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In Reply:—Dr. Ramsay is certainly correct when he elicits concern over the real potential for respiratory depression in the spontaneously breathing patient receiving potent opioids. He criticizes our use of respiratory rate and oxygen saturation as a reflection of respiratory depression. However, Ramsay *et al.* must have missed reading the protocol, which indicated that we also studied end-tidal CO_2 using an oxygen delivery CO_2 sampling nasal cannula. Not only did we find no difference between the two groups, we were able to assess only a minimal increase in $P_{ET}CO_2$ in both groups. However, more important is the fact that such end-tidal CO_2s are trends only and somewhat inaccurate when sampled from a nasal cannula; this can be the only way that we measure an increase in end-tidal CO_2. We do not insert an endotracheal tube or an LMA in patients during MAC. Therefore, because of editorial exigencies, we did not report actual $P_{ET}CO_2$ trends.

Further, we cannot justify the insertion of an arterial line in a MAC patient to derive a better reflection of respiratory depression, *via* an increase in $PaCO_2$. On the other hand, an average of two or three investigators were in the operating room constantly talking to the patient during the procedure. Therefore, in addition to respiratory rate, level of oxygen saturation, CO_2 sampling by nasal cannula, an important reflection of ventilatory depression was contact with the patient and level of sedation. We believe we could accurately diagnose respiratory depression or lack thereof despite Dr. Ramsay's references.

We note that Dr. Ramsay uses remifentanil "effectively" in the management of surgical pain and "it is being used more frequently in our clinical practice." Does Dr. Ramsay use an arterial line with continuous sampling of $PaCO_2$ during MAC? Does he use an oxygen delivery CO_2 sampling nasal cannula? If not, we suspect Dr. Ramsay uses his clinical acumen, careful measurement of respiratory rate and oxygen saturation.

We thank Dr. Ramsay and his colleagues for bringing home the point that an infusion of intravenous narcotics may be associated with ventilatory depression if used in excess, and we thank the Editor-in-Chief for the opportunity to reply.

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In Reply:—Dr. Ramsay and his colleagues question the measurement of the adequacy of respiratory function in the above two studies. They state that adequacy of respiratory function cannot be ascertained by measuring the respiratory rate and O_2 saturation alone. I agree. That is why the studies were performed with anesthesia personnel in attendance at all times during spontaneous ventilation with concomitant remifentanil infusion. Respiratory pattern and wakefulness were noted, and verbal contact (no less than once per 5-min interval) was also maintained at all times.

The Gold *et al.* paper compared intraoperative analgesic doses of remifentanil with and without midazolam. Spontaneous ventilation was maintained, and end-tidal CO_2 was measured. The Yarmush *et al.* paper compared analgesic doses of remifentanil with intravenous morphine in the post-anesthesia care unit (PACU). Spontaneous ventilation was maintained, but end-tidal CO_2 was not measured. This was consistent with standard PACU monitoring techniques.

These same concerns were obviously on the mind of the Food and

Drug Administration (FDA). The indication for postoperative analgesic infusion of remifentanil stipulates that it must be administered under the direct supervision of an anesthesia practitioner.

To measure PCO_2 in the PACU with arterial blood gases would have required a far more invasive technique than was warranted for these patients. Future investigations could include end-tidal CO_2 and a more detailed analysis of respiratory pattern.

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Predicting the Severity of Carbon Monoxide Poisoning at Varying FIO_2

To the Editor:—Frink et al.[1] showed that extremely high carboxyhemoglobin (COHb) concentrations can be produced in 25-kg pigs by the inhalation of desflurane through a breathing circuit with dried CO_2 absorbents, which produce carbon monoxide (CO) through anesthetic breakdown. These experiments were performed at 40% inspired oxygen, although in clinical practice, inspired oxygen concentrations can vary from less than 25% to nearly 100%, impacting the resultant severity of CO poisoning as measured by COHb concentrations.

We attempt to exemplify the clinical effect of various inspired oxygen concentrations during the rapidly changing CO concentrations produced by desflurane breakdown by using mathematical modeling to extrapolate the data of Frink *et al.* We interpolated parts per million CO from the graphic data presented in figures 3 and 4 of the study by Frink *et al.* and applied these data to the equation developed by Coburn *et al.*,[2] (CFK equation) to determine the COHb concentration. Good experimental fit of this equation has been documented by Peterson *et al.*[3] We used this equation to calculate the accumulation of COHb as a function of the absorption and elimination of CO through respiration using Microsoft Excel (Microsoft Corporation) for the MacIntosh (Apple Computer, Inc., Cupertino, CA). The implementation of the CFK equation required that assumptions be made to complete the respiratory profile, as shown in table 1. We used the CFK equation to calculate the COHb concentration by an iterative method using CO concentrations interpolated from the graphic data of Frink *et al.* To validate the appropriateness of using the CFK equation, we graphically show the correlation of the calculated COHb data to the measured experimental data of Frink *et al.* in figure 1. This results in an r^2 value of 0.997 for the descending data points and an overall r^2 value of 0.967. Several possible explanations exist for the slight deviation of the experimental and calculated data during the ascending phase of the curve. It is possible that physiologic parameters (*e.g.*, cardiac output) were severely compromised during this phase of the experiment, and differences reflected changes in circulation in the experimental animals from those for which the

Table 1. Assumed Values for Experimental Data

Parameter	Value
Weight	25 kg
Tidal volume	275 ml
Respiratory rate	10 min^{-1}
Dead space	68 ml
Body surface area	0.6 m^2
Hemoglobin	14.0 g/dl
Barometric pressure	750 mmHg
Blood volume	1875 ml
DL_{CO}	5.9 $ml \cdot min^{-1} \cdot mmHg^{-1}$

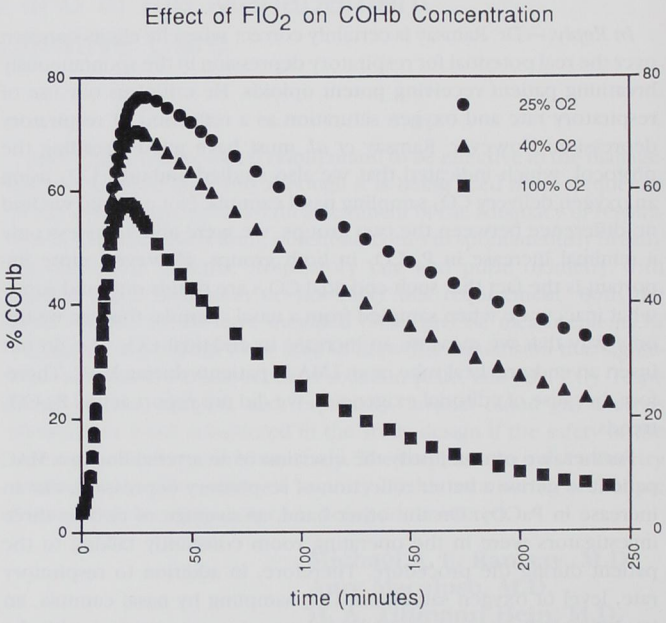

Fig. 1. Shows the concentration of CO *(open circles)* in parts per million (ppm) interpolated from the graphic data of Frink *et al.* This figure also compares COHb % calculated at 40% inspired oxygen from these data *(small triangles)* using the CFK equation for comparison with the experimental COHb % taken from the graphic data of Frink *et al.* The experimental COHb data correlate well with those predicted by the CFK equation.