Haemodynamic and respiratory outcomes for pressure controlled ventilation and volume-controlled ventilation in patients submitted to laparoscopic surgery


ABSTRACT

Background: Mechanical ventilation strategies are used to prevent lung damage, optimizing gas exchange. Recently, has been described that mechanical support limited by volume and pressure reduces lung overdistention. The aim of our study was to compare pressure control and volume control modalities in patients underwent to different laparoscopic approaches. Methods: With local ethics committee approval and written informed consent, 40 patients (Class I/II ASA) undergoing elective laparoscopic surgery were included in the study. The patients were fasted from midnight before the day of surgery. Anesthesia was induce with phentanyl (2 μg/kg), propofol (2 mg/kg) and atracurium (150 μg/kg). Endotracheal intubation was performed after complete relaxation evaluated with train of four (TOF). Anesthesia was mantained with sevoflurane (1 MAC). Patients were randomized to receive PCV or VCV. Results: Haemodynamic parameters were similar in both groups during the different periods of time recorded. Systolic, diastolic and mean pressure were similar. During pneumoperitoneum SpO₂ increased up to 97.61 ± 1.29 for PCV compared to 97.25 ± 1.2 for VCV (p 0.368). Ten minutes after insufilation SpO₂ remained similar for both groups (p 0.368). Conclusion: In summary we conclude that PCV and VCV are both well tolerated ventilation modalities for patients submitted to laparoscopic surgery.

Key words: Pressure controlled ventilation, volume controlled ventilation, laparoscopic surgery, haemodynamic an respiratory outcomes.

INTRODUCTION

Pressure positive mechanical ventilation was developed in the mild 20’s as a part of the support in anesthesia during thoracic surgery.1 Recently, mechanical ventilation has been described to
have many clinical utilities due to reduced breath work and the fact that it improves acidosis status and hypoxemia. Mechanical ventilation is a useful tool, but it has morbidity and mortality related to its employment. This is the reason for trying to find new strategies improving gas exchange and to reduce these complications.2

Morbidity and mortality associated to mechanical ventilation represents the effect on volume and pressure outcomes. We know that positive pressure causes acute lung injury.3-6 It seems to be obvious that lung inflation causes injury if airway pressure is high enough. Webb and Tierney, ventilated mice during 1 hour at different levels of pressure observing moderate perivascular edema in those which received peak inspiratory pressure (PIP) at 30 cm/H2O while those receiving 45 cm/H2O developed severe hypoxia after the first hour.7 Another cause of lung damage is lung overdistention; this peculiar condition causes an increased on vessels permeability and dysfunction on the surfactant, leading to lung bleeding and hyaline membrane development. The use of low tidal volume and inspiratory peak pressure decreases lung damage caused by overdistention.8 On the other side, ventilation with volume and pressure at lower levels next to alveolar volume has been described as a cause of lung damage related to mechanical forces and subsequently affecting surfactant function associated to changes on the alveolar surface.8,9 Since the early 90’s the use of mechanical ventilation has changed. Recently, the use of low tidal volumes reduced mortality associated to respiratory dystress.10 Mechanical ventilation strategies are used to prevent lung damage, optimizing gas exchange. Recently, has been described that mechanical support limited by volume and pressure reduce lung overdistention.11-15

Haemodynamic changes associated to mechanical ventilation are caused by increasing on the mean intrathoracic pressure and reduction on the venous return and preload volume. Vascular resistance, also is increased and positive ventilation could cause direct compression on the heart.16 There are four haemodynamic conditions associated, those are heart rate, pre-load volume, post-load volume and contractility. These conditions could be modified as result of lung volume and intrathoracic pressure. There are several mechanisms including tone autonomic changes, lung vascular resistance, direct mechanical compression and increased in the abdominal pressure.16 Considering the need to maintain a healthy patient under mechanical ventilation during a very short time as occurs in elective surgical procedures (laparoscopic surgery) it is important to prevent lung damage. Mechanical ventilation during an anesthetic procedure could be performed in many forms.

Pressure controlled ventilation (PCV) and Volume controlled ventilation (VCV) are both an alternative modes of ventilation which are used widely in severe respiratory failure. Those ways of ventilations could be used in patients submitted to anesthesia procedures.

Laparoscopic surgery usually requires a pneumoperitoneum by insufflating the abdominal cavity with carbon dioxide (CO2).

Pneumoperitoneum for laparoscopic surgery has been shown to induce increased systemic arterial pressure and filling volumes. The interpretation of increased central venous and pulmonary capillary wedge pressures associated with pneumoperitoneum remains controversial.16-20 They may in fact, reflect increased cardiac filling but may also be the consequence of elevated intrathoracic pressure due to increased intraabdominal pressure, and hence even result in reduced cardiac filling. The intravascular volume state, positioning and the amount of intraabdominal pressure appear to be important factors with respect to venous return and the interpretation of increased filling pressures.6

The aim of our study was to compare pressure control and volume control modalities in patients underwent to different laparoscopic approaches.

PATIENTS AND METHODS

With local ethics committee approval and written informed consent, 40 patients (Class I/II ASA) undergoing elective laparoscopic surgery were in-
cluded in the study. None of the patients had history or signs of cardiopulmonary disease. The present study is a prospective, randomized, longitudinal and descriptive one.

**Anesthesia.** The patients were fasted from midnight before the day of surgery. Anesthesia was induced with fentanyl (2 µg/kg), propofol (2 mg/kg) and atracurium (150 µg/kg). Endotracheal intubation was performed after complete relaxation evaluated with train of four (TOF). Anesthesia was maintained with sevoflurane (1 MAC). Patients were randomized to receive PCV or VCV. In the VCV group tidal volume was adjusted to maintain 8 mL/kg (ideal body weight) without PEEP (positive end expiratory pressure). At the beginning PCV was given at 10 cm H2O of peak pressure and during the third ventilation it was adjusted to maintain 8 mL/kg (ideal body weight) after that the pressure was modified only if the volume increases or decreases. Train of four monitoring was used to assess the depth of paralysis. If the train of four increased 30% it was administered only 20% of the initial dose from atracurium in both groups. During the study FiO2 was maintained at 60% (3 lts/minute). Respiratory rate was adjusted to obtain an end tidal CO2 of 30 mm Hg + 2. The lungs were ventilated using a Datex Ohmeda Aestiva 5000 ventilator.

Once steady 5 minutes after intubation state was achieved, respiratory parameters (plateau pressure, end tidal CO2 inspiratory and expiratory, inspiratory and expiratory tidal volume, minute volume, lung compliance, respiratory frequency and main airway pressure) 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 ventilator. Inspiratory plateau pressure (Pplt) was measured during the last 0.3 s of each hold maneuver. The haemodynamic outcomes such as heart rate, mean arterial pressure, systolic pressure, SPO2 and diastolic pressure were measured.

**Statistical analysis.** Data were analyzed by a biostatistic using SPSS 13.0 for windows software and are presented as mean ± standard deviation. The significance of differences was tested by a two way analysis of variance for repeated measurements. A p value < 0.05 was considered statistically significant.

**RESULTS**

Patients data are shown in table I. We evaluated 40 consecutively patients randomized to receive PCV or VCV (20 and 20 respectively). Age, gender, weight, height, BMI and ASA were similar in both groups. We found 4 non heavy smokers in each group. Surgical procedure were similar for both groups (Table II). Surgical time was of 1.8 ± 0.80 and 1.7 ± 0.74 for PCV and VCV respectively. All patients completed the study protocol which allowed a comparative analysis of the two modes of ventilation.

**Table I. Clinical characteristics.**

<table>
<thead>
<tr>
<th></th>
<th>PCV</th>
<th>VCV</th>
<th>p</th>
</tr>
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<tbody>
<tr>
<td>Age</td>
<td>43 ± 10</td>
<td>42 ± 9</td>
<td>0.341</td>
</tr>
<tr>
<td>Sex</td>
<td>F 10 M 10</td>
<td>F 10 M 10</td>
<td>0.398</td>
</tr>
<tr>
<td>Weight</td>
<td>75 ± 16</td>
<td>75 ± 17</td>
<td>0.665</td>
</tr>
<tr>
<td>Height</td>
<td>1.67 ± 0.86</td>
<td>1.69 ± 0.97</td>
<td>0.796</td>
</tr>
<tr>
<td>BMI</td>
<td>26 ± 3</td>
<td>25 ± 4</td>
<td>0.565</td>
</tr>
<tr>
<td>ASA</td>
<td>I-II</td>
<td>I-II</td>
<td>0.716</td>
</tr>
<tr>
<td>Smokers</td>
<td>4</td>
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</table>

PCV = Pressure controlled ventilation. VCV = Volume controlled ventilation. F = Female. M = Male. BMI = Body mass index.

* Values are expressed as median and standard deviation
Respiratory outcomes. Baseline peak pressure was of 14.15 ± 2.79 for PCV and 16.40 ± 4.62 for VCV group (p 0.43). During pneumoperitoneum, peak pressure increased up to 15.90% (PCV) compared to 18.56% in the VCV arm (p 0.004). Peak airway pressure after ten minutes from pneumoperitoneum was 15.5 ± 3.23 for PCV and 17.90 ± 4.65 for VCV (p 0.59).

Baseline plateau pressure was of 13.85 ± 2.68 and 15.80 ± 4.47 for PCV and VCV respectively. During pneumoperitoneum Plateau pressure was increased up to 18.09 ± 3.27 and 21.19 ± 4.85 for PCV and VCV (p 0.017) and after pneumoperitoneum PCV exhibited a Plateau pressure of 14.90 ± 3 compared with 17.4 ± 4.85 in VCV.

Lung compliance was similar before insufflation between PCV and VCV. During insufflation, it was higher in PCV patients (36.73 ± 8.49 compared to 32.34 ± 7.8, p 0.052).

Baseline mean airway pressure was higher in the PCV group (6.45 ± 1.14 versus 5.86 ± 1.42). During pneumoperitoneum and after itself mean airway pressure was of 7.79 ± 1.36 versus 7.10 ± 1.47 and 6.65 ± 1.22 versus 5.95 ± 1.43 for PCV and VCV respectively (Table III).

Baseline expiratory volume was of 529 ± 79 be-

<table>
<thead>
<tr>
<th>Surgical procedures and PCV/VVC.</th>
<th>PCV</th>
<th>VCV</th>
</tr>
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<tbody>
<tr>
<td>Cholecystectomy</td>
<td>12</td>
<td>9</td>
</tr>
<tr>
<td>Funduplication</td>
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<td>8</td>
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<tr>
<td>Inguinal Hernioplasty</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Surgical time*</td>
<td>1.80 ± 0.80</td>
<td>1.70 ± 0.74</td>
</tr>
<tr>
<td>Sevofluorane</td>
<td>13</td>
<td>11</td>
</tr>
<tr>
<td>Desfluorane</td>
<td>7</td>
<td>9</td>
</tr>
<tr>
<td>Atracurium</td>
<td>16</td>
<td>14</td>
</tr>
</tbody>
</table>

* Values are expressed as median and standard deviation.

<table>
<thead>
<tr>
<th>Respiratory outcomes during PCV and VCV.</th>
<th>PCV*</th>
<th>VCV*</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>PIP baseline</td>
<td>14.15 ± 2.79</td>
<td>16.40 ± 4.62</td>
<td>.043</td>
</tr>
<tr>
<td>PIP pneum</td>
<td>18.58 ± 3.32</td>
<td>22.03 ± 5.03</td>
<td>.004</td>
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<tr>
<td>PIP post-pneumo</td>
<td>15.55 ± 3.23</td>
<td>17.90 ± 4.65</td>
<td>.059</td>
</tr>
<tr>
<td>PP baseline</td>
<td>13.85 ± 2.68</td>
<td>15.80 ± 4.47</td>
<td>.093</td>
</tr>
<tr>
<td>PP pneum</td>
<td>18.09 ± 3.27</td>
<td>21.19 ± 4.85</td>
<td>.017</td>
</tr>
<tr>
<td>PP post-pneumo</td>
<td>14.90 ± 3.00</td>
<td>17.40 ± 4.85</td>
<td>.063</td>
</tr>
<tr>
<td>Compl baseline</td>
<td>49.60 ± 11.49</td>
<td>45.80 ± 14.46</td>
<td>.356</td>
</tr>
<tr>
<td>Compl pneum</td>
<td>36.73 ± 8.49</td>
<td>32.34 ± 7.80</td>
<td>.052</td>
</tr>
<tr>
<td>Compl post-pneum</td>
<td>44.65 ± 12.20</td>
<td>40.30 ± 10.29</td>
<td>.208</td>
</tr>
<tr>
<td>Paw baseline</td>
<td>6.45 ± 1.14</td>
<td>5.85 ± 1.42</td>
<td>.117</td>
</tr>
<tr>
<td>Paw pneum</td>
<td>7.79 ± 1.36</td>
<td>7.10 ± 1.47</td>
<td>.058</td>
</tr>
<tr>
<td>Paw post-pneum</td>
<td>6.65 ± 1.22</td>
<td>5.95 ± 1.43</td>
<td>.069</td>
</tr>
</tbody>
</table>

PCV = Pressure controlled ventilation. VCV = Volume controlled ventilation. PIP = Peak inspiratory pressure. PP = Plateau pressure. Compl = Lung compliance. Paw = Main airway pressure. pneum = Mean time during pneumoperitoneum.

* Values are expressed as median and standard deviation.
fore pneumoperitoneum, 545 + 81 during pneumoperitoneum and 541 +/- 91 after pneumoperitoneum for PCV versus 548 +/-107, 552 + 93.4 and 569 + 103 for VCV at the same stages (Table IV). Minute volume in PCV and VCV is seen in Table IV.

Haemodynamic outcomes. Haemodynamic parameters were similar in both groups during the different periods of time recorded. Systolic, diastolic and mean pressure were similar. Baseline \( \text{SpO}_2 \) was similar for both groups (p 0.937) (Table V). During pneumoperitoneum \( \text{SpO}_2 \) increased up to 97.61 + 1.29 for PCV compared to 97.25 + 1.2 for VCV (p 0.368). Ten minutes after insufflation \( \text{SpO}_2 \) remained similar for both groups (p 0.368) (Table V).

DISCUSSION

Ventilation modes are often described as volume-controlled or pressure controlled depending on whether tidal volume or maximum airway pressure is the specified target for the end of active inspiration. In current practice, pressure controlled modes build pressure rapidly and attempt to maintain pressure constant through the remainder of the high pressure phase. Decelerating inspiratory flow characterizes such rectilinear pressure waveforms, characteristic that may improve the distribution of ventilation and limit the maximal inspiratory and regional pressure among lung units with heterogeneous ventilatory time constants.\(^{21,22}\)
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. Uniform distribution of inspired gas with PCV is the major cause of better arterial oxygenation in patients with respiratory failure. Volume controlled ventilation (VCV) in the other side is the traditional method of performing procedures such as one lung anesthesia. 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.

Our study was designed to evaluate the outcomes in terms of respiratory parameters in patients undergoing laparoscopic surgery. Laparoscopic surgery is usually performed by the intra-abdominal insufflation of carbon dioxide, i.e. pneumoperitoneum. Insufflation of carbon dioxide intra-abdominally causes a rise in arterial carbon dioxide tension (PaCO₂) and possibly acidosis, by diffusion through peritoneum. In order to maintain normocapnia, the respiratory rate often needs to be increased intraoperatively. In cases of severe hypercapnia or acidosis, conversion of the laparoscopic approach into an open procedure has been reported. It has already been demonstrated that anesthesia per se results in a reduced functional residual capacity and shifting of the diaphragm cranially. Furthermore, atelectasis development occurring in dependent regions of the lungs during anesthesia impairs the ventilation-perfusion match. This has been evaluated by the multiple inert gas technique as increased shunting of lung blood flow. Based on indirect methods it has been claimed that pneumoperitoneum causes an increase in dead space in pigs and in healthy patients. At our study we compared 2 modalities of ventilation patients undergoing to laparoscopic surgery. VCV offers the possibility to reduce airway peak pressure at tidal volume of 8 mL/kg maintaining an adequate gas exchange and avoiding alveolar overdystention. Campbell et al, previously reported that PCV is not better than VCV. We found that during pneumoperitoneum plateau pressure increased much less in the PCV mode (p < 0.017). Lung compliance is another important outcome in anesthesia ventilation. Previous reports described a compliance reduction during pneumoperitoneum. We reported an slighty better outcome in Lung compliance for those patients underwent to PCV (49 cm H₂O vs 45).

One problem related to PCV is the possibility to reduce tidal volume and subsequently minute volume, impairing gas exchange. At our study we maintained a tidal volume upper 8 mL/kg requiring an slightly lower respiratory rate in the PCV group. In summary we conclude that PCV and VCV are both well tolerated ventilation modalities for patients submitted to laparoscopic surgery.

REFERENCES


