Title: EFFECTS OF SODIUM BISULFITE ON SPRAGUE-DAWLEY RAT SCIATIC NERVE: A PRELIMINARY REPORT

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Purpose:

Sodium bisulfite is the antioxidant contained in the commercial preparations of several local anesthetics. Although experimental studies on the toxic effects of this compound in the subarachnoid space have been reported, investigations into the toxicity of this agent on peripheral nerves has received little attention. The purpose of this study was to determine if sodium bisulfite caused any neural damage when injected in vivo into the sciatic nerve.

Materials and Methods:

Fifteen Sprague-Dawley male rats weighing 250-300 grams were used. Ketamine, 100mg/Kg was given for the procedure. The animals were divided randomly into five groups: 1 cc, 1% (Group I); 1 cc, 2.5% (Group II); and 1 cc, 5% (Group III) solution sodium bisulfite in normal saline was injected blindly to the sciatic nerve. Two control groups were used: 1 cc normal saline injected as above (Group IV) and no injection (Group V).

After injection, all animals were left to recuperate and observed for motor and sensory changes. After a predetermined interval of 48, 96 hours and 20 days, animals were sacrificed. Sections for histopathologic study included nerve and adjacent muscle. Two portions were fixed in formalin for routine light microscopy, one embedded in cross section, the second longitudinally.

Results:

Paralysis and loss of sensation of the limb injected, was seen in all groups except controls. Motor function was recovered approximately 48 hours after injection in all groups affected.

Histopathology: Sections from both control groups (I and II) were unremarkable. The 1% and 2.5% solution of sodium bisulfite produced changes suggestable of disorganization of nerve bundles, inflammation with destruction of muscular fibers and granulation tissue encroaching nerve bundles. Samples obtained at the 20 day interval, showed reparative changes with glial cells proliferating in nerve tissue. The 5% solution showed the most profound changes. Nerve fibers appeared in disarray, with vacuolar degeneration and pyknotic changes. Muscle fibers adjacent to the above also showed early coagulative necrosis.

Conclusions:

In this study, despite the use of the antioxidant directly into the nerve and muscle tissue, all animals recovered motor and sensory function independent of the solution concentration. The histopathologic changes seen were short-lived as signs of regeneration were observed in all groups affected at the 20 day interval.

Though generally believed to be safe, sodium bisulfite has been known to have adverse mutagenic properties and show local irritation in multiple tissues. Chronic paralysis has been produced after repeated subarachnoid injections of anesthetics containing this compound, and axonal degeneration has been reported with the use of bupivicaine. The damage seemed to be augmented by the addition of epinephrine, a situation in which the preservative sodium bisulfite seems to have played a pivotal role.

The dilemma of whether or not sodium bisulfite should continue to be incorporated in drug preparations has major significance. This substance is contained in may foods, beverages and pharmaceuticals. Further studies should be done to elucidate the long term effect of sodium bisulfite on peripheral nerves.

References:

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