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.

Christoph H. Kindler, M.D.
Staff Anesthesiologist
Department of Anaesthesia
Andreas J. Bircher, M.D.
Staff Allergist and Dermatologist
Department of Dermatology
University of Basel, Kantonsspital
CH-4031 Basel, Switzerland

References

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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.1 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 prematurity 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

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Fig. 1. A 3.4-mm fiberoptic bronchoscope jacketed with a 4.0-mm internal diameter endotracheal tube placed through a modified standard nipple.

with an otolaryngologist standing by to perform emergency tracheotomy if necessary. After applying topical anesthesia to the child’s oropharynx, using 2% lidocaine via atomizer, two attempts were made to place a 3.4-mm outer diameter bronchoscope (Pentax FB-10 X, Pentax Industries, Orangeburg, New York) into the patient’s trachea. On each occasion, the child struggled and the bronchoscopy was aborted. At this point, a nipple from a baby bottle (Standard Nipple Unit, Ross Laboratories, Columbus, Ohio) was produced and a 0.8-mm hole was cut obliquely into the end of the nipple (fig. 1). A mark was made on the rim of the nipple in alignment with the hole, to facilitate orientation of the hole inferiorly. When introduced into the child’s mouth, the nipple was eagerly accepted. Using the nipple as a conduit for the fiberoptic bronchoscope, an excellent view of the superior aspect of the epiglottis and larynx was obtained. The supraglottic region was anesthetized with 2% lidocaine injected through the auxiliary port of the bronchoscope. The bronchoscope was then advanced inferiorly past the vocal cords. A 4.0-mm internal diameter uncuffed endotracheal tube was then passed over the bronchoscope, through the nipple, and past the vocal cords. After securing the endotracheal tube, the child underwent MRI examination, which revealed a hemangioblastoma with cervical nerve root involvement. After tumor excision, the patient had minimal residual neurologic impairment.

Rarely, the need arises to secure the airway of an awake infant without the use of a laryngoscope. We describe the use of a modified nipple as an oropharyngeal-laryngeal conduit to aid fiberoptic intuba- tion of the trachea in this challenging context.

Randall Guskowicz, M.D.
Assistant Clinical Professor of Anesthesiology
Electronic mail: rguskowicz@ucsd.edu

Henri G. Colt, M.D.
Associate Professor of Medicine
Department of Anesthesiology
University of California, San Diego
200 West Arbor Drive MC 8380
San Diego, California 92103

Louis D. Youules, M.D.
Anesthesia Associates of Boise
Staff Anesthesiologist
St. Luke’s Regional Medical Center
Boise, Idaho 83702

Reference


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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\(^{1,2}\) 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 2.4–3.2 mM (instead of 6.24–0.52 mM) isoflurane, which compare to a 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.

Danuta Kosk-Kosicka, Ph.D.
Associate Professor
Anesthesiology and Critical Care Medicine
The Johns Hopkins University School of Medicine
600 North Wolfe Street/Blalock 1404-C
Baltimore, Maryland 21287-4961

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