severe anaphylactoid reaction during open heart surgery probably caused by protamine. The findings of an elevated tryptase and positive protamine skin tests remain inadequate to answer questions concerning the mechanism of this severe reaction.

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References


(Accepted for publication August 6, 1996.)

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Fiberoptic Tracheal Intubation Using a Nipple Guide

To the Editor.—Fiberoptic tracheal intubation of the infant may be assisted via a laryngeal mask airway (LMA), a standard mask, or a ventilating mask. Of these devices, only the LMA acts as an oropharyngeal laryngeal conduit, through which a flexible fiberoptic bronchoscope may be placed directly above the vocal cords. Unfortunately, the LMA is poorly tolerated by the awake infant. We describe an alternate device that facilitated fiberoptic bronchoscopic tracheal intubation of an infant with an unstable cervical spine who could not be safely anesthetized before intubation.

A 7-month-old premature infant with a history of bronchopulmonary dysplasia, apnea and bradycardia of prematurity, and chronic respiratory failure that required prolonged intubation was admitted with rapidly progressive upper extremity weakness. A magnetic resonance imaging (MRI) examination was indicated to rule out a space-occupying lesion that involved the spinal cord. The combination of the patient's medical history and his remote position while in the MRI scanner necessitated tracheal intubation with controlled ventilation. Because of his progressive paralysis, we were compelled to assume that his cervical spine was unstable, and that direct laryngoscopy might result in permanent neurologic damage. In summary, we were confronted with a 7-month-old boy with an unstable cervical spine who could not sustain more than mild sedation for the fiberoptic placement of an endotracheal tube.

Fiberoptic bronchoscopy was performed in the operating room using a fiberoptic bronchoscope, which was passed through a LMA. This technique may be used as an alternative in the management of infants who require fiberoptic bronchoscopy.
Inhibition of Plasma Membrane Ca$^{2+}$-ATPase by Volatile Anesthetics

To the Editor:—We recently realized that the concentrations of volatile anesthetics reported in our publications$^{2,3}$ are incorrect. It follows that the activity of the plasma membrane Ca$^{2+}$-ATPase (PMCA) is half-maximally inhibited (IC$_{50}$ values) at 2.2–3.0 mM (instead of 0.22–0.30 mM) halothane and 2.1–3.2 mM (instead of 0.21–0.52 mM) isoflurane, which compare to 7–5–12 minimum alveolar anesthetic concentrations.

Although we cannot claim that PMCA is inhibited by volatile anesthetics at their clinical concentrations, all other findings presented in our papers are unaffected by the concentration error. These are: 1) analogous dose-dependent inhibition of PMCA activity by volatile anesthetics in neuronal and red cell membranes; 2) significantly higher sensitivity of the PMCA as compared with three other plasma membrane ATPases to the inhibitory action of halothane and isoflurane; and 3) lack of difference in sensitivity of PMCA versus the other ATPases to sodium pentobarbital, which inhibits them at 100 to 200 fold above its anesthetic concentrations.

With these in mind, we use the PMCA as a model of a membrane protein on which molecular events of anesthetic action could be elucidated.

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(Accepted for publication August 6, 1996)

Anesthesiology
1996; 85:1211–2
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Lippincott-Raven Publishers