after intracameral administration. Data were analyzed using the paired *t*-test.

Outcomes
The mean systolic and diastolic blood pressures rose by 2.7 and 0.8 mm Hg post-I/C PE administration, respectively. Neither the systolic nor the diastolic blood pressure changes were statistically significant. The mean of the mean arterial pressures recorded a minor but statistically significant ($P = 0.015$) rise of 1.4 mm Hg, from 106.6 mm Hg to 108.0 mm Hg. We therefore concluded that I/C PE in the specified dose of 0.25 ml of 2.5% has negligible and clinically insignificant effects on blood pressure, and can be safely used during cataract surgery.

Comment
Intracameral administration of mydriatics provides several advantages compared with topical ones, including a more rapid and sustained effect, less glare and discomfort, and increased cost-effectiveness. This makes it useful not only as an alternative, but also as an adjunct for patients with poor topical mydriasis. One particular concern with topical instillation is the greater systemic absorption, which has been demonstrated to carry potential cardiovascular risk.[1] Hypertension is a comorbidity for half of patients undergoing cataract operations, and even if medically controlled these patients are already at risk of preoperative rises in blood pressure.[2]

Similar studies investigating the blood pressure effects of intracameral adrenaline found no intraoperative fluctuations in blood pressure as compared to or as adjuncts to topical mydriatics.[3,4] The only existing literature regarding complications of I/C PE indicates that its use is not associated with any statistically significant changes in surgical outcomes,[5] although the study in question did not directly measure the circulatory effects. Group sizes for these studies were limited, ranging from 25 to 50.

Conflict of interest

The authors declare no conflict of interest.

References

1 Lundberg B, Behndig A. Intracameral mydriatics in phacoemulsification cataract surgery. *J Cataract Refract Surg* 2003; **29**(12): 2366–2371.
2 Lira RP, Nascimento MA, Arieta CE, Duarte LE, Hirata FE, Nadruz W. Incidence of preoperative high blood pressure in cataract surgery among hypertensive and normotensive patients. *Indian J Ophthalmol* 2010; **58**(6): 493–495.
3 Liou SW, Yang CY. The effect of intracameral adrenaline infusion on pupil size, pulse rate, and blood pressure during phacoemulsification. *J Ocul Pharmacol Ther* 1998; **14**(4): 357–361.
4 Hempel S, Senn P, Pakdaman F, Schmid MK, Suppiger M, Schipper I. [Perioperative circulatory side effects of topical 5% phenylephrine for mydriasis]. *Klin Monbl Augenheilkd* 1999; **215**(5): 298–304.
5 Williams GS, Radwan M, Kadare S, Williams CP. The short to medium-term risks of intracameral phenylephrine. *Middle East Afr J Ophthalmol* 2012; **19**(4): 357–360.

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Sir,
Selective serotonin reuptake inhibitors and perioperative bleeding in endoscopic dacryocystorhinostomy

During a recent endoscopic dacryocystorhinostomy procedure on a patient taking selective serotonin reuptake inhibitors (SSRIs), we noticed engorgement of the nasal mucosa as well as severe early post-op epistaxis. We write to remind readers of the potential association between SSRIs and perioperative hemorrhage.

SSRIs (e.g. Citalopram, Fluoxetine, Fluvoxamine, and Sertraline) are commonly used to treat mood disorders such as depression, anxiety, and obsessive–compulsive disorders. Their popularity in the treatment of mood disorders stems from their side effect profile, which is better tolerated than the classic treatments (monoamine oxidase inhibitors, tricyclic antidepressants, etc).

One of the side effects of SSRIs of particular interest to us is the increased risk of bleeding perioperatively. It has already been documented that SSRIs increase the risk of gastrointestinal bleeding[1] and intracranial hemorrhage.[2] de Abajo[3] summarizes the mechanism by which the SSRIs potentiate bleeding. Platelets cannot synthesize serotonin; rather, serotonin is stored in platelets and released by certain stimuli to induce vasoconstriction and platelet activation, and to enhance fibrin formation. This important neurotransmitter also helps in generating coated platelets, a subgroup of platelets with important procoagulant activity. As SSRIs inhibit the serotonin transporter, which is responsible for the uptake of serotonin into platelets, it could be predicted that SSRIs would deplete platelet serotonin, leading to a reduced ability to form clots and a subsequent increase in the risk of bleeding.

Although some clinical practice references suggest holding SSRIs for 2 or more weeks before surgery, it is difficult to frame a detailed strategy based on the available evidence.[4,5] Discontinuing SSRIs could lead to discontinuation syndrome, increased sensitivity to pain, and relapsing depression postoperatively. Furthermore, although the morbidity may be greater in patients under SSRIs, the mortality is still quite low.[5] Consultation with a psychiatrist is recommended when there is high risk of morbidity from perioperative bleeding.

We write to remind readers to consider this potential association between the use of SSRIs and increased risk of hemorrhage, especially during the consent process for lacrimal surgery.

Conflict of interest

The authors declare no conflict of interest.

References

1 Yamamoto T, Abe K, Kuyama Y. Selective serotonin reuptake inhibitor and gastrointestinal hemorrhage. *Nihon Rinsho* 2013; **71**(4): 751–756.
2 Hackam DG, Mrkobrada M. Selective serotonin reuptake inhibitors and brain hemorrhage: a meta-analysis. *Neurology* 2012; **79**(18): 1862–1865.
3 de Abajo FJ. Effects of selective serotonin reuptake inhibitors on platelet function: mechanisms, clinical outcomes and implications for use in elderly patients. *Drugs Aging* 2011; **28**(5): 345–367.
4 Muluk V, Macpherson DS. Perioperative medication management. In Basow D ed. *UpToDate*. UpToDate: Waltham, MA, 2013. http://www.uptodate.com/contents/perioperative-medication-management (accessed 11 April 2014).
5 Auerbach AD, Vittinghoff E, Maselli J, Pekow PS, Young JQ, Lindenauer PK. Perioperative use of selective serotonin reuptake inhibitors and risks for adverse outcomes of surgery. *JAMA Intern Med* 2013; **173**(12): 1075–1081.

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Sir,
Concerning central retinal artery occlusion (CRAO) and cerebral stroke

I read with interest the correspondence on central retinal artery occlusion (CRAO) and cerebral stroke regarding Varma *et al*'s article 'A review of central retinal artery occlusion: clinical presentation and management.'[1]
In a paper published in this journal in 1992 we looked at the relationship between central retinal artery and ocular neovascularisation in eight patients.[2]

We agree with McLeod[3] that penumbral retinal tissue viability can persist as half of our patients did not fall into the carotid occlusive disease group. This concurs with Kottow and Hendrickson,[4] who found anterior segment neovascularisation following CRAO, as well as Brown,[5] who reported a case of neovascularisation following CRAO due to presumed single mitral valve embolus in the absence of carotid disease.

Varma *et al* also refer to preventing ocular neovascularisation following CRAO. The relationship between these two events is more complex than this paper implies, as we described in a table which I reproduce here for the sake of clarity (Table 1). As well as considering CRAO alone, it considers the concept of double embolism as reported by Wolter[6] as well as CRAO in a setting of chronic ocular ischaemia due to carotid occlusive disease.[7,8]

We do, however, strongly agree with Varma *et al* that such patients should be reviewed at regular intervals for at least 3 months following the diagnosis of CRAO.

Table 1 A classification of the proposed mechanisms relating central retinal artery occlusion (CRAO) to ocular neovascularisation (NVN)

1. Chronic ocular ischaemia alone leading to ocular NVN	→Rubeotic glaucoma[a]
↓	
Low CRA perfusion pressure	→CRAO
2. Chronic ocular ischaemia + coincidental CRAO	→Ocular NVN
3. Double embolism: CRAO + PCAO	→Ocular NVN
4. CRAO alone	→Ocular NVN

Abbreviation: PCAO, posterior ciliary artery occlusion.
[a]Rubeotic glaucoma is usually characterised by a low intraocular pressure.

Conflict of interest

The author declares no conflict of interest.

References

1 Varma DD, Cugati S, Lee AW, Chen CS. A review of central retinal artery occlusion: clinical presentation and management. *Eye* 2013; **27**: 688–697.
2 Jacobs NA, Trew DR. Occlusion of the central retinal artery and ocular neovascularization: an indirect association? *Eye* 1992; **6**: 599–602.
3 McLeod D. Letter to the editor: partial central retinal artery occlusion offers a unique insight into the ischemic penumbra. *Clin Ophthalmol* 2012; **6**: 9–22.
4 Kottow M, Hendrickson P. Iris angiographic findings in retinal arterial occlusions. *Can J Ophthalmol* 1974; **9**: 435–443.
5 Brown GC. Isolated central retinal artery obstruction in association with ocular neovascularization. *Am J Ophthalmol* 1983; **96**: 10–11.
6 Wolter JR. Double embolism of the central retinal artery followed by secondary haemorrhagic glaucoma. *Am J Ophthalmol* 1972; **73**: 652–657.
7 Sturrock GD, Mueller HR. Chronic ocular ischaemia. *Br J Ophthalmol* 1984; **68**: 716–723.
8 Jacobs NA, Ridgway AEA. Syndrome of ischaemic ocular inflammation; six cases and review. *Br J Ophthalmol* 1985; **69**: 681–687.