Changes in plasma potassium concentration during carbon dioxide pneumoperitoneum

A. Perner1*, K. Bugge1, K. M. Lyng2, S. Schulze2, P. A. Kristensen3 and A. Bendtsen1

Department of Anaesthesia1 and Department of Surgery2, Sundby Hospital, University of Copenhagen, DK-2300 Copenhagen, Denmark. 3Department of Anaesthesia, Gentofte Hospital, University of Copenhagen, DK-2900 Hellerup, Denmark

*To whom correspondence should be addressed

Hyperkalaemia with ECG changes has been noted during prolonged carbon dioxide pneumoperitoneum in pigs. We have compared plasma potassium concentrations during surgery in 11 patients allocated randomly to undergo either laparoscopic or open appendectomy and in another 17 patients allocated randomly to either carbon dioxide pneumoperitoneum or abdominal wall lifting for laparoscopic colectomy. Despite an increasing metabolic acidosis, prolonged carbon dioxide pneumoperitoneum resulted in only a slight increase in plasma potassium concentrations, which was both statistically and clinically insignificant. Thus hyperkalaemia is unlikely to develop in patients with normal renal function undergoing carbon dioxide pneumoperitoneum for laparoscopic surgery.

Br J Anaesth 1999; 82: 137–9

Keywords: surgery, laparoscopy; surgery, laparotomy; carbon dioxide, pneumoperitoneum; ions, potassium; complications, hyperkalaemia

Accepted for publication: August 10, 1998

A linear increase in plasma potassium concentrations during carbon dioxide pneumoperitoneum, with concomitant ECG changes consistent with hyperkalaemia, has been noted in pigs after 2–3 h of carbon dioxide insufflation.1 It was hypothesized that the hyperkalaemia was caused by movement of intracellular potassium into the plasma as a result of acidosis induced by absorption of carbon dioxide from the peritoneal cavity. Respiratory acidosis did not develop, as insufflation of carbon dioxide was accompanied by an increase in mechanical ventilation. Alternatively, ischaemia of the abdominal wall or oliguria as a result of increased intra-abdominal pressure could have caused hyperkalaemia during pneumoperitoneum.1

If this effect occurred in patients during laparoscopic surgery, the resultant cardiac arrhythmias could be life-threatening. Thus in this study, we have compared changes in plasma potassium concentrations during carbon dioxide pneumoperitoneum for laparoscopic surgery with a control group undergoing surgery without the use of carbon dioxide pneumoperitoneum.

Methods and results

The study was approved by the Ethics Committee of Copenhagen and all participants gave informed, written consent. The study population consisted of patients included in two surgical studies: (1) patients allocated to either laparoscopy (carbon dioxide pneumoperitoneum) or laparotomy for suspected appendicitis and; (2) patients allocated to either carbon dioxide pneumoperitoneum or abdominal wall lifting for laparoscopic colectomy. Patients were allocated by random numbers to each group using sealed envelopes. Patients receiving blood transfusions, insulin, diuretics, potassium, beta-blocking agents or beta-agonists 24 h perioperatively were excluded, as were those with impaired renal function (serum creatinine >120 µmol litre−1).

Before anaesthesia, at surgical incision and every 30 min during the procedure, arterial blood (2 ml) was obtained into a syringe (Qs90, Radiometer, Denmark) and analysed for pH, P\text{aCO}_2 and plasma potassium concentrations (ABL 610, Radiometer, Denmark) within 15 min. Oesophageal temperature was measured (DM 852, El-Lab, Denmark) after tracheal intubation and at the end of surgery.

During the procedure, all patients underwent artificial ventilation via a semi-closed circle system using a MCM 801 respirator (Dameca, Denmark). End-tidal carbon dioxide partial pressure (Oscar II SC-123 Datex, Finland) was maintained constant at 4.0–6.0 kPa throughout the procedure to prevent respiratory acidosis causing an increase in plasma potassium concentrations.5 Mean arterial pressure (MAP) was measured non-invasively (study 1) or invasively (study 2) (Propaq 106, Protocol Systems Inc., USA). Patients received isotonic saline and hydroxyethyl starch
60 mg ml⁻¹ (Haes-Sterile, Fresenius, Germany (study 2)) to replace basic requirements and surgical loss. Hypotension (MAP < 60 mm Hg) was treated with ephedrine 5–10 mg i.v., repeated if necessary.

Data are presented as median (range). The Wilcoxon test (paired data) or Mann–Whitney U test (unpaired data) was used for hypothesis testing and the Spearman rank order test for calculations of correlation coefficients. \( P < 0.05 \) was considered significant.

**Study 1—appendectomy**

We studied 11 ASA I patients suspected of appendicitis (eight females; aged 33 (18–53) yr). Seven patients were allocated randomly to undergo laparoscopy (intraperitoneal carbon dioxide pressure < 1.83 kPa) and four to laparotomy (controls). Seven patients had appendicitis, one salpingitis, one mesenteric adenitis and two patients had a ruptured ovarian cyst. General anaesthesia was induced with thiopental 1.5 mg kg⁻¹. Anaesthesia was maintained with 1–2% isoflurane and 50% nitrous oxide in oxygen, with fentanyl 50–150 \( \mu \)g ml⁻¹ (lignocaine) 20 mg ml⁻¹ with epinephrine (adrenaline) 50 \( \mu \)g ml⁻¹. Regional anaesthesia was ensured by needle prick infiltration with ropivacaine 0.75%. Only one patient in the colectomy-control group had surgery for longer than 150 min. Data are medians with 25th and 75th percentiles.

**Study 2—colectomy**

We studied 17 ASA I–IV patients undergoing colectomy (appendectomy, \( n = 11 \); broken lines) and in study 2 (colectomy, \( n = 17 \); solid lines). Only one patient in the colectomy-control group had surgery for longer than 150 min. Data are medians with 25th and 75th percentiles.

Changes in plasma concentrations of potassium during anaesthesia can have deleterious cardiac effects. 3 In this study, plasma potassium concentrations were unaffected by carbon dioxide pneumoperitoneum in young and relatively
healthy patients during a short laparoscopic procedure. Furthermore, prolonged carbon dioxide pneumoperitoneum in older patients (study 2) induced only a minor increase in plasma potassium concentrations, which was both statistically and clinically insignificant, despite development of a progressive metabolic acidosis. However, it is possible that movement of intracellular potassium into the blood is enhanced during carbon dioxide pneumoperitoneum, but that an aldosterone-mediated increase in urinary potassium excretion prevents hyperkalaemia. We did not measure urinary potassium concentrations, but an inverse correlation between plasma potassium and urinary output was not found (study 2, data not shown), suggesting that aldosterone activity was not increased markedly in these patients. As firm evidence of the potential role of aldosterone is lacking, care should be taken in patients treated with aldosterone inhibitors, such as potassium-sparing diuretics, and in patients with impaired renal function during prolonged carbon dioxide pneumoperitoneum.

The development of progressive metabolic acidosis during carbon dioxide pneumoperitoneum, as observed in study 2, has been suggested to be caused by decreased peripheral perfusion. This theory is supported by our finding of an inverse correlation between pH and MAP, as changes in MAP could reflect changes in peripheral vascular resistance during carbon dioxide pneumoperitoneum.

Even though there is a risk of a type II error, we can conclude that clinically relevant hyperkalaemia is unlikely to develop in patients with intact renal function during carbon dioxide pneumoperitoneum for laparoscopic surgery, when respiratory acidosis is avoided.

References

2 Hassan H, Gjessing J, Tomlin PJ. Hypercapnia and hyperkalaemia. Anaesthesia 1979; 34: 897–99
4 Chiu AW, Chang LS, Birkett DH, Babayan RK. Changes in urinary output and electrolytes during gaseous and gasless laparoscopy. Urol Res 1996; 24: 361–6