Hyperkalaemia associated with prolonged insufflation of carbon
dioxide into the peritoneal cavity

M. R. B. PEARSON AND M. L. SANDER

SUMMARY
We measured plasma potassium concentrations in five anaesthetized pigs over a 3.5 h period during which a carbon dioxide pneumoperitoneum was maintained. During this period the abdominal pressure was kept at 1.96 kPa and normocapnia was maintained by intermittent positive pressure ventilation. The potassium concentration was found to increase with time to a mean of 5.63 (SD 0.44) mmol litre⁻¹. Consideration should be given to monitoring plasma potassium concentration in patients undergoing prolonged laparoscopic surgery. (Br. J. Anaesth. 1994; 72: 602-604)

KEY WORDS

Laparoscopic procedures necessitating insufflation of gas into the peritoneal cavity have been used for short gynaecological examinations and minor procedures for many years. More recently, laparoscopic surgery has been developed to avoid conventional open laparotomy. Laparoscopic cholecystectomy is now commonplace and more complex and time-consuming procedures, such as Nissen fundoplication and colonic resection, are being performed regularly. While there have been several investigations into the effects of short periods (about 30 min) of intraperitoneal carbon dioxide insufflation, the effects of more prolonged insufflation have not been described.

METHODS AND RESULTS
The study was approved by the University of Sydney Animal Care and Ethics Committee. Five pigs (mean weight 45 (SD 7.29) kg) were anaesthetized on three occasions, 1 week apart. On each occasion, the pigs were premedicated with ketamine 6 mg kg⁻¹ i.m., atropine 0.04 mg kg⁻¹ i.m. and droperidol 0.4 mg kg⁻¹ i.m. combined with fentanyl 10 µg kg⁻¹ i.m. Anaesthesia was induced with i.v. thiopentone and halothane in oxygen by mask. Intubation of the trachea was facilitated by spraying the larynx with 2 ml of a 2 % lignocaine solution. Anaesthesia was maintained with halothane in oxygen, delivered via a semi-closed circle system, to give an end-tidal halothane concentration of 1.3 %. The animals' lungs were ventilated mechanically with a Campbell ventilator (Ulco, Australia). An initial tidal volume of 10 ml kg⁻¹ was delivered and an arterial partial pressure of carbon dioxide of 5.20–5.47 kPa was maintained.

Throughout the study, radiant heat lamps were used to maintain the animals at 38 °C. They were monitored using lead II ECG, direct arterial pressure measurement (via a catheter inserted percutaneously into the aortic arch), pulmonary artery pressure and temperature (7-French gauge Thermodilution Catheter, Abbott, U.S.A. and Lifescope 9, Nihon Kohden, Japan) and measurement of inspired and end-tidal concentrations of carbon dioxide, halothane and oxygen (Capnomac Ultima, Datex, Finland). Single measurements of arterial blood-gas tensions, acid-base state and electrolyte concentrations were made intermittently using an automated blood-gas and electrolyte analyser (ABL 505, Radiometer, Denmark). The reproducibility of the potassium electrode was tested on two occasions during the study when 10 measurements were made on one sample. On both occasions the SD obtained was less than 0.05, with means of 3.54 and 3.77.

In order to trigger malignant hyperthermia in susceptible animals, suxamethonium 1.5 mg kg⁻¹ was given i.v. after the physiological measurements had stabilized. The pigs were then observed for 30 min and any animal that developed signs suggestive of malignant hyperthermia was excluded from the study. Throughout the anaesthetic period, each pig received an i.v. infusion of a balanced electrolyte solution 2.2 ml kg⁻¹ h⁻¹ (Plasmalyte 148, Baxter, Australia) using an infusion pump (Imed 927).

An 18-gauge Verres needle was introduced into the peritoneal cavity through a 0.5-cm sub-umbilical incision. Another 30 min was allowed to ensure that the physiological measurements were stable before collecting control blood samples. Each pig was then given one of three treatments allocated on a random basis. The control treatment consisted of inserting the Verres needle. In the other treatments, carbon dioxide was insufflated into the peritoneal cavity with a surgical insufflator to maintain an intra-peritoneal pressure of 1.96 kPa. During one treatment, the increase in ventilation required to...
HYPERKALAEMIA AND CO₂ INSUFFLATION

After 2–3 h of insufflation, all pigs receiving insufflation had developed ECG changes consistent with early hyperkalaemia (reduced Q–T interval, increased T wave amplitude, widened QRS interval). No ECG changes were seen in the controls. Increases in the concentration of CK ranged from 0–123%. However, insufficient measurements were made to permit meaningful statistical analysis.

COMMENT

The changes in mean arterial pressure between treatments are difficult to explain but as the magnitude of the changes were small, they are of little clinical significance.

Among the potential causes of the progressive hyperkalaemia found in this study, the administration of suxamethonium- and potassium-containing i.v. fluids may be discounted, as both were given with each treatment. Translocation of potassium from intracardial to extracellular fluid may follow tissue damage and hypoxia [1]. Ischaemic damage to the muscles of the abdominal wall may have occurred as increased intra-abdominal pressure has been shown to cause a significant reduction in abdominal wall blood flow in pigs [2]. It is unlikely, however, that this mechanism alone would have caused the observed changes in plasma potassium, as only trivial increases in plasma concentrations of CK were found. Oliguric renal failure associated with intra-abdominal pressures of between 2.00 and 2.67 kPa has been described [3] and the association between renal failure and hyperkalaemia is well known. This process may have contributed to the hyperkalaemia observed in the present study, although the rate of increase of the potassium concentration suggests that this mechanism alone is insufficient to be solely responsible [4].

Another possible cause is the diffusion of carbon dioxide out of the peritoneal cavity producing local intracellular acidosis sufficient to cause movement of intracellular potassium into the blood.

If pneumoperitoneum were to continue beyond 3 h the plasma potassium concentration might well increase to a level at which immediate treatment is recommended [5]. This concentration would be expected to be reached sooner if there were concurrent mechanisms operating, such as acidosis or treatment with potassium-sparing diuretics, that would themselves contribute to hyperkalaemia [6].

The results of this study suggest that monitoring of plasma potassium concentration is advisable in patients undergoing prolonged laparoscopic procedures.

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REFERENCES


