CORRESPONDENCE


(Accepted for publication April 4, 1995.)

In Reply.—We appreciate the useful suggestion to our study expressed by Introna et al. Based on their study using spectral analysis of heart rate variability, which showed that both sympathetic and parasympathetic activity reduced after cardiac sympathectomy with spinal block, we agree that vagal dominance after epidural anesthesia might not exist, although we did not evaluate sympathovagal balance after thoracic epidural blockade. In our dysrhythmogenic experiments, bilateral vagotomy did not affect the dysrhythmogenic threshold significantly in dogs without thoracic epidural blockade, suggesting that vagal stimulation alone induced by baroreceptor pressor response after epinephrine infusion was not enough to affect halothane-epinephrine dysrhythmias. However, vagal stimulation played a significant role in preventing the dysrhythmias in dogs with thoracic sympathetic blockade, although vagal outflow may have been reduced through the central nervous system. These observations suggested that sympathetic activity as well as sympathovagal balance might be important in the myocardial sensitization by halothane.

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References

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Parkinsonian Signs May Be Related to Bupivacaine Excess

To the Editor:—Murerchick and Smith described Parkinsonian signs in a patient after general anesthesia. Of interest, bupivacaine was used, both for intercostal blockade as well as for wound infiltration in a total dose of 2.25 mg (45 ml of 0.5%). This is the maximum dose that can be used. Indeed, Wood considers 2 mg/kg the highest safe limit, which, in the reported case (80 kg), would have been 160 mg bupivacaine. The rate of injection and rapidity with which blood concentrations of bupivacaine are achieved can alter its toxicity signs. The use of epinephrine could have delayed the absorption of bupivacaine so that a toxic concentration would have been reached.

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only at the end of the procedure. Furthermore, hypercarbia at the conclusion of the case (due to incomplete reversal) would have reduced the central nervous system toxicity/convulsive threshold to bupivacaine. The signs noticed in this case, i.e., dystonic movement and prolonged emergence from anesthesia, thus might have been related to a higher blood concentration of bupivacaine. It was unfortunate that the blood bupivacaine concentration was not measured.

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In Reply.—Elias implies that the unusual Parkinsonian emergence from anesthesia we described in a case report1 could have been simply a manifestation of central nervous system toxicity. However, the relevant facts do not support this hypothesis because (1) the total dose of bupivacaine did not exceed the recommended maximum, a recommendation that is understandably conservative; (2) the appearance of manifestations of local anesthetic toxicity several hours, rather than minutes, after intercostal nerve block is contrary both to our own extensive clinical experience and to current concepts in anesthetic practice; and (3) the event we described could not have been influenced significantly by the adequacy of the patient’s ventilation because continuous monitoring of neuromuscular blockade and end-tidal carbon dioxide throughout anesthesia and emergence confirmed there was no evidence of the incomplete reversal or the hypercarbia presumed to exist by Elias.

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To the Editor.—A 47-yr-old woman and her 70-yr-old mother experienced difficult intubations within 2 months of each other while undergoing general endotracheal anesthesia.

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Unanticipated Difficult Intubation in Two Family Members

Preoperatively, the daughter’s airway revealed a three-finger breadth mouth opening with partial visualization of her uvula, three-finger breadth thyroid-mental distance, adequate neck mobility, and...