

CORRESPONDENCE

payments. Indeed, the income from the annual meeting allows the ASA to keep its dues structure lower than that of many professional organizations.

The ASA imposes no unreasonable financial demands on the exhibitors at its annual meeting. Furthermore, no exhibitor expends \$500,000 for the exclusive purpose of a display at the ASA annual meeting. Most displays are used many times for other meetings. Some exhibits include *very* expensive anesthesia delivery systems (machines) and monitoring devices that cannot be included in the actual cost of exhibiting. An exhibiting company that chooses to send 20 or more people to the meeting clearly has the option of sending a smaller delegation.

Next we come to the bells and whistles aspects of commercialism. I refer to the pens, flashlights, butterscotch candies, *etc.* that work a disturbing magic upon the intellect and behavior of the scientific pilgrims who seek knowledge and spiritual enrichment on the floor of the exhibit hall. The ASA imposes significant restraints upon the activities of those companies that participate in its annual meeting

as regards receptions, galas, and "perks." It is difficult to achieve a balance that satisfies the needs of all our members and at the same time serves the legitimate commercial interests of our colleagues in industry. It is an open market economy, and we are part of it.

The excesses on both sides of the equation—the rapacious response of our members to shiny objects and cocktail wieners, and the seductive blandishments of salesmen determined to corrupt the conscience of humankind—should be restrained. Whether this is best achieved by regulation or moral persuasion is an open question.

Let's try moral persuasion: I call on all those involved to put economy and value before personal benefit. Our patients deserve it.

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Does Diabetes Reduce Local Anesthetic Requirement?

To the Editor:—Kalichman's laboratory has pioneered sophisticated neuroanatomic techniques quantitating nerve injury from exposure to local anesthetics. And they show that nerve of streptozotocin-treated rats is injured more severely by lidocaine than by saline.¹ The 4% lidocaine solution appears particularly damaging and, perhaps for that reason, is restricted to surface (topical) application in humans and labeled "not for injection."

My quibble is one of implication. It is not whether streptozotocin produces hyperglycemia (clearly, blood sugar level was increased 2.5 times that of control), but whether hyperglycemia necessarily implies diabetic neuropathy—a segmental demyelinating or axonal degenerative process of slow onset (in humans).² The author's figure 6, for instance, depicts well myelinated, densely packed intact fibers, a "... normal-appearing diabetic rat nerve ..." per the legend.

Muscle action potential amplitude appears to have more than doubled by streptozotocin treatment (amplifier sensitivity was reduced from 200 μ V to 500 μ V in fig. 1). Could reduced recorder sensitivity perhaps explain the apparent enhanced potency of procaine (but not of lidocaine) in hyperglycemic "diabetic" rats?

This still leaves unresolved whether 1% lidocaine or procaine is more neurotoxic in treated than in untreated rats, and whether the outcome was due to increased serum glucose levels or whether the results reflect the subtle neurotoxicity of streptozotocin. One wonders

"... whether these empiric findings in animal models have a clinical parallel."

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