VENTILATORY EFFECTS OF LAPAROSCOPY UNDER GENERAL ANAESTHESIA

G. D. PURI AND H. SINGH

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 ($V_{D}^{phys}$), arterial to end-tidal carbon dioxide partial pressure difference ($P_{a}C_{O_{2}} - P_{E}C_{O_{2}}$) and $P_{a}C_{O_{2}}$. $V_{C_{O_{2}}}$ increased after insufflation of carbon dioxide with a mean (SD) maximum increase of 32 (28)% compared with the preinsufflation value. $P_{a}C_{O_{2}}$ increased also, with a mean (SD) maximum increase of 0.6 (0.58) kPa immediately before carbon dioxide deflation. $V_{D}^{phys}$ and ($P_{a}C_{O_{2}} - P_{E}C_{O_{2}}$) 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 ($V_{C_{O_{2}}}$) [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 ($P_{a}C_{O_{2}} - P_{E}C_{O_{2}}$) during laparoscopy, their study did not include measurement of baseline ($P_{a}C_{O_{2}} - P_{E}C_{O_{2}}$). The present study was planned to evaluate the changes in ($P_{a}C_{O_{2}} - P_{E}C_{O_{2}}$) deadspace: tidal volume ratio ($V_{T}/V_{T}$) and physiological deadspace ($V_{D}^{phys}$) in non-pregnant females during laparoscopy under general anaesthesia.

METHODS AND RESULTS

We studied 14 female patients (ASA grade I) 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 ($P_{E}C_{O_{2}}$) was measured with a Siemens Elema 930 calibrated according to the manufacturer's specifications. In addition, a Lung Mechanics Calculator (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 ($V_{T}$) was adjusted to 100–120 ml kg$^{-1}$, to maintain $P_{E}C_{O_{2}}$ 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 ml) was taken with the patient in the supine position, before starting insufflation of carbon dioxide (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: $P_{a}C_{O_{2}}$, minute production of carbon dioxide ($V_{C_{O_{2}}}$), ineffective tidal volume ($V_{T}^{ineff}$), effective tidal volume ($V_{T}^{eff}$), tidal production of carbon dioxide ($V_{T}P_{C_{O_{2}}}$), compliance of the respiratory system (Crs) and airway pause pressure ($P_{aw}$). $V_{T}$ and $V_{T}$ were calculated from $V_{T}^{eff}$, $V_{T}^{ineff}$ and ventilatory frequency.

Arterial samples were analysed with a Radiometer ABL2 blood-gas analyser which was calibrated every hour. $V_{D}^{phys}$ was calculated according to the equation:

$$V_{D}^{phys} = V_{T} \times \frac{(P_{a}C_{O_{2}} - P_{E}C_{O_{2}})}{P_{aw}}$$

where $P_{aw}$ = barometric pressure.

Laparoscopy was performed using a standard
technique. The patient was placed in the modified lithotomy position with a 15–20° Trendelenburg tilt and carbon dioxide 2 litre was insufflated into the peritoneal cavity at a rate of approximately 1 litre min⁻¹. After the start of laparoscopy, carbon dioxide was insufflated intermittently, if needed, to maintain an intra-abdominal pressure (IAP) of 12–14 mm Hg, sufficient to allow a clear view of the pelvic organs. Total carbon dioxide insufflated was not more than 3 litre in any subject. IAP was measured with an anaerobic manometer attached to the Verres needle which was used to insufflate carbon dioxide. Simultaneous measurements from the carbon dioxide analyser and Lung Mechanics calculator were obtained. Arterial blood sampling was undertaken 10 min after the start of laparoscopy (stage I) and 20 min after the start of laparoscopy or just before the end of insufflation, whichever occurred earlier (stage III). The duration of laparoscopy did not exceed 25 min in any patient. Observations were repeated 10 min after abdominal decompression with the patient supine (stage IV). Arterial blood gas analysis was repeated 15 min after tracheal extubation (approximately 30 min after deflation) in the recovery room with the patient breathing room air. Statistical analysis of results was performed using Student’s paired t test and linear regression analysis.

The patients’ mean age was 30.1 yr (range 24–36 yr); mean (SD) duration of laparoscopy was 19 (3.75) min. Carbon dioxide elimination increased after insufflation of the gas, and remained significantly increased 10 min after deflation (table I). PaCO₂ increased also in comparison with baseline after insufflation of carbon dioxide and it was significantly greater (P < 0.01) throughout stages II–IV. V̇OD/V̇T and V̇O₂phys increased slightly but insignificantly after insufflation of carbon dioxide and decreased towards baseline after deflation. (PaCO₂ - ṖE CO₂) did not change from the pre-insufflation (stage I) value. PaO₂ was not changed by laparoscopy. IAP increased after insufflation and this correlated with the change in airway pause pressure (Paw) (r = 0.85, P < 0.01). There was no significant correlation between the change in Paw and those in V̇O₂/V̇T or PaO₂. The increase in V̇CO₂ was related inversely to the increase in Paw (r = -0.67, P < 0.05).

**TABLE I. Respiratory variables (mean (sd)) at different stages of laparoscopy. **P < 0.01 compared with stage I**

<table>
<thead>
<tr>
<th></th>
<th>V̇D/ḢK (ml kg⁻¹)</th>
<th>V̇CO₂ (ml kg⁻¹)</th>
<th>(PaCO₂ - ṖE CO₂) (kPa)</th>
<th>Crs (ml/cm H₂O)</th>
<th>PaCO₂ (kPa)</th>
<th>PaO₂ (kPa)</th>
<th>Paw (cm H₂O)</th>
<th>IAP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before insufflation</td>
<td>3.46 (0.86)</td>
<td>3.6 (0.51)</td>
<td>0.61</td>
<td>51.27</td>
<td>5.03</td>
<td>19.1</td>
<td>7.9</td>
<td>8.5</td>
</tr>
<tr>
<td>During laparoscopy</td>
<td>3.55 (0.86)</td>
<td>4.26** (0.55)</td>
<td>0.62</td>
<td>40.26**</td>
<td>5.5**</td>
<td>18.03</td>
<td>10.6**</td>
<td>13.0**</td>
</tr>
<tr>
<td>During laparoscopy</td>
<td>3.69 (0.8)</td>
<td>4.37** (0.56)</td>
<td>0.71</td>
<td>41.4**</td>
<td>5.63**</td>
<td>18.5</td>
<td>10.54**</td>
<td>12.2**</td>
</tr>
<tr>
<td>After deflation</td>
<td>3.5 (0.8)</td>
<td>4.70** (0.65)</td>
<td>0.63</td>
<td>51.3</td>
<td>5.56**</td>
<td>18.2</td>
<td>8.38</td>
<td>9.5</td>
</tr>
<tr>
<td>Recovery room</td>
<td></td>
<td></td>
<td></td>
<td>4.7</td>
<td>5.26**</td>
<td>12.1</td>
<td>8.5</td>
<td>(0.25)</td>
</tr>
</tbody>
</table>

**COMMENT**

Respiratory changes during laparoscopy occur because of the Trendelenburg position and i.p. insufflation of carbon dioxide. Increase in VCO₂ occurs because of absorption from the peritoneal cavity [5]. In some patients the increase in VCO₂ 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 VCO₂ resulted in an increase in ṖE CO₂. The slight decrease in ṖE CO₂ 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 ṖE CO₂ after extubation was 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 excreted and partly stored, resulting in increased ṖE CO₂. Diaphragmatic elevation resulting from insufflation of carbon dioxide decreases FRC, which increases V̇Q mismatch and so decrease PaO₂, so that the alveolar dead-space (V̇D/alv). Anatomical deadspace (V̇D/anat) may decrease because of a decrease in the size of airways as a result of decreased FRC. Allocation of V̇D physis to V̇D anat and V̇D alv was not possible, as we did not record expired carbon dioxide graphically [6].

Our finding of an insignificant overall change in (ṖE CO₂ - ṖE 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 (ṖE CO₂ - ṖE CO₂) in the present study was variable. A negative value of (ṖE CO₂ - ṖE CO₂) in some patients was not surprising, in view of the temporal mismatching [6].

There was no significant change in PaO₂ during laparoscopy. The head-down position, together with peritoneal insufflation decreases FRC, which may increase V̇Q mismatch and so decrease PaO₂. 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-
VENTILATORY EFFECTS OF LAPAROSCOPY

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


