

CORRESPONDENCE

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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.*¹ could find no differences in the number of insertion attempts between consultants and trainees, despite the latter's limited previous experience. Johnston *et al.*² reported a success rate of 67% on the first attempt.

According to Rowbottom *et al.*,³ despite adequate depth of anesthesia, 19% of cases had partial and 2% had total airway obstruction after LMA insertion, as confirmed by fiberoptic endoscopy.³

Brimacombe and Berry question the possibility that pulmonary aspiration may occur with the LMA. Carlsson and Islander⁴ found a 20% incidence rate of acidic material regurgitation in the pharynx during general anesthesia. This was confirmed recently by Barker *et al.*,⁵ who found a 25% incidence of regurgitation with the LMA.

As shown by Criswell and John,⁶ 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.⁷

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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References

1. Brodrick PM, Webster NR, Nunn JF: The laryngeal mask airway: A study of 100 patients during spontaneous breathing. *Anaesthesia* 44:238–241, 1989
2. Johnston DF, Wrigley SR, Robb PJ, Jones HE: The laryngeal mask airway in pediatric anaesthesia. *Anaesthesia* 45:924–927, 1990
3. Rowbottom SJ, Simpson DL, Grubb D: The laryngeal mask airway in children: A fiberoptic assessment of positioning. *Anaesthesia* 46:489–491, 1991
4. Carlsson C, Islander G: Silent gastropharyngeal regurgitation during anaesthesia. *Anesth Analg* 60:655–657, 1981
5. Barker P, Langton JA, Murphy PJ, Rowbotham DJ: Regurgitation of gastric contents during general anaesthesia using the laryngeal mask airway. *Br J Anaesth* 69:314–315, 1992
6. Criswell J, John R: The laryngeal mask: Cautionary tales. *Anaesthesia* 45:168, 1990
7. Stehling LC: Management of the airway, *Clinical Anesthesia*. Edited by Barash PG, Cullen BF, Stoelting RK. Philadelphia, JB Lippincott, 1989, p 557

* Unpublished data.

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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.*¹ 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 (MABP) decreased from 92 ± 5 to 81 ± 6 mmHg and from 92 ± 5 to 82 ± 4 mmHg in the fentanyl and sufentanil groups, respectively, whereas ICP increased from 9.7