CORRESPONDENCE

INADVERTENT ADMINISTRATION OF 100% OXYGEN DURING ANAESTHESIA

Sir,—I read Dr Paymaster’s letter (1978) with interest as I have encountered similar difficulties with the Cape Waine, Mark III Anaesthetic Machine. Here again there is a “push-pull” switch situated on the front panel of the machine for the delivery of emergency oxygen. This button has been left pushed to the “on” position after use and this has not been noticed by another anaesthetist using the machine immediately afterwards.

The position of the switch is a hazard. Located at the front of the machine, it is liable to be pushed to the “on” position by any member of the theatre staff who leans against the machine. The switch is noiseless and the anaesthetist may be unaware that a high concentration of oxygen is being delivered to the patient. Two cases of awareness during anaesthesia have occurred as a result of this problem.

Many similar incidents must have occurred and I would support the suggestion of a warning device to guard against the machine inadvertently delivering 100% oxygen.

This problem has been notified to Cape Waine Ltd, who propose the fitting of a metal shroud to prevent accidental activation of the emergency oxygen switch.

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REFERENCE


NALOXONE AND ACID–BASE BALANCE IN MAN

Sir,—In a recent study (Khanna and Pleuvry, 1978) naloxone was shown to produce significant changes in the acid–base state of the rabbit. Since such changes would be of considerable interest if they occurred in man, a double-blind study was undertaken in healthy volunteers to compare saline with naloxone 6 μg kg⁻¹ i.v.

Blood samples were taken for blood-gas analysis from the back of the hand which had been warmed in an electric blanket to ensure rapid circulation. Arterialized venous blood pH and PCO₂ were measured using a Radiometer micro-analysser; haemoglobin concentration was measured using standard hospital techniques and pulse and arterial pressure were measured at frequent intervals throughout the study.

After consistent baseline values had been obtained the volunteers were given saline or naloxone i.v. Further blood samples were taken at 10-min intervals for the subsequent 30 min and also at 60 min after injection. Drug or saline injections were always given in the arm opposite to that used for blood sampling. At least 1 week elapsed between each of the two treatments and six volunteers (three of each sex) took part in the trial.

Naloxone had no significant effects upon the measurements obtained (table I) and thus the acid–base problems seen in the rabbit would not appear to be relevant to man.

V. K. KHANNA
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REFERENCE


RECTAL KETAMINE IN PAEDIATRIC ANAESTHESIA

Sir,—The induction of anaesthesia in the frightened or difficult child can be difficult and unpleasant for all involved. In most European countries rectal thermometers are used frequently for children who, therefore, accept rectal application of drugs without undue distress. We have used ketamine rectally for induction of anaesthesia in 150 children undergoing surgery for inguinal hernia (61), ectopic testis (37), hydrocoele (25) and various other operations.

Atropine and diazepam 0.2 mg kg⁻¹ were given i.m. 1 h before the rectal administration of ketamine 8–10 mg kg⁻¹ (to a maximum of 150 mg). A 1% solution

Table I. Effect of saline and 6 μg kg⁻¹ naloxone on the arterialized venous blood pH and PCO₂ in man. *Readings are expressed as the change from the values before injection for each individual. Results are means ± standard errors of six experiments

<table>
<thead>
<tr>
<th>Time after injection</th>
<th>Saline</th>
<th>Naloxone</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH (unit)</td>
<td>7.459 ± 0.016</td>
<td>7.462 ± 0.009</td>
</tr>
<tr>
<td>PCO₂ (kPa)</td>
<td>4.82 ± 0.30</td>
<td>5.16 ± 0.07</td>
</tr>
<tr>
<td>Before injection</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pH (unit)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCO₂ (kPa)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10 min</td>
<td>+0.027 ± 0.023</td>
<td>-0.018 ± 0.011</td>
</tr>
<tr>
<td>20 min</td>
<td>-0.05 ± 0.19</td>
<td>-0.03 ± 0.16</td>
</tr>
<tr>
<td>30 min</td>
<td>-0.06 ± 0.017</td>
<td>-0.006 ± 0.013</td>
</tr>
<tr>
<td>60 min</td>
<td>+0.02 ± 0.20</td>
<td>+0.12 ± 0.08</td>
</tr>
<tr>
<td>PCO₂ (kPa)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30 min</td>
<td>-0.039 ± 0.015</td>
<td>+0.007 ± 0.021</td>
</tr>
<tr>
<td>60 min</td>
<td>-0.09 ± 0.12</td>
<td>+0.19 ± 0.21</td>
</tr>
<tr>
<td>PCO₂ (kPa)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30 min</td>
<td>+0.36 ± 0.27</td>
<td>+0.08 ± 0.08</td>
</tr>
</tbody>
</table>
was used for children weighing less than 15 kg and a 5% solution for those weighing more. When the required level of sedation was obtained, anaesthesia was maintained and controlled by the use of enflurane in nitrous oxide and oxygen, with spontaneous ventilation via a face mask. In 48 patients the trachea was intubated following the administration of suxamethonium. In 22 children artificial ventilation was employed with the aid of competitive neuromuscular blocking drugs.

The interval from rectal administration of ketamine to loss of verbal contact was 378 ± 9 s (n = 67)—an interval similar to that following i.m. administration.

The children did not tolerate the face mask without moving until at least 8 min (average 9 min 28 s ± 18 s (n = 59)). Heart rate increased from 97 ± 11 to 110 ± 14 beat min⁻¹ and arterial pressure from 94 ± 0.8 to 101 ± 1 mm Hg. The duration of these effects could not be determined, since inhalation anaesthesia was started immediately.

The concentrations of enflurane required were 40% less than those required in the absence of ketamine. Surgical conditions were not changed by the use of ketamine.

The children responded to simple commands 18 min 36 s ± 16 s after terminating the administration of enflurane. Excessive salivation was frequent. It was concluded that a larger dose of atropine should be given before administering ketamine rectally. Hypotonia, without respiratory depression, occurred in four very young children. Vomiting occurred after operation in 16. No rectal irritation was observed.

Induction was judged excellent in 139, good in seven and fair in four instances. Amnesia for induction was complete in 75% of the patients.

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LITHIUM, ANAESTHESIA AND ECT

Sir,—Jephcott and Kerry (1974) have described a patient who had taken lithium carbonate and who could not be roused for more than 2 h after modified ECT and who was drowsy for the remainder of the day. The plasma lithium concentration during the episode was 3.4 mmol litre⁻¹ and it seems likely that the unusually prolonged effect of the anaesthetic was associated with this. Subsequently, Mannisto and Saarnivaara (1976) found that, in mice, lithium prolonged methohexitone-induced sleep. Jephcott and Kerry (1974) were unable to explain the high plasma lithium concentration in their patient, since she had previously been well controlled. Since anaesthetics affect membrane permeability, it may be postulated that such drugs cause short-term changes in the distribution of lithium.

Six patients suffering from manic depression who were undergoing modified ECT and who were taking lithium carbonate were investigated. ECT treatment was given at 11.00 h, lithium carbonate having been withheld from 14.00 h the previous evening. Venous blood samples were taken immediately before and 1 h and 3 h after ECT. Ammonium heparin was used as anticoagulant. Premedication was with atropine 0.6 mg given 30 min before ECT. Methohexitone sodium 100 mg followed by suxamethonium bromide 30 mg i.v. were used for anaesthesia. Plasma lithium concentrations were determined by atomic emission spectrometry.

FIG. 1. Decay curves of plasma lithium concentration in six patients treated by ECT at time 0.

In no patient (fig. 1) was treatment associated with a significant deviation from the expected exponential concentration decay curves. We conclude that there is no evidence that modified ECT affects blood lithium concentrations.

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REFERENCES