Sir,
Reply to Gurbaxani and Packard

We read with interest the paper by Gurbaxani and Packard¹ on the use of intra-cameral phenylephrine to prevent floppy iris syndrome during cataract surgery. The authors acknowledge that there are limitations to the study but still claim that 'all patients had a significant reduction in the signs of floppy iris syndrome', whereas what they have demonstrated is that intra-cameral phenylephrine dilates the pupil. Whether this is in addition to the effect of pre-operative topical application of phenylephrine drops is not recorded.

The intra-operative floppy iris syndrome (IFIS) describes a spectrum of signs seen intraoperatively in patients on alpha-1a antagonists.² This can range from a small pupil with rather poor iris tone to the full IFIS where the iris is billowing and prolapsing into and through all corneal incisions. There is no current evidence that IFIS will occur in everyone on tamsulosin, and a small pupil can be associated with other common conditions such as diabetes. IFIS has also been encountered in patients with no history of alpha-1a antagonist use (personal communication). It does not follow that a patient on tamsulosin with a small pupil is a certain candidate to develop IFIS, and therefore no treatment or manoeuvre can be confidently stated to prevent IFIS.

We would agree, however, that intra-cameral phenylephrine is a useful option in the management of IFIS, and thank the authors for bringing this technique to a wider audience. It should be considered as one of an array of available options for use sooner rather than later, if the surgeon is suspicious that IFIS may develop.

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Sir,
Response to Nguyen et al

We thank Dan Nguyen and co-authors for their comments. As rightly pointed out, our paper intends to bring to our colleagues this simple but very effective tool for combating IFIS.

The pupillary dilatation achieved with intracameral phenylephrine is in addition to that achieved with the preoperative 2.5% topical phenylephrine drops used. In addition to increasing pupillary dilatation, intracameral phenylephrine increases the tone of the iris, thus reducing the 'floppiness' of the iris that significantly contributes to the effectiveness of this technique.

We do not set out to identify the incidence of IFIS in our paper but various papers quote this to be between 43%¹ and 65%² in patients taking Tamsulosin. The range of signs also varies from patient to patient.

Ophthalmic surgeons need to know if their patients are taking Tamsulosin so that they can anticipate and prevent complications due to IFIS. Our technique is part of a growing body of methods to deal with IFIS.

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Sir,
Crystalline retinopathy associated with chronic macular hole; a report of two cases

Crystalline retinopathy has been associated with a variety of toxic, metabolic hereditary conditions, chronic retinal detachment (RD), and retinal dialysis.^{1–4}

The origin of the crystalline opacities in patients with RD has been speculative and controversial.^{1,3,4}

We describe two cases of crystalline retinopathy in patients with no history of systemic illness or drug use, with chronic full thickness macular holes (FTMHs).

Case 1

A 73-year-old woman complaining for blurred vision in her left eye (LE). Best-corrected visual acuity (BCVA) was 20/200 LE. Anterior segments examination and funduscopy of the right eye (RE) were unremarkable. Funduscopy of LE revealed a stage-5 FTMH and multiple superficial refractile crystals within the vascular arcades at the level of the inner retina (Figure 1). Conservative management was undertaken and 8 months later, the distribution and appearance of crystals remained unchanged.

Case 2

A 71-year-old Afro-Caribbean woman presented with decreased vision LE; BCVA was 20/200. Anterior segments examination was unremarkable. Fundoscopy in the LE disclosed a stage-5 FTMH. Few superficial refractile crystals were visible within the vascular arcades situated at the level of the inner retina (Figure 2). The patient decided against any surgical intervention and 11 months later, the distribution and appearance of crystals remained unchanged.

Comment

Few previous reports have described the association of inner retina refractile opacities with long-standing RDs.^{1,2,5} Ahmed *et al*¹ and Ciardella *et al*² postulated that the crystalline deposits were granules of haemosiderin-laden macrophages adjacent to the internal limiting membrane. Banaee³ had postulated that photoreceptor outer segment particles can be released into the vitreous cavity and this may explain the

deposition of crystals in the cases presented by Ahmed *et al*¹ and Ciardella *et al*.³

On the basis that our patients and the majority of those reported in previous studies^{1,2,4} did not have a history of vitreous haemorrhage, it seems unlikely that the crystals deposition can be attributed to blood-break-down products.

A similar mechanism to that described by Banaee could explain the appearance of crystals in our patients with FTMHs.

Our cases illustrate the rare association of chronic FTMH with the deposition of small refractile crystals on the superficial retina which to the best of our knowledge has never been reported before. The association of crystals with FTMHs strengthens the view that crystal deposits in noninherited, metabolic, or toxic crystalline retinopathy may be attributed to photoreceptor degeneration rather than degenerative by-products of erythrocytes.

Figure 1 Patient 1-LE. Fundus photography showing a FTMH and crystalline deposits in the posterior pole.

Figure 2 Patient 2-LE. Fundus photography depicting small crystalline deposits close to a stage 5 FTMH.

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Sir,
Estimating the potential impact of using intravitreal angiostatic agents for wet macular degeneration on service capacity in a large retinal referral centre
We performed a retrospective review of the angiographic subtype of patients who attended their first fluorescein angiography within a 3-month period in our department, which is a photodynamic therapy (PDT)-providing unit serving a 1.8 million population.

We identified 42 eyes with classic or predominantly classic choroidal neovascularisation (CNV), 40 eyes with minimally classic CNV or occult CNV, and nine eyes with