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.

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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