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In Reply—We agree with Gajraj et al.’s statement that repeated attempts at inserting the laryngeal mask airway (LMA) must not be made at the expense of adequate ventilation and oxygenation. Nevertheless, despite the difficulties, we did insert the LMA successfully. During the attempts at insertion (60–90 s total time required), the patient’s oxygenation as reflected by pulse oximetry (SpO2) remained unchanged, and only a few minutes later the SpO2 decreased to 90–92%, suggesting postobstructive pulmonary edema as the cause of hypoxia.

In reply to Stiff et al., in both cases we employed inhaled anesthesia for induction, and the LMA was inserted under deep halothane anesthesia. The upper airway obstruction persisted about 5 min, and pulmonary edema was diagnosed about 30 min later in the post-anesthesia care unit. Certainly, we agree with Stiff et al. that the airway obstruction induced pulmonary edema in our two patients and not the LMA per se.

In reply to the remarks by Brimacombe and Berry, we usually insert the LMA under deep halothane anesthesia. To date, no difficulties have been encountered with this technique in more than 1,000 patients. Regarding the skills for insertion of the LMA, Broderick et al. could find no differences in the number of insertion attempts between consultants and trainees, despite the latter’s limited previous experience. Johnston et al. reported a success rate of 67% on the first attempt.

According to Rowbottom et al., despite adequate depth of anesthesia, 19% of cases had partial and 2% had total airway obstruction after LMA insertion, as confirmed by fiberoptic endoscopy. Brimacombe and Berry question the possibility that pulmonary aspiration may occur with the LMA. Carlson and Islander found a 20% incidence rate of acidic material regurgitation in the pharynx during general anesthesia. This was confirmed recently by Barker et al., who found a 25% incidence of regurgitation with the LMA. As shown by Criswell and John, the LMA does not prevent soiling of the larynx by stomach contents in the event of regurgitation, and the vomitus is preferentially directed by the LMA from the esophagus into the larynx.

Brimacombe and Berry also recommend the rapid use of a fiberoptic bronchoscope if airway obstruction develops after LMA insertion. In our opinion, fiberoptic endoscopy in this situation not only could be difficult because of secretions and blood but could further compromise the airway and worsen hypoxemia. Fiberoptic examination requires more time than does conventional laryngoscopy even when performed by an expert.

Finally, the fact (as stated by Brimacombe and Berry) that, despite worldwide use of the LMA in millions of patients, no pulmonary edema was reported does not mean that it did not occur. Whenever persistent hypoxemia is observed after insertion of the LMA, postobstructive pulmonary edema might be considered.

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Cerebrovascular Autoregulation May Be the Probable Mechanism Responsible for Fentanyl- and Sufentanil-induced Increases in Intracranial Pressure in Patients with Head Trauma

To the Editor—Sperry et al. gave 3 μg/kg fentanyl or 0.6 μg/kg sufentanil to nine patients with head trauma and increased intracranial pressure (ICP) in a randomized, double-masked, crossover design study. Mean arterial blood pressure (MAP) decreased from 92 ± 5 to 81 ± 6 mm Hg and from 92 ± 5 to 82 ± 4 mm Hg in the fentanyl and sufentanil groups, respectively, whereas ICP increased from 9.7

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* Unpublished data.
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± 1.8 to 17.4 ± 2.9 mmHg and from 7.1 ± 1.8 to 13.2 ± 2.8 mmHg in the fentanyl and sufentanil groups, respectively.

The ICP zenith and MAP nadir occurred 6 and 10 min, respectively, after fentanyl and 4 and 8–15 min, respectively, after sufentanil administration.

They offered five explanations: (1) direct cerebrovascular smooth muscle dilatation, (2) an increase in the cerebral metabolic rate for oxygen, (3) systemic hypotension producing cerebral ischemia, (4) histamine release, and (5) effects of opioids on cerebral spinal fluid production and/or absorption.

They did not discuss cerebrovascular autoregulation, which may be a reasonable explanation for their findings.

Changes in cerebral perfusion pressure (CPP), within the autoregulatory range, have little effect on cerebral blood flow (CBF) because cerebral vasodilatation occurs; however, this compensatory mechanism also increases cerebral blood volume (CBV) and the CBV/ CBF ratio. In comatose patients with head trauma, pharmacologic tests of cerebrovascular autoregulation of CBF, using phenylephrine or trimethaphan, resulted in a steep increase in ICP (from 20 ± 3 to 30 ± 2 mmHg) when MAP was decreased (from 120 ± 13 to 91 ± 10 mmHg) in patients with intact autoregulation, whereas in those with impaired autoregulation, decreasing MAP (from 112 ± 19 to 90 ± 17 mmHg) decreased ICP (from 16 ± 5 to 11 ± 7 mmHg). Increasing MAP in these patients did not change ICP, presumably because autoregulatory vasocostriction results in a maximum 10% decrease of baseline diameter, whereas autoregulatory vasodilatation may result in up to 65% increase in baseline diameter. In another study, the cerebrovascular responses to CPP changes were blunted in animals anesthetized with pentobarbital, presumably because of the cerebrovasoconstricting effect of this drug.

In summary, cerebrovascular autoregulation, intracranial elastance, and cerebrovascular effects of anesthetic drugs may result in complex interactions in patients with head trauma. The increases in ICP observed by Sperry et al. may be due to opioid-induced increased CBV in patients with compromised intracranial elastance and intact autoregulation and deserve further investigation.

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In Reply—Posing an alternative mechanism for our finding,1 DeLima describes a study of cerebrovascular autoregulation in patients with head trauma.2 In patients with intact autoregulation, a decrease in blood pressure correlated with an increase in intracranial pressure (ICP). In patients with impaired autoregulation, a decrease in blood pressure did not correlate with an increase in ICP. Our study was small, and we did not believe we could legitimately separate different patient groups. However, in our results we found that there probably were two patient populations. Five of the nine patients accounted for 86% of the variation of ICP from baseline when administered fentanyl and 74% of the variation when administered sufentanil.

It is possible, although by no means proved in our study, that these two patient populations represent those described in the cited study. Further work is required to delineate this possibility.

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