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Development of atelectasis and arterial to end-tidal P_{CO_2}-difference in a porcine model of pneumoperitoneum

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Background. Intraperitoneal insufflation of carbon dioxide (CO_2) may promote collapse of dependent lung regions. The present study was undertaken to study the effects of CO_2-pneumoperitoneum (CO_2-PP) on atelectasis formation, arterial oxygenation, and arterial to end-tidal P_{CO_2}-gradient ($Pa\text{-}E'_{CO_2}$).

Methods. Fifteen anaesthetized pigs [mean body weight 28 (SD 2) kg] were studied. Spiral computed tomography (CT) scans were obtained for analysis of lung tissue density. In Group 1 (n=5) mechanical ventilation (V_T=10 ml kg $^{-1}$, $F_{I_{O_2}}$=0.5) was applied, in Group 2 (n=5) $F_{I_{O_2}}$ was increased for 30 min to 1.0 and in Group 3 (n=5) negative airway pressure was applied for 20 s in order to enhance development of atelectasis. Cardiopulmonary and CT data were obtained before, 10, and 90 min after induction of CO_2-PP at an abdominal pressure of 12 mmHg.

Results. Before CO_2-PP, in Group 1 non-aerated tissue on CT scans was 1 (1)%, in Group 2 3 (2)% (P<0.05, compared with Group 1), and in Group 3 7 (3)% (P<0.05, compared with Group 1 and Group 2). CO_2-PP significantly increased atelectasis in all groups. $Pa_{O_2}/F_{I_{O_2}}$ fell and venous admixture ('shunt') increased in proportion to atelectasis during anaesthesia but CO_2-PP had a varying effect on $Pa_{O_2}/F_{I_{O_2}}$ and shunt. Thus, no correlation was seen between atelectasis and $Pa_{O_2}/F_{I_{O_2}}$ or shunt when all data before and during CO_2-PP were pooled. $Pa\text{-}E'_{CO_2}$, on the other hand correlated strongly with the amount of atelectasis (r^2=0.92).

Conclusions. Development of atelectasis during anaesthesia and PP may be estimated by an increased $Pa\text{-}E'_{CO_2}$.

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Intraperitoneal insufflation of carbon dioxide (CO_2) is commonly used for induction of pneumoperitoneum (PP) during laparoscopic surgery.[1] An increased intra-abdominal pressure may shift the end-expiratory position of the diaphragm, decrease functional residual capacity, and induce collapse of dependent lung regions, and even more so in patients with morbid obesity.[2 3] Perfusion of non-ventilated alveoli strongly affects oxygenation of blood[4] and CO_2-elimination may also be impaired. In anaesthetized patients, PP causes an increase in atelectasis but need not increase shunt or lower arterial oxygenation.[5 6] This apparent paradox has not yet been explained. Another consequence of the absence of a correlation between atelectasis and oxygenation is that measurement of Pa_{O_2} or calculation of shunt cannot be used to assess the amount of collapsed lung. The present study was undertaken to study the effects of CO_2-PP on atelectasis formation and arterial to end-tidal P_{CO_2}-gradient ($Pa\text{-}E'_{CO_2}$). We hypothesized that there was a correlation between regional lung collapse and CO_2-elimination in a porcine model of PP.

Methods

After approval by the local animal ethics committee, 15 2-month-old healthy piglets [mean body weight 28 (2) kg,

eight males and seven females) of the Hampshire, Yorkshire, and Swedish country breeds from a local breeder were studied.

Anaesthesia and mechanical ventilation

All pigs were anaesthetized by an i.m. injection of xylazine (2.2 mg kg^{-1}, Rompun®; Bayer, Leverkusen, Germany), tiletamine/zolazepam (6 mg kg^{-1}, Zoletil®; Virbac, Carros, France), and atropine (0.04 mg kg^{-1}, NM Pharma, Stockholm, Sweden). The pigs' lungs were mechanically ventilated after intubation with an ID 7.0 mm cuffed tracheal tube (Mallinckrodt, Athlone, Ireland). Anaesthesia was maintained by continuous administration of fentanyl (5 μg kg^{-1} h^{-1}, Leptanal®; Janssen-Cilag AB, Sweden), pancuronium (0.3 mg kg^{-1} h^{-1}, Pavulon®; Organon, Oss, The Netherlands), ketamine (25 mg kg^{-1} h^{-1}, Ketaminol vet.®; Intervet, Boxmeer, The Netherlands), and propofol (3 mg kg^{-1} h^{-1}, Diprivan®; Astra, Södertälje, Sweden). Before experimentation, adequate depth of surgical anaesthesia was confirmed by absence of both the hind limb flexion reflex and corneal reflex responses according to the laboratory standard of the Animal Ethics Committee of Uppsala University. Ringer's acetate (Pharmacia AB; Stockholm, Sweden) was infused with an average rate of 5 ml kg^{-1} h^{-1} to maintain a constant haemoglobin concentration and stable systemic arterial blood pressure.

Mechanical ventilation was initiated in volume-controlled mode [intermittent positive pressure ventilation (IPPV); Servo i; Maquet Critical Care AB, Solna, Sweden]. Ventilatory frequency was adjusted to achieve normocapnia (Pa_{CO_2}=4.7–6 kPa). Tidal volume (V_t), airway pressures (P_{aw}), and flow were continuously recorded. Static compliance (C_{rs}) of the total respiratory system was calculated as $C_{rs}=V_t$ ($P_{aw\ plateau} - P_{aw\ end\text{-}expiration}$).

Monitoring

For pressure measurements and arterial blood sampling, an 18-gauge catheter (Hydrocath™; Becton Dickinson, Franklin Lakes, NJ, USA) was inserted into the left carotid artery. A thermistor-tipped Swan-Ganz catheter (CritiCath™ SP5107H-14 TD; Becton Dickinson) and an 18-gauge catheter (Hydrocath™; Becton Dickinson) were introduced into the left external jugular vein. Systemic, pulmonary arterial, and central venous pressures were displayed on a monitor (SC 9000 XL; Maquet Critical Care AB, Solna, Sweden) and were recorded with reference to atmospheric pressure at the midthoracic level during end-expiration. End-expiratory CO_2 tension ($P_{E'_{CO_2}}$) was measured by capnography implemented in the ventilator (Servo i; Maquet Critical Care AB, Solna, Sweden).

Arterial and mixed venous blood samples were analysed with ABL 300 blood gas analyser and OSM 3 oximeter (Radiometer, Copenhagen, Denmark). Cardiac output (CO) was determined by thermodilution. The thermal indicator was 10 ml of saline 8–10°C and was injected into the right atrium. The first measurement was ignored and the cardiac output was derived from the mean of three consecutive measurements. The injections were evenly distributed over the respiratory cycle. Venous admixture ('shunt') was calculated according to the standard shunt equation, based on the calculation of oxygen content in arterial, mixed venous, and pulmonary end-capillary blood.

Computed tomography

A frontal tomogram of the chest was obtained during ventilation to determine the borders of the lung. An end-expiratory transversal spiral computed tomography (CT; 140 kV, 111 mA) covering the whole lung, with 1 mm slice thickness was acquired with a Somatom Plus 4 CT scanner (Siemens, Erlangen, Germany). The scanning time for the transverse images was approximately 3 s. The CT scanning was analysed using the custom-made software package MALUNA (Mannheim Lung Analysis Tool). The total lung volume was calculated by creating a region of interest (ROI) around each lung scan excluding the mediastinum and the big vessels. Each voxel of the CT scan is characterized by a CT number, which is related to the tissue density and numerically expressed in Hounsfield units (HU). The scale ranges from +1000 HU (bone) to 0 HU (water) and −1000 HU (air). For example, a voxel with −200 HU consists of 20% gas and 80% tissue, and a voxel with −700 HU consists of 70% gas and 30% tissue. For further analysis, the lung was divided into four categories: areas with densities ranging from −1000 to −850 HU were classified as over-aerated, from −850 to −500 HU as normally aerated, from −500 to −100 HU as poorly aerated, and from −100 to +100 HU as non-aerated (atelectasis).

Study protocol

Allocation to the groups was made using sealed envelopes. In Group 1 (n=5), pigs were ventilated with a V_T of 10 ml kg^{-1}, $F_{I_{O_2}}$ of 0.5 and PEEP of 5 cm H_2O. Based on data from the literature and results from our laboratory minor development of atelectasis was expected. In Group 2 (n=5), $F_{I_{O_2}}$ was increased to 1.0 for 30 min and in Group 3 (n=5) a negative pressure of −15 cm H_2O was applied to the tracheal tube for 20 s in order to produce a large range of atelectasis similar to findings in anaesthetized normal-weight and morbidly obese patients. After these procedures, Group 2 and Group 3 were given the same ventilation as Group 1.

Pneumoperitoneum was created by insufflation of CO_2 into the abdominal cavity via a VERRES needle with a common CO_2 insufflator (7060-Insufflator Pelvi Pneu Semm Systems; Wisap, Munich, Germany) until the abdominal pressure (P_{abd}) reached 12 mmHg. Mechanical