

Title: EFFECT OF PREMEDICATION ON PREOPERATIVE ARTERIAL OXYGEN SATURATION IN CHILDREN WITH CONGENITAL HEART DISEASE

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INTRODUCTION: Arterial oxygen desaturation is a frequent finding in patients with congenital heart disease. Its magnitude is influenced by the relative changes in the systemic and pulmonary vascular resistances, as well as by oxygen consumption, respiration, and cardiac output. We and others have documented the high incidence of postoperative hypoxia after general anesthesia in children (1), but the effects of premedication on preoperative oxygen saturation in children have not been fully investigated. In adults undergoing coronary artery bypass grafting premedication resulted in significant decreases in oxygen saturation due to ventilatory depression during placement of invasive monitors (2). In children with congenital heart disease, the sedation and decrease in oxygen consumption caused by premedication may offset its respiratory depressant effect and thereby prevent arterial desaturation. This study was undertaken to determine the effects of premedication on the arterial oxygen saturation in pediatric cardiac patients about to undergo surgery.

METHODS: After approval from our institutional review board 20 patients, aged 3.5 months to 7 years, 10 with cyanotic congenital heart disease (CCHD) and 10 with noncyanotic congenital heart disease (NCHD) were studied. The age in months (mean \pm SE) in the CCHD and NCHD groups was 21.2 ± 5.8 and 41.0 ± 7.6 , respectively. Hemoglobin level was 17.9 ± 0.6 and 12.4 ± 0.2 g/dl, respectively.

Preoperative baseline SaO_2 (%) was measured with a pulse oximeter (Nellcor, Inc., Hayward, CA), usually on the right hand (preductal); if this was not possible, the left hand or a foot was used. Average dose of premedication, administered 1-2 h before surgery, was morphine 0.1 mg/kg, scopolamine 13 mcg/kg, and seconal 2.5 mg/kg given IM. There was no significant difference in dose between the two groups. The SaO_2 was recorded until the patient entered the operating room. The degree of sedation (1 = awake, 2 = tired, 3 = sleepy, 4 = asleep but arousable, 5 = asleep, nonarousable) and airway patency (1 = normal, 2 = snoring, 3 = severe obstruction) was also recorded. The paired Student t-test was used for statistical comparison.

RESULTS AND DISCUSSION: In the NCHD group the mean baseline SaO_2 was $97.8 \pm 0.4\%$ and the postpremedication SaO_2 measured during the deepest state of sedation was $96.6 \pm 0.4\%$ ($p < 0.01$). Eight of 10 patients scored 4 or higher on the sedation scale. Three patients snored; no patient had severe airway obstruction.

The mean baseline SaO_2 for the CCHD group was $70.5 \pm 3.8\%$, while the postpremedication value was $71.6 \pm 3.9\%$ ($p > 0.1$). All patients had patent airways. Seven of the 10 patients were sedated to score 4 or 5; in 5 of these patients (71%) SaO_2 increased, while in 2 it decreased. In 4 of these 7 patients, SaO_2 changed markedly ($\geq 10\%$ absolute change); in 2 of these SaO_2 decreased and in 2 it increased.

The slight but significant decrease in mean SaO_2 in the NCHD group was not clinically significant. The lowest SaO_2 in this group during deepest sedation was 94%, a decrease from 96%.

In the CCHD group the effect of premedication on SaO_2 appears to be unpredictable, as dramatic changes in both directions were seen. These results contrast with the findings of Stow et al. (3). We, therefore, recommend monitoring by pulse oximetry and/or supplying supplemental oxygen when patients with CCHD are given premedication.

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