INHALATION INDUCTION OF ANAESTHESIA WITH ISOFLURANE IN CHILDREN

Sir,—The article concerning the incidence of respiratory complications and hypoxic episodes during inhalation induction with isoflurane in children [1], raises several interesting points.

The authors compared the incidences of three respiratory complications: coughing, breath-holding and laryngospasm during induction of anaesthesia; unfortunately, these complications were not clearly defined. Were they severe enough to be clinically significant, and was intervention (100% oxygen, neuromuscular blocking drugs, tracheal intubation) required at any time? Clearly, all three may vary from mild to complete airway obstruction.

Warde, Nagi and Raftery [1] also presented the results of minimum arterial oxygen saturation (minimum SpO₂) during induction as incidence within the following ranges: < 71, 71-80, 81-90 and 91-100 %. The majority of the children were within the uppermost range. Why were these ranges chosen; could the range 91-100 % be further subdivided? Other investigators have used widely differing ranges: Sampaio and colleagues [2] used the ranges < 95 % and 95-100 % for minimum SpO₂ to compare airway complications, and Phillips, Brimacombe and Simpson [3] used an uppermost range of ≥ 85 % as part of a scoring system, but also presented the raw data for the minimum SpO₂ during induction.

At what point does oxygen desaturation become clinically significant? Motoyama and Glazener [4] investigated hypoxia in a group of children after general anaesthesia: 43 % developed an SpO₂ of ≤ 91 % in the early postoperative period. These authors stated that (assuming a normal acid-base balance and haemoglobin-oxygen affinity) an SpO₂ of < 91 % corresponds to a PaO₂ of < 8.0 kPa, which is the value at which the hypoxic ventilatory response becomes evident in awake adult humans. Two children in their study developed an SpO₂ in the range 70-74 %. Neither developed cyanosis, bradycardia or increased ventilation; one child was "lightly snoring", the other did not have any evidence of upper airway obstruction.

Finally, which pulse oximeter was used during the study by Warde, Nagi and Raftery [1]? Two recent articles [5, 6] have reviewed potential errors in pulse oximetry. I would suggest that errors in measurement during an investigation might be sufficient to alter the incidences of minimum SpO₂ values recorded.

Respiratory complications leading to "severe" obstruction result rapidly in oxygen desaturation in children, and pulse oximetry rightly has come to be regarded as essential monitoring during anaesthesia. The relationship between oxygen saturation and "minor" respiratory complications is less clear, and oximetry perhaps is less useful if it distracts the anaesthetist from assessing and managing the airway. Potential errors and significance of pulse oximetry during induction of anaesthesia must also be considered carefully both in clinical practice and in experimental study.

I. H. LEWIS  
Southampton

REFERENCES