

the remaining eye, no evaluation was possible for 2 weeks because of vitreous haemorrhage. In the six eyes with BRAO, preoperative visual acuity was: hand motion, counting fingers, 20/400, 20/200, 20/200 and 20/25; at 48 h after surgery it was 20/200, 20/100, 20/100, 20/200, 20/100 and 20/30, respectively, and the final visual acuity was 20/50, 20/30, 20/80, 20/200, 20/25 and 20/25, respectively. In the eye with CRAO, visual acuity remained hand motion throughout.

It is worth noting, initially, that the usefulness of a conclusion based on six eyes without control is limited. However, the apparent improvement in visual acuity claimed by the authors deserves comment.

(1) All six of the eyes had temporal BRAO. In the vast majority of eyes with temporal BRAO, the border between the ischaemic and non-ischaemic retina passes through the fovea, as figs 2 and 3 of Garcia-Arumi *et al*¹ show. Also the fluorescein angiogram in fig 2 shows a patent cilioretinal artery. These are extremely important facts in determining whether the visual improvement noted was the result of removal of the embolus or simply the natural history of the disease. Having studied the natural history of about 200 eyes with BRAO, I recently stated: "In cilioretinal artery occlusion, branch retinal artery occlusion and CRAO with cilioretinal artery sparing, the junction between the infarcted and normal retina may often pass through the fovea. In many of these eyes, I have seen marked spontaneous visual acuity improvement occurring within several days or weeks, from almost 20/200 or worse to even normal. This spontaneous improvement is often erroneously attributed to an advocated treatment."²

When the junction between the normal and infarcted retina passes through the fovea, in such eyes, the retinal oedema associated with retinal infarction most probably also involves to some extent the adjacent normal foveal retina shortly after the occlusion; over the following weeks the normal foveal retina recovers spontaneously, resulting in natural visual improvement. In the series of Garcia-Arumi and colleagues, a significant visual improvement at final visit seen in four of six eyes is not uncommon in such eyes as part of the natural history, in my experience. Moreover, the eye in fig 2, not only had the junction between the normal and infarcted retina passing through the fovea but also had a patent cilioretinal artery, which is an important factor in natural improvement in visual acuity not only in BRAO but also in CRAO.³

(2) Having studied more than 450 patients with BRAO and CRAO, I have found another common confounding factor involving visual acuity testing. At the initial visit, because of sudden visual loss, the patient is emotionally upset, and tends to test poorly; for instance, a patient with only temporal BRAO with the border between the ischaemic and normal retina passing through the fovea should not have hand motion or even 20/400 visual acuity if tested properly because the other half of the macula and retina is still functioning normally. Moreover, later on it is not unusual to find patients with central scotoma learning to fixate eccentrically and show better visual acuity, which may erroneously be interpreted as genuine improvement. In my studies on various ocular vascular occlusive diseases, I have found that, unless improvement in visual acuity corresponds to improvement in central scotoma, it is not a genuine improvement but due to eccentric fixation.⁴

(3) As visual acuity testing assesses the function of only the fovea and not the entire

involved retina, visual fields, particularly with a Goldmann perimeter, provide much better information about the extent of visual loss and change. In my study, every eye with BRAO had visual fields plotted with a Goldmann perimeter, which showed that in eyes with BRAO there is often a reduction in the size of the visual field defect as part of the natural history. Garcia-Arumi and colleagues state that they recorded the visual fields with the Humphrey perimeter but give no information on the visual fields of their cases. Moreover, unlike the Goldmann perimeter, the Humphrey perimeter provides information about only the central 24° to 30° and not the entire involved retina.

(4) In the series of Garcia-Arumi and colleagues, six of seven eyes had had acute retinal ischaemia for 12–33 h and one for 4 h. We evaluated the retinal tolerance time to acute ischaemia experimentally in rhesus monkeys⁵ and found that, in CRAO, ischaemic retina can recover normal function from acute ischaemia of 97 min, but, after that, the longer the ischaemia, the more extensive the irreversible damage, so that acute ischaemia lasting 240 min results in massive irreversible retinal damage. Therefore, it does not seem logical that restoration of circulation in BRAO 4–33 hours after the occlusion would restore function in an already irreversibly damaged retina. Moreover, they found restoration of circulation in four of the six eyes on fluorescein angiography first performed 48 h after surgery. They argue that "in branch RAO ... some degree of perfusion at the macular area may be supplied by the contralateral temporal artery."¹ This may be true, but it may also be another factor in the spontaneous marked visual recovery in such eyes as part of the natural history.

In conclusion, on the basis of my studies on the natural history of eyes with BRAO, I believe that the improvement in visual acuity attributed by Garcia-Arumi and colleagues to embolotomy simply represents natural history.

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Competing interests: None declared.

References

- 1 Garcia-Arumi J, Martinez-Castillo V, Boixadera A, *et al*. Surgical embolus removal in retinal artery occlusion. *Br J Ophthalmol* 2006;**90**:1252–5.
- 2 Hayreh SS. Prevalent misconceptions about acute retinal vascular occlusive disorders. *Prog Retin Eye Res* 2005;**24**:493–519.
- 3 Hayreh SS, Zimmerman B. Central retinal artery occlusion: visual outcome. *Am J Ophthalmol* 2005;**140**:376–91.
- 4 Hayreh SS, Zimmerman B, Kardon RH. Visual improvement with corticosteroid therapy in giant cell arteritis. *Acta Ophthalmol Scand* 2002;**80**:355–67.
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NOTICES

Glaucoma

The latest issue of *Community Eye Health* (No 59) discussing new treatments for glaucoma in the developing world, with an editorial by leading specialist Richard Wormald. For further information please contact: Journal of Community Eye Health, International Resource Centre, International Centre for Eye Health, Department of Infectious and Tropical Diseases, London School of Hygiene and Tropical Medicine,

Keppel Street, London WC1E 7HT, UK (tel: +44 (0)20 7612 7964; email: Anita.Shah@lshtm.ac.uk; online edition: www.jceh.co.uk). Annual subscription (4 issues) UK £28/US\$45. Free to developing country applicants.

9th IOIS International symposium

The 9th International Ocular Inflammation Society international symposium will be held in Paris from 17–20th September 2007. For further information on registration please call +33 (0)1 70 08 69 82, or fax +33 (0)1 42 93 29 28, or email andrelamy1@wanadoo.fr. Or you can visit the website www.iois-paris-2007.com.

IAPB 8th General Assembly – 2008

The 2008 International Agency for the Prevention of Blindness Eighth General Assembly: "Excellence and Equity in Eye Care" will be taking place at the Centro de Convenções Rebouças, in Sao Paulo, Brazil on 28 July–2 August. For further information please email: agency@lvpei.org.

Second Sight

Second Sight would like to hear from experience Indian eye surgeons returning to India after training/working in the UK. Second Sight is a London based charity dedicated to the elimination of cataract blindness in India. For further information please contact Dr Lucy Mathen, email: lucymathen@yahoo.com.

Singapore National Eye Centre

The Singapore National Eye Centre will be holding its 18th Anniversary International Meeting from 14–17 March 2008 at Suntec City Convention Centre, Singapore. For further information please call +65 6322 8374, fax +65 6227 7290 or email meet@snecc.com.sg.

Inaugural Asia Cornea Society Scientific Meeting

The Asia Cornea Society will be holding its inaugural meeting on 13–14 March 2008 at the Shangri La's Rasa Sentosa Resort, Singapore. For further information please fax +65 6227 7291 or email acs@snecc.co.sg.

International Ocular Blood Flow Symposium

The International Ocular Blood Flow Symposium will be taking place on 13 October 2007 at the Sutton Place Hotel, Toronto, Canada. For further information please telephone +416 978 2719 or +1 888 512 8173, fax +416 946 7028 or email ce.med@utoronto.ca.

Neuro-Ophthalmology and Strabismus – 2008 EUPO Residents' Course

The 2008 European Professors in Ophthalmology (EUPO) Course will be held in Geneva, Switzerland, on September 5–6, 2008. The course organized by Prof. Avinoam B. Safran will provide an overview and an update on recent advances in Neuro-Ophthalmology and Strabismus. Further information on website: <http://eupo.eu>.

CORRECTION

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In the paper by Tranos *et al* (*Br J Ophthalmol* 2006;**90**:1107–10) the spelling of the third author is incorrect. The correct spelling is Zambarakji. We apologise for this error.