REPLY

Drs. Smelt, de Lange and Booij have written in regard to changes in end-tidal carbon dioxide following both air embolism and alterations in cardiac output. In Figure 1, they have shown a decrease in ETCO₂ and a marked rise in PAP, without change in either CVP or arterial pressure. Figure 2 shows a minor decrease in ETCO₂ and a marked fall in systemic pressure. It is unfortunate that they make no comment regarding:

(i) volumes of air required to give the pattern seen in Figure 1, and

(ii) any alterations in cardiac output.

The change in cardiac output is obviously the important aspect of this whole question.

Small emboli may be sufficient to cause local changes to PA pressures without significantly altering central venous or systemic pressures or affecting cardiac output. Conversely, larger emboli may cause changes in both PA pressure and cardiac output, with secondary alterations in central venous and arterial pressures. The time course for resolution of haemodynamic insult and return to "normal" of ETCO₂ may be quite different, as was evident in our original article.

The possibility of imprecise placement of the precordial doppler probe has been raised; however, it did detect the changes in heart sounds, but only where arterial pressure and presumably cardiac output were falling. The difficulties in reliably placing the doppler in all instances (e.g. prone or sitting) should make one hesitant about its sole use to detect embolism. I would suggest an end-tidal monitor is both easier to place and more consistently reliable.

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Epidural morphine for postoperative analgesia

To the Editor:

We read with interest the paper by Writer *et al.*,[1] which reported the results of a multicentre study of epidural morphine for postoperative analgesia. We are in entire agreement with their hypothesis that continuing analgesia from longer acting local anaesthetic agents, such as bupivacaine, allows time for epidural morphine to reach peak effect. Bupivacaine (0.5%) and lidocaine (with 1:400,000 epinephrine) are the local anaesthetic agents most frequently used in epidural anaesthesia for Caesarean section, in our institution. Only very rarely is any request made for analgesia as the epidural block regresses.

We are interested in their observation that 2 of the 103 patients (2%) who had abdominal surgery and 2 of the 35 patients (7%) who had lower limb orthopedic surgery developed clinically significant bradypnea. It would be of interest to know the nature of the abdominal surgery done on the 2 patients, in that group, who developed bradypnea; and whether they had received any other medication. The protocol in our institution requires that the anaesthetist on duty be informed of any patient with a respiratory rate of less than 10/minute, after receiving epidural morphine. We have used this technique for post-Caesarean section analgesia in over 3000 patients, only 2 of whom developed bradypnea. Neither of these patients was distressed, cyanosed or exhibited any other signs of hypoventilation or hypoxia. One patient was noted to have a respiratory rate of 9/minute about 5 hours after receiving 5 mg epidural morphine. She received 2.5 mg droperidol intra-operatively and 3 mg perphenazine (in 1 mg doses) in the recovery room. She was given naloxone 0.15 mg I.V. on two occasions, with no immediate change in respiratory rate; suggesting a droperidol and/or perphenazine effect.

The other patient was observed by the nurse, on a routine visit, to have a respiratory rate of 8/minute, while asleep, 12 hours after epidural morphine (5 mg) had been administered. Again there was no other clinical evidence of hypoventilation or hypoxia. She was easily roused and in no distress. However, she was given 0.1 mg naloxone I.V., with immediate and sustained increase in her respiratory rate. She was noted to have received dimenhydrinate for nausea, 1 hour prior to the bradypnea.

It would appear that parturients, who are young, are generally in good health and tend to receive little intercurrent medication, are less prone to develop delayed respiratory depression after receiving epidural morphine. They do seem to have a higher incidence of pruritus (mainly in the distribution of the trigeminal nerve), but this is usually mild.

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REFERENCE

1 *Writer WDR, Hurtig JB, Evans D, Needs RE, Hope CE, Forrest JB.* Epidural morphine prophylaxis of postoperative pain: report of a double-blind multi-centre study. Can Anaesth Soc J 1985; 32: 330–8.

REPLY

Thank you for the opportunity to reply to the points raised by Drs. McMorland and Douglas. Our further clinical experience with epidural morphine 5 mg following Cae-sarean section, lends support to our original hypothesis concerning the benefits of long-action local anaesthetics, such as bupivacaine. Mothers who receive chloroproc-aine as the primary agent for epidural anaesthesia commonly experience pain in the recovery room as the epidural block regresses, and one of us (WDRW) routine-ly administers epidural bupivacaine, 0.25 per cent, before recovery from chloroprocaine, in order to extend postoperative analgesia. Epidural morphine then reach-es its peak effect, before the bupivacaine wears off.

Concerning the incidents of bradypnea following abdominal surgery, we cannot exclude the possibility of additive effects from other medications. One patient, aged 43 years, underwent abdominal hysterectomy for menorrhagia. She took ibuprofen, 400 mg t.i.d., during her heavy menses, but had received none in the seven days before surgery. After premedication with diazepam, 10 mg p.o., 1 h preoperatively, general anaesthesia was induced with thiopentone, 375 mg, and succinylcholine, 120 mg. Anaesthesia was maintained with nitrous oxide and isoflurane. The patient also received metocurine, 5 mg, pancuronium, 1 mg, and droperidol, 1 mg. Epidural morphine, 5 mg, was administered 40 min before the conclusion of surgery, and the isoflurane was discon-tinued prior to reversal of the neuromuscular blockade with atropine, 1.2 mg, and neostigmine, 2.5 mg. Despite apparently adequate neuromuscular reversal, the patient remained apneic for 45 min and in the recovery room would not breathe unless instructed. However, the apnea was significantly improved after IV naloxone, 0.2 mg.

The 69-year-old male who developed transient respira-tory depression (9/min) 4½ hours after epidural morphine underwent choledochojejunostomy for obstructive jaun-dice. He also received diazepam, 10 mg, preoperatively, and the anaesthetic technique included thiopentone, succinylcholine, pancuronium, nitrous oxide, oxygen and enflurane. A single dose of IV naloxone, 0.2 mg, was given.

Although butyrophenones, for example droperidol, have powerful central sedative effects, they do not appear to potentiate the respiratory depressant effects of systemic narcotics.[1] Diazepam, in contrast, may enhance respira-tory depression, although this is usually associated with intravenous administration. Respiratory depressant drugs administered to our two patients during anaesthesia, for example thiopentone and volatile agents, may have con-tributed to the bradypnea, but the possibility of such interactions has not been addressed in epidural morphine

studies to date. Age and debility, as in the second patient, increase the risk of respiratory depression. The prompt response of both patients to IV naloxone, 0.2 mg, suggests a narcotic effect.

Bradypnea does not necessarily imply respiratory depression. Periods of apnea occur commonly during sleep in apparently healthy individuals not suffering from disabling or life-threatening illness.[2] Conversely, sig-nificant and prolonged depression of central respiratory regulation, with impaired ventilation but without signific-ant bradypnea, may follow administration of epidural morphine.[3] These findings underline the inadequacy of the respiratory rate as an indicator of respiratory depression, and emphasize the need for close surveil-lance of patients, as described in our paper.

Finally, McMorland and Douglas, after extensive ex-perience with epidural morphine, suggest that parturients, because of their youth and generally good health, are less prone to develop delayed respiratory depression after epidural morphine. Shnider's group reported no respiratory depression in 276 consecutive parturients who received epidural morphine for Caesarean section,[4] and this has been the experience to date in the Grace Maternity Hospital, Halifax, Nova Scotia. There is a need for studies to determine if the well-documented increase in alveolar ventilation during pregnancy pro-tects the parturient against the respiratory depression from epidural narcotics.

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