and cautioned that a larger series is needed before one can comment on the true incidence of ocular motility dysfunction following this complication. Therefore, it may be misleading to conclude that 'the migrating encircling band is not usually linked to ocular motility disturbance'. We suggest that it is important to consider migration of encircling elements in the differential diagnosis of diplopia in patients with previous scleral buckling procedures to ensure that important clinical signs are not overlooked in examination.

References

- 1 Kreis AJ, Klainguti G, Bovey EH, Wolfensberger TJ. Transmuscular migration of 240 silicone encircling band. *Eye* 2006; **20**: 1456–1458.
- 2 Seaber JH, Buckley EG. Strabismus after retinal detachment surgery: etiology, diagnosis, and treatment. *Semin Ophthalmol* 1995; **10**: 61–73.
- 3 Macleod JDA, Morris RJ. Detached superior rectus following scleral buckling: anatomy and surgical management. *Eye* 1997; **11**: 30–32.
- 4 Maguire AM, Zarbin MA, Elliott D. Migration of solid silicone encircling element through four rectus muscles. *Ophthalmic Surg* 1993; **24**: 604–607.
- 5 Lanigan LP, Wilson-Holt N, Gregor ZJ. Migrating scleral explants. *Eye* 1992; **6**: 317–321.

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Sir,
Eccentric macular hole formation associated with macular hole surgery

We read with interest the article by Polkinghorne and Roufail¹ on eccentric macular hole formation associated with macular hole surgery. From their series of four eyes, they described that the 'risk appears higher in eyes operated on by vitreoretinal fellows' and christened it the 'fellow eye syndrome,' which is a clever wordplay that hopefully is not derogatory or uncharitable. However, Rubinstein *et al*² in their seminal report of four patients stated that 'one experienced vitreoretinal surgeon performed all the operations (RB)'. Likewise in our experience with two patients, the development of eccentric macular hole did not occur in the hands of fellows, but in the hands of a more experienced vitreoretina fellowship-trained surgeon (KGAE). Incidentally, one of our two patients was also a myope with associated myopic chorioretinal degeneration who developed multiple eccentric macular holes after undergoing standard pars plana vitrectomy and internal limiting membrane peeling for macular hole.

Although we agree with the authors' suggestion that the eccentric macular hole probably results 'from excessive manipulation,' we believe that the culprit is not necessarily the direct iatrogenic insult alone. We concur with their opinion that 'outer retinal degenerative changes may increase the risk of eccentric macular hole formation.'¹ Concomitant predisposing degenerative weaknesses as in the presence of 'extensive drusen'¹ or myopic chorioretinal degeneration (as in our case) as well as weakening of the glial structure of the retina caused by decapitation of the Muller cells³ may also play a role in the development of eccentric macular hole.

References

- 1 Polkinghorne PJ, Roufail E. Eccentric macular hole formation associated with macular hole surgery. *Eye* 2007; **21**: 122–123.
- 2 Rubinstein A, Bates R, Benjamin L, Shaik A. Iatrogenic eccentric full thickness macular holes following vitrectomy with ILM peeling for idiopathic macular holes. *Eye* 2005; **19**: 1333–1335.
- 3 Steven P, Laqua H, Wong D, Hoeraf H. Secondary paracentral holes following internal limiting membrane removal. *Br J Ophthalmol* 2006; **90**: 293–295.

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Sir,
Reply to Sangtam *et al*

We thank Sangtam *et al* for their useful comments on our observations regarding eccentric macular holes as a complication of macular surgery. In our letter, we described this phenomenon occurring subsequent to surgery for epiretinal membrane formation.¹

Rubinstein *et al*² first reported this complication in a series of patients who had undergone macular hole surgery. Our report suggested that other surgeries might also precipitate this outcome and identified three possible contributing factors, namely, excessive manipulation, outer retinal degenerative changes and experience of the surgeon.

A former fellow coined the term the *fellow eye syndrome*, a term which has become synonymous with the complication in this region. We certainly did not intend for Sangtam *et al* or others to take offence at this suggested name, but in absence of Rubinstein providing an eponym we believe this appellation might be an acceptable intitution.

References

- 1 Polkinghorne PJ, Roufail E. Eccentric macular hole formation associated with macular hole surgery. *Eye* 2007; **21**: 122–123.
- 2 Rubinstein A, Bates R, Benjamin L, Shaik A. Iatrogenic eccentric full thickness macular holes following vitrectomy with ILM peel for idiopathic macular holes. *Eye* 2005; **19**: 1333–1335.
- 3 Harris A, Arend O, Kagemann L, Garrett M, Chung HS, Martin B. Dorzolamide, visual function and ocular hemodynamics in normal-tension glaucoma. *J Ocul Pharmacol Ther* 1999; **15**: 189–197.
- 4 Harris A, Arend O, Chung HS, Kagemann L, Cantor L, Martin B. A comparative study of betaxolol and dorzolamide effect on ocular circulation in normal-tension glaucoma patients. *Ophthalmology* 2000; **107**: 430–434.
- 5 Harris A, Jonescu-Cuyppers CP, Kagemann L, Nowacki EA, Garzosi H, Cole C *et al*. Effect of dorzolamide timolol combination versus timolol 0.5% on ocular bloodflow in patients with primary open-angle glaucoma. *Am J Ophthalmol* 2001; **132**: 490–495.
- 6 Harris A, Migliardi R, Rechtman E, Cole CN, Yee AB, Garzosi HJ. Comparative analysis of the effects of dorzolamide and latanoprost on ocular hemodynamics in normal tension glaucoma patients. *Eur J Ophthalmol* 2003; **13**: 24–31.

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Sir,

The clinical utility of colour Doppler imaging

We read, with great interest, the recent article by Zeitz, Vilchez, Matthiessen, Richard, and Klemm titled 'Volumetric colour Doppler Imaging: a useful tool for the determination of ocular blood flow in glaucoma patients?' Two issues within the manuscript require attention. The authors state that the only technology providing quantification of volumetric flow as volume per unit time in a specific vessel is fluorescein angiography. They have ignored the Canon laser blood flowmeter. The technology is FDA approved, has been reported in the literature in multiple publications,¹ and its very existence contradicts the authors' statement.

More importantly, the authors conclude that since they were unable to detect significant dorzolamide-induced increases in retinal blood flow with a volumetric CDI measurement, the technology is inapplicable in ophthalmology. This is a sweeping statement that is unsupported by research findings. In the present study, the authors' findings are consistent with those in the literature. In multiple studies, we also failed to detect significant changes in CDI parameters.^{2–5} The present findings support the existing literature suggesting that arterio-venous passage times are more sensitive to changes in retinal haemodynamics than CDI.

References

- 1 Sato E, Feke GT, Menke MN, Wallace MJ. Retinal haemodynamics in patients with age-related macular degeneration. *Eye* 2006; **20**: 697–702.

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Sir,

Reply to A Harris

We would like to take the opportunity to reply to the comment by Alon Harris on our recent publication on volumetric colour Doppler imaging (vCDI). We agree with Alon Harris that the Canon laser blood flowmeter is to measure blood flow, but this is only the case in visible retinal vessels. The ciliary arteries or the ophthalmic artery cannot be assessed by the Canon laser blood flowmeter. This is an important limitation of this method.

In general, we agree with the comment of Alon Harris that the reports on effects of dorzolamide on CDI parameters are nonuniform. Nevertheless, it is commonly thought and also shown by the publications of Harris cited in his letter to the editor that dorzolamide increases ocular blood flow. Therefore, we applied dorzolamide as a standard in our study since a method that is applicable in clinic and research should detect such changes. vCDI failed to do so, which led us to the negative conclusion on vCDI's usefulness. In accordance with this conclusion, Harris is probably right that the