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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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Paul J. Sharek, MD, MPH
Division of General Pediatrics
Department of Pediatrics
Lucile Packard Children's Hospital
Stanford Medical School
Palo Alto, CA 94304

Laura K. Barger, PhD
Division of Sleep Medicine
Department of Medicine
Brigham and Women's Hospital
Harvard Medical School
Boston, MA 02115

Melanie Eisner, BSc
Department of Pediatrics
Children's National Medical Center
George Washington University
Washington, DC 20010

Sarah Edwards, BA
Division of Sleep Medicine
Department of Medicine
Brigham and Women's Hospital
Harvard Medical School
Boston, MA 02115

Department of Medicine
Children's Hospital Boston
Harvard Medical School
Boston, MA 02115

Vincent W. Chiang, MD
Department of Medicine
Children's Hospital Boston
Harvard Medical School
Boston, MA 02115

Bernhard L. Wiedermann, MD
Department of Pediatrics
Children's National Medical Center
George Washington University
Washington, DC 20010

Theodore C. Sectish, MD
Department of Medicine
Children's Hospital Boston
Harvard Medical School
Boston, MA 02115

Division of General Pediatrics
Department of Pediatrics
Lucile Packard Children's Hospital
Stanford Medical School
Palo Alto, CA 94304

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

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 led 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.

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-

dural sedation irrespective of their training background. To be clear, when desaturation develops, diagnosis and treatment of the problem causing the hypoxemia is warranted and likely includes oxygen administration as a temporizing measure. However, oxygen administration alone treats the hypoxemia without addressing the root cause.^{4,5} Therefore, we stand by our conclusions as stated.

Dietrich Gravenstein, MD

Department of Anesthesiology
University of Florida College of Medicine
Gainesville, FL 32610-0254

Ilan Keidan, MD

Avner Sidi, MD

Department of Anesthesiology
University of Florida College of Medicine
Gainesville, FL 32610-0254

Sheba Medical Center

Sackler School of Medicine

Tel-Hashomer 52621, Israel

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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 β_2 receptor stimulation, 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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