Augmented spontaneous breathing and pulmonary gas exchange during pneumoperitoneum

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Summary

Background and objective: Ventilation of the lungs with positive end-expiratory pressure during pneumoperitoneum has been shown to improve the arterial partial pressure of oxygen. The implications of spontaneous breathing on pulmonary gas exchange remain unknown in this setting. We therefore sought to examine the influence of pressure-support ventilation with spontaneous breathing on gas exchange during simulated laparoscopy.

Methods: Ten pigs were subjected to pneumoperitoneum at a pressure of 15 cmH2O. Animals received, in a random order, pressure-support and pressure-controlled ventilation for 60 min per mode. Inert gas and haemodynamic measurements were performed before changing to a subsequent mode.

Results: Pressure-support ventilation was more efficient than pressure-controlled ventilation regarding perfusion of normal VA/Q lung areas (78 ± 4% vs. 72 ± 5%) (P < 0.05), alveolar–arterial partial pressure of oxygen difference (9.73 ± 1.3 vs. 11.2 ± 1.2 kPa) and arterial partial pressure of oxygen (14.93 ± 1.6 vs. 13.7 ± 2.0 kPa) (P < 0.05).

Conclusions: Pressure-support ventilation resulted in significantly better gas exchange than pressure-controlled ventilation in this model of simulated laparoscopy.

Keywords: RESPIRATION, respiratory transport, respiratory gas exchange, ventilation–perfusion ratio; SURGICAL PROCEDURES, minimally invasive, endoscopy, laparoscopy.

Introduction

Peritoneal insufflation (pneumoperitoneum) during laparoscopic procedures leads to increased intrapulmonary shunt and thus to a decreased arterial partial pressure of oxygen (PaO2). Positive end-expiratory pressure (PEEP), on the other hand, has been shown to improve pulmonary gas exchange during pneumoperitoneum [1]. What else can be done to diminish the influence of abdominal distension on pulmonary gas exchange? In an animal model with lung injury, spontaneous efforts during mechanical ventilation resulted in improved ventilation–perfusion (VA/Q) matching [2]. The beneficial effect of spontaneous breathing during mechanical ventilation is apparently due to diaphragmatic contractions, generating a redistribution of ventilation to basal, less aerated lung units [3]. As pneumoperitoneum equally results in VA/Q defects including right-to-left shunt and reduced blood flow to lung areas with a normal VA/Q ratio [1], spontaneous breathing may improve VA/Q matching during laparoscopy. However, spontaneous breathing on a certain level of continuous positive airway pressure (CPAP) alone is not feasible during pneumoperitoneum as thoracic excursions and the descent of the diaphragm are limited by the abdominal distension [4]. However, a newer mode of
augmented spontaneous breathing known as pressure-support ventilation (PSV) may be considered during pneumoperitoneum. Pressure-support ventilation was originally designed to decrease the work of breathing and to prevent diaphragmatic fatigue during weaning from mechanical ventilation in the intensive care unit. Pressure-support ventilation is not commonly used in the operating room. The aim of this study was to examine the V_A/Q distribution during PSV and pressure-controlled ventilation (PCV) in the setting of simulated laparoscopy. The hypothesis was that PSV leads to better V_A/Q matching compared with PCV in a porcine model with an air pneumoperitoneum at 15 cmH_2O inflation pressure.

Methods

Animal preparation and general set-up

The Animal Care Committee of the Austrian Ministry of Science approved this investigation. Ten 12–16-week-old domestic pigs of either gender weighing 35–38 kg were studied. Anaesthesia was introduced with ketamine (15 mg kg^{-1} intramuscularly (i.m.)) and maintained by a continuous infusion of propofol (6 mg kg^{-1} h^{-1}) and bolus injections of piritramide (pirinitramide) as needed. A 3% gelatine solution (4 mL kg^{-1} h^{-1}) was administered continuously throughout the study. All pigs were placed in the supine position. The trachea was intubated with an endotracheal tube (i.d., 6.5–7.5 mm). The lungs were then ventilated using an Evita-4® ICU ventilator (Dräger, Lübeck, Germany) according to the study protocol as described below. During the preparatory phase, the minute volume of ventilation was adjusted to maintain a PaCO_2 5.5 kPa. Body temperature was maintained between 38.0 and 39.0°C using an electric heating blanket. Venous catheters were inserted percutaneously into auricular veins for infusion of inert gas and continuous infusion of propofol. A 7 F thermistor-tipped flow-directed pulmonary artery catheter was inserted directly into the right internal jugular vein and advanced into a main pulmonary artery using direct-pressure monitoring. This permitted measurements of cardiac output, pulmonary arterial pressure, pulmonary capillary wedge pressure, mixed venous blood sampling and collection of inert gas blood samples. The left femoral artery was cannulated for measurement of systemic arterial pressure, arterial blood gas sampling and collection of inert gas blood samples. Mean arterial, central venous, pulmonary arterial and pulmonary capillary wedge pressures were measured by use of an intensive care monitor (Servomed®; Hellige GmbH, Freiburg, Germany) and standard pressure transducers that had been zeroed to the level of the right atrium. Measurements were taken immediately before each collection of blood and expired samples for inert gas determination. Cardiac output was measured using the thermodilution technique; the mean of three serial measurements was recorded. Arterial and mixed venous samples (2 mL each) were collected immediately after collection of each set of inert gas arterial and mixed venous samples and immediately analyzed for PO_2, PCO_2, pH, oxygen saturation, haemoglobin concentration and haematocrit using a Ciba Corning 806® blood gas analyzer (Ciba-Geigy, Basel, Switzerland). All values were corrected to body temperature. In all animals, the abdominal cavity was inflated with purified air to a pressure of 15 cmH_2O. The pressure in the abdominal cavity was measured with a manometer that had been originally designed to assess the cuff-pressure of tracheal tubes and maintained at 15 cmH_2O in all animals.

Study protocol and measurements

Animals were subjected in random order to PSV and PCV in serial order. Randomization was performed using a standard computer algorithm. Muscular relaxation during PCV was established using intra-venous (i.v.) boluses of mivacurium 0.25 mg kg^{-1}. This was performed when switching to PCV to stop spontaneous ventilation. Positive end-expiratory pressure was set to 8 cmH_2O in all animals during the whole study. After changing to the different mode, 60 min were allowed for equilibration before the subsequent set of measurements was taken. Each set included measurement of heart rate, mean arterial (MAP), central venous (CVP), pulmonary arterial (MPAP), pulmonary capillary wedge pressure (PCWP) and the respiratory measurements described below. Inert gas as well as arterial and mixed venous samples were collected at the same time.

Inert gas elimination technique

The distributions of ventilation and perfusion were determined using the multiple inert gas elimination technique (MIGET) as described [5,6]. A mixture of six inert gases, including sulphur hexafluoride, ethane, cyclopropane, halothane, diethyl ether and acetone, was dissolved in saline and infused into a peripheral vein at 3 mL min^{-1}. This infusion was started 1 h ahead of the first set of measurements. Duplicate 10 mL blood samples were collected in heparinized glass syringes from the pulmonary artery and the left femoral artery. A total of 30 mL mixed expired gas samples was obtained from a heated mixing chamber into warmed gas-tight glass syringes.

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All samples were kept at 39°C and immediately prepared for analysis. Inert gas extraction was carried out according to Wagner and colleagues [5]. Concentrations of inert gases were measured using gas chromatography (HP-5890®, Series II; Hewlett-Packard, Germany). Ventilation–perfusion distributions were then determined from inert gas data using the 50-compartment model of Wagner and colleagues [6,7].

Distributions of \( V_A/Q \) were as follows:

- Blood flow to unventilated lung units, \( \text{shunt} \) \( (V_A/Q < 0.005) \).
- Blood flow to poorly ventilated lung units, \( \text{low} \) \( V_A/Q (V_A/Q > 0.005-0.1) \).
- Blood flow to normally ventilated lung units, \( \text{normal} \) \( V_A/Q (V_A/Q > 0.1-10) \).
- Ventilation of poorly perfused lung units, \( \text{high} \) \( V_A/Q (V_A/Q > 10-100) \).
- Ventilation of unperfused lung units, \( \text{dead space} \) \( (V_A/Q > 100) \).

In addition, the alveolar–arterial PO\(_2\) difference (\( Aa\Delta PO_2 \)) was calculated.

**Mode of augmented spontaneous breathing and ventilator settings**

To achieve comparable ventilation, the following settings were chosen. In all animals, PEEP was set to 8 cmH\(_2\)O. During PSV, the flow-trigger was set to 5 L min\(^{-1}\), pressure support above PEEP was set to 10 cmH\(_2\)O. Settings for PCV were lower pressure level 8 cmH\(_2\)O, upper pressure level 18 cmH\(_2\)O, inspiration time 1.8 s and expiration time 2.2 s. Inspiratory oxygen fraction was 0.3 throughout the study.

**Respiratory measurements**

Airway pressures, expiratory tidal volume, respiratory minute volume and respiratory rate measurements, and carbon dioxide elimination were read from the ventilator display.

**Study protocol and schedule**

All animals were subjected to both modes in a random order. After allowing 60 min for equilibration in each mode, a complete set of measurements including haemodynamics, inert gas and respiratory measurements was taken. The mode was then changed and another 60 min were allowed for equilibration. Two inspiratory holds of 20 s each were performed immediately after each set of measurements to prevent non-specific atelectasis.

**Statistical analysis**

Intergroup differences were examined by a two-factor repeated measurement analysis of variance (two-tailed). Post-hoc comparisons were conducted using the Newman–Keuls test. \( P < 0.05 \) was considered as significant. Results are shown as mean ± SD.

**Results**

**Inert gas measurements**

Data are shown in Table 1. A plot with characteristic distributions of ventilation and perfusion in this experimental setting is shown in Figure 1. The intergroup differences in blood flow to lung units with low or zero \( V_A/Q \) ratio (shunt) did not reach significance, while blood flow to lung units with a normal \( V_A/Q \) ratio was higher during PSV when compared with PCV.

**Arterial blood-gas data**

Data are given in Table 1. Compared with PCV, \( PaO_2 \) was significantly higher during PSV, while \( PaCO_2 \) was significantly lower.

**Haemodynamic measurements**

Data are shown in Table 1. There was no intergroup difference in any haemodynamic parameter observed.

**Respiratory measurements**

Respiratory minute volume (RMV) was 6.6 ± 1.2 L min\(^{-1}\) during PSV and 5.3 ± 1.1 L min\(^{-1}\) during PCV \( (P < 0.01) \). Respiratory rate was 14.2 ± 4 min\(^{-1}\) during PSV and 15 min\(^{-1}\) during PCV (fixed rate) \( (P = 0.8) \). Other respiratory variables were not significantly different in intergroup comparison.

<table>
<thead>
<tr>
<th></th>
<th>PSV</th>
<th>PCV</th>
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<tbody>
<tr>
<td>Shunt (% of cardiac output)</td>
<td>15 ± 2</td>
<td>17 ± 5</td>
</tr>
<tr>
<td>Low ( V_A/Q ) (% of cardiac output)</td>
<td>7 ± 5</td>
<td>10 ± 7</td>
</tr>
<tr>
<td>Normal ( V_A/Q ) (% of cardiac output)</td>
<td>78 ± 4*</td>
<td>72 ± 5</td>
</tr>
<tr>
<td>High ( V_A/Q ) (% of cardiac output)</td>
<td>0.1 ± 0.2</td>
<td>0.1 ± 0.1</td>
</tr>
<tr>
<td>Deadspace (% of RMV)</td>
<td>32 ± 11.0</td>
<td>32 ± 9</td>
</tr>
<tr>
<td>( Aa\Delta PO_2 ) (kPa)</td>
<td>9.7 ± 1.3*</td>
<td>11.2 ± 1.2</td>
</tr>
<tr>
<td>( PaO_2 ) (kPa)</td>
<td>14.9 ± 1.6*</td>
<td>13.7 ± 2.0</td>
</tr>
<tr>
<td>( PvO_2 ) (kPa)</td>
<td>7.3 ± 1.1</td>
<td>7.1 ± 0.5</td>
</tr>
<tr>
<td>( PaCO_2 ) (kPa)</td>
<td>6.0 ± 0.7*</td>
<td>6.9 ± 0.8</td>
</tr>
<tr>
<td>( ETCO_2 ) (kPa)</td>
<td>4.8 ± 1.1*</td>
<td>3.9 ± 1.3</td>
</tr>
<tr>
<td>( CO_2 ) elimination (mL min(^{-1}))</td>
<td>212 ± 27</td>
<td>196 ± 22</td>
</tr>
</tbody>
</table>

RMV, respiratory minute volume; shunt represents blood flow to the lung units with a zero \( V_A/Q \) ratio; low \( V_A/Q \) blood flow to lung units with a low \( V_A/Q \) ratio; normal \( V_A/Q \) blood flow to the lung units with a normal \( V_A/Q \) ratio, and high \( V_A/Q \) blood flow to lung units with a high \( V_A/Q \) ratio; \( Aa\Delta PO_2 \), alveolar–arterial partial pressure difference of oxygen; \( PaO_2 \), \( PaCO_2 \), arterial and mixed venous partial pressures of oxygen; \( ETCO_2 \), end-tidal partial pressure of carbon dioxide. Values are mean ± SD; *\( P < 0.05 \) in intergroup comparisons.
The aim was to evaluate the influence of augmented spontaneous breathing during pneumoperitoneum on pulmonary gas exchange. Pressure-support ventilation was superior to PCV regarding blood flow to lung areas with a normal Vₐ/Q lung units during PCV when compared with PSV. The latter may be of importance in patients with gas exchange disorders. Pressure-support ventilation during anaesthesia may counteract the lung function impairments described by Hedenstierna, being, among others, cephalad displacement of the diaphragm, and redistribution of blood flow to lower, less ventilated lung areas [8]. Nevertheless, one must remember that PSV may also be understood as a patient-triggered controlled ventilation. Two limitations of this study should be mentioned. The results cannot be fully extrapolated to humans as the pig lacks collateral ventilation and as the supine position is not a physiological position for the animal. It is concluded that PSV results in significantly better gas exchange than PCV in a porcine model with an air pneumoperitoneum at a pressure 15 cmH₂O.

Discussion

Equal numbers of animals received each mode first.

Table 2. Haemodynamic variables.

<table>
<thead>
<tr>
<th></th>
<th>PSV</th>
<th>PCV</th>
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<tbody>
<tr>
<td>CI (mL min⁻¹ kg⁻¹)</td>
<td>220 ± 20</td>
<td>214 ± 214</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>112 ± 13</td>
<td>115 ± 13</td>
</tr>
<tr>
<td>CVP (mmHg)</td>
<td>7 ± 3</td>
<td>7 ± 1</td>
</tr>
<tr>
<td>MPAP (mmHg)</td>
<td>20 ± 4</td>
<td>22 ± 4</td>
</tr>
<tr>
<td>PCWP (mmHg)</td>
<td>14 ± 2</td>
<td>14 ± 2</td>
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CI: cardiac index; MAP: mean arterial pressure; CVP: central venous pressure; MPAP: mean pulmonary arterial pressure; PCWP: pulmonary capillary wedge pressure. Values are mean ± SD.

References