Patients Under Sedation Should Always Be Monitored by Well-Trained Personnel and Should Be Given Supplemental Oxygen

Anique E. A. Hemelaar and Joris Lemson

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To the Editor.—

With great interest we read the article by Keidan et al1 concerning the use of supplemental oxygen during sedation in simulated pediatric patients. They concluded that “[h]ypoventilation and apnea are detected more quickly when patients undergoing sedation breathe only air” and that “[s]upplemental oxygen not only does not prevent oxygen desaturation but also delays the recognition of apnea.”

We congratulate them for their excellent study. Their work shows that pediatric patient simulation, apart from clinical training, can attribute to research in the pediatric field. For several reasons, however, we think their conclusions are debatable.

First, in their experiment, apnea was detected more quickly in patients breathing air only if the attending physician was inexperienced in monitoring a child under sedation. The anesthesiology residents detected apnea significantly faster (3.5–7 times), although their simulated patients received oxygen. Second, in the third group (trained anesthesiology residents), the transcutaneous oxygen saturation (SpO2) value at the time of clinical detection of apnea was higher than in the group without oxygen supplementation, although this was not statistically significant (P < .06). We believe this lack of significance is a result of a lack of power, because this subgroup was much smaller (n = 10 in group 3 versus 15 in groups 1 and 2). Increasing group 3 to n = 15 would most likely have lead to a significance in the SpO2 level. Also, it is unfortunate that Keidan et al did not include a fourth group of anesthesiology residents using only room air.

Among anesthesiologists it is well known that, especially in younger children, oxygen supplementation prolongs the time to desaturation when hypoventilation occurs.2,3 This is important, because it is hypoxia that is potentially life-threatening, not hypercapnia. Unlike Keidan et al we consider SpO2 monitoring to be useful only for monitoring oxygenation levels but not for detecting hypoventilation. The performance of the trained residents in anesthesia (group 3) underscores our opinion.

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Therefore, we think the most appropriate conclusion of the article would be that well-trained personnel detect hypoventilation and apnea much more quickly than untrained personnel irrespective of the use of supplemental oxygen. Hence, patients under sedation should always be monitored by well-trained personnel and should be given supplemental oxygen.

Anique E. A. Hemelaar, MD
Joris Lemson, MD
Department of Intensive Care Medicine
Radboud University Nijmegen Medical Centre
6500 HB Nijmegen, Netherlands

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In Reply.—
We appreciate the interest of Drs Hemelaar and Lemson in our report.1 We agree that our study supports the utility of pediatric simulators for clinical research and, perhaps, for a role in education and training monitoring skills. We have no disagreement with the notion that more experienced clinicians generally will perform better, at whatever task, than those with less expertise. Furthermore, proper preparation, proper evaluation, and proper recovery should help lead to safe and successful sedation of children.2

It is unrealistic to expect and impossible to provide anesthesia-trained personnel for every sedation case. Regardless, it is well understood that even experts may have substandard performance on occasion.3 Although Hemelaar and Lemson use oximetry only for oxygenation, in our opinion it is a missed opportunity to derive additional value from the device and to exploit the alveolar gas equation for the benefit of their patients. After all, hypoxemia in children in sedation scenarios is most commonly secondary to hypoventilation but has other causes as well. Simple supplementation with oxygen can mask conditions that would otherwise produce desaturation early after their occurrence. If supplemental oxygen is withheld, the pulse oximeter can serve as a redundant monitor of ventilation, complementing the bedside clinical skills.

Capnometry is likely used by most anesthesiologists, but this monitoring modality is seldom available or used by nonanesthesiologists. The incorporation into clinical care of strategies that will allow early detection of hypoventilation, such as withholding supplemental oxygen until it is needed, is an opportunity to improve the clinical surveillance provided by all who perform proce- 

Omega-3 Fatty Acids, Prematurity, and Autism

To the Editor.—
I was interested to read about the high prevalence of autism in children born prematurely and weighing <1500 g.1 Birth weight had more effect than gestational age, indicating nutritional and/or placental function factors. Chorioamnionitis was a strong risk factor, which could be both a stress effect and a placental dysfunction effect. Stressful situations such as chorioamnionitis and placental hemorrhage cause β2 receptor simulation, which also occurs with agents used to stop premature labor, such as terbutaline. Such stimulation increases the risk for autism.2 It would be interesting to know the incidence of terbutaline use in this study.

Another important potential cause of autism that would go along with low birth weight, prematurity, and
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