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(Accepted for publication January 30, 1986.)

Anesthesiology
65:119, 1986

Neurotoxicity of Local Anesthetics

To the Editor:—The altered perineural permeability, edema, and nerve fiber injury after local anesthetics as described by Myers *et al.*[1] provide interesting reading. It is unfortunate that the two local anesthetics incriminated in causing some degree of nerve damage were not plain local-anesthetic solutions. The 3% 2-chloroprocaine HCl used contained 0.2% sodium sulfite and the 1% tetracaine HCl contained 0.2% sodium bisulfite. The amide local anesthetics used contained no antioxidants.

As there already exists the question of possible neurotoxicity from the antioxidants rather than the local anesthetic,[2,3] perhaps it would be more relevant if the control group in the article by Myers *et al.*[1] had been the antioxidant with sodium chloride. If the results were unchanged, then their conclusion, that the two ester local anesthetics are less safe than the two amide local anesthetics, is given more credence.

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3. Wang BC, Hillman DE, Turndorf H: Chronic neurological deficit and Nesacaine-CE: an effect of the anesthetic, 2 chloroprocaine or the antioxidant, sodium bisulfite? Anesth Analg 63:445–447, 1984

(Accepted for publication March 12, 1986.)

Anesthesiology
65:119–120, 1986

In reply:—Our investigations into the neurotoxicity of local anesthetics were stimulated by the clinical reports and debate concerning the neurotoxicity of Nesacaine-CE® following inadvertent injection into the subarachnoid space. As noted by Dr. Cartwright, Wang *et al.*[1] have developed a rabbit model to mimic the clinical problem and have shown that sodium bisulfite is neurotoxic when evaluated with clinical measurements of function. Many of the initial investigations by other laboratories appeared to be inconsistent, variably reporting that neurotoxicity might be the result of the Nesacaine-CE® vehicle,[1] the local anesthetic 2-chloroprocaine,[2] or any local anesthetic.[3] These results appeared to be resolved by the observations of Gissen *et al.*,* who attributed nerve injury to sodium bisulfite at low *p*H. Unfortunately, their model does not explain cases in which nerve injury was observed with other local anesthetics (*e.g.*, reference 3) or in which neurotoxicity was not differentially produced by the bisulfite-containing 2-chloroprocaine solution when compared with other local anesthetics.[4]

In the discussion we stated that, "On the basis of these findings, we would not agree that local anesthetics of the ester type are relatively more safe than the amide type. Dose–response studies are necessary, however, to further test this hypothesis." We have subsequently found no evidence for distinguishing between the toxicity of ester- and amide-linked local anesthetics in this model.[5],† Commercial preparations of four amide-linked and three ester-

* Gissen AJ, Datta S, Lambert D: The chloroprocaine controversy. II. Is chloroprocaine neurotoxic? Regional Anesthesia 9:135–145, 1984.

† Kalichman MW, Powell HC, Myers RR: Pathology of local anesthetic–induced nerve injury. (submitted for publication).