Nitrous oxide anaesthesia and intraocular gases

Editor—I read with great interest the article by Lee,1 and like to thank the author for highlighting the important issue of nitrous oxide anaesthesia and intraocular gases. Long-acting gases, such as sulphur hexafluoride or perfluoropropane, have been used in the treatment of retinal detachment for over 30 years,2 and animal experiments with intraocular long-acting gas showed that the intraocular pressure could rise by 100% during nitrous oxide anaesthesia (75%) in an average of 24.1 min.3 This may lead to retinal artery occlusion, retinal ischaemia, and eventually visual loss. The first case of adverse effect of nitrous oxide anaesthesia on intraocular pressure in a patient with intraocular gas was reported in 1975.4 To my knowledge, the longest reported duration between a retinal operation with intraocular gas injection and subsequent visual loss from nitrous oxide anaesthesia was 6 weeks (42 days),5 which is a longer period than the one reported by Lee.

With such a long documented history of problem, it is unfortunate and worrying that the risk of visual loss from nitrous oxide anaesthesia in patients with intraocular gas is not better recognized. One of the reasons may be that the majority of the reports on this complication is published in ophthalmic journals. Our unit had previously addressed this complication and recommended such patients should carry cards giving details of possible complications of intraocular gas.6 The use of information cards or bracelets may be useful in patients who are not forthcoming with their history of recent intraocular surgery, or in the event of emergency anaesthesia. Lastly, vitrectomy for retinal detachment is not the only way to introduce long-acting gases into the eye. Pneumatic retinopexy for repair of retinal detachment involves injecting a small amount of long-acting gas into the vitreal cavity followed by laser treatment or cryotherapy.1 The gas bubble will enlarge slowly over a few days to partially fill the vitreal cavity. This is arguably a clinic/office based procedure, and patients who undergo this procedure may not feel they have had an ‘operation’, and therefore may not inform the anaesthetist before a subsequent anaesthetic.5 Unfortunately, the resultant expanding gas bubble poses the same danger to the eye in nitrous oxide anaesthesia as the intraocular gas after vitrectomy operation. It is important to enquire about all ocular procedures before the use of nitrous oxide anaesthesia in patients with intraocular gas.

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1 Lee EJ. Use of nitrous oxide causing severe visual loss 37 days after retinal surgery. Br J Anaesth 2004; 93: 464–6
Regional cerebral oximetry after oxygen administration

Editor—We read with interest the report of Stoneham and Martin showing that administration of oxygen 100% can antagonize the neurological deficits after carotid cross-clamping. This was attributed to an increase of the blood oxygen content that could be enough to increase the mitochondrial $P_{O_2}$ above the critical level so that oxidative phosphorylation in ischemic cerebral neurons could restart.

Near infrared spectroscopy has been recently used to monitor regional cerebral oximetry ($R_S_{O_2}$) during carotid endarterectomy. Cerebral spectroscopy, whilst having limitations in terms of absolute measurement, may give continuous noninvasive assessments of the cerebral oxygen supply–demand balance.

We investigated the effect of administration of oxygen 100% with a tight-fitting anaesthetic face mask in six awake patients who were scheduled for coronary artery bypass grafting. The mean (sd) $R_S_{O_2}$ in the awake patients during breathing room air was 62 (9.5)% which increased significantly to 67.8 (10.6)% after oxygenation. After subsequent induction of general anaesthesia using thiopental, fentanyl and rocuronium, there was a further significant increase of $R_S_{O_2}$ up to 80.2 (9.7)%. The 8% increase of $R_S_{O_2}$ after 100% oxygenation matches the increase of the total oxygen content as calculated by Stoneham and Martin. The subsequent induction of general anaesthesia resulted in a further increase in $R_S_{O_2}$ of about 20%. The combination of an increased oxygen delivery and general anaesthesia, which decreases the cerebral oxygen consumption, has an additive or even a synergistic effect that could have a significant impact on the neurons close to their ischaemic threshold.

Our results suggest that breathing oxygen 100% by the awake patients or induction of general anaesthesia using a high $F_{O_2}$, while monitoring the cerebral oxygen supply–demand by cerebral oximetry can enhance the cerebral oxygen supply–demand balance and may decrease the need for shunting in patients who are liable to develop neurological deficits during carotid endarterectomy.

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Ketamine for treatment of catheter-related bladder discomfort

Editor—We read with great interest the article by Dal and colleagues on the efficacy of prophylactic ketamine in preventing postoperative shivering. We have observed this technique as very effective and useful in the postoperative setting. We wish to report another incidental finding which we observed in two patients in whom we used ketamine 0.5 mg kg$^{-1}$ for treatment of postoperative shivering.

Two adult female patients who had undergone upper abdominal surgery, had shivering on arrival in the post-anesthesia care unit. Both patients had a urinary catheter that had been in place before commencement of surgery and complained of urinary catheter-related bladder discomfort. These patients were administered i.v. ketamine 0.5 mg kg$^{-1}$ as treatment for their shivering. Marked reduction in postoperative shivering was observed in both patients. To our surprise there was also marked reduction in the urinary catheter-related bladder discomfort in both these patients. We presume that ketamine might be effective in reducing catheter-related bladder discomfort by inhibition of the cholinergic system, which supplies to trigone of the urinary bladder. Further work is needed to evaluate this action of ketamine.

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