Arterial to End-tidal Carbon Dioxide Pressure Difference during Laparoscopic Surgery in Pregnancy

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Background: There is controversy about whether capnography is adequate to monitor pulmonary ventilation to reduce the risk of significant respiratory acidosis in pregnant patients undergoing laparoscopic surgery. In this prospective study, changes in arterial to end-tidal carbon dioxide pressure difference (PaCO₂–PETCO₂), induced by carbon dioxide pneumoperitoneum, were determined in pregnant patients undergoing laparoscopic cholecystectomy.

Methods: Eight pregnant women underwent general anesthesia at 17–30 weeks of gestation. Carbon dioxide pneumoperitoneum was initiated after obtaining arterial blood for gas analysis. Pulmonary ventilation was adjusted to maintain PETCO₂ around 32 mmHg during the procedure. Arterial blood gas analysis was performed during insufflation, after the termination of insufflation, after extubation, and in the postoperative period.

Results: The mean ± SD for PaCO₂–PETCO₂ was 2.4 ± 1.5 before carbon dioxide pneumoperitoneum, 2.6 ± 1.2 during, and 1.9 ± 1.4 mmHg after termination of pneumoperitoneum. PaCO₂ and pH during pneumoperitoneum were 35 ± 1.7 mmHg and 7.41 ± 0.02, respectively. There were no significant differences in either mean PaCO₂–PETCO₂ or PaCO₂ and pH during various phases of laparoscopy.

Conclusions: Capnography is adequate to guide ventilation during laparoscopic surgery in pregnant patients. Respiratory acidosis did not occur when PETCO₂ was maintained at 32 mmHg during carbon dioxide pneumoperitoneum. (Key words: Capnography; carbon dioxide pneumoperitoneum.)

IT is assumed that maintaining end-tidal carbon dioxide pressure (PETCO₂) around 32–34 mmHg prevents significant respiratory acidosis during laparoscopic surgery in pregnant patients.1–5 Successful outcome after laparoscopic surgery has been reported in several parturients (no spontaneous abortions, preterm labors, or premature deliveries reported in 67 parturients).5–11 However, Amos et al.12 documented four fetal deaths in seven pregnant women who underwent laparoscopic cholecystectomy or appendectomy (in these patients, ventilation was adjusted to maintain PETCO₂ around 32–34 mmHg). Although Amos et al. did not measure arterial blood gases, respiratory acidosis was stated as a possible factor contributing to fetal loss.12 Based on studies in pregnant ewes,13,14 Cruz et al.13 questioned the validity of the current practice of estimating arterial carbon dioxide pressure (PaCO₂) by capnography in pregnant women undergoing laparoscopic surgery. Maternal and fetal acidosis occurred in pregnant ewes when PETCO₂ was used to guide ventilation during carbon dioxide insufflation.13,14 In the absence of studies evaluating PaCO₂–PETCO₂ in pregnant patients undergoing laparoscopic surgery, we studied arterial PaCO₂ and PaCO₂–PETCO₂ differences during carbon dioxide insufflation.

Methods

Eight healthy pregnant women at 17, 20, 23, 23, 24, 26, and 30 weeks of gestation scheduled for laparoscopic cholecystectomy for recurrent gallstone cholecystitis in pregnancy were studied. The study protocol was approved by the hospital ethical committee, and all patients provided written informed consent. Fetal heart rate was evaluated by ultrasound before induction of general anesthesia. After treatment with sodium citrate (30 ml administered orally) and denitrogenation of the lungs with 100% oxygen, a rapid sequence induction was performed with sodium pentathol and succinylcholine, and the trachea was intubated with a 7.0-mm endotracheal tube. General anesthesia was maintained with

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desflurane in an air–oxygen mixture, fentanyl and cisatracurium. Left uterine displacement was assured during anesthesia by placing a rolled bed sheet under the right lumbar region. Intraoperative maternal monitoring included continuous electrocardiogram, pulse oximetry, capnography, oropharyngeal temperature, and noninvasive blood pressure measurements. The Ohmeda capnograph (5250 RGM; Ohmeda, Englewood, CO) was calibrated before the commencement of each anesthesia. After induction of general anesthesia, a 20-gauge arterial cannula was introduced into the radial artery, observing sterile precautions. Initial pulmonary ventilation was adjusted to maintain $\text{PETCO}_2$ around 32 mmHg. After obtaining arterial blood for baseline blood gas measurements, peritoneal insufflation of carbon dioxide limited to peak inflation pressure of 15 mmHg was used. Pulmonary ventilation was adjusted to maintain $\text{PETCO}_2$ around 32 mmHg by increasing both the respiratory rate (from 10 to 12 breaths/min) and tidal volume as is currently the practice at our institution (inspiratory time:expiratory time ratio 1:2). Additional samples for measurements of arterial blood gas tensions were obtained during and after the end of carbon dioxide insufflation (approximately every 10 min) and in the postanesthesia care units. Arterial blood gas analysis was performed at $37^\circ C$ using a Nova blood gas analyzer (Stat Profile Ultra; Nova Biomedical, Waltham, MA) after two-point calibration, and the results were corrected to the patient’s temperature. Intravenous ephedrine (10-mg bolus doses) was used if the systolic blood pressure decreased below 20% of the baseline measurements. Fetal heart rate was reassessed 10 min after the patient emerged from general anesthesia and continued into the recovery room until discharge. One-way analysis of variance was used to determine if the difference between the variables in each phase of laparoscopic surgery was statistically significant. A $P$ value $<0.05$ was considered significant. The differences in variables that were significant according to analysis of variance were examined using the Student $t$ test for paired observations.

**Results**

There was no significant differences in mean $\text{PETCO}_2$, $\text{PaCO}_2$, $\text{PaCO}_2–\text{PETCO}_2$, $\text{pH}$, bicarbonate ($\text{HCO}_3$), and base excess–deficit during various phases of laparoscopy (table 1) except in minute volume and peak inspiratory pressures ($P < 0.01$, analysis of variance). The peak inspiratory pressures and minute volume were higher ($P < 0.05$, paired $t$ test) during insufflation compared with before or after carbon dioxide pneumoperitoneum. The mean $\text{PaCO}_2$ value observed during insufflation (fraction of inspired oxygen = 40%) was 245 mmHg (SD, 55), and in the postoperative period was 150 mmHg (SD, 36). The duration of anesthesia was 85.7 min (SD, 13.5; range, 70–110 min), and insufflation period was 36.3 min (SD, 11.6; range, 25–60 min). The mean oropharyngeal temperature was 35.5°C (SD, 0.3; range, 35.1–36°C), and the mean carbon dioxide abdominal insufflation pressure was 11.7 mmHg (SD, 1.3). All parturients had uneventful progress of pregnancy except one patient (30 weeks’ gestation) who had mild uterine contractions without cervical effacement in the immediate postoperative period that required magnesium therapy for 12 h.

**Discussion**

Our results in eight pregnant patients show that there were no significant differences in $\text{PaCO}_2–\text{PETCO}_2$ during carbon dioxide pneumoperitoneum as compared with preinsufflation values during laparoscopic surgery. This is in contrast to results obtained by Cruz et al.15 (nine pregnant ewes) and Hunter et al.14 (four pregnant ewes), in which the difference increased by a mean of 10 mmHg during insufflation. Among the 28 observations obtained in eight patients during carbon dioxide insufflation in our study, the highest $\text{PaCO}_2–\text{PETCO}_2$ observed was 5.1 mmHg as compared with 16 and 25 mmHg observed by Cruz et al.15 and Hunter et al.14 in ewes. The results observed in our study are consistent with our earlier observations of derived $\text{PaCO}_2–\text{PETCO}_2$ of 6–7 mmHg from transcutaneous $\text{PCO}_2$ monitoring in laparoscopic surgery in a parturient.4 Therefore, the physiologic consequences of pneumoperitoneum are different in humans from those in pregnant ewes, as seen by a lowered $\text{PaCO}_2–\text{PETCO}_2$ during insufflation in pregnant patients.

Although the sheep model is used for obstetric research, there could be physiologic differences between the two species. For example, the preinsufflation $\text{PaCO}_2–\text{PETCO}_2$ in pregnant ewes ranges from 6 to 15 mmHg, whereas it is lower in pregnant humans (0.6 mmHg; range, −2.5 to 5.1 mmHg; occasionally $\text{PETCO}_2$ exceeds $\text{PaCO}_2$).16,17 In our study, the values varied from 0 to 5 mmHg, similar to those reported in pregnant subjects undergoing cesarean section and in women during postpartum sterilization.16,17 The smaller preinsufflation $\text{PaCO}_2–\text{PETCO}_2$ in humans translates into a lower
Preinflation alveolar dead space in pregnant patients is lower than in pregnant ewes. A lower preinsufflation alveolar dead space in pregnant patients probably results in a smaller subsequent change in alveolar dead space during carbon dioxide insufflation, and thus to a smaller difference in arterial and end-tidal carbon dioxide (

\[ \text{PaCO}_2 - \text{PETCO}_2 \]

) in pregnant patients than in pregnant ewes. Amos et al. suggested that fetal loss in their series (four of seven fetal deaths, three during first postoperative week and another four weeks postoperatively) could have been a result of prolonged respiratory acidosis despite maintaining end-tidal carbon dioxide (ET\text{CO}_2) in the low to mid 30s. However, they did not have direct information on hypercarbia or acidosis. It is important to note that patients in their series had other conditions, including such risk factors as pancreatitis and perforated appendix, which are generally believed to increase the risk of fetal loss and could have contributed to increased fetal mortality.

Because of the relatively infrequent nature of laparoscopic surgery in pregnancy, it is difficult to accumulate a large number of patients, as surgery is preferably delayed until the conclusion of pregnancy. However, our observations in this prospective study suggest that capnography is adequate to guide ventilation during carbon dioxide insufflation in pregnant patients. Assuming that the maximum difference in arterial and end-tidal carbon dioxide (\( \text{PaCO}_2 - \text{PETCO}_2 \)) of approximately 7 mmHg (as observed in our study) could occur during anesthesia, a ET\text{CO}_2 of 32 mmHg should allow \( \text{PaCO}_2 \) in the range not greater than \( \text{PaCO}_2 \) that is usually encountered in the postoperative period after laparoscopic surgery in pregnant patients. At our institution, the outcome has been successful in 23 patients (retrospective study, 10 subjects, 1991–1995; case report, 1 subject, 1997; present prospective study, 8 subjects, 1998–1999; and 4 subjects outside of these studies) who underwent laparoscopic surgery during pregnancy.

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| Table 1. Mean (SD) Values for \( \text{PaCO}_2, \text{PETCO}_2, \text{PaCO}_2 - \text{PETCO}_2, \text{pH}, \text{Bicarbonate (HCO}_3\text{)}, \text{Base Deficit–Excess, Minute Ventilation, and Peak Inspiratory Pressures} |
|-------------------------------------------------|---------------------------------|---------------------------------|-----------------|-----------------|
| Preinsufflation | During Insufflation | Postinsufflation | Postextubation | Postanesthesia Care Unit |
| \( \text{PETCO}_2 \) (mmHg) | 32.1 (1.6) | 32.4 (1.1) | 32.7 (1.4) | 32.1 (1.6) |
| \( \text{N} = 17 \) | \( \text{N} = 28 \) | \( \text{N} = 12 \) | \( \text{N} = 8 \) | \( \text{N} = 12 \) |
| \( \text{PaCO}_2 \) (mmHg) | 34.5 (2.6) | 35 (1.7) | 34.6 (2.5) | 34.9 (2.9) |
| \( \text{N} = 17 \) | \( \text{N} = 28 \) | \( \text{N} = 12 \) | \( \text{N} = 4 \) | \( \text{N} = 8 \) |
| \( \text{PaCO}_2 - \text{PETCO}_2 \) (mmHg) | 2.4 (1.5) | 2.6 (1.2) | 1.9 (1.4) | 2.4 (1.5) |
| \( \text{N} = 17 \) | \( \text{N} = 28 \) | \( \text{N} = 12 \) | \( \text{N} = 4 \) | \( \text{N} = 8 \) |
| \( \text{pH} \) | 7.42 (0.02) | 7.41 (0.02) | 7.40 (0.02) | 7.41 (0.02) |
| \( \text{N} = 17 \) | \( \text{N} = 28 \) | \( \text{N} = 12 \) | \( \text{N} = 4 \) | \( \text{N} = 8 \) |
| \( \text{HCO}_3 \) (mm) | 25.2 (1.6) | 24.6 (1.4) | 24.5 (1.5) | 24.5 (1.4) |
| \( \text{N} = 17 \) | \( \text{N} = 28 \) | \( \text{N} = 12 \) | \( \text{N} = 4 \) | \( \text{N} = 8 \) |
| Base deficit | 0.1 (1.5) | \(-0.29 (1.3)\) | \(-0.5 (1.3)\) | \(-0.8 (1.3)\) |
| \( \text{N} = 17 \) | \( \text{N} = 28 \) | \( \text{N} = 12 \) | \( \text{N} = 4 \) | \( \text{N} = 8 \) |
| Minute ventilation (l/min) | 5.6 (1.2) | 7.2 (1.2) | 5.8 (1.2) | 5.6 (1.2) |
| \( \text{N} = 26 \) | \( P < 0.01^* \) | \( \text{N} = 26 \) | \( \text{N} = 26 \) | \( \text{N} = 8 \) |
| Peak inspiratory pressures (cm H_2O) | 19.5 (4.6) | 24.6 (5) | 19.8 (4.2) | 19.5 (4.6) |
| \( \text{N} = 26 \) | \( P < 0.01^* \) | \( \text{N} = 26 \) | \( \text{N} = 26 \) | \( \text{N} = 8 \) |

Values are mean (SD), number of observations (N), and 95% confidence intervals.

* Significant difference from preinsufflation and postinsufflation values.
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