time taken to zero the transducers, measure intracranial elastance, possibly insert head pins, etc. Because MAC for nitrous oxide is greater than 1 atmosphere, the authors seem to be relying rather heavily on some unspecified residual amount of thiopental and/or lidocaine, after an unspecified period of time, if they, indeed, believe they were maintaining general anesthesia in these patients at the time of craniotomy.

In the absence of a proper control group, cannot one conclude with at least equal justification that isoflurane protects against rises in intracranial pressure in patients whose general anesthesia is "maintained" with 70% nitrous oxide in oxygen, because intracranial pressure did not increase in eight of the 14 patients studied?

If the effects of isoflurane on intracranial pressure had been studied in a steady state, i.e., with a constant end-tidal (rather than inspired) isoflurane concentration, would intracranial pressure have increased in even fewer patients?

A. Donald Finck, M.D.
Associate Professor
Department of Anesthesiology
College of Physicians and Surgeons
Columbia University
630 West 168th Street
New York, New York 10032

REFERENCE

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In reply:—With all due respect, we submit that Dr. Finck missed the stated goal of our clinical report. Adams et al., using patients with brain tumors as their own control, demonstrated quite adequately that isoflurane increases cerebrospinal fluid pressure during normocarbic steady-state anesthesia. We had no intention of simply reduplicating their work. The purpose of our study, as stated in the introduction, was to identify which patients with intracranial neoplasms are at risk for developing increases in intracranial pressure (ICP) during inhalation of isoflurane, compared with those with intracranial neoplasms who are not at risk. Although inclusion of a control group not receiving isoflurane might have yielded interesting results, we cannot imagine how it would have aided us in reaching a conclusion concerning a question that we felt to be clinically pertinent.

Speaking of clinical relevance, Dr. Finck takes us to task for not achieving steady-state conditions during the period of time we studied the effects of isoflurane. We submit that at the time of skin incision, most clinicians administer anesthetics based on patients’ responses rather than numbers. In paralyzed patients it hardly matters whether an end-tidal concentration of isoflurane sufficient to prevent movement in one-half the patients (1 MAC) had been achieved as long as a dose of isoflurane was administered that was sufficient to block adrenergic responses to surgical stimulation. This is clearly depicted in our figure 1, where blood pressure and ICP were 120/80 and 15, respectively, at the time isoflurane and surgery were begun, and where the corresponding values were 115/80 and 25, respectively, at the time isoflurane was discontinued (followed by a prompt reduction in ICP). If Dr. Finck wishes to conclude "that isoflurane protects against rises in intracranial pressure," then we repeat: he has missed the point.

Kenneth Grosslight, M.D.
Robert F. Bedford, M.D.
Department of Anesthesiology
University of Virginia Medical Center, Box 238
Charlottesville, Virginia 22908

REFERENCE

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