Retrograde Intubation through a Laryngeal Mask Airway

To the Editor.—Previously described methods of replacing the laryngeal mask airway (LMA) with an endotracheal tube have relied on antegrade passage of a Cook airway exchange catheter or flexible guidewire, either blindly or with fiberoptic bronchoscope assistance. The potential exists for the unsecured distal portion of these devices to become dislodged when passed antegrade into the trachea, either during removal of the LMA or attempted passage of the endotracheal tube. Blind intubation through the LMA also may be complicated by esophageal intubation. Recently, we managed a difficult airway using a combined laryngeal mask airway and retrograde wire technique to facilitate tracheal intubation.

A 35-yr-old, 100-kg man with an uncomplicated medical history presented for exploratory laparotomy. On preoperative anesthetic evaluation of the airway only; one half of the uvula was visible, but the examination was otherwise normal. In the operating room after placement of the standard monitors, anesthesia was induced with fentanyl, thiopental, and succinylcholine. Laryngoscopy failed to permit visualization of the laryngeal structures despite using a variety of types and sizes of laryngoscope blades. A #5 LMA (Geesa) was easily inserted, and adequate ventilation was begun. A 6.0-mm endotracheal tube (ETT) was passed, in its entirety, through the LMA. Fiberoptic bronchoscopy (Olympus LF-1) revealed intralaryngeal placement of the tube cuff. Although the LMA adaptor was removed, the patient’s head and neck repositioned, and the volume of air adjusted in the LMA, intralaryngeal ETT cuff inflation remained. Withdrawal of the LMA over a blindly placed tube exchange catheter (Cook) intended to facilitate tracheal intubation resulted in dislodgement of the exchange catheter, necessitating replacement of the LMA. After sterile preparation, the cricothyroid membrane was entered with an 18-gauge introducer needle and a sterile 1-½” 0.38-inch (0.97-mm) diameter, 110-cm guidewire (Cook Critical Care retrograde guidewire kit, Cook) passed cephalad. Resistance was encountered at the level of the LMA aperture bar, but the wire easily passed with slight manipulation. A 11.0 French Teflon guide catheter (TFE) was threaded antegrade over the wire into the LMA until skin tenting was noted at the cricothyroid membrane and the LMA removed. An 8.0-mm ETT was passed over the TFE catheter until skin tenting was observed again. A fiberoptic elbow adapter (Portex) was attached to the ETT and connected to a capnograph, to verify placement. The TFE catheter and guidewire were removed, and ventilation was achieved through the endotracheal tube.

The possibility of intralaryngeal or supraglottic ETT cuff positioning after insertion of a standard ETT through the LMA was described previously. Suggested solutions to this problem have included the use of the extra-long Mallinckrodt microcuffed laryngeal tube (St. Louis, MO) or Mallinckrodt reinforced tracheal tube, deflation of the LMA cuff and advancement of the ETT and LMA together, insertion of a smaller diameter ETT into the cut end of the primary tube, cutting of the LMA shaft and advancement of the ETT, and use of the shortened version of the LMA, the ST-LMA (Intravent International SA, Henley-on-Thames, England). In the case described here, insertion of an extra-long ETT most likely would have prevented this situation from occurring, but, one was not readily available and we were forced to use retrograde intubation to attain satisfactory tracheal intubation. Retrograde intubation is invasive and not without limitations or complications, although one advantage may be that guidewire fixation at the cricothyroid membrane may decrease the likelihood of dislodgement of a tracheal tube passed through a laryngeal mask airway. This technique also might be modified to include the use of a fiberoptic bronchoscope passed antegrade over the guidewire. The combined technique of retrograde intubation via the laryngeal mask airway should be considered another alternative to achieve tracheal intubation through the laryngeal mask airway when conventional methods are unsuccessful.

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Cerebral Protection: Are all Barbiturates Created Equal?

To the Editor.—We enjoyed the recent article by Warner et al.1 They have had the audacity to challenge a verse of the paediatrics catechism that has been chanted faithfully for years. They are to be congratulated (or burned at the stake) for demonstrating that, in the setting of focal ischemia, a maximal cerebroprotective effect can be achieved with only one third of the dose of pentobarbital required to achieve electroencephalographic burst-suppression. Their observations refute the notion that a maximal cerebral protective effect requires a dose sufficient to achieve complete electroencephalographic suppression. But their observations lead to another heretical question.

It has been tacitly accepted, with respect to cerebral protection, that one barbiturate is equivalent to another. In the many studies of cerebral protection, several barbiturates (thiopental, methohexitol, pentobarbital) have been used, invariably, without any attempt to justify the choice on a pharmacodynamic basis. However, the data of Warner et al. should force us to reexamine that assumption of protective equivalence. That assumption seemed reasonable when it was accepted that cerebral metabolic rate suppression was the important protective mechanism because the barbiturates appeared very homogenous in their capacity to suppress cerebral metabolic rate. However, if, as suggested by Warner et al., we must begin to suspect some other pharmacologic effect, then is it reasonable to assume that all barbiturates share that effect (whatever it is) equally? Is it possible that one barbiturate is a more effective protector than others or that some barbiturates are no more protective than other general anesthetics?

These questions are highlighted by material in the very thorough discussion section of the current paper. Warner et al. candidly point out that the protective effect they observed (expressed as a percentage reduction in infarct volume) was smaller in their current investigation of pentobarbital than was the case in an earlier, though methodologically similar, study that included a group anesthetized with halothane.2 Both studies used an awake control state. Infarct volume was reduced by approximately 25% in the pentobarbital groups in the current study and by 40% in the investigation involving halothane. In an even earlier investigation by Warner and his colleagues, it was demonstrated that methohexitol provided greater protection (again expressed as percentage reduction in infarct volume) than did 1 MAC halothane anesthetic.3 From these investigations, one might be tempted to construct a “protective hierarchy” as follows: methohexitol > halothane > pentobarbital (another heresy)? On the basis of nonconcurrency alone, such a conclusion would be unreasonable. However, we think that, in the uncertainty, there is justification for a concurrent investigation of the effects of volatile agents and several barbiturates on reduction of infarct size. It no longer appears reasonable to assume that all barbiturates are equal in their cerebral protective potential or, for that matter, to rest assured that they are all superior to anesthesia with a volatile agent.

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