Table 1 Comparing shield and shield-less cohorts

	Shieldless		*Shield*		P-*value*
Mean age (years)	72.8	±7.7	73.4	±7.2	
Total patients	425	30.2%	982	69.8%	
Scleral tunnel	298	70.1%	668	68.0%	
Clear corneal	127	29.9%	314	32.0%	
Uveitis	6	1.4%	19	1.9%	0.661
Corneal oedema	5	1.2%	9	0.9%	0.770
IOP >21 mm Hg	5	1.2%	8	0.8%	0.548
Iris prolapse	1	0.2%	1	0.1%	0.513
Endophthalmitis	0	0.0%	1	0.1%	1.000
Macular oedema	3	0.7%	15	1.5%	0.302

Abbreviation: IOP, intraocular pressure.

($n=1407$). One surgeon used no shields throughout this period (regardless of patient factors; $n=425$). All other cases ($n=982$) wore a Cartella shield overnight for three weeks. Both groups contained similar demographics and wound construction (Table 1). The shield-less regime conferred no safety disadvantage. All adverse events had nonsignificant *P*-values with Fisher's exact test (Table 1).

A total of 46 patients responded by anonymous questionnaire; 59% stating shields were 'uncomfortable' and 43% would have 'preferred to not wear' one. Comments included 'If it helps I will wear it' and 'I assume I was given it for a reason'. With the recent advances in wound construction, surgical outcomes and complication rates is the routine use of shields without evidence still necessary in 2011?

Conflict of interest
The authors declare no conflict of interest.

References

1 El-Hindy N, Johnston RL, Jaycock P, Eke T, Braga AJ, Tole DM *et al*. The cataract national dataset electronic multi-centre audit of 55,567 operations: anaesthetic techniques and complications. *Eye* 2009; **23**(1): 50–55.
2 Perkins RS, Olson RJ. A new look at postoperative instructions following cataract extraction. *Ophthalmic Surg* 1991; **22**(2): 66–68.
3 Mayer S, Wirbelauer C, Haberle H, Altmeyer M, Pham DT. Evaluation of eye patching after cataract surgery in topical anaesthesia. *Klin Monbl Augenheilkd* 2005; **222**(1): 41–45.

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Sir,

Response to: Idiopathic uveal effusion syndrome causing unilateral acute angle closure in a pseudophakic patient

We read with interest the case report of presumed idiopathic uveal effusion syndrome (IUES) associated with unilateral acute angle closure (AAC) in a pseudophakic patient.[1]

The authors propose that the case occurred in the absence of pupil block, however, the anterior segment OCT image presented shows iris convexity implying pupil block. We note that no posterior synechiae were seen clinically, however, the B-scan ultrasound images suggest adhesions between the posterior iris and the anterior capsule, consistent with seclusio pupillae. Pseudophakic pupil block with synechiae not visible at the pupillary margin can occur.[2] Furthermore, the case resolved with pupil dilation and medical intraocular pressure control supporting a pseudophakic pupil block mechanism.

The association of uveal effusion with AAC is well recognized and has been reported to occur in up to 58% cases of acute primary angle closure.[3] As stated by the authors, IUES is a diagnosis of exclusion; and is typically associated with serous retinal detachment.[4] No serous retinal detachment is seen in the case presented.

Their case is certainly unusual with respect to the fact that AAC occurred with an IOL placed in the capsular bag with presumed correct orientation. We would suggest the authors consider prophylactic peripheral laser iridotomy in their case to reduce the risk of a repeat AAC episode.

Conflict of interest
The authors declare no conflict of interest.

References

1 Bhogal M, Mitry D, Restori M, Subak-Sharpe I. Idiopathic uveal effusion syndrome causing unilateral acute angle closure in a pseudophakic patient. *Eye* 2011; **25**: 1236–1238.
2 Naveh N, Wysenbeek Y, Solomon A, Melamed S, Blumenthal M. Anterior capsule adherence to iris leading to pseudophakic pupillary block. *Ophthalmic Surg* 1991; **22**(6): 350–352.
3 Sakai H, Morine-Shinjyo S, Shinzato M, Nakamura Y, Sakai M, Sawaguchi S. Uveal effusion in primary angle-closure glaucoma. *Ophthalmology* 2005; **112**(3): 413–419.
4 Elagouz M, Stanescu-Segall D, Jackson TL. Uveal effusion syndrome. *Surv Ophthalmol* 2010; **55**(2): 134–145.

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Sir,

Response to Day and Foster

We value the interest Day and Foster[1] have expressed in our case.[2] The reported cases of seclusio pupillae in

pseudophakia are accompanied by clear, clinical descriptions of an immobile iris that is tethered to the anterior capsule where the rhexis edge lies, typically in the mid periphery.[3–5] In the largest case series, Gaton *et al* describe 'iris bombé and a shallow anterior chamber with a fixed, non-reacting pupil and increased IOP (40 and 60 mm Hg)'. In our case, the pupil was mobile, and ~2–3 mm smaller than the adequately sized anterior rhexis. The iris did not have the typical bombé appearance and pupil dilated uniformly without any signs of pigmentation on the anterior capsule suggestive of prior iridocapsular adhesion.

Choroidal effusions are increasingly being implicated in primary angle closure. It remains unclear if they are a causative factor or consequence. We could find no reports where choroidal effusions were seen in the context of seclusio pupillae, and the findings in phakic primary angle closure may not be directly transferrable to our case. In the paper by Sakai *et al*,[6] inclusion into the acute primary angle closure (APAC) group required bilateral narrow angles, IOP >40 mm Hg, nausea/vomiting, and corneal oedema in phakic patients. This is quite different from the case we present. Of their APAC patients, only 2 of 70 had grade 3 effusions and they make no mention of these extending beyond the equator. Our patient had large choroidal detachments, with folds visible at the posterior pole through an undilated pupil.

Utilizing the facilities available to us, we performed dynamic ultrasonography and were happy that the iris was fully mobile. High-resolution ultrasound biomicroscopy would have been helpful in confirming the pathology, however, this modality was unavailable at our institution.

Peripheral iridotomy is not without risk. It is only of value in cases of pupil block and as such would be ineffective in this case.

We were fortunate enough to be able to document what we recognized as an unusual case and have included a video highlighting some of the salient features that we believe support our interpretation (Supplementary Video).

Conflict of interest
The authors declare no conflict of interest.

References

1 Day AC, Foster PJ. Response to: Idiopathic uveal effusion syndrome causing unilateral acute angle closure in a pseudophakic patient. *Eye* 2011; **25**(12): 1660.
2 Bhogal M, Mitry D, Restori M, Subak-Sharpe I. Idiopathic uveal effusion syndrome causing unilateral acute angle closure in a pseudophakic patient. *Eye* 2011; **25**: 1236–1238.
3 Gaton DD, Mimouni K, Lusky M, Ehrlich R, Weinberger D. Pupillary block following posterior chamber intraocular lens implantation in adults. *Br J Ophthalmol* 2003; **87**(9): 1109–1111.
4 Sathish S, MacKinnon JR, Atta HR. Role of ultrasound biomicroscopy in managing pseudophakic pupillary block glaucoma. *J Cataract Refract Surg* 2000; **26**(12): 1836–1838.
5 Naveh N, Wysenbeek Y, Solomon A, Melamed S, Blumenthal M. Anterior capsule adherence to iris leading to pseudophakic pupillary block. *Ophthalmic Surg* 1991; **22**(6): 350–352.
6 Sakai H, Morine-Shinjyo S, Shinzato M, Nakamura Y, Sakai M, Sawaguchi S. Uveal effusion in primary angle-closure glaucoma. *Ophthalmology* 2005; **112**(3): 413–419.

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Sir,

Spontaneous resolution of early postoperative intraocular lens opacification in a patient with uveitis

Opacification of intraocular lenses (IOLs) is a complication of cataract surgery that may result in IOL explantation.[1,2] We describe a patient with uveitis who developed early postoperative IOL opacification, which subsequently resolved spontaneously.

Case Report

A 50-year-old Chinese female with quiescent idiopathic intermediate uveitis underwent left uneventful 1. 8 mm clear cornea phacoemulsification with in-the-bag implantation of a hydrophilic acrylic IOL (Akreos MI60, Bausch and Lomb, Rochester, NY, USA) in 2007. Posterior synechiae and pupillary membrane were absent. She received prophylactic intracameral dexamethasone 0.4 mg/0.1 ml at the end of surgery.

During postsurgery visits at 1 day and 1 week, there was 1+ anterior chamber (AC) cells, no flare, and the IOL was clear. Her unaided visual acuity (UAVA) was 6/9.

A haze was seen on the anterior surface of the IOL, within the capsulorhexis opening but distinct from the plane of the anterior capsule margin, at 1 month postsurgery (Figures 1a and b). There was 1+ AC and anterior vitreous cells. She was asymptomatic with 6/9 UAVA.

The central optic haze cleared by 2 months (Figure 1c). At six months, the IOL had completely cleared (Figure 1d) and has since remained clear despite further uveitis recurrences, resulting in 6/7.5 UAVA.

Comment

The IOL opacification seen in our patient differs in its appearance and clinical course from other causes including calcification,[2] whitening, glistening,[3] and lens epithelial cell (LEC) outgrowth.[4]

Calcium phosphates deposits, described in older hydrophilic IOLs, usually appear more than 1 year postoperatively.[2] LEC outgrowth onto the anterior IOL surface starts early postoperatively.[4] It is thought that hydrophilic IOLs with higher water content may promote LEC migration.[5] Calcifications