In Reply:

We thank Dr. Kempen for his interest in our study[1] and questions and we have attempted to address them in order. The baseline minute ventilation was as given in table 1 for both groups. The ventilator was a Datex-Ohmeda Aestiva ventilator set (GE Healthcare, Madison, WI) during surgery and emergence to volume control with an inspired:expired ratio of 1:2. It has compliance compensation functionality, but despite this there were significantly higher expired tidal volumes in the N_2O group during the first 5 min of emergence phase (118 ml higher at 1 min and 110 ml at 5 min, $P < 0.05$ at each time point with Bonferroni correction), explained by the rapid volume washout of N_2O.

In the control group there were six abdominal, three head and neck, and one orthopedic surgeries, with one patient in the prone position. In the N_2O group there were five abdominal, two spinal, and three orthopedic surgeries, with two patients in the prone position.

The means (SD) of the blood/gas partition coefficients were 0.73 (0.14) for the control group and 0.69 (0.18) for the N_2O group ($P = 0.37$ on the two-tailed Student *t* test). The trend toward a higher partition coefficient in the control group might suggest that the difference in blood partial pressures between the two groups we measured was a slight underestimate.

Ventilatory and hemodynamic disturbances due to coughing, straining, *etc.*, would have disrupted gas elimination and affected our results, as Dr. Kempen points out. We deliberately restricted data sampling during emergence to the first 5 min after cessation of anesthetic gas administration, and at 30 min in the postanesthesia care unit so that this was successfully avoided. We agree that an unblinded study of time to emergence is prone to observer bias, but we found time to eye opening to command a robust endpoint, and time to extubation correlated closely with this. We reiterate that these were secondary endpoints in the study, but were prospectively studied in our protocol, and so were reported.

We believe our intention to fashion the study around a typical general anesthetic protocol with similar depth of anesthesia across the two groups was a sensible one. The different sevoflurane concentrations in the two groups were an inevitable consequence of this, as in standard anesthetic practice. Given that the *relative* change from baseline in sevoflurane partial pressures was the primary outcome variable, we believe this approach was appropriate. Dr. Kempen's suggestion for a study using identical sevoflurane concentrations and balancing depth of anesthesia with propofol in the control arm is a valid one, and we look forward to the results of such a study. However, we would encourage researchers in the field to take the trouble to measure blood partial pressures rather than just expired concentrations in these types of pharmacokinetic investigations, because of the significant effect of ventilation-perfusion scatter on alveolar-arterial partial pressure gradients. More meaningful quantitative data are obtained and the implications of the findings of a study are clearer.

Finally, we agree entirely that it is possible to accelerate elimination of volatile agent and emergence by deliberately increasing expired alveolar ventilation, which emulates the effect of N_2O washout. As Dr. Kempen proposes, turning on N_2O near the end of surgery is a useful maneuver that the primary author frequently uses to gain the benefit of the minimum alveolar concentration-sparing and washout effects without the potential side effects of prolonged N_2O administration. The intent of our study was to demonstrate the pharmacokinetic principles underlying the administration of inhalational anesthesia with N_2O, and its potential implications for speed of emergence. Clearly, the detailed conduct of anesthesia so as to achieve rapid and smooth emergence is a larger issue than simply whether or not to include a single agent such as N_2O, and this complex formulation should always be left to the judgment of the skilled anesthesiologist.

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Evidence Does Not Show that Pregnancy Is a Risk Factor for Latex Allergy

To the Editor:

I read with interest the report by Draisci *et al.* on the prevalence of latex allergy in obstetrical patients.[1] Although there was a statistical difference between the obstetrical patients (OB) and the nonobstetrical women (non-OB), I must disagree with the author's conclusion that the results prove that higher prevalence is due to the factor of pregnancy alone.

Unfortunately, the two groups were not matched for risk factors. As noted in the same issue of ANESTHESIOLOGY, Sampathi and Lerman described the risk factors for developing latex allergy: congenital abnormalities (spina bifida, genitourinary abnormalities), multiple surgeries, atopy to drugs or food, exposure to latex, and healthcare worker.[2]

In table 1, Draisci *et al.* list the prevalence of latex sensitization and the risk factor prevalence. The authors did not find a statistical difference for risk factors between the two groups (OB and non-OB). But if one looks closer, one would see that there might be a difference that could result in the difference in latex sensitization found.

In absolute numbers, the OB group had 15 patients who were positive for latex sensitization, whereas the non-OB group had 5 such patients, with both groups having 294 patients in each. In other words, the OB group had 10 more patients who tested positive for latex allergy than did the non-OB. Looking at the risk factors listed, one finds that the OB group had six to seven more patients with positive results than did the non-OB: specifically, drug allergy (atopy), seven more; food allergy (atopy), six more; other allergy (atopy), seven more; multiple surgeries, six more; and healthcare workers, six more. If this difference of six to seven patients accounts for the majority of difference in latex sensitization, the findings of higher prevalence in the OB group is because of the higher prevalence of risk factors.

Unfortunately, the authors do not discuss this confounding issue in their report. Thus, I must conclude that the authors did not have enough evidence to make the conclusion that pregnancy is a risk factor for latex allergy.

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Remove Latex from the Labor and Delivery Suite

To the Editor:

The recent article by Draisci *et al.*[1] showing an increased incidence of increased serum concentrations of specific rubber latex immunoglobulin antibodies among pregnant women is very important. We were interested, however, to know whether the two patients who actually exhibited anaphylaxis had increased concentrations of latex immunoglobulin E antibodies and/or positive latex skin tests. In the methods, it is noted that "skin-prick tests and intradermal tests with oxytocin or other drugs administered in the study were performed to exclude drug allergy in patients who experienced adverse reactions." It is possible that these two patients could have in fact been allergic to other allergens and this information was not reported. The treatment of anaphylaxis, especially in a pregnant patient with a potentially difficult airway, who is exhibiting facial edema and "throat closure," may also require adrenaline and a low threshold for intubation.[2] It would also be of interest to know whether among the pregnant women with latex hypersensitivity the serum concentrations of rubber latex immunoglobulin E became normal after pregnancy. Although the reasons behind the increased serum concentrations of rubber latex immunoglobulin E, potentially increasing the incidence of latex hypersensitivity among pregnant women, are pure speculation, as discussed,[1] the danger is clear. The way to avoid this life-threatening problem altogether is to remove latex (gloves or catheters) from the operating room in the labor and delivery suite.

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In Reply:

We appreciate Dr. Abouleish's deep attention in revising our article[1] and we thank him for his comments. To investigate a possible history of allergy in our patients, we designed our questionnaire according to data in the literature.[2–5] All risk factors (multiple surgical procedures, high-risk work, atopy, cross-reacting fruits/vegetables, previous history of allergy) associated with latex sensitization were analyzed. The same factors were recently described by Sampathi and Lerman as risk factors for developing latex allergy in children.[6] In table 1, we reported the statistical differences between pregnant and nonpregnant patients. Even if the two groups showed different frequencies or means for all variables, those differences were not significant ($P > 0.05$), that is, the pregnant and nonpregnant groups were omogenous. In contrast with previous data reported by Chen *et al.*,[7] we found no significant correlations between accepted risk factors and latex sensitization in our study.

We also thank Dr. Weiniger for the interest in our work. In our data, the two patients who experienced an adverse reaction previously experienced allergic disease and hand hitching after the use of rubber gloves. In studies performed before surgery, both patients revealed a sensitization to latex, presenting with a latex immunoglobulin E serum concentration of 100 kilo units/l and 5.33 kilo units/l, respectively. After adverse reaction, skin-prick and intradermal tests were performed to detect latex allergy: both tests were positive. Oxytocin and other drugs were administered and tested, and other drug allergies were excluded. After pregnancy, high-latex immunoglobulin E serum concentration was reported, and the patients were managed with desensitizing treatment.