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Correspondence

Tension Pneumocephalus and the Sitting Position

To the Editor:—The recent report of tension pneumocephalus[1] following a sitting posterior-cranial-fossa exploration with nitrous oxide–relaxant anesthesia called to our attention a similar experience.

In May 1975, a 43-year-old man who had right hemiparesis secondary to cervical spondylosis underwent a sitting posterior-cervical laminectomy (C3 to C7). Anesthesia consisted of nitrous oxide–oxygen–*d*-tubocurarine, with controlled ventilation. Postoperatively, complete left hemiplegia, not present prior to operation, was evident. Bilateral retrograde carotid arteriograms ruled out intravascular air as the etiology. A large bilateral prefrontal and subfrontal extracerebral collection of air, presumably subdural, was demonstrated.

We felt that this episode was a bizarre experience with little general clinical application. We call attention to the fact that this complication of the sitting surgical position may be more common than previously presumed. The potential deleterious effect of nitrous oxide in such circumstances must be given careful consideration.

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REFERENCE

1. Kitahata LM, Katz JD: Tension pneumocephalus after posterior-fossa craniotomy, a complication of the sitting position. ANESTHESIOLOGY 44:448–450, 1976

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Nitroprusside-induced Metabolic Acidosis

To the Editor:—The April issue contained two papers on the interesting subject of metabolic acidosis occurring during and after administration of sodium nitroprusside.[1,2] Once again, a high proportion of baboons, five of nine, bit the dust when given this agent. In the experiments of McDowall *et al.*[3] four of eight baboons died.

These figures contrast strangely with the experience in our center, where more than 1,000 patients have received sodium nitroprusside. There has been no fatality, and only one patient has had severe metabolic acidosis, which responded to treatment.[4] Could it be that some inherent metabolic defect exists in baboons which makes them susceptible to the agent? Such a defect could be a species difference, but it is also possible that the well-known in-breeding of experimental animals has produced a strain with a peculiar susceptibility. This would be analogous to the strain of pig that so readily develops malignant hyperthermia, and could be a factor in the high mortality rate. If this were so, it does not invalidate the results obtained in the experimental situation. It might indeed, spur the research workers on to attempt to elucidate the nature of the metabolic disorder and thereby make it predictable in man.

The second aspect of note is the small but growing literature on deaths following the now characteristic pattern of "tachyphylaxis" followed by severe metabolic acidosis. So far as I am aware, our paper[4] gives the only report of recovery following such a sequence of events. This may be because most people do not report near-misses, or it could be because lesser degrees of metabolic acidosis go unnoticed. However, were it not for our