Neonatal Resuscitation Using the Laryngeal Mask Airway


PATERSON et al. raise two issues worthy of attention in this issue of ANESTHESIOLOGY (page 1248). First, this clinically relevant study has the potential to ultimately influence clinical management as it opens the door for more extensive evaluation of the laryngeal mask airway (LMA) in emergent resuscitation of neonates and infants. Second, it illustrates the frequently unappreciated role of anesthesiologists acting as consultants to safely disseminate life-saving techniques to primary/emergency-care teams, as opposed to arriving intermittently to “put out fires” and then departing. This consultant role, appropriately, should increase as attention is focused on clinical situations in which maintenance of airway patency during sedation for painful procedures or imaging, for example, may be problematic.

The LMA, now commonly found in the operating theater in Great Britain, has a number of attractive features, most importantly, relative ease of placement without requiring direct visualization of the larynx to produce a patent airway for spontaneous ventilation. The attendant potential drawbacks include the lack of an intratracheal seal to allow positive-pressure ventilation and prevent aspiration. In addition, one cannot ignore the economic implications of purchasing large numbers of LMAs for, at present, uncertain effects on morbidity or mortality. The authors have demonstrated that pediatricians, anesthesiologists, and anesthesia residents with “expert skills in neonatal resuscitation” could rapidly learn how to successfully insert and use an LMA for resuscitating mildly to moderately depressed newborns with a gestational age of at least 35 weeks and weight 2.2–4.4 kg. This study represents an ethically appropriate beginning to evaluating whether the LMA has a significant role to play in this specific setting and in other clinical settings outside the operating room. By training only those who could (and did in 3 of the 21 cases) intubate the trachea when medically indicated, the authors provided maximum safety for the study patients. The authors’ results make it clear that the LMA is not a panacea but can be successful in this patient population. No comparison was made as to whether conventional (but difficult for the untrained) positive-pressure mask ventilation would have been successful. The finding that, despite peak airway pressures of 37 cmH₂O with audible leaks at an average of 22 cmH₂O, gastric distention was not clinically observed during the brief resuscitative efforts (approximately 2 min) suggests that the LMA may have some advantage. However, the average time for placement was 30 s, compared to presumptively immediate commencement possible with a mask and bag.

No evaluation of the ease of training and success rate of health-care providers with less initial expertise was carried out. The “trainees” in this study have more experience and a greater understanding of anatomy and physiology than lesser trained medical or paramedical personnel. It remains to be studied whether, analogous to the widely used training programs for these groups in advanced life support, training in animals and mannequins will be satisfactory and whether considerable aggregate experience in humans would be needed for safe implementation. This study raises the potential for extrapolation of this technique to less experienced M.D. and non-M.D. health-care providers in the delivery room, emergency room, ward, and field, specifically for neonates and infants, but for other patient populations as well.

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Volatile Anesthetic-induced Efflux of Calcium from IP₃-gated Stores in Clonal (GH₃) Pituitary Cells

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FOR about 150 yr, we have wondered how general anesthetics work. As our knowledge and sophistication of neurobiology and neurochemistry have grown, so our speculations about the specific targets of general anesthetics have advanced. Ample evidence testifies to the actions of inhalational and intravenous anesthetics

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on voltage-gated channels\textsuperscript{1,2} and on both excitatory\textsuperscript{3,4} and inhibitory\textsuperscript{5-7} ligand-gated channels. Although there is broad acceptance of synapses between neurons as likely locales for anesthetic action, there is also evidence that presynaptic elements\textsuperscript{8} and postsynaptic receptors are directly engaged in the integrated pharmacologic effects. In this issue of Anesthesiology, Hosain and Evers (page 1379) demonstrate that an important step in many pathways of signal transduction, including those obligated in the release of certain neurotransmitters, is modified by general anesthetics. They show that the release of calcium ions from intracellular storage compartments, a process regulated by the second messenger inositol trisphosphate (IP\textsubscript{3}),\textsuperscript{9} is accelerated by the anesthetic agents halothane and isoflurane and by octanol, at “clinically relevant” concentrations. Anesthetic-induced depletion of these calcium stores greatly reduces the large phasic rise of intracellular Ca\textsuperscript{2+} that is essential for coordinated neuronal signaling.\textsuperscript{10} Furthermore, the elevation of resting intracellular Ca\textsuperscript{2+} that follows the release probably is deleterious for many cytoplasmic enzymes that require a high Mg\textsuperscript{2+}/Ca\textsuperscript{2+} ratio to function.

Two noteworthy features of this observation are, first, that an additional site for general anesthetics is now identified on an organelle membrane, within the enveloping plasma membrane, and second, that this mechanism predicts a transient, stimulated phase of transmitter release followed by a persistent, inhibited release phase. Perhaps these neurochemical phases correspond to the sequence of excited and quiescent stages during anesthetic induction. It is also interesting to relate these actions, which, in neuroendocrine cells, may contribute to the therapeutic aspects of general anesthesia, to the analogous actions in skeletal muscle, which, in the case of genetically predisposed imbalances in Ca\textsuperscript{2+} transport, lead to the pathologic condition of malignant hyperthermia.\textsuperscript{11}

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References

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