Respiratory mechanics and arterial blood gases during and after laparoscopic cholecystectomy

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Purpose: *The purpose of this study was to assess the effects of increased intra-abdominal pressure due to CO_2 insufflation on the mechanical characteristics of the respiratory system and arterial blood gases during and after laparoscopic cholecystectomy.*
Methods: *Respiratory mechanics and arterial blood gases were examined in 12 patients undergoing laparoscopic cholecystectomy with CO_2 insufflation. Respiratory mechanics were continuously monitored with in-line spirometry. In the recovery room, $PaCO_2$ was measured in this group at 30 min and compared with $PaCO_2$s in 23 patients who had undergone open cholecystectomy retrospectively, to evaluate the effects of insufflation on CO_2 elimination.*
Results: *Minute ventilation was decreased by about 500 $ml \cdot min^{-1}$ during abdominal insufflation. Dynamic lung compliance decreased from 49.6 ± 4.7 to 30.7 ± 2.3 (mean ± SEM) $ml \cdot cmH_2O^{-1}$ with abdominal insufflation ($P < 0.005$), and returned to 45.1 ± 3.1 after the release of pneumoperitoneum. Peak inspiratory pressure increased from 15.9 ± 0.9 to 18.9 ± 1.0 cmH_2O with abdominal insufflation ($P < 0.05$). Arterial blood gas determinations indicated a decrease in arterial pH, with CO_2 retention during insufflation and in the recovery room ($P < 0.05$). $PaCO_2$ of the laparoscopic patients was higher than that of the open patients in the recovery room.*
Conclusion: *The results indicate that respiratory acidosis was caused during CO_2 insufflation for laparoscopic cholecystectomy, that was due to (1) decreased compliance, (2) increased CO_2 load and (3) insufficient ventilation. Accumulated CO_2 during laparoscopic cholecystectomy increased $PaCO_2$ level in the recovery room.*

Objectif: *Evaluer les effets de l'augmentation de pression intraabdominale provoquée par l'insufflation de CO_2 sur les caractéristiques du système respiratoire et des gaz du sang artériel pendant et après la cholécystectomie laparoscopique.*
Méthode: *La mécanique respiratoire et les gaz du sang artériels ont été étudiés chez 123 patients soumis à une cholécystectomie laparoscopique avec insufflation de CO_2. La mécanique respiratore a été monitorée en continu par spirométrie. A la salle de réveil, la $PaCO_2$ a été mésurée à la 30^e min de l'admission et comparée rétrospectivement à la $PaCO_2$ de 23 patients qui avaient subi une cholécystectomie ouverte, dans le but d'évaluer les effets de l'insufflation sur l'élimination du CO_2.*
Résultats: *La ventilation minute a diminué d'environ 500 $ml \cdot min^{-1}$ pendant l'insufflation abdominale. La compliance dynamique pulmonaire diminuait de 49,6 ± 4,7 à 30,7 ± 2,3 (moyenne ± SEM) $ml \cdot cmH_2O^{-1}$ avec l'insufflation ($P < 0,005$) et revenait à 45,1 ± 3,1 après le relâchement du pneumopéritoine. L'analyse des gaz artériels a révélé une diminution du pH artériel avec rétention de CO_2 pendant l'insufflation et à la salle de réveil ($P < 0,005$). La $PaCO_2$ des patients opérés sous laparoscopie était plus élevée que celle des patients opérés par chirurgie ouverte.*
Conclusion: *Ces résultats indiquent que l'insufflation de CO_2 pour la cholécystectomie laparoscopique provoque de l'acidose respiratoire causée 1) par la baisse de la compliance, 2) l'augmentation du volume de CO_2 et 3) l'insuffisance ventilatoire. L'accumulation du CO_2 pendant la cholécystectomie laparoscopique augmente la $PaCO_2$ à la salle de réveil.*

Key words
MONITORING: dynamic lung compliance, peak inspiratory pressure, carbon dioxide tension;
SURGERY: laparoscopic cholecystectomy.

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Laparoscopic cholecystectomy has gained worldwide acceptance because of shortened hospital stays and improved patient satisfaction.[1,2] This surgical laparoscopic procedure involves change in patient position from supine to reverse Trendelenburg, and requires intraperitoneal carbon dioxide (CO_2) insufflation.

Laparoscopic cholecystectomy may require longer periods of peritoneal insufflation than previously reported in gynaecological procedures. It is conceivable that the longer CO_2 insufflation might impede diaphragmatic movement and increase the CO_2 load for ventilation.[3,4] Respiratory mechanics and blood gases must be monitored to identify the mechanical and ventilatory effects of insufflation.

However, the reverse Trendelenburg position may be accompanied by respiratory advantages,[2] because arterial hypercapnia may be caused not only by the transperitoneal absorption of CO_2 but also by the decreased dynamic lung compliance during laparoscopic cholecystectomy.[3] To test this hypothesis, we assessed the effects of increased intra-abdominal pressure due to CO_2 insufflation on the mechanical characteristics of the respiratory system and on arterial blood gas analysis during and after laparoscopic cholecystectomy. The effects of CO_2 load during laparoscopic cholecystectomy on $PaCO_2$ in the recovery room was compared with that in patients undergoing open cholecystectomy.

TABLE I Data on patients

	Laparoscopic cholecystectomy	*Open cholecystectomy*
n	12	23
Age (yr)	50.8 ± 3.4	55.1 ± 2.8
Weight (kg)	57.0 ± 3.5	57.7 ± 1.7
Sex (M/F)	4/8	9/14
Duration of anaesthesia (min)	142.5 ± 10.1	134.3 ± 8.8
Duration of operation (min)	110.8 ± 8.9	97.6 ± 6.1

Data are mean ± SEM. For any variables, $P > 0.05$.

Methods

The study was approved by the Institutional Ethics Committee. Informed consent was obtained from 12 patients (eight women, four men) scheduled for laparoscopic cholecystectomy. Retrospective following-up of the anaesthesia records was also approved by the Ethics Committee. None had functionally limiting respiratory, cardiac, or metabolic disease. All were classified as American Society of Anesthesiology (ASA) physical status I.

All patients were premedicated with hydroxyzine (50 mg) and atropine (0.5 mg) *im* 30 min before transfer to the operating theatre. Anaesthesia was induced with thiamylal sodium (4–5 $mg \cdot kg^{-1}$). Vecuronium bromide (0.1–0.15 $mg \cdot kg^{-1}$) was given for muscle relaxation. After tracheal intubation, general anaesthesia was maintained with 60% nitrous oxide in oxygen and sevoflurane (1.0–2.0%) as needed. Adequate muscle relaxation was achieved by incremental injection of vecuronium when the train-of-four ratio, which was monitored by the force of contraction of the adductor pollicis, exceeded 75%. The lungs were mechanically ventilated with the same volume-cycled ventilator using a semi-closed circle system incorporating a carbon dioxide absorber (NARKOMED 2B). The ventilator was inspected for leaks before each case and the same type of disposable breathing tubing was used throughout the study. Tidal volume was set at 10 $ml \cdot kg^{-1}$, with a rate of 10 $breaths \cdot min^{-1}$. This was maintained throughout the study period. An Ultima SV respiratory in-line monitor (Datex Instrumentarium Corp, Helsinki, Finland) was used to collect respiratory data.[5] This monitor, which undergoes regular maintenance and calibration, analyses respiratory gases (inspiratory and expiratory oxygen and carbon dioxide concentrations) and ventilatory variables (expiratory tidal volume, dynamic lung compliance, peak and plateau inspiratory pressure). (O_2 linearity: <2%, CO_2 linearity: <2%, airway pressure error: ±1.5 cmH_2O, tidal volume error: ±6%). These values were displayed breath-by-breath. Arterial blood gas samples were analyzed immediately using an infrared spectrometer (ABL-3, Radiometer Inc, West Lake, Ohio), which was autocalibrated regularly (two point calibration every four hours, one point calibration every two hours). The measurements were made just before (approximately 15 min after induction of anaesthesia), and one hour after the establishment of a pneumoperitoneum with the patient positioned in the reverse Trendelenburg (10°) position with a left tilt (5°). Measurements were also made just after abdominal desufflation with the patient in the supine position. Arterial blood was also sampled in the recovery room (30 min after tracheal extubation). Carbon dioxide was used for peritoneal insufflation, and abdominal pressure was maintained at 8 to 10 mmHg.

We also compared, retrospectively, the $PaCO_2$ in the recovery room (30 min after tracheal extubation) in similar unselected patients ($n = 23$, ASA physical status I) who had undergone open cholecystectomy to examine the effects of insufflation on CO_2 elimination.

Results are reported as the mean ± SEM. The effects of successive steps in the procedure were evaluated by one-way ANOVA and Fisher's Protected Least Significant Difference. Linear regression analysis was performed by the method of least squares. Comparison between end-tidal and arterial CO_2 was performed with the Bland and Altman method.[6] Significance was assumed at the $P < 0.05$ level.

Results

As shown in Table I, there was no differences in age,

TABLE II Effects of carbon dioxide insufflation on respiratory mechanics during laparoscopic cholecystectomy

	Pre-insufflation	*1 hr after insufflation*	*Post-desufflation*
Dynamic lung compliance (ml · H_2O^{-1})	49.6 ± 4.7	30.7 ± 2.3**	45.1 ± 3.1†
Peak inspiratory pressure (cmH_2O)	15.9 ± 0.9	18.9 ± 1.0*	17.5 ± 1.3
Inspiratory plateau pressure (cmH_2O)	13.2 ± 1.1	17.4 ± 1.1**	14.9 ± 1.0
Expiratory tidal volume (ml)	543.3 ± 33.4	496.5 ± 37.7	546.2 ±33.3
$PaCO_2$ (mmHg)	34.5 ± 0.7	42.8 ±1.0**	37.5 ± 1.1†
End-tidal CO_2 (mmHg)	34.0 ± 0.8	42.5 ± 1.4**	37.3 ± 1.1†
Pa-$P_{ET}CO_2$ (mmHg)	0.06 ± 0.47	0.24 ± 0.49	0.01 ± 0.56

Compared with pre-insufflation ($P < 0.01$, **$P < 0.005$)
†Compared with 1 hour after insufflation (†$P < 0.01$)

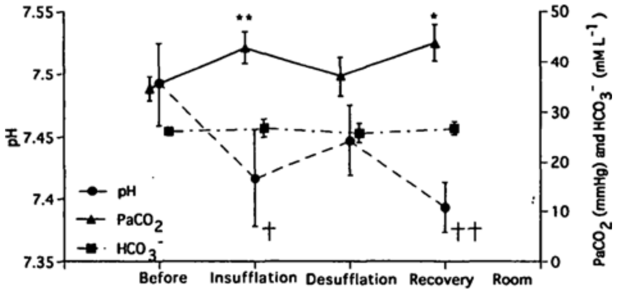

FIGURE 1 Changes in arterial blood pH, carbon dioxide tension ($PaCO_2$), bicarbonate concentration (HCO_3^-) during laparoscopic cholecystectomy. Pre: prior to insufflation, Insufflation: 1 hour after establishment of pneumoperitoneum, Desufflation: just after release of pneumoperitoneum, Recovery Room: 30 min after tracheal extubation. Significant differences compared with pre-insufflation (*$P < 0.05$, **$P < 0.01$, †$P < 0.005$, ††$P < 0.001$).

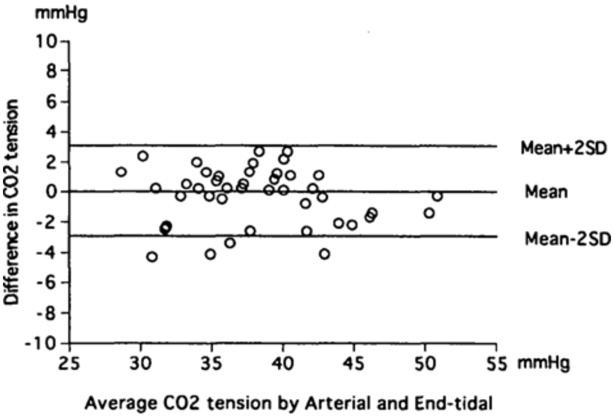

FIGURE 2 Scatterplot of differences (*y axis*) and average (*x axis*) of end-tidal and arterial CO_2 during laparoscopic cholecystectomy. *Horizontal lines* indicate the mean difference ± 2SD (95% confidence limits of agreement) between end-tidal and arterial CO_2.

weight, sex, anaesthesia or operation time between laparoscopic and open cholecystectomy patients.

Peritoneal insufflation decreased the dynamic lung compliance compared with pre-insufflation and post-desufflation values (Table II). Both peak and plateau inspiratory pressures were increased with insufflation compared with baseline. The expiratory tidal volume was not decreased with insufflation.

All patients maintained adequate oxygen saturation during the study. However, arterial and end-tidal carbon dioxide tensions increased during insufflation compared with pre-insufflation and decreased with desufflation. Blood gas analysis revealed a decrease in arterial pH and an increase in arterial carbon dioxide tension ($PaCO_2$) with insufflation compared with pre-insufflation. These values returned toward normal after desufflation, but were again abnormal in the recovery room (Figure 1). Bicarbonate concentration was constant throughout the study. Increases in individual patient $PaCO_2$ measurements were accompanied by corresponding increases in simultaneously obtained end-tidal CO_2 measurements ($r = 0.937$, $P < 0.0001$). Results with the Bland-Altman method are shown in Figure 2. The mean difference between end-tidal and arterial CO_2 was 0.3 ± 0.3 mmHg (range −4.3 to 2.7). There was a high negative correlation between the perioperative change in pH and $PaCO_2$ ($r = -0.789$, $P < 0.0001$).

The $PaCO_2$ in the recovery was higher in patients who had undergone laparoscopic cholecystectomy than in those who had an open cholecystectomy (44.6 ± 1.1 vs 38.4 ± 1.0 mmHg, $P < 0.0004$).

Discussion

This study shows that dynamic lung compliance decreased and arterial as well as end-tidal PCO_2 increased during insufflation. The values returned to baseline after release of pneumoperitoneum but the $PaCO_2$ increased again in the recovery room to levels which were higher than those seen after open cholecystectomy.

During laparoscopic procedures, pneomoperitoneum may cause respiratory embarrassment due to the mechanical effect of the increased intra-abdominal pressure.[7,8] Dynamic lung compliance was reduced by about 40% with increased peak inspiratory and inspiratory plateau pressures during insufflation. Abdominal distension may impede the movement of diaphragm and restrict lung expansion. The increase in airway pressure we observed in the presence of adequate muscle relaxation reflects the decrease in compliance associated with the elevated intra-abdominal pressure.[3] We assume that the decrease in dynamic lung compliance is due to cephalad shift of the diaphragm caused by the insufflated gas during the procedure.[9]

With the increased intra-abdominal pressure and the cephalad shift of the diaphragm, increased airway pressures are needed to maintain a constant minute ventilation. In this study, the value of the expiratory tidal volume decreased by 50 ml at insufflation. The circuit losses from stretching can be 6 $ml \cdot H_2O^{-1}$.[10] Thus, with an airway pressure of 40 mmHg, 240 ml or more may be lost due to circuit distension. In a small patient with a high intra-abdominal pressure during laparoscopy, this may exceed the tidal volume. Loss of about 50ml per breath is equal to 500 $ml \cdot min^{-1}$ cumulative loss which, together with the calculated loss due to tube distension, may cause insufficient ventilation. This may result in CO_2 retention. Therefore, it is important to use continuous in-line spirometry for routine intraoperative monitoring during laparoscopic cholecystectomy.

Although laparoscopic cholecystectomy is purported to be minimally invasive,[11-13] uptake of CO_2 from the pneumoperitoneum can cause clinically relevant hypercapnia. In this prospective study, monitoring of respiratory mechanics did not reveal a change in the expiratory tidal volume. Hence, we concluded that the carbon dioxide retention we observed was mainly the result of the pneumoperitoneum. Associated with this hypercapnia was a decrease in arterial pH. These changes in pH and $PaCO_2$ were well correlated. Because bicarbonate concentration did not change throughout the study, the decrease in pH was not metabolic. Blobner *et al.* have also reported an increased CO_2 load (40%) during pneumoperitoneum, which was felt to be caused by resorption of CO_2 from the abdominal cavity.[4] Thus the need for careful monitoring of respiratory function, especially the extent of CO_2 retention is clear. However, one of the most controversial issues regarding the management of patients during laparoscopy is whether radial arterial cannulation should be performed to assess the degree of hypercapnia and the effectiveness of oxygenation.[14,15] In our study of healthy patients with mechanical ventilation, end-tidal CO_2 and $PaCO_2$ were well correlated. The mean difference between end-tidal and arterial PCO_2 was also small in our study. These results suggest that end-tidal PCO_2 monitoring is sufficient in this patient population. In contrast, patients with cardiac or pulmonary disease have exhibited differences between $PaCO_2$ and end-tidal PCO_2.[15] Therefore, radial artery cannulation for frequent arterial blood gas analysis should be considered in patients with preoperative cardiopulmonary disease.

Both $PaCO_2$ and pH immediately recovered to preinsufflation values with the release of the pneumoperitoneum. Following the successful completion of the laparoscopic cholecystectomy, neuromuscular blockade was reversed with neostigmine (2 mg) and atropine (1 mg), which was followed by tracheal extubation. Of particular interest was the observation that $PaCO_2$ was increased and pH was decreased again in the recovery room. In addition, the $PaCO_2$ in the recovery room was higher in patients who underwent laparoscopic than after open cholecystectomy. This indicates that the hypercapnia induced by pneumoperitoneum persists after extubation as the carbon dioxide is gradually eliminated from the tissue. In our group, the operative time was long, almost double that noted in a recent report.[17] This prolonged operative time might increase absorbed CO_2 during insufflation, which may influence the second increase of CO_2 in the recovery room. Although laparoscopic cholecystectomy has many advantages, particularly minimal effects on respiratory function,[18] diaphragmatic function was inhibited in the early postoperative period.[19,20] Impaired diaphragmatic function immediately after laparoscopic surgery may also cause hypercapnia early in the recovery room period. Furthermore, subcutaneous emphysema, which is one of the most important complications, can increase end-tidal PCO_2.[21] Fortunately, we could find no emphysematous complication during this study. In the anaesthetic management of the laparoscopic cholecystectomy, hypercapnia after tracheal extubation must be considered.

In summary, insufflation of the abdominal cavity with carbon dioxide during laparoscopic cholecystectomy has several consequences including decreased lung compliance, decreased expiratory minute volume, and high peak airway pressures. Hypercarbia and acidosis were due to (1) absorption of CO_2 via the peritoneum, (2) decreased lung compliance and (3) insufficient ventilation. The hypercarbia and acidosis may persist in the recovery room. The anaesthetist should carefully observe and document respiratory mechanics and arterial blood gases in these patients.

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