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Ventilatory effects of laparoscopic cholecystectomy under general anesthesia

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Abstract

Purpose. To investigate the ventilatory effect of laparoscopic cholecystectomy in patients under general anesthesia with epidural block.

Methods. We measured arterial blood gas, pulmonary carbon dioxide elimination ($\dot{V}ECO_2$), the dead space/tidal volume ratio (VD/VT), and the alveolar-arterial PO_2 difference [(A-a)DO_2] just before and 5, 10, 20, 40, and 80 min after peritoneal insufflation in eight patients who underwent laparoscopic cholecystectomy under general anesthesia with epidural block. The effect of laparoscopic cholecystectomy on these values was evaluated. The patients were ventilated on the controlled mode by Servo 900C with a constant tidal volume (VT 10 ml·kg^{-1}) and frequency (respiratory rate 12 breaths·min^{-1}) throughout the study.

Results. After starting peritoneal insufflation the $PaCO_2$ showed a sudden increase during the initial 10 min of about 4 mmHg followed by a gradual increase thereafter. The increase in $\dot{V}ECO_2$ was about 30 ml·min^{-1} (20%) on average during the initial 20 min, and a plateau was reached within 20–40 min after peritoneal insufflation. Neither VD/VT nor (A-a)DO_2 showed significant changes during the study.

Conclusion. These results suggest that (1) transperitoneal absorption of CO_2 may be the main cause of hypercarbia, and the hypercarbia is not attributed to the increase in VD/VT; and (2) oxygenation is not impaired during pneumoperitoneum.

Key words: Laparoscopic cholecystectomy, Peritoneal insufflation, Pulmonary carbon dioxide elimination, Deadspace/tidal volume ratio, Alveolar-arterial PO_2 difference

Introduction

Laparoscopic cholecystectomy has been accepted as a relatively noninvasive surgical procedure. It affords a shorter hospital stay, faster recovery to daily life, and lower medical expenses, as well as smaller surgical incisions and less postoperative pain.

Taking anesthesiology management into consideration, however, laparoscopic cholecystectomy may not always be "noninvasive" compared with traditional open cholecystectomy. Subcutaneous emphysema [1,2], pneumothorax [3,4], air embolism [5], hypoxemia [6], and pulmonary atelectasis sometimes occur, and hypercarbia during pneumoperitoneum may be a relatively common complication when carbon dioxide (CO_2) is used for peritoneal insufflation.

During gynecologic laparoscopy, it has been generally accepted that hypercarbia is the result of peritoneal CO_2 absorption and mechanical impairment of ventilation caused by the Trendelenburg tilt and pneumoperitoneum [7]. Diaphragmatic elevation resulting from insufflation of CO_2 decreases functional residual capacity, which may increase the ventilation/perfusion (V/Q) mismatch. There is also a report that the dead space/tidal volume ratio (VD/VT) slightly increases during pneumoperitoneum [8], and this increase in VD/VT might impair pulmonary CO_2 elimination ($\dot{V}ECO_2$) and promote hypercarbia. The ventilatory effect of laparoscopic cholecystectomy has not been sufficiently examined.

In the present study we measured arterial blood gas, $\dot{V}ECO_2$, VD/VT, and the alveolar-arterial PO_2 difference [(A-a)DO_2] under constant ventilation in patients undergoing laparoscopic cholecystectomy. The effects of peritoneal insufflation on pulmonary gas exchange have been rarely studied during laparoscopic cholecystectomy [9]. The objective of this study was to evaluate the effects of pneumoperitoneum with CO_2 on these values in patients under general anesthesia with epidural block.

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Materials and methods

Patients scheduled to undergo laparoscopic cholecystectomy with American Society of Anesthesiologists (ASA) physical status 1 or 2 without evidence of circulatory or respiratory disease were included in this study. There were four men and four women with a mean age of 51.0 ± 8.8 (SD) years and a mean body weight of 59.1 ± 7.7 kg. Neither preoperative spirometry nor arterial blood gas analysis showed abnormal values in these patients. The study was approved by the university's ethical committee.

Preoperative medication with 50 mg of hydroxyzine and 0.5 mg of atropine was administered intramuscularly 30 min before transfer to the operating room. Epidural anesthesia was performed in the T12–L1 interspace using a midline approach with the patient in the lateral position. An epidural catheter was inserted 3–5 cm in a cephalad direction, and 8–12 ml of 1% lidocaine was injected after a test dose of 2 ml of 1% lidocaine. General anesthesia was induced with intravenous thiamylal 5 mg·kg^{-1}, and endotracheal intubation was facilitated with intravenous vecuronium 0.2 mg·kg^{-1}. Anesthesia was maintained with 60% nitrous oxide and 0.5–1.0% isoflurane with oxygen; 0.25% bupivacaine was administered epidurally during the operation for analgesia when blood pressure or heart rate (or both) started to increase. The concentration of isoflurane was controlled to maintain a constant blood pressure. An arterial catheter was inserted into the radial artery to measure blood pressure and collect blood samples. The rectal temperature of the patient was monitored and was kept constant by controlling the operating room temperature and circulating a warm-water blanket.

After endotracheal intubation, patients were ventilated mechanically on the controlled mode by Servo 900C (Siemens, Solna, Sweden) with a constant tidal volume (V_T 10 ml·kg^{-1}) and frequency (12 breaths·min^{-1}). A square-wave flow pattern was used with an inspiration time of 25% and a 10% inspiratory pause. The side-stream sampling line of the expired gas analyzer (Ultima; Datex, Helsinki, Finland) was connected in-line with the endotracheal tube for measurement of end-tidal carbon dioxide pressure ($P_{ET}CO_2$). A Nortech Omniflator, model 7400 (Northgate Technologies, Arlington heights, IL, USA) was used to insufflate CO_2 at a constant intraabdominal pressure (12 cmH_2O). PaO_2, $PaCO_2$, CO_2 partial pressure of mixed expired gas (P_ECO_2), peak inspiratory pressure (PIP), and V_T were measured and recorded just before and 5, 10, 20, 40, and 80 min after the pneumoperitoneum. PaO_2, $PaCO_2$, and P_ECO_2 were measured by a STAT Profile 5 gas analyzer (NOVA Biomedical, Waltham, MA, USA).

Mixed expired gas was collected during five breaths into a Douglas bag on the expiratory limb of the nonrebreathing circuit. $\dot{V}_ECO_2$ was calculated by the following formula.

$$\dot{V}_ECO_2\ [\text{ml (STPD)}] = V_T(\text{ml}) \times \text{respiratory are} \times (P_ECO_2(\text{mmHg})/\text{barometric pressure (mmHg)} \times [273(°\text{K})/\text{body temperature (°K)}]$$

where STPD represents the volume of gas at standard temperature and pressure that contains no water vapor.

V_D/V_T was calculated by the following formula.

$$V_D/V_T(\%) = [PaCO_2(\text{mmHg}) - P_ECO_2(\text{mmHg})]/PaCO_2(\text{mmHg}) \times 100$$

(A-a)DO_2 was calculated using the formula for determining alveolar oxygen partial pressure

$$PaO_2 = (FiO_2 \times 713) - PaCO_2/R$$

where R is the respiratory exchange ratio (assumed to be 0.8).

Blood pressure, heart rate, PaO_2, $PaCO_2$, P_ECO_2, $\dot{V}_ECO_2$, PIP, V_T, V_D/V_T, and (A-a)DO_2 at different times were analyzed by one-way analysis of variance (ANOVA) with a subsequent Tukey multiple comparison test. We arbitrarily defined $\Delta\dot{V}_ECO_2(t)$ as the increase in $\dot{V}_ECO_2$ compared to the values before pneumoperitoneum, where t is the time (minute) after starting pneumoperitoneum. We defined $\Delta PIP(t)$ in the same manner. Both $\Delta\dot{V}_ECO_2(t)$ and $\Delta PIP(t)$ were analyzed by Kruskal-Wallis's test to evaluate the effect of CO_2 pneumoperitoneum on $\dot{V}_ECO_2$ and PIP. $\Delta\dot{V}_ECO_2(t)$ was also analyzed by fitting a monoexponential function.

$$\Delta\dot{V}_ECO_2(t) = (1 - e^{-t/\tau}) \cdot \Delta\dot{V}_ECO_2(\text{max})$$

where $\Delta\dot{V}_ECO_2(\text{max})$ is the maximum $\Delta\dot{V}_ECO_2(t)$ during peritoneal insufflation in each patient, and τ is the time constant calculated by the least-squares exponential equation. Statistical difference was considered significant at $P < 0.05$.

Results

The anesthesia and surgical procedure proceeded without complications. The mean durations of peritoneal insufflation and controlled ventilation were 128 ± 43 min and 184 ± 40 min, respectively. Neither blood pressure nor heart rate showed any significant changes throughout the study (Table 1).

The main results are summarized in Table 2. $PaCO_2$ showed a sudden increase during the initial 10 min of about 4 mmHg after peritoneal insufflation followed by a gradual increase thereafter. $PaCO_2$ at 40 and 80 min

Table 1. Various parameters before and after CO_2 insufflation

Parameter	Before	5 min	10 min	20 min	40 min	80 min
SBP (mmHg)	118.3 ± 8.5	120.4 ± 12.3	119.4 ± 16.5	111.0 ± 11.6	122.5 ± 16.4	117.8 ± 15.1
DBP (mmHg)	67.6 ± 10.9	69.0 ± 14.6	68.5 ± 12.1	65.3 ± 11.2	72.8 ± 16.4	69.5 ± 14.2
HR (beats·min^{-1})	78.4 ± 12.5	80.9 ± 19.4	84.0 ± 23.0	78.5 ± 19.4	79.8 ± 17.3	78.4 ± 16.4

SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate.

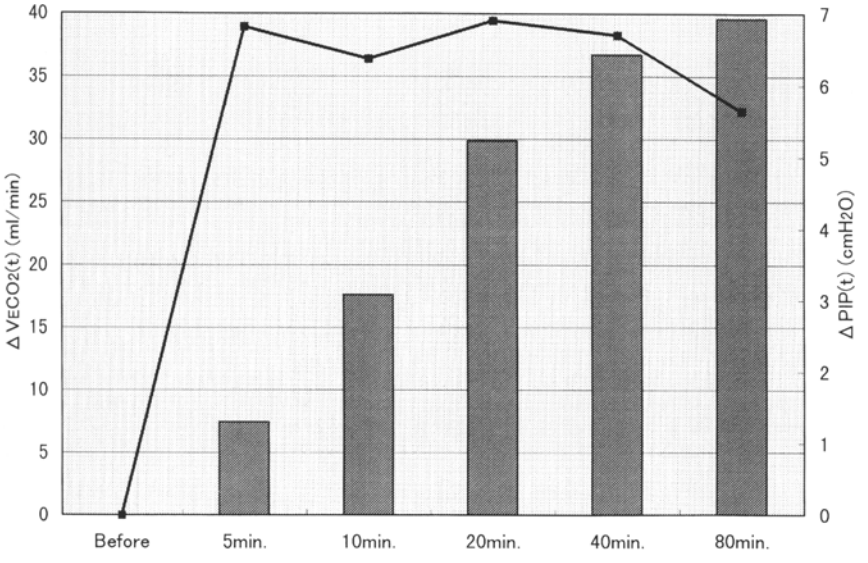

Fig. 1. Sequential changes of mean $\Delta\dot{V}ECO_2(t)$ (*bars*) and mean $\Delta PIP(t)$ (*solid line*) before and after CO_2 insufflation. They showed an increase with time after induction of pneumoperitoneum ($P < 0.01$, Kruskal-Wallis's test)

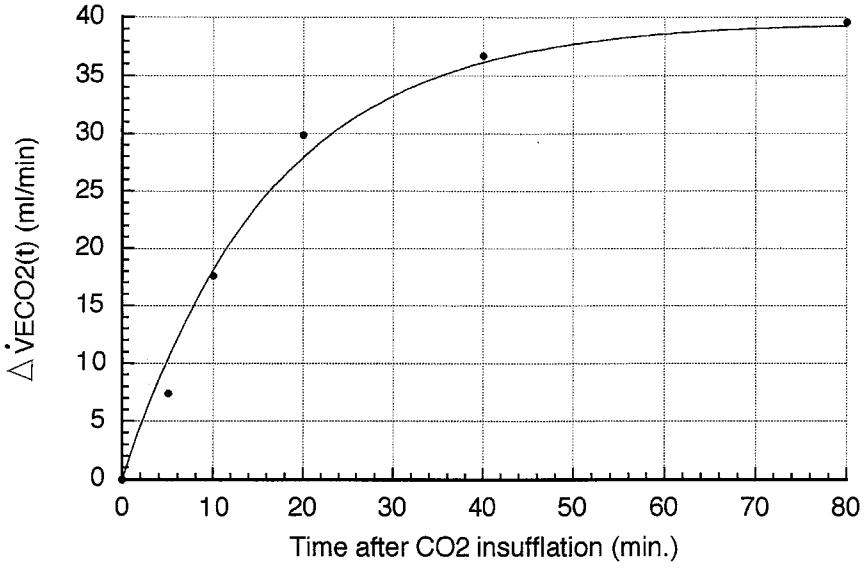

Fig. 2. Sequential changes of mean $\Delta\dot{V}ECO_2(t)$ before and after CO_2 insufflation. Mean $\Delta\dot{V}ECO_2(t)$ changed following a monoexponential function. $\Delta\dot{V}ECO_2(t) = (1 - e^{-t/16.4}) \times 39.6$ (ml/min); R = 0.995

after peritoneal insufflation were significantly higher than the preinsufflation value. The increase in $\dot{V}ECO_2$ was approximately 30 ml·min^{-1} (20%) on average during the initial 20 min and reached a plateau within 20–40 min after the pneumoperitoneum. $\Delta PIP(t)$ indicated an abrupt increase, and $\Delta\dot{V}ECO_2(t)$ showed a gradual increase with time after induction of pneumoperitoneum ($P < 0.01$, Kruskal-Wallis's test) (Fig. 1). In

Table 2. Various parameters, before and after CO_2 insufflation

Parameter	Before	5 min	10 min	20 min	40 min	80 min
PaO_2 (mmHg)	190.3 ± 25.3	193.1 ± 21.0	199.4 ± 27.7	199.0 ± 14.6	201.3 ± 16.9	211.0 ± 12.9
$PaCO_2$ (mmHg)	32.0 ± 3.1	33.1 ± 4.2	35.7 ± 4.1	37.3 ± 4.3	40.4 ± 4.4*	42.7 ± 5.7**
$PECO_2$ (mmHg)	18.1 ± 1.6	18.6 ± 1.4	20.1 ± 1.6	21.3 ± 1.3**	22.0 ± 1.6**	23.4 ± 2.0**
$VECO_2$ (ml/min)	152.7 ± 24.1	160.1 ± 25.9	170.3 ± 28.1	182.5 ± 32.2	189.4 ± 31.9	192.3 ± 36.7
PIP (cmH_2O)	16.9 ± 5.8	23.7 ± 9.2	23.3 ± 8.5	23.8 ± 7.6	23.6 ± 6.3	22.6 ± 6.0
VT (ml)	595.8 ± 77.1	585.3 ± 80.5	583.0 ± 80.5	585.8 ± 87.6	591.3 ± 75.2	564.3 ± 91.5
VD/VT (%)	43.7 ± 5.4	42.6 ± 8.6	41.6 ± 7.7	40.9 ± 5.4	44.9 ± 7.7	44.0 ± 9.1
(A-a)DO_2 (mmHg)	53.2 ± 28.9	49.2 ± 25.8	40.1 ± 32.1	37.6 ± 13.5	29.4 ± 12.5	20.2 ± 13.9

$PECO_2$, CO_2 partial pressure of mixed expired gas; $VECO_2$, pulmonary carbon dioxide elimination; PIP, peak inspiratory pressure; VT, tidal volume; VD/VT, dead space/tidal volume ratio; (A-a)DO_2, alveolar-arterial PO_2 difference.
* $P < 0.05$ *vs* before CO_2 insufflation; ** $P < 0.01$ *vs* before CO_2 insufflation.

five of eight patients, $\Delta\dot{V}ECO_2(t)$ increased exponentially during CO_2 insufflation. The mean values of $\Delta\dot{V}ECO_2(max)$ and τ in our patients were 50.1 ± 19.5 ml/min and 35.3 ± 23.2 min, respectively. The mean $\Delta\dot{V}ECO_2(t)$ also changed following a monoexponential function (Fig. 2). $PECO_2$ showed a greater increase during the initial 20 min than the rest of the 60 min. $PECO_2$ at 20 min after peritoneal insufflation was significantly higher than the preinsufflation value. Neither VD/VT nor VT showed any significant changes during the study. The (A-a)DO_2 gradually decreased after pneumoperitoneum, but the change was also not significant. No patient suffered from hypoxemia, and the mean PaO_2 was maintained at around 200 mmHg throughout the study.

Discussion

Many surgeons prefer to use CO_2 for pneumoperitoneum during laparoscopic cholecystectomy because of its higher solubility in blood than nitrous oxide [10] and a lower frequency of venous air embolism. However, peritoneal insufflation with CO_2 often brings about unfavorable effects, such as hypercarbia and high peak airway pressure.

Three factors may contribute to the increase in $PaCO_2$ during pneumoperitoneum with CO_2. First, the VT may be substantially reduced owing to the increase in PIP during pneumoperitoneum. Because the compression volume is relatively large in a ventilator with a semiclosed system, the expired respiratory volume might be far less than the preset volume. To avoid this problem and maintain a constant VT we used a Servo 900C ventilator, which allows minimum compression volume. The second factor is transperitoneally absorbed CO_2. With this absorption of CO_2, $PaCO_2$ rises if the preset volume of the ventilator is kept constant. Third, the diaphragm is shifted cephalad, and respiratory compliance may be reduced with peritoneal insufflation. Because of the effect of peritoneal insufflation on the respiratory system, redistribution of inspired gas to underperfused or nonperfused areas is likely to occur, and a ventilation—perfusion inequality may develop. In the present study the VD/VT was unchanged, suggesting that the ability of the respiratory system to eliminate CO_2 did not deteriorate with a constant tidal volume and respiratory rate during pneumoperitoneum.

During pneumoperitoneum with CO_2 the $PaCO_2$ increases during both gynecological laparoscopy [11,12] and laparoscopic cholecystectomy [13–16]. If the respiratory minute volume is constant, the $PaCO_2$ increases with time during pneumoperitoneum by 34–39% [11–13]. In the present study the $PaCO_2$ increased about 33%, a result similar to those in other studies [11–13].

The $\dot{V}ECO_2$ rapidly increased during the initial stage of pneumoperitoneum, and this increase was blunt and less rapid during the latter half of the study course. Our results suggest that the $\Delta\dot{V}ECO_2(t)$ changes following a monoexponential function. This increase in $\dot{V}ECO_2$ consists of transperitoneally absorbed CO_2 (extrinsic CO_2) and CO_2 produced in the patient's body (intrinsic CO_2). Although intrinsic CO_2 cannot be distinguished from extrinsic CO_2 in this clinical setting, we presumed that the change in intrinsic CO_2 may be minimum because the patients were anesthetized and hemodynamically stable during the study. Thus extrinsic CO_2 may account for most of the increase in $\dot{V}ECO_2$ in our patients. This hemodynamic stability may have been contributed to by epidural anesthesia, which inhibited the sympathetic response to surgical stimuli. Our results suggest the clinical usefulness of the combination of general anesthesia and epidural anesthesia. In terms of total intravenous anesthesia, the study by Girardis et al. [9] failed to keep mean arterial pressure (MAP) constant during laparoscopic cholecystectomy.

The shape of the $\dot{V}ECO_2$ curves during pneumoperitoneum was similar to those in other studies [17,18]. During the initial stage of pneumoperitoneum the $PaCO_2$ showed a sudden rise, which suggests that transperitoneal absorption of CO_2 far exceeded the increase in $\dot{V}ECO_2$ and the CO_2 body store abruptly increased. At 40–80 min after pneumoperitoneum, both $PaCO_2$ and $\dot{V}ECO_2$ showed little increase. At this time the increase in $\dot{V}ECO_2$ and transperitoneal absorption may have been in equilibrium, based on the assumption that elimination of intrinsic CO_2 showed no or little change during pneumoperitoneum.

Oxygenation was not impaired during pneumoperitoneum in the present study. Because laparoscopic cholecystectomy is performed in reverse Trendelenburg position, diaphragmatic elevation may be attenuated and a V/Q mismatch may not develop as has been observed during gynecologic laparoscopy. The result may also be explained by the relation between alveolar PCO_2 and hypoxic pulmonary vasoconstriction (HPV). Hypocapnia has been thought to inhibit regional HPV directly, and hypercapnia has been thought to enhance regional HPV directly [19]. Ooka et al. observed a transient decrease in PaO_2 with N_2O pneumoperitoneum followed by the PaO_2 regaining a normal level during CO_2 insufflation [14]. The increase in $PaCO_2$ during CO_2 pneumoperitoneum found in our study may have enhanced the HPV and avoided the decrease in PaO_2 observed during N_2O pneumoperitoneum [14].

In conclusion, the $\dot{V}ECO_2$ showed a sudden rise at the induction of pneumoperitoneum and reached a plateau within 20–40 min after the induction in patients undergoing laparoscopic cholecystectomy. Transperitoneal absorption of CO_2 may be the main cause of hypercarbia, and the hypercarbia is not attributed to the increase in V_D/V_T during pneumoperitoneum. Oxygenation was not impaired throughout the study.

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