Editor—We are obliged to Dr Webb and colleagues whose comments support our article on percutaneous PFO closure before neurosurgery in the sitting position. We have nothing to add other than that the benefit of PFO closure is not limited to the day of neurosurgery. In fact, it persists for the rest of the patient's life and potentially protects against subsequent paradoxical stroke, myocardial infarction, or other systemic embolism. In addition, patients with PFO often suffer from migraine, independent of the problem requiring neurosurgery, and migraine symptoms may improve.

A.-R. Fathi
P. Eshtehardi
B. Meier*
Bern, Switzerland
*E-mail: bernhard.meier@insel.ch

- I Fathi AR, Eshtehardi P, Meier B. Patent foramen ovale and neurosurgery in sitting position: a systematic review. Br J Anaesth 2009; 102: 588-96
- 2 Calvert P, Klein A. Anaesthesia for percutaneous closure of atrial septal defects. Contin Educ Anaesth Crit Care Pain 2008; 8: 16-20
- 3 Duke DA, Lynch JJ, Harner SG, Faust RJ, Ebersold MJ. Venous air embolism in sitting and supine patients undergoing vestibular schwannoma resection. *Neurosurgery* 1998; 42: 1282-6
- 4 Orliaguet GA, Hanafi M, Meyer PG, et al. Is the sitting or the prone position best for surgery for posterior fossa tumours in children? Paediatr Anaesth 2001; 11: 541-7
- 5 Black S, Ockert DB, Oliver WC Jr, Cucchiara RF. Outcome following posterior fossa craniectomy in patients in the sitting or horizontal positions. *Anesthesiology* 1988; 69: 49–56

doi:10.1093/bja/aep185

Hyperbaric oxygen therapy for arterial gas embolism

Editor—The June issue of the British Journal of Anaesthesia contained three informative articles discussing various aspects of gas embolism. 1-3 In their report of paradoxical air embolus from a central venous catheter, Eichhorn and colleagues² incorrectly state that 'no specific treatment is available' for the management of arterial gas embolism (AGE). Hyperbaric oxygen therapy (HBOT) has been an established treatment for both diving-related and iatrogenic AGE for many years. Although there are no randomized controlled trials demonstrating the positive effect of HBOT, there are numerous case reports and case series and also sound mechanistic principles that support its use in AGE.^{4 5} The high oxygen tension promotes the resorption of nitrogen from the bubble and the elevated ambient pressure reduces the size of the bubbles in accordance with Boyle's law. At 282 kPa (a conventional HBOT treatment pressure), spherical gas bubble diameter will be reduced to 82% with a resulting 45% decrease in volume,

such that bubble passage through the microcirculation and resolution of embolic phenomena may occur. In their review of 27 case series, Moon and Gorman⁶ report substantial improvement in outcome in patients with AGE treated with HBOT. Three hundred and forty-six (78%) of the 441 who received HBOT fully recovered and 20 (4.5%) died. Of the 288 with no recompression therapy, 74 (26%) fully recovered and 151 (52%) died. We feel it is incumbent upon clinicians anaesthetizing patients at risk of AGE (such as those undergoing neurosurgery in the sitting position) to be aware of what, if any, services are available for the administration of HBOT should this complication occur. There are obvious limitations to HBOT, including the location of chambers (necessitating the transfer of critically ill patients) and the ability of those chambers to care for critically ill patients during the therapy. However, if facilities are available, HBOT should be considered immediately in cases of iatrogenic AGE as early treatment may improve outcome.⁴

M. E. Edsell*
J. Kirk-Bayley
London, UK

*E-mail: markedsell@doctors.org.uk

Editor-We thank Dr Edsell and Kirk-Bayley for their constructive comments on our manuscript. We do not doubt that an HBOT therapy may be useful in patients with acute AGE. The mechanism of reducing the size of the air bubbles by an elevated ambient pressure in a pressure chamber is clearly described. The resorption of nitrogen from the bubbles by high oxygen tension is also beneficial. These mechanisms are indubitably helpful in the case of an AGE. However, our hospital, which is close to the North and Baltic Seas, and therefore involved in the management of nautical accidents, is 100 km away from the nearest pressure chamber with 24 h availability. We still believe that for a sudden AGE, the time from the (often late) diagnosis to an HBOT treatment is unacceptably long and not reasonable. This is further stressed by avoidance of air transportation which makes the transport longer and more complicated as well. For the reasons mentioned above, we still believe that there is no reasonable specific treatment for an AGE in a typical clinical scenario where the AGE emergency is unexpected. For high-risk operations, like the sitting position in neurosurgery, it would be helpful to have the algorithm for AGE management including the possible decompression in a pressure chamber available.

V. Eichhorn*
A. Bender
D. A. Reuter
Hamburg, Germany
*E-mail: v.eichhorn@uke.de

- I Fathi A-R, Eshterhardi P, Meier B. Patent foramen ovale and neurosurgery in sitting position: a systematic review. Br J Anaesth 2009; 102: 588-96
- 2 Eichhorn V, Bender A, Reuter DA. Paradoxical air embolism from a central venous catheter. *Br J Anaesth* 2009; 102: 717–8
- 3 Fuentes-Garcia D, Hernandez-Palazon J. Intracoronary air embolism detected by transoesophageal echocardiography for aortic valve replacement. *Br J Anaesth* 2009; 102: 718–9
- 4 Blanc P, Boussuges A, Henriette K, Sainty JM, Deleflie M. latrogenic cerebral air embolism: the importance of an early hyperbaric oxygenation. *Intensive Care Med* 2002; **28**: 559–63
- 5 Trytko BE, Bennett MH. Arterial gas embolism: a review of cases at Prince of Wales Hospital, Sydney, 1996 to 2006. Anaesth Intensive Care 2008: 36: 60–4
- 6 Moon R, Gorman D. Treatment of the decompression disorders. In: Brubank A, Neuman T, eds. Physiology and Medicine of Diving (5th Edn). London: Saunders, 2003; 600-50

doi:10.1093/bja/aep186

Subanaesthetic sevoflurane by a helmet for bronchospasm after tracheal extubation

Editor—We would like to report a case of successful treatment of bronchospasm after tracheal extubation with sub-anaesthetic sevoflurane delivered by a helmet. A 59-yr-old female was undergoing elective laparoscopic adrenalectomy for a 7 mm tumour. The patient was taking pantoprazole for gastro-oesophageal reflux disease and had no

history of asthma or allergy to medication. After induction of general anaesthesia, the anaesthetist intubated the trachea (Cormack and Lehane Grade IIIA) at the third attempt and after the insertion of a Frova airway intubating introducer[®] (Cook Critical Care, Bjaeverskov, Denmark). Some clear liquid was sucked out from the mouth and the trachea, suggesting that pulmonary aspiration may have occurred and a 16 Salem gastric tube was introduced into the stomach. Anaesthesia was maintained with sevoflurane 1–1.4% and nitrous oxide 65% in oxygen. Fentanyl and vecuronium were administered i.v. to ensure analgesia and muscle relaxation. Ranitidine 100 mg and methylprednisolone 125 mg were given i.v. to reduce gastric secretion and bronchial hyperreactivity. No wheezing was audible during anaesthesia.

At the end of an uneventful surgical procedure, the trachea was extubated and the patient was transferred to the postoperative recovery room fully awake, free of pain, and haemodynamically stable, with adequate gas exchange and without wheezing audible in different sites of auscultation. Half an hour later, the patient developed restlessness, increasing breathing difficulties, bilateral wheezing, hypoxaemia, and hypercapnia (Fig. 1). The patient was given oxygen 10 litre min⁻¹ through a non-re-breathing mask, methylprednisolone 250 mg, and nebulized salbutamol 2.5 mg and ipratropium 500 μg. Thirty minutes later, the respiratory distress was worsening (Fig. 1). A helmet (CaStar, Starmed, Mirandola, Italy) was connected to a ventilator and placed on the patient. Non-invasive positive

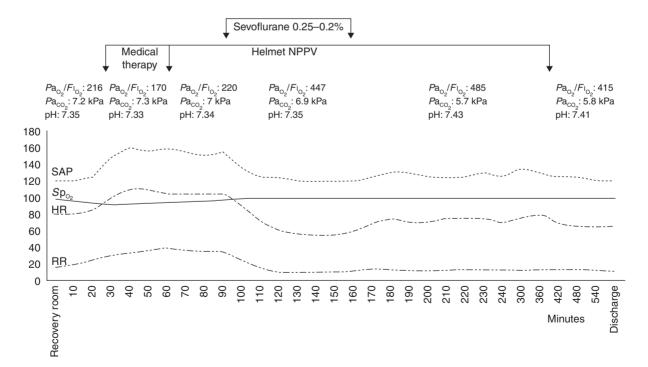


Fig 1 Systolic arterial pressure (mm Hg), heart rate (beats min⁻¹), ventilatory frequency (bpm), pulse oximetry (Sp_{o_2}). Medical therapy: oxygen via face mask, i.v. methylprednisolone, and inhaled ipratropium and salbutamol. Helmet NPPV, non-invasive pressure positive ventilation delivered by means of a helmet. Sevoflurane 0.25–0.2%, subanaesthetic concentration of sevoflurane delivered for the first 10 min at 0.25% and then at 0.2% during helmet NPPV.