Intraabdominal Carbon Dioxide Insufflation in the Pregnant Ewe

Uterine Blood Flow, Intraamniotic Pressure, and Cardiopulmonary Effects

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Background: Laparoscopic surgical procedures are being performed in pregnant women with increasing frequency. Maternal-fetal physiology changes occurring during intraabdominal carbon dioxide insufflation are poorly understood, and maternal-fetal safety is of concern during carbon dioxide pneumoperitoneum. A previous pilot study using end-tidal carbon dioxide-guided ventilation resulted in maternal and fetal acidosis and tachycardia during carbon dioxide pneumoperitoneum. Using serial arterial Pco2 to guide ventilation, this study was designed to evaluate maternal-fetal cardiopulmonary status, uterine blood flow, and the intraamniotic pressure effects of intraabdominal carbon dioxide insufflation in singleton pregnant ewes between 120 and 135 days of gestation.

Methods: In a prospective randomized cross-over study, nine ewes were to receive either abdominal insufflation with carbon dioxide to an intraabdominal pressure of 15 mmHg (n = 9; insufflation group) or receive no insufflation (n = 9; control group). Anesthesia was induced with thiopental and maintained with end-tidal halothane (1 to 1.5 minimum alveolar concentration/100% oxygen). Mechanical ventilation was guided by serial maternal arterial blood gas analysis to maintain Pao2 between 35 and 40 mmHg. Data from insufflated animals were collected during insufflation (60 min) and after desufflation (30 min). Control group data were collected and matched to similar time intervals for 90 min. Ewes were allowed to recover, and after a rest period (48 h) they were entered in the cross-over study.

Results: During insufflation there was a significant increase (P < 0.05) in maternal Pao2 to end-tidal carbon dioxide gradient and minute ventilation, with concomitant decreases in maternal end-tidal carbon dioxide and PaCO2. Intraamniotic pressure increased significantly during insufflation. No significant changes were observed in maternal hemodynamic variables, fetal variables, or in uterine blood flow during the study. There were no fetal deaths or preterm labor in any of the animals during the experiment.

Conclusions: During the 1-h insufflation, a marked increase in Pao2 to end-tidal carbon dioxide gradient was observed, suggesting that capnography may be an inadequate guide to the pressure changes observed during the carbon dioxide pneumoperitoneum in the pregnant patient. No other significant circulatory changes were observed. (Key words: Anesthesia, obstetrics; maternal; fetal; cardiovascular effects. Carbon dioxide pneumoperitoneum. Laparoscopy. Pregnancy. Uterine blood flow.)

LAPAROSCOPIC surgery has become a common therapeutic approach to treat various surgical conditions, particularly cholecystectomy. Conditions such as cholecystitis, ovarian adnexal mass, or appendicitis may require surgical intervention during pregnancy, but abdominal pain during pregnancy can present a diagnostic challenge, and the use of laparoscopy as a diagnostic tool has been reported to reduce the incidence of laparotomies.1

Previous studies have evaluated the cardiopulmonary effects of intraabdominal carbon dioxide (IACO2) insufflation and shown it to be well tolerated in healthy
nongravid patients.\textsuperscript{2,3} Laparoscopic cholecystectomy, appendectomy, and ovarian cystectomy have been reported during pregnancy.\textsuperscript{4,5} Physiologic changes of pregnancy, predominantly in the respiratory and cardiovascular systems,\textsuperscript{6} may contribute to a unique cardiopulmonary response during carbon dioxide pneumoperitoneum in the pregnant patient and should be considered before implementing laparoscopic techniques in these patients.

Studies in animal models have been published focusing on maternal and fetal cardiopulmonary changes during IACO\textsubscript{2} insufflation.\textsuperscript{9,10} Such studies using end-tidal carbon dioxide (ETCO\textsubscript{2})-guided ventilation in gravid models have shown maternal and fetal acidosis and tachycardia during IACO\textsubscript{2} insufflation,\textsuperscript{9,10} and the effects of carbon dioxide pneumoperitoneum on maternal-fetal well being remain a well-founded concern.

This prospective randomized cross-over study was designed to characterize the effects of IACO\textsubscript{2} insufflation on maternal-fetal cardiopulmonary status, uterine blood flow (UBF), and intraamniotic pressure (IAP) using serial maternal PA\textsubscript{CO\textsubscript{2}}, to guide ventilation to maintain maternal normocarbia.

Materials and Methods

Animal Preparation

Nine Dorset-cross ewes with singleton pregnancies of 120 to 135 days' gestation (full-term at 150 days) were used in this study, which was approved by the Animal Care Protocol Review Committee, University of Saskatchewan, and conducted under guidelines of the Canadian Council of Animal Care guidelines. The ewes were fasted for 18 h and deprived of water for 8 h before instrumentation and study.

A lumbar epidural injection of 10 ml lidocaine hydrochloride with epinephrine (lidocaine with epinephrine 2%; Langford, Guelph, Ontario, Canada) and 0.07 mg/kg xylazine hydrochloride (Rompun; Bayvet Division, Chemagro Ltd., Etobicoke, Ontario, Canada) was aseptically administered. Animals were placed in dorsal recumbency with oxygen (inspired oxygen concentration 1.0) \textit{via} face mask (5 l/min) and received balanced electrolyte solution (5% dextrose and 10 ml·kg\textsuperscript{-1}·h\textsuperscript{-1} Ringers) intravenously. After a ventral midline laparotomy, the pregnant uterine horn was identified and a 10-cm hysterotomy incision was made to exteriorize the fetal hindlimb. Using local infiltration with 2% lidocaine and a surgical cutdown, the fetal femoral artery was isolated and catheterized with a 21-gauge over-the-needle catheter. Using the Seldinger technique, a 5-French, 6-cm customized silastic infant feeding tube (Bard Canada, Mississauga, Ontario, Canada) was placed over a pediatric J-wire (central vein catheterization set; Arrow International, Reading, PA) and inserted through the femoral artery into the descending aorta. The infant feeding tube was connected to arterial pressure tubing (Abbott Laboratories, North Chicago, IL) and secured with sutures and adhesive (Vetbond; 3M Canada, Toronto, Ontario, Canada). A 12-French Kaslow stomach tube (Baxter Healthcare Corporation, Deerfield, IL) was placed in the amniotic cavity to monitor IAP. The two catheter sets were exteriorized through one end of the hysterotomy incision. The fetal surgical site and uterine incision were closed with 2-0 absorbable sutures. A calibrated transit time ultrasonic flow probe (model T201; Transonic Systems, Ithaca, NY) was placed around the uterine artery of the pregnant horn just proximal to the arterial bifurcation. The two intrauterine catheters and the Transonic cable were exteriorized through the ewe's right flank and the laparotomy site was closed. One hundred twenty milliliters of warm saline solution with 500 mg of sodium ampicillin (Penbritin 500; Ayerst Laboratories, Montreal, Quebec, Canada) were infused into the amniotic cavity through the amniotic catheter to replace amniotic fluid lost during the procedure.

After local infiltration and surgical cutdown, the maternal carotid artery was catheterized using a 16-gauge, 14-cm catheter (Angiocath, Becton-Dickinson, Sandy, UT) and attached to noncompliant pressure tubing. An 8-French pulmonary artery catheter introducer (Cords Introducer, Daig Corp., Minnetonka, MN) was placed and secured in the jugular vein. Postoperative analgesia consisted of 0.2 mg/kg butorphanol tartrate (Torbugesic; Ayerst Laboratories) intramuscularly, after which ewes were returned to their pen. Antimicrobial therapy consisted of 25,000 iu/kg procaine penicillin (Ethacillin; Rogar/STB, London, Ontario, Canada) given intramuscularly and an intraamniotic infusion of 500 mg sodium ampicillin once daily.

Experimental Design

The animals were randomized to receive two treatments: insufflation to an intraabdominal pressure of 15 mmHg and no insufflation (controls). No laparoscopic surgery was done.

Ewes were rested for 48 h between implantation and the time of the study. Once the first study was complete, the ewes rested for 3 h, and then entered the second phase.

Before incision, arterial blood samples were drawn. Arterial pH was normally 7.35 to 7.45. 

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The ewes recovered from anesthesia, rested for 48 h, and then entered the cross-over study.

Before inducing anesthesia, fetal and maternal arterial blood samples were drawn. If fetal acidemia (pH < 7.2) was present, the study was postponed until the fetal pH was normal. After anesthesia was induced with 10 to 15 mg/kg sodium thiopental (Pentothal, Abbott Laboratories, Montreal, Quebec, Canada), the trachea was intubated and the animal maintained at a constant end-tidal halothane (MTC Pharmaceuticals, Cambridge, Ontario, Canada) administered at 1 to 1.5 minimum alveolar concentration (MAC; 1 MAC was taken as 0.78%[1]) and 100% oxygen. Continuous skeletal muscle relaxation was provided with 0.01 mg/kg intravenous vecuronium bromide (Norcuron; Organon Canada Ltd., West Hill, Ontario, Canada) and monitored using a peripheral nerve stimulator (Digi Stim III, Neurotechnology, Houston, TX) placed over the peroneal nerve.

In both insufflation and control groups, changes in mechanical ventilation were made to maintain $P_{acO_2}$ between 35 and 40 mmHg. A volume-cycled ventilator (Drager AV Ventilator, North American Drager, Telford, PA) was used to control and maintain ventilation by adjusting tidal volume up to 20 ml/kg and then respiratory rate, guided by intraoperative maternal arterial blood gas analysis. Minute ventilation was obtained from ventilator settings of tidal volume and respiratory rate. Circuit airway peak pressure was measured from the Bourdon gauge of the breathing circuit. Maternal $ETC0_2$, end-tidal halothane, and respiratory rate were recorded with a gas analyzer (Ohmeda 5250 respiratory gas monitor; Ohmeda, Louisville, KY).

A 7-French triple-lumen pulmonary artery catheter (Edwards Swan-Ganz catheter; Baxter Corp., Toronto, Ontario, Canada) was advanced through the catheter introducer and positioned in the pulmonary artery to measure cardiac output, pulmonary arterial wedge pressure, mean pulmonary arterial pressure, and central venous pressure. Correct placement was confirmed by observing characteristic pressure waveform. Maternal mean arterial pressure (MAP), pulmonary artery wedge pressure, mean pulmonary arterial pressure, central venous pressure, IAP, heart rate (HR), and fetal MAP and HR were recorded with a multichannel computer system (Hewlett-Packard M1092A, Sorrona, Italy). Cardiac output was determined using the thermodilution technique, using 10 ml room temperature 5% dextrose and a cardiac output computer (Gould Hemodynamic Profile Computer SP1445; Gould, Cardiovascular Products Division, Oxnard, CA). Three measurements were averaged and recorded. Blood samples were drawn from the fetus and the maternal carotid and pulmonary arteries to measure blood gases (Copenhagen Radiometer Acid-Base Laboratory, Copenhagen, Denmark).

The intraamniotic, fetal, and maternal lines were connected to a pressure transducer computer (Hewlett Packard M1092A) and the Transonic flow cable was connected to a computerized monitor (model T201; Transonic Systems).

Carbon dioxide pneumoperitoneum to a pressure of 15 mmHg was achieved by insufflating carbon dioxide through a Verres needle placed in the supraumbilical area using a standard procedure. An automatic laparoscopic insufflator (SOLOS Endoscopy, Atlanta, GA) was used to monitor the intraperitoneal pressure during insufflation. Baseline measurements were taken 30 min after anesthesia was induced. Data were collected at regular intervals during the 60-min insufflation period and during the first 30 min after desufflation.

Corrected fetal MAP was calculated by subtracting the IAP from the measured fetal MAP. Maternal and fetal heart rate (mHR, fHR), maternal MAP, and UBF were measured every 5 min. Central venous pressure, mean pulmonary artery pressure, Pulmonary artery wedge pressure, cardiac output, $ETC0_2$, maternal and fetal serum lactate, m-ABG, and f-ABG measurements were taken every 15 min. Respiratory rate, tidal volume, airway peak pressure, fresh gas flow, and IAP were measured every 20 min. All pressures and cardiac output

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Table 1. Maternal Blood Gas Data ETCO₂ and Serum Lactate (Mean ± SD) during Control (C) and Insufflation (I)

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>Pao₂ (mmHg)</th>
<th>Paco₂ (mmHg)</th>
<th>ETCO₂ (mmHg)</th>
<th>pH</th>
<th>Bicarbonate (mEq/L)</th>
<th>Lactate (mmol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>306 ± 67</td>
<td>335 ± 68</td>
<td>35 ± 3</td>
<td>29 ± 3</td>
<td>25 ± 4</td>
<td>7.42 ± 0.05</td>
</tr>
<tr>
<td>0</td>
<td>299 ± 74</td>
<td>206 ± 94*</td>
<td>35 ± 4</td>
<td>28 ± 4</td>
<td>25 ± 4</td>
<td>7.44 ± 0.06</td>
</tr>
<tr>
<td>15</td>
<td>278 ± 71</td>
<td>136 ± 49*</td>
<td>35 ± 2</td>
<td>28 ± 2</td>
<td>25 ± 4</td>
<td>7.44 ± 0.04</td>
</tr>
<tr>
<td>30</td>
<td>264 ± 83</td>
<td>135 ± 36*</td>
<td>35 ± 2</td>
<td>28 ± 2</td>
<td>25 ± 2*</td>
<td>7.44 ± 0.05</td>
</tr>
<tr>
<td>45</td>
<td>254 ± 70</td>
<td>149 ± 45*</td>
<td>35 ± 3</td>
<td>28 ± 2</td>
<td>23 ± 4*</td>
<td>7.44 ± 0.04</td>
</tr>
<tr>
<td>60</td>
<td>240 ± 77</td>
<td>152 ± 46*</td>
<td>36 ± 2</td>
<td>28 ± 2</td>
<td>22 ± 4</td>
<td>7.45 ± 0.05</td>
</tr>
<tr>
<td>75</td>
<td>244 ± 77</td>
<td>209 ± 96*</td>
<td>36 ± 3</td>
<td>29 ± 3</td>
<td>20 ± 2</td>
<td>7.44 ± 0.05</td>
</tr>
<tr>
<td>90</td>
<td>229 ± 74</td>
<td>226 ± 95</td>
<td>37 ± 3</td>
<td>31 ± 3</td>
<td>23 ± 2</td>
<td>7.44 ± 0.04</td>
</tr>
</tbody>
</table>

NM = not measured.
*P = 0.05.

Discussion:

The results of the study showed that there were significant changes in maternal arterial pH and lactate levels during insufflation compared to control conditions. Insufflation led to a significant decrease in maternal arterial pH and an increase in serum lactate levels. These findings are consistent with previous studies that have reported an increase in lactate production during insufflation, which may be due to increased myocardial oxygen demand. The decrease in arterial pH suggests that there was a decrease in maternal systemic oxygenation, which may be due to decreased maternal cardiac output or increased maternal systemic vascular resistance. These findings have important implications for the management of patients undergoing insufflation, as they may require additional monitoring and intervention to ensure maternal oxygenation and prevent maternal acidosis.
than baseline pressure, resulted during insufflation, whereas values remained constant in the control group (fig. 4).

No significant differences were found between insufflation and control groups with respect to MAP, HR, central venous pressure, pulmonary artery wedge pressure, mean mean pulmonary artery pressure, cardiac index, systemic vascular resistance, pulmonary vascular resistance, UBF, or venous admixture.

**Fetal Variables**

Fetal PAO2 remained within physiologically normal limits in both groups (table 2). Corrected fetal MAP to account for IAP changes was not different between groups (fig. 5). No significant differences in HR were noted between study groups, nor were there changes in acid-base status (table 2). There were no fetal deaths and no preterm labor was detected in any of the animals in this study.

**Discussion**

Fetal death, spontaneous abortion, and preterm labor have been reported in pregnant patients shortly after undergoing laparoscopic surgery. However, uncomplicated completion of pregnancy after laparoscopic surgery has also been reported.

The use of laparoscopic surgical procedures during pregnancy remains controversial. Potential risks include maternal and fetal acid-base disturbances and utero-placental perfusion alterations secondary to carbon dioxide pneumoperitoneum. Physiologic implications of pregnancy must be considered in addition to the known effects of IACO2 insufflation before considering the use of laparoscopic techniques in pregnant women.

Previous studies in pregnant animal models have shown maternal and fetal acidosis and tachycardia using ETCO2-guided ventilation. In our previous pilot study, maternal acidosis confounded the interpreta-

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tion of the fetal acidosi. In the present study, PaCO₂-guided ventilation was used to maintain maternal normocarbia and define the effects of carbon dioxide pneumoperitoneum on fetal acid-base status and maternal cardiopulmonary parameters, UBF and IAP changes.

Precise control of ventilation during the insufflation and early desufflation periods is required to avoid maternal hypercarbia and hypocarbia, respectively. During desufflation, ventilatory parameters must be regulated because hyperventilation can result in decreased UBF, and hypocarbia may increase maternal hemoglobin oxygen affinity with potential detrimental effects on fetal oxygenation.16,17

During carbon dioxide pneumoperitoneum, increased intraabdominal pressure can result in decreased diaphragmatic excursion, reduced pulmonary compliance, and increased deadspace ventilation.18 In addition, absorption of carbon dioxide from the peritoneal cavity19,20 can result in increased PaCO₂ unless appropriate ventilatory adjustments are made to eliminate the excess carbon dioxide and overcome the increased deadspace ventilation.21 The marked increase in the PaCO₂-ETCO₂ gradient observed in our study reflects the inadequacy of ETCO₂ to estimate PaCO₂ accurately.

Decreases in maternal oxygenation probably was a reflection of decreased functional residual capacity and increased ventilation/perfusion ratio mismatch. Despite the decrease in maternal oxygenation, the fetal PaO₂ remained within normal limits. The absence of fetal acidosi in this model would suggest that fetal acidosi reported in other studies10,22 has been due to the concurrent maternal respiratory acidosis resulting from ineffective ventilation.

As in other gravid animal studies,16,22,23§ no significant changes occurred in maternal hemodynamics during a 1-h insufflation period to an intraabdominal pressure of 15 mmHg in dorsal recumbency. Galan and associates8 reported increases in pulmonary artery wedge pressure, central venous pressure, and mean pulmonary artery pressure at 20 mmHg but not at 10 mmHg insufflation pressure using the gravid baboon model.

In nongravid patients, wide variations in hemodynamic responses to insufflation have been observed. Most studies report an increase in systemic vascular resistance, MAP, and right atrial pressure and a decrease in cardiac index insufflation pressure of study.16 Other changes in cardiac output may increase in MABP.

Increased intragastric pressure can increase chest pressure into the thoracic cavity and cause increased symptoms.

We noted no significant change in carbon dioxide partial pressure during insufflation. However, no changes in pressure, fetal HR, or Apgar score were noted.

We observed that the ICAO₂ insufflation gradient during breathing of PaCO₂ and CO₂. Additional studies are needed to identify pathophysiologic mechanisms involved.

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in cardiac index.\textsuperscript{24,25} Venous return is reduced when
insufflation pressures approach those used in our study.\textsuperscript{20} Other studies did not report a significant change in cardiac output, although there was an increase in MAP, central venous pressure, and HR.\textsuperscript{27}

Increased intrathoracic pressure also may develop in pregnant patients during insufflation because of decreased chest compliance as the diaphragm is pushed into the thorax. Although it is an inaccurate reflection of true intrathoracic pressure, circuit pressure was increased significantly in this study during insufflation.

We noted no changes in UBF. In a previous study,\textsuperscript{25} carbon dioxide pneumoperitoneum at an intrabdominal pressure of 20 mmHg resulted in pressure-dependent decreased perfusion of the maternal placenta. However, no changes were seen in maternal arterial pressure, fetal cardiopulmonary, or acid-base status.

We observed a significant increase in $\text{PaCO}_2$-ETCO$_2$ gradient during insufflation. A considerable underestimation of $\text{PaCO}_2$ can occur if ETCO$_2$ is used to monitor the adequacy of ventilation of pregnant patients undergoing IACO$_2$ insufflation. There were no changes in fetal MAP, HR, acid-base status, or serum lactate.

Additional studies investigating delayed fetal effects of IACO$_2$ insufflation, cardiopulmonary effects of patient positioning, and laparoscopic surgery should be done to identify pathophysiologic changes in pregnant women.

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Intestinal Gas and the Gut Barrier

M. M. Pearlman

Background:

achieve enteral feeding, although this remains to be systematized. Their role in the immediate postoperative period is uncertain. We examined the effects of enteral feeding using different opioids.

Methods:

phine, chlorpromazine, and haloperidol; and that had received no opioid, chlorpromazine, or haloperidol. Response was defined as the time to the first dose of morphine in the group that had received morphine in addition to chlorpromazine or haloperidol. Data were analyzed using a fixed-factor repeated measures analysis of variance.

Results:

in a dose-response manner with a minimum level of r = 1.0 and complete inhibition at r = 2.0 doses or the highest dose analyzed for each opioid. The effect of each opioid (dose ratio) was conditionally independent, suggesting a common mechanism of action.

Conclusions:

clude several factors that affect opioid potencies. Isolated investigations of mechanisms of action in the context of the combined effects of opioids.

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