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## A New Look at Old Anesthesia Circuits

*To the Editor:*—While some may believe that my journals lie unread for months, this is not the reason I comment only now on Dr. Edsall's letter in March 1981 issue of ANESTHESIOLOGY.[1] My copy, for reasons totally obscure, only reached me on December 8. I am old enough to remember an earlier technology which made it possible to predict with certainty that a letter mailed in New York on a Monday would reach its addressee in London, England, before Friday noon of the same week. Our modern technology does not seem able to rival those achievements of yesterday. Perhaps because so many of us are blind to what history can teach us.

Why, I wonder, have we not yet applied to the clinical situation in anesthesia, those lessons learned by the deep-sea diver with his self-contained under-water breathing apparatus? Unable to carry extra equipment such as $O_2$ and $CO_2$ analyzers along with him, he uses a demand-feed system which supplies an atmosphere prepared in advance to meet expected requirements. He voids all exhaled gas from his breathing system and manages to live and work safely in a very hostile environment.

In the days of the earlier technology, EI McKesson (1910) was busy developing this concept in anesthesia when Dennis Jackson (1915), Ralph Waters (1923), and Brain Sword (1928), wishing to economize, in particular to enable cyclopropane to be used clinically, distracted attention from demand-feed systems which had given preselected dosage on a breath-to-breath basis.[2] Such systems still exist.[3] They can, and should, be further refined and be adopted universally, not only because they permit far greater precision in prescribing and administering inhalational anesthetics, but also because the voiding of all exhaled gases will allow the development of a total scavenging system to salvage, and store for later re-use, nitrous oxide as well as volatile anesthetic agents. At present all anesthetic gases and vapors, while they may not grossly contaminate the operating suite, certainly do dilute the general atmosphere outside the hospital, representing not only a health threat but a total and major financial loss.

The cryogenic technology essential to achieve massive recycling of anesthetic agents is already available. Its application to anesthesia could effect far more significant savings than the mathematically insignificant difference in anesthetic costs as between the closed and the semi-closed systems discussed by Dr. Edsall.

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### REFERENCES

1. Edsall DW: Economy is not a major benefit of closed-system anesthesia. ANESTHESIOLOGY 54:258, 1981
2. Keys TE: The History of Surgical Anesthesia. New York, Dover Publications, 1963
3. Jones CS: Modification of a Bird® Mark-14 anesthesia ventilator for greater versatility. Anesth Analg (Cleve) 55:879, 1976

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## Epidural Morphine and Ventilatory Depression

*To the Editor:*—It is recognized that the single most important factor limiting the conventional use of narcotics to treat severe pain is the risk of excessive ventilatory depression. Despite initial speculation to the contrary, it is now evident that small doses of narcotic administered by the epidural route to healthy patients may be complicated several hours later by life-threatening ventilatory depression.[1–4] However, a recent study reported in ANESTHESIOLOGY concludes that the ventilatory depressant effect of 10 mg epidural morphine alone is less than that associated with comparable doses of morphine given parenterally, and suggests that severe depression may be of real concern only in patients with "diminished ventilatory reserve" or in those receiving other drugs which reduce breathing.[5]

We wonder if these conclusions are justified. This report contains data on $CO_2$ responses only, responses were determined after epidural morphine only during the period of analgesic effect. Fundamental ventilatory variables, *i.e.*, ventilation, $P_{ET_{CO_2}}$ or $Pa_{CO_2}$, are not pre-

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 $P_{ET_{CO_2}}$ 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 study[1] 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,