Correspondence

CNS Depression from Intrathecal Morphine

To the Editor: — Recent months have witnessed increasing clinical interest in the use of narcotic analgesics injected into the subarachnoid and epidural spaces. Initial observations suggested that these techniques could provide good analgesia without respiratory depression. Two communications recently published in the Lancet contradict this view.

Encouraged by our own results using morphine and fentanyl via the epidural route and having read with interest the preliminary report from Wang,2 we evaluated intrathecal morphine given at the same time as a spinal anesthetic for postoperative analgesia.

Five patients undergoing elective surgical procedures, ages ranging from 20 to 77 years, consented to the procedure. Each received 4 ml of 0.5 per cent isobaric bupivacaine and 1 mg morphine injected into the lumbar subarachnoid space. Satisfactory analgesia was achieved in each case. No other narcotic was given, and all five patients appeared alert on discharge from the recovery room. None of the patients requested or received postoperative medication for pain relief. All patients were nursed in the supine position. Approximately ten hours after administration of the spinal anesthetic, three patients (aged 20, 60, and 77 years) experienced bradycardia, decreased respiratory rate, and pinpoint pupils. Two patients subsequently had respiratory arrest, which responded to naloxone given intravenously.

Initial clinical reports2,3 and extensive animal work4-10 failed to demonstrate that intrathecal narcotics could spread cephalad to the brain stem and higher centers. Our observations and those of Glynn et al.4 and Liolios et al.6 suggest that such spread does occur. The time intervals between injection and respiratory depression indicate that the effect may be a function of cerebrospinal fluid flow, of which little is known. Glynn et al. used 3 mg and 5 mg of morphine. Liolios et al. used 15 mg. We have demonstrated that even as little as 1 mg of morphine is not a safe dose when injected into the subarachnoid space. The observations of Wang2 and Samii2 concerned patients with chronic pain who may have been tolerant to narcotics.

We feel that intrathecal narcotics may have a role in postoperative analgesia. However, the dose needed to produce analgesia without also causing undesirable side effects has yet to be established.

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REFERENCES

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Incompatibility of a New Pediatric Endotracheal Tube with Existing Fittings

To the Editor:—This letter is to call attention to the incompatibility of new McGaw pediatric endotracheal tubes* that have a raised flange inside the standard 15-mm adaptor and the Norman mask elbow. The raised flange on the McGaw endotracheal tube impinges on the gas flow inlet inside the Norman elbow, making ventilation with the circuit impossible. Further-

*McGaw Respiratory Therapy Division of American Hospital Supply Corporation, Irvine, California.
more, the adaptor flange and the inlet in the Norman elbow serve the same purpose of minimizing dead space in the system, only one is useful.

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Statistical Error?

To the Editor:—In a recent letter describing their investigation into the possibility of naloxone reversal of the sedation induced by diazepam, Drs. Christensen and Hüttel found the difference between control and treated patient groups to be $P = 0.02$. They said there was no statistically significant difference; this is not so. In biological practice a probability between 0.01 and 0.05 is considered statistically significant. Their conclusion about naloxone is, therefore, incorrect.

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Mortality Rates and Exposure to Anesthetics

To the Editor:—I read with interest the article by Lew, “Mortality Experience Among Anesthesiologists, 1954–1976,” in the September 1979 issue.1 The results of this study of mortality rates are comforting in that mortality rates of male anesthesiologists are approximately 88 per cent of that for all physicians and 70 per cent of that for all white males in the general population. The death rates for female anesthesiologists are similar to those for males although the number of deaths reported, 39, was too small to permit drawing significant conclusions.

One statement in the paper, however, should not go unchallenged. The first sentence of the abstract states: “To determine whether anesthesiologists, because of prolonged exposure to halothane or other inhalational anesthetics, might have higher death rates from all causes, from cancer, and from hepatic or renal diseases than other physicians, the mortality rates of male and female members of the American Society of Anesthesiologists (ASA) for the period of 1954 to 1976 were examined” (italics are mine). Persons reading this article should understand that examination of the mortality rates for anesthesiologists can never determine whether the observed death rates—be they higher or lower than those of the control groups—were due to prolonged exposure to halothane or to other inhalational anesthetics. Mr. Lew’s report is purely descriptive and does not address the question of cause and effect. To date, there is no definitive evidence that anesthetic agents are hazardous to individuals who work in operating rooms. Death rate analyses can never establish such a relationship.

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Reference


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