Ventilatory requirements during laparoscopic cholecystectomy

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The purpose of this clinical study was to determine: (1) the increase in minute ventilation required to maintain preinsufflation arterial carbon dioxide tension ($PaCO_2$) during laparoscopic cholecystectomy, and (2) whether end-tidal PCO_2 ($PETCO_2$) can be used as an index of $PaCO_2$ and, therefore, of the adequacy of minute ventilation during the pneumoperitoneum. We measured $PaCO_2$, $PETCO_2$, expired minute volume (V_{exp}) standardized for body surface area (SA), airway and intra-abdominal pressure (Paw, Pabd) during general anaesthesia for laparoscopic cholecystectomy just before and 30 min after the creation of a CO_2 pneumoperitoneum in 28 healthy (ASA class 1 and 2) consenting adults. They were in the reverse Trendlenburg position (20°) with a 5° lateral tilt. Expired minute volume was increased from 3.75 (SEM ± 0.12) to 4.19 (0.15) L · min^{-1} · m^{-2} to maintain $PaCO_2$ close to control levels: 38.9 (0.8) vs 40.1 (0.6) mmHg 5.19 (0.1) vs 5.35 (0.08) kPa). In most of the patients (23/28), $PETCO_2$ was less than 41 mmHg with a correlation between $PaCO_2$ and $PETCO_2$. In ten of these patients, $(Pa-PET)CO_2$ was greater than the normal range. In 5/28, $(Pa-PET)CO_2$ was negative. The "driving pressure" (Paw-Pabd) increased from 8.7 (1.0) to 10.4 (1.1) cm H_2O, without any correlation between the increase in Paw-Pabd and that in $\dot{V}exp$. The results indicate the need for extra ventilatory requirement during laparoscopy and that $PETCO_2$ is an imperfect index of $PaCO_2$ under these circumstances.

Cette étude clinique visait à déterminer: (1) l'augmentation de ventilation-minute nécessaire à maintenir au niveau préinsufflation la pression artérielle en gaz carbonique ($PaCO_2$) pendant la cholécystectomie laparoscopique; (2) si la mesure de la $PaCO_2$ télé-expiratoire ($PETCO_2$) peut être utilisée comme un indicateur de la $PaCO_2$ et en conséquence de l'efficacité ventilatoire pendant le pneumopéritoine. Nous avons mesuré la $PaCO_2$, la $PETCO_2$, le volume-minute expiré ($\dot{V}$ exp) normalisé à la surface corporelle, la pression sur les voies respiratoires et la pression abdominale (Paw, Pabd) pendant l'anesthésie générale administrée pour cholécystectomie par laparoscopie immédiatement avant et 30 minutes après la création du pneumopéritoine avec du CO_2 chez 28 adultes non tarés, consentant et classés ASA 1 et 2. Ils étaient en position Trendélembourg inversé (20°) avec une inclinaison latérale de 5°. Nous avons augmenté le volume expiré de 3,75 (SEM ± 1,12) à 4,19 (0,05) L · min^{-1} · m^{-2} de façon à maintenir la $PaCO_2$ près du niveau des contrôles: 38,9 (0,8) vs 40,1 (0,6) mmHg. Pour la plupart des patients (23/28), la $PETCO_2$ fut inférieure à 41 mmHg avec une bonne corrélation entre $PaCO_2$ et $PETCO_2$. Pour dix de ces patients, la $(PaCO_2-PETCO_2)$ dépassait les valeurs normales. Chez 5/28, la $(Pa-PET)CO_2$ était négative. La pression effective (Paw-Pabd) a augmenté de 8,7 (1,0) à 10,4 (1,1) cm H_2O sans corrélation entre l'augmentation de la différence Paw-Pabd et celle de $\dot{V}_{exp}$. Ces résultats démontrent qu'il faut augmenter la ventilation pendant la laparascopie et que la $PETCO_2$, dans ces circonstances, ne réflète pas toujours la $PaCO_2$.

Key words

CARBON DIOXIDE: hypercontia, measurement, tension, arterial, end-tidal;
SURGERY: laparoscopy;
VENTILATION: carbon dioxide tension.

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Laparoscopic cholecystectomy has gained acceptance because of the minimal postoperative morbidity and markedly reduced hospital stay.[1] However, the pneumoperitoneum, which may be as much as 25 to 30 L CO_2 in the first 30 min, can have undesirable cardio-respiratory effects, particularly in patients with pulmonary disease in whom severe respiratory acidosis has been reported.[2] If cardiac output is reduced, as reported in one study,[1] adverse effects on CO_2 homeostasis and oxygenation can occur. Also, insufflation of CO_2 is an increased "load" on ventilation due the volume of CO_2 added by transperitoneal absorption and to the increased abdominal volume impeding diaphragmatic descent and reducing total compliance, even though the reverse Trendlenburg position may reduce the effects of the increased abdominal volume. Appropriate measures, in particular increasing

minute ventilation, must be taken to counteract the mechanical and ventilatory effects. During pelvic laparoscopy in the Trendlenburg position, a 20–30% increase in minute volume was necessary to maintain normocarbia.[3] The purpose of this study was to determine the increase in minute ventilation needed to maintain $PaCO_2$ constant and the extent to which $PETCO_2$ can be used as a reliable, non-invasive index of $PaCO_2$.

TABLE Biometric data

	Range	*Mean*	*SE*
Age (yr)	23–85	48.0	3.4
Weight (kg)	40–115	71.4	3.5
Height (m)	1.35–1.8	1.63	0.16
Surface area (m^2)	1.27–2.40	1.70	0.05

SA = Square root of {[weight (Kg) × height (cm)]/3600}.

Methods

The sample consisted of 28, ASA physical class 1 and 2, healthy adults (24 women, 4 men). The patients gave their verbal consent to be included in the study which was approved by the hospital's Research Ethics Committee. The biometric data are given in the Table.

The anaesthetic technique was similar in all cases and consisted of narcotic premedication, thiopentone induction, tracheal intubation facilitated by succinyl choline and maintenance with N_2O:O_2 and enflurane or isoflurane in a partial rebreathing system with CO_2 absorption. Adequate muscle relaxation was achieved by either atracurium or vecuronium. An Ohio V_5 anaesthesia ventilator was used for artificial ventilation. The measurements were made just before (approximately 15 min after induction of anaesthesia) and 30 min after the start of the pneumoperitoneum with the patients positioned in the reverse Trendlenburg (20°) position with a left tilt (5°).

The initial minute volume was based on 10 $ml \cdot kg^{-1}$ × 10 $breaths \cdot min^{-1}$ and adjusted to a $PETCO_2$ 32–36 mmHg (4.3–4.8 kPa). Expiratory minute volume was increased during insufflation by tidal volume increments sufficient to maintain $PETCO_2$ at or near control levels.

An Ohio 5250 RGM® (Respiratory Gas Monitor) monitor was used. This monitor, which receives regular maintenance checks and calibrations, and which has a built-in automatic autozeroing subroutine, measures inspired and expired gas concentrations, expired minute volume (tidal volume, frequency and minute volume) airway pressure, $PETCO_2$, peripheral oxygen saturation and heart rate. The side-stream sampling line for gas concentrations and $PETCO_2$ was placed between the micropore filter/water trap and a Ruben® one-way valve. The accuracy of the infra-red analyzer is 1% with minimal thermal drift. The spirometer reads tidal volume and minute volume (as the sum of tidal volumes during the preceding minute). The accuracy is ±40 ml per breath. The spirometer and Paw transducer (5% accuracy) line were placed distal to the valve. Since each patient acted as his own control, we felt that further calibrations were not needed.

Arterial blood was sampled anaerobically from a radial artery which was tested beforehand by Allen's test and the samples were packed in ice and analyzed within five to ten minutes by an Instrumentation Laboratory Analyzer Model 1312®. The analyzer is calibrated repeatedly every day with gas mixtures with known CO_2 concentrations and also has an automatic self-calibration system. A Storz Electronic Laparoflator 26012® was used to insufflate CO_2 at a constant abdominal pressure. The initial Paw was recorded when Pabd was 16 cm H_2O and insufflation had just begun. The statistical analyses were by Student's t test and least squares regression.

Results

Prior to insufflation, mean $\dot{V}$ exp/SA (in $L \cdot min^{-1} \cdot m^{-2}$) was 3.75 (SEM 0.12), mean $PETCO_2$ was 34.75 (0.8) mmHg (4.64 (0.1) kPa) and mean $PaCO_2$ was 38.96 (0.8) mmHg (5.19 (0.1) kPa). The mean difference between Paw and Pabd (with the latter held constant at 16 cm H_2O) was 8.7 (1.0) cm H_2O (0.1). Following insufflation with an average volume of 27.13 (2.48) L CO_2, during which tidal volume was increased in an attempt to maintain $PETCO_2$ constant, the mean values of the four measurements was compared with control:

$\dot{V}$ exp/SA: 4.19 (0.15)	$P < 0.001$
$PaCO_2$: 40.1 mmHg (0.6) [5.35 (0.08) kPa]	$P < 0.2$
$PETCO_2$: 36.7 mmHg (0.8) [4.89 (0.10) kPa]	$P < 0.002$

Figure 1 shows the $\dot{V}$ exp and $PaCO_2$ data. During the pneumoperitoneum, the relationship between $PaCO_2$ and $\dot{V}$ exp/SA was $PaCO_2 = 47.5 - (\dot{V}\text{ exp/SA} \times 1.76)$ ($r = 0.5$).

The difference between Paw and Pabd increased from 8.7 (1.0) to 10.4 (1.1) cm H_2O ($P < 0.05$). The anaesthesia ventilator was not a constant flow generator and did not have an inspiratory hold. Since we recorded minute volume, but not tidal volume, the values of Paw cannot be used to assess changes in pulmonary mechanics. Although mean Paw-Pabd and mean $\dot{V}$ exp increased, there was no correlation between the increases in the two measurements, with a marked disproportion in the change in the values between patients.

The relationship between $PETCO_2$ and $PaCO_2$ following insufflation is shown in Figure 2a. The statistical significance of the correlation between the two measurements in the entire sample is poor ($r = 0.34$). The data

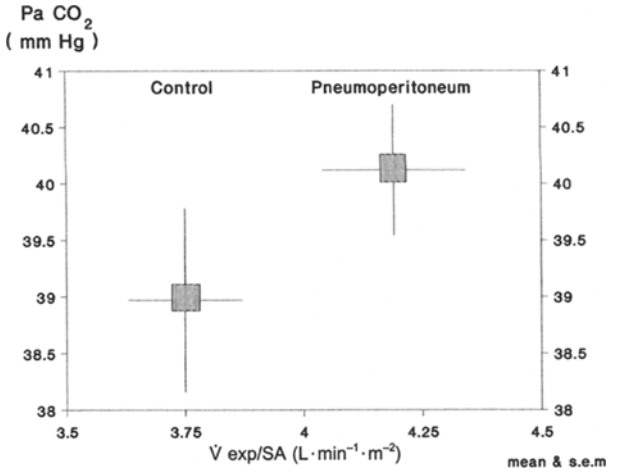

FIGURE 1 Expired minute volume and $PaCO_2$ Mean values (± SEM) values of the two measurements before and during the pneumoperitoneum are shown. Note that also the increase in minute volume was statistically different.

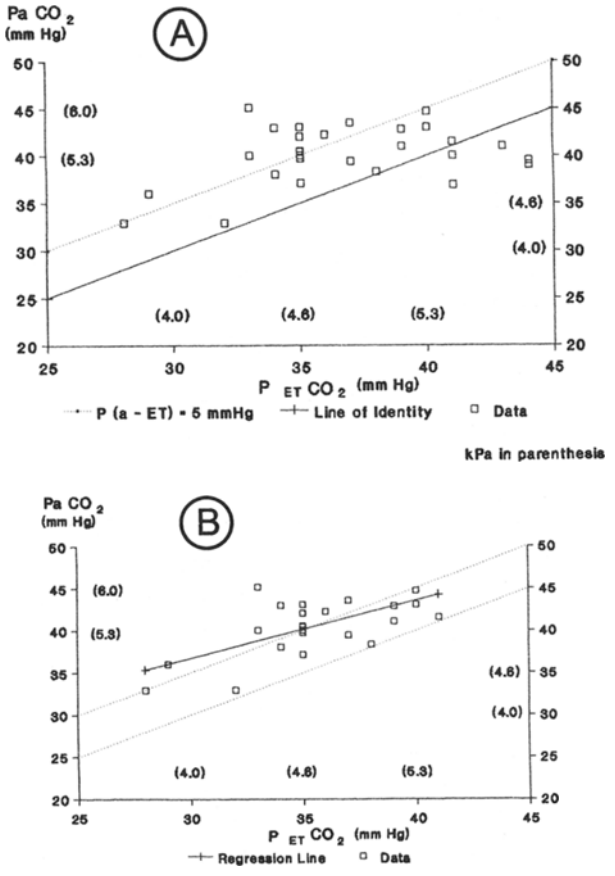

FIGURE 2 Pa and $PETCO_2$ during laparoscopy. (A) The values measured in the entire sample are shown together with the lines of identity and that of the upper limit of the normal difference between the two tensions. (B) The values in the sub-set of 23 patients with the line of best fit and the two lines in (A).

are best examined in their relationship to two lines: (a) the line of identity which joins identical Pa and $PETCO_2$s, that is whenever $Pa-PETCO_2 = 0$ (the dark line in the Figure) and (b) the upper range of the normal 3–5 mmHg difference (the interrupted line in the Figure). In five patients $(Pa-PET)CO_2$ was negative. The $PETCO_2$ in these patients was 41 mmHg (5.3 kPa) or more, in contrast with the rest, in whom $PETCO_2$ was 40 mmHg or less. These five patients required an average of 32.5 L CO_2, while the average volume insufflated in the rest was 25.2. The measured values of Pa and $PETCO_2$ in the 23 patients with $PETCO_2 < 41$ mmHg and the calculated line of best fit $\{PaCO_2 = 16.21 + [0.68 \times PETCO_2] (r = 0.63)\}$ are shown in Figure 2b together with the two lines mentioned above. It must be noted that in ten of these 23 patients $Pa-PETCO_2$ was larger than normal.

Discussion

Increasing minute ventilation by 12–16% (from 3.75 ± 0.12 to 4.19 ± 0.15 $L \cdot min^{-1} \cdot m^{-2}$) during laparoscopic cholecystectomy maintained the $PaCO_2$ close to the pre-insufflation levels. Under these circumstances $PETCO_2$ is not a satisfactory non-invasive index of $PaCO_2$ if $PETCO_2$ if $>$ 41 mmHg and large volumes of CO_2 are insufflated. Otherwise $PETCO_2$ provides a reasonable approximation of $PaCO_2$.

The elimination of the insufflated CO_2 depends on cardiac output, ventilation:perfusion ratios and alveolar ventilation. Cardiac output is the delivery system of CO_2 to the lungs for elimination and is a determinant of the overall ratio of ventilation to perfusion. The results of two studies[1,4] which measured the changes in cardiac output during laparoscopy are different. The first group measured cardiac output three times during the creation of the pneumoperitoneum and also during cholecystectomy in 25 patients. During the whole period, cardiac output was decreased in most (53–80%) patients by 20–26%, but the magnitude of the reduction decreased with time. The second group reported that cardiac output was unchanged during the procedure with a slight but statistically insignificant increase in cardiac output after approximately one hour in 16 healthy adults. Both groups used transoesophageal ultrasonography and there is no obvious reason for the difference in the results. In both studies, blood pressure and heart rate were not reduced. One would have expected a universal and sustained reduction in cardiac output due to the combined effects of anaesthesia, posture and the abdominal distention. It

is not known whether the CO_2 induces changes in plasma catecholamine concentrations. The data presented in the first report suggest that we should expect interpatient and intrapatient variability, particularly during the first 30 min of pneumoperitoneum.

The second component in the elimination of CO_2 is the ventilation: perfusion relationship which will be influenced by cardiac output and lung mechanics. Nothing is known about the latter during laparoscopic cholecystectomy. During pelvic (gynaecological) laparoscopy, total respiratory compliance and diaphragmatic excursion are reduced,[5] with a progressive decrease in compliance during insufflation.[6] The chest wall component of total compliance in particular is affected, to about 50% of the values before positioning and insufflation.[7] The pneumoperitoneum simultaneously decreases functional residual capacity (FRC) by 20%.[7] Because we did not keep a record of tidal volume in all patients, and did not have an end-inspiratory hold, we cannot use our Paw measurements to estimate altered compliance. The obvious differences between laparoscopic cholecystectomy and gynaecological laparoscopy are the duration of the procedures, the volume of gas insufflated and the surgical position. The reverse Trendlenburg position during anaesthesia increases FRC[8] and, presumably compliance. Until information about FRC and lung mechanics is reported, we assume that the changes in FRC and compliance during the two operations are different.

Alveolar (and hence end-tidal) PCO_2 is determined by the ratio of the determinants of $\dot{V}CO_2$/alveolar ventilation, where the former are CO_2 production and delivery to the lungs and the latter is minute volume minus dead space ventilation. Dead space ventilation is directly affected by cardiac output. Although an increase in minute ventilation does not necessarily cause a proportionate increase in alveolar ventilation, increasing minute volume is the most common method of handling the extra CO_2 absorbed through the peritoneum and the effects of any reduction in compliance. However, the increase in ventilation could decrease cardiac output and impair oxygenation[5,6,9] and alter the magnitude of dead space. A recent study[3] reported that adequate oxygenation and CO_2 homeostasis during gynaecological laparoscopy could be achieved by a 20–30% increase in minute volume. The measured additional CO_2 load to the lungs was about 40 ml · min^{-1} in the first 30 min. Other authors believe that absorption plays a major role.[2,4] The data in our report do not differentiate between the mechanical effect of the insufflated gas and transperitoneal absorption. The fact that our patients needed a more modest increase (12–16%) in minute ventilation in spite of the much larger volumes of CO_2 used is probably due to the different surgical positions. In the present report, the modest slope of 1.76 mmHg · L^{-1} minute volume indicates that other factors (probably cardiac output) markedly influence $PaCO_2$ during the pneumoperitoneum.

There are two reports which deal with ventilation and $PaCO_2$ during laparoscopic cholecystectomy. In the first,[2] the investigators measured minute ventilation, $PaCO_2$ and oxygenation during laparoscopic cholecystectomy in two groups of patients: 20 healthy adults were compared with ten patients of ASA class II and III. In the healthy group, in which the initial minute volume (100.0 (±22) ml · kg^{-1} · min^{-1}) was not increased, mean $PaCO_2$ and $P_{ET}CO_2$ increased from 30.7 mmHg (SD = 2.9) and 28.6 mmHg (3.3) respectively to 36.6 mmHg (5.1) and 30.7 (3.3). Statistical analysis of these data was not performed. The authors averaged the data collected for each patient over three hours and used that single value in the analysis. Thus, comparisons with our data are impossible. They noted that patients with severe lung disease develop severe respiratory acidosis ($PaCO_2$ = 46.0 (9.2) mmHg, pHa = 7.33 (0.06)) in spite of a 20–25% increase in minute ventilation. The other report was a study of arterial blood gases, $P_{ET}CO_2$ and haemodynamic variables in 16 healthy adults.[4] In spite of an unstated increase in tidal volume, which was initially 10–12 ml · kg^{-1} at an unspecified rate, the $PaCO_2$ at the end of the procedure was increased from 33.3 (0.7) to 43.7 (1.2) mmHg with pure respiratory acidosis. The peak increase occurred after one hour. The authors did not examine the relationship between $PaCO_2$ and $P_{ET}CO_2$ during insufflation although they did examine the values before and after the pneumoperitoneum. The authors concluded that $P_{ET}CO_2$ must be closely monitored and ventilation increased to maintain $P_{ET}CO_2$ below 35 mmHg (4.7 kPa). Also they suggested that the definite, although statistically insignificant, increase in cardiac output is the result of transperitoneal absorption of CO_2.

In the present study, $(Pa–P_{ET})CO_2$ was zero or negative in 5/28 patients (Figure 2a). The mean $P_{ET}CO_2$ in that subgroup was 42.3 mmHg (5.64 kPa) which is higher than that in the other 23 patients: 32.2 mmHg (4.29 kPa). A negative $(Pa–P_{ET})CO_2$ gradient has been reported in some normal patients during anaesthesia when large tidal volumes at slow rates were used. Negative $(Pa–P_{ET})CO_2$ gradients were also reported during pregnancy, following open heart operations and in children. The $P_{ET}CO_2$ is increased when cardiac output and CO_2 production (and delivery presumably) are increased and whenever a wide range of spatial and temporal mismatching of ventilation:perfusion exists. During large tidal volume ventilation, the very large breaths lead to (a) improved ventilation of dependent alveoli, akin to the opening of closed units, and (b) to faster emptying of slow lung units. It is tempting to use this to explain our observation but

a time lag exists between incremental and decremental changes in minute volume and changes in $P_{ET}CO_2$.[11] The half-life of a reduction in $P_{ET}CO_2$ is three minutes. It is possible that we did not record the correct $P_{ET}CO_2$ in these five instances and that later readings may have been lower in this subgroup, in which larger volumes of CO_2 were insufflated. During the surgical procedure, the repeated insertion and withdrawal of the surgical instruments and the suction-irrigation device result in loss of gas to the atmosphere. Thus, the use of large volumes does not mean that all the insufflated gas necessarily remains in the body. The first half-hour of the operation is the time during which the most severe changes in cardiorespiratory function occur.[11]

The correlation between Pa and $P_{ET}CO_2$ (Figure 2b) indicated that $P_{ET}CO_2$, if < 41 mmHg, can be used as an index of $PaCO_2$ with the proviso that the clinician be aware that an increased $(Pa-P_{ET})CO_2$, which reflects reduced cardiac output,[10] may exist in some of the patients.

In summary, we report that increasing minute ventilation by 12–16% during laparoscopic cholecystectomy in a healthy population sample maintained $PaCO_2$ at acceptable levels and that $P_{ET}CO_2$ monitoring should be used as an estimate of $PaOC_2$ with caution.

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