One of the interesting observations noted in the subgroup of patients with preoperative moderate/marked inferior oblique overaction and binocular single vision, was the observed trend in postoperative inferior oblique muscle overaction in the inferior oblique recession and inferior oblique myectomy groups between 2 months and 12 months postoperatively.[1] There was a greater likelihood of a recurrence of some inferior oblique muscle overaction in patients who underwent an inferior oblique muscle recession. Should this trend continue, then this could lead to not only a clinical but a functionally different long-term outcome between these two procedures in this subgroup of patients with overacting inferior obliques. The anatomical differences between the described myectomy and recession procedures may well be one explanation for this observational difference.

We fully agree with Shankar and Thompson that a difference of 1.25 prism dioptres, while statistically significant, is not likely to be clinically significant in this group of patients who are expected to have normal/supranormal vertical fusion ranges.

The statistically significant differences between the two groups, as a whole, is very much more likely to be genuine rather than attributable to small changes in head positioning as all the measurements in the three gaze positions for the recession and myectomy patients were carried out under the same clinical conditions by the same experienced orthoptist. None of the patients in either group were unhappy postoperatively.

While Table 2 indicated that a single case had a measurable but functionally asymptomatic contralateral inferior oblique muscle underaction, Table 3 reflected the changes that occurred in the hyperdeviation after the immediate postoperative period, namely from 2 weeks to 12 months postoperatively. This table demonstrated the variability in the range of primary position and contralateral gaze measurements in the myectomy and recession groups. These data do not support the view that recessions are more predictable. Accordingly, we disagree with Shanker and Thompson: our conclusions have not been overstated.

## References

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Sir,

**Acute glaucoma in the unoperated eye after macular hole surgery**

We read with interest the article by Bansal *et al*[1], and would like to report another case of angle closure glaucoma following pars plana vitreous surgery. This, however, occurred in the unoperated eye of a 68-year-old man following macular hole surgery.

### *Case report*

The patient was initially referred 8 years earlier with a persisting inferotemporal retinal detachment in his right eye, following an unsuccessful buckling with cryotherapy a month earlier. A vitrectomy with gas and oversewing of the buckle was performed. The retina was attached but within 2 months developed an epiretinal membrane that was peeled. His vision improved from 6/60 to 6/12. At 3 years following this procedure, he was discovered to have raised intraocular pressure (IOP) in his right eye, with a narrow but open angle. His left IOP was normal and his angle was slightly narrow. He was commenced on a topical beta -blocker to his right eye. After 4 years, he developed a symptomatic cataract in the right eye and underwent phacoemulsification with an intraocular lens implantation. Over the next few months, he developed right metamorphopsia and his vision fell from 6/9 to 6/36 and was found to have developed a macular hole.

Therefore, a right internal laminar membrane peel was performed. An internal search revealed a retinal break at 12 o'clock that was lasered. An air/16% C3F8 exchange was performed. The postoperative instruction was to lie face down.

On the first postoperative day, the patient was found to have an IOP of 45 mmHg in his right eye with an attached retina. Systemic acetazolamide and topical beta blockers and alpha agonists were given and the IOP fell to 38 mmHg. The patient, however, complained of feeling unwell and of an ache over his forehead, which he blamed on pressure on his forehead from posturing.

Further topical therapy was given and the right IOP fell to 32 mmHg, but the patient still complained of feeling unwell. The IOP in his left eye was checked and found to be 52 mmHg with an associated shallow anterior chamber. A diagnosis of acute angle closure glaucoma was made. Further systemic and topical treatment was given and the respective IOPs fell to 31 and 21 mmHg in the right and left eyes with associated relief of the symptoms. A left YAG peripheral iridotomy was subsequently performed.

**Comment**

The most likely cause of the acute glaucoma in the unoperated eye was the prolonged posturing in the face down position. Indeed, one provocative test for glaucoma is to place patients in the prone position.[2] The mechanism for this is the shifting of the lens-iris diaphragm anteriorly. This shallows the anterior chamber and narrows the angle. In our patient, the problem was compounded by the dilatation of the eye. Although it is unlikely that the episode of angle closure was solely precipitated by dilatation as both eyes had been dilated previously at vitreo-retinal clinic without incident. Also, gonioscopy had found a slightly narrow angle and the axial length was not particularly short. Biometry prior to the cataract surgery found axial lengths of 24 and 23.7 mm in the right and left eyes, respectively. Furthermore, it is unlikely that there was a phacomorphic component as there was no significant cataract in the left eye.

Raised IOPs can, therefore, be found in both the operated and unoperated eyes following pars plana vitreous surgery. Indeed for the unoperated eye this is not surprising, as vitreoretinal surgery often requires prone posturing and dilatation, both of which may precipitate angle closure glaucoma in those at risk.

**References**

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Sir,

**A case of postoperative candida endophthalmitis**

Candida endophthalmitis is a potentially devastating complication of cataract surgery.[1] It is an important opportunistic infection of intravenous (iv) drug abusers and debilitated patients[2] and it is the most common cause of endogenous endophthalmitis.[3] Generally, visual outcome from this disease is poor and candida endophthalmitis requires aggressive management. Therapy is controversial regarding whether surgery is required, choice of antifungal agent, administration route, frequency and what individual and total dosage can be given. We present a case of candida endophthalmitis in which clinical deterioration occurred despite intravitreal and iv amphotericin B, vitrectomy, and lens implant extraction. Subsequent improvement was noted after repeated multiple intracameral injection of amphotericin B.

***Case report***

An 85-year-old well-controlled noninsulin-dependent diabetic male on latanoprost 0. 005% nocte for open-angle glaucoma underwent uneventful left phacoemulsification with lens implant. A visual acuity of 6/6 was achieved. Vision remained at count fingers in the right eye secondary to glaucomatous damage. A left low grade, painless anterior uveitis subsequently developed and a latanoprost-induced uveitis was considered as the underlying cause. The latanoprost was withdrawn and the uveitis treated with topical dexamethasone hourly and atropine 1% bd. Despite treatment vision gradually dropped to 2/60 and a fibrinous anterior uveitis with a hypopyon developed. Anterior chamber aspiration was preformed and tissue plasminogen 25 $\mu$g/0.1 ml given intracamerally in an attempt to disintegrate the hypopyon. Negative Gram stain and culture of aqueous fluid was reported. Visual acuity improved to 6/24 but