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Chloroprocaine–Echothiophate Interaction

To the Editor:—The recent case report by Brodsky and Campos[1] includes a general statement about the safety of chloroprocaine in patients with low levels of active plasma cholinesterase, based upon one successful caudal anesthetic use in a patient being treated with a plasma cholinesterase inhibitor (echothiophate iodide eye drops) and given chloroprocaine, 10 mg/kg, twice with a 105-min interval. This is a total dosage that is lower than that given intravenously in less than one-fourth the time to ten healthy volunteers by Foldes *et al.*[2] (1 mg/kg/min over 22.8 min) in preconvulsive studies. Thus, despite the fact that the patient of Brodsky and Campos[1] had a cholinesterase concentration somewhat lower than normal (1,216 mU; normal range 1,900–3,800) the metabolic degradation of chloroprocaine should be sufficient to avoid toxic symptoms with the reported dosage. Patients treated with echothiophate have been shown to have cholinesterase activities as low as less than 5 per cent of normal,[3] with a much slower breakdown of ester-linked local anesthetics than in the patient reported.

The authors make the point that the normal durations of anesthesia they observed indicate safety of chloroprocaine in patients with low cholinesterase levels. It is not surprising that the duration of anesthesia was normal. This was previously observed in a similar case by Raj *et al.*[4] The difference between minimum blocking concentration at the nerve for a drug such as procaine (2 mg/ml) and the toxic blood level (0.01–0.015 mg/ml) is substantial. A hypothetical tenfold increase in blood level due to decreased metabolism would have little impact on the concentration gradient removing drug from the blocked nerve. Therefore, the duration of the block would not be affected, but the likelihood of toxicity would be greatly increased. The latter is a function of activity at the target organ (CNS, heart) and is closely correlated with arterial blood levels. It is surprising that Brodsky and Campos[1] use the study by Raj *et al.*[4] to confirm their view, as the patient with atypical cholinesterase in the latter study in fact did have a prolonged increase in the arterial blood chloroprocaine level.

The authors correctly restrict their conclusion to patients with low levels of active cholinesterase (some patients with atypical cholinesterase do not metabolize some ester anesthetics at all[5]). Still, this case report does not warrant the generalization they make that chloroprocaine probably is a safe anesthetic for all such patients.

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In reply:—The comments in the above letters are valid. Since clinical experience with chloroprocaine in patients with depressed levels of pseudocholinesterase is limited, I feel that the case report fulfilled its purpose, *i.e.*, to report such an experience. The generalized statements made in the discussion section of this report cannot be substantiated by a single case. When ester-linked local anesthetics are used in patients with decreased or abnormal pseudocholinesterase levels, careful monitoring and extreme caution are, of course, necessary.

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