Correspondence

Respiratory arrest after recovery from anaesthesia supplemented with sufentanil

To the Editor:

I wish to report a case of acute respiratory arrest following apparently satisfactory recovery from general anaesthesia supplemented with sufentanil.

The patient was a 61-year-old, 68 kg female presenting for hemicolectomy. She was premedicated with oxazepam 30 mg PO and morphine 8 mg IM 90 minutes before surgery. Thiopentone 100 mg, sufentanil 150 μg and succinylcholine 120 mg were administered IV and the trachea was intubated. The patient was ventilated with nitrous oxide 60 per cent and oxygen 40 per cent and pancuronium 4 mg was administered IV. Thirty minutes later, following the surgical incision, sufentanil 25 μg was administered in response to increases in pulse and blood pressure. Isoflurane 0.25–0.5 per cent was added in response to similar responses to surgical stimulation. At the end of surgery pyridostigmine 20 mg and atropine 1.2 mg were administered IV. One hundred and sixty-five minutes after anaesthetic induction the patient was awake and extubated herself in the operating room.

When the patient was transferred to the recovery room she was breathing well, oriented and talking to the anaesthetist. In the recovery room, ten minutes following extubation, the patient's vital signs were normal, including respiratory rate of $18 \cdot min^{-1}$. She was conscious, oriented and talking with the nurses. Fifteen minutes following extubation the patient was noted to be apnoeic, cyanotic and unresponsive to commands or deep pain.

The patient responded immediately to the administration of naloxone 0.2 mg IV and within 60 seconds she was awake, breathing spontaneously and responding appropriately. Her subsequent course was uneventful.

Respiratory depression after anaesthesia with a narcotic is well recognized. The rapidity with which this patient became apnoeic and the depth of narcosis was unusual. Chang *et al.*[1] reported a similar case of post-anaesthetic respiratory depression with associated chest wall rigidity following sufentanil, while Goldberg *et al.*[2] described a more gradual onset of respiratory depression, again associated with chest wall rigidity. Both reports concluded that the use of moderately high doses of sufentanil (2–5 $\mu g \cdot kg^{-1}$) as a supplement to anaesthesia could be associated with significant respiratory depression in the early postoperative period. In this dose range, the duration of respiratory depression produced by sufentanil is said to be comparable to that seen with fentanyl. In our department, where fentanyl is the most commonly used narcotic, we had never experienced such profound postoperative renarcotisation.

The pharmacokinetics of sufentanil were found by Bovill *et al.*[3] to be similar to fentanyl in having a three compartment open model characterised by triexponential decay. The third compartment acts as a reservoir, limiting return of the drug to the central compartment from which elimination is assumed to occur.

Becker *et al.*[4] observed respiratory depression in the postoperative period after fentanyl anaesthesia. He found a consistent decrease in the slope of the carbon dioxide response curve. This in itself would not be surprising but these authors also described a recurrence of respiratory depression in the recovery room, after normality had apparently been achieved. Following this report, secondary peaks in plasma fentanyl levels were subsequently observed. Stoeckel *et al.*[5] noted high initial plasma fentanyl concentrations following a bolus dose IV and in many patients a secondary increase in plasma concentration was seen. McQuay *et al.*[6] demonstrated a second peak of fentanyl in the plasma of many patients.

McClain and Hug[7] suggested that the cumulation and persistence of fentanyl may cause unexpectedly prolonged ventilatory depression. Recurrent respiratory depression might result from mobilisation of fentanyl from tissue stores and from reduced stimulation.

The recommended induction dose of sufentanil for major surgery with an anticipated duration of two hours is 1.5–2.0 $\mu g \cdot kg^{-1}$. Maintenance doses of 10–25 μg are given, based on changes in clinical signs. Total dose requirements should average 1 $\mu g \cdot kg^{-1} \cdot hr^{-1}$. Respiratory depression produced by a sufentanil dose of 25 μg IV is said to last 25–30 minutes.

Our patient received 2.6 $\mu g \cdot kg^{-1}$ for a 2.5-hour procedure, with the last increment being administered 120 minutes prior to the end of the procedure and 140 minutes before the respiratory arrest.

The manufacturer of sufentanil warns: "Patients who arrive in the recovery room awake and alert may lapse into a sedated state with diminished breathing if left unstimulated." Our patient by comparison was comatose and apnoeic, presumably due to a secondary narcotic peak.

The probable explanation is that with increased muscle tone, voluntary movement and increased body temperature there was increased perfusion of skeletal muscle with re-uptake by the blood of sufentanil and increased plasma concentration. Stimulation during emergence and transfer to the recovery room may antagonise the ventilary depressant effects of residual low-dose sufentanil and respiratory depression may only become evident when the patient is relatively unstimulated in the postoperative period.

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REFERENCES

1 *Chang J, Fish KJ.* Acute respiratory arrest and rigidity after anesthesia with sufentanil: a case report. Anesthesiology 1985; 63: 710–1.
2 *Goldberg M, Ishak S, Garcia C, McKenna J.* Post-operative rigidity following sufentanil administration. Anesthesiology 1985; 63: 199–201.
3 *Bovill JG, Sebel PS, Blackburn CL, Oei-Lim V, Heykants JJ.* The pharmacokinetics of sufentanil in surgical patients. Anesthesiology 1984; 61: 502–6.
4 *Becker LD, Paulson BA, Miller RD, Severinghaus, JW, Eger EI II.* Biphasic respiratory depression after fentanyl–droperidol or fentanyl used to supplement nitrous oxide anesthesia. Anesthesiology 1976; 44: 291–6.
5 *Stoeckel H, Hengstmann JH, Schuttler J.* Pharmacokinetics of fentanyl as a possible explanation for recurrence of respiratory depression. Br J Anaesth 1979; 51: 741–4.
6 *McQuay HJ, Moore RA, Patterson GMC, Adams AP.* Plasma fentanyl, fentanyl concentration and clinical observations during and after operation. Br J Anaesth 1979; 51: 543–50.
7 *McClain DA, Hug CC.* Intravenous fentanyl kinetics. Clin Pharmacol Ther 1980; 28: 106–14.

Airway assessment in obstetrical patients

To the Editor:

After reading Dr. McIntyre's article on difficult tracheal intubation,[1] I would like to emphasize that any anaesthetist dealing with pregnant patients should assess their airway with the patient *recumbent*. In this position, large pendulous breasts will often rise up to rest under the chin and thus make both constant cricothyroid pressure and tracheal intubation difficult, if not impossible. Taping the breasts out of the way and/or use of a short handled (Datta) laryngoscope or a laryngoscope with a "polio blade" may facilitate intubation.

Obstetrical emergencies often require anaesthetizing a patient who has recently ingested a full meal. The risk of encountering a difficult or failed intubation as well as pulmonary aspiration of excessively acid gastric material is much greater in the pregnant patient.[2,3] Careful preoperative airway assessment may help to prevent these potential disasters.

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REFERENCES

1 *McIntyre JWR.* The difficult tracheal intubation. Can J Anaesth 1987; 34: 204–13
2 *Tomkins J, Turnbull A, Robson G et al.* Report on Confidential Enquiries into Maternal Deaths in England and Wales. 1976–1978. Department of Health and Social Security. Her Majesty's Stationery Office. 1982.
3 *Gibbs CP.* Maternal Physiology. Clin Obstet Gynecol 1981; 24: 538.

REPLY

One purpose of the article was to emphasise the value of a careful preanaesthesia examination regarding potential difficulties of tracheal intubation. From this viewpoint the problems that may be presented by the pregnant patient do not differ substantially from certain others such as the very obese person. As for the position in which the patient is examined, Dr. Ross's point is well taken and it is reasonable to state that any patient should be examined in the position in which intubation will be attempted. Other matters she mentions, though vitally important, fall within the realm of selection of anaesthesia conditions and state of consciousness under which intubation is done rather than the technique of intubation.

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Facial nerve paralysis following mask anaesthesia

To the Editor:

The incidence of facial nerve paresis following mask anaesthesia is rare. Azar and Lear[1] and Glauber[2] in 1986, described three patients who developed sensory and motor nerve dysfunction of the face, following mask