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Ventilatory effects of laparoscopic cholecystectomy

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Background: During laparoscopic cholecystectomy the arterial–end-tidal CO_2 gradient (Fa-$ETCO_2$) has been variously shown to be unchanged, increased, decreased or even negative. The goal of this study was to evaluate Fa-$ETCO_2$, and to determine the proper contribution of $\dot{V}ECO_2$ and $\dot{V}A$ in regard to the increase of $FETCO_2$.
Methods: Ventilatory patterns were studied in 15 ASA 1-2 patients (mean age±SD: 48.5±15.0) undergoing laparoscopic cholecystectomy, with intraperitoneal CO_2 insufflation limited to 12 mmHg, 15° head-up position, during general anaesthesia and controlled ventilation. The following were studied before, during, and after the pneumoperitoneum: $FaCO_2$, $FETCO_2$, nasopharyngeal temperature; dead space ventilation, and expired volumes using the Single Breath Test for CO_2. $\dot{V}A$ was calculated as the alveolar fraction of expired VT multiplied by the respiratory frequency.
Results: During pneumoperitoneum it is shown that: 1) Fa-$ETCO_2$ either decreases and becomes even negative (n=8) ($P<0.01$), or stays unchanged (n=7), but never elevates; 2) $\dot{V}ECO_2$ increases (peak value : + 22.6%) ($P<0.01$); 3) $\dot{V}A$ is unchanged, and 4) dead space ventilation, determined in 7 patients, remains unchanged.
Conclusion: We conclude that only exogenous CO_2 loading, and not $\dot{V}A$, can explain such increase in $FETCO_2$ and $FaCO_2$, in cases of limited CO_2 insufflating pressure in ASA 1-2 patients.

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Key words: Equipment: laparoscopy; measurement techniques: single breath test CO_2; monitoring: carbon dioxide, arterial minus end-tidal carbon dioxide gradient; ventilation: alveolar ventilation, dead spaces; surgery: laparoscopic cholecystectomy.

An increase in end-tidal CO_2 fraction ($FETCO_2$) is commonly observed with CO_2 intraperitoneal insufflation during laparoscopic cholecystectomy (1-3). This results from the diffusion of CO_2 from the peritoneal cavity to the blood and from the pulmonary CO_2 elimination ($\dot{V}ECO_2$) (3). A similar evolution of $FETCO_2$ and $\dot{V}ECO_2$ has been observed in ASA physical status 1-2 patients (3). This suggests that an increase in $\dot{V}ECO_2$ is the leading cause of rising $FETCO_2$, and that a change in alveolar dead space is unlikely. If this assumption is right, the arterial–end- tidal CO_2 gradient (Fa-$ETCO_2$) may stay unchanged, and changes in $FETCO_2$ may then represent changes in $FaCO_2$.

However, striking variations in Fa-$ETCO_2$ have been reported during CO_2 intraperitoneal insufflation in ASA 1-2 patients. This gradient was found to be either slightly increased (2, 4), nonsignificantly increased (5-7), or even negative (2). It is therefore possible that alveolar ventilation is reduced in some ASA 1-2 patients. To date, only Puri and Singh (7) and Tan et al. (1) have evoked this hypothesis during gynaecological procedures. They have found either an unsignificant increase or no change in physiological dead space (VD phys). However, these two studies have been performed during pelvic laparoscopy in a Trendelenburg position. Results observed in these conditions may differ from those during a laparoscopic cholecystectomy with an head-up position. On the one hand, the increases in FRC and respiratory compliance with head-up position during anaesthesia (8) are expected to minimize the effect of abdominal insufflation on ventilation; on the other hand, an increase in dead space ventilation is expected since a decrease in as much as 50% of the cardiac output may occur after abdominal insufflation

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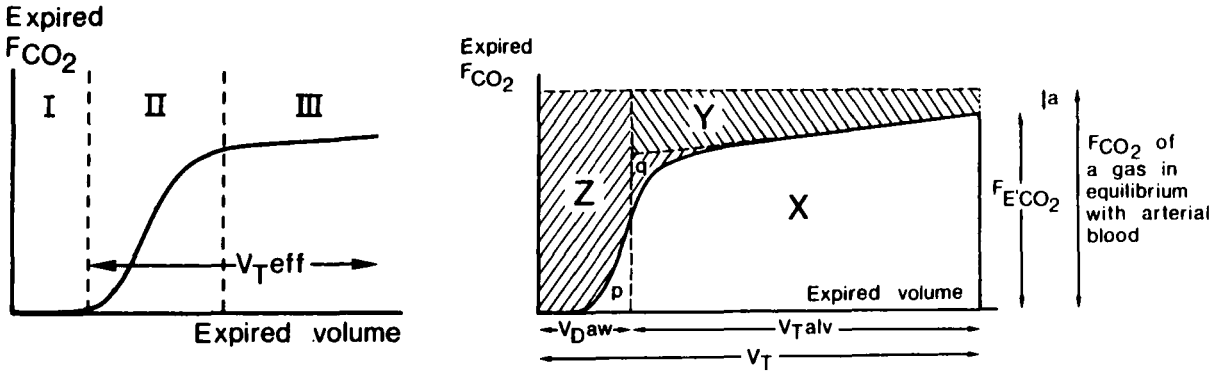

Fig. 1. Illustration of the single breath test for CO_2 modified by R. Fletcher et al. (9). (Reproduced with kind permission of the authors and publishers). Left side: The three phases of SBT-CO_2. Phase I represents CO_2-free gas from the airway dead space. Phases II and III together are the CO_2 - containing part of the breath, or effective tidal volume (VTeff). Phase II represents the transition between airway and alveolar gas. Phase III represents the CO_2 - rich gas from the alveoli.
Right side: $FaCO_2$ is added on the figure (dotted line) since it can represent FCO_2 of a gas in equilibrium with arterial blood. The a-value equals the arterial– end-tidal FCO_2 difference. Physiological dead space and its subdivisions can be determined. Triangles p and q are of equal area. Area X is the volume of CO_2 in the breath ($VTCO_2$), whilst areas Z and Y are defects in CO_2 elimination which represent wasted ventilation due to airway dead space (VDaw) and VDalv respectively. Volume of CO_2 expired in one minute ($\dot{V}ECO_2$) is equal to: $VTCO_2$×respiratory rate. Expired TV is the addition of VDaw and alveolar fraction of the TV (VTalv).

with an head-up position (5). The calculation of VD-phys in gynaecological surgery (1, 7) is also based on a questionable assumption that $PaCO_2$ represents alveolar CO_2 tension. In fact, alveolar ventilation has not been evaluated during laparoscopic cholecystectomy.

We have therefore undertaken this study in order to evaluate the Fa-$ETCO_2$ changes, and the ventilatory components of this gradient, during CO_2 intraperitoneal insufflation with a head-up position during laparoscopic cholecystectomy in ASA 1-2 patients. It is for this purpose that we have used the single breath test for CO_2 according to the method described by Fletcher et al. (9).

Patients and methods

General procedure

Fifteen ASA physical status 1-2 adult patients undergoing laparoscopic cholecystectomy with CO_2 intraperitoneal insufflation were selected on the basis of written informed consent, after approval by our local Consultative Committee for the Protection of Patients in Biomedical Research. A positive Allen test was an exclusion criteria. All patients were premedicated with hydroxyzine 100 mg orally 1h before surgery. General anaesthesia was induced with propofol 2.5 $mg.kg^{-1}$, vecuronium 0.1 $mg.kg^{-1}$, and fentanyl 3-6 $\mu g.kg^{-1}$. After tracheal intubation, ventilation was controlled (Servo Siemens Elema 900 D) with these constant settings throughout the study: tidal volume: 8 $ml.kg^{-1}$, respiratory rate: 12 min^{-1}, I : E ratio = 1 : 2. A total non-rebreathing anaesthetic circuit was used as well as an artificial nose ("Humid-vent filter", Gibeck Respiration, Chicago IL). Anaesthesia was maintained with continuous propofol infusion on the basis of 0.16 $mg.kg^{-1}.min^{-1}$, N_20: 0_2 = 50%, fentanyl and vecuronium as needed. Usual monitoring included nasopharyngeal temperature, circuit oximetry, pulse oximetry, and capnometry. Hypothermia was prevented with a warm water blanket.

Specific monitoring

A radial artery was cannulated for repeated blood gas analysis as well as for blood pressure monitoring. The pulse oximeter was placed on a finger related to radial artery vascularization. Atmospheric pressure in the operating room was determined with a barometer. The pneumoperitoneum was induced by the surgeon with a 12 mmHg electronically regulated intra-abdominal pressure using a CO_2 insufflator (Olympus - Walz Electronik Gmb H D-7271 Rohrdorf, Germany). The capnometer Siemens-Elema 930 was chosen because of its low time constant previously found to be 28 ms. The capnograph was calibrated before each operation according to manufacturer's recommendations regarding the N_20 concentration and atmospheric pressure.

The Servo Ventilator pneumotacograph and the Capnometer were connected to a Servo Computer Module 990, which enabled the analogic-digital transformation of the signals of expired volume and CO_2. These signals were instantaneously transmitted

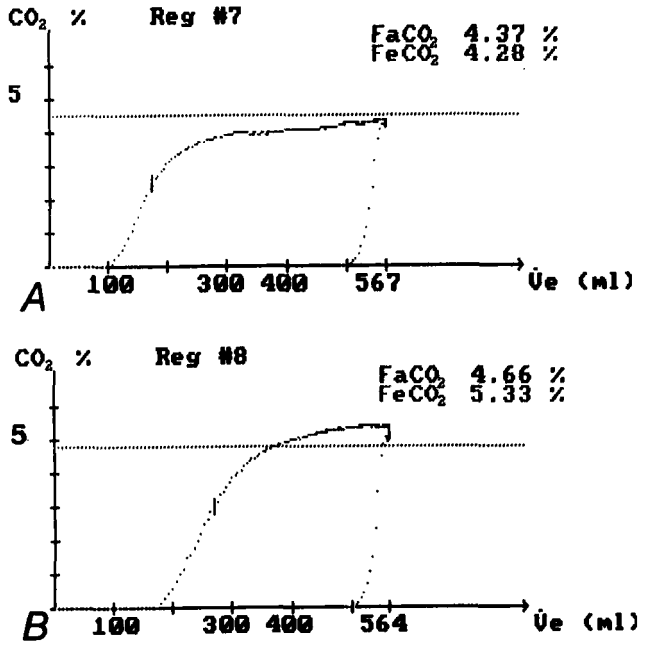

Fig. 2 . Typical records of expired CO_2 versus expired volume obtained from patient no. 4.
A) Fa-ETCO$_2$ is positive. Dotted line stands for FaCO$_2$ corrected for the temperature. This tracing was obtained before CO_2 insufflation in this patient.
B) After CO_2 insufflation Fa-ETCO$_2$ has become negative. Note that phase III is upsloping. This is interpreted as increases in scatter in time constants of alveoli and effect of CO_2 loading.

to a PC microcomputer (Siemens Nixdorf PCD-3NSX), which allowed an on-line representation of the expired CO_2 (vol %) versus VE (ml) relationship for each respiratory cycle, according to the principle of the single breath test for CO_2 (10), modified by Fletcher et al. (9). Curves representing the different recordings were printed out (printer Canon BJ10).

Principle of the single breath test for CO_2 (SBT-CO_2)
Briefly, the plot of expired CO_2 fraction against expired volume, applied to each single expiration, is the SBT-CO_2. It was first described by Aitken and Clarke-Kennedy (10), using six successive samples of expired gas. Fletcher et al. (9) did significant additions to the method in order to determine alveolar ventilation and dead space volumes. Sixteen pairs of values were used during the expiratory phase to obtain a more reliable curve of expired CO_2 versus expired volume. Fletcher's method to determine physiological dead space and its components is described in Fig. 1. It is shown that measured $PaCO_2$ by blood gas analysis must be added to the SBT-CO_2 curve to determine areas Z and Y. Area X represents the CO_2 volume in a single breath ($VTCO_2$). Assuming inspired CO_2 is zero in the non-rebreathing circuit, this method allows a determination of pulmonary CO_2 elimination in 1 min ($\dot{V}ECO_2$): $\dot{V}ECO_2 = VTCO_2 \times$ respiratory frequency. Measurements are based on the integration of signals of CO_2 concentration in the expired gas, and the expiratory flow.

Studied parameters
The following parameters were measured: $FETCO_2$, plateau inspiratory pressure, pH, $PaCO_2$ and nasopharyngeal temperature. The Fa-$ETCO_2$ was calculated with correction for temperature. The SBT-CO_2 curve was constructed with 16 pairs of values (expired volume and $FECO_2$) during each expiratory phase. After blood-gas analysis (Instrumentation Laboratory 482), corrected $PaCO_2$ was entered into the microcomputer, in order to determine the $FaCO_2$.

The following parameters were determined by the microcomputer's calculator according to the SBT-CO_2 principle (Fig. 1): expiratory VT or VTexp, area Z or VDaw, $VTCO_2$ and $\dot{V}ECO_2$, alveolar fraction of VT or VTalv, area Y or VDalv, and VD phys equals VDaw + VDalv. $\dot{V}A$ was also calculated, as VTalv×respiratory rate.

STPD and BTPS conditions
The pneumotacograph of the ventilator was calibrated at a standard dry temperature and pressure (STPD), whereas clinical measurements of volumes were taken at a body temperature saturated with water vapour (BTPS). According to Gravenstein et al. (11), the difference between these two conditions is given by F such as: volume of pulmonary gases = volume of expired gases×F.

$$F = \frac{273 + \text{body temperature}}{273 + \text{room temperature}} \times \frac{PB - PH_2O \text{ in the room}}{PB - PH_2O \text{ in the body}}$$

(273 is 273°K = 0°C; body temperature: nasopharyngeal temperature; PB: atmospheric pressure). Body temperature and PB were entered in the microcomputer before any measurement. The expired humidity was considered to be 47 mmHg throughout the study. A hygroscopic filter was used to keep a constant humidity.

Conversion of $PaCO_2$ to $FaCO_2$
A principle of the $SBTCO_2$ curve is to consider the $PaCO_2$ as the PCO_2 of a gas in equilibrium with arterial blood (see Fig. 1). To express the concentration of carbon dioxide in the alveoli, not as a partial pressure of all the gases exerting a pressure in the alveoli, but as a percentage, the calculator used a water

Table 1

Main clinical and surgical characteristics of the patients

Age (years)	48.5±15.0
Weight (kilograms)	64.1±8.9
Insufflation pressure (mmHg)	11.9±0.5
Duration of operation (min)	104±26
Duration of CO_2 insufflation (min)	92±29

Results are mean±SD

vapour of 47 mmHg and the barometric pressure given by the investigator.

Timing of the study

Studied parameters were recorded at the following time points: 30 min after anaesthetic induction and before CO_2 insufflation (T1); at the end of CO_2 insufflation (T2); 10 min after tilting in 15° head-up position (T3); 30 min after the beginning of CO_2 insufflation (T4); 10 min after the patient was once again in the horizontal position (T5); and finally 10 min after CO_2 deflation (T6).

Statistical analysis

Respiratory parameters were compared at different times using a one-way analysis of variance and the Wilcoxon test for nonnormal distributions. A linear regression analysis was used to study the relationship between variations in Fa-$ETCO_2$ and $\dot{V}ECO_2$. A value of $P<0.05$ was judged as significant.

Results

Main clinical and surgical characteristics of all patients are shown in Table 1.

Mean variations in $FETCO_2$, $FaCO_2$ and Fa-$ETCO_2$ in the 15 patients are shown in Table 2. During the pneumoperitoneum a significant increase in both $FETCO_2$ and $FaCO_2$ ($P<0.05$) and a significant decrease in temperature ($P<0.05$) were observed. The mean value of Fa-$ETCO_2$ became slightly negative after CO_2 insufflation, and was significantly decreased at T2 ($P < 0.05$). $FETCO_2$ was thus greater than $FaCO_2$.

Individual variations in Fa-$ETCO_2$ have shown two different trends. In 7 of 15 patients, Fa-$ETCO_2$ remained positive and unchanged after CO_2 insufflation, as shown in Table 3. An example of this type of $SBTCO_2$ curve is represented in Fig. 2A. This situation permits a graphic determination of VDalv. Table 3 shows that neither VDalv nor VDphys were changed in these patients after CO_2 insufflation. In 8 of 15 patients, Fa-$ETCO_2$ became negative and decreased significantly after CO_2 insufflation (Table 4).

Table 2

Variations in ventilatory parameters in 15 ASA 1-2 patients during laparoscopic cholecystectomy

Parameters	T1	T2	T3	T4	T5	T6
		Pneumoperitoneum				
	After induction	After insufflation	Head-up position	30 min after insufflation	Horizontal position	After deflation
$FETCO_2$ (vol %)	4.11 ±0.72	4.32 ±0.97	4.83* ±1.14	4.95* ±1.27	4.98* ±1.24	4.68 ±1.25
$FaCO_2$ (vol %)	4.39 ±0.52	4.29 ±0.82	4.71 ±0.92	4.91 ±0.95	4.97** ±0.86	4.80 ±0.94
Fa-$ETCO_2$ (vol %)	0.27 ±0.62	−0.04* ±0.72	−0.13* ±0.74	−0.03 ±0.84	−0.01 ±0.91	0.12 ±0.73
pH	7.42 ±0.05	7.41 ±0.07	7.38** ±0.08	7.36** ±0.07	7.35** 0.07	7.37** ±0.07
Temperature (°C)	36.3 ±0.7	36.1** ±0.7	35.9** ±0.6	35.6** ±0.6	35.4** ±0.6	35.6** ±0.7
Plateau inspiratory pressure (cmH_20)	10.1 ±3.6	15.4** ±4.7	16.6** ±4.8	15.8** ±4.0	15.4** ±4.0	12.4* ±5.5
$\dot{V}ECO_2$ ($ml.min^{-1}$)	168 ±38	172 ±40	194** ±47	201** ±44	206** ±55	192* ±51
$\dot{V}A$ ($l.min^{-1}$)	4.0 ±0.8	4.1 ±1.0	4.1 ±0.9	4.2 ±1.1	4.3 ±1.1	4.2 ±1.1

Variations in ventilatory parameters considered in 15 patients.
Abbreviations used: $FETCO_2$, end-tidal expired fraction in CO_2; $FaCO_2$, arterial fraction in CO_2; Fa-$ETCO_2$, arterial minus end-tidal expired fraction difference in CO_2 ; pH, arterial pH ; temperature, nasopharyngeal temperature; $\dot{V}ECO_2$, ventilatory CO_2 elimination; $\dot{V}A$, alveolar ventilation; Timing of measurements after the induction of anaesthesia (T1), after CO_2 insufflation (T2), 10 min after 15° head-up position (T3), 30 min after the beginning of CO_2 insufflation (T4), 5 min after the patient was once again placed in the horizontal position (T5), and 10 min after CO_2 deflation (T6).
Results are mean±SD
*$P<0.05$, **$P<0.01$, as compared to T1.

Table 3

Mean ventilatory parameters in the 7 patients with positive $Fa\text{-}ETCO_2$

Parameters	T1	T2	T3	T4	T5	T6
		Pneumoperitoneum				
	After induction	After insufflation	Head-up position	30 min after insufflation	Horizontal position	After deflation
$FETCO_2$ (vol %)	3.69 ±0.81	3.75 ±0.90	4.27 ±1.03	4.17 ±0.84	4.02 ±0.70	3.71 ±0.83
$FaCO_2$ (vol%)	4.39 ±0.62	4.31 ±0.87	4.69 ±0.91	4.78 ±0.50	4.83 ±0.46	4.39 ±0.45
$Fa\text{-}ETCO_2$ (vol %)	0.70 ±0.42	0.56 ±0.49	0.42 ±0.56	0.61 ±0.50	0.81 ±0.42	0.68 ±0.47
pH	7.44 ±0.05	7.44 ±0.06	7.40 ±0.07	7.39* ±0.05	7.38* ±0.05	7.41 ±0.06
Temperature (°C)	36.4* ±0.8	36.2* ±0.8	35.9* .±0.8	35.7* ±0.8	35.5* ±0.9	35.6* ±0.9
Plateau inspiratory pressure (cmH_20)	9.4 ±2.8	15.4* ±4.7	15.9* ±3.6	15.2* ±3.7	17.2* ±5.5	13.9 ±7.2
VDalv (ml))	76 ±42	71 ±50	63 ±43	66 ±41	77 ±27	64 ±29
VDphys (ml)	275 ±17	267 ±38	265 ±41	278 ±36	291 ±25	280 ±34
$\dot{V}ECO_2$ ($ml.min^{-1}$)	150 ±32	158 ±46	172* ±52	172* ±42	167 ±49	160 ±37
$\dot{V}A$ ($l.min^{-1}$)	4.2 ±0.5	4.5 ±0.8	4.5 ±0.8	4.7 ±1.1	4.8 ±1.2	4.7 ±1.2

Abbreviations used: $FETCO_2$, $FaCO_2$, $Fa\text{-}ETCO_2$, $\dot{V}ECO_2$ and $\dot{V}A$, as in Table 2 ; VDalv, alveolar dead space volume ; VDphys, physiological dead space volume.
Signification of times (T1.....T6) are as in Table 2.
Results in mean±SD
*$P<0.05$ compared to T1.

An example of this type of $SBTCO_2$ curve is represented in Fig. 2B. This situation precludes a graphic determination of VDalv. Subcutaneous emphysema caused by a parietal insuffflation was observed in 3 patients of this group. In these 3 patients, an increase in PaCO2 above 5.5 kPa (40 mmHg) ((6.1 - 7.5 kPa)

Table 4

Mean ventilatory parameters in the 8 patients with negative $Fa\text{-}ETCO_2$

Parameters	T1	T2	T3	T4	T5	T6
		Pneumoperitoneum				
	After induction	After insufflation	Head-up position	30 min after insufflation	Horizontal position	After deflation
$FETCO_2$ (vol%)	4.43 ±0.38	4.81 ±0.77	5.32* ±1.05	5.61* ±1.09	5.70* ±0.97	5.48* ±1.21
$FaCO_2$ (vol %)	4.37 ±0.45	4.26 ±0.84	4.72 ±0.99	5.11 ±1.21	5.09* ±1.12	5.16* ±1.13
$Fa\text{-}ETCO_2$ (vol %)	-0.03 ±0.55	-0.53* ±0.39	-0.63* ±0.49	-0.51 ±0.67	-0.62* ±0.57	-0.30 ±0.56
pH	7.41 ±0.04	7.38 ±0.07	7.36* ±0.08	7.33* ±0.07	7.31* ±0.06	7.34* ±0.07
Temperature (°C)	36.2 ±0.5	36.0* ±0.6	35.8* ±0.6	35.5* ±0.5	35.3* ±0.4	35.5* ±0.4
Plateau inspiratory pressure (cmH_20)	10.9 ±4.4	15.3* ±5.1	17.2* ±6.0	16.5* ±4.5	13.7 ±4.5	10.9 ±3.2
$\dot{V}ECO_2$ ($ml.min^{-1}$)	181 ±36	188 ±31	217* ±34	221* ±30	235* ±39	217* ±43
$\dot{V}A$ ($l.min^{-1}$)	3.8 ±1.0	3.8 ±1.1	3.8 ±1.0	3.8 ±0.9	3.9 ±0.8	3.8 ±0.8

Abbreviations used, and signification of times are as in previous Tables.
Results in mean ± SD
*$P<0.05$, **$P<0.01$, compared to T1.

(45-55 mmHg)) occurred during the pneumoperitoneum. Nevertheless, ventilatory settings were kept constant because hypercapnia was well tolerated by the patients.

$\dot{V}ECO_2$ was increased after CO_2 insufflation (Tables 2, 3, 4). The mean peak value of $\dot{V}ECO_2$ according to T1 values during the pneumoperitoneum was + 22.6% in the 15 patients (Table 2), + 14.6% in the 7 patients with positive Fa-$ETCO_2$ (Table 3), and + 29.8% in the 8 patients with negative Fa-$ETCO_2$ (Table 4). A significant but weak negative relationship was found between $\dot{V}ECO_2$ and Fa-$ETCO_2$ ($r=-0.51$, $P<0.001$). No significant variation in mean $\dot{V}A$ was met throughout the study (Table 2), at any Fa-$ETCO_2$ value (Tables 3 and 4).

Discussion

Our study mainly shows that the increase in $FETCO_2$ and $FaCO_2$ observed after CO_2 peritoneal insufflation for cholecystectomy in ASA 1 - 2 patients is only due to the CO_2 loading, without any change in alveolar ventilation. This finding is consistent with the lack of increase in Fa-$ETCO_2$ observed.

During intra-abdominal CO_2 insufflation under general anaesthesia (controlled ventilation), most studies have reported either a moderate increase (2) or a nonsignificant increment trend (5-7) in Fa-$ETCO_2$ in ASA physical status 1 or 2 patients. Results from these studies suggest that a moderate increase in alveolar dead space might exist during intraperitoneal CO_2 insufflation. A drop in cardiac output (5) during head-up position, and basal lung compression (12) in a Trendelenburg positioning, have been advocated as possible reasons. In contrast, two other results in the literature suggest an unlikely occurence of increase in alveolar dead space. A parallel evolution of $\dot{V}ECO_2$ and $PETCO_2$ was observed by Mullet et al. (3) and a negative gradient was noted in some patients by Wahba and Mamazza (2). This question brings potential drawbacks since $PETCO_2$ is usually monitored to explore the clinical changes in $PaCO_2$. In our patients, the mean trend in Fa-$ETCO_2$ was a slight decrease, and individual variations showed no increase in Fa-$ETCO_2$. This particular point conflicts with some previous studies showing an increase in Fa-$ETCO_2$ in ASA 1-2 patients (2, 5-7). A possible explanation for this discrepancy can be that blood gases must be corrected according to the patient's body temperature, otherwise the $PaCO_2$ will be systematically overestimated if the patient is colder than the measuring apparatus. This surgery is prone to overestimations of $PaCO_2$ because of intraperitoneal insufflation and irrigation, and various durations. For a maximal decrease of 0.9°C in body temperature (Table 2) in our patients, uncorrected blood gas values leads to an overestimation of 0.65 vol% (about 4.9 mmHg) in mean Fa-$ETCO_2$. Temperature correction must be used when samples in the gas and blood phases are compared, the more so as the influence of temperature is only 0.3%/°C for exhaled gas, whereas the solubility of CO_2 in the blood has a relatively high temperature coefficient (4.5%/°C) (13).

Fa-$ETCO_2$ had become negative in 8 out of 15 patients, after initiation of the pneumoperitoneum and head-up position. Such inversion of the gradient may be regarded as an intringuing phenomenon. In fact, it has already been observed, especially in healthy patients during laparoscopic cholecystectomy by an anaesthesiologist team (2), and during exercice by a physiologist team (14). Several mechanisms may explain the negative gradient observed during laparoscopy. First, the upsloping phase III on the capnogram (Fig. 2) may indicate inhomogeneity in the emptying rate of alveolar units. A more substantial contribution to alveolar exchanges could be given by low $\dot{V}/\dot{Q}$ units, in this case (9). On the capnogram (Fig. 2), it is hypothetized that the portion of phase III above the $FaCO_2$ might be due to expired CO_2 from dependent, late-emptying, well-perfused alveoli. The portion of phase III under $FaCO_2$ might be explained by a fast emptying of the non-dependent alveoli. During cholecystectomy with head-up position and intraperitoneal insufflation, increments in the spread of low $\dot{V}/\dot{Q}$ units are thought to occur, since a decrease in thoraco-pulmonary compliance has been demonstrated (15,16). A similar decrease in semi-static thoraco-pulmonary compliance occurred in our study since intraperitoneal insufflation was associated with an increase in plateau airway pressure at constant inspiratory tidal volume. This fact may be regarded as indirect evidence of increase in spread of low $\dot{V}/\dot{Q}$ units in our patients. Second, the ventilatory settings of the ventilator may also have promoted exhalation of CO_2 from these dependent low $\dot{V}/\dot{Q}$ units. We used a rather low respiratory frequency, a factor prone to promote emptying of alveoli with a low time constant; and large tidal volumes are another factor that improves the ventilation of dependent alveoli and a faster emptying of slow lung units. A third factor is the exogenous CO_2 loading. Several data suggest that an important CO_2 loading

favours an inversion in Fa-$ETCO_2$. Wahba and Mamazza (2) have found a nil or negative gradient when $PETCO_2$ was greater than 41 mmHg during laparoscopic cholecystectomy. Jones et al. (14) have shown that Fa-$ETCO_2$ is directly related to the CO_2 output during exercise. We found a similar result since a negative relationship exists between Fa-$ETCO_2$ and $\dot{V}ECO_2$ in our study. In addition, an extraperitoneal insufflation occurred in 3 patients.This has led to an hypercapnia associated with a negative Fa-$ETCO_2$ difference. These facts support the hypothesis that large volumes of insufflated CO_2 favour a negative Fa-$ETCO_2$. However, this hypothesis needs a persistent perfusion of the alveoli during the head-up position. In ASA 1-2 patients with intraperitoneal insufflation pressure near 12 mmHg and subsequent head-up position, Cunningham et al.'s data suggest that cardiac output is minimally decreased (17). Their clinical conditions were comparable to ours. The reduction in cardiac output can then be thought not to exceed 10% (17) in our patients. Finally, we think that the inversion of Fa-$ETCO_2$ observed in 8 of our patients may result from several causes: 1) an increase in spread of dependent low $\dot{V}/\dot{Q}$ units due to basal lung compression; 2) a large exogenous CO_2 loading and/or wound CO_2 insufflation; 3) an enhancement of CO_2 elimination by dependent alveoli due to a low respiratory frequency with large tidal volume, and a good alveolar perfusion.

No change in dead space ventilation was found. Variations in dead space ventilation during CO_2 intraperitoneal insufflation and head-up position were previously evaluated in experimental conditions only (18). Lister et al. (18) showed in pigs that the increase in $PaCO_2$ is explained by an increase in exogenous CO_2 loading, and not by an increase in dead space, when the intraperitoneal pressure is limited to 10 mmHg. Our results are consistent with this finding. However, the role of dead space ventilation was demonstrated with the use of higher intraperitoneal pressures (18). Previous clinical estimations of VDphys were done in two studies performed during gynaecological procedures with Trendelenburg positioning (1, 7). This position could be thought to induce more marked basal lung compression and increased venous return. However, these authors found that VDphys was unsignificantly increased or remained unchanged (1, 7). The calculation of VDphys was based in these studies on the hypothesis that $PaCO_2$ may represent $PACO_2$ during laparoscopy. This is a questionable point in cases of asynchronous emptying of alveoli and $\dot{V}/\dot{Q}$ scatter. The use of the capnograph may in fact not be sufficient to rule out a sequential inhomogeneity in lung regions. A sequential inhomogeneity in lung regions may exist even in a case of pseudo-plateau during phase III on the capnograph. Fletcher et al. (9) showed that only a tracing of expired CO_2 vs expired volume is accurate in this area. The reason for this is that as expiratory flow decreases with time, the late percentage of expiratory flow can occupy as much as half the time available to phase III. Then a pseudo-plateau situation may exist on tracing vs time, whilst phase III of the $SBTCO_2$ curve may be upsloping. $SBTCO_2$ with our patients showed an upsloping phase III most of the time during the pneumoperitoneum, indicating a $\dot{V}/\dot{Q}$ scatter. $PaCO_2$ may then not represent $PACO_2$. Our study shows that alveolar ventilation plays no role in the increase in $FETCO_2$ and $FaCO_2$ observed during laparoscopic cholecystectomy in healthy patients. This agrees with the stability in both VDalv and VDphys found in all 7 patients with a positive Fa-ETCO2. Finally, our study indicates that the CO_2 exogenously administered plays a far more important role in the increase of arterial and alveolar CO_2 than do changes in alveolar ventilation during laparoscopic cholecystectomy. This confirms what is strongly implied during gynaecological laparoscopy.

Significant clinical drawbacks are brought by the fact that CO_2 parietal insufflation was associated with an increase in $PaCO_2$ above 40 mmHg and a negative Fa-$ETCO_2$ gradient. It is suggested that in cases of elevated $PETCO_2$ during insufflation, the Fa-$ETCO_2$ difference could be of value when calculated according to body temperature. A reduced or even negative gradient could indicate either a large volume of insufflated CO_2, or an extraperitoneal insufflation. Ventilation was not increased during insufflation in our study because the aim was to determine the ventilatory effects of both insufflation and posture *per se*. In patients with an elevated $PaCO_2$, we kept constant the minute ventilation because acidaemia was clinically well tolerated.

One of the methodological problems encountered in the study was the inability to determine VDalv (and thus VDphys) in the 8 patients with a $FETCO_2$ greater than $FaCO_2$. As shown by Fig. 1, the linear upsloping of the phase III suggests that a sufficient increase in VTeff would result in a negative VDalv (area Y) (see Fig. 2). Negativity of VDalv is impossible, since the inhomogeneity in $\dot{V}/\dot{Q}$ ratios always implies the presence of VDalv (19). This is indeed a limitation of the $SBTCO_2$ method. Another problem

is the accuracy of the method. The accuracy of volume measurements by the pneumotacograph is 10% at low frequency, and total similarity with test gas mixtures was found for $FETCO_2$ after previous calibration at barometric pressure and $0_2 : N_20 = 50 : 50$ (20). Since several aspects of the method can affect the accuracy of volume measurements, we think it more appropriate to consider within-patient comparaisons at constant ventilor settings, rather than absolute values.

In conclusion, controlled ventilation with a tidal volume of 8 $ml.kg^{-1}$ and a respiratory frequency of 12 min^{-1} allows a good control of $FaCO_2$ during CO_2 pneumoperitoneum during laparoscopic cholecystectomy in healthy patients. In these conditions, $FETCO_2$ is often greater than $FaCO_2$, due to an inhomogeneity in $\dot{V}/\dot{Q}$ ratios associated with a greater CO_2 loading. An increase in $FaCO_2$ and $FETCO_2$ is associated with the pneumoperitoneum and proclive position of the patient. Such increases can only be due to the CO_2 loading with no change in alveolar dead space ventilation when an insufflating pressure of 12 mmHg is used.

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