

CORRESPONDENCE

Finally, Aronson and Cook imply that one cannot separate the contribution of providers and their technology to the process and outcome of anesthesia care. Our study did look at providers working with their technology in the context of actual patient care. Thus, although future studies may be able to look more deeply into the cognition of providers using TEE, our study is fully consistent with the strategy of investigating the process of care in its natural context.

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Use of Remifentanyl in Patients Breathing Spontaneously during Monitored Anesthesia Care and in the Management of Acute Postoperative Care

To the Editor:—Two multi-center studies evaluate the use of remifentanyl in patients breathing spontaneously during monitored anesthesia care and in the management of acute postoperative care.^{1,2} Neither addresses the real potential for respiratory depression in a sound manner. The conclusions by Gold *et al.*¹ and Yarmush *et al.*² that patients receiving remifentanyl maintained adequate respiratory function during their studies cannot be supported with the reported data.

Adequacy of ventilation was assessed by monitoring respiratory rate and oxygen saturation as recorded by pulse oximetry. A respiratory rate of <8 breaths/min for ≥ 1 min, or O₂ saturation <94% on oxygen supplementation were the thresholds for defining respiratory depression, yet both of these values correlate poorly with ventilatory depression.³⁻⁵ Arterial blood oxygen saturations have been shown to be well maintained at the time of peak respiratory depression in patients receiving supplemental oxygen.⁶ Under these circumstances, oxygen desaturation may be a late sign of respiratory depression. This failure to detect hypoventilation until oxygen desaturation occurs is an example of the false sense of security seen with pulse oximetry. Normal readings of oxygen saturation in the presence of increased inspired oxygen have been shown to give no information about the adequacy of ventilation.^{7,8} Supplemental oxygen may mask carbon dioxide retention.⁹ The oxygen supplementation in these two studies was also not defined. The only noninvasive parameters that have been shown to correlate well with depressed ventilation are respiratory pattern, (*i.e.*, paradoxical respiration) end-tidal CO₂, or, if the depression is severe, level of consciousness.¹⁰ Sedation is a well-known accompaniment of hypercapnia with somnolence and unconsciousness occurring when PaCO₂ levels reach 80 mmHg.

Variations in respiratory pattern may be subtle and difficult to assess. End-tidal CO₂ measurements in the extubated patient monitored *via* nasal cannulae, in our experience, do not always correlate well with arterial PaCO₂. When the patient is obtunded, nasal breathing is often reduced, and low recordings of end-tidal CO₂ are obtained. On the other hand, sedation level correlates with severe respiratory depression.

Although we have found remifentanyl to be effective in the management of surgical pain and although it is being used more frequently in our clinical practice, careful assessment of the adequacy of respiration is required when using potent narcotics in spontaneously breathing extubated patients. Respiratory rate and pulse oximetry with oxygen supplementation do not meet this requirement. Both the cited studies should have included more precise measurements of ventilatory depression to be able to draw the conclusion that significant depression did not occur. A sedation scale, end-tidal CO₂, respiratory pattern analysis, and, especially, arterial blood gas analysis should have been considered in the study design if the safety of the technique was the goal of the study. The emphasis on pulse oximetry reinforces the false sense of security of this monitoring technique when supplementary oxygen is being administered.

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CORRESPONDENCE

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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 CO₂ using an oxygen delivery CO₂ 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}CO₂ in both groups. However, more important is the fact that such end-tidal CO₂s 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 CO₂. 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}CO₂ 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 PaCO₂. 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, CO₂ 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 remifentanyl "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 PaCO₂ during MAC? Does he use an oxygen delivery CO₂ 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 O₂ saturation alone. I agree. That is why the studies were performed with anesthesia personnel in attendance at all times during spontaneous ventilation with concomitant remifentanyl 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 remifentanyl with and without midazolam. Spontaneous ventilation was maintained, and end-tidal CO₂ was measured. The Yarmush *et al.* paper compared analgesic doses of remifentanyl with intravenous morphine in the post-anesthesia care unit (PACU). Spontaneous ventilation was maintained, but end-tidal CO₂ was not measured. This was consistent with standard PACU monitoring techniques.

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