Pressure-controlled ventilation and intrabronchial pressure during one-lung ventilation†


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RESPIRATION AND THE AIRWAY

Key points
- PCV may reduce peak airway pressure when compared with VCV during one-lung ventilation.
- This study compared airway pressure in the breathing circuit with that in the dependent lung bronchus during VCV followed by PCV.
- PCV reduced both circuit pressure and bronchial pressure. The effect on circuit pressure was much larger in the circuit.
- Small reduction in bronchial airway pressure during PCV is probably not clinically significant.

Background. Pressure-controlled ventilation (PCV) has been suggested to reduce peak airway pressure (Ppeak) and intrapulmonary shunt, thereby limiting the risk of barotrauma and improving oxygenation, respectively.1-3 Compression of small intra-alveolar vessels during inflation increases the resistance to pulmonary blood flow in the dependent lung and can divert blood to the non-dependent lung.4 However, this phenomenon is more closely related to the mean airway pressure and more specifically to the mean alveolar pressure.5-6 The potential benefits of PCV over volume-controlled ventilation (VCV) during OLV remain controversial, and recent studies have failed to demonstrate an improvement in oxygenation when PCV is used.7-8 These divergent results can be explained by the fact that PCV principally induces a decrease in Ppeak with a much smaller effect on mean alveolar pressure.8-10 Moreover, pressure measurements are usually recorded within the respirator breathing circuit, proximal to the double-lumen tube (DLT), and it is widely recognized that a substantial part of the resistive pressure originates in the tracheal tube.11 Consequently, at the same tidal volume (Vt), the decrease in airway pressure that is observed in the breathing circuit and displayed on the ventilator monitor is not necessarily associated with a decrease in the bronchus of the dependent lung.

Methods. This observational study included 15 consecutive subjects who were ventilated with VCV followed by PCV at constant Vt. Airway pressure was measured simultaneously in the breathing circuit and main bronchus of the dependent lung after 20 min of ventilation.

Results. PCV induced a significant decrease in Ppeak (mean (SD)) measured in the breathing circuit [36 (4) to 26 (3) cm H2O, P<0.0001] and in the bronchus [23 (4) to 22 (3) cm H2O, P=0.01]. However, the interaction (ventilatory mode × site of measurement) revealed that the decrease in Ppeak was significantly higher in the circuit (P<0.0001). Although the mean percentage decrease in Ppeak was significant at both sites, the decrease was significantly lower in the bronchus [5 (6)% vs 29 (3)%].

Conclusions. During PCV for OLV, the decrease in Ppeak is observed mainly in the respiratory circuit and is probably not clinically relevant in the bronchus of the dependent lung. This challenges the common clinical perception that PCV offers an advantage over VCV during OLV by reducing bronchial Ppeak.

Keywords: monitoring, ventilation; respiratory, mechanics; surgery, thoracic; ventilation, one-lung

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References
Methods

Study population

This prospective observational study was carried out in the Department of Anaesthesia and Critical Care of Bordeaux University Hospital in September and October 2008. After approval by our institutional review board (Comité de Protection des Personnes Sud-Ouest et Outre Mer III), 15 consecutive subjects undergoing elective open-chest thoracotomy for pulmonary resection were prospectively enrolled. Informed consent was obtained from all subjects. Exclusion criteria included forced expiratory volume < 70% predicted for age and height in 1 s. This study was designed as an intra-individual comparison of VCV and PCV, each subject being his/her own control and undergoing VCV before PCV.

Perioperative management

Premedication consisted of midazolam 5 mg given orally. Preoperative medications were continued until the morning of surgery with the exception of angiotensin-converting enzyme-inhibitors. Routine monitoring included electrocardiography, pulse oximetry, capnography, and non-invasive arterial pressure. Bispectral index (BIS) was monitored continuously using a BIS monitor (Aspect Medical Systems, Newton, MA, USA). After pre-oxygenation, anaesthesia was induced with propofol 1–3 mg kg\(^{-1}\), sufentanil 0.3 \(\mu\)g kg\(^{-1}\) and cisatracurium 0.15 mg kg\(^{-1}\). Anaesthesia was maintained with sevoflurane and repeated boluses of sufentanil. End-tidal concentrations of sevoflurane were titrated to maintain BIS values between 45 and 55 throughout the intraoperative period. Additional boluses of cisatracurium were given intraoperatively as necessary. The trachea and bronchi were intubated with a left-sided DLT (Broncho-part; Rusch, Kermen, Germany: 39 F for male and 37 F for female subjects). Fibreoptic bronchoscopy was systematically used to confirm correct positioning of the DLT immediately after blind insertion; subjects were then placed in the lateral position. Haemodynamic instability was treated with fluids, ephedrine, or both. The lungs were ventilated with an Engström Carestation Ventilator (GE Healthcare, Helsinki, Finland) in square-wave flow VCV mode and an inspired oxygen fraction (\(F_{\text{IO}}\)) of 1.0. \(V_T\) was set at 8 ml kg\(^{-1}\) of ideal body weight, with maximal plateau pressure (\(P_{\text{plat}}\)) limited to 32 cm H\(_2\)O. The inspiratory pressure was adjusted to maintain end-tidal carbon dioxide tension (\(c_{\text{etCO}}\)) between 4.0 and 4.6 kPa. Positive end-expiratory pressure (PEEP) was arbitrarily set at 5 cm H\(_2\)O. OLV was initiated with VCV immediately after skin incision without modifying the respiratory parameters. This method of ventilation was maintained for 20 min and the ventilator was then switched to PCV mode. The inspiratory pressure was then adjusted to maintain a similar \(V_T\) (i.e. 8 ml kg\(^{-1}\)). The ventilatory frequency, \(F_{\text{in}}\), and \(I/E\) ratio also remained unchanged. After a 20 min period of PCV, the ventilatory mode of the patient was left at the discretion of the attending anaesthesiologist. During OLV, the lumen of the non-ventilated lung was opened to room air. Haemodynamic data (non-invasive arterial pressure, heart rate) and ventilatory data (\(V_T\), \(P_{\text{peak}}\), \(P_{\text{plat}}\) and mean airway pressure, respiratory frequency and compliance) were recorded at the end of each 20 min period of ventilation. A 3 s inspiratory pause was maintained in order to obtain a stable \(P_{\text{plat}}\). Intrinsic PEEP (iPEEP) was measured during end-expiratory pause. All measurements were made during radical lymphadenectomy and thus before any of the pulmonary vessels of the non-dependent lung were ligated. If \(SpO_2\) decreased below 90%, surgery was temporarily stopped to resume two-lung ventilation.

Airway pressure was measured simultaneously in the breathing circuit and at the end of the DLT within the main bronchus of the lung being ventilated as illustrated in Figure 1. Bronchial airway pressure was measured using a low-compliant pressure catheter (external diameter 2 mm, internal diameter 0.9 mm), which was positioned through a swivel connector and connected to a pressure transducer (Engstrom\(^{10}\); General Electric, USA).\(^{12}\) The position of the pressure line was controlled by fibreoptic bronchoscopy.

Data analysis

Data are expressed as mean (SD). Comparison of means was carried out using either unpaired or paired two-sample \(t\)-tests as required. Comparison of the decrease in respiratory parameters between the two sites was performed using two-way analysis of variance (ANOVA). A \(P\)-value < 0.05 was used to rule out the null hypothesis. Statistical analysis was performed with NCSS (Statistical Solutions Ltd).

Results

Fifteen consecutive subjects (13 males and two females) were enrolled. The patient characteristics and surgical procedures are summarized in Table 1. No patient exhibited \(SpO_2\) < 90% requiring interruption of the surgical procedure and two-lung ventilation. \(V_T\) was comparable in both modes of ventilation (Table 2). Mean airway pressure and \(P_{\text{plat}}\) were also unaffected by the change in ventilation mode.

The switch from VCV to PCV induced a significant decrease in \(P_{\text{peak}}\) measured in the breathing circuit and in the main bronchus of the dependent lung (Table 2 and Figure 2). The interaction analysis (ventilatory mode \(\times\) site of measurement) revealed that the decrease in \(P_{\text{peak}}\) was significantly greater when measured in the breathing circuit (Fig. 2).

Proximal to the DLT (at the circuit Y-piece), the mean reduction in \(P_{\text{peak}}\) was 10.7 (1.3) cm H\(_2\)O. Inside the main bronchus, distal to the DLT, the mean reduction in \(P_{\text{peak}}\) was only 1.2 (1.5) cm H\(_2\)O, corresponding to a 5 (6)% reduction (\(P<0.05\)) (Fig. 3). The remaining respiratory
mechanics variables such as thoracopulmonary compliance, iPEEP, or ventilatory frequency were comparable in both ventilatory modes (Table 2). Similarly, no significant differences in alveolar ventilation and hemodynamics were observed (Table 2). As shown in Figure 4, the waveforms of the pressure–time curves recorded in the main bronchus of the dependent lung were similar in both ventilatory modes.

**Discussion**

The results of this observational study show that a switch from VCV to PCV markedly decreased $P_{\text{peak}}$ in the breathing circuit with only a small decrease in the main ventilated bronchus.

$P_{\text{peak}}$ depends on inspiratory flow but also on the tracheal tube diameter, which is the major source of resistance during mechanical ventilation. As a DLT has a smaller internal diameter, the resistance during OLV is even higher. Our aim was to demonstrate that the decrease in $P_{\text{peak}}$ observed in the breathing circuit is different from that found inside the main bronchus of the dependent lung, therefore unmasking the artifactual nature of $P_{\text{peak}}$ measurements in the circuit. For an identical $V_t$, the mean reduction in $P_{\text{peak}}$ was only 1.2 (1.5) cm H$_2$O, which was far less than that following ventilator monitoring [mean decrease in $P_{\text{peak}}$ 10.7 (1.3) cmH$_2$O].

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**Table 2** Respiratory and hemodynamic variables at the end of each ventilation period during OLV: VCV vs PCV ($n=15$). Data are expressed as mean (SD). $V_t$, tidal volume; $P_{\text{peak}}$, peak airway pressure; $P_{\text{plat}}$, plateau airway pressure; $P_{\text{mean}}$, mean airway pressure; iPEEP, intrinsic PEEP; $C_{\text{dyn TP}}$, thoraco-pulmonary compliance; $E_{\text{CO}_2}$, end-tidal CO$_2$; SAP, systolic arterial pressure; HR, heart rate

<table>
<thead>
<tr>
<th>Parameter</th>
<th>VCV</th>
<th>PCV</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$V_t$ expired (ml)</td>
<td>539 (71)</td>
<td>541 (75)</td>
<td>0.86</td>
</tr>
<tr>
<td>$P_{\text{peak}}$ circuit (cm H$_2$O)</td>
<td>36 (4)</td>
<td>26 (3)</td>
<td>$&lt;0.0001$</td>
</tr>
<tr>
<td>$P_{\text{peak}}$ bronchus (cm H$_2$O)</td>
<td>23 (4)</td>
<td>22 (3)</td>
<td>0.01</td>
</tr>
<tr>
<td>$P_{\text{plat}}$ circuit (cm H$_2$O)</td>
<td>20 (3)</td>
<td>20 (3)</td>
<td>0.22</td>
</tr>
<tr>
<td>$P_{\text{plat}}$ bronchus (cm H$_2$O)</td>
<td>21 (3)</td>
<td>20 (3)</td>
<td>0.16</td>
</tr>
<tr>
<td>$P_{\text{mean}}$ circuit (cm H$_2$O)</td>
<td>12 (2)</td>
<td>12 (1)</td>
<td>0.004</td>
</tr>
<tr>
<td>$P_{\text{mean}}$ bronchus (cm H$_2$O)</td>
<td>11 (2)</td>
<td>12 (3)</td>
<td>0.33</td>
</tr>
<tr>
<td>iPEEP (cm H$_2$O)</td>
<td>2 (2)</td>
<td>2 (2)</td>
<td>1</td>
</tr>
<tr>
<td>$C_{\text{dyn TP}}$ (ml cm H$_2$O$^{-1}$)</td>
<td>37 (8)</td>
<td>38 (8)</td>
<td>0.09</td>
</tr>
<tr>
<td>$E_{\text{CO}_2}$ (kPa)</td>
<td>4.13 (0.4)</td>
<td>4.13 (0.4)</td>
<td>0.78</td>
</tr>
<tr>
<td>SAP (mm Hg)</td>
<td>98 (12)</td>
<td>100 (10)</td>
<td>0.09</td>
</tr>
<tr>
<td>HR (beats min$^{-1}$)</td>
<td>80 (13)</td>
<td>81 (11)</td>
<td>0.75</td>
</tr>
</tbody>
</table>

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**Table 1** Patient characteristics and operative characteristics of the patients ($n=15$). Data are expressed as mean (SD or range) or number of patients

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>56 (20–81)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>75 (15)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>173 (7)</td>
</tr>
<tr>
<td>Male/female</td>
<td>13/2</td>
</tr>
<tr>
<td>ASA physical status I/II/III/IV</td>
<td>3/7/4/1</td>
</tr>
<tr>
<td>Right/left-sided thoracotomy</td>
<td>10/5</td>
</tr>
<tr>
<td>Lobectomy/pneumonectomy</td>
<td>12/3</td>
</tr>
</tbody>
</table>
Our results are consistent with those of Unzueta and colleagues who compared PCV and VCV in 58 patients undergoing thoracotomy. These authors observed a significant reduction in $P_{\text{peak}}$ (in the breathing circuit) in the same range as that observed in our study, but without any improvement of oxygenation with PCV. Compression of small intra-alveolar vessels might increase resistance to pulmonary blood flow in the dependent lung and is a matter for concern. However, this is closely related to alveolar inflation, overdistension, or both, which are linked to mean alveolar pressure and not to $P_{\text{peak}}$. Limiting $P_{\text{plat}}$ would then be more physiologically relevant than a decrease in $P_{\text{peak}}$. This can be achieved in both PCV and VCV by decreasing the $V_T$. Our data demonstrate that PCV results in a minimal reduction in $P_{\text{peak}}$ during OLV at constant $V_T$ (Fig. 4).

These results agree with those of other authors who failed to find a decrease in plateau and mean airway pressure during PCV. It should be pointed out that in other studies, where the decrease in plateau and mean airway pressure was significant, the values were very similar in both ventilatory modes. Finally, some authors have attributed the improvement in oxygenation to respiratory settings more than to a specific ventilatory mode. This study challenges the use of PCV as a way to reduce $P_{\text{peak}}$ in the airways at constant $V_T$ and consequently its recommendation as a rational mode of ventilation in OLV, as suggested by several authors. Another feature of PCV is that minute ventilation cannot be guaranteed safely over a prolonged period of time. During thoracic surgery,
unpredictable changes in the respiratory system can be expected, such as variations in airways resistance and thoracopulmonary compliance. Therefore, repeated adjustments at the patient’s side in the operating room are required to avoid either derecruitment or overdistension. While PCV offers multiple advantages and useful properties in anaesthesia,10 17 18 it is concerning that its recommended use in OLV could be based on a pathophysiological misconception. Guidelines therefore appear to recommend the use of a more challenging ventilatory mode during OLV without the expected beneficial effects.4

The following points should be considered when assessing the clinical relevance of our results. First, this was a prospective non-blinded non-randomized study. However, each subject served as his/her own control to avoid confounding factors, and adequate power was obtained with a limited number of subjects. Because it was not a real cross-over design, an order effect or a carry-over effect cannot be excluded, but previous studies using a randomized order to compare PCV and VCV did not report these problems.2 4 Second, the impact of ventilatory mode on inotropic oxygenation was not evaluated, and only mechanical respiratory parameters were studied. Third, the pressure transducer line used might have increased the resistive pressure between the Y-piece and bronchus by 10%, although this resistance did not change during the comparison of VCV and PCV.12

At constant V_T, this study demonstrates that the reduction in P_{peak} induced by PCV during OLV is probably not clinically relevant when measured in the main ventilated bronchus. The reduction in P_{peak} with PCV compared to VCV is essentially linked to the circuit resistance as the pressure dissipates almost entirely at the concentration of the DLT. Our results challenge the common clinical perception that PCV offers an advantage over VCV during OLV by reducing bronchial P_{peak}.

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Conflict of interest
None declared.

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