VENTILATORY EFFECTS OF LAPAROSCOPY UNDER GENERAL ANAESTHESIA

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SUMMARY

We have studied 14 female patients undergoing elective laparoscopy under general anaesthesia with peritoneal insufflation of carbon dioxide in order to examine changes in physiological deadspace \((V_{Dphys})\), arterial to end-tidal carbon dioxide partial pressure difference \((P_{A,CO_2} - P_{E,CO_2})\) and \(P_{ACO_2}\). \(V_{CO_2}\) increased after insufflation of carbon dioxide with a mean (SD) maximum increase of 32 (28)% compared with the preinsufflation value. \(P_{ACO_2}\) increased also, with a mean (SD) maximum increase of 0.6 (0.58) kPa immediately before carbon dioxide deflation. \(V_{Dphys}\) and \((P_{ACO_2} - P_{E,CO_2})\) increased during laparoscopy, but this was not significant \((P > 0.05)\).

KEY WORDS


In recent years, laparoscopy has become common clinical practice for diagnostic procedures and tubal ligation. The Trendelenburg position, together with increased intra-abdominal pressure, secondary to i.p. insufflation of carbon dioxide results in an increase in central blood volume [1] and a decrease in functional residual capacity \((FRC)\) [2]. Insufflation of carbon dioxide also increases carbon dioxide production \((V_{CO_2})\) [3]. There may also be a change in \(V/Q\) distribution as a result of basal lung compression and redistribution of hydrostatic forces. Although Brampton and Watson [4] observed a decreased arterial to end-tidal carbon dioxide partial pressure difference \((P_{A,CO_2} - P_{E,CO_2})\) during laparoscopy, their study did not include measurement of baseline \((P_{A,CO_2} - P_{E,CO_2})\). The present study was planned to evaluate the changes in \((P_{A,CO_2} - P_{E,CO_2})\) deadspace: tidal volume ratio \((Vb/Vt)\) and physiological deadspace \((V_{Dphys})\) in non-pregnant females during laparoscopy under general anaesthesia.

METHODS AND RESULTS

We studied 14 female patients \((ASA grade 1)\) admitted for day-case diagnostic laparoscopy for primary infertility. Consent was obtained from each patient and local Institutional Ethics Committee approval was obtained. Premedication comprised oral diazepam 0.1 mg kg\(^{-1}\) on the morning of surgery, followed by morphine 0.15 mg kg\(^{-1}\) and promethazine 0.5 mg kg\(^{-1}\) 1 h before induction of anaesthesia. Anaesthesia was induced with thiopentone 4–5 mg kg\(^{-1}\) given slowly over 1 min, and morphine 0.1 mg kg\(^{-1}\). Pancuronium 0.12 mg kg\(^{-1}\) was given to facilitate tracheal intubation and the lungs were ventilated with 67% nitrous oxide in oxygen using a Servo 900B ventilator. The end-tidal carbon dioxide concentration \((\epsilon_{CO_2})\) was measured with a Siemens Elema 930 calibrated according to the manufacturer’s specifications. In addition, a Lung Mechanics Calculator \((\text{Siemens Elema 940})\) was used to measure pause pressure and compliance of the respiratory system. A square-wave flow pattern was used with an inspiration time of 25% and a 10% inspiratory pause. The lungs were ventilated at approximately 15 b.p.m. and minute ventilation \((V)\) was adjusted to 100-120 ml kg\(^{-1}\), to maintain \(\epsilon_{CO_2}\) at about 5%, and kept constant after the initial setting. A radial artery catheter was inserted after induction of anaesthesia to permit withdrawal of arterial blood for analysis. An arterial sample \((2 \text{ ml})\) was taken with the patient in the supine position, before starting insufflation of carbon dioxide \((\text{stage I})\) but at least 10 min after stable ventilation was achieved after tracheal intubation.

The following variables were noted at the time of arterial sampling: \(\epsilon_{CO_2}\), minute production of carbon dioxide \((V_{CO_2})\), ineffective tidal volume \((V_{T ineffective})\), effective tidal volume \((V_{T effective})\), tidal production of carbon dioxide \((V_{T,CO_2})\), compliance of the respiratory system \((Crs)\) and airway pause pressure \((Paw)\). \(V\) and \(Vt\) were calculated from \(V_{T effective}, V_{T ineffective}\) and ventilatory frequency.

Arterial samples were analysed with a Radiometer ABL2 blood-gas analyser which was calibrated every hour. \(V_{Dphys}\) was calculated according to the equation:

\[
V_{Dphys} = V_T \times (P_{A,CO_2} - P_{E,CO_2})/P_b,
\]

where \(P_b\) = barometric pressure.

Laparoscopy was performed using a standard...
TABLE I. Respiratory variables (mean (SD)) at different stages of laparoscopy. **P < 0.01 compared with stage I

<table>
<thead>
<tr>
<th></th>
<th>V̇Dphys (ml kg⁻¹)</th>
<th>V̇CO₂ (ml kg⁻¹)</th>
<th>(P̄aco₂ – PE’co₂) (kPa)</th>
<th>Crs (ml/cm H₂O)</th>
<th>P̄aco₂ (kPa)</th>
<th>P̄ao₂ (kPa)</th>
<th>P̄aw (cm H₂O)</th>
<th>IAP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before insufflation (stage I)</td>
<td>3.46 (0.86)</td>
<td>3.6 (0.51)</td>
<td>0.61</td>
<td>51.27 (13.76)</td>
<td>5.03 (0.55)</td>
<td>19.1</td>
<td>7.9</td>
<td>8.5</td>
</tr>
<tr>
<td>During laparoscopy (stage II)</td>
<td>3.55 (0.86)</td>
<td>4.26** (0.55)</td>
<td>0.62</td>
<td>40.26** (11.3)</td>
<td>5.5** (0.81)</td>
<td>18.03</td>
<td>10.6**</td>
<td>13.0**</td>
</tr>
<tr>
<td>During laparoscopy (stage III)</td>
<td>3.69 (0.8)</td>
<td>4.37** (0.56)</td>
<td>0.71</td>
<td>41.4** (11.6)</td>
<td>5.63** (0.92)</td>
<td>18.5</td>
<td>10.54**</td>
<td>12.2**</td>
</tr>
<tr>
<td>After deflation (stage IV)</td>
<td>3.5 (0.8)</td>
<td>4.70** (0.65)</td>
<td>0.63</td>
<td>51.3 (10.65)</td>
<td>5.56** (0.8)</td>
<td>18.2</td>
<td>8.38</td>
<td>9.5</td>
</tr>
<tr>
<td>Recovery room</td>
<td></td>
<td></td>
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</tbody>
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4.7 (0.25)  12.1 (1.0)

COMMENT

Respiratory changes during laparoscopy occur because of the Trendelenburg position and i.p. insufflation of carbon dioxide. Increase in V̇CO₂ occurs because of absorption from the peritoneal cavity [5]. In some patients the increase in V̇CO₂ was much greater after deflation than during the period of inflation, indicating that absorption continued after the IAP decreased. As minute ventilation was kept constant, the increase in V̇CO₂ resulted in an increase in P̄aco₂. The slight decrease in P̄aco₂ occurring at stage IV in comparison with that at stage III may be a result of improved V̇O₂/V̇T after deflation in the supine position. The decrease in P̄aco₂ after extubation was caused either by decreased absorption of carbon dioxide with time or by the ability of spontaneously breathing patients in the recovery room to compensate for increased production of carbon dioxide, or both [1, 5].

Carbon dioxide absorbed from the peritoneum is partly inhaled and partly stored, resulting in an increase in P̄aco₂. Diaphragmatic elevation resulting from insufflation of carbon dioxide decreases FRC, which increases V̇O₂/Q mismatch and alveolar dead-space (V̇ḊaSW). Anatomical dead-space (V̇ḊaSW) may decrease because of a decrease in the size of airways as a result of decreased FRC. Allocation of V̇Ḋphys to V̇ḊaSW and V̇ḊaSW was not possible, as we did not record expired carbon dioxide graphically [6].

Our finding of an insignificant overall change in (P̄aco₂ – PE’co₂) after insufflation of carbon dioxide is at variance with the finding of Brampton and Watson [4], who found a decrease of 0.44 kPa during laparoscopy. In addition, the direction of change in (P̄aco₂ – PE’co₂) in the present study was variable. A negative value of (P̄aco₂ – PE’co₂) in some patients was not surprising, in view of the temporal mismatching [6].

There was no significant change in P̄ao₂ during laparoscopy. The head-down position, together with peritoneal insufflation decreases FRC, which may increase V̇O₂/Q mismatch and so decrease P̄ao₂. The absence of a significant change during laparoscopy may be the result of a decreased alveolar to arterial oxygen partial pressure difference caused by an increase in cardiac output which occurs after insuf-
flation of carbon dioxide and continues even after deflation [1].

ACKNOWLEDGEMENT

We thank Dr D. Shaw for her help in the preparation of this manuscript.

REFERENCES


