Postoperative Supplemental Oxygen and High Intraoperative Inspired Oxygen: Effect and Cause?

To the Editor:

The optimal concentration of oxygen that should be delivered during a routine anesthetic is a controversial issue that continues to provoke interest, debate, and wide variations of clinical practice. Given that evidence has been published reporting both benefit and harm from high inspired concentrations of oxygen (F_IO_2), we paid particular attention to the article recently published by Mackintosh, *et al.*[1] that presented data addressing this controversy. These authors randomized patients to four treatment groups, on the basis of intraoperative oxygen supplementation, as well as level of positive end-expiratory pressure (PEEP), and analyzed the effect of these variables on the likelihood for postoperative hypoxemia and need for inspired oxygen supplementation. In short, the article presented evidence that, in low-risk operations, neither the provision of high F_IO_2, nor the provision of 0–5 cm H_20 PEEP had a demonstrable effect on the need for postoperative inspired oxygen supplementation. These authors further speculated that absorption atelectasis induced by high concentrations of inspired oxygen was "not sufficient to produce hypoxemia" beyond that associated with routine anesthetic care.

Although we share these authors' belief in the importance of this topic as an area for scientific inquiry, we do not agree that this study adequately addresses the controversy. It is important to note that all patients in this study received preoxygenation with 100% oxygen for "at least 3 min" before induction of general anesthesia; after placement of a tracheal tube, each patient was immediately ventilated according to the group randomization (0.9 *vs.* 0.3 F_IO_2; 3–5 cm H_20 *vs.* zero PEEP).

The effect of such a preoxygenation strategy on absorption atelectasis has been studied, and recently reviewed.[2] With as little as 3 min of preoxygenation with 100% oxygen, patients have been reported to have, on average, 15–20% atelectasis of total lung tissue.[3] As a similar strategy was adopted by Mackintosh, *et al.*, we speculate that all patients in all study groups had significant atelectasis before implementation of the study protocol. In addition, as all patients received 5 cm H_20 PEEP or less, and no alveolar recruitment maneuvers were performed, any such atelectasis would be unlikely to be reversed during the surgical procedure.[4] Finally, even if recruitment and adequate PEEP had been implemented, the second period of oxygen breathing at the end of the anesthetic would have eliminated any beneficial effect thereof.

Pulmonary atelectasis constitutes areas of zero ventilation/perfusion ratios that affect hypoxemia unresponsive to a small increase in F_IO_2. Hence, atelectasis cannot be quantified with oxygen titration as described by Macintosh *et al.* Because the effects of atelectasis on oxygenation should not be reversible with low-flow oxygen *via* a nasal cannula, another factor must explain the hypoxemia detected in practically all their patients. Of the two remaining likely causes, low but finite ventilation/perfusion ratios and hypoventilation, we suggest the latter, primarily because the hypoxemia was short lived and readily reversible with little supplemental oxygen. Unfortunately, Macintosh *et al.* provide no information on the adequacy of postoperative ventilation in their patients.

After a properly managed anesthetic in patients with normal preoperative lung function, few patients need oxygen supplementation to maintain adequate oxygen saturations in the recovery room.[5] Room air breathing also carries the added benefit of alveolar stability and allows the pulse oximeter to reflect adequacy of ventilation. Judicious use of oxygen pre, intra, and postoperatively, and proper management of ventilation, are known to minimize atelectasis throughout the perioperative period. Unfortunately, the study by Macintosh *et al.* provides neither an adequate control group nor the tools to detect any postoperative detrimental effects of hyperoxygenation, and thus leaves the central hypothesis of the article untested.

Michael E. Nemergut, M.D., Ph.D.,* Jukka Räsänen, M.D., John B. Downs, M.D. *Mayo Clinic, Rochester, Minnesota. nemergut.michael@mayo.edu

References

1. Mackintosh N, Gertsch MC, Hopf HW, Pace NL, White J, Morris R, Morrissey C, Wilding V, Herway S: High intraoperative inspired oxygen does not increase postoperative supplemental oxygen requirements. Anesthesiology 2012; 117:271–9
2. Hedenstierna G: Oxygen and anesthesia: What lung do we deliver to the post-operative ward? Acta Anaesthesiol Scand 2012; 56:675–85
3. Edmark L, Kostova-Aherdan K, Enlund M, Hedenstierna G: Optimal oxygen concentration during induction of general anesthesia. Anesthesiology 2003; 98:28–33
4. Tusman G, Böhm SH, Vazquez de Anda GF, do Campo JL, Lachmann B: 'Alveolar recruitment strategy' improves arterial oxygenation during general anaesthesia. Br J Anaesth 1999; 82:8–13
5. Fu ES, Downs JB, Schweiger JW, Miguel RV, Smith RA: Supplemental oxygen impairs detection of hypoventilation by pulse oximetry. Chest 2004; 126:1552–8

(Accepted for publication November 28, 2012.)

In Reply:

We appreciate the thoughtful comments of Dr. Nemergut. Although we agree our study does not provide a final answer, it does add evidence for the safety of using greater than 90% inspired oxygen intraoperatively. Dr. Nemergut raises several issues, which we will address in turn.

As we noted in the original article[1], preoxygenation for induction and emergence are confounding variables in our study. Given the safety margin provided by preoxygenation during these two critical periods, we felt it would not be ethical to include a control group with reduced inspired oxygen during induction and emergence. Excluding potential subjects with recognized potential difficult airway management would have reduced our ability to recruit subjects and the generalizability of the results. Because use

of 100% oxygen during induction and emergence is standard practice for the vast majority of anesthetists, our study reflects the impact of high intraoperative inspired oxygen in actual practice.

The use of 100% oxygen for induction and emergence also likely had minimal effect on our results because the degree of atelectasis induced by administration of 100% oxygen for only a few minutes is relatively minor. As noted in our article, Edmark *et al.*[2] (which is reference number 3 in Dr. Nemergut's letter) found 1–20% atelectasis in subjects preoxygenated with 100% oxygen whereas Benoit *et al.*[3] found approximately 8% in subjects administered 100% oxygen for 10 min before emergence. Of note, in the preoxygenation study, volunteers in the 100% group were apneic for approximately twice as long (7 min *vs.* 3.5 min) before the measurement of atelectasis, which may have exaggerated the effect of 100% oxygen.

Dr. Nemergut notes that the degree of atelectasis cannot be quantified with oxygen titration, and we agree. Oxygen supplementation in our study was used as a safety measure to prevent hypoxemia, because supplemental oxygen can overcome the combined effects of atelectasis and hypoventilation. Although at sea level it is likely we could have safely obtained room air arterial oxygen saturation by pulse oximetry measurements in most subjects,[4] this is not the case at our hospital, which is at an altitude of approximately 4,700 feet (1,433 m). Barometric pressure averages 635 mmHg (85 kPa). During room air breathing at this pressure, even mild hypoventilation (arterial partial pressure of carbon dioxide ~ 45 mmHg), likely present in all patients in the Post-Anesthesia Care Unit, makes hypoxemia likely: alveolar partial pressure of oxygen = 0.21(635–647) – 45/0.8 = 67 mmHg.

Therefore, the requirement for supplemental oxygen in our subjects does not suggest greater than normal hypoventilation or unusual anesthetic management. As noted in our article, oxygen requirement was minimal in all but a handful of subjects, in whom more severe hypoventilation and worse preexisting lung function were common, but there was no relationship with intraoperative inspired oxygen concentration. Although supplemental oxygen interferes with detection of hypoventilation (but not hypoxemia) by pulse oximetry,[4] hypoventilation can be detected by other monitors, and low dose (<30%) supplemental oxygen provides a safety margin for postoperative patients[5,6] in whom atelectasis and hypoventilation are common and difficult to avoid completely.

In conclusion, despite some limitations, our published randomized controlled trial adds to the evidence supporting a lack of harm from brief exposures to inspired oxygen concentrations greater than 90%.

Matthew C. Gertsch, M.D., Natalie Mackintosh, M.D., Harriet W. Hopf, M.D.* *University of Utah School of Medicine, Salt Lake City, Utah. harriet.hopf@hsc.utah.edu

References

1. Mackintosh N, Gertsch MC, Hopf HW, Pace NL, White J, Morris R, Morrissey C, Wilding V, Herway S: High intraoperative inspired oxygen does not increase postoperative supplemental oxygen requirements. Anesthesiology 2012; 117:271–9
2. Edmark L, Kostova-Aherdan K, Enlund M, Hedenstierna G: Optimal oxygen concentration during induction of general anesthesia. Anesthesiology 2003; 98:28–33
3. Benoît Z, Wicky S, Fischer JF, Frascarolo P, Chapuis C, Spahn DR, Magnusson L: The effect of increased FIO(2) before tracheal extubation on postoperative atelectasis. Anesth Analg 2002; 95:1777–81
4. Fu ES, Downs JB, Schweiger JW, Miguel RV, Smith RA: Supplemental oxygen impairs detection of hypoventilation by pulse oximetry. Chest 2004; 126:1552–8
5. Stone JG, Cozine KA, Wald A: Nocturnal oxygenation during patient-controlled analgesia. Anesth Analg 1999; 89:104–10
6. Govinda R, Kasuya Y, Bala E, Mahboobi R, Devarajan J, Sessler DI, Akça O: Early postoperative subcutaneous tissue oxygen predicts surgical site infection. Anesth Analg 2010; 111:946–52

(Accepted for publication November 28, 2012.)

Resident Research and Graduate Medical Education Funding

To the Editor:

I sincerely enjoyed the recent article by de Oliveira *et al.*, which analyzed various factors associated with successfully matching to a residency in anesthesiology.[1] I also enjoyed the accompanying Editorial, written by four academic leaders in our specialty.[2] I strongly agree with the editorialists' sentiment that the future of anesthesiology must be built upon scholarly investigation into the basic and clinical sciences.

As the editorialists do not specifically articulate it, it is important to remind the readers of the complex process by which Graduate Medical Education is funded in the United States and how this process may affect research during residency training. The Center for Medicare & Medicaid Services (CMS) makes two types of Graduate Medical Education payments to support residency programs and teaching hospitals. Direct Graduate Medical Education payments compensate teaching institutions for costs directly related to resident education (*e.g.*, resident salaries). Indirect Medical Education payments are intended to compensate teaching hospitals for higher inpatient costs and are calculated as a percentage add-on to basic Medicare per case diagnosis-related group payments. In 2011, CMS Direct Graduate Medical Education and Indirect Medical Education payments totaled approximately $3 billion and $6.5 billion, respectively.

To the surprise of many, CMS does not automatically continue to fund a resident if he/she decides to participate in research during the course of residency training. In