Title: HYPOXEMIA IN CHILDREN FOLLOWING GENERAL ANESTHESIA

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INTRODUCTION In infants and children the resting lung volume (FRC) is reduced markedly under general anesthesia (1). Consequently, airway closure and increases in A-aDO_2 ensue which may continue into the postanesthetic period. Postoperative hypoxemia in adults has been reported (2). However, postoperative hypoxemia in infants and children, its frequency of occurrence, magnitude, and duration have not been well documented to date because of the lack of a noninvasive monitoring technique. Pulse oximetry, which has recently become available for clinical use (Nellcor Pulse Oximeter N-100, Haywood, CA) is a simple, noninvasive, and reliable method of measuring arterial oxygen saturation (SaO_2) using a fingertip sensor (3). The purpose of this investigation was to obtain such information in pediatric patients during the postoperative period.

METHODS As part of clinical monitoring, we measured pre- and postoperative SaO_2 using a pulse oximeter in 65 ASA PS: I infants and children with the average age of 4.4 years (range: 1 month to 17 years) who underwent simple, elective surgical procedures. Those patients who had surgery involving the thorax and abdomen were excluded. Fifty-five patients did not receive premedication, while the remaining 10 children received narcotics before or during anesthesia. Of the 55 without premedication, 45 patients received inhalation anesthesia (halothane or isoflurane with N_2O)only; 10 additional patients in this group received in addition non-depolarizing muscle relaxants which were reversed at the end of the surgery with atropine and neostigmine. In all patients SaO_2 was measured preoperatively by placing a sensor on a fingertip or a toe and the measurement was repeated postoperatively on the same site in the postanesthetic recovery room (PAR) shortly after arrival from the operating room. Monitoring of SaO_2 was continued intermittently until the patient was awake and active or until SaO_2 returned to the preanesthetic level. Those patients who required supplemental O_2 clinically (respiratory depression, airway obstruction, etc.) were excluded from this study. None of the children included exhibited a clinical sign of airway obstruction.

RESULTS AND DISCUSSION The average SaO_2 during the pre anesthetic period, when most of the patients were awake and calm, was 97.5 ± 0.22 (SEM)%. Postoperatively, on arrival to the PAR SaO_2 decreased to 93.3 ±0.54% (estimated PaO_2:70 torr, Severinghaus's nomogram) and ranged between 100% and 84% (PaO_2:51 torr). The reduction in SaO_2 was highly significant ($p < 0.001$). Five to fifteen minutes later (mean: 8.4 ± 0.5 minutes), SaO_2 increased slightly to 94.7 ± 0.5% but was still significantly ($p < 0.001$) lower than the preanesthetic levels. During this period SaO_2 increased in most of the patients who awoke (SaO_2: 95.6 ±7%) while in those who were still asleep hypoxemia persisted (SaO_2: 93.7 ± 0.7%). In these patients hypoventilation was not the cause of hypoxemia since their airways were patent clinically, ventilation seemed adequate, and transcutaneous PCO_2, when it was monitored, was below 40 torr. There was no correlation between the duration of anesthesia (10 to 158 min) and the severity of oxygen desaturation postoperatively, indicating that even 10 minutes of general anesthesia is sufficient to produce postoperative hypoxemia in room air. There was also no correlation between the age of the patients and the degree of postoperative hypoxemia, at least in our preliminary data. In the second group of 10 patients, who received narcotics and were sedate in the PAR, the mean SaO_2 (91.6 ± 2.8%) tended to be even lower than in those in the first group, who did not receive narcotics ($p < 0.1$). The lowest SaO_2 in this group was 74% (PaO_2:39 torr) in room air.

Thus most pediatric patients who are asleep and breathing adequately following general anesthesia, regardless of its duration, are likely to become hypoxemic even without premedication or narcotics. Since a trace concentration of inhalation anesthetics may depress hypoxic drive in these patients (4), there is a potential danger of further hypoxemia and respiratory depression. It appears imperative that all pediatric patients receive supplemental oxygen at least until they are awake and active in the PAR.

REFERENCES

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