Carbon dioxide absorption and gas exchange during pelvic laparoscopy

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Twelve ASA physical status I–II patients undergoing pelvic laparoscopy for infertility were enrolled in a study to quantify the effects of CO_2 insufflation and the Trendelenburg position on CO_2 elimination and pulmonary gas exchange, and to determine the minute ventilation required to maintain normocapnia during CO_2 insufflation. Measurements of O_2 uptake ($\dot{V}O_2$), CO_2 elimination ($\dot{V}CO_2$), minute ventilation (VE), FIO_2, and respiratory exchange ratio (RQ) were made during three steady states: control (C) taken after 15 min of normoventilation but before CO_2 insufflation, after 15 min (L_1) and 30 min (L_2) of hyperventilation during CO_2 insufflation. The FIO_2 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 CO_2 insufflation, the volume of CO_2 absorbed from the peritoneal cavity was estimated at 42.1 ± 5.1 and 38.6 ± 6.6 (SEM) $ml \cdot min^{-1}$ respectively, increasing CO_2 elimination through the lungs by about 30%. Hyperventilation of the lungs by a 20–30% increase in minute ventilation maintained normocapnia. Despite the CO_2 pneumoperitoneum and Trendelenburg position, there was no impairment of pulmonary oxygen exchange as estimated by $(A–a)DO_2$. This study demonstrated that a 30% increase in minute ventilation, achieved by increasing tidal volume to more than 10 $ml \cdot kg^{-1}$, is sufficient to eliminate the increased CO_2 load and maintain normal pulmonary O_2 exchange during pelvic laparoscopy.

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 let effets conjoints de l'insufflation du CO_2 et de la position de Trendelenbourg sur l'élimination du CO_2 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'O_2 ($\dot{V}O_2$), d'élimination de CO_2 ($\dot{V}CO_2$), de ventilation minute (VE), de FIO_2 et de quotient respiratoire (RQ). Ces moments sont : 15 minutes après une ventilation normale et précédant l'insuffiation de CO_2 (C) ; 15 minutes (L_1) et 30 minutes (L_2) d'hyperventilation accompagnant l'insufflation du CO_2. La FIO_2 maintenue à 0,5 et la mesure des gaz artériels ont permis de calculer les échanges pulmonaires dépendant des pressions partielles en O_2. Quinze et 30 minutes après l'insufflation du CO_2, la quantité de CO_2 absorbée par la cavité péritonéale a été évaluée successivement à 42,1 ± 5,1 $ml \cdot min^{-1}$ et à 38,6 ± 6,6 (SEM) $ml \cdot min^{-1}$, 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 CO_2 dans le péritoine et la position de Trendelenbourg, il n'y a pas eu d'altération des échanges pulmonaires en O_2 estimés par la DO_2 (A–a). Cette étude prouve qu'une augmentation de ventilation-minute de 30%, obtenue par l'augmentation du volume courant à plus de 10 $ml \cdot kg^{-1}$ suffit pour éliminer le surplus de CO_2 et maintenir des échanges respiratoires normaux en O_2 pendant une laparoscopie pelvienne.

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

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Laparoscopy for visualisation of the pelvic organs was described in the early 20th century[1] 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, appendicectomy 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 CO_2 pneumoperitoneum: this further increases abdominal pressure and exaggerates the $\dot{V}A/\dot{Q}$ imbalance due to the Trendelenburg posture.

3 CO_2 absorption: CO_2 is absorbed transperitoneally and the combination of hypercapnia and alveolar hypoventilation can lead to cardiac arrhythmias and even cardiac arrest.[4]

Desmond and Gordon[5] demonstrated the superiority of controlled hyperventilation over spontaneous respiration or controlled normoventilation for maintaining normal PCO_2. However, they did not quantify the amount of CO_2 absorbed or the increase in ventilation needed to maintain normocapnia.

This clinical study was undertaken to quantify the CO_2 absorption and the effects on gas exchange in patients undergoing laparoscopy with CO_2 insufflation in the Trendelenburg position. We also determined the increase in minute ventilation necessary to maintain normocapnia during CO_2 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 $mg \cdot kg^{-1}$; fentanyl 2–3 $\mu g \cdot kg^{-1}$ and succinylcholine 1–2 $mg \cdot kg^{-1}$ was used to facilitate tracheal intubation. Anaesthesia was maintained with a continuous propofol infusion (6–10 $mg \cdot kg^{-1} \cdot hr^{-1}$) and intermittent doses of fentanyl. Muscle paralysis was achieved with atracurium 0.3 $mg \cdot kg^{-1}$ 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/O_2 with a FIO_2 of 0.5.

All patients were placed in the supine Trendelenburg tilt with CO_2 insufflated through a trocar passed into the peritoneal cavity. Prior to CO_2 insufflation patients' lungs were ventilated with a minute volume of about 80 $ml \cdot kg^{-1}$ (tidal volume set at 8 $ml \cdot kg^{-1}$, respiratory rate of ten per minute), adjusted to maintain an end-tidal CO_2 ($FETCO_2$) of 5%. During CO_2 insufflation, the minute volume was increased to at least 100 $ml \cdot kg^{-1}$ (tidal volume increased to 10 $ml \cdot kg^{-1}$ with a respiratory rate kept at ten per minute) to maintain $FETCO_2$ 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 CO_2 insufflation. The second (L_1) and third (L_2) sets were taken 15 and 30 min after beginning CO_2 insufflation.

Airway pressures were recorded from the ventilator and $FETCO_2$ from a Siemens CO_2 analyzer Model 930. A Datex Deltatrac Metabolic Monitor was used to measure O_2 uptake ($\dot{V}O_2$), CO_2 elimination ($\dot{V}CO_2$), minute ventilation ($\dot{V}E$), FIO_2 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 O_2 and the CO_2 analyzer of the metabolic monitor were calibrated with medical grade calibration gas containing 5% CO_2 and 95% O_2 before each study.

Pulmonary gas exchange was assessed by calculating the alveolar-arterial O_2 tension gradient $(A–a)DO_2$ based on FIO_2, temperature corrected PaO_2 and calculated PaO_2.

The alveolar O_2 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 H_2O 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 CO_2 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.

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

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

The alveolar CO_2 fraction, $FACO_2$ was calculated as follows:

The model for CO_2 removal from the body describes

TABLE I Conventional ratio ventilation: Mean ± 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 (± 0.3)	10.4 (± 0.2)*	10.8 (± 0.2)*
Temperature °C	36.4 (± 0.1)	36.2 (± 0.1)	36.1 (± 0.1)
Heart rate	77.9 (± 3.5)	74.7 (± 3.2)	70.1 (± 3.4)
Systolic BP mmHg	125.9 (± 4.1)	132.3 (± 3.3)	130.3 (± 4.5)
Diastolic BP mmHg	71.1 (± 2.5)	74.6 (± 2.2)	73.3 (± 5.2)
pH	7.34 (± 0.01)	7.34 (± 0.01)	7.34 (± 0.02)
$PaCO_2$ mmHg	42.3 (± 1.0)	42.1 (± 1.1)	43.0 (± 1.0)
PaO_2 mmHg	227.0 (± 19.6)	236.4 (± 14.1)	241.4 (± 22.6)
Base excess	−2.8 (± 0.6)	−3.0 (± 0.6)	−3.5 (± 1.0)

*$P < 0.01$ compared with C.

TABLE II Pulmonary ventilation and gas exchange (mean ± SEM). Different study conditions with resultant pulmonary ventilation and gas exchange variables. Metabolic CO_2 and absorbed CO_2 – see text for further details.

	C	L_1	L_2
$\dot{V}CO_2$ ml · min^{-1}	146.0 (± 5.9)	183.3 (± 5.0)*	172.0 (± 6.2)*
$\dot{V}O_2$ ml · min^{-1}	168.9 (± 3.8)	164.1 (± 5.3)	153.8 (± 10.3)
RQ	0.86 (± 0.2)	1.12 (± 0.2)*	1.13 (± 0.04)*
VA ml · kg^{-1}	3.12 (± 0.13)	4.05 (± 0.19)*	3.68 (± 0.13)*
VD/VT	0.32 (± 0.2)	0.31 (± 0.1)	0.32 (± 0.02)
(A–a) DO_2 mmHg	72.9 (± 16.1)	72.0 (± 11.9)	73.2 (± 21.2)
Metabolic CO_2	141.1 (± 5.2)	141.2 (± 6.6)	133.3 (± 9.9)
Absorbed CO_2	–	42.1 (± 5.1)	38.6 (± 6.6)
% ↑ CO_2 absorbed	–	30%	29%
% ↑ (VA)	–	30%	28%

*$P < 0.01$ compared with C.

$\dot{V}O_2$ as a function of alveolar ventilation (VA) and alveolar CO_2 concentration ($FACO_2$):

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

The alveolar CO_2 concentration can be estimated to be equal to the arterial CO_2 concentration and the equation written as:

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

where k_2 is a constant that converts arterial CO_2 partial pressure ($PaCO_2$) to CO_2 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 $PaCO_2$ 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 $PaCO_2$ is expressed in kPa when

$k_2 = 8.16$

and $FaCO_2$ is expressed as a percentage fraction.

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

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

Physiological dead space

$$VD = VE - VA\ (BTPS)$$

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

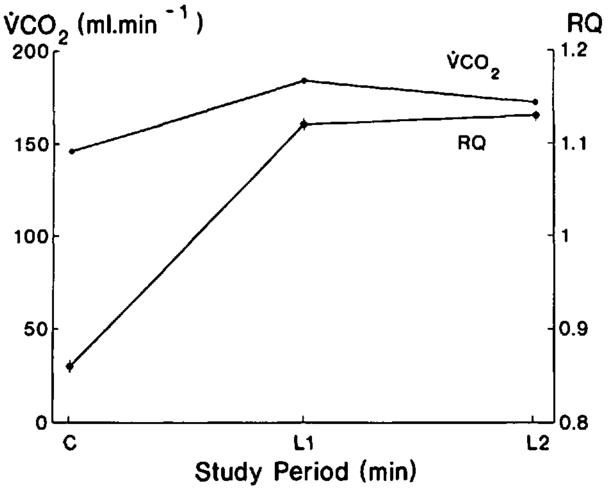

FIGURE Effects of time on $\dot{V}CO_2$ and RQ.

$ml \cdot kg^{-1}$ and was increased to 10.4 ± 0.2 and 10.8 ± 0.2 $ml \cdot kg^{-1}$ at L_1 and L_2, during CO_2 insufflation. Respiratory rate was held constant. Both $\dot{V}CO_2$ and RQ were significantly increased at L_1 and L_2, reflecting an increased CO_2 load presented to the lungs due to CO_2 absorption from the peritoneal cavity. The calculated rate of absorption of CO_2 at L_1 and L_2 was 42.1 ± 5.1 and 38.7 ± 6.6 $ml \cdot min^{-1}$ respectively. Arterial $PaCO_2$ was maintained constant by an increase in effective alveolar ventilation estimated as 30 and 18% during L_1 and L_2 respectively.

There was no significant difference in gas exchange as assessed by the (A–a) DO_2 between the C and the study periods L_1 and L_2 in spite of the CO_2 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 L_1 ($P < 0.001$) but no further change between L_1 and L_2.

Discussion

Desmond and Gordon[5] 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 CO_2. 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 CO_2. However, they did not quantify the excess CO_2 load nor the level of ventilation required to maintain normocapnia.

Our study was done using a total intravenous anaesthetic technique and an air-O_2 mixture so as to eliminate the effect of nitrous oxide on the infra-red CO_2 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 CO_2 load due to CO_2 absorption from the peritoneal cavity. This required a 30% increase in alveolar ventilation to maintain normocapnia. The volume of CO_2 absorbed appeared to reach a plateau and there was no further increase between 15 and 30 min. Rapid uptake of CO_2 is due to the highly diffusible nature of CO_2, 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 CO_2 than on the less soluble gases like N_2O and O_2, so that $\dot{V}A/\dot{Q}$ mismatching in the lungs during anaesthesia would have less effect on $PaCO_2$.

The plateau in the CO_2 elimination curve between 15 and 30 min, in conjunction with constant levels of $PaCO_2$, suggests that the excess CO_2 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 Ventilation[11] and Positive End Expiratory Pressure.[12] In the present study we expected that the increased intra-abdominal pressure due to the CO_2 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 O_2 exchange after CO_2 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 CO_2 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.

Acknowledgements

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