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A Method for Minimizing Leakage during Positive Pressure Ventilation after Intubation through Laryngeal Mask Airway

To the Editor:—When a laryngeal mask airway (LMA) is used as a conduit for tracheal intubation in children, the internal lumen of the LMA usually limits the size of the endotracheal tube (ETT) that can be used.[1] This often forces the anesthesiologist to place a tube that is smaller than might otherwise be used, with a large leak during positive pressure ventilation being the result. We hypothesized that inflating the LMA cuff and/or sealing the gap between the LMA and the ETT might substantially reduce this leak.

After obtaining Institutional Review Board approval and informed parental consent, 32 pediatric patients were studied. The patients were all < 8 yr of age, weighed 10–30 kg, and were undergoing procedures for which LMA use was appropriate. Anesthesia was induced with thiopental, and paralysis was achieved with vecuronium. An appropriate-sized uncuffed ETT was then inserted *via* direct laryngoscopy, and thereafter anesthesia was maintained with oxygen–air–isoflurane. The leak around this initially placed ETT was then assessed. If the leak occurred at a pressure < 20 cm H_2O, then the tube was exchanged for a larger uncuffed size; if the leak occurred at a pressure > 30 cm H_2O, it was exchanged for a smaller uncuffed tube. This process resulted in the empirical determination of the "appropriate-sized" ETT for each child.

When this sizing procedure was completed, the ETT was removed, and an appropriate-sized LMA was inserted in the conventional manner. The trachea was thereafter intubated under direct fiberoptic control, using the largest uncuffed ETT that could be advanced through the LMA (table 1). Patients were then classified into one of three groups: (1) group 1: the inserted ETT was 0.5 mm smaller than the tube size that had previously been determined as appropriate; (2) group 2: the ETT was 1.0 mm smaller than appropriate; (3) or group 3: the ETT was 1.5 mm smaller than the appropriate tube size.

The ETT was connected to a volume ventilator with tidal volume set at 10 ml/kg and and inspiratory-to-expiratory ratio of 1:2. A pneumotachometer flow transducer (Bicore Monitoring Systems, Irvine, CA) was placed between the patient's ETT adapter and the Y-connector and was used to measure expiratory tidal volume and peak inspiratory pressure. A series of four measurements were made, all at the same ventilator setting: the LMA cuff deflated or maximally inflated (based on manufacturers recommendations) and with the gap between the ETT and LMA sealed or not sealed. This seal was achieved by wrapping a transparent dressing (3M Company, St. Paul, MN) around the proximal end of the LMA at the connector. This dressing was manually rubbed to insure that the plastic adhered well to the ETT.

The Friedman test was used to compare expiratory tidal volume and peak inspiratory pressure among steps (α error was taken as 0.01), followed by multiple comparison. $P < 0.01$ was considered significant.

Results are shown in table 2. With the LMA cuff inflated and the LMA/ETT gap sealed with plastic, an expiratory tidal volume approximately equivalent to that achieved with an appropriately sized ETT was observed, even when the actual size of the ETT was as much as 1.5 mm smaller than "appropriate."

With the LMA cuff inflated and the gap sealed, we could not detect air entry into the stomach using a stethoscope, even when airway pressure was increased to 40 cm H_2O. No gastric insufflation was noted by either palpation or percussion, even after 1 h of positive pressure ventilation. However, we did not study longer procedures because of concerns about complications related to the maximally inflated LMA cuff.

The LMA cuff can play the same role as gauze packing in the oral cavity to cope with leakage encountered during the surgical procedure. The LMA must be positioned optimally, and leaks can be further

Table 1. Patient Characteristics

	Group 1 (n = 9)		Group 2 (n = 15)		Group 3 (n = 8)
Age (months)	53 ± 29 (17–94)		54 ± 25 (23–98)		69 ± 14 (43–84)
Weight (kg)	18.6 ± 6.3 (11–28)		19.2 ± 5.3 (12–29)		19.3 ± 1.2 (17–20)
	LMA Size				
	2 (n = 4)	2.5 (n = 5)	2 (n = 10)	2.5 (n = 5)	2 (n = 8)
The largest ETT size[2] (mm) inserted through LMA	ID 4.5 (n = 4)	ID 5.5 (n = 1) or 6.0 (n = 4)	ID 4.5 (n = 10)	ID 5.5 (n = 5)	ID 4.5 (n = 8)
Appropriate ETT size (mm) for the patients	ID 5.0 (n = 4)	ID 6.0 (n = 1) or 6.5 (n = 4)	ID 5.5 (n = 10)	ID 6.5 (n = 5)	ID 6.0 (n = 8)

Age and weight data given as mean ± SD (range).

LMA = laryngeal mask airway; ETT = endotracheal tube.

Table 2. Comparison of Tidal Volume and Peak Inspiratory Pressure When Sealing between Laryngeal Mask Airway and Endotracheal Tube and/or Laryngeal Mask Airway Cuff Inflation was Performed

		Largest ETT Inserted through an LMA			
		Without Sealing		With Sealing	
Group	Appropriate-sized ETT	Deflation [D]	Inflation [I]	Deflation [DS]	Inflation [IS]
Total (N = 32)					
Expiratory V_T (ml)	199 (47)	144 (45)*	146 (42)*	175 (46)*†	191 (46)
PIP (cm H_2O)	14 (2)	15 (2)‡	15 (2)‡	16 (2)‡§	16 (2)‡§
1 (n = 9)					
Expiratory V_T (ml)	199 (65)	183 (61)	180 (59)	188 (67)	190 (65)
PIP (cm H_2O)	14 (2)	15 (2)	15 (2)	15 (2)	15 (2)
2 (n = 15)					
Expiratory V_T (ml)	201 (50)	130 (34)*	133 (28)*	169 (43)*†	189 (46)
PIP (cm H_2O)	14 (2)	15 (3)	15 (2)	16 (2)	17 (2)
3 (n = 8)					
Expiratory V_T (ml)	195 (21)	133 (17)*	138 (28)*	173 (27)*†	196 (24)
PIP (cm H_2O)	14 (1)	15 (2)	15 (2)	16 (2)	16 (2)

Data are mean (SD). Each step is described in squared parenthesis. Statistics for PIP were obtained only for the total group.

V_T = tidal volume; PIP = peak inspiratory pressure; ETT = endotracheal tube; LMA = laryngeal mask airway.

* $P < 0.0001$ *versus* ETT and IS step.

† $P < 0.0001$ *versus* all other steps.

‡ $P < 0.0001$ *versus* ETT step.

§ $P < 0.0001$ *versus* ETT, D, and I step.

reduced by sealing the gap between the LMA and ETT. This method was successful and apparently safe, at least in these short surgical procedures.

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Neuron-specific Enolase as a Marker of Fatal Outcome in Patients with Severe Sepsis or Septic Shock

To the Editor:—The mortality rate of septic patients increases progressively with increasing severity of encephalopathy caused by sepsis.[1] Several mechanisms have been suggested as the cause of septic encephalopathy, including direct central nervous system infection, endotoxin and cytokine effects on the brain, inadequate cerebral perfusion, metabolic derangements, or complications of medical therapy.[2,3] Neuropathologic examinations of 12 septic patients who died during protracted, severe septic encephalopathy showed microabscesses in eight cases and vascular lesions in approximately half.[4]

We assessed the ability of the neuron-specific enolase (NSE), the neuronal isomer of the glycolytic enzyme 2-phospho-D-glycerate hydrolase, and the central nervous system–specific isoforms of S100 to predict mortality in patients with severe sepsis or septic shock. Both NSE and S100 have been shown to be specific parameters for the assessment of