Figure 2 At 3 weeks after steroid pulse therapy, late-phase fluorescein angiography of the right eye (a) and the left eye (b) of the patient. Late-phase fluorescein angiography showed resolution of serous retinal detachment and fluorescein leakage (a, b).

This case is noteworthy for two reasons. First, the ophthalmologist can be the first medical contact, before systemic symptoms appears in PNH. Had the patient not been checked with laboratory examination in our case, other problems may have ensued. Second, this is the first report of PNH with bilateral serous retinal detachment.

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Sir,

Alcohol debridement for recurrent corneal erosions

We read with interest the paper by Ramamurthi *et al*¹ on recurrent corneal erosions. The authors should be congratulated for such a comprehensive review of this common condition. However, we feel that the use of alcohol debridement of the epithelium as an alternative treatment method should have been included.

Alcohol debridement has been mentioned in the literature since 2000.² Its use first gained popularity after it was used in LASEK. It was noted that alcohol debridement cleaved a smooth plane and was associated with faster visual rehabilitation and reduced postoperative haze.³ Dua *et al*⁴ started to use this technique for recurrent erosion after noticing that following alcohol debridement there was increased difficulty debriding the epithelium if repeat LASEK was required. In their study,⁴ on the use of alcohol debridement for recurrent erosions, 75% had complete resolution of symptoms after 1 month of treatment.

We ourselves have used alcohol debridement as a treatment method in York since 2003 with comparable success rates. The technique we use is similar to that described by Dua *et al*.⁴ We apply 20% alcohol in a corneal well for 60 s. The epithelium is then rinsed with balanced salt solution and the loose epithelium removed. We then insert a bandage contact lens for 1 week or until the epithelium is healed.

Mah, as quoted by Lipner,⁵ felt that alcohol debridement does not present a valid alternative for recurrent erosions, feeling that it is more complicated and that the results are the same as those with mechanical epithelial debridement or microstromal puncture. However, we disagree. While we acknowledge that it is not a suitable treatment option for all patients, for recurrent erosion caused by localised trauma, in the absence of a dystrophy, it is a cheap, successful and

readily accessible treatment method. It should therefore be mentioned as a valid alternative in units who do not have easy access to PTK.

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Sir,

Minimising blinding complications of cyclodiode laser in high risk and only eyes

We read with great interest the article by Tay *et al*¹ reporting the first case of suprachoroidal haemorrhage as a blinding complication of cyclodiode laser. The use of cyclodiode laser has been increasing over the last decade as an effective management of complicated glaucoma. This phenomenon has unmasked uncommon but potentially blinding complications such as suprachoroidal haemorrhage,¹ scleral perforations with² or without^{3,4} choroidal haemorrhage, necrotising scleritis,⁵ malignant glaucoma,⁶ and pan-ophthalmitis.⁴

Figure 1 Debris arising from the ciliary body post-cyclodiode laser (arrow).

Certainly ultrasonically, debris can be seen arising from the ciliary body post-cyclodiode (Figure 1).

It is worthwhile noting that these reported complications had occurred in the setting of scleral thinning and/or high laser power setting (2000 mW power and 2000 ms duration) with audible 'pops'. Tay *et al*¹ treated their eye (only functioning eye, other eye count fingers) with 60 applications of 2000 mW power and 2000 ms duration to 360°. It was suggested that hypotony was the initiating event resulting in suprachoroidal haemorrhage. Our default laser setting is 1500 mW and 1500 ms, titrated down by 250 mW if there is an audible 'pop'.

In only eyes and in high-risk eyes, such as a history of hypotony post-cyclodiode laser treatment to the same or fellow eye, large eyes (eg buphthalmos, high myopes), aphakes, uveitic eyes, history of multiple ocular surgery, and connective tissue disease, we propose a sequential titrated cyclodiode laser treatment. In these eyes, we initially start with one quadrant, leaving the other three quadrants untreated. We may even start at laser setting of 1000 mW and 1000 ms. Palmer *et al*⁷ found that about 40% less energy is required to achieve ciliary photocoagulation in thin sclera compared to normal thickness sclera in cadaveric eyes. Another important practical point is checking the tip of the probe so that there is no debris or carbonisation, which increases laser delivery. Even with the recommended single use probe (a new probe for each eye), carbonisation can still occur during any one of the applications on the one eye.

We understand that this may involve more treatment sessions to achieve the target intraocular pressure. However the alternative regimen of sequential titrated cyclodiode laser treatment offers an increased safety profile in high-risk eyes prone to hypotony and may help to prevent the devastating blinding complications that can occur in these eyes.