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Original Article

Effects of different inhaled oxygen concentration and end-expiratory positive pressure on Pa-etCO_2 in patients undergoing gynecological laparoscopic surgery

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Abstract: There is some disagreement about whether the $PetCO_2$ is reliable in predicting the $PaCO_2$ in laparoscopic procedures with CO_2 inflation. The aim of the present study is to measure the Pa-etCO_2 differences over time in healthy patients undergoing gynecological laparoscopic surgery with different ventilation methods. Methods: After intubation, patients were randomized into three groups. The patients in group A were ventilated with air/O_2 (FiO_2 = 50%) and supplied with PEEP (5cmH_2O). The patients in group B were ventilated with 50% oxygen while the patients in group C were ventilated with 100% oxygen. All patients were conducted with volume-controlled ventilation. $PetCO_2$ and $PaCO_2$ were measured at baseline, one minute after intubation, every 30 min thereafter and 5 minutes after deflation of pneumoperitoneum. Results: The differences in Pa-etCO_2 between groups A, B and C were insignificant one minute after intubation. The Pa-etCO_2 in group B and C significantly increased compared with that in group A at other time points after intubation. Conclusion: Oxygen concentration and positive end-expiratory pressure could affect Pa-etCO_2 in laparoscopic surgery patients.

Keywords: FiO_2, PEEP, $PaCO_2$, $PetCO_2$, laparoscopic surgery

Introduction

The relatively good hemodynamic and respiratory tolerance to abdominal CO_2 inflation has mostly been observed in healthy patients during short-lasting laparoscopic procedures. However, long-term laparoscopic surgery requires prolonged CO_2 pneumoperitoneum, which may facilitate CO_2 absorption and its accumulation in blood and tissues, eventually causing hypercapnia.

Anesthetists use end-tidal PCO_2 ($PetCO_2$) values routinely to monitor the adequacy of ventilation. However, there is some disagreement about whether the $PetCO_2$ is reliable in predicting the $PaCO_2$ ($PaCO_2$) in laparoscopic procedures with CO_2 inflation. In some reports, $PetCO_2$ is accepted as a reliable method of measuring $PaCO_2$ [1-4] in the absence of cardio-respiratory disease. But in other studies, the Pa-etCO_2 differences increase [5-7].

The clinical utility of $PetCO_2$ as a surrogate for $PaCO_2$ depends on the gradient between $PaCO_2$ and $PetCO_2$. This gradient depends primarily on the degree of alveolar dead space. Larson and Severinghaus reported that the administration of O_2 to awake sitting subjects led to an increase in Pa-etCO_2 [8]. As we know, inhaled oxygen concentration and positive end-expiratory pressure (PEEP) affect the volume of alveolar dead space in mechanical ventilation during general anesthesia [9, 10]. We hypothesized that, during pneumoperitoneum, $PetCO_2$ differs from $PaCO_2$ and the difference between $PaCO_2$ and $PetCO_2$ might be affected by oxygen concentration and PEEP. The aim of the present study is to measure the Pa-etCO_2 differences over time in healthy patients undergoing gynecological laparoscopic surgery with different ventilation methods.

Materials and methods

With approval by the Institutional Ethics Committee and written informed consent, 90 consecutive healthy, ASA physical status I~II, and non-smoking patients undergoing gyneco-

Table 1. Comparison of General Information of Three Groups (Mean ± SD)

Group	Age (yr)	Weight (kg)	Height (cm)	Duration of operation (min)
A (n = 30)	37 ± 7	59 ± 8	160 ± 6	122 ± 13
B (n = 30)	39 ± 8	55 ± 7	158 ± 5	130 ± 16
C (n = 30)	38 ± 6	56 ± 5	160 ± 4	120 ± 21

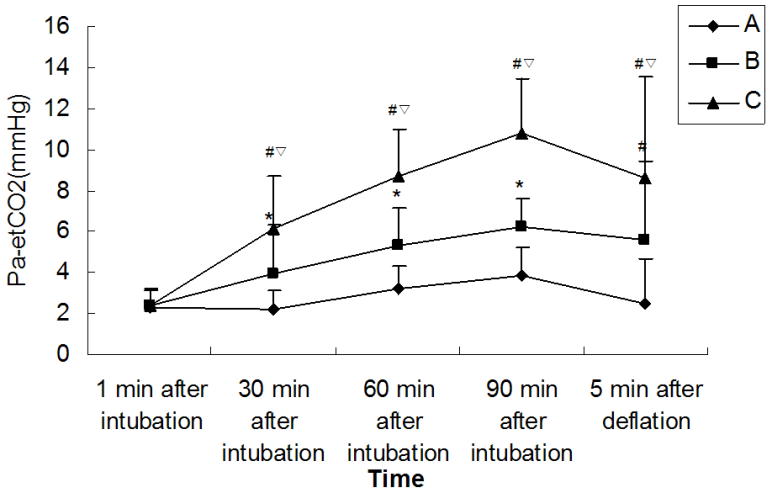

Figure 1. Comparison of Pa-etCO_2 at Different Time Points Among Three Groups. All values represent the Mean ± SD. *P < 0.05, #P < 0.01, compared with Group A at the same time point. ▽P < 0.05, △P < 0.01, compared with Group B at the same time point.

logical laparoscopic surgery were enrolled in the study. The exclusion criteria were signs of cardiopulmonary disease, acute infection and body mass index ≥ 30.

No pre-medication was used before anesthesia induction. Patients were monitored with ECG, pulse oximetry and non-invasive blood pressure measurement and received a pre-operative fluid challenge (Lactated Ringer, 10 ml kg^{-1}). Before anesthesia induction, a radial artery catheter was inserted to collect blood samples.

Anesthesia was induced with sufentanil (0.5 μg kg^{-1}) and propofol (2 mg kg^{-1}), and orotracheal intubation was facilitated by rocuronium (0.6 mg kg^{-1}). Anesthesia was maintained with propofol and remifentanil to obtain stable hemodynamic parameters (i.e., pulse rate and blood pressure variations < 20% of the pre-induction values).

After intubation, patients were randomized into three groups. The patients in group A were ventilated with air/O_2 (FiO_2 = 50%) and supplied with PEEP (5cmH_2O). The patients in group B were ventilated with 50% oxygen. The patients in group C were ventilated with 100% oxygen. All patients under volume-controlled ventilation by means of the Datex-Ohmeda machine (Aestiva/5; Datex-Ohmeda, USA) were adapted to obtain a $PetCO_2$ value between 4.0 and 5.5 kPa by adjusting minute ventilation up to an airway pressure limit (30cmH_2O). $PaCO_2$ was measured at baseline (before general anesthesia), one minute after intubation, every 30 min thereafter and 5 minute after deflation of pneumoperitoneum by using the blood gas analyser (i-STAT1, ABBOTT, USA). Heart rate, blood pressure, pulse oximetry, $PetCO_2$, airway pressure and minute ventilation were recorded at baseline (before general anesthesia), one minute after intubation, every 30 min thereafter and 5 minutes after deflation of pneumoperitoneum. The patient's body temperature was continuously monitored via nasopharynx and maintained at 36° to 37°C by external heating. The room temperature was maintained at 24° to 26°C.

During anesthesia, hydration was provided with Lactate-Ringer solution, 7.5 ml kg^{-1} h^{-1} and modified thereafter to obtain a diuresis ≥ 1 ml kg^{-1} h^{-1}. Intra-abdominal pressure was steadily maintained between 12 and 14 mmHg by a CO_2 inflator (Karl Storz, Germany). Following CO_2 inflation and till the end of surgery, patients were placed in a fixed 20° Trendelenburg position.

Statistical analysis

Quantitative data were presented as mean ± SD. Statistical analysis was conducted by using SPSS Version 13.0. A value of $P < 0.05$ was considered statistically significant.

Results

All patients enrolled in the study completed the protocol. The three groups did not differ with respect to age, weight, height and duration of operation (**Table 1**).

Compared with 1 minute after intubation, the difference of Pa-etCO_2 in group B and C obviously increased 30 minute after intubation. Among these groups, there were significant differences in Pa-etCO_2 60 minutes after intubation and thereafter (**Figure 1**).

Discussion

The number of laparoscopic gynecological procedures in clinical practice has increased rapidly because of their advantages that include minimal invasiveness, reduced pain, and fast recovery. Accurate evaluation of $PaCO_2$ is necessary in anesthesia management during the aforementioned surgery to assess ventilation efficacy and to avoid hypercapnia, which may lead to sympathetic excitation, hypertension, tachycardia, and other complications.

$PetCO_2$ cannot reliably estimate $PaCO_2$ under these circumstances because CO_2 inflated into the abdominal cavity may cause pulmonary atelectasis, resulting in decreased functional residual capacity and ventilation-perfusion mismatch [11]. Klopfenstein et al. [5] measured $PaCO_2$ and $PetCO_2$ simultaneously during laparoscopic colon surgery, with prolonged CO_2 pneumoperitoneum, and concluded that the correlation between $PaCO_2$ and $PetCO_2$ was inconsistent after the initiation of CO_2 inflation. Xue et al. [6] found that $PetCO_2$ was correlated with $PaCO_2$ only at baseline, before pneumoperitoneum, and underestimated $PaCO_2$ when CO_2 pneumoperitoneum was established. The results of our study indicate that the differences of Pa-etCO_2 increase with duration of operation extended.

In our study, inhaled 50% oxygen, after anesthesia induction, decreased Pa-etCO_2. We presume that high FiO_2 increases the volume of alveolar dead space and reduces vascular resistance in high perfusion alveolar areas, resulting in the redistribution of blood flow away from low perfusion alveolar areas as proposed by Larson [8]. Thus, the changes in Pa-etO_2 were partially due to the dilution effect of $PACO_2$ with gas from alveolar dead space.

A hyperoxia-induced reduction in cardiac output may partially explain an increase in Pa-etO_2. Anderson [12] reported that cardiac output decreased by 10.3% when FiO_2 was changed from 0.21 to 1.0. Johann [13] reported that cardiac output decreased by 10.6% after a change in FiO_2 from ≤ 0.6 to 1.0 in patients after coronary artery bypass surgery. The decrease in cardiac output increases Pa-etO_2 by causing the decrease in CO_2 elimination from alveoli and the redistribution of pulmonary blood flow. Although we did not detect any changes in heart rate and arterial pressure during our study, it is likely that a small decrease in cardiac output contributed to the increase in Pa-etCO_2.

Intrapulmonary shunt can lead to the increase in Pa-etO_2 as a consequence of the increase in $PaCO_2$ with venous admixture. Administration of PEEP reduced the differences of Pa-etCO_2 in our study. Development of atelectasis is a major cause for intrapulmonary shunt during general anesthesia [14]. Dantzker [15] reported that breathing pure O_2 induced absorption atelectasis within 9 min and that pulmonary shunt increased abruptly when FiO_2 increased above 0.8. Brismar [16] found that in chest computed tomography study, atelectasis rapidly developed in the dependent lung regions during the induction of general anesthesia, probably with pure O_2 preoxygenation, but it did not progress with time or increased FiO_2.

There are some limitations in our study. The first is that all patients went through preoxygenation with 100% oxygen, which is a standard clinical practice to ensure patient safety. Nevertheless, preoxygenation with 100% oxygen would result in the development of absorption atelectasis. Although an inflation of the lungs after induction to airway pressure of 3.92 kPa [17] or an application of PEEP of 0.98 kPa [18] can eliminate the atelectasis, we did not perform them. The atelectasis during the induction of anesthesia must have persisted, even if FiO_2 was changed. Secondly, all patients in our study were female.

In conclusion, oxygen concentration and positive end-expiratory pressure could affect Pa-etCO_2 in gynecologic laparoscopic surgery patients. The accuracy of $PetCO_2$ values as a surrogate for $PaCO_2$ should be adjusted according to ventilation methods.

Disclosure of conflict of interest

None.

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References

[1] Joris JL, Noirot DP, Legrand MJ, Jacquet NJ and Lamy ML. Hemodynamic changes during laparoscopic cholecystectomy. Anesth Analg 1993; 76: 1067-1071.

[2] Wahba RW, Beique F and Kleiman SJ. Cardiopulmonary function and laparoscopic cholecystectomy. Can J Anaesth 1995; 42: 51-63.

[3] Junghans T, Modersohn D, Dorner F, Neudecker J, Haase O and Schwenk W. Systematic evaluation of different approaches for minimizing hemodynamic changes during pneumoperitoneum. Surg Endosc 2006; 20: 763-769.

[4] Nguyen NT, Anderson JT, Budd M, Fleming NW, Ho HS, Jahr J, Stevens CM and Wolfe BM. Effects of pneumoperitoneum on intraoperative pulmonary mechanics and gas exchange during laparoscopic gastric bypass. Surg Endosc 2004; 18: 64-71.

[5] Klopfenstein CE, Schiffer E, Pastor CM, Beaussier M, Francis K, Soravia C and Herrmann FR. Laparoscopic colon surgery: unreliability of end-tidal CO2 monitoring. Acta Anaesthesiol Scand 2008; 52: 700-707.

[6] Xue Q, Wu X, Jin J, Yu B and Zheng M. Transcutaneous carbon dioxide monitoring accurately predicts arterial carbon dioxide partial pressure in patients undergoing prolonged laparoscopic surgery. Anesth Analg 2010; 111: 417-420.

[7] Hirvonen EA, Nuutinen LS and Kauko M. Ventilatory effects, blood gas changes, and oxygen consumption during laparoscopic hysterectomy. Anesth Analg 1995; 80: 961-966.

[8] Larson CP Jr and Severinghaus JW. Postural variations in dead space and CO2 gradients breathing air and O2. J Appl Physiol 1962; 17: 417-420.

[9] Yamauchi H, Ito S, Sasano H, Azami T, Fisher J and Sobue K. Dependence of the gradient between arterial and end-tidal P(CO(2)) on the fraction of inspired oxygen. Br J Anaesth 2011; 107: 631-635.

[10] Satoh D, Kurosawa S, Kirino W, Wagatsuma T, Ejima Y, Yoshida A, Toyama H and Nagaya K. Impact of changes of positive end-expiratory pressure on functional residual capacity at low tidal volume ventilation during general anesthesia. J Anesth 2012; 26: 664-669.

[11] Kazama T, Ikeda K, Kato T and Kikura M. Carbon dioxide output in laparoscopic cholecystectomy. Br J Anaesth 1996; 76: 530-535.

[12] Anderson KJ, Harten JM, Booth MG and Kinsella J. The cardiovascular effects of inspired oxygen fraction in anaesthetized patients. Eur J Anaesthesiol 2005; 22: 420-425.

[13] Harten JM, Anderson KJ, Kinsella J and Higgins MJ. Normobaric hyperoxia reduces cardiac index in patients after coronary artery bypass surgery. J Cardiothorac Vasc Anesth 2005; 19: 173-175.

[14] Bendixen HH, Hedley-Whyte J and Laver MB. Impaired Oxygenation in Surgical Patients during General Anesthesia with Controlled Ventilation. A Concept of Atelectasis. N Engl J Med 1963; 269: 991-996.

[15] Dantzker DR, Wagner PD and West JB. Instability of lung units with low Va/Q ratios during O2 breathing. J Appl Physiol 1975; 38: 886-895.

[16] Brismar B, Hedenstierna G, Lundquist H, Strandberg A, Svensson L and Tokics L. Pulmonary densities during anesthesia with muscular relaxation--a proposal of atelectasis. Anesthesiology 1985; 62: 422-428.

[17] Hedenstierna G and Rothen HU. Atelectasis formation during anesthesia: causes and measures to prevent it. J Clin Monit Comput 2000; 16: 329-335.

[18] Tokics L, Hedenstierna G, Strandberg A, Brismar B and Lundquist H. Lung collapse and gas exchange during general anesthesia: effects of spontaneous breathing, muscle paralysis, and positive end-expiratory pressure. Anesthesiology 1987; 66: 157-167.