

reassuring them through touch and eye contact were able to reduce the child's distress. It may be that effective methods of training can be developed for parental presence during induction of anesthesia. Therefore, in our center, we do not offer parental presence to all patients, but rather respond to each request based on the individual child, parent, and anesthesiologist.

We agree that separation anxiety is a major problem after surgery in children. This postoperative separation anxiety, however, is reflective of the behavioral response of the child to the entire perioperative experience and not only to the preoperative separation period. Therefore, to conclude that by preventing preoperative separation we will in fact prevent postoperative separation problems may be premature. Moreover, in two previous randomized controlled trials involving parental presence, we followed-up children for 2 weeks after surgery,^{3,4} measuring postoperative behavior with the Post Hospitalization Behavior Questionnaire. We demonstrated that children whose parents were present during induction of anesthesia were equally as likely to develop postoperative separation anxiety as children who were not accompanied by a parent. Therefore, we must deduce, based on the scientific data, that parental presence during induction of anesthesia does not decrease the incidence of postoperative behavioral changes in general, and postoperative separation anxiety in particular.

In conclusion, we believe that parental presence during induction of anesthesia may have a place in a child's perioperative experience, but significant work is needed to determine what role parents should play and how best to prepare parents to be most helpful to their children in the operating room setting. As it stands, parental presence increases parental satisfaction⁹ but does not affect a child's immediate perioperative anxiety or long-term behavior.

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Causes of Nitrous Oxide Contamination in Operating Rooms

To the Editor:—We read with interest the investigation revealing causes of nitrous oxide contamination in operating rooms.¹ The authors observed occupational exposure to trace amounts of a waste anesthetic gas, nitrous oxide, and showed a number of sources that were responsible for abnormally high workplace concentrations. In addition to insufficient or lacking air conditioning systems and scavenging devices, inhalational mask induction and leakage during use of uncuffed tubes have widely been proved as the most important factors with regard to exposure to both nitrous oxide and volatile agents.^{2,3}

However, we feel some points of the recent study require further discussion. The air samples were taken at the air conditioning exhaust grill at a distance of approximately 3 m from the sources of contamination. Therefore, the measurements only reflect air contamination at

a given point, not actual exposure of an individual, which is far more important in the evaluation of workplace safety and eventual health hazards. Actual exposure to an individual was not measured because anesthetic gases are distributed within the room and thus—depending on the distance from the source of contamination—are diluted in significant manner.^{3,4}

To estimate dilution of nitrous oxide, we checked leakage 62 wall-mounted gas outlet sockets (Draeger, Luebeck, Germany) that provide nitrous oxide from the high-pressure central gas system to the anesthesia machines in 17 operating rooms in our hospital. All rooms were well air conditioned by laminar flow and an air exchange rate ranging from 19.2-21.3/h without recirculation of exhaust air. Measurements were taken continuously for 6 min with a directly displaying infrared

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spectrometer (Brüel & Kjaer 1302, Naerum, Denmark) at a distance of 1 cm from the sockets. To determine the baseline contamination of the operating rooms, levels of nitrous oxide were measured in the middle of the rooms, approximately 3.5 m away from the sockets.

Our measurements revealed that no socket was absolutely gas-tight. Mean leakage was 181.6 ± 624.0 ppm (range, 1.6–4,730.2 ppm). Despite high concentrations of nitrous oxide close to the sockets, overall room contamination was low, which can be attributed to dilution. In the center of the operating rooms, nitrous oxide levels were only 4.38 ± 2.30 ppm and showed that at a distance from the leakage comparable to that in which Kanmura *et al.* took their air samples, the concentrations were approximately 40-fold lower than those measured close to the leakage. Therefore, in the recent study, exposure of personnel who worked close to the sources of contamination could have been much higher than the concentrations actually measured at the exhaust grill.

Although having already undergone distribution and dilution, concentrations of nitrous oxide at the exhaust grill were disproportionately high when compared with recent studies conducted in equally climatized operating rooms.^{3–5} Such high exposure may be plausible only in cases of undetected massive leakage from the central gas system or malfunction of air conditioning or scavenging devices,⁶ which resulted in relatively high baseline contaminations of the operating rooms close to the 50-ppm threshold set by the authors.¹ During use of nitrous oxide, relatively low concentrations from anesthesia itself added to the high baseline level and triggered alarm. Furthermore, high concentrations may result from the careless use of nitrous oxide by anesthesiologists.^{3,6}

Nonetheless, the recent study shows that there is still a lot of work to be done from the standpoint of occupational health and workplace safety. Furthermore, our results show that there are undetected sources of leakage that led to occupational exposure. Because exposure to naturally produced nitrous oxide is estimated to be only approximately 0.31 ppm,⁷ occupational exposure in contaminated rooms caused by leakage in our study was approximately 12 times as high. Thus, air-conditioning systems and scavenging devices, as well as

anesthetic equipment, should be checked in short intervals to prevent or repair malfunction and leakage. Only increased awareness of this problem will keep occupational exposure as low as possible.

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In Reply:—We thank Drs. Byhahn and Westphal for the attentive reading they have accorded our article.¹ We agree with their comment that our data represent air contamination at the air conditioning exhaust grill, not the concentration around the medical workers in the operating rooms. Because of the air conditioning system present (laminar flow system), the concentration of contaminant anesthetic gas near the anesthesiologists is bound to be greater than at the periphery of the operating rooms.

We did not measure regional differences in contaminant anesthetic gases in our operating rooms, but Wood *et al.*² reported that the nitrous oxide exposure of anesthesiologists was five to six times higher than the level at the air conditioning exhaust grill. On this basis, the actual exposure of individuals in the operating rooms might well be greater than we reported. We should again like to emphasize the frequent occurrence of anesthetic gas contamination in operating rooms in routine practice.

To estimate the dilution of contaminant anesthetic gases in operating rooms, Byhahn and Westphal investigated the difference in nitrous

oxide concentration between sites close to the wall-mounted gas outlet sockets and sites in the middle of the operating room. They reported a 40-fold difference between these two locations, but such a large difference will not be applicable to our investigation. In operating rooms, air flow is usually from the center of the ceiling to the periphery near the floor. In their study, the source of nitrous oxide contamination was at the wall, so little nitrous oxide would have been able to reach the center of the room against the air flow. In the clinical situation, Wood *et al.*² reported a regional difference in nitrous oxide concentration in operating rooms of fivefold to sixfold.

Byhahn and Westphal claim that our data for the concentration of nitrous oxide at the exhaust grill are disproportionately high,³ and they attribute this to a possible undetected massive leakage of nitrous oxide from the central gas system or malfunction of air-conditioning or scavenging devices. For the purposes of our study, we actually checked the baseline concentrations of nitrous oxide in our operating rooms, and the average concentration was 3.0 ppm. There was no

massive leakage of nitrous oxide from the central gas system or malfunction of air-conditioning or scavenging devices. Furthermore, the values for contaminant nitrous oxide in clinical practice reported by other investigators are in the same range as those reported in our article. For example, Wood *et al.*² reported that the mean concentration of nitrous oxide at the exhaust grill during pediatric anesthesia was 87 ppm with the use of scavenging system. Kant *et al.*⁴ reported a mean time-weighted concentration of nitrous oxide in the periphery of their operating room of 41.2 ppm, whereas Davenport *et al.*⁵ reported that the mean concentration of nitrous oxide at the periphery of the operating room was 136 ppm without scavenging precautions, but this value was reduced to 13 ppm with an active scavenging system. To judge from these reports, we do not think that our values are disproportionately high.

In our study, we did not attempt to measure the actual concentrations of anesthetic gases near the medical workers in the operating rooms; instead, we assessed the frequency with which contamination occurred, and we established that nitrous oxide contamination was common during routine circumstances. The other point we would like to emphasize is the important role of scavenging systems in preventing anesthetic gas contamination.

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Minimizing Venous Air Embolism from Reinfusion Bags

To the Editor:—Drs. Ho and Ling's excellent Medical Intelligence article, "Systemic Air Embolism after Lung Trauma,"¹ provides an important teaching service to the anesthesia community. However, one totally preventable and recurring cause of potentially fatal venous air embolism that was not mentioned in the article (particularly in the last section entitled, "Other Causes of Air Embolism in Nontraumatic and Traumatic Clinical Setting") is externally pressurizing a reinfusion blood bag that has been filled with blood from a surgical field scavenging-blood processing system. In most of the Haemonetics blood scavenging-processing models (Cell-Saver; Haemonetics Corp., Braintree, MA), the unit sends an 80-ml column of air ahead of the first column of blood into the reinfusion blood bag. If the reinfusion blood bag, which contains air, is then externally pressurized, a venous air embolism may occur. The obvious, but life-saving, take-home message of these considerations is that reinfusion blood bags that have been filled with blood from an autotransfusion system should never be externally pressurized.

The old warning on the Haemonetics Cell-Saver reinfusion bag of "Do Not Use With Pressure Cuff" was changed to "Do Not Use Pressure Cuff. The Use of Pressure Cuff May Lead to Fatal Infusion of Air" in 1995 for obvious reasons. However, I am aware that the practice of externally

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pressurizing Cell-Saver reinfusion blood bags is still common and widespread. Consequently, other additional solutions to minimize the risk of fatal venous air embolism appear to be desirable and necessary. The potential solutions include insertion of an audible alarm in the infusion line to the patient, routine use of a double reinfusion bag (transfer pack) system, redesign of the processing unit to exclude air from being sent to the reinfusion bag (mandatory use of a purge mechanism), and insertion of a nondependent air escape valve in the reinfusion bag. All these solutions involve an additional response to the problem by the manufacturer. Certainly any two in-series solutions (e.g., the printed warning on the reinfusion bag plus one of the above suggestions) for the air in the reinfusion bag problem will greatly minimize the risk of venous air embolism.

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