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Carbon dioxide absorption and gas exchange during pelvic laparoscopy

Twelve ASA physical status I–II patients undergoing pelvic laparoscopy for infertility were enrolled in a study to quantify the effects of CO₂ insufflation and the Trendelenburg position on CO₂ elimination and pulmonary gas exchange, and to determine the minute ventilation required to maintain normocapnia during CO₂ insufflation. Measurements of O₂ uptake ($\dot{V}O_2$), CO₂ elimination ($\dot{V}CO_2$), minute ventilation (VE), FIO₂, and respiratory exchange ratio (RQ) were made during three steady states: control (C) taken after 15 min of normoventilation but before CO₂ insufflation, after 15 min (L₁) and 30 min (L₂) of hyperventilation during CO₂ insufflation. The FIO₂ was controlled at 0.5 and arterial blood gases were used to calculate the oxygen tension-based indices of pulmonary gas exchange. After 15 min and 30 min of CO₂ insufflation, the volume of CO₂ absorbed from the peritoneal cavity was estimated at 42.1 ± 5.1 and 38.6 ± 6.6 (SEM) ml · min⁻¹ respectively, increasing CO₂ elimination through the lungs by about 30%. Hyperventilation of the lungs by a 20–30% increase in minute ventilation maintained normocapnia. Despite the CO₂ pneumoperitoneum and Trendelenburg position, there was no impairment of pulmonary oxygen exchange as estimated by (A–a)DO₂. This study demonstrated that a 30% increase in minute ventilation, achieved by increasing tidal volume to more than 10 ml · kg⁻¹, is sufficient to eliminate the increased CO₂ load and maintain normal pulmonary O₂ exchange during pelvic laparoscopy.

Key words

ANAESTHESIA: diagnostic;
CARBON DIOXIDE: absorption, elimination, respiratory quotient;
SURGERY: laparoscopy;
VENTILATION: alveolar, tidal volume.

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Douze patientes de la classe ASA I ou II subissant une laparoscopie pelvienne pour infertilité ont été incluses dans une étude qui visait d'une part, à quantifier les effets conjoints de l'insufflation du CO₂ et de la position de Trendelenburg sur l'élimination du CO₂ ainsi que sur les échanges gazeux pulmonaires, et d'autre part, de déterminer la ventilation requise pour maintenir une normocapnie pendant l'insufflation. Trois moments d'équilibre ont été déterminés pour faire des mesures de captation d'O₂ ($\dot{V}O_2$), d'élimination de CO₂ ($\dot{V}CO_2$), de ventilation minute (VE), de FIO₂ et de quotient respiratoire (RQ). Ces moments sont : 15 minutes après une ventilation normale et précédant l'insufflation de CO₂ (C) ; 15 minutes (L₁) et 30 minutes (L₂) d'hyperventilation accompagnant l'insufflation du CO₂. La FIO₂ maintenue à 0,5 et la mesure des gaz artériels ont permis de calculer les échanges pulmonaires dépendant des pressions partielles en O₂. Quinze et 30 minutes après l'insufflation du CO₂, la quantité de CO₂ absorbée par la cavité péritonéale a été évaluée successivement à 42,1 ± 5,1 ml · min⁻¹ et à 38,6 ± 6,6 (SEM) ml · min⁻¹, entraînant une augmentation d'élimination pulmonaire de 30%. Une augmentation de ventilation minute de 20 à 30% a permis de maintenir une normocapnie. Malgré la présence de CO₂ dans le péritoine et la position de Trendelenburg, il n'y a pas eu d'altération des échanges pulmonaires en O₂ estimés par la DO₂ (A–a). Cette étude prouve qu'une augmentation de ventilation-minute de 30%, obtenue par l'augmentation du volume courant à plus de 10 ml · kg⁻¹ suffit pour éliminer le surplus de CO₂ et maintenir des échanges respiratoires normaux en O₂ pendant une laparoscopie pelvienne.

Laparoscopy for visualisation of the pelvic organs was described in the early 20th century¹ but its usefulness in gynaecological surgery was first recognized by Steptoe in 1964.² Today, laparoscopy has moved beyond the realm of pelvic surgery to include abdominal surgery (e.g., laparoscopic cholecystectomy, appendectomy and vagotomy).

Laparoscopy for pelvic surgery introduces three major physiological alterations:³

1 Trendelenburg position: usually with a 30° head-down tilt to allow better visualisation of the pelvic viscera.

The Trendelenburg position causes cardio-vascular and gas exchange impairment.

- 2 CO₂ pneumoperitoneum: this further increases abdominal pressure and exaggerates the \dot{V}_A/\dot{Q} imbalance due to the Trendelenburg posture.
- 3 CO₂ absorption: CO₂ is absorbed transperitoneally and the combination of hypercapnia and alveolar hypoventilation can lead to cardiac arrhythmias and even cardiac arrest.⁴

Desmond and Gordon⁵ demonstrated the superiority of controlled hyperventilation over spontaneous respiration or controlled normoventilation for maintaining normal PCO₂. However, they did not quantify the amount of CO₂ absorbed or the increase in ventilation needed to maintain normocapnia.

This clinical study was undertaken to quantify the CO₂ absorption and the effects on gas exchange in patients undergoing laparoscopy with CO₂ insufflation in the Trendelenburg position. We also determined the increase in minute ventilation necessary to maintain normocapnia during CO₂ insufflation.

Methods

The protocol was approved by the Departmental and Institutional Ethics Review Committee. Verbal consent was given by the patients for the study and for cannulation of the radial artery.

The study sample was 12 young women (age 33.5 ± 4.8 yr, weight 57.7 ± 10.2 kg) of ASA physical status I and II undergoing laparoscopic examination for infertility under general endotracheal anaesthesia. Routine pre-anaesthetic work-up included history, physical examination and routine full blood count. Patients with a history of recent pulmonary disease, abnormal physical findings or who smoked were excluded from the study.

Most patients received midazolam premedication. Those who were not premedicated were day surgery cases. Total intravenous anaesthesia was used which was induced with propofol $2 \text{ mg} \cdot \text{kg}^{-1}$; fentanyl $2\text{--}3 \text{ } \mu\text{g} \cdot \text{kg}^{-1}$ and succinylcholine $1\text{--}2 \text{ mg} \cdot \text{kg}^{-1}$ was used to facilitate tracheal intubation. Anaesthesia was maintained with a continuous propofol infusion ($6\text{--}10 \text{ mg} \cdot \text{kg}^{-1} \cdot \text{hr}^{-1}$) and intermittent doses of fentanyl. Muscle paralysis was achieved with atracurium $0.3 \text{ mg} \cdot \text{kg}^{-1}$ and controlled intermittent positive pressure ventilation (IPPV) with a Siemens 900C servo ventilation at a rate of ten per minute in the volume-controlled mode. The anaesthetic circuit was a non-rebreathing system with a mixture of air/O₂ with a FiO₂ of 0.5.

All patients were placed in the supine Trendelenburg tilt with CO₂ insufflated through a trocar passed into the peritoneal cavity. Prior to CO₂ insufflation patients' lungs were ventilated with a minute volume of about $80 \text{ ml} \cdot \text{kg}^{-1}$ (tidal volume set at $8 \text{ ml} \cdot \text{kg}^{-1}$, respiratory rate of ten per

minute), adjusted to maintain an end-tidal CO₂ (FETCO₂) of 5%. During CO₂ insufflation, the minute volume was increased to at least $100 \text{ ml} \cdot \text{kg}^{-1}$ (tidal volume increased to $10 \text{ ml} \cdot \text{kg}^{-1}$ with a respiratory rate kept at ten per minute) to maintain FETCO₂ constant. Conventional ratio ventilation was used with inspiratory time of 2.5 sec, and an inspiratory pause of 1 sec.

There were three study periods. The first set of measurements control (C) was taken 15 min after IPPV was started and before CO₂ insufflation. The second (L₁) and third (L₂) sets were taken 15 and 30 min after beginning CO₂ insufflation.

Airway pressures were recorded from the ventilator and FETCO₂ from a Siemens CO₂ analyzer Model 930. A Datex Deltatrac Metabolic Monitor was used to measure O₂ uptake (\dot{V}_{O_2}), CO₂ elimination (\dot{V}_{CO_2}), minute ventilation (VE), FiO₂ and respiratory exchange ratio (RQ). Metabolic measurements were recorded at one-minute intervals and the final five measurements during each study period were averaged. At the end of each study period, an arterial blood sample was drawn from the indwelling radial artery cannula and analyzed immediately in a Nova Stat Profile Blood Gas analyzer. Heart rate (HR) was recorded from the ECG monitor and blood pressure (BP) by an automatic BP monitor (Dinamap) at three-minute intervals. All measuring instruments were properly calibrated. The O₂ and the CO₂ analyzer of the metabolic monitor were calibrated with medical grade calibration gas containing 5% CO₂ and 95% O₂ before each study.

Pulmonary gas exchange was assessed by calculating the alveolar-arterial O₂ tension gradient (A-a)DO₂ based on FiO₂, temperature corrected PaO₂ and calculated PaO₂.

The alveolar O₂ tension was calculated by the formula adopted by Pappenheimer *et al.*⁶

$$PAO_2 = FiO_2(PB - Pa_{H_2O}) - PaCO_2 \left[FiO_2 + \frac{1 - FiO_2}{RQ} \right]$$

Since Singapore is at sea level, the barometric pressure (PB) was assumed to be 760 mmHg and alveolar H₂O vapour pressure to be 47 mmHg. Day to day variation in barometric pressure in Singapore is less than 1% according to the Meteorological Service of Singapore.

The metabolic CO₂ production was calculated from the measured \dot{V}_{O_2} by assuming that the ratio between metabolic carbon dioxide production and oxygen consumption remained constant and equal to the control RQ.

$$RQ (\text{Control-C}) \times \dot{V}_{O_2} = \text{metabolic } \dot{V}_{CO_2}$$

Hence the amount of CO₂ absorbed per min = measured \dot{V}_{CO_2} - metabolic \dot{V}_{CO_2} , and the percentage increase in absorbed CO₂ = absorbed \dot{V}_{CO_2} /metabolic \dot{V}_{CO_2} .

The alveolar CO₂ fraction, FACO₂ was calculated as follows:

The model for CO₂ removal from the body describes

TABLE I Conventional ratio ventilation: Mean \pm SEM (*n*). Different study conditions with resultant haemodynamic, arterial blood gas analyses and tidal volume variables.

	C (12)	L ₁ (12)	L ₂ (4)
Tidal volume VT ml · kg ⁻¹	8.6 (\pm 0.3)	10.4 (\pm 0.2)*	10.8 (\pm 0.2)*
Temperature °C	36.4 (\pm 0.1)	36.2 (\pm 0.1)	36.1 (\pm 0.1)
Heart rate	77.9 (\pm 3.5)	74.7 (\pm 3.2)	70.1 (\pm 3.4)
Systolic BP mmHg	125.9 (\pm 4.1)	132.3 (\pm 3.3)	130.3 (\pm 4.5)
Diastolic BP mmHg	71.1 (\pm 2.5)	74.6 (\pm 2.2)	73.3 (\pm 5.2)
pH	7.34 (\pm 0.01)	7.34 (\pm 0.01)	7.34 (\pm 0.02)
PaCO ₂ mmHg	42.3 (\pm 1.0)	42.1 (\pm 1.1)	43.0 (\pm 1.0)
PaO ₂ mmHg	227.0 (\pm 19.6)	236.4 (\pm 14.1)	241.4 (\pm 22.6)
Base excess	-2.8 (\pm 0.6)	-3.0 (\pm 0.6)	-3.5 (\pm 1.0)

**P* < 0.01 compared with C.TABLE II Pulmonary ventilation and gas exchange (mean \pm SEM). Different study conditions with resultant pulmonary ventilation and gas exchange variables. Metabolic CO₂ and absorbed CO₂ – see text for further details.

	C	L ₁	L ₂
$\dot{V}CO_2$ ml · min ⁻¹	146.0 (\pm 5.9)	183.3 (\pm 5.0)*	172.0 (\pm 6.2)*
VO ₂ ml · min ⁻¹	168.9 (\pm 3.8)	164.1 (\pm 5.3)	153.8 (\pm 10.3)
RQ	0.86 (\pm 0.2)	1.12 (\pm 0.2)*	1.13 (\pm 0.04)*
$\dot{V}A$ ml · kg ⁻¹	3.12 (\pm 0.13)	4.05 (\pm 0.19)*	3.68 (\pm 0.13)*
VD/VT	0.32 (\pm 0.2)	0.31 (\pm 0.1)	0.32 (\pm 0.02)
(A-a) DO ₂ mmHg	72.9 (\pm 16.1)	72.0 (\pm 11.9)	73.2 (\pm 21.2)
Metabolic CO ₂	141.1 (\pm 5.2)	141.2 (\pm 6.6)	133.3 (\pm 9.9)
Absorbed CO ₂	-	42.1 (\pm 5.1)	38.6 (\pm 6.6)
% \uparrow CO ₂ absorbed	-	30%	29%
% \uparrow (VA)	-	30%	28%

**P* < 0.01 compared with C.

$\dot{V}O_2$ as a function of alveolar ventilation (VA) and alveolar CO₂ concentration (FACO₂):

$$\dot{V}CO_2 = VA \cdot FACO_2 \quad (\text{Eq. 1})^7$$

The alveolar CO₂ concentration can be estimated to be equal to the arterial CO₂ concentration and the equation written as:

$$\dot{V}CO_2 = k_2 \cdot VA \cdot PaCO_2 \quad (\text{Eq. 2})^8$$

where *k*₂ is a constant that converts arterial CO₂ partial pressure (PaCO₂) to CO₂ concentration and the $\dot{V}CO_2$ to standard pressure (760 mmHg) dry gas. When VA is given in L · min⁻¹, body temperature (37°C) and fully saturated with water vapour, and PaCO₂ in kPa, *k*₂ = 8.16.

From equation 1 and 2 combined,

$$VA \cdot FACO_2 = k_2 \cdot VA \cdot PaCO_2$$

Therefore

$$FACO_2 = k_2 \cdot PaCO_2$$

$$\% FACO_2 = \frac{PaCO_2}{7.5} \times \frac{8.16}{1000}$$

as PaCO₂ is expressed in kPa when

$$k_2 = 8.16$$

and FaCO₂ is expressed as a percentage fraction.

Effective alveolar ventilation (VA, BTPS) was then calculated as

$$VA = \dot{V}CO_2 / FACO_2, L \cdot \text{min}^{-1} \text{ (BTPS)}$$

Physiological dead space

$$VD = VE - VA \text{ (BTPS)}$$

Physiological dead space to tidal volume ratio:

$$\frac{VD}{VT} = 1 - \frac{VA}{VE}$$

Measured and calculated variables from the three different study periods were compared by ANOVA. Two sample comparisons were by non-parametric tests: Wilcoxon signed rank test for paired data and the Mann-Whitney U test for unpaired data.

Results

The vital signs (BP and HR), temperature, and arterial blood gases were stable throughout with no significant changes from the base-line values (Table I).

Gas exchange and ventilatory data for the three measurement periods is presented in Table II. During the control (C) measurement, tidal volume was 8.6 ± 0.3

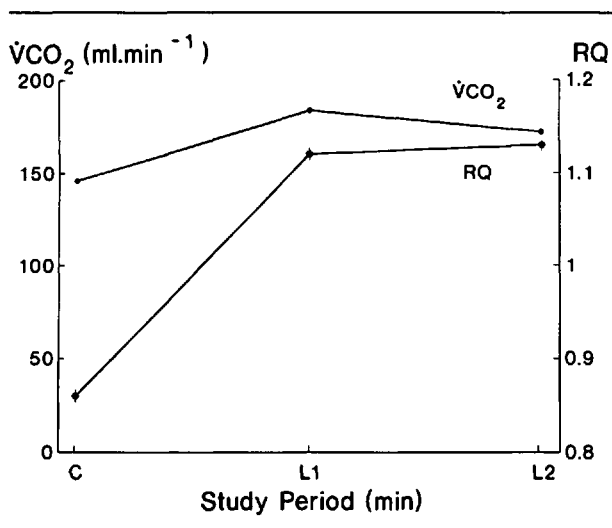


FIGURE Effects of time on $\dot{V}CO_2$ and RQ.

ml · kg⁻¹ and was increased to 10.4 ± 0.2 and 10.8 ± 0.2 ml · kg⁻¹ at L₁ and L₂, during CO₂ insufflation. Respiratory rate was held constant. Both $\dot{V}CO_2$ and RQ were significantly increased at L₁ and L₂, reflecting an increased CO₂ load presented to the lungs due to CO₂ absorption from the peritoneal cavity. The calculated rate of absorption of CO₂ at L₁ and L₂ was 42.1 ± 5.1 and 38.7 ± 6.6 ml · min⁻¹ respectively. Arterial PaCO₂ was maintained constant by an increase in effective alveolar ventilation estimated as 30 and 18% during L₁ and L₂ respectively.

There was no significant difference in gas exchange as assessed by the (A-a) DO₂ between the C and the study periods L₁ and L₂ in spite of the CO₂ pneumoperitoneum and the combined Trendelenburg lithotomy tilts (Table II). The physiological dead space to tidal volume ratio (VD/VT) also remained constant.

The effect of time on $\dot{V}CO_2$ and RQ are seen in the Figure. There was an increase in both $\dot{V}CO_2$ and RQ between C and L₁ ($P < 0.001$) but no further change between L₁ and L₂.

Discussion

Desmond and Gordon⁵ studied the effects of different ventilatory modes during laparoscopy, comparing spontaneous respiration with controlled normoventilation and with hyperventilation. They used a general anaesthetic with nitrous oxide and halothane for the spontaneously breathing group and added curare for controlled ventilation. Arterial blood was sampled and expired gases collected in a Douglas bag before and after the insufflation of CO₂. They concluded that spontaneous respiration was dangerous because the large pneumoperitoneum with the steep Trendelenburg tilt and splinted diaphragm led to hypoventilation and hypercapnia and advocated routine

hyperventilation to remove the excess CO₂. However, they did not quantify the excess CO₂ load nor the level of ventilation required to maintain normocapnia.

Our study was done using a total intravenous anaesthetic technique and an air-O₂ mixture so as to eliminate the effect of nitrous oxide on the infra-red CO₂ sensor in the Datex Metabolic Monitor. Inhalational agents were also omitted as they can affect the flow measurement. The Servo 900C ventilator completely separates the inspiratory and expiratory gases, and is necessary for accurate collection of the expired gases. The performance of the Datex Deltatrac metabolic monitor has been validated recently.⁹

We demonstrated a 30% increase in CO₂ load due to CO₂ absorption from the peritoneal cavity. This required a 30% increase in alveolar ventilation to maintain normocapnia. The volume of CO₂ absorbed appeared to reach a plateau and there was no further increase between 15 and 30 min. Rapid uptake of CO₂ is due to the highly diffusible nature of CO₂, but other factors such as the splanchnic circulation, the concentration gradient between the peritoneal cavity and the venous blood, and the amount of venous shunting in the splanchnic vascular bed also play a role. $\dot{V}A/\dot{Q}$ shunting in the lung has less effect on CO₂ than on the less soluble gases like N₂O and O₂, so that $\dot{V}A/\dot{Q}$ mismatching in the lungs during anaesthesia would have less effect on PaCO₂.

The plateau in the CO₂ elimination curve between 15 and 30 min, in conjunction with constant levels of PaCO₂, suggests that the excess CO₂ absorbed from the peritoneal cavity had reached equilibrium with that removed by the increase in alveolar ventilation.

It has been shown that the well-known impairment gas exchange during anaesthesia is, to a large extent, attributable to early formation of atelectasis which in turn produces shunt.¹⁰ This impairment of gas exchange is much improved with Continuous Positive Pressure Ventilation¹¹ and Positive End Expiratory Pressure.¹² In the present study we expected that the increased intra-abdominal pressure due to the CO₂ peritoneum and Trendelenburg tilt would worsen the cephalad shift of the diaphragm and the resultant compression atelectasis and pulmonary venous shunting caused by anaesthesia. However, we found no significant deterioration of pulmonary O₂ exchange after CO₂ insufflation and the Trendelenburg-lithotomy tilt. The reasons, we believe, are that the patients were young, non-obese and healthy, and that high tidal volume ventilation (there was a 30% increase in VT) had a protective effect.

In conclusion, this study demonstrated that during pelvic laparoscopy there was a rapid rise of about 30% in the CO₂ load eliminated by the lungs. This quickly reached a plateau and could be compensated for by hyperventilation of the lungs with a 30% increase in minute ventilation.

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