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In Reply:—We thank Dr. Yemen for his interest in our publication. Our adult patients have not experienced the episodes of "coughing" associated with the administration of sufentanil that he describes in children. The literature on opioid-induced difficult ventilation in neonates and children is at best confused and consists mostly of isolated case reports.

Baraka¹ described post-extubation laryngospasm after opioid-based anesthesia in a 4-yr-old child. Naloxone terminated the laryngospasm. MacGregor *et al.*² described difficult ventilation after initiation of a fentanyl infusion in an intubated neonate. They extubated the child, fearing an endotracheal tube obstruction. They could not ventilate the extubated child. A cardiorespiratory arrest resulted. Nalaxone was administered and restored the ability to ventilate. They ascribed the difficult ventilation to chest wall rigidity.

Perhaps the chest wall component plays a larger role in causing difficult ventilation in infants and children than in adults. The only way to clarify the issue is to conduct a prospective study in children similar to that done in adults.³

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Is Phenylephrine or Sodium Bisulfite Neurotoxic?

To the Editor:—We have read with interest the article by Sakura et al., in which they attributed the transient neurologic deficit seen in patients after spinal anesthesia to phenylephrine added to tetracaine solution. Regrettably the authors failed to acknowledge that the commercially available 0.5% phenylephrine solution (Kowa, Nagoya, Japan) contained 0.1% sodium bisulfite, well known for its neurotoxicity when administered neuraxially. The amount of sodium bisulfite given in their patients ranged from 0.5 to 0.75 mg, approximately half the dose that caused permanent hind-limb paralysis in rabbits and approximately one tenth the concentration that caused irreversible spinal monosynaptic reflex in rats. Unless preservative-free

phenylephrine solution is used in combination with tetracaine, phenylephrine, *per se*, cannot be regarded as an etiology of the reported transient neurologic sequelae.

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Transient Neurologic Symptoms when Phenylephrine Is Added to Tetracaine Spinal Anesthesia—An Alternative

To the Editor: — The article by Sakura et al. regarding transient neurologic symptoms after tetracaine spinal anesthesia with phenylephrine was informative.¹ Why do anesthesiologists add a vasoconstrictor to prolong the duration of the anesthesia? As the authors indicate, "Prolongation is thought to result, at least in part, from a decrease in nerve blood flow resulting in reduced vascular uptake of the local anesthetic."² Thus, more local anesthetic is available for neuronal penetration, causing a more profound and prolonged block. It also is implied that this allows the anesthesiologist to limit the amount of local anesthetic injected, in turn decreasing cephalad spread. But if the vascular absorption of the local anesthetic decreases, is not that the equivalent of injecting more local anesthetic? Because there is an apparent adverse effect of adding phenylephrine to tetracaine spinal anesthesia,¹ why not just inject a little more tetracaine to prolong the anesthesia and omit the phenylephrine?

The Editorial View that accompanied the Sakura paper was confusing.³ Dr. Rowlingson agrees that Sakura *et al.*¹ have furthered our knowledge regarding transient neurologic symptoms after spinal anesthesia. He then goes a step further and states, "Perhaps tetracaine is a better (safer) drug for spinal anesthesia, because compared to lidocaine and bupivacaine it increases spinal cord blood flow." That conclusion, based on one dog study,² is a big leap. Tetracaine, like lidocaine, caused cauda equina syndrome after continuous spinal anesthesia,⁴⁻⁶ and it is neurotoxic to isolated nerve.⁷ Further, compared with bupivacaine, tetracaine is associated with a significantly higher incidence of tourniquet pain during lower extremity orthopedic operations.⁸ It is for these reasons that I do not share or understand Dr. Rowlingson's enthusiasm for tetracaine spinal anesthesia.

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