

CORRESPONDENCE

tory CO₂ sensitivity after intravenous and epidural morphine in volunteers. *Anesth Analg* 1983; 62:633-40

4. Rawal N, Wattwil M: Respiratory depression after epidural morphine—An experimental and clinical study. *Anesth Analg* 1984; 63:8-14

5. Barkin JS, Krieger B, Blinder M, Bosch-Blinder L, Goldberg RI, Phillips RS: Oxygen desaturation and changes in breathing pattern in patients undergoing colonoscopy and gastroscopy. *Gastrointest Endosc* 1989; 35:526-30

6. Ready LB, Oden R, Chadwick HS, Benedetti C, Rooke GA, Caplan R, Wild LM: Development of an anesthesiology-based postoperative pain management service. *ANESTHESIOLOGY* 1988; 68:100-6

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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

7. Hutton P, Clutton-Brock T: The benefits and pitfalls of oximetry. *BMJ* 1993; 307:157-8

8. Davidson J, Hosie H: Limitations of pulse oximetry: respiratory insufficiency—A failure of detection. *BMJ* 1993; 307:372-3

9. Council on Scientific Affairs, American Medical Association: The use of pulse oximetry during conscious sedation. *JAMA* 1993; 270:1463-8

10. Wiklund L, Hök B, Stahl K, Jordeby-Jönsson A: Postanesthesia monitoring revisited: Frequency of true and false alarms from different monitoring devices. *J Clin Anesth* 1994; 6:182-8

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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