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(Accepted for publication October 22, 1983)

Intraoperative Hyperpyrexia—Keep an Open Mind

To the Editor:—Malignant hyperthermia (MH) is probably the first diagnosis contemplated when an intraoperative pyrexia is encountered because effective treatment requires early diagnosis. However, the consequences of this diagnosis are far reaching, both for the patient and the family, and it therefore behooves the clinician to make an accurate diagnosis prior to instituting therapy.

REPORT OF A CASE

A 33-yr-old ASA 1 woman, weighing 60 kg, with documented chol- lelithiasis was scheduled for cholecystectomy. Past history included six general anesthetics without complications and a negative family history of anesthetic-related problems. Diazepam 10 mg po and cefazolin 1 g iv were given 1 h prior to surgery. Anesthesia was induced with thi- malyl 280 mg followed by pancuronium 7 mg. Ventilation was easily controlled with 100% oxygen, and tracheal intubation was performed without difficulty. Anesthesia was maintained with nitrous oxide 70% in oxygen and enfurane 0.5%. Levophanol 2 mg was administered for analgesia. An esophageal stethoscope and temperature probe were positioned. Vital signs remained stable until 45 min into the case when esophageal temperature rose rapidly from 37.2° C to 39° C. Sweating was absent and skin color unchanged. Inspiratory wheezing was noted but pulmonary compliance appeared unchanged. The following maneuvers were instituted urgently: Enfuranze was discontinued, and hyperventilation with 100% oxygen was commenced; an arterial blood sample was drawn, the endotracheal tube withdrawn 2 cm, the cooling mattress turned on, and the MH treatment box requested. The esophageal probe and temperature monitor were checked, but the recorded temperature remained high. Blood pressure was 120/95, heart rate 105 beats/min, PaO2 73.4 mmHg, PaCO2 34 mmHg and pH 7.42. Inspiratory wheezing had subsided, but esophageal temperature had risen to 41.3° C. Repeat blood-gas analysis showed PaO2 422 mmHg, PaCO2 20.9 mmHg, and pH 7.54. Enfuranze was recommenced, and esophageal temperature was 57.7° C by the end of the otherwise uneventful procedure. There were no further febrile episodes during the hospitalization.

Our initial reaction was that we were confronted with a case of MH. The diagnosis of MH requires a complex of physical signs and symptoms that includes muscle rigidity, tachycardia, metabolic and respira-


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(Accepted for publication October 22, 1983)

Isoflurane and the Oxyhemoglobin Dissociation Curve in Humans

To the Editor:—Data concerning the influence exerted by inhalation anesthetic on the oxyhemoglobin dissociation curve (ODC) are contradictory concerning the results obtained by investigators working with N2O and halo-

Anesthesiology
64:413–415, 1986
TABLE I. Effect of Isoflurane on Oxyhemoglobin Dissociation In Vitro and In Vivo

<table>
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<tr>
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<th>F1</th>
<th>F2</th>
<th>F3</th>
<th>F4</th>
<th>C-4</th>
<th>C-3</th>
<th>C-2</th>
<th>C-1</th>
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</thead>
<tbody>
<tr>
<td>P4 (mmHg)</td>
<td>26.5 ± 0.3</td>
<td>26.6 ± 0.3</td>
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<td>26.4 ± 0.3</td>
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<tr>
<td>P5 (mmHg)</td>
<td>2.73 ± 0.03</td>
<td>2.78 ± 0.03</td>
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<tr>
<td>P6 (mmHg)</td>
<td>2.75 ± 0.04</td>
<td>2.72 ± 0.03</td>
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<tr>
<td>P7 (mmHg)</td>
<td>1.83 ± 0.07</td>
<td>2.58 ± 0.06</td>
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</table>

Values are mean ± SE. In vitro: B = basal samples; F1, F2, F3, F4 = samples exposed to increasing concentrations, (from 1% to 4%) for 40 min; C1, C2, C3, C4, C-4 = controls.

In vivo: values of P4, P5, and P7 were determined at 10 min after isoflurane exposure.

To increase the concentration of isoflurane from 1% to 4%, the gas mixture was increased in 0.5% increments.

1. Differences were also found between in vitro and in vivo effects of agents such as N₂O. Many of the inconsistencies may be accounted for by the oxidizing effect of inhalation anesthetics on the Clark electrode, which is used in P₅O₂ determination for the ODC; this was recognized only a few years ago. This phenomenon is quite evident in the range of potential differences in commercially available electrodes and causes determinations carried out in the presence of inhalation anesthetics to be quite unreliable.² This problem was bypassed by our method, which allows determination of the curve by measuring only oxygen saturation (Sₒ₂) and pH, values which are not altered in the presence of inhalation anesthetics.

Our investigation was carried out to determine the in vitro and in vivo effect of isoflurane on ODC of nine subjects of both sexes aged between 20 and 37 yr. The patients, classified as ASA I, had not taken any drugs for at least 3 months before surgery (appendectomy) and showed a normal basal P₅₀ ranging from 25.5 to 28.5 mmHg.

For the in vitro studies samples of venous blood (10 ml) were collected using heparinized, ice-cooled syringes on the day before surgery. Aliquots of 1 ml were used for basal ODC determination. The remaining 9 ml were used for the determination of ODC after exposure to isoflurane. Samples were placed in a series of 110 µl cuvettes covered with paraffin and maintained in ice-cold water. To determine the basal ODC, two microcuvettes were exposed to two humified mixtures of three gases, respectively, using a microtonometer kept at 37°C. The first mixture contained 3.8% O₂, 5.6% CO₂, and 90.6% N₂; the other one contained 4.9% O₂, 5.6% CO₂, and 89.5% N₂. Sₒ₂ and the pH were measured after 10 min. Further tonometry was carried out using a mixture containing 1.9% O₂, 5.6% CO₂, and 92.5% N₂ in aliquots in which Sₒ₂ had been found to be higher than 85% after exposure to 4.9% O₂. P₅₀ and Hill's n were calculated from the values found, using Hill's equation after having adjusted the pH to 7.4. For determining the effects of isoflurane on ODC, individual samples were exposed for 10 min to an isoflurane concentration ranging from 1% to 4%. One sample, in addition, was exposed to isoflurane in a continuous sequential concentration (from 1% to 4%) for a total of 40 min.

For the in vivo studies, a 5-ml sample (T0) of venous blood was collected on the day of surgery before administering 0.01 mg·kg⁻¹ of flunitrazepam. A second 5-ml sample (T1) was collected 15 min later. Anesthesia was induced by increasing inspired doses of isoflurane (from 0.25% to 4%) and oxygen. On disappearance of the eyelid reflex, anesthesia was maintained by 2% isoflurane in O₂ for 10 min, and a third 5-ml sample (T2) was collected. After succinylcholine administration (1 mg·kg⁻¹), orotracheal intubation and skin incision were finally carried out. Anesthesia was maintained with 1.5% isoflurane in air enriched with 60% oxygen, while muscle relaxation...
Airway Rupture with a Disposable Double-lumen Tube

To The Editor:—The recent report by Wagner et al. alerts us to a potentially serious complication of the new, disposable polyvinylchloride (PVC) double-lumen endobronchial tubes. Their observation of tracheal rupture follows another report of rupture of the left main-stem bronchus associated with a PVC double-lumen tube. In the latter case, as in most instances of airway trauma with double-lumen tubes, injury occurred from overdistention of the bronchial cuff.

When a PVC double-lumen tube is in proper position, the bronchial cuff usually requires less than 2 ml of air. If more than 3 ml is necessary to avoid a leak, the cuff is probably herniating above or is entirely in the carina. The low-pressure bronchial cuff of the PVC double-lumen tube assumes the high-pressure characteristics of red-rubber tube cuffs with volumes greater than 3 ml.* There have been several reports of airway rupture from double-lumen tube cuffs that were initially inflated with small amounts of air but then became further distended during prolonged nitrous-oxide anesthetics. If nitrous oxide is being used, it is important to deflate these cuffs periodically to avoid excessive pressure buildup on the bronchial mucosa. Nitrous oxide was not used in Wagner's case, and the intubation was apparently traumatic so the cause of their patient's airway injury remains a mystery.

It should be reemphasized that even with stiff, low-compliant, red-rubber double-lumen tubes, tracheobronchial rupture is a very infrequent occurrence. Guernelli et al. reported only five cases of airway rupture among 2,700 patients intubated with cuffed Carlen tubes. This rare complication, now reported with PVC double-lumen tubes, should not discourage anesthesiologists from routinely using these tubes to facilitate surgery during intra-thoracic procedures.

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REFERENCES
