂O) (P = 0.01).

Mean PaO₂ 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 PaO₂ value with OLV-PCV had an FVC lower than 77%, whereas only six of 17 (35%) patients who had an improved PaO₂ with OLV-VCV had an FVC lower than 77% (P = 0.02). Also, 19 of 31 patients (61%) whose PaO₂ improved with OLV-PCV had a FEV₁ lower than 77%, whereas five of 17 (30%) patients whose PaO₂ improved with OLV-VCV had an FEV₁ lower than 77%. On the basis of linear regression analysis “individual PaO₂ 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 FEV₁ (r = −0.24, P = 0.09).

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

**Discussion**

VVC 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, 1,5,10 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 PaO₂ tends to increase over 1 h after the onset of one-lung anaesthesia, 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 H₂O 9 and from 43 to 40 cm H₂O 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.45 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 

Hypoxic pulmonary vasoconstriction is an autoregulatory 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 of hypoxic lung is intermediate (30–70%), that is the amount of lung that is hypoxic during one-lung anaesthesia.30

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.41617 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, 

References


