LETTERS TO THE EDITOR

EPIEDURAL TEST DOSE

We would like to respond to the comments made by F.J. Spielman and C.B. Watson. While 3 ml of bupivacaine 0.75% was used as a test dose in one of the cases reported, we did not recommend a specific volume to be used as a test dose. Moore's recent report on the use of bupivacaine in spinal anaesthesia gives the mean height of block achieved after 1.6 ml of bupivacaine 0.75% as T4 ± 2. Thus, the amount of bupivacaine 0.75% to be used as a test dose should probably be in the range of 1.5–2.0 ml (1.25–1.5 mg).

We did not consider the importance of a test dose in demonstrating the possibility of an intravascular injection. Another recent report by Moore discussed the effects of 3 ml of bupivacaine 0.75% ± epinephrine when injected intravenously. Bupivacaine without epinephrine only caused signs of slight toxicity in five out of forty patients but with epinephrine it consistently caused tachycardia and 50 per cent complained of transient palpitation. Thus, to detect intravenous injection, a local anaesthetic containing epinephrine would appear preferable.

The suggestion that 2-chloroprocaine would be preferable as a test dose is not helpful in Canada, as this drug is not regularly available. Also, 2 ml of 2 or 3 per cent 2-chloroprocaine might not give a very obvious spinal block. Covino, et al. recently recommended 4–5 ml of 2 per cent or 3 ml of 3 per cent 2-chloroprocaine in their editorial, which also cautioned against the use of larger volumes of this drug when injected intrathecally because of reports of prolonged sensory and motor deficits.

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REFERENCES


SEIZURE ACTIVITY ASSOCIATED WITH ENFLURANE

Sir,

Ng is to be congratulated for bringing the attention of the anaesthesia community to the problem of seizure activity in the immediate postoperative period, associated with enflurane. Kruczek, et al. recently reported a generalized tonic/clonic seizure after an enflurane anaesthetic in a healthy young female with no personal or family history of seizures. Arterial blood gases immediately after the seizure revealed normocarbia. Electroencephalographic examination twelve days postoperatively showed focal irregularities in the posterior right temporal lobe with the EEG returning to normal after forty days.

Both of these patients received thiopentone for induction, which has been shown to exacerbate electroencephalographic seizure activity during light enflurane anaesthesia. These two case reports and that reported by Ohm, et al. suggest that the development of delayed seizure activity may be due to enflurane or one of its metabolites producing an irritative focus that manifests itself only after the administration of the anaesthetic.

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REFERENCES


