

sented. We know of no method of estimating ventilation and P_{CO_2} from CO_2 response slopes and intercepts (obtained by either steady-state or Read techniques) since there is an unpredictable relationship among them, especially in the presence of pharmacologic depressants.^{6,7}

In healthy pain-free volunteers, 3.5 or 7.0 mg epidural morphine alone caused a progressive reduction of ventilation and increase of P_{ETCO_2} as the analgesic effect waned.⁸ Hypoventilation was clearly greater than that which followed the same doses of morphine given to the same subjects subcutaneously, and it persisted for twenty-four hours. Ventilatory depression that continues for many hours after analgesic effect dissipates would be particularly hazardous to patients, because repeated doses of narcotic based upon analgesic requirement could result in cumulative depressive effects on breathing.

On the basis of experimental and clinical data available to date, we think that all patients receiving epidural narcotics should be monitored closely for respiratory depression, not only for the duration of analgesia but for 12 to 24 hours thereafter, and especially when administrations are repeated.

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REFERENCES

1. Reiz S, Westberg M: Side-effects of epidural morphine. *Lancet* 2:203-204, 1980
2. Christensen V: Respiratory depression after extradural morphine. *Br J Anaesth* 52:841, 1980
3. McDonald AM: Complication of epidural morphine. *Anaesth Intensive Care* 8:490-491, 1980
4. Boas RA: Hazards of epidural morphine. *Anaesth Intensive Care* 8:377-378, 1980
5. Doblar DD, Muldoon SM, Abbrecht PH, et al: Epidural morphine following epidural local anesthesia: Effect on ventilatory and airway occlusion pressure responses to CO_2 . *ANESTHESIOLOGY* 55:423-428, 1981
6. Lambertsen CJ: Effects of drugs and hormones on the respiratory response to carbon dioxide, *Handbook of Physiology*, Section 3, Respiration, Vol. 1. Edited by Fenn WO, Rahn H. Washington, American Physiological Society, 1964, pp 545-555
7. Larson CP, Eger EI, Muallem M, et al: The effects of diethyl ether and methoxyflurane on ventilation: A comparative study in man. *ANESTHESIOLOGY* 30:174-184, 1969
8. Knill RL, Clement JL, Thompson WR: Epidural morphine causes delayed and prolonged ventilatory depression. *Can Anaesth Soc J* 28:537-543, 1981

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In reply:—Our study¹ was undertaken to quantitate the effects of epidural morphine *alone* on ventilatory control in healthy subjects because we were concerned with the reports of severe ventilatory depression associated with the use of epidural and intrathecal narcotics.

Our conclusion that epidural morphine causes less depression of ventilatory sensitivity to CO_2 than parenterally administered morphine is independent of the other ventilatory variables mentioned above and is based on the comparison of the average slopes of the ventilatory response to CO_2 in our patients with those reported for parenteral morphine.²⁻⁴ We cautioned, however, that because of considerable individual variability in the ventilatory response to CO_2 , extrapolation of averaged data to a specific clinical setting might be hazardous. We did not state, as suggested in the letter above, that severe depression may be of real concern only in patients with diminished ventilatory reserve or in those receiving other drugs which depress ventilation. Instead, we expressed

concern that based on the depressant effects we found in healthy subjects, other factors such as diminished ventilatory reserve or concomitant use of parenteral narcotics might result in even greater depression of the CO_2 response than we found. A similar concern has been expressed recently as the result of experience with combined intrathecal and parenteral narcotics.⁵ The study of Knill *et al.* provides additional data on the duration of the effect in pain-free subjects and raises concern that prolonged monitoring should accompany epidural narcotic use.

The association of epidural and intrathecal use of narcotics and severe depression of ventilation or apnea is apparent from the available case reports. The influence of age, physical status, presence of pain, concomitant use of parenteral narcotics, or respiratory depressants, and the effects of repeated doses of epidural narcotics on the magnitude and duration of ventilation depression must be determined as further studies continue. In the interim,

the safe use of epidural and intrathecal narcotics demands monitoring for as long as the effect on ventilatory control can be shown to be significant in order to prevent serious, avoidable complications. We agree, therefore, with Knill *et al.* that based on their findings the minimum duration of monitoring should be twelve to twenty-four hours following a single dose of epidural morphine.

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REFERENCES

1. Doblal DD, Muldoon SM, Abbrecht PH, et al: Epidural morphine following epidural local anesthesia: Effect on ventilatory and airway occlusion pressure responses to CO₂. ANESTHESIOLOGY 55:423-428, 1981
2. Santiago TV, Johnson J, Riley DJ, et al: Effects of morphine on ventilatory response to exercise. J Appl Physiol 47:112-118, 1979
3. Weil JV, McCullough RE, Kline JS, et al: Diminished ventilatory response to hypoxia and hypercapnia after morphine in normal man. N Engl J Med 292:1103-1106, 1975
4. Nielson CH, Camporesi EM, Bromage PR, et al: CO₂ sensitivity after epidural and I.V. morphine. ANESTHESIOLOGY 55:A372, 1981
5. Odom JA, Sih IL: Respiratory depression after intrathecal morphine. Anesth Analg (Cleve) 61:70, 1982

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Why Routinely Pretreat?

To the Editor:—The recent article by Blitt *et al.*¹ evaluating nondepolarizing neuromuscular blockers for pretreatment prior to administering succinylcholine (SCh) has prompted this letter. Although the authors demonstrated that metocurine was as effective as gallamine and *d*-tubocurarine in preventing the muscle fasciculations that follow SCh, they failed to demonstrate any real clinical benefit from the use of metocurine for this purpose. “Self-taming,” a technique using a small dose of succinylcholine prior to administering a larger paralyzing dose of SCh is also effective in preventing fasciculations.^{2,3}

Blitt *et al.* stated that the low incidence of postoperative myalgia they observed may be related to studying “patients in whom the likelihood of muscle pain was not high.” Postoperative muscle pain occurs in 0.2–89 per cent of patients who receive SCh.⁴ The many factors believed to influence postoperative muscle pains, such as age and sex of patient, anesthetic technique, length and type of surgical procedure, intraoperative patient position, postoperative care, medications, and time and extent of ambulation all differ between studies and often within the same study making it difficult to compare reports.⁵ None of these factors were addressed by Blitt. The extremely low incidence of myalgia they noted only emphasizes how uncommon the problem is. SCh-myalgias are believed to be more frequent after outpatient,⁶ dental,⁷ or other “minor” procedures.^{4,8} Muscle pains following these minor procedures may be modified by a variety of techniques including pretreatment with a

subparalyzing dose of nondepolarizing muscle relaxant,^{4,6,7,9} intravenous lidocaine,^{10,11} thiopental,⁸ vitamin C,¹² and/or diazepam.¹³ Pretreatment with nondepolarizing relaxants did not reduce the incidence of postoperative myalgias in two well-controlled studies of patients undergoing major abdominal surgery.^{5,14} Are SCh-myalgias after major surgery a true clinical entity? There were no differences in postoperative myalgia after staging laparotomy in patients receiving only pancuronium for intubation compared to patients receiving only SCh.¹⁵

This gets me to the point I wish to make. Why do we continue the routine practice of pretreating with a nondepolarizing muscle relaxant prior to the use of SCh? SCh-myalgias after major surgery, if they really do occur, are very rare. Certainly, if one wishes to avoid fasciculations, “self-taming” with SCh will do this and provide excellent intubating conditions as well.^{2,3} I do not feel comfortable using the pretreatment technique for a patient with an open eye injury, or for a patient at risk from hyperkalemia since pretreatment may not be totally effective and does not completely protect susceptible patients.¹⁶ In these situations it is safer to avoid SCh completely. As for the rise in intragastric pressure with SCh, the potential dangers of pretreatment may offset any benefit.¹⁷ Nondepolarizing muscle relaxants and SCh are antagonistic, their combination may delay the onset of paralysis and result in an incomplete block. I prefer the sure, rapid onset of paralysis with SCh alone, plus cricoid pressure to minimize the risk of aspiration in patients with a full stomach.