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.7 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 operation.8 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 as an anaesthetic agent. I hope with Lee’s latest report, we have drawn the attention of our anaesthetic colleagues, especially those in training, to the danger of nitrous oxide anaesthesia and 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

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Postoperative visual loss following prone spinal surgery

Editor—We read with interest the article by Kamming and Clarke1 on a case report of postoperative visual loss in a 60-yr-old man after prolonged decompressive laminectomy in the prone position. We completely agree with the discussion about aetiology of postoperative visual loss, but we would like to add some commentaries on retrobulbar circulation physiology. The authors assumed that the vessels involved in vascular supply of the posterior portion of the optic nerve (branches of the ophthalmic artery) are ‘incapable of autoregulatory control’. As we discussed in a recent article published in the British Journal of Anaesthesia,2 although the ocular circulation does not have any autonomic nerve supply, the ophthalmic and central retinal arteries have an autoregulation of their own blood flow.3,4 The central retinal artery is tightly auto-regulated by retinal endothelium derived factors as prostaglandin or endothelin.5 The ophthalmic artery may have a different autoregulatory mechanism and vasoactivity seems to be lower but the autoregulation in this artery is also efficient.6 Effects of anaesthetic agents (i.v. or inhaled) on retrobulbar circulation are not well known. We demonstrated in children that high alveolar concentrations of sevoflurane (2 MAC) may enhance alterations in ophthalmic artery blood flow.2 At 2 MAC sevoflurane, end diastolic velocity in the ophthalmic artery decreased to 70% of that at 1 MAC, whereas mean arterial pressure decreases only by 4%. In the case reported by Kamming and Clarke,7 anaesthesia was induced with midazolam, propofol and remifentanil, and maintained with isoflurane (0.9–1.2%) and remifentanil for more than 6 h. In their case, with only a minor anaesthesia-related decrease in systolic blood pressure, the oculovascular effect of inhaled anaesthetic such as isoflurane may have contributed to alterations in ophthalmic artery blood flow, even if a major decrease in perfusion pressure to the eye did not occur. In addition, isoflurane has a more important intrinsic cerebral vasodilatory effect compared with sevoflurane.7 The specific oculovascular effect of isoflurane is unknown, but we can hypothesize a similar or greater effect than sevoflurane. The discussion of this case report should consider the possibility of important vasodilatory effect of isoflurane in ophthalmic artery. If perfusion pressure decreases in an artery vasodilated by isoflurane, the ocular blood flow will be compromised, and optic nerve ischaemia may occur.

In conclusion, and in addition to this carefully discussed case report of postoperative visual loss, we could suggest for an