General Anesthesia

Gastric air tonometry during laparoscopic cholecystectomy: a comparison of two $PaCO_2$ levels

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Purpose: Pneumoperitoneum can cause disturbances in acid-base balance and splanchnic perfusion. We studied the effect of ventilation on acid-base balance and gastric mucosal tonometric values in patients undergoing laparoscopic cholecystectomy.

Methods: Twenty-four patients (ASA I-II) were randomly allocated into two groups. In the fixed ventilation group, ventilation was constant allowing free increase in PCO_2, while in the constant CO_2 group end-tidal PCO_2 was fixed with ventilatory adjustment. Intraabdominal pressure was limited to 12 mmHg. Arterial acid-base balance, automated air tonometric variables and gastric mucosal to arterial PCO_2 gap were determined frequently from anesthesia induction until three hours postoperatively.

Results: During pneumoperitoneum, in the fixed ventilation group arterial PCO_2 changed from 5.0 ± 0.2 to 6.6 ± 0.4 kPa and pH from 7.43 ± 0.03 to 7.33 ± 0.04, tonometric PCO_2 from 5.1 ± 0.5 to 6.9 ± 0.4 and pH from 7.44 ± 0.04 to 7.33 ± 0.04. In the constant CO_2 group these variables remained at control levels ($P <$ 0.01 between groups). The PCO_2 gap remained unchanged without any differences between the groups. In the recovery room all measured variables were within normal range in both groups.

Conclusion: Despite inter-group differences in arterial and tonometric PCO_2 and pH values during CO_2 pneumoperitoneum, the patients did not develop splanchnic hypoperfusion detectable by air tonometric method, as indicated by normal PCO_2 gap in both groups throughout the study.

Objectif : Le pneumopéritoine peut causer des perturbations de l'équilibre acido-basique et de l'irrigation splanchnique. Nous avons étudié l'effet de la ventilation sur l'équilibre acido-basique et la tonométrie de la muqueuse gastrique chez des patients devant subir une cholécystectomie laparoscopique.

Méthode : Vingt-quatre patients (ASA I-II) ont été répartis au hasard en deux groupes. Dans le premier groupe, à ventilation fixe, la ventilation était constante et permettait une augmentation libre de la PCO_2, tandis que dans le second groupe, au CO_2 constant, la PCO_2 de fin d'expiration était fixe par ventilation adaptée. La pression intra-abdominale était limitée à 12 mmHg. L'équilibre acido-basique artériel, les variables automatisées de la tonométrie gazeuse de la muqueuse gastrique, pour calculer l'écart de PCO_2 artériel, ont été déterminés fréquemment depuis l'induction de l'anesthésie jusqu'à trois heures après l'opération.

Résultats : Pendant le pneumopéritoine, dans le groupe à ventilation fixe, la PCO_2 artérielle est passée de 5,0 ± 0,2 à 6,6 ± 0,4 kPa et le pH de 7,43 ± 0,03 à 7,33 ± 0,04; la PCO_2 tonométrique est passée de 5,1 ± 0,5 à 6,9 ± 0,4 et le pH de 7,44 ± 0,04 à 7,33 ± 0,04. Dans le groupe au CO_2 constant, ces variables ont conservé les valeurs témoins ($P <$ 0,01 intergroupe). L'écart de PCO_2 est demeuré inchangé et sans différence intergroupe. Dans la salle de réveil, toutes les variables mesurées étaient dans les limites de la normale pour les patients des deux groupes.

Conclusion : Malgré des différences intergroupes de PCO_2, artérielle et tonométrique, et de pH pendant le pneumopéritoine au CO_2, les patients n'ont pas présenté d'hypoperfusion splanchnique détectable par tonométrie gazeuse, comme l'indique l'écart normal de PCO_2 chez les patients des deux groupes.

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INTRAPERITONEAL insufflation of carbon dioxide during laparoscopic surgery leads to possibly harmful physiologic alterations, such as increased airway pressures and hypercarbia. Disturbances in acid-base balance have been suggested to be caused by increased intraabdominal pressure or by absorption of CO_2 from the abdominal cavity. Some of the observed acidotic changes might be of non-respiratory origin.[1,2] Circulatory disturbances, such as increase in central venous pressure,[3] development of venous stasis in lower limbs[4] or reduced cardiac index[5] might result in metabolic acidosis. Furthermore, pneumoperitoneum has been associated with disturbances in splanchnic micro-circulation depending on the level of intraabdominal pressure.[6]

Gastrointestinal saline tonometry was introduced in the late 1980s.[7] The method has been improved to a clinically feasible on-line monitoring of splanchnic perfusion by new automated air tonometry.[8] Results of tonometric measurements during laparoscopic cholecystectomy have been conflicting, varying from reports of splanchnic ischemia[9] or deterioration[10] to no detectable changes.[11]

We studied gastric air tonometry together with simultaneous measurement of arterial acid-base balance during laparoscopic cholecystectomy and immediate postoperative period. We compared the influence of two distinct $P_{ET}CO_2$ levels, obtained by ventilatory arrangements, on acid-base balance and tonometric variables.

Patients and methods

The protocol was approved by the IRB of the hospital, and written informed consent was obtained from 24 ASA I-II patients scheduled for elective laparoscopic cholecystectomy. They were randomly allocated using a sealed envelope method to one of the two study groups. Any respiratory disease or body mass index > 30 were taken as exclusion criteria.

Anesthesia

Patients were premedicated with 10 mg diazepam *po* one hour before surgery. Acetated Ringer's solution was given *iv*, 10 $ml \cdot kg^{-1}$ before surgery and 5 $ml \cdot kg^{-1} \cdot hr^{-1}$ throughout the operation. Following 0.2 mg glycopyrrolate, anesthesia was induced with 2 $\mu g \cdot kg^{-1}$ remifentanil and 2.5 $mg \cdot kg^{-1}$ propofol and maintained with 10 $mg \cdot kg^{-1} \cdot hr^{-1}$ propofol and initial 5 $\mu g \cdot kg^{-1} \cdot hr^{-1}$ remifentanil infusion. During surgery, the infusion rates were adjusted to maintain values of systolic arterial blood pressure and heart rate within ± 25 % from the control values.

Tracheal intubation was facilitated with 0.6 $mg \cdot kg^{-1}$ rocuronium. Neuromuscular block was maintained at 80-90 % level with 10 mg rocuronium increments, as evaluated using transcutaneous train-of-four stimulation of the ulnar nerve.

Ketorolac, 30 mg *iv*, was given during closure of trocar wounds. The patients were kept free of pain by giving meperidine, in increments of 10 mg, before transport to the recovery room. Postoperative pain was treated with oxycodone and nausea with droperidol given by recovery room nurses when needed.

Ventilation

The lungs were ventilated using a Sulla 909V® (Drëgerwerk AG, Lübeck, Germany) ventilator with a rebreathing circuit incorporating a CO_2 absorber. A continuous fresh gas flow of 4 $L \cdot min^{-1}$ (1.5 L O_2 and 2.5 L air), an inspiratory to expiratory ratio of 1:2 and zero end-expiratory pressure were applied. In both groups, respiratory frequency was set to 10 $breaths \cdot min^{-1}$ and inspiratory tidal volume adjusted to provide an end-tidal carbon dioxide tension ($P_{ET}CO_2$) of 4.5 kPa before the start of surgery. Thereafter, in the FV (Fixed Ventilation) group, ventilation was left unaltered with fixed ventilatory settings. In the CC (Constant end-tidal Carbon dioxide) group, instead, a constant $P_{ET}CO_2$ was maintained by adjusting the inspiratory tidal volume of ventilation until the end of anesthesia.

Surgery

Carbon dioxide pneumoperitoneum was introduced and maintained with a Laparoflator Electronic 3059® (F. M. Wiest Medizintechnik GmbH, Germany) device. Intraabdominal insufflation pressure was limited to 12 mmHg with computer control. After introducing trocars the patients were placed to a head up and right side up lateral tilt, 10° each. When the pneumoperitoneum was evacuated, the patients were returned to the horizontal position.

Measurements

After anesthetic induction a radial artery was cannulated, and a TRIP® Tonometry Catheter, 16F with stopcock (Datex-Ohmeda Div./Instrumentarium Corp., Helsinki, Finland), was introduced via the nasogastric route. Correct positioning of the catheter in the stomach was evaluated, first by estimating the distance from the nostril to the left upper abdominal quadrant, second by injecting air into the catheter while auscultating the abdomen, and third by aspiration of gastric contents. The catheter was connected to the Tonocap™ Monitor for automated air tonometry through the TRIP® Catheter Sampling Line (Datex-Ohmeda).

TABLE I Demographic, operative, anesthetic and postoperative pain treatment data of the patients as total numbers or mean ± SD.

	Group FV	*Group CC*
Sex (m / f)	5 / 7	3 / 9
Age (yr)	49.3 ± 12.8	49.3 ± 15.3
BMI ($kg \cdot m^{-2}$)	25.8 ± 3.2	25.5 ± 3.2
ASA status (I / II)	7 / 5	8 / 4
Pneumoperitoneum (min)	59.6 ± 19.2	76.0 ± 47.5
Propofol ($mg \cdot kg^{-1} hr^{-1}$)	9.7 ± 1.6	9.4 ± 1.0
Remifentanil ($\mu g \cdot kg^{-1} hr^{-1}$)	8.3 ± 2.2	7.6 ± 2.8
Meperidine (mg)	41.5 ± 14.0	38.1 ± 3.3
Oxycodone (mg)	10.8 ± 5.4	13.3 ± 6.5

Before surgery, gastric mucosal PCO_2 ($PgCO_2$) was determined three times at 10 min intervals, the last of which served as control value. Intraoperatively, the measurements were performed at 10 min intervals until 20 min after desufflation of pneumoperitoneum, and thereafter until three hours in the recovery room. The arterial blood samples, from which oxygen and carbon dioxide tensions ($PaCO_2$), pH (pHa), bicarbonate and base excess were determined, were analyzed simultaneously with the tonometric measurements. The gastric mucosal to arterial PCO_2 gradient, $P(g\text{-}a)CO_2$, i.e. the PCO_2 gap, was calculated as the difference between tonometric PCO_2 ($PgCO_2$) and arterial PCO_2. Tonometric pH, i.e., gastric mucosal pH (pHg), was calculated using a modification of the Henderson-Hasselbalch equation.[1 2]

In addition, from anesthesia induction until tracheal extubation, the following measurements were continuously performed (Datex-Ohmeda AS / 3™ Anesthesia Monitor): respiratory gas concentrations (inspiratory and expiratory CO_2 and O_2), respiratory rate, respiratory volumes (inspiratory and expiratory tidal and minute volume), airway pressures (peak inspiratory, end-inspiratory, end-expiratory), SpO_2, ECG, HR, invasive arterial blood pressures, as well as core temperature from rectum and skin temperatures from big toe, upper third of ventral antebrachium and middle finger.

In the recovery room, besides the arterial samples and tonometry as described above, ECG, HR, SpO_2, respiratory frequency and invasive arterial blood pressures were continuously recorded from 20 min to three hours following extubation.

Statistics

Statistical analyses were performed with Systat® statistical program and a freeware power calculation program. Patient group size calculations were based on an earlier study,[1 0] according to which a minimum of nine patients in each group would be needed to detect a 1.0 kPa PCO_2 gap difference with 95% sensitivity and 80% specificity. Thus, we decided to use a sample size of 12 patients in each group. Patient characteristics were compared with analysis of variance and Chi-square test. The effects of intervention (pneumoperitoneum) *vs* time and study group on repeated measurements were tested with multivariate repeated measures analysis. A posteriori analyses for repeated measurements within the groups *vs* between the groups were done using Tukey-type multiple comparisons test *vs* analysis of variance with Tukey's HSD (Honest Significant Difference) test. $P < 0.05$ was considered as statistical significance. Unless stated otherwise, all results are given as mean or mean ± SD.

Results

There were no differences between the two groups with regard to demographic, operative, or anesthetic data (Table I).

Ventilatory measurements during laparoscopic cholecystectomy are shown in Table II. During pneumoperitoneum, $P_{ET}CO_2$ increased in the FV group from 4.4 to 5.6 kPa ($P < 0.01$), while in the CC group it was maintained constant. In the FV group, expiratory tidal volume of ventilation remained unchanged. In the CC group maintenance of constant $P_{ET}CO_2$, instead, required an increase of 47% in the tidal volume, that is, from 467 ± 119 to 685 ± 148 ml. Peak inspiratory airway pressure increased 20% in the FV and 50% in the CC group. The differences in ventilatory variables between the groups were significant during pneumoperitoneum ($P < 0.01$).

Arterial and tonometric PCO_2 and pH values are shown in Table III. During pneumoperitoneum, $PaCO_2$ increased in the FV group from 5.0 to 6.6 kPa ($P < 0.01$), while in the CC group it remained within 5.1-5.3 kPa. In the FV group, $PgCO_2$ increased from 5.1 to 6.9 kPa ($P < 0.01$) and in the CC group from 5.2 to 5.8 kPa ($P < 0.05$). In the FV group, both pHa and pHg decreased ($P < 0.01$), whereas in the CC group the values remained at the control levels. Arterial and tonometric CO_2 and pH values differed between the groups ($P < 0.01$). Before pneumoperitoneum, the mean $P(g\text{-}a)CO_2$ gradient value in the FV or CC group was 0.0 and 0.1 kPa, respectively. At the end of pneumoperitoneum, the figures were 0.3 and 0.6 kPa, respectively (NS within and between groups)(Figure).

During cholecystectomy, arterial bicarbonate concentrations varied in the FV or CC group from 25.3 to 26.4 and from 25.1 to 25.6 $mmol \cdot l^{-1}$, and base excess from 1.6 to 0.7 and from 1.9 to 1.1 $mmol \cdot l^{-1}$, respectively (NS within and between groups).

TABLE II Ventilatory variables of the study groups during laparoscopic cholecystectomy.

	C	*P1*	*P2*	*P3*	*P4*	*PX*	*X1*	*X2*
$P_{ET}CO_2$								
FV	4.4 ± 0.2	5.0 ± 0.4*	5.4 ± 0.4†	5.5 ± 0.5†	5.6 ± 0.5†	5.6 ± 0.5†	5.5 ± 0.6†	5.3 ± 0.5†
CC	4.4 ± 0.2	4.4 ± 0.2 §	4.4 ± 0.2 §	4.3 ± 0.2 §	4.3 ± 0.3 §	4.3 ± 0.2 §	4.2 ± 0.1 §	4.4 ± 0.2 §
MV								
FV	4.7 ± 0.9	4.7 ± 0.8	4.7 ± 0.8	4.8 ± 0.8	4.8 ± 0.9	4.8 ± 0.9	5.0 ± 0.8	5.0 ± 0.9
CC	4.5 ± 1.1	5.4 ± 0.9	6.5 ± 1.5* §	6.6 ± 1.3* §	6.5 ± 1.4† §	6.8 ± 1.4† §	5.7 ± 1.3	5.2 ± 1.2
PIP								
FV	15.0 ± 2.2	18.7 ± 2.7*	18.8 ± 3.2*	18.8 ± 3.1*	18.8 ± 2.7*	18.7 ± 2.7*	15.2 ± 2.4	15.2 ± 2.4
CC	15.1 ± 2.4	21.7 ± 3.1† ‡	22.4 ± 3.0† §	22.2 ± 3.1† §	22.3 ± 3.0† §	22.2 ± 2.4† §	16.1 ± 2.3	15.1 ± 2.7

FV group with fixed ventilation, CC group with constant end-tidal CO_2.
$P_{ET}CO_2$ end-tidal PCO_2 (kPa), MV expiratory minute volume (L), PIP peak inspiratory airway pressure(cmH_2O).
C control before pneumoperitoneum (P), P1-4 10-40 min during P, PX start of exsufflation of P, X1-2 10-20 min after exsufflation of P.
‡$P < 0.05$, §$P < 0.01$ between the groups; *$P < 0.05$, †$P < 0.01$ within a group as compared with C.

TABLE III Arterial and tonometric PCO_2 and pH values during laparoscopic cholecystectomy.

	C	*P1*	*P2*	*P3*	*P4*	*PX*	*X1*	*X2*
$PaCO_2$								
FV	5.0 ± 0.2	5.7 ± 0.2†	6.1 ± 0.3†	6.3 ± 0.3†	6.4 ± 0.4†	6.6 ± 0.4†	6.3 ± 0.4†	6.2 ± 1.5†
CC	5.1 ± 0.5	5.2 ± 0.5 §	5.2 ± 0.4 §	5.2 ± 0.4 §	5.1 ± 0.4 §	5.2 ± 0.4 §	5.1 ± 0.3 §	5.3 ± 0.4 §
$PgCO_2$								
FV	5.1 ± 0.5	5.6 ± 0.5	6.2 ± 0.5†	6.5 ± 0.5†	6.8 ± 0.4†	6.9 ± 0.4†	6.9 ± 0.5†	6.7 ± 0.5†
CC	5.2 ± 0.3	5.4 ± 0.3	5.7 ± 0.4 ‡	5.7 ± 0.5 §	5.8 ± 0.6* §	5.8 ± 0.5* §	5.7 ± 0.5 §	5.7 ± 0.4 §
pHa								
FV	7.43 ± 0.03	7.39 ± 0.02	7.37 ± 0.03†	7.35 ± 0.03†	7.34 ± 0.05†	7.33 ± 0.04†	7.34 ± 0.04†	7.35 ± 0.04†
CC	7.43 ± 0.02	7.42 ± 0.03 §	7.42 ± 0.03 §	7.42 ± 0.03 §	7.42 ± 0.03 §	7.41 ± 0.03 §	7.42 ± 0.03 §	7.41 ± 0.02 §
pHg								
FV	7.44 ± 0.04	7.42 ± 0.03	7.38 ± 0.04†	7.35 ± 0.03†	7.33 ± 0.04†	7.33 ± 0.04†	7.32 ± 0.05†	7.33 ± 0.05†
CC	7.44 ± 0.02	7.42 ± 0.03	7.40 ± 0.04	7.39 ± 0.05 ‡	7.39 ± 0.05 §	7.38 ± 0.04* §	7.39 ± 0.05 §	7.40 ± 0.04 §

FV group with fixed ventilation, CC group with constant end-tidal CO_2.
$PaCO_2$ arterial, $PgCO_2$ tonometric PCO_2 in kPa.
C control before pneumoperitoneum (P), P1-4 10-40 min during P, PX start of exsufflation of P, X1-2 10-20 min after exsufflation of P.
‡$P < 0.05$, §$P < 0.01$ between the groups; *$P < 0.05$, †$P < 0.01$ within a group as compared with C.

TABLE IV Arterial and tonometric PCO_2 and pH values in the recovery room after cholecystectomy.

	R2	*R4*	*R6*	*R8*	*R10*	*R12*	*R15*	*R18*
$PaCO_2$								
FV	5.5 ± 0.9	5.3 ± 0.9	5.3 ± 1.1	5.6 ± 0.9	5.5 ± 0.7	5.6 ± 0.6	5.6 ± 0.5	5.5 ± 0.5
CC	6.1 ± 0.5	6.1 ± 0.5	5.9 ± 0.6	5.9 ± 0.4	6.0 ± 0.5	5.8 ± 0.5	5.9 ± 0.5	5.9 ± 0.5
$PgCO_2$								
FV	6.0 ± 1.0	6.1 ± 1.0	6.1 ± 0.9	6.3 ± 1.0	6.4 ± 0.9	6.5 ± 0.7	6.5 ± 0.7	6.6 ± 0.6
CC	6.2 ± 0.4	6.4 ± 0.4	6.5 ± 0.5	6.6 ± 0.4	6.7 ± 0.6	6.6 ± 0.7	6.4 ± 0.9	6.5 ± 0.7
pHa								
FV	7.39 ± 0.06	7.41 ± 0.08	7.41 ± 0.08	7.39 ± 0.06	7.39 ± 0.05	7.39 ± 0.03	7.39 ± 0.03	7.40 ± 0.03
CC	7.36 ± 0.03	7.36 ± 0.02	7.37 ± 0.03	7.37 ± 0.02	7.37 ± 0.02	7.38 ± 0.03	7.37 ± 0.03	7.38 ± 0.03
pHg								
FV	7.38 ± 0.06	7.37 ± 0.06	7.36 ± 0.06	7.36 ± 0.05	7.35 ± 0.06	7.34 ± 0.04	7.35 ± 0.04	7.34 ± 0.04
CC	7.37 ± 0.04	7.36 ± 0.03	7.35 ± 0.04	7.35 ± 0.03	7.34 ± 0.03	7.34 ± 0.04	7.36 ± 0.06	7.35 ± 0.04

FV group with fixed ventilation, CC group with constant end-tidal CO_2.
$PaCO_2$ arterial, $PgCO_2$ tonometric PCO_2 in kPa.
R2-18 20-180 min after endotracheal extubation.

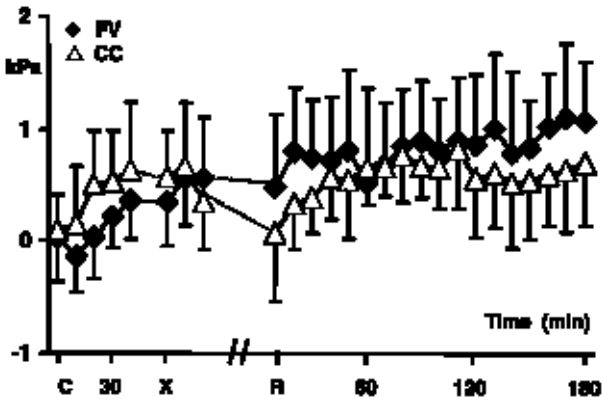

FIGURE Tonometric to arterial PCO_2 difference, the P(g-a)CO_2 gap (kPa), before, during and after laparoscopic cholecystectomy.
Filled diamond = the FV group with fixed ventilation.
Open triangle = the CC group with constant end-tidal CO_2.
C = control before pneumoperitoneum, X = exsufflation of pneumoperitoneum, R = arrival at recovery room.
The gap remained unchanged during surgery and in the immediate postoperative period, and there were no differences between the groups.

Hemodynamic variables showed similar courses in both groups (NS between the groups). Heart rate increased to its maximum after creation of the pneumoperitoneum (in the FV group to 77 ± 14 and in the CC group to 65 ± 13 beats·min^{-1}). The MAP increased in the FV group from the control 73 ± 12 to 96 ± 14 ($P < 0.01$) after creation of pneumoperitoneum, and in the CC group from 70 ± 6 to 90 ± 7 mmHg ($P < 0.01$). At the end of the pneumoperitoneum, the values of MAP were 87 ± 16 and 90 ± 11 mmHg in the FV and CC group.

The changes in body temperature were similar in the two groups. Rectal core temperature decreased, 0.7°C in the FV and 0.8°C in the CC group ($P < 0.01$ within groups). Skin temperatures increased after induction of anesthesia and remained at the elevated levels; arm by 2°C, finger and toe by 6°C ($P < 0.01$ within groups).

Postoperatively, during three hours in the recovery room, $P_{ET}CO_2$ remained within 4.9-5.6 kPa in both groups. Respiratory frequency varied between 12.6-15.1 breaths·min^{-1} in both groups. As shown in Table IV, in the FV group, $PaCO_2$ remained between 5.3-5.6, and in the CC group between 5.8-6.1 kPa. The corresponding values for $PgCO_2$ were 6.0-6.6 and 6.2-6.7 kPa. In the FV group, P(g-a)CO_2 varied between 0.5-1.1, and in the CC group between 0.3-0.8 kPa (Figure). Arterial pH remained between 7.36 and 7.41 and pHg between 7.34 and 7.37 in both groups (Table IV). The variations in $P_{ET}CO_2$, respiratory frequency, $PaCO_2$, $PgCO_2$, P(g-a)CO_2 (Figure), pHa or pHg were not significant within or between groups. Arterial bicarbonate levels remained in the FV and CC groups between 25.2-26.1 and between 25.8-26.2 mmol·l^{-1}, and base excess levels between 1.1-1.3 and 0.9-1.0 mmol·l^{-1}, respectively, NS. Heart rate varied between 64-80 beats·min^{-1}, and MAP between 80–112 mmHg (NS within and between groups).

Postoperative nausea occurred in three patients in the FV group and in one patient in the CC group.

Discussion

In this study, fixed ventilation during intraabdominal insufflation of CO_2 resulted in slight, clinically acceptable hypercarbia and decrease in pH. The changes were reflected in corresponding tonometric values. Both acid-base balance and tonometric values remained unchanged, when minute volume of ventilation was increased to maintain constant end-tidal PCO_2. Postoperatively, there were no differences between the groups in acid-base balance or gastric tonometric variables, the values of which showed normal physiological variation. This implicated rapid recovery of the intraoperative changes observed in the FV group. Despite these inter-group differences during pneumoperitoneum, the gastric mucosal to arterial PCO_2 gradient was similar in both groups. Nor did the gradient change in either group during the study period.

Splanchnic ischemia is defined as critical hypoperfusion of splanchnic organs causing anerobic metabolism. A decrease in tissue oxygen consumption, tension and development of anerobic metabolism was suggested to occur at a critical gastric mucosal to arterial PCO_2 gradient of 3.3 kPa.[13] In studies focused on examination of normal values for gastric tonometry, PCO_2 was considered normal up to 6.6 kPa and the PCO_2 gap up to 1.1 kPa.[14] Further, the lower limit of normal gastric mucosal pH was 7.32.[7,15] Thus, the PCO_2 gap and gastric mucosal pH values of our patients remained within these normal ranges. Furthermore, the gap between regional and arterial PCO_2 did not change, strongly suggesting the unharmful nature of the surgical procedure.

During pneumoperitoneum, in the FV group, where CO_2 was allowed to accumulate in the tissues, the increase in $PaCO_2$ was 1.6 kPa. At the end of the pneumoperitoneum, the patients were slightly hypercarbic with the maximum $PaCO_2$ of 6.6 kPa. Correspondingly, arterial pH decreased from 7.43 to 7.33. The decrease in arterial pH was of respiratory

origin. Through a 47% increase in minute ventilation the values of $PaCO_2$ and acid-base balance were maintained at the control level. Previously, during laparoscopic chlocystectomy, a 66% increase in minute ventilation was needed to keep $P_{ET}CO_2$ at preoperative control,[16] or a 48% increase to maintain $PaCO_2$ at the control.[17] There are also reports, where smaller increases were sufficient.[18] The need of ventilatory change might depend on many factors, such as the preoperative CO_2 balance of the patient, hemodynamic alterations and the exact aim of the adjustment.

In the recovery room, $PaCO_2$ and pHa levels of our patients remained within the normal range of 5.3-6.1 kPa and 7.36-7.41, respectively, and respiratory frequency between 12-15 breaths·min^{-1}. Despite differences during pneumoperitoneum the values of $PaCO_2$ in both groups were similar in the recovery room. Thus, the findings of our study do not support claims of excessive accumulation of CO_2 in the tissues during pneumoperitoneum followed by gradual postoperative elimination and delayed disturbances in acid-base balance.[1,19] Rather, our results are in agreement with those of Kazama *et al.*[20] demonstrating that excess CO_2 output evoked by pneumoperitoneum decreased steeply already during the first 30 min after evacuation of intraabdominal CO_2. However, we do not recommend unadjusted ventilation during laparoscopic surgery. In clinical practice, we strictly maintain normocarbia using either $P_{ET}CO_2$ or $PaCO_2$ as reference. The observed increase in airway pressure may be reduced by increasing frequency of ventilation instead of tidal volume.

During pneumoperitoneum, the occurrence and extent of metabolic acidosis might depend on the level and duration of intraabdominal pressure. In contrast to the present study with a pressure of 12 mmHg, metabolic acidosis was reported during laparoscopic cholecystectomy with the pressure maintained between 13-15 mmHg.[2] During prolonged laparoscopic surgery, the extent of metabolic acidosis seemed to be influenced both by the level and duration of intra-abdominal pressure. Accordingly, at 10 mmHg pressure, only slight metabolic acidosis of short duration was observed, whereas at 15 mmHg profound acidosis of long duration and increased level of plasma lactate became evident after 90 min.[21]

In the previous reports of tonometry during laparoscopic cholecystectomy intraabdominal pressure was 12 mmHg,[9] 12-13 mmHg[10] or 15 mmHg.[11] Surprisingly, with saline tonometry, very low gastric intramucosal pH (7.15)[9] was seen during 12 mmHg and normal pH during 15 mmHg intraabdominal pressure.[11] In the third paper, where air tonometry was applied, the lowest gastric mucosal pH, 7.24, occurred at 60 min during recovery.[10] Direct comparison with the values of our study is difficult as the PCO_2 gap was not included in these reports. Controversial results may have several reasons, such as various details of patients, anesthetic, surgical and measurement techniques. On the other hand, the PCO_2 gap of our patients was similar to that of patients undergoing open colon resection, where gastric mucosal to arterial PCO_2 gap remained < 1 kPa during the first hour of surgery. However, during succeeding hours the gap increased significantly. Exposure to ambient air might have contributed to the development of impaired intestinal perfusion.[22] Splanchnic blood flow, as assessed by estimated hepatic blood flow, was not affected in healthy patients during laparoscopic cholecystectomy with an intraabdominal pressure of 11-13 mmHg.[23]

Splanchnic perfusion is influenced by several local and systemic factors related to the patient and anesthetic and surgical techniques. Intraabdominal pressure might affect splanchnic perfusion directly or via hemodynamic changes, such as decreased cardiac output. Creation of pneumoperitoneum for laparoscopic cholecystectomy resulted in variable hemodynamic changes: cardiac index decreased more during 15 mmHg than 7.5 mmHg intraabdominal pressure.[24] A small increase was reported at 12 mmHg,[25] a substantial decrease[5] at 14 mmHg. Furthermore, the change for a particular patient seems to be unpredictable.[26] On the other hand, the PCO_2 gap of intensive care patients was not affected by variations of alveolar ventilation unless cardiac output changes were associated.[27] Recently, the Haldane effect was suggested as an alternative explanation for increase in $P(g\text{-}a)CO_2$ gradient in various circumstances. Changes of mucosal oxygen saturation influence the relationship between carbon dioxide content and PCO_2: at a given carbon dioxide content, mucosal PCO_2 increases with increasing mucosal oxygen saturation.[28] While gastrointestinal mucosal pH was originally suggested to constitute an index of the adequacy of splanchnic mucosal perfusion, the regional PCO_2 measured by tonometry may simply reflect the balance between the metabolic production of CO_2 in the tissue and the transport of CO_2 away from the tissue by the circulation.[29,30] Moreover, the regional PCO_2 will inevitably be influenced by arterial PCO_2. Thus, evaluation of tonometrically measured PCO_2 should always be performed against PCO_2 in the arterial blood.[27,31]

Two distinct ventilatory arrangements allowed us to compare the effects of two different levels of systemic PCO_2 on tonometric values. The results

obtained from frequent, simultaneous tonometric and arterial measurements showed the importance of relating the values of gastric mucosal PCO_2 to those of arterial blood. In the presence of constant gap, the higher $PgCO_2$ in the FV group compared with that in the CC group obviously reflected the level of arterial PCO_2 instead of implicating inadequate gastric perfusion. Indeed, mere statistical significance between some few sets of tonometric measurements do not justify any kind of straightforward conclusions about splanchnic circulation. Owing to the complex variety of systemic and local physiological changes occurring in the splanchnic circulation during anesthesia and surgery, a cautious attitude is needed when making observation statements based on tonometric data.[32]

Our study was carried out in healthy patients undergoing uneventful laparoscopic cholecystectomy. During prolonged and more complex laparoscopic surgery, however, especially patients with impaired cardiovascular or pulmonary function may well be in danger of disturbances in acid-base balance and regional circulation. Therefore, before judging the critical usability of online air tonometry, further investigations are required to clarify the potential of the device for early warning of decrease in gastric mucosal perfusion.

Our patients, undergoing elective laparoscopic cholecystectomy, did not show any detectable disturbances in splanchnic perfusion, as evidenced by the constant PCO_2 gap in normal range throughout the study in both groups. In the fixed ventilation group, which developed respiratory acidosis during pneumoperitoneum, tonometric evaluation of splanchnic perfusion entirely from the gastric mucosal PCO_2 and pH, without calculation of the PCO_2 gap, might have led to a false assumption of declining gastric intramucosal blood flow.

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