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*In reply:*—This is in response to the letter from Dr. Rumley. All of his points are almost meticulously correct and are well-taken, including the statements of implied cause and effect. However, we have considered the detrimental effects of improperly cleansed colons and this was clearly discussed in paragraph four of the discussion.

When the risks of parenteral administration equal the risks of long-term pulmonary aspiration in esophageal and dysphagic problems, peroral bulk of any kind must be avoided. Obviously, in these patients, orally administered antibiotic bowel preps also most likely will be less than optimum in terms of fermentative bacterial growth inhibition.

The conspiracy of factors including inadequate control of bacterial flora, fermentation of a nitrogen-carbohydrate substrate, explosive gas trapped by a cutting stapler, application of heat and spark, are cause and effect. One might add that we sometimes are not masters of our technology, and then in a sequential fashion get lulled and hoisted by our own petard. All of the explosion hazard and O.R. designs would not help this patient.

Certainly Ensure does not cause explosions. Sparks do. Since spectrophotometric studies were not done of

the explosion, it is impossible to determine the absolute components of the explosive mixture. Nevertheless, we will no longer open the colon with electrocautery, whether it be empty or full, clean or dirty, prepared or unprepared—unless it is vented or suctioned. We will certainly continue our efforts according to the excellent principles of bowel preparation expressed by Dr. Rumley, including the use of Ensure or an equivalent.

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## Nitrous Oxide and the Prevention of Tension Pneumocephalus after Craniotomy

*To the Editor:*—In a recent clinical report,<sup>1</sup> Artru states that his intraoperative observations of increased ICP with introduction of N<sub>2</sub>O and decreased ICP with discontinuation of N<sub>2</sub>O after dural closure in the sitting position fail to support my proposal for prevention of tension pneumocephalus in the *postoperative* period. I suggested that “it may be advantageous to maintain anesthesia with high inspired concentrations of nitrous

oxide until dural closure so that a pneumocephalus that formed intraoperatively would contain nitrous oxide that would then be reabsorbed rapidly when nitrous oxide was discontinued.<sup>2</sup> What Artru actually noted was a dramatic increase in ICP when nitrous oxide was *continued* after dural closure. There was then a rapid decrease in ICP (within five minutes) after discontinuing nitrous oxide. Had nitrous oxide been discontinued

upon dural closure as I had suggested, the problem would not have arisen in the first place. This finding agrees with the experience of Saidman and Eger,<sup>3</sup> and demonstrates that a pneumocephalus resulting from craniotomy may accumulate a significant amount of nitrous oxide. As Artru's experience demonstrates, this can be reabsorbed within minutes if nitrous oxide is discontinued. Artru's report supports rather than vitiates my conclusion that nitrous oxide does not play a significant role in the pathogenesis of tension pneumocephalus occurring in the *postoperative* period. If pneumocephalus is present at the moment of dural closure, its reabsorption may be accelerated if it is made to contain some fraction of nitrous oxide by administering the gas prior to closure, and then promptly discontinuing nitrous oxide following closure of the dura.

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### Nitrous Oxide and Intraoperative Tension Pneumocephalus

*To the Editor:*—Artru's conclusion that nitrous oxide plays a direct role in the development of tension pneumocephalus intraoperatively seems most reasonable.<sup>1</sup> In fact, several other factors may combine to make this a much more common complication than has been realized previously.

The transphenoidal approach to the pituitary gland generally is performed with the patient maintained in a slightly head-up position. A lumbar subarachnoid catheter or needle frequently is inserted, through which 2–3 ml air may be injected intraoperatively to confirm, fluoroscopically, complete extirpation of the tumor. We also have used this system as a convenient means to measure lumbar subarachnoid pressure (LSAP). Following injection of 3 ml air to a patient anesthetized with nitrous oxide, LSAP was noted to increase from 5–15 mmHg over a seven-minute period. Nitrous oxide was discontinued and the pressure returned to baseline levels. Approximately 15 minutes later, nitrous oxide again was introduced to the system and again the pressure promptly increased, although no more air had been injected. We assumed that the initial infusion of air increased the pressure by the well-recognized nitrous oxide/nitrogen displacement mechanism. A larger gas pocket thus was created. With discontinuation of nitrous

oxide, this gas was then absorbed, creating negative pressure which then sucked more air through the surgical wound. The phenomenon probably was aggravated by the upright position as fluid could not be used to flood the field. Reintroduction of nitrous oxide some minutes later thus caused further pressure increases.

It has become our practice to eliminate nitrous oxide from the anesthetic regimen some 10–15 minutes prior to the anticipated injection of air and not to introduce it again for the duration of the procedure. It may well be that routine use of nitrous oxide in any neurosurgical procedure should be more completely examined.

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