Letters to the Editor

Prevention of Nitrous Oxide-Induced Increases in Endotracheal Tube Cuff Pressure

To the Editor:

I read with interest the letter by Brandt (1) in the February edition of your journal. I wish to propose an even simpler method of preventing excessive rise in endotracheal tube cuff pressure during the administration of a nitrous oxide anesthetic. If one simply fills the endotracheal tube cuff with the mixture of nitrous oxide and oxygen to be used during anesthesia administration, then there is no diffusion gradient by which nitrous oxide can enter the cuff. There will therefore effectively be no change in cuff pressure during anesthesia. This simple system is remarkably effective.

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References

Back Pain in Volunteers After Epidural Anesthesia With Chloroprocaine

To the Editor:

I found the recent report by Hynson et al. (1) very interesting. I frequently use 2% and 3% 2-chloroprocaine in my practice while performing diagnostic and therapeutic anesthetic blocks for patients in chronic pain.

I also have seen severe back pain and muscle spasm after these blocks but my experience would not implicate ethylenediaminetetraacetic (NaEDTA) acid as the precipitating agent of this phenomenon as was suggested. I frequently use 2% 2-chloroprocaine in performing my blocks and only rarely have a problem despite the use of relatively large volumes (20–60 mL injected in approximately 5-mL increments), whereas on the few occasions I have needed to progress to 3% 2-chloroprocaine, there seems to be a high incidence of muscle spasm and severe pain as the block wears off—despite the use of smaller volumes overall. As NaEDTA is present in both formulations in the same concentration, based on my own experience it is unlikely that it is the sole source of this phenomenon. However, I agree it is a real phenomenon and something seen only rarely with the previous formulation of this drug.

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Reference

In Response:

In our discussion, we suggested several potential etiologies for back pain after epidural chloroprocaine administration, but did not endorse one in particular. The possible causes listed included the theory proposed by Fibuch and Opper (1) that pain is caused by EDTA. In support of this etiology, Wang et al. recently reported that hourly lumbar epidural injections of 0.1 mL of 0.1% EDTA in rats result in tetanic contractures of the lower extremities and focal neuropathologic changes when the total dose exceeded 0.1 mg (2). For comparison, the total dose of disodium EDTA after a 35-mL lumbar epidural injection of Nesacaine-MPF in humans would be 3.5 mg. (The concentration of disodium EDTA in Nesacaine-MPF is 0.011%.) Although not definitely implicating EDTA as the causative agent, this study suggests that further investigation of its neurophysiologic effects, neurotoxicity, and myotoxicity is warranted.

Dr. Day's personal observations of back pain after the use of chloroprocaine in the pain clinic are interesting. One consistency in the published reports, as well as the experience of Dr. Day and other clinicians with whom we have spoken, is that this characteristic back pain after 3% chloroprocaine has been observed primarily in patients treated on an outpatient basis. The recent emergence of this problem could in fact be unrelated to the introduction of Nesacaine-MPF. Rather, the increased number of outpatients receiving epidural blocks in recent years may simply have helped to uncover the association of back pain with epidural chloroprocaine.

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References