Letter to the Editor:

Etiology of Hypoxemia Often Overlooked

To the Editor:

I was struck by a seeming dichotomy in two sections of the Winter 2003-04 *APSF Newsletter*, the columns on postoperative hypoxemia vs. the current HRO initiatives.

To a critical care physician, it is readily apparent that in most clinical settings, it is not the **what** that is important, but the **why**. I can imagine few things more fundamental to accomplishing the goals of HRO than a thorough understanding of the physiologic mechanisms underlying any clinical scenario. Hypoxemia may be the best example, for the following reasoning: Hypoxemia, itself, is virtually benign medically. Rather, in almost all clinical scenarios, it is not the specific pO_2 of a patient that is important, but rather, why the pO_2 is what it is.

In Murphy and Vender's article reviewing the 2003 ASA scientific papers, they discuss a paper regarding postoperative hypoxemia. In this review, they note the cause (intensive opiate analgesia), but focus on the hypoxemia without noting that the real problem is the ventilatory defect. This is more than just semantics. Frequently, clinicians pay more attention to hypoxemia, thinking it deleterious; therefore they apply oxygen, without an understanding that it's not the secondary hypoxemia that will hurt the patient, but the primary defect (ventilatory or pulmonary parenchymal) that really needs to be addressed to "save" the patient.

In similar fashion, the ASA's *Practice Guidelines for Sedation and Analgesia by Non-Anesthesiologists* are remarkable for underemphasizing the importance of breathing. The guidelines even state, "If hypoxemia develops during sedation/analgesia, supplemental oxygen should be administered." In my experience, such a concept is a recipe for respiratory arrest. It is a common occurrence in the ICU to review a chart of a newly intubated patient from the floor who has hypoxemia documented in their chart, followed by the application of oxygen, without sufficient (or any) investigation of the cause of the hypoxemia. In the case of the above, I would suggest that during sedation/analgesia, if hypoxemia develops, the first and foremost emphasis should be immediate determination of the cause of the hypoxemia. If the patient's physical breathing appears adequate and the airway is determined to be patent, then the procedure can be continued with oxygen supplementation. In fact, the entire 14-page guideline document could arguably have been summarized by one short sentence: BREATHING (via a patent airway) IS THE ONLY THING THAT COUNTS.

In short, hypoxemia is virtually always a secondary issue, a simple but highly important signal that something is wrong. In terms of patient safety, the focus on hypoxemia itself, and consideration of it as harmful, is misplaced, and may even be injurious to the patient by distracting the clinician from a focus on the primary clinical problem, which may or may not be apparent. Subsequent application of oxygen may be doubly dangerous by raising the SpO_2, thereby masking, often temporarily, the underlying pathology. Not that oxygen should not be applied when the SpO_2 reaches some low level, but dogged attention must not be diverted from determining and addressing the cause of the hypoxemia.

In addition to the guidelines statement cited above, the guidelines also recommend that supplemental oxygen be administered to all patients undergoing deep sedation "unless specifically contraindicated." I'm not sure what the latter means, but the guidelines are silent on the complications of this practice, which render oxygen, in my opinion, one of the most dangerous drugs used in acute care medicine.

Specifically, in many settings, practitioners use the pulse oximeter as the de facto monitor of adequacy of ventilation, without realizing that they are doing so. By my observation, this includes many who are not anesthesia providers, yet practice sedation, along with nurses in the recovery room and ICU, and even during monitored anesthesia care. These individuals may fail to understand that only if the patient breathes room air does the SpO_2 correlate closely with alveolar ventilation (with only a lag of a few breaths). Once added inspired oxygen is applied, even one or two liters by nasal cannulae, the patient is moved to the right on the Hb-oxygen dissociation curve, and the pO_2 no longer linearly correlates with the SpO_2; the SpO_2 therefore no longer correlates with alveolar ventilation. In fact, the higher the percentage of inspired oxygen, the less the SpO_2 moves with even large changes in pO_2, and therefore pCO_2—all the way up to apneic oxygenation.

Thus, the use of oxygen may mask the onset and delay the recognition of inadequate ventilation, apnea, and/or airway obstruction, as detected by pulse oximetry. Accordingly, probably the best way for the non-anesthesia provider to stay out of trouble during IV sedation is to titrate drugs slowly to a patient breathing room air. The decline in SpO_2 to the point of the practitioner's comfort would thus preclude further administration of drug doses or combinations that would cause further hypoventilation or apnea. (Note that the level of induced hypoventilation directly reflects the degree of sedation; in turn, in a sedated patient breathing room air, the SpO_2 correlates with the ventilatory status. Thus, in such a patient the SpO_2 offers an indirect but quantitative measure of the patient's level of sedation, and as a practical matter, sedative administration can therefore be titrated to the SpO_2, but only if the patient is breathing room air.)

For those who have any doubt about the benign consequences of hypoxemia itself, the following reports and case histories from anesthesiologists and critical care physicians from around the world who participate in the GasNet and CCM-L web-discussion groups are presented for consideration:

- Fourteen-year-old with severe hypoxic ephalopathy after choking. After terminal extubation, SpO_2 25-40% for 18 hours. Normal HR and BP, good perfusion, normal ECG, no metabolic acidosis on ABG despite a PaO_2 of 24 mmHg (pH 7.30, $PaCO_2$ 59).
- The FAA requires that flight crews wear oxygen only for altitudes above 12,500 feet. For non-pressurized aircraft, the FAA requires that passengers be offered supplemental oxygen for altitudes only above 14,000 ft, which corresponds to a SaO_2 below 82%.
- "In research sleep studies, saturations varied in the 80-90% range. People with sleep apnea go for many years prior to diagnosis, spending most of every night with saturations lower than that, and it doesn't bother them very much. And they are mostly people with co-morbidity as well. I've spent a number of experimental periods myself with a saturation of 80% and barely noticed the difference."
- "When flying at 5500 feet my SpO_2 was 91%. I remember thinking to myself that here I was, making life-threatening decisions in real-time, with a PaO_2 at the low end of the scale."
- "My little pulse oximeter has traveled widely and highly. This pulse oximeter was originally purchased for studies on Mt. McKinley at 14,000-18,000 ft, and accompanied me on a highly enlightening trip to Bolivia. In sea-level Miami I boarded the plane at 98% saturation, by mid-flight it had dropped to 85%, and by the time I got off in La Paz the next morning, my oxygen saturation had dropped to 60%. I carried my own luggage through customs. I felt short of breath but euphoric and herculean. The average O_2 saturations of lifelong residents of Lake Titicaca (13,000 ft) and Bolivia's Altiplano (13,000-17,000 ft) were 82-85%."
- "Hiking the Inca trail, I found that my wimpy saturations in the mid-high 80s were for unknown reasons higher than those of most of the Quechuan porters."
- "Annually in Colorado at Independence Pass (roughly 12,000 ft), I spend a couple of hours tricking tourists into exercising while wearing a

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pulse oximeter. O_2 saturations run as high as 90% or as low as the mid-70s. Most are in the high-80s."

- "The take-home message to me, however, was that people function quite well with traditionally considered "inadequate" saturation. Or at least they can be easily duped into carrying their own luggage."

Proof of lack of detriment does exist. There have been studies in thousands of patients showing that when anesthesiologists are not aware of the SpO_2, moderate levels of hypoxemia (i.e., SpO_2s in the 80s) occur commonly, with no adverse effect to patients.[1,2]

From the above and other studies, and from knowledge and insight gained since the advent of pulse oximetry in the OR, there can be little doubt that during the majority of years that anesthesiology has been practiced (i.e., from the mid 1800s to the mid-1980s), episodic undiagnosed hypoxemia was rampant, yet patients weren't dying, having myocardial infarctions, or stroking out by the hundreds, unless the hypoxemia was prolonged. The latter cases virtually always occurred as a result of a major ventilatory problem, recognized or not (in particular, unrecognized esophageal intubation, apnea, or airway obstruction).

There is no consistent evidence, even in patients with stable coronary artery disease, that low levels of acute hypoxemia (or even chronic hypoxemia in some settings) are, in themselves, dangerous. On the contrary, there is much anecdotal evidence of no harm coming to such patients. Evidence from the years previous to the development of the pulse oximeter would overwhelmingly corroborate that. What is potentially dangerous are the reasons for which the SpO_2 drops acutely in acute care settings.

Finally, "A Focus on History" (*ASA Newsletter* September 2001) states: "Inhalation anesthesia of the early 1920s consisted of either breathing anesthetic gases and vapors via a mask and bag or by open-drop of volatile liquids (ether or chloroform) on a gauze mask. There were no intravenous agents to speed induction. One hundred percent nitrous oxide was administered for gas induction and attainment of maximum anesthesia. Induction was usually accomplished within 2 to 3 minutes and was followed by addition of 10 percent to 15 percent oxygen, or more, to avoid cyanosis. 'Too much oxygen' was shunned to avoid diluting the nitrous oxide ."

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2. Moller JT, Johannessen NW, Espersen K, et al. Randomized evaluation of pulse oximetry in 20,802 patients: II. Perioperative events and postoperative complications. *Anesthesiology* 1993;78:445-53.

Editor's Note: ***While this letter makes several valid and important points, it is important to NOT withhold supplemental oxygen from those patients for whom it is indicated by clinical judgment. The*** **APSF Newsletter** ***invites readers' thoughts and comments on this topic.***

APSF Executive Committee Invites Collaboration

From time to time the Anesthesia Patient Safety Foundation reconfirms its commitment of working with all who devote their energies to making anesthesia as safe as humanly possible. Thus, the Foundation invites collaboration from all who administer anesthesia, and all who provide the settings in which anesthesia is practiced, all individuals and all organizations who, through their work, affect the safety of patients receiving anesthesia. All will find us eager to listen to their suggestions and to work with them toward the common goal of safe anesthesia for all patients.

Proper Patient Selection Most Important With Forehead Sensor

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above the eyebrow so that it is centered slightly lateral of the iris. Placement also includes use of the headband, as Branson's work indicates. Patient position for use of the forehead sensor *excludes* patients in Trendelenberg's position due to venous pulsations. The product literature carries this admonition, but its importance may have not been given due emphasis in clinical settings. Recently published studies of poor performance with forehead monitoring all tend to overlook the importance of placement site, patient orientation, and headband use.[10-12]

A third and likely most important consideration is appropriate patient selection. With cost containment an abiding consideration, clinicians must be mindful that, no matter how convenient—site access on the forehead, rather than buried under surgical drapes—the forehead sensor is not for general use. It is intended for patients with poor peripheral circulation. In this group of patients the advantages of the forehead sensor have been well-documented.

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