- 3 De Becker I, Walter M, Noel LP. Phenotypic variations in patients with a 1630 A>T point mutation in the PAX6 gene. *Can J Ophthalmol* 2004;**39**:272–8.
- 4 Ritch R, Forbes M, Hetherington J Jr, et al. Congenital ectropion uveae with glaucoma. *Ophthalmology* 1984;**91**:326–31.
- 5 Futterweit W, Ritch R, Teekhasaene C, et al. Coexistence of Prader-Willi syndrome, congenital ectropion uveae with glaucoma, and factor XI deficiency. *JAMA* 1986;**255**:3280–2.
- 6 Trovo-Marqui AB, Goloni-Bertollo EM, Teixeira MF, et al. Presence of the R1748X mutation in the NF1 gene in a Brazilian patient with ectropion uveae. *Ophthalmic Res* 2004;**36**:349–52.
- 7 Azuma N, Yamada M. Missense mutation at the C terminus of the PAX6 gene in ocular anterior segment anomalies. *Invest Ophthalmol Vis Sci* 1998;**39**:828–30.
- 8 Mirzayans F, Pearce WG, MacDonald IM, et al. Mutation of the PAX6 gene in patients with autosomal dominant keratitis. *Am J Hum Genet* 1995;**57**:539–48.
- 9 Nelson LB, Spaeth GL, Nowinski TS, et al. Aniridia. A review. *Surv Ophthalmol* 1984;**28**:621–42.

Fourth time lucky: a case of multiple recurrence of a macular hole

We present the case of a patient who has undergone four macular hole operations for late multiple recurrences. The same experienced ophthalmologist confirmed the presence of each macular hole and performed each operation.

Case report

A 55 year old female myope, −3.75 D right eye and −3.5 D left eye, was referred to the vitreoretinal clinic with a 2 month history of right visual disturbance. On presentation, her visual acuities were 6/9 right eye and 6/60 left eye. Fundal examination and fluorescein angiography showed a right stage I macular hole and a left stage IV macular hole. As the left macular hole was long standing, it was decided not to operate on this eye. Two months later the right visual acuity was reduced further to 6/36 because of progression to a stage II macular hole and she underwent a right vitrectomy and 18% C₃F₈ gas tamponade. At the time of surgery, the posterior hyaloid was detached and the absence of an epiretinal membrane was confirmed but no inner limiting membrane (ILM) peel was performed. Following this and each subsequent vitrectomy, the patient postured face down for 2 weeks. Postoperatively, right visual acuity improved to 6/6 but 8 months later the hole reopened, and her right visual acuity dropped to 6/18. A further vitrectomy and 20% C₃F₈ gas tamponade was carried out. During this, a silicon tipped aspiration needle was used to confirm the absence of vitreous at the macula and a barbed blade used to show that no epiretinal membrane was present. The hole closed and the vision improved to 6/12. Two years later the patient underwent uneventful right phacemulsification cataract surgery with posterior chamber intraocular lens implantation and her acuity was restored to 6/9. After another 2 years right visual acuity fell to 6/36, the hole had reopened. The patient underwent a third macular hole operation consisting of ILM peel and 18% C₃F₈ tamponade. The hole remained closed and the vision improved to 6/9 for a further 3 years when the hole reopened and the vision decreased to 6/24. A fourth procedure was performed consisting of trypan blue

assisted ILM peel to two disc diameters from the hole and 18% C₃F₈ gas tamponade.

When last seen, 16 months after surgery and 9 years after presentation, the right visual acuity was 6/12 and the macula hole confirmed as closed on ocular coherence tomography.

Comment

Previous studies have shown that repeat macular hole surgery is successful in closing recurrent macular holes and improving vision.^{1,2} Late reopening of successfully closed macular holes has been reported to occur in between 4.8% and 6.9% of eyes.^{3–4} The success of repeat surgery did not depend on whether the hole reopened early or late as in this case.^{1,2} A third recurrence of a macular hole is a less common event. The results of repeat macular hole surgery are better in those eyes in which one of the previous operations had successfully closed the macula hole.¹ In those eyes that had previous successful surgery the mean final visual acuity was 20/40. By comparison, in those eyes in which surgery failed to close the macula hole, the success of final closure rate was 56% and the mean final visual acuity was 20/100.⁵

The pathophysiology for failure of closure and reopening remain unclear. Early failure is thought to result from the lack of formation of a stable glial plug that is essential for hole closure.⁶ The causes for late reopening remain unclear as in this case, it may be a spontaneous event.^{2,7} Some authors have suggested that chronic cystoid macular oedema, cataract surgery, or the development of an epiretinal membranes resulting in tractional forces may be involved.^{2,4} This was not the case in this patient.

In summary, this case suggests that the visual outcome can be favourable following multiple repeat macular hole operations especially if there has been previous successful closure.

H M Hussin, R H Y Asaria, N E Knox
Cartwright, R H B Grey

Bristol Eye Hospital, Lower Maudlin Street, Bristol BS1 2LX, UK

Correspondence to: Mr Hussin M Hussin, Bristol Eye Hospital, Lower Maudlin Street, Bristol BS1 2LX, UK; h.m.hussin@bristol.ac.uk

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References

- 1 Smiddy WE, Sjaarda RN, Glaser BM, et al. Reoperation after failed macular hole surgery. *Retina* 1996;**16**:13–18.
- 2 Paques M, Massin P, Blain P, et al. Long-term incidence of reopening of macular holes. *Ophthalmology* 2000;**107**:760–5.
- 3 Duker JS, Wendel R, Patel AC, et al. Late reopening of macular holes after initially successful treatment with vitreous surgery. *Ophthalmology* 1994;**101**:1373–8.
- 4 Christmas NJ, Smiddy WE, Flynn HW Jr. Reopening of macular holes after initially successful repair. *Ophthalmology* 1998;**105**:1835–8.
- 5 Thompson JT, Sjaarda RN. Results of macular hole surgery in patients over 80 years of age. *Retina* 2000;**20**:433–8.
- 6 Funata M, Wendel RT, de la CZ, et al. Clinicopathologic study of bilateral macular holes treated with pars plana vitrectomy and gas tamponade. *Retina* 1992;**12**:289–98.
- 7 Thompson JT, Sjaarda RN. Surgical treatment of macular holes with multiple recurrences. *Ophthalmology* 2000;**107**:1073–7.

MAILBOX

Vision restoration therapy and raising red flags too early

Vision restoration therapy (VRT) has been the subject of considerable controversy,^{1–3} and with Horton's latest editorial³ it has now turned rather personal. The editorial is filled with numerous inaccuracies and misrepresentations and this cannot go unanswered. Just as a scientific debate should not focus on fiction but on facts, comments by critics must be evaluated with the same rigor as the original work itself. Only by keeping track of all of the scientific evidence rather than by a personal and limited review of some selected facts will we get closer to the truth.

Firstly, it is a fundamental misrepresentation by Horton that we are trying to "salvage neurons" at the fringe of a lesion. Rather, we believe that VRT activates surviving neurons in areas of residual vision (ARVs)—that is, areas that are partially damaged.^{4–7} Repetitive use through daily training increases neuronal activation,⁸ which is greatly enhanced by attentional processes.⁹ No neuron is "salvaged."

It is correct that at the time Horton wrote his editorials no one had published studies on eye movements. But since then the first eye movement studies have been published in abstract form. It actually turns out that eye movements, when measured with an eye tracker, are not altered after VRT.¹⁰ There is also other indirect evidence, which is incompatible with the hypothesis that eye movements explain visual field improvements:

- Fixation performance in standard automated perimetry is an accepted quality control measure and our patients typically show >90% stable fixation, which is measured by counting the number of hits at fixation. Because this does not change after VRT (it usually improves slightly, see Kasten et al.,⁴ Poggel et al.,⁹ Reinhard et al.,¹¹ Mueller et al.,¹² and Sabel et al.¹³) it is unlikely that patients moved their eyes more after VRT.
- Horton erroneously believes that temporal field loss prevents blind spot measurements. This is true only for one eye. In our studies, the position of the blind spot remains unchanged in monocular measurement after VRT.¹³ Thus, neither eccentric fixation occurred after VRT nor are there more large saccades after VRT.¹⁰ The position of the blind spot is a reliable parameter of eccentric fixation.¹⁴
- If eye movements were responsible for the visual field shifts, then the entire visual field border should shift in parallel. In most patients this is not the case. The border of the visual field may shift in one place (say the lower quadrant) and remain invariant in another (upper quadrant). Also, highly localised border shifts are induced by attention cues,⁹ and horizontal border shifts may occur in the absence of border shifts at fixation. All of these observations are not compatible with the proposal that eye movements cause the visual field border shifts. In fact, in the periphery the border shifts tend to be more pronounced, which is explained by the cortical magnification factor—that is, by visual cortex architecture.