References

1 *Tiso RL, Thomas PS, Macadaeg K.* Epidural catheter direction and local anesthetic dose. Reg Anesth 1993; 18: 308–11.
2 *Igarashi T, Hirabayashi Y, Shimizu R, Saitoh K, Fukuda H, Mitsuhata H.* The lumbar extradural structure changes with increasing age. Br J Anaesth 1997; 78: 149–52.
3 *Hirabayashi Y, Shimizu R, Matsuda I, Inoue E.* Effect of extradural compliance and resistance on spread of extradural analgesia. Br J Anaesth 1990; 65: 508–13.
4 *Gotou M, Yokoyama K.* Location of PERIFIX SOFT epidural catheter investigated by X-ray (Japanese). Masui 1993; 42: 922–5.
5 *Hendriks GW, Hasenbos MA, Gielen MJ, van Egmond J, Barentsz JO.* Evaluation of thoracic epidural catheter position and migration using radio-opaque catheters. Anaesthesia 1997; 52: 457–9.

Is there a need for the epiglottic bars in the laryngeal mask airway?

To the Editor:

The laryngeal mask airway (LMA-Classic) has been used widely in clinical practice including as a conduit for fibreoptic-aided intubation.[1] Fibreoptic- aided intubation is preferred to blind intubation due to the lower risk of soft tissue trauma and the ability to confirm correct positioning of the tracheal tube. Bars at the junction of the mask and the tube were designed to prevent the epiglottis from occluding the airway,[2] but may obstruct the passage of the tracheal tube.[3]

After approval of Ethics Committee, we evaluated the effect of the absence of the bars on the positioning of the LMA-Classic and its effect on the clinical management of the airway in adults. The manufacturer's instructions regarding the size of the LMA were followed. One hundred and sixty patients were divided into two groups; LMAs with and without bars. Anesthesia was induced using fentanyl and propofol and supplemented with oxygen, nitrous oxide and sevoflurane, with rocuronium when surgically indicated. The LMA was inserted one minute after completion of induction and following the loss of lash reflex and the relaxation of the jaw. The anesthesiologist who inserted the LMAs (B.A.-S.), blinded to the type of the LMA, used the standard technique of insertion[4] applying standardized clinical tests to evaluate the correct placement of the LMA.[5] Using a fibrescope with its tip located at the inner aperture of the LMA, another anesthesiologist (D.P.) who was not present during the placement of the LMA, assessed the position of the epiglottis using a standardized four-point scale.[5] The incidence of laryngeal spasm, both endoscopically and clinically, was documented. Statistical analysis was with Chi-squared test. LMAs with no bars were used in a further 300 patients undergoing gynecological and orthopedic procedures.

TABLE Endoscopic evaluation of the epiglottis: grade 4, only vocal cords seen; grade 3, vocal cords and posterior epiglottis seen; grade 2, vocal cords and anterior epiglottis seen; grade 1, vocal cords not seen and grade 0, failure to function where the vocal cords not seen. Grades 4 and 3 are correct. Grades 2, 1 and 0 are suboptimal.[5]

	Bars group n (%)	*No bars group n (%)*
Grade 4	37 (46)	40 (50)
Grade 3	19 (24)	17 (21)
Grade 2	14 (18)	16 (20)
Grade 1	10 (12)	7 (9)
Grade 0	0 (0)	0 (0)

No patient was excluded from the study. All 160 patients, in both groups, had a patent and clinically acceptable airway from the first attempt of the LMA insertion. There was no significant difference between the two groups in the incidence of correct and suboptimal positioning of the LMAs as shown in the Table. The LMAs were inserted and placed successfully from the first attempt in all 300 patients. None of the 460 patients had laryngeal spasm during the insertion of the LMA.

In conclusion, despite the absence of the epiglottic bars, we found no adverse effects on the airway management of the 380 patients we studied.

Baha Al-Shaikh FFARCSI
David Pilcher MRCP
Kent, UK

References

1 *Benumof JL.* Laryngeal mask airway and the ASA difficult airway algorithm. Anesthesiology 1996; 84: 686–99.
2 *Brain AI.* The development of the Laryngeal Mask – a brief history of the invention, early clinical studies and experimental work from which the Laryngeal Mask evolved. Eur J Anaesthesiol 1991; Suppl 4: 5–17.
3 *McNeillis NJ, Timberlake C, Avidan MS, Sarsang K, Choyce A, Radcliffe JJ.* Fibreoptic view through the

laryngeal mask and the intubating laryngeal mask. Eur J Anaesthesiol 2001; 18: 471–5.
4 *Brain AIJ*. The Intavent Laryngeal Mask Instruction Manual, 4th edition. Intavent; 1999.
5 *Joshi S, Sciacca RR, Solanki DR, Young WL, Mathru MM*. A prospective evaluation of clinical tests for placement of laryngeal mask airways. Anesthesiology 1998; 89: 1141–6.

Hyperbaric oxygen therapy (HBOT) in a child with suspected influenza-associated encephalopathy

To the Editor:

A three-year-old girl felt feverish, and the next day became comatose with convulsions. A computed tomography scan revealed diffuse cerebral edema. Encephalopathy associated with influenza (EI) was suspected. On day two she was transferred to the intensive care unit where she underwent hypothermia therapy under artificial ventilation. On day eight, influenza-A-positive antigen was identified in her nasal discharge. She recovered from her critical state and was extubated. However, her consciousness remained disturbed with no signs of recovery over the next six days. In Japan, hypoxic encephalopathy is one of the indications for HBOT. She had status epilepticus, which might also be suggestive of brain hypoxia. With this in mind, we decided that HBOT was a treatment option. After obtaining informed consent from her parents, HBOT (one session per day) was started on day 14. After the second HBOT session, she made eye contact and said one word. Six HBOT sessions were performed in total, and the patient was discharged home on day 34 with no sign of brain dysfunction. No factor other than HBOT readily explained the dramatic recovery, a situation analogous to that described in a previous study.[1] For some years, HBOT has been used for the treatment of coma due to post-anoxic encephalopathy,[2] and the active use of HBOT for global cerebral ischemia and coma has also been stressed. However, the benefit of HBOT for coma associated with EI remains unclear. EI is a severe condition which can result in serious brain damage and cause rapid death within a few days. Mortality is as high as 26.7–43.8%[3,4] and, even in survivors, the incidence of neurological sequelae is 20.3–25.8%.[3,4] Although ways of preventing EI-induced death and sequelae are urgently needed, reliable methods do not yet exist. Using hypothermia against EI is a possible approach and its effectiveness is currently being evaluated in Japan. Actually, HBOT is not considered an appropriate indication for patients with closed head injury (CHI).[5] However, since no standard therapy exists for EI-associated coma, and since EI is in some respects different from CHI, we suggest that HBOT may be of use to treat a coma that persists after initial therapy in the intensive care. Even delayed HBOT appears to be worthy of consideration.

Hiroshi Dohgomori MD PhD
Kazuhiro Arikawa MD PhD
Yuichi Kanmura MD PhD
Kagoshima, Japan

References

1 *Wherrett CG, Mehran RJ, Beaulieu MA*. Cerebral arterial gas embolism following diagnostic bronchoscopy: delayed treatment with hyperbaric oxygen. Can J Anesth 2002; 49: 96–9.
2 *Oriani G*. Acute indications of HBO therapy – final report. *In*: Oriani G, Marroni A, Wattel F (Eds.) Handbook on Hyperbaric Medicine, 1st ed. Milano: Springer-Verlag; 1996: 99–109.
3 *Togashi T, Matsuzono Y, Narita M*. Epidemiology of influenza-associated encephalitis-encephalopathy in Hokkaido, the northernmost island of Japan. Pediatr Int 2000; 42: 192–6.
4 *Kasai T, Togashi T, Morishima T*. Encephalopathy associated with influenza epidemics (Letter). Lancet 2000; 355: 1558–9.
5 *Rockswold GL, Ford SE, Anderson DC, Bergman TA, Sherman RE*. Results of a prospective randomized trial for treatment of severely brain-injured patients with hyperbaric oxygen. J Neurosurg 1992; 76: 929–34.