Figure 3 (a) Magnetic resonance imaging of the brain and orbits, illustrating the large left eye with bulging of the temporal (left) side. No other pathology apart from the staphyloma seen in the left eye. (b) Note also the right intraocular lens implant and 'funnel' retinal detachment with failed scleral buckling (retinal detachment attempted correction procedure) in right eye.

perforation with needle local anaesthesia (retrobulbar or peribulbar anaesthetic³). Preferred techniques that minimise this risk or overcome the problem completely include topical, sub-Tenon's or general anaesthesia.^{1,4}

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Sir,

Recovery of stereoacuity 27 years after trauma: an unusual case

The ability of adults with long-standing monocular visual loss and associated secondary strabismus to regain stereoacuity is poorly understood. Although there are reports of patients regaining a high level of binocular function many years following the onset of strabismus they all had a good level of visual acuity with no prior period of visual deprivation.^{1–3} We report the case of a patient who regained a high level of stereoacuity following correction of traumatic aphakia and secondary exotropia 27 years after a penetrating eye injury.

Case report

A 64-year-old female was referred for further assessment of a secondary exotropia associated with aphakia, which developed following a penetrating injury to her left eye 27 years earlier. Her acuity in this eye was hand movements only, although with an aphakic correction it improved to 6/12. However, with this correction she complained of constant horizontal diplopia and as a result she had never worn a contact lens. In addition, she had a secondary exotropia measuring 45 prism dioptres (Δ) for near and 30 Δ for distance. Her visual acuity was 6/5 in her right eye with normal ophthalmic examination. There were concerns that if she underwent anterior segment reconstruction with secondary intraocular lens implantation followed by possible strabismus surgery she was at risk of intractable diplopia. She therefore had a botulinum toxin injection to her left lateral rectus muscle, which reduced her exotropia to 12 Δ for near and 6 Δ for distance. Following this she was encouraged to wear her

contact lens as often as possible; whereas she was initially aware of intermittent diplopia this improved over a period of several weeks by which stage she was noted to have a non-specific exophoria with left suppression. These findings remained stable over a period of several months and it was therefore deemed to be safe to proceed with implantation of a secondary intraocular lens combined with iris prosthesis. Post-operatively her acuity gradually improved to 6/9 with orthoptic assessment revealing a non-specific exophoria. Although she was initially aware of diplopia this gradually settled and over the following 4 months her best corrected acuity improved to 6/6 and she regained 110 arc sec (Frisby). At her most recent clinic visit 1 year after her surgery her examination is unchanged.

Comment

Recovery of a high level of stereoacuity following such a long period of visual deprivation and longstanding strabismus is a very rare occurrence. Although there have been reports of patients regaining up to 40 arc sec of stereoacuity more than 30 years following the onset of their strabismus,^{1,2} in contrast to our patient they all had acuities of at least 6/9 in the squinting eye with no reported history of visual loss. It is thought by some that the ability of patients with chronic acquired strabismus to regain binocular vision is dependent upon their previous capacity for binocularity.⁴ As our patient regained such a high level of stereoacuity it is reasonable to assume that this was present before her original injury. However, the fact that this occurred after such a long period of poor vision is most unusual and would indicate that the visual system has a surprising capacity for recovery.

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Sir, Acute leber hereditary optic neuropathy in a 73-year-old man

Sudden onset of optic neuropathy in the elderly is most frequently vascular in aetiology; however, when bilateral, the diagnosis of LHON should still be considered. We report the oldest documented case of visual loss due to LHON.

Case report

A 73-year-old man presented with severe painless sequentially bilateral visual loss over the past few weeks. He recorded counting fingers vision bilaterally, but ocular examination was unremarkable with only mild dry age-related macular degeneration, normal retinal vessels, and healthy looking optic discs. Investigations including fluorescein angiography, CT scan of head and orbits, and extensive blood testing failed to provide any explanation.

He was a known hypertensive with a history of previous myocardial infarction and peripheral vascular disease requiring bilateral femoral bypass grafting. Carotid Doppler ultrasound scanning demonstrated internal carotid artery stenosis of >70% on the side of the initially affected eye, but no significant stenosis on the other side.

A family history of LHON was elicited. Genetic screening to investigate this possible diagnosis was undertaken which found a guanine to adenine mutation at position 11778 on the mitochondrial DNA. His optic discs had become pale by the 10th week after presentation and he could see only hand movements.

Comment

LHON is a maternally inherited condition caused by point mutations in the mitochondrial DNA. Penetrance, independent of genotype, is estimated at 25–50% in males and 5–11% in females,¹ visual loss becoming bilateral within 1 year in 97% of cases. The typical interval between eyes is 2–4 months, although simultaneous bilateral involvement is reported.²