weight of the patient are not given, this limited spread is consistent with that found commonly in thin parturients. Second, subarachnoid injection of a dose of tetracaine larger than that currently recommended for cesarean section extended the sensory loss cephalad only by two segments, to T6. This suggests that the subarachnoid injection was ineffective and the increased sensory loss was due to the further spread of the epidurally administered local anesthetic. It is not stated whether aspiration from the subarachnoid space was done after the tetracaine had been given. Progressive sensory loss from 0.75 per cent bupivacaine administered epidurally can continue for more than an hour after injection. Third, the block was unilateral on the nondependent side. Unsatisfactory epidural analgesia can be due to limited cephalad spread bilaterally/unilaterally on the dependent side or unilaterally on the nondependent side. Review of our data shows that in 5 per cent of unsatisfactory epidural blocks, the sensory loss was considerably less on the dependent side.

From the evidence given, the most likely cause of the unilateral analgesia in this case is limited spread of the epidurally administered local anesthetic.

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REFERENCES


(Accepted for publication April 29, 1980.)

Biotransformation of Nitrous Oxide

To the Editor:—Dr. Hong and co-workers1 have performed an elegant and meticulous study of nitrous oxide metabolism by intestinal contents, and have shown that one more “inert gas” can be biotransformed in the body. There is no evidence in their article, however, to support their conclusion that N2 is the major metabolite of N2O. They analyzed only the headspace gas incondensable at -195.8 C. This treatment would remove NO, NO2, and NH3, all possible gaseous biotransformation products. Neither were water-soluble, nonvolatile nitrogen compounds (e.g., NO2-, NO3-, NH4+) sought in the aqueous incubation mixture.

The decrease in N2 production at higher oxygen tensions that they observed could reflect not a decrease in N2O metabolism, but rather an increase in the production of oxygenated metabolites.

Certainly, N2 is a product of bacterial metabolism of N2O. It remains to be demonstrated that it is the major or only metabolite. When this question is answered, we will still be left with the more intriguing and perhaps unanswerable question, can mammalian systems bio-transform N2O?

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(Accepted for publication April 29, 1980.)

In reply:—Drs. Linde and Avram correctly state that we have asserted that nitrogen is the major metabolite of nitrous oxide without providing evidence of a search for other possible metabolites. Research on the metabolism of nitrous oxide in these laboratories was begun in 1972. As a result, we have much information not incorporated in our recent article.1 In fact, we have looked for 15N-containing metabolites in in