The Changes of Ventilatory Parameters in Laparoscopic Colecystectomy

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—— Abstract ——

We investigated the ventilatory changes in healthy patients without cardiopulmonary pathology during elective laparoscopic cholecystectomy in the head-up position. During surgery, intraabdominal pressure was maintained at 15 mmHg by a CO_2 insufflator, and minute ventilation was controlled with a constant tidal volume and fixed respiratory rate. $P_{ET}CO_2$ was monitored continuously and recorded every minute. Basic hemodynamic and ventilatory parameters were measured before anesthesia; after induction of anesthesia; at 5 min, 10 min and 30 min after peritoneal insufflation; and 5 min and 10 min after exsufflation. Arterial blood samples were obtained 3 times to calculate $D_{(a-A)}CO_2$, V_D/V_T, and $\dot{V}co2$. The latent period of $P_{ET}CO_2$ change was 2.9 min, the ascending period was 12.6 min, and the descending period was 12.2 min. During the 71.5 min of pneumoperitoneum, V_I, V_E, peak and plateau Paw increased, while Cdyn decreased significantly. Peritoneal insufflation or exsufflation also resulted in a significant change of $D_{(a-A)}CO_2$, $D_{(a-A)}O_2$, and $\dot{V}co2$. The anesthesiologist must be aware of both hemodynamic and ventilatory changes and must be ready to respond promptly and adequately.

Key Words: Ventilatory effect, laparoscopy, head-up position

INTRODUCTION

Although laparoscopy was introduced early in the 20th century and developed in the 1970s for gynecologic procedures, its recent extension to gastrointestinal surgical procedures has created new interest and considerations in anesthetic management. Many benefits were reported on laparoscopy[1,2] and explained its increasing success and the efforts to introduce the endoscopic approach for other surgical procedures. However, peritoneal insufflation with CO_2, creating a pneumoperitoneum necessary for laparoscopy, induces intraoperative hemodynamic[3] and ventilatory changes[4,5] which complicates anesthetic management. The position of the patient (head-down or head-up) required during these procedures also contributes to the changes.[4,6]

Three main ventilatory problems can occur during laparoscopy: an increase in $PaCO_2$, pneumothorax[7] although rare, and gas embolism.[8] It is generally accepted that hypercarpnea observed during laparoscopy results from peritoneal CO_2 absorption, mechanical impairment of ventilation caused by the pneumoperitoneum, and depression of ventilation by anesthetic agents and neuromuscular relaxants,[9-11] but still remains a controversy. During intraperitoneal insufflation of CO_2 under a constant minute volume of ventilation, increase in both $PaCO_2$ and $P_{ET}CO_2$ were observed in animals[12] and humans.[5,10] Elevation of the diaphragm due to pneumoperitoneum resulted in the mismatch of pulmonary ventilation and perfusion.[12] The head-up position is generally considered to be more favorable to respiration.[13,14] However, the actual effect on V_A/Q, when the patient is in a head-up tilt position receiving mechanical ventilation and general anesthesia, has not been investigated.

We investigated the basic hemodynamic and ventilatory changes in healthy patients without cardiopulmonary pathology during laparoscopic cholecystectomy in the head-up position.

MATERIALS AND METHODS

In this study, 22 fully informed healthy patients (ASA physical status I) underwent elective laparoscopic cholecystectomy. Exclusion criteria were as follows: obesity, asthma, extreme age (<15 or >65), acute cholecystitis, and cardiorespiratory disease.

All patients were premedicated with an intramuscular injection of midazolam ($2.5-3$ mg) and glycopyrrolate (0.2 mg) just before being transferred to the operating room. $1-2$ minutes before induction of anesthesia, fentanyl (1.5 μg/kg) and lidocaine (1 mg/kg) were administered intravenously and one radial artery was cannulated. They received thiopental sodium (5 mg/kg) and succinylcholine (1 mg/kg) for induction of anesthesia and this was followed by intravenous pancuronium (0.1 mg/kg), 50% nitrous oxide and $1-2$ vol% enflurane in oxygen, administered via a circle system with carbon dioxide absorption. After intubation, the cuffs of the endotracheal tubes were filled with air until the intraballoon pressure reached 30 cmH2O by using an endotracheal pressure checker (National Catheter Co. Mallinckrodt Inc., New York, U.S.A.). Minute ventilation was controlled (Ohmeda 7000 Ventilator, BOC Health Care, Medison) with a constant tidal volume (10 ml/kg) and respiratory rate (10/min) in adjustment to keep the initial expired end-tidal CO_2 ($P_{ET}CO_2$) between $33-35$ mmHg. A basal IV infusion (4 mg/kg/hr) of lactated Ringer's solution was given to compensate for the fasting state and intraoperative losses.

During surgery, the $P_{ET}CO_2$ was monitored continuously by a Datex Capnomac sidestream analyzer (Datex, Instrumentarium Co., Helsinki, Finland) and recorded every minute. The latent ascending and descending periods were recorded. The latent period meant the duration from the point of CO_2 insufflation to the point when $P_{ET}CO_2$ started to increase. The ascending period was the period after $P_{ET}CO_2$ started to increase, until it reached a plateau (maintain constant $P_{ET}CO_2$ for 5 min), and finally the descending period was the time needed to wash out the end-tidal CO_2 to a constant level for at least 5 min after exsufflation of CO_2.

During the laparoscopic procedure, intraabdominal pressure (IAP) was automatically maintained at 15 mmHg by a CO_2 insufflator (Sirakawa Olympus Co., Kawasaki, Japan). The following variables were collected: before induction (T0); 5 min after intubation (T1); 5 min (T2), 10 min (T3), and 30 min (T4) after insufflation of CO_2; and 5 min (T5) and 10 min (T6) after exsufflation of CO_2; mean arterial pressure (MAP); heart rate (HR); peripheral oxygen saturation (SpO_2); inspiratory and expiratory tidal volume (ins/exp V_T); peak airway pressure (P_{aw}); plateau pressure (P_{plt}); mixed expired CO_2 pressure (P_ECO_2), CO_2 concentration in mixed expired gas (F_ECO_2) and dynamic compliance (C_{dyn}) by a Datex Capnomac sidestream analyzer.

Carbon dioxide was measured with a dual-beam nondispersive infrared optical system with pneumatic detection (Beckman LB2 CO_2 analyzer, Fullerton, Calif., U.S.A.). Calibration of the gas sensors were automatically accomplished with a zero gas (100% N_2) and a calibration gas (4% CO_2, 21% O_2 in N_2).

Arterial blood sampling was done at the same time to measure arterial PCO_2 ($PaCO_2$) in order to calculate $D_{(A-a)}CO_2$, physiologic dead space ventilation ratio (V_D/V_T), and CO_2 production ($\dot{V}CO_2$). These data were obtained 3 times; 5 min after intubation (T1), 10 min after CO_2 insufflation (T3), and 5 min after exsufflation (T5).

CO_2 production was calculated as
$$\dot{V}CO_2 = V_E \times F_ECO_2,$$
F_ECO_2; CO_2 concentration in the mixed expired gas.
V_E; expired minute volume
Physiologic dead space ventilation ratio was measured as

$$V_D/V_T = (PaCO_2 - P_ECO_2)/PaCO_2$$

All patients were tilted into a $10°$ head-up position $6-7$ min after CO_2 insufflation, and were repositioned to a supine position just after exsufflation. A basal intravenous infusion (4 ml/kg/hr) of lactate Ringer's solution was given to compensate for the fasting state and intraoperative losses.

Results were reported as mean $\pm$ standard deviation (SD). Data were analyzed by a one way analysis of variance for repeated measures followed by Newman-Keuls test and independent unpaired T-test. Results were considered to be statistically significant at the 5% critical level.

RESULTS

Our study included 22 patients. The mean age was

Table 1. Hemodynamic Changes during Laparoscopic Cholecystectomy

Hemodynamic changes	T0 before induction	T1 5 min after induction	T2	T3	T4	T5 5 min after exsufflation	T6 10 min after exsufflation
			Pneumoperitoneum				
			5 min	10 min	30 min		
MAP, mmHg	97.2±18.7	84.6±19.8*	105.8±17.4*	107.1±13.6*†	100.3±17.4†	96.3±12.4†	95.7±17.5†
HR, beat/min	75.6±13.4	81.4±14.4*	84.1±13.6*†	82.8±15.2*†	83.9±14.5*†	81.8±15.6*†	80.4±15.7†
S_PO_2, %	98.5±0.9	98.3±0.8	98.3±1.0	98.3±0.9	98.1±1.4	97.9±1.5	98.3±1.2

Hemodynamic changes were measured before the induction of anesthesia (T0), 5 min after the induction of anesthesia (T1), 5 min (T2), 10 min (T3), and 30 min (T4) after the beginning of insufflation, and 5 min (T5), 10 min (T6) after exsufflation. Results are mean±SD.
Abbreviation used are: MAP, mean arterial pressure; HR, heart rate; S_PO_2, peripheral oxygen saturation.
* $p < 0.05$ as compared with T0.
† $p < 0.05$ as compared with T1.

Table 2. Ventilatory Changes during Laparoscopic Cholecystectomy

Ventilatory changes	T1 5 min after induction	T2	T3	T4	T5 5 min after exsufflation	T6 10 min after exsufflation
		Pneumoperitoneum				
		5 min	10 min	30 min		
V_I, L/min	5.55±88.9	4.93±82.4†	4.91±83.7†	4.91±80.2†	5.28±81.7	5.32±80.8
V_E, L/min	5.19±81.8	4.85±73.0†	4.91±76.8†	5.04±80.2	5.43±62.4	5.53±62.3
Peak P_{aw}, cmH$_2$O	15.1±2.8	20.7±3.8†	20.7±3.8†	20.7±4.0†	16.6±3.2†	16.4±3.0†
Plateau P_{aw}, cmH$_2$O	14.6±3.4	19.8±4.0†	20.2±3.6†	20.2±4.0†	15.9±3.3†	15.6±3.5†
Cdyn, L/kPa	44.3±7.2	28.4±6.6†	27.6±6.0†	29.4±7.1†	41.5±7.0†	43.0±8.5
PaCO$_2$, mmHg	35.5±3.9		40.9±4.6†		40.6±4.7	
PaO$_2$, mmHg	219.0±28.7		215.7±28.2		193.9±46.9	
$D_{(a-A)}CO_2$, mmHg	3.14±2.0		3.95±2.7†		3.42±2.2*	
$D_{(a-A)}O_2$, mmHg	93.4±29.5		89.6±28.4		111.9±45.9*†	
V_D/V_T, %	44.1±9.2		44.1±5.8		43.8±7.5	
$\dot{V}co_2$, mL/min	219.2±30.4		237.9±24.9		272.8±19.4*†	

Expired end-tidal CO$_2$ (P$_{ET}$CO$_2$), inspired minute volume (V$_I$), expired minute volume (V$_E$), peak and plateau airway pressure (Paw), and dynamic lung compliance (Cdyn) were measured 5 min after the induction of anesthesia (T1), 5 min (T2), 10 min (T3), 30 min (T4) after the beginning of insufflation, and 5 min (T5), 10 min (T6) after exsufflation.
Arterial alveolar carbon dioxide difference [D$_{(a-A)}$O$_2$], arterial alveolar oxygen difference [D$_{(a-A)}$O$_2$], tidal volume ratio (V$_D$/V$_T$), and carbon dioxide output (V̇co$_2$) were calculated 5 min after the induction of anesthesia (T1), 10 min after the beginning of insufflation (T3), and 5 min after exsufflation (T5) from observed ventilatory parameters.
Results are mean±SD.
* $p < 0.05$ as compared with T3.
† $p < 0.05$ as compared with T1.

47.7±14.6 yr with a mean height and weight of 161.1±7.3 cm and 61.5±8.5 kg, respectively. The duration of CO_2 insufflation was 71.5±25.4 min.

Hemodynamic changes during surgery are shown in Table 1. There were no significant changes in SpO_2 after induction, insufflation or exsufflation of CO_2, and changes in patient position. MAP decreased after induction, but increased after peritoneal insufflation and even after deflation of CO_2. HR values were significantly greater than the baseline value after induction, during pneumoperitoneum, and after exsufflation.

The time lag from CO_2 insufflation to the emergence of $P_{ET}CO_2$ change (latent period) was 2.90±1.78 min. The $P_{ET}CO_2$ changed from baseline to a plateau (ascending period) which was 12.64±4.12 min. The $P_{ET}CO_2$ declined from plateau to baseline after CO_2 deflation (descending period) which was 12.21±3.78 min. The initial baseline $P_{ET}CO_2$ was 33.7±1.9 mmHg, the initial plateau of $P_{ET}CO_2$ was 40.8±2.7 mmHg, the last plateau of $P_{ET}CO_2$ just before deflation was 42.6±3.1 mmHg, and the last $P_{ET}CO_2$ was 37.4±2.5 mmHg.

The changes of ventilatory parameters during surgery are shown in Table 2. After insufflation, V_I, V_E, peak and plateau P_{aw} increased significantly, while C_{dyn} decreased significantly. Ten minutes after exsufflation, V_I, V_E, C_{dyn} had returned to pre-insufflation values, while peak and plateau P_{aw} did not. Peritoneal insufflation or exsufflation also resulted in a significant change of $D_{(a-A)}CO_2$ and $D_{(a-A)}O_2$. An 8% increse in production of CO_2 ($\dot{V}CO_2$) during pneumoperitoneum further increased to 24.5%, though the gas was deflated. There was no clinical evidence of hypoxia during or after anesthesia, and recovery occurred within 15 minutes in all cases. There were no postoperative complications.

DISCUSSION

This study demonstrates that peritoneal CO_2 insufflation with an intraabdominal pressure of 15 mmHg necessary for laparoscopic cholecystectomy induces hemodynamic and ventilatory changes in healthy patients.

The changes in blood pressure and heart rate observed in this study complemented the results of recent studies during gynecologic laparoscopy in the

head-down position.[3,15] After the induction of anesthesia, MAP decreased and HR increased. Anesthetic induction drugs might depress the myocardium and therefore reduced MAP. Tilting patients to the head-up position reduced venous return, and MAP further decreased. After peritoneal insufflation with CO_2, MAP increased significantly during pneumoperitoneum and was sustained after exsufflation.

Joris et al. reported that only an increase of SVR could explain the increase in MAP observed after insufflation despite the significant reduction of cardiac index (CI).[16] Increased venous resistance and compression of the abdominal aorta may contribute to the increase of cardiac afterload.[3,15] However, it is unlikely that the increase in MAP and SVR was entirely related to mechanical factors because the correction of these changes were gradual and took 10 min. or more when the intraabdominal pressure was released suddenly at the end of the operation. Whereas these cardiovascular changes should not be hazardous in healthy patients, special care is necessary for patients with impaired cardiac function.

$P_{ET}CO_2$ is generally considered to provide reliable information on $PaCO_2$ during laparoscopy.[17,18] However, two points need to be emphasized. First, the $D_{(a-A)}CO_2$ varied from patient to patient.[19,20] Second, although mean $D_{(a-A)}CO_2$ did not change significantly during peritoneal insufflation of CO_2 in some studies,[5,18] individual patient data, including this study, regularly showed an increase of this difference during pneumoperitoneum.[21] The choice of anesthetic technique and duration of pneumoperitoneum influenced the extent of the increase in $PaCO_2$. Kenefick et al.[22] did not report any significant increase of $PaCO_2$, but the insufflation periods were shorter than in the study of Hodgson et al.,[10] Lewis et al.,[11] and ours. Several studies reported that it took 15−25 min for $PaCO_2$ to plateau.[5,10] In our study, we got similar results at 12.6 min of the ascending period. The unique pattern of increasing $P_{ET}CO_2$ is a useful predictor for anesthesiologists, who not only must prevent and adequately respond to these changes, but also must evaluate and prepare the patients preoperatively in the light of these disturbances. Also, patients with preoperative cardiopulmonary disease demonstrated a significantly larger increase in $PaCO_2$ than patients without underlying disease.[21]

With the patient in the head-up position for laparoscopic cholecystectomy, the effect of CO_2 insuf-

flation has led to some contradictory results. In this study, the actual effect of head-up tilt on V_A/Q was not investigated.

The mechanism of the increase of $PaCO_2$ is controversial. During CO_2 pneumoperitoneum, absorption of CO_2 from the peritoneal cavity, impairment of ventilation by mechanical factors such as abdominal distension, position of the patient, and volume-controlled mechanical ventilation are all factors.

The observation of an increase in $PaCO_2$, when CO_2 not nitrous oxide was used as the peritoneal insufflating gas, suggests that absorption of CO_2 from the peritoneal cavity was a potential mechanism for the $PaCO_2$ increase.[12] This hypothesis was supported by the increased production of CO_2 ($\dot{V}co_2$) observed in our study, and by the increased $PaCO_2$ when CO_2 not nitrous oxide was used as an insufflating gas.[23] However, other studies failed to demonstrate any significant changes in $\dot{V}co_2$ during pneumoperitoneum with CO_2.[4,11] The limited increase in $PaCO_2$ actually observed can be explained by the capacity of the body to store CO_2 and by impaired local perfusion. Interestingly, $P_{ET}CO_2$ declined abruptly after deflation of CO_2, but $\dot{V}co_2$ increased continuously up to 24.5% even after exsufflation. The reason for that is beyond the scope of this study.

Regardless, an augmentation of the arterioalveolar CO_2 difference $[D_{(a-A)}CO_2]$, reflected an increase in the physiologic dead space. This would indicate that if controlled ventilation is not adjusted in response to the increased dead space, induced hypercapnea may aggravate homeostasis of the patient. Although increased $PaCO_2$ may be well tolerated by young, healthy patients, the extent to which hypercapnia is acceptable has not been determined and probably varies according to the patient's physical status. Therefore, it is wise to maintain $PaCO_2$ within physiologic values by adjusting controlled mechanical ventilation. Also these indicate the need for gasless laparoscopy or low CO_2 insufflation pressure in patients with impaired cardiac or pulmonary function scheduled for laparoscopy.

In conclusion, these results highlight the fact that laparoscopy induces significant hemodynamic and ventilatory changes even in healthy patient and creates increases of MAP and HR, $D_{(a-A)}CO_2$, $\dot{V}co_2$, V_D/V_T, and a reduction of C_{dyn}. Whereas these changes may not be harmful in healthy patients, special care and monitoring are mandatory for patients with impaired cardiopulmonary function. In these patients, the postoperative benefits of laparoscopy should be balanced against intraoperative risks.

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