pharynx. In very rare entities such as megaesophagus and esophageal diverticulum, cricoid pressure may not be effective and may be hazardous. In retropharyngeal abscess, cricoid compression may result in rupturing the abscess and flooding the tracheobronchial tree, especially if excessive cricoid pressure is applied and, therefore, should be avoided.[5]

It is unlikely that early postmortem changes of the cricopharyngeal and esophageal musculature and/or localized edema might have contributed to superior conditions in our experiments. It is also unlikely that sagittal localization of the nasogastric tube hinders the efficacy of cricoid compression. In a previous study in anesthetized pediatric patients,[6] roentgenographic films of nasogastric tubes previously filled with contrast material and tied at both ends showed displacement of the nasogastric tubes during cricoid compression. Because the maneuver was effective in sealing the esophagus around the nasogastric tubes, we believe that sagittal localization of nasogastric tubes is of no significance.

Finally, our investigation was not intended to compare rapid-sequence induction to awake intubation in the management of patients with a full stomach. We merely evaluated the efficacy of cricoid compression in obliterating the esophageal lumen in the presence of an esophageal tube.

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Isoflurane and Intracranial Pressure

To the Editor:—Grosslight *et al.*[1] have shown that intracranial pressure rose in six of fourteen patients who were paralyzed, breathing 70% nitrous oxide, and given isoflurane, 1% inspired, for several min at the onset of craniotomy. If one is going to ascribe the rise in intracranial pressure to an effect of isoflurane, as the authors tentatively conclude, then at least two sets of conditions must be met—which were not.

First, a control group consisting of patients not given isoflurane is needed. It was not appropriate to use patients given isoflurane but whose intracranial pressure did not rise as a control group. What happens to intracranial pressure in paralyzed patients whose general anesthesia is "maintained with nitrous oxide, 70% in oxygen" during the beginning of a craniotomy? Or, is the answer given in figure 1, which shows a clear increase in both systemic arterial as well as intracranial pressures? And how does one reconcile the *increased* systemic arterial pressure shown in figure 1 with the *decrease* of 30 mmHg in mean arterial pressure shown in table 1?

Second, steady-state conditions should apply, and they clearly do not in this study. The patients were studied at a time when alveolar and arterial isoflurane concentrations were dynamically changing, rather than in a steady state, not to mention the confounding influence of the surgical stimulation, which commenced at the same time isoflurane was added to the inspired mixture.

Although these unpremedicated patients received thiopental, 3 mg/kg for induction and then 2 mg/kg for tracheal intubation, along with 1.5 mg/kg of lidocaine, considerable time had to elapse prior to skin incision for craniotomy. The airway had to be secured, the head turned to one side and "prepped and draped," a burr hole made, a subarachnoid bolt inserted aseptically, and the head repositioned to the other side and prepped and draped at the very minimum. This does not account for

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?

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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.*,[1] 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.

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