References

1   Jonas JB, Akkoyun I, Kamppeter B, Kreissig I, Degenring RF. Branch retinal vein occlusion treated by intravitreal triamcinolone acetonide. *Eye* 2005; **19**: 65–71.
2   Altman DG. *Practical Statistics for Medical Research*, 1st edn. Chapman & Hall: London, 1991.

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Sir,
Reply to intravitreal triamcinolone acetonide as treatment of branch retinal vein occlusion

We would like to thank Dr Wilkins for his interest in our study.[1] We do agree with him that the statistical basis of the study is relatively weak. As he pointed out, multiple comparisons were performed so that Bonferoni's method to correct for multiple comparisons might have been necessary. On the other hand, the number of patients in the study group was rather low ($n = 10$), despite of which the difference in visual acuity between baseline measurement and measurement at 1 month after injection was marginally significant ($P = 0.027$). Additionally, he difference between visual acuity at baseline of the study and the best visual acuity during follow-up was significant in the study group, but not in the control group. Furthermore, the study fits with other investigations on the intravitreal use of triamcinolone acetonide for a number of diseases associated with cystoid macular oedema including branch retinal vein occlusion.[2–4] In all of these studies, a decrease in macular oedema, and in most of the studies, an increase in visual acuity was observed. In conclusion, we appreciate very much Dr Wilkins' comments and consider the present study as a precursor of ongoing randomized controlled trials on intravitreal triamcinolone acetonide as treatment of retinal vein occlusions.

References

1   Jonas JB, Akkoyun I, Kamppeter B, Kreissig I, Degenring RF. Intravitreal triamcinolone acetonide as treatment of branch retinal vein occlusion. *Eye* 2005; **19**: 65–71.

2   Chen SD, Lochhead J, Patel CK, Frith P. Intravitreal triamcinolone acetonide for ischaemic macular oedema caused by branch retinal vein occlusion. *Br J Ophthalmol* 2004; **88**: 154–155.
3   Özkiris A, Evereklioglu C, Erkilic K, Ilhan Ö. The efficacy of intravitreal triamcinolone acetonide on macular edema in branch retinal vein occlusion. *Eur J Ophthalmol* 2005; **1**: 96–101.
4   Jonas JB, Kreissig I, Degenring RF. Intravitreal triamcinolone acetonide for treatment of intraocular proliferative, exudative and angiogenic diseases. *Prog Ret Eye Res* 2005 (forthcoming).

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Proprietary interest: none

Sir,
Macrophthalmos as a long-term outcome of severe open globe injury

Long-term sequelae of open globe injuries include cataract, glaucoma, phthisis bulbi, and sympathetic ophthalmia. We present a case of a severe open globe injury in childhood resulting in macrophthalmos as an adult.

Case report

A 39-year-old man presented with gradual increased prominence of his left eye, which had suffered a corneal penetrating eye injury from a wooden stick at age 7 years and had undergone primary repair.

On examination, best-corrected visual acuities were 6/7.5 OD and perception of light OS. The appearance of the left eye is shown in Figure 1. Intraocular pressures were 16 mmHg OD and 28 mmHg OS. The left eye was aphakic.

Thyroid function tests were normal. An orbital CT scan revealed an elongated left axial length of 33 mm,

Figure 1 (a) Left pseudoproptosis especially prominent on downgaze. (b) Scleral thinning with choroid visible under superotemporal bulbar conjunctiva. (c) Nasal corneal neovascularisation and scarring with central climatic droplet keratopathy. The anterior chamber was markedly disrupted.
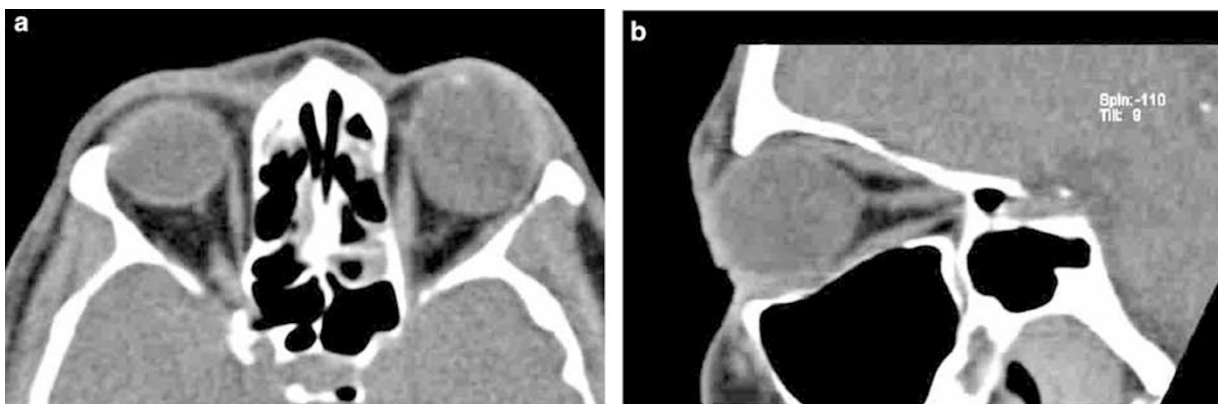
Figure 2 (a) Axial view of orbital CT scan showing left macrophthalmos measuring 33 mm in the anteroposterior axis, compared to the normal right eye measuring 26 mm. (b) Sagittal view of orbital CT scan of left eye showing pseudoproptosis secondary to an enlarged, deformed left globe.

compared to 26 mm in the right eye (Figure 2a and b). There was no evidence of an intraorbital mass or extraocular muscle enlargement.

Comment

Ocular trauma is the leading cause of monocular blindness in children.[1–3] In a series of 50 eyes that had suffered severe globe rupture and were not removed within 2 weeks of injury,[4] the majority (70%) became phthisical. No eyes developed macrophthalmos.

In normal eyes, after the rapid growth in axial length in the first 5 years of life, a slow juvenile phase lasting until age 13 years results in an increase of only 1.3–1.4 mm.[5] In a series of 13 adult patients with unilateral childhood traumatic cataract, all injured eyes

had greater axial lengths than their normal fellow eyes (range 0.1–11.5 mm, mean 2.48 mm).[6] Interestingly, the patient with an injured eye 11.5 mm longer than the normal fellow eye had suffered the injury at age 3 years.

The authors believe that the reported case appears to be the first reported case of severe macrophthalmos following trauma in a juvenile. This could be due to gradual stretching of a biomechanically weakened sclera from previous trauma in response to raised intraocular pressure and extraocular muscle tension. This case suggests that since the dynamic process of emmetropisation continues after the age of 7 years, the axial length might remain malleable, if this process is interrupted by trauma and visual impairment.

References

1   Maw R, Pineda R, Pasquale LR, Dana MR. Traumatic ruptured globe injuries in children. *Int Ophthalmol Clin* 2002; **42**: 157–165.
2   Jandeck C, Kellner U, Bornfeld N, Foerster MH. Open globe injuries in children. *Graefes Arch Clin Exp Ophthalmol* 2000; **238**: 420–426.
3   Rudd JC, Jaeger EA, Freitag SK, Jeffers JB. Traumatically ruptured globes in children. *J Pediatr Ophthalmol Strabismus* 1994; **31**: 307–311.
4   Brackup AB, Carter KD, Nerad JA, Folk JC, Pulido JS. Long-term follow-up of severely injured eyes following globe rupture. *Ophthalmol Plast Reconstr Surg* 1991; **7**: 194–197.
5   Larsen JS. The sagittal growth of the eye. IV. Ultrasonic measurement of the axial length of the eye from birth to puberty. *Acta Ophthalmol* 1971; **49**: 873–886.
6   Calossi A. Increase of ocular axial length in infantile traumatic cataract. *Optom Vis Sci* 1994; **71**: 386–391.

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Sir,
Glutamate excitotoxicity in glaucoma: throwing the baby out with the bathwater?

The recently published study by Kwon *et al* provides some interesting and valuable data concerning the possible role of glutamate in neuronal cell death in glaucoma.[1] As pointed out in the accompanying editorial, this is a field of study that has been bedevilled by conflicting results and even scientific fraud.[2] The editorial[2] is right to draw attention to these matters and to state that 'it is important to re-evaluate the evidence for and against glutamate being causal in the development of glaucoma.' Additionally, the editorial goes on to imply that there is thus no role for glutamate and its receptors (particularly NMDA receptors) in glaucomatous neuronal death. However, it may be wise to urge caution in the interpretation of these new data, as the lack of vitreal glutamate elevation does not necessarily mean that there is no role for glutamate and its receptors in glaucoma. Indeed, Kwon *et al*[1] clearly discuss some of the relevant issues. Although an elevation of vitreal glutamate *might* be expected in glaucoma models, there is no *a priori* reason for assuming that this must be so, and indeed it may be that such an increase is not as central to the disease as implied in the editorial:[2] there is a danger here of throwing the baby out with the bathwater.

It is well known that glutamate is an amino acid that is abundant in all cells and that it is intimately involved in many metabolic processes, with only a relatively small proportion being involved in neurotransmission and the activation of the receptors involved in this.[3] This compartmentalisation of glutamate, maintained by active transport processes, makes it difficult to measure changes in tissue levels of glutamate related to activation of glutamate receptors unless relatively noninvasive sampling techniques with good temporal resolution are applied close to the source of glutamate (eg microdialysis or push–pull perfusion). Thus, sampling of retinal tissue post-mortem or sampling of vitreal glutamate, as performed by Kwon *et al*,[1] might not yield information directly relevant to the activation of glutamate receptors on threatened neurones.

A second issue is the nature of the NMDA receptor (the glutamate receptor that is thought to be involved in the neurodegenerative aspects of glutamate pathophysiology) itself. A feature of the NMDA receptor is that its ion channel is largely blocked by magnesium ions when cell membranes are maintained at a healthy resting membrane potential.[4] If tissue is compromised (eg injury or anoxia), then the membrane potential will depolarise and this will then relieve the magnesium ion block of the NMDA receptors: the consequence of this is that even relatively 'normal' levels of glutamate will have a much greater effect and there will be a substantial inward current into cells carried by sodium and calcium ions. Thus, large increases in glutamate levels may not be