Clinical Reports

Transcutaneous PCO_2 monitoring during laparoscopic cholecystectomy in pregnancy

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Purpose: Respiratory acidosis during carbon dioxide (CO_2) insufflation has been suggested as a cause of spontaneous abortion and preterm labour following laparoscopic cholecystectomy during pregnancy. Capnography may not be adequate as a guide to adjust pulmonary ventilation during laparoscopic surgery and hence arterial carbon dioxide ($PaCO_2$) monitoring has been recommended. We report the feasibility and benefits of transcutaneous carbon dioxide monitoring ($PtcCO_2$) as an approach to optimise ventilation during laparoscopic surgery in pregnancy.

Method: A healthy parturient received general anaesthesia for laparoscopic cholecystectomy. Pulmonary ventilation was adjusted to maintain end-tidal carbon dioxide (conventional $P_{ET}CO_2$) at 32 mmHg during CO_2 insufflation. A $PtcCO_2$ monitor was used to trend $PaCO_2$ throughout the procedure. Mechanical ventilation was interrupted every five minutes to obtain an end-tidal PCO_2 value at large tidal volume (squeeze $P_{ET}CO_2$).

Results: The $PtcCO_2$ increased from 39 mmHg before induction to 45 mmHg after CO_2 insufflation. This corresponds to an estimated maximum $PaCO_2$ of 39–40 mmHg during insufflation. The $PtcCO_2$ gradually returned to pre-induction baseline values one hour after the termination of CO_2 insufflation. Squeeze $P_{ET}CO_2$ values approximated $PtcCO_2$ more closely than did conventional $P_{ET}CO_2$ values ($P < 0.01$).

Conclusion: Continuous $PtcCO_2$ measurements as well as squeeze $P_{ET}CO_2$ may be of clinical value in trending and preventing hypercarbia during laparoscopic surgery.

Objectif : L'acidose respiratoire durant l'insufflation au CO_2 a été proposée comme cause d'avortement spontané et de travail prématuré à la suite de cholécystectomie laparoscopique durant la grossesse. La capnométrie peut ne pas être un guide adéquat pour ajuster la ventilation durant la chirurgie laparoscopique et de ce fait le monitoring du CO_2 artériel a été recommandé. Cette étude rapporte la faisabilité et les bénéfices du monitorage du CO_2 par voie transcutanée ($PtcCO_2$) comme mesure d'optimalisation de la ventilation durant la chirurgie laparoscopique pendant la grossesse.

Méthode : Une parturiente en bonne santé a subi une cholécystectomie par laparoscopie sous anesthésie générale. La PCO_2 en fin d'expiration ($P_{ET}CO_2$ conventionnelle) a été maintenue à 32 mmHg durant l'insufflation de CO_2 en ajustant la ventilation pulmonaire. Un moniteur de PCO_2 transcutanée a été utilisé pour établir la tendance durant la procédure. A toutes les cinq minutes, la ventilation mécanique était interrompue pour mesurer la PCO_2 de fin d'expiration suite à un volume courant élevé ("Squeeze" $P_{ET}CO_2$).

Résultats : La $PtcCO_2$ était de 39 mmHg avant l'induction et a augmenté à 45 mmHg après l'insufflation avec le CO_2. Ceci correspond à une $PaCO_2$ maximale estimée de 39–40 mmHg durant l'insufflation. La $PtcCO_2$ a retrouvé progressivement les valeurs de base une heure après la fin de l'insufflation de CO2. La PETCO2 suite à un volume courant élevé a une meilleure corrélation avec la $PtcCO_2$ que la $P_{ET}CO_2$ normale ($P < 0,01$).

Conclusion : Les mesures de la $PtcCO_2$ de même que celles de la $P_{ET}CO_2$ suite à un volume courant élevé peuvent être cliniquement utiles durant la chirurgie laparoscopique pour établir la tendance et prévenir l'hypercarbie.

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CHOLECYSTECTOMY is the second most common nonobstetric operation during pregnancy (1 to 8 of every 10,000 pregnancies).[1] In one series, up to 50% were performed laparoscopically.[2] Laparoscopic cholecystectomy is less invasive than conventional open cholecystectomy and allows for earlier recovery, less postoperative pain, shorter hospital stay and decreased hospital cost.[1–6] Although uncomplicated completion of pregnancy after laparoscopic surgery has been noted in several reports,[1–6] others[7,8] have reported foetal death, spontaneous abortion, and preterm labour in pregnant patients shortly after undergoing laparoscopic surgery. A possible cause of such morbidity is respiratory acidosis secondary to carbon dioxide (CO_2) insufflation to produce pneumoperitoneum.[7,9,10]

Based on studies in a gravid ewe model, Cruz *et al.*,[9] and Hunter *et al.*[10] suggested that capnography may not be adequate to guide ventilation during CO_2 insufflation in parturients, and they recommended arterial catheterisation for CO_2 monitoring.[9,10] In contrast, maintenance of $P_{ET}CO_2$ at 32–36 mmHg has been used by others to adjust pulmonary ventilation during laparoscopic cholecystectomy in pregnant women without apparent untoward effects on the mother or baby.[2,3] Furthermore, Pucci and Seed[11] suggested laparoscopic surgery would have little effect on arterial PCO_2 ($PaCO_2$) or pH, although they did not comment on $P_{ET}CO_2$ measurements during surgery. Amos *et al.*,[7] however, reported four foetal deaths in seven pregnant women who underwent laparoscopic cholecystectomy or appendectomy (in these patients ventilation was adjusted to maintain $P_{ET}CO_2$ in the low to mid 30s), but $PaCO_2$ levels were not measured. Thus, there is a controversy regarding the need for $PaCO_2$ measurements in parturients undergoing laparoscopic surgery,[3,7,9,10] since no studies have evaluated arterial to end-tidal gradient ($PaCO_2 - P_{ET}CO_2$) in pregnant patients undergoing laparoscopic surgery, and successful outcomes have been reported following laparoscopic surgery without measurement of $PaCO_2$.

One approach to this problem is to use transcutaneous CO_2 monitoring ($PtcCO_2$) to estimate $PaCO_2$, as well as $PaCO_2$-$P_{ET}CO_2$ during laparoscopic cholecystectomy in parturients. We report a case illustrating the feasibility and potential benefits of this approach. The use of transcutaneous CO_2 monitoring in laparoscopic surgery has not been described previously.

Case report

A healthy 27-yr-old pregnant woman (20-wk gestation) with acute cholelithiasis presented for elective cholecystectomy. Foetal heart rate was evaluated by ultrasound prior to induction of general anaesthesia. Following treatment with sodium citrate (30 ml *po*) and denitrogenation of the lungs with oxygen 100%, a rapid sequence induction was performed with thiopentone and succinylcholine, and the trachea was intubated with a 7.0 mm endotracheal tube. General anaesthesia was maintained with isoflurane in air/oxygen mixture, fentanyl and cisatracurium. Intraoperative monitoring included ECG, pulse oximetry, capnography, oropharyngeal temperatures and non-invasive blood pressure measurements every one to three minutes during the case. Pulmonary ventilation was adjusted to maintain the $P_{ET}CO_2$ (conventional $P_{ET}CO_2$) at 32 mmHg by varying both respiratory rate and tidal volume (Ohmeda ventilator 7850). Mechanical ventilation was interrupted every five minutes with manually administered 900 ml (large tidal volume) breaths, and adequate expiratory time was allowed for registering the maximum values of end-tidal CO_2 (squeeze $P_{ET}CO_2$).[12] The capnographic data and oropharyngeal temperatures were recorded every 20 sec and saved on computer disc (Ohmeda disk logging software). Peritoneal insufflation of CO_2 was limited to a peak inflation pressure of 15 mmHg. At the end of the procedure, isoflurane was discontinued and muscle relaxation was reversed using neostigmine and glycopyrrolate. Foetal heart rate was re-assessed 10 min after the patient emerged from general anaesthesia.

Transcutaneous PCO_2 ($PtcCO_2$) was measured with a Novametrix® 850 TCO_2M monitor. Following a two point calibration using known concentrations of CO_2 from gas cylinders, adequate time (15 min) was allowed to produce consistent stable $PtcCO_2$ measurements with the sensor placed over the forearm. Once stabilisation was achieved preoperatively, $PtcCO_2$ measurements were recorded every minute during anaesthesia, and at 15 min intervals in the postoperative period until $PtcCO_2$ values returned to the preoperative baseline $PtcCO_2$ value. Transcutaneous CO_2 measurements were corrected for the temperature difference between the heated sensor and oropharyngeal temperature (anaerobic heating coefficient of blood).[13,14]

An arterial catheter was not placed in our patient in keeping with our institution's approach to the perioperative management of parturients undergoing laparoscopic surgery.[3] We decided to perform arterial blood gas analysis should there be clinical manifestations of respiratory acidosis or an increase in $PtcCO_2$ greater than >10 mmHg from baseline values.

Results

The figure shows the one minute trend of conventional $P_{ET}CO_2$ and $PtcCO_2$ measurements in addition to squeeze $P_{ET}CO_2$ values during anaesthesia. The table shows minute ventilation, peak inspiratory pressures,

TABLE The $P_{ET}CO_2$ (conventional), $PtcCO_2$, $PtcCO_2$-conventional $P_{ET}CO_2$, $PtcCO_2$-squeeze $P_{ET}CO_2$, VE (minute ventilation), and PIP (peak inspiratory pressure) values (mean ± SEM) at various phases of laparoscopic surgery.

	mmHg	*$P_{ET}CO_2$ mmHg*	*$PtcCO_2$ mmHg*	*$PtcCO_2$- $P_{ET}CO_2$ Squeeze $P_{ET}CO_2$*	*$PtcCO_2$- VE (L/min)*	*PIP (cm water)*
Pre-induction	32.5 ± 0.3	39.1	6.6 ± 0.3			
Pre-insufflation	32.4 ± 0.1	39.1 ± 0.1	6.7 ± 0.1	6.6	4.3	14
During insufflation	34.8 ± 0.3	42.0 ± 0.3	7.2 ± 0.2		4.7 ± 0.2	16.4 ± 0.4
During insufflation/increased ventilation	32.8 ± 0.3	45.7 ± 0.1	12.9 ± 0.3	8.7 ± 0.8	7.4 ± 0.2	22.5 ± 0.5
After insufflation	30.9 ± 0.2	44.7 ± 0.1	13.7 ± 0.2	12.2 ± 0.4	4.8 ± 0.2	16 ± 0.5
Spontaneous breathing	39.4 ± 0.6	45.8 ± 0.1	6.4 ± 0.6		4.6 ± 0.9	

conventional $P_{ET}CO_2$, $PtcCO_2$, and differences between $PtcCO_2$ and both conventional and squeeze $P_{ET}CO_2$ (several recordings of each variable were made during each phase of the procedure). The $PtcCO_2$-$P_{ET}CO_2$ changed from 6.7 ± 0.6 mmHg to 7.2 ± 0.2 mmHg during the first four minutes after CO_2 insufflation. Thereafter, the $P_{ET}CO_2$ began to rise, and minute ventilation was progressively increased to maintain $PETCO_2$ at 32 mmHg (maximal increase approximately 60%; tidal volume from 570 to 661 ml, respiratory rate from 8 to 11 bpm). The maximum difference observed between $PtcCO_2$ and conventional $P_{ET}CO_2$ during insufflation was 12.9 ± 0.3 mmHg (Figure). Each of the squeeze $P_{ET}CO_2$ values (at tidal volumes 903 ± 40.2 ml) was higher than the corresponding conventional $P_{ET}CO_2$ determination (Mean squeeze = 36.6 ± 0.5 mmHg, higher than the mean conventional = 32.5 ± 0.5 mmHg, t test, $P < 0.01$, Figure). Thus, there was less of a difference between $PtcCO_2$ and squeeze $P_{ET}CO_2$ than between $PtcCO_2$ and conventional $P_{ET}CO_2$ during the 22 min of pneumoperitoneum (8.7 ± 0.8 mmHg *vs* 12.9 ± 0.3, t test, $P < 0.01$). There were no changes in the shape of capnograms during or after insufflation of CO_2. Transcutaneous CO_2 measurements returned to preoperative levels by one hour following the termination of anaesthesia. Heart rate and blood pressure remained within 15% of baseline values throughout the procedure. No arterial blood gas measurement were performed because there were neither clinical manifestations of respiratory acidosis nor large increases in $PtcCO_2$ (>10 mmHg) from baseline values. In addition, no abnormalities occurred on postoperative foetal heart rate monitoring. The patient's pregnancy proceeded uneventfully and a healthy baby was delivered at 38-wk gestation with Apgar scores of 9 and 9 at one and five minutes.

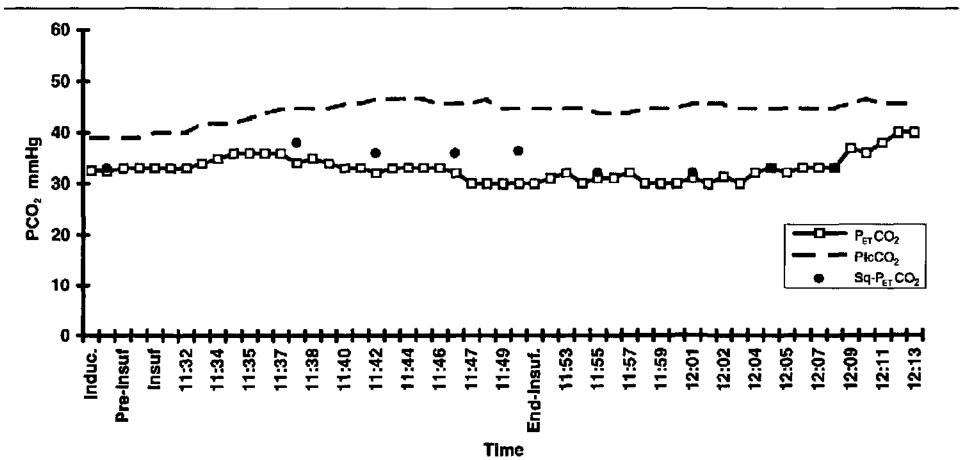

FIGURE Trends of transcutaneous PCO_2 ($PtcCO_2$), conventional end-tidal PCO_2 ($P_{ET}CO_2$), and squeeze $P_{ET}CO_2$ (sq- $P_{ET}CO_2$) during the course of anaesthesia.

Discussion

Transcutaneous CO_2 is measured with a sensor that utilises an unique pH electrode based on the Stow-Severinghaus principle.[14–16] The CO_2 sensor is composed of two parts:[14] (1) the sensor itself which consists of a pH electrode, reference electrode, electrolyte and a CO_2 permeable membrane, and (2) a heating element with precision thermistors to measure and control the sensor temperature. When the sensor is exposed to CO_2, the CO_2 molecules diffuse through the membrane, react with the electrolyte solution, and alter its pH. This in turn alters the potential difference between pH and reference electrodes. The voltage change is amplified and converted to CO_2 tension at the sensor/membrane interface, and displayed as $PtcCO_2$ value. The CO_2 sensor is heated to improve the response time of the measurements. Increased temperature, however, increases local blood and tissue PCO_2 values. Thus, a temperature correction factor (anaerobic heating coefficient of blood) is applied to measured $PtcCO_2$ values. Corrected $PtcCO_2$ measurements correlate well, virtually in all clinical conditions, with $PaCO_2$ values, and are typically higher by 5.2–6.4 mmHg;[16–18] the difference is attributable to skin metabolism and arteriole-cellular CO_2 difference.

Reid *et al.* studied the correlation between $PtcCO_2$ values and $PaCO_2$ values (over a wide range of $PaCO_2$ values 28–62 mmHg) in adults undergoing general anaesthesia. They concluded that transcutaneous devices provide an effective measure of monitoring CO_2 in situations where continuous, precise control of $PaCO_2$ levels is desired.[19] Similarly, Drummond *et al.* concluded that $PtcCO_2$ monitoring following craniotomy can be helpful to identify spontaneously breathing adult patients who are at risk of developing hypercarbia in the postoperative period.[20]

The underutilisation of valuable $PtcCO_2$ technology probably relates to the following disadvantages of transcutaneous CO_2 measurements: skin burns from heated electrodes, injury to skin by adhesive, excessive drift of electrodes, erratic behaviour in the presence of acidosis, long calibration and stabilisation intervals, and the need to change the sensor site every two to four hours.[21] However, short term use of newer monitors with rapid initial stabilisation (10–15 min) and response times (changes in $PtcCO_2$ values tend to lag one to two minutes behind $PaCO_2$ changes) utilising lower electrode temperatures (39° *vs* 45°C) can minimise the listed disadvantages of $PtcCO_2$ monitors.

Since $PtcCO_2$ values typically exceed $PaCO_2$ by 5–6 mmHg,[16–18] the predicted $PaCO_2$-$P_{ET}CO_2$ at preinsufflation in our patient was most likely 0.5–1.5 mmHg ($PtcCO_2$-$P_{ET}CO_2$ = 6.6 mmHg). This appears to be within the normal range for pregnant women (-1 to 0.75 mmHg),[12,22,23] and is lower than in pregnant ewes (6–15 mmHg).[9,10] It is noteworthy that the maximum increase in $PtcCO_2$ observed in our patient was 6.6 mmHg (corresponding to a $PaCO_2$ of approximately 39–40 mmHg) following insufflation of CO_2. This implies that the increase in $PaCO_2$-$P_{ET}CO_2$ during CO_2 insufflation was about 6–7 mmHg, which is lower than that reported in gravid ewes (16 mmHg).[9,10] However, since we did not measure $PaCO_2$ in our case, we are unable to confirm our speculation about the relationship between $PaCO_2$ and $P_{ET}CO_2$ in parturients during laparoscopic surgery.

Interestingly, "squeeze PCO_2 breaths" were associated with a higher end-tidal PCO_2 (by 4.2 mmHg), thus resulting in smaller transcutaneous to end-tidal PCO_2 difference and, hence, smaller arterial to end-tidal PCO_2 difference than with conventional mechanical breaths. Inadequate end-tidal PCO_2 sampling, which increases arterial to end-tidal PCO_2 difference, is not uncommon when decreases in total thoracic compliance (e.g., pneumoperitoneum) coupled with increases in respiratory rate are used to prevent pneumoperitoneum-induced hypercarbia.[24,25] This is because gas from those alveoli with the highest PCO_2 remains within the airways (anatomical deadspace) and does not reach the CO_2 sensor. In such cases, intermittently assisted, squeeze breaths can increase end-tidal PCO_2 (squeeze $P_{ET}CO_2$) and better reflects alveolar PCO_2 at end-expiration.

The $PtcCO_2$, which increased upon CO_2 insufflation from 39 to 45 mmHg (corresponding to $PaCO_2$ from 33–34 to 39–40 mmHg), remained higher than pre-insufflation values in the immediate postoperative period. This may have resulted from residual anaesthetic effects on pulmonary ventilation. The $PtcCO_2$ gradually returned to pre-insufflation values within one hour, and this delayed return was not associated with abnormalities in postoperative foetal heart rate monitoring. Nonetheless, our findings suggests that respiratory acidosis may persist into the early postoperative period, and further studies are needed to determine the extent and frequency of respiratory acidosis in parturients undergoing laparoscopic surgery.

Amos *et al.*[7] suggest that the foetal loss in their series (4/7 foetal deaths, three during the first postoperative week and another four weeks postoperatively) could have been due to prolonged respiratory acidosis despite maintenance of $P_{ET}CO_2$ of low to mid 30s. They did not, however, measure $PaCO_2$ or $PtcCO_2$ and have no direct information on hypercarbia or acidosis. Moreover, it is important to note that parturients in their series had other conditions, including such risk factors as pancreatitis and perforated appendix, which are generally felt to increase the risk of foetal loss and could have contributed to increased foetal mortality.[7]

The normal pH and $PaCO_2$ in the human foetus are 7.35 and 45 mmHg respectively.[10] Normal carbon dioxide and pH gradients across the placenta are 8–10 mmHg and 0.05-0.08 respectively.[10,26] A linear relationship exists between maternal $PaCO_2$ and foetal $PaCO_2$.[26] Therefore, a change in maternal $PaCO_2$ of 6–7 mmHg produces a change < 0.1 in foetal pH, which is probably within safe limits, as it is generally felt that foetal pH > 7.25 is not worrisome, and isolated foetal hypercarbia and concomitant low pH is not as detrimental to the foetus as the presence of metabolic acidosis consequent to hypoxia.[10,27] Furthermore, a mild foetal acidosis may benefit the foetus by improving tissue oxygen unloading by right-shifting the foetal haemoglobin dissociation curve.[10,28]

Although long term effects of a transient episode of foetal acidosis during pregnancy are unknown, it may be considered to be benign, as evidenced by successful outcome following laparoscopic surgery in several parturients (no spontaneous abortions, preterm labours or premature deliveries reported in 67 parturients).[1–6] On the other hand, excessive hypercarbia ($PaCO_2$ > 60 mmHg) in parturients has been associated with decreased uterine and umbilical circulation, and foetal heart rate changes (foetal PCO_2 > 60 mmHg).[26,29] Hence, a $PtcCO_2$ monitor, if available, can provide important trend information about maternal $PaCO_2$ and facilitates the prevention of excessive foetal hypercarbia.

Conclusion

We feel continuous $PtcCO_2$ measurements may be of clinical value for the prevention of hypercarbia in parturients undergoing laparoscopic operations. By adjusting pulmonary ventilation to achieve $P_{ET}CO_2$ values of 32 mmHg, we did not observe excessive hypercarbia (via $PtcCO_2$ monitoring) in our patient during CO_2 pneumoperitoneum. Squeeze end-tidal PCO_2 monitoring provides an additional guide to adequacy of ventilation during CO_2 insufflation in parturients, although a larger study is required to validate the use of capnographic data for predicting either $PaCO_2$ or $PtcCO_2$.

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