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STUDY OF THE ASSOCIATION OF BLOOD CARBON DIOXIDE OUTPUT IN LAPAROSCOPIC CHOLECYSTECTOMYIN AT RIMS RAIPUR CHATTISGARH STATE

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ABSTRACT We studied patients undergoing elective laparoscopic cholecystectomy. Carbon dioxide output, oxygen uptake, respiratory exchange ratio (RER), expired minute ventilation (VE), deadspace to tidal volume ratio (VD/VT ratio) and arterial to end-tidal carbon dioxide partial pressure difference (PaCO2-PE'CO2) were determined before induction, and during anaesthesia, pneumoperitoneum and recovery. By controlling ventilatory frequency (f) every 1 min, PaCO2 was adjusted to concentrations before induction. Constant monitoring of end-tidal carbon dioxide partial pressure (PE'CO2) and intermittent measurement of (PaCO2-PE'CO2) (15-min intervals) were conducted to predict PaCO2). Carbon dioxide output and oxygen uptake decreased significantly from mean values of 83.5 (SEM 5.2), 101.6 (5.1) to 68.5 (4.2), 81.1 (4.6) ml min-1 m-2 (ATPS, P < 0.05) with sevoflurane anaesthesia, and RER did not change. During carbon dioxide pneumoperitoneum (intra-abdominal pressure 8 mm Hg), carbon dioxide output increased by 49% (102.4 (5.0) ml min-1 m-2) (P < 0.05) while oxygen uptake remained stable and RER increased from 0.84 (0.02) to 1.16 (0.03) (P < 0.05). It was necessary to increase VE during pneumoperitoneum by 1.54 times that during anaesthesia to maintain individual PaCO2 values constant. After removal of carbon dioxide from the abdominal cavity, the regression equation of excess carbon dioxide output/BSA best fitted a two-compartment model. The time constants of the rapid and slow compartments were 8.2 and 990 min, respectively. Excess carbon dioxide output/BSA was still 5.5 ml min-1 m-2, 30 min after pneumoperitoneum.

KEYWORDS : Surgery, laparoscopy. Anaesthetics, volatile, sevoflurane. Ventilation, carbon dioxide response. Ventilation, deadspace. Oxygen, uptake.

INTRODUCTION

In pneumoperitoneum, carbon dioxide eliminated in expired gas (carbon dioxide output) contains both metabolic and absorbed carbon dioxide from the peritoneal cavity. When elimination of carbon dioxide is much higher than carbon dioxide output, storage of tissue carbon dioxide and arterial carbon dioxide concentrations change. Finally, the rate of carbon dioxide eliminated in expired gas is not a match for the real rate of metabolic production and absorbed carbon dioxide from the peritoneal cavity. During and after insufflation of carbon dioxide, changes in carbon dioxide output were elucidated under constant arterial carbon dioxide pressure (PaCO2), the same as the preinduction level.

MATERIAL AND METHOD

We conducted this study in RIMS RAIPUR to assess excess carbon dioxide output evoked by pneumoperitoneum with stable carbon dioxide storage maintained by keeping arterial CO2 Pa constant at preinduction levels and to investigate continued carbon dioxide load after pneumoperitoneum using a pharmacokinetic model. We studied 20 consenting, ASA I or II patients, undergoing elective laparoscopic cholecystectomy.Patients were in good general health, with no signs or laboratory findings of renal, pulmonary or hormonal disease, or obesity (defined as a body mass index 29). All patients fasted overnight and were premedicated with midazolam 0.1 mg/kg and atropine 0.5 mg 1 h before induction of anaesthesia. An i.v. cannula was using during local anaesthesia. I.v. infusion of sodium lactate solution was started, and the rate of infusion was adjusted to 8–10 ml kg-1 h-1 during the study. Heart rate and ECG were monitored continuously. An arterial cannula was inserted into the radial or dorsalis pedis artery using local anaesthesia for arterial pressure monitoring. An arterial blood sample was obtained from the indwelling arterial cannula anaerobically and analysed immediately. Rectal temperature was displayed continuously on a temperature module (Hewlett-Packard) using a thermistor probe inserted 10 cm into the rectum. Body temperature was maintained at 36–37 °C by external heating. The temperature of the operating room was maintained at 24–26°C. The sampling cannula for measuring end-tidal the mixing chambers, was measured by mass spectrometer in order to exclude the effect of ageing on carbon dioxide absorber. The equations for calculation are valid even in the presence of anaesthetic gas. The ratio of dead space to tidal volume(VD/VT) was calculated using Bohr's equation Before induction of anaesthesia, all subjects were permitted to breathe room air through the anaesthesia mask for 20 min and mean expired and inspired concentrations of oxygen, carbon dioxide, nitrogen and expiratory minute volume were measured to obtain baseline carbon dioxide output and oxygen uptake. Anaesthesia was induced with Propofol 2 mg/kg i.v. and non-depolarizing neuromuscular blocker (vecuronium 0.1 mg kg-1). Tracheal intubation was performed orally using a cuffed tracheal tube. Inspired concentrations of oxygen and nitrous oxide were adjusted to 30 %, and 70 %, respectively, during anaesthesia. The inspired sevoflurane mixture was adjusted to give an end-tidal sevoflurane concentration of 1.37 % (0.8 MAC). The fresh gas flow rate was 2–3 litre min-1 . The lungs of all patients were ventilated mechanically with the same ventilator equipped with an anaesthesia machine. Tidal volume was set at 10 ml kg-1 . Ventilatory frequency (f) was adjusted mainly each minute to maintain Pa CO2 at preinduction levels with continuous monitoring of end-tidal carbon dioxide pressure (PE CO2) and intermittent measurement of arterial to end-tidal carbon dioxide partial pressure difference PaCO2 –PE'CO2. Arterial blood samples were obtained every 15 min after stabilization of the ventilator settings and analysed for pH, PaCO2, PaO2 and base excess. Before skin incision, baseline carbon dioxide output and oxygen uptake were obtained after they reached steady state. Neuromuscular block was maintained with additional increments of vecuronium 0.025 mg kg-1 when the train-of four ratio exceeded 75 %. Carbon dioxide was introduced into the peritoneal cavity by a carbon dioxide Pneu pressure limiting automatic insufflation apparatus and abdominal pressure was maintained at 12 mm Hg in all patients. After insufflation, multiple incremental changes in minute volume were made to maintain PaCO2 at preinduction levels. The table was tilted about 5° of the reverse. Trendelenburg position and then slightly to the left lateral position. Laparoscopic cholecystectomy was performed by a standard procedure. Intraperitoneal gas was evacuated after cholecystectomy and regression of carbon dioxide output in the postinsufflation state was measured 30 min under stable P E CO2

every 1 min by manual control of ventilatory frequency. After carbon dioxide output returned to control level, neuromuscular block was antagonized with neostigmine 2.0–2.5 mg i.v. and atropine 1.0 mg i.v. The trachea was then extubated. Anaesthesia was divided into five periods: (1) preinduction phase, (2) anaesthesia phase (control period of anaesthesia before skin incision), (3) pneumoperitoneum phase (during laparoscopy at intra-abdominal pressure of 12 mm Hg), (4) postpneumoperitoneum phase (0–30 min after evacuation of carbon dioxide from the peritoneal cavity), and (5) recovery phase (40 min after extubation). To fit the regression curve for carbon dioxide output during the post-pneumoperitoneum phase to multicompartment models, the least squares method was used and the number of compartments was determined by minimum AIC (an information criterion) based on the maximum likelihood estimation method . Statistical analysis was carried out using analysis of Student's t test, where appropriate. P 0.05 was considered statistically significant. All values are expressed as mean (SEM).

DISCUSSION

During laparoscopy, the large volume of carbon dioxide for pneumoperitoneum is passively absorbed from the peritoneal cavity into the blood and most of it is removed from the circulation by hyperventilation. The quantity of carbon dioxide and bicarbonate ion in the body is large. When ventilation is not in accord with carbon dioxide output, hypercapnia continues. Sustained elevation in Pa CO2 , typical of that associated with pneumoperitoneum, probably recruits whole body storage depots, such as skeletal muscle and bone , in addition to intraperitoneal organs. Thus to study carbon dioxide output during laparoscopic cholecystectomy, the body storage of carbon dioxide must not be allowed to change. Although it is not certain that a constant PaCO2 necessarily implies constant storage, it is a reasonable assumption. In the present study, VE was adjusted to maintain PaCO2 constant at the preinduction level with constant monitoring of PE CO2 and intermittent measurement of (PaCO2- P E CO2).This method for estimating PaCO2 is invalid if the alveolar dead space increases with (PaCO2- P E CO2), a situation that may occur during the period of carbon dioxide insufflation. Bramton and Watson reported that monitoring of CO2 PE during laparoscopy could be used to reflect , CO2 Pa except at the start of pneumoperitoneum . Fi tzlerald and colleagues demonstrated that there were no changes in (PaCO2- P E CO2)during the pneumoperitoneum in mechanical ventilation . In the present study, VD/VT was almost constant except during preinduction when an anaesthesia mask was used for ventilation. PaCO2 during pneumoperitoneum was thought to be nearly constant, since PE CO2 and (PaCO2- P E CO2) were nearly constant.During anaesthesia, the metabolic rate is reduced by about 15–30 % below that of the awake state . In this study, oxygen uptake and carbon dioxide output decreased 20.2 % and 18.0 % respectively. Thus RER remained constant, even during anaesthesia. Surgical stimulation may alter carbon dioxide output and oxygen uptake by increasing metabolic rate. End-tidal total MAC multiples of sevoflurane and nitrous oxide during pneumoperitoneum were maintained at 1.39, which was sufficient to prevent the effect of surgical stimulation on metabolism, as oxygen uptake did not change during surgery. The increased intra-abdominal pressure may have interfered with the elimination of carbon dioxide from abdominal organs and lower extremities by causing the veins to collapse. Carbon dioxide output and oxygen uptake increase after completion of pneumoperitoneum in this situation. However, an intra-abdominal pressure of 12 mm Hg, which was maintained automatically, did not hinder elimination of carbon dioxide, as oxygen uptake during and after pneumoperitoneum remained stable.In the present study, VD/VT was almost constant except during preinduction when an anaesthesia mask was used for ventilation.PaCO2 during pneumoperitoneum was thought to be nearly constant, since PE CO2 and (PaCO2- P E CO2) were nearly constant. During anaesthesia, the carbon dioxide output of 102.4 (5.0) ml min-1 m-2 and excess carbon dioxide output of 34.1 (3.9) ml min-1 m-2 (54.0 (4.6) ml min-1) observed in the present study, possibly because of the following three factors: a decrease in tissue storage of carbon dioxide as a result of hyperventilation before insufflation of carbon dioxide, hypoventilation after insufflation and unstable conditions for measuring carbon dioxide output. The carbon dioxide insufflated to establish pneumoperitoneum diffuses into the abdominal organs and abdominal wall through the peritoneum, partly accumlates in tissue, and is then carried by the blood to the lungs. Therefore, the rapid compartment represents abdominal circulatory blood, liver, kidneys and other well-perfused tissues. The slow compartment represents tissues with low blood flow. Although the carbon dioxide load in the present study was clinically safe in patients without cardiac or pulmonary disease with controlled ventilation during and after pneumoperitoneum,excess VCO2/BSA was still 5.5 ml min-1 m-2 , 30 min after pneumoperitoneum. Therefore, patients receiving sedative drugs, patients with significant cardiac or pulmonary disease, or those with impaired ventilation in the pneumoperitoneum phase may be adversely affected by hypercapnia associated with carbon dioxide insufflation, even in the postoperative phase .

RESULT

We studied 20 premedicated patients, who were essentially normal in the pre-anaesthesia state. Steady states for VCO2/BSA and VO2/BSA were obtained during the following phases: preinduction, anaesthesia, pneumoperitoneum and recovery. Although it was difficult to maintain PaCO2 constant during the 10 min after tracheal intubation and the start of PE CO2 surgery, concentrations were maintained at 5.0–5.4 kPa. Compared with the preinduction phase, minute ventilation (VE) decreased from 5.26 (0.33) to 3.33 (0.27) litre min-1 (P 0.05) in the anaesthetic phase and increased to 5.14 (0.30) litre min-1 in the pneumoperitoneum phase (P 0.05). Respiratory minute volume during pneumoperitoneum thus had to be made 1.54 times that in the anaesthesia phase to maintain PaCO2 constant. Carbon dioxide output and oxygen uptake decreased, respectively, from 83.5 (5.2), 101.6 (5.1) to 68.5 (4.2), 81.1(4.6) ml min-1 m-2 (ATPS, P 0.05) with sevoflurane anaesthesia and RER did not change. During pneumoperitoneum, carbon dioxide output increased 49 % (102.4 (5.0) ml min-1 min-2 , (P 0.05) while oxygen uptake remained stable and RER increased from 0.84 (0.02) to 1.16 (0.03) (P 0.05). These variables returned to preinduction levels during recovery. PaCO2 , pH, base excess and SaO2a in the preinduction phase were normal and were maintained throughout the experiment. After induction of anaesthesia there were decreases in heart rate, systolic arterial pressure, mean arterial pressure and diastolic arterial pressure. Significant increases in arterial pressure in the pneumoperitoneum phase were noted, but no cardiac arrhythmia was detected during induction, pneumoperitoneum or after pneumoperitoneum. The regression equation for excess VCO2/BSA after removal of carbon dioxide from the abdominal cavity were stable. Time constants of the first (rapid) and second (slow) compartments were 8.2 and 990 min, respectively. At the end of pneumoperitoneum, the rapid compartment was thought to be equilibrated, as * T was short (5.7 min) and carbon dioxide output was constant. The excess rate of carbon dioxide output/BSA was still 5.5 ml min-1 m-2 , 30 min after removal of carbon dioxide from the peritoneal cavity. There was no clinical evidence of hypoxaemia, no change in standard bicarbonate before and after peritoneal insufflation, and no postoperative complications.

CONCLUSION

PaCO2 , pH, base excess and SaO2a in the preinduction phase were normal and were maintained throughout the experiment. After induction of anaesthesia there were decreases in heart rate, systolic arterial pressure, mean arterial pressure and diastolic arterial pressure. Significant increases in arterial pressure in the pneumoperitoneum phase were noted, but no cardiac arrhythmia was detected during induction, pneumoperitoneum or after pneumoperitoneum. During pneumoperitoneum, carbon dioxide output increased 49 % (102.4 (5.0) ml min-1 min-2 , (P 0.05) while oxygen uptake remained stable and RER increased from 0.84 (0.02) to 1.16 (0.03) (P 0.05). There was no clinical evidence of hypoxaemia, no change in standard bicarbonate before and after

peritoneal insufflation, and no postoperative complications..

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