Unfortunately, subsequent investigations could not corroborate the initial findings when they failed to detect elevated level of vitreous glutamate in both human and animal models of glaucoma.[10–12] Thus, the original evidence that stimulated the theory of glutamate excitotoxicity in glaucoma is now in serious doubt. Salt and Cordeiro, and indeed many others in the glaucoma community, are asking whether it is still possible that glutamate excitotoxicity plays a significant role in glaucoma. The answer is unclear. What *is* clear is that additional, reproducible, experimental support will be required for glutamate excitotoxicity to be accepted as a significant factor in glaucoma development.

References

1 Kwon YH, Rickman DW, Baruah S, Zimmerman MB, Kim CS, Boldt HC *et al.* Vitreous and retinal amino acid concentrations in experimental central retinal artery occlusion in the primate. *Eye* 2005; **19**: 455–463.

2 Adachi K, Kashii S, Masai H, Ueda M, Morizane C, Kaneda K *et al.* Mechanism of the pathogenesis of glutamate neurotoxicity in retinal ischemia. *Graefes Arch Clin Exp Ophthalmol* 1998; **236**: 766–774.

3 Louzada-Junior P, Dias JJ, Santos WF, Lachat JJ, Bradford HF, Coutinho-Netto J. Glutamate release in experimental ischaemia of the retina: an approach using microdialysis. *J Neurochem* 1992; **59**: 358–363.

4 Muller A, Villain M, Bonne C. The release of amino acids from ischemic retina. *Exp Eye Res* 1997; **64**: 291–293.

5 Iijima T, Iijima C, Iwao Y, Sankawa H. Difference in glutamate release between retina and cerebral cortex following ischemia. *Neurochem Int* 2000; **36**: 221–224.

6 Bull ND, Barnett NL. Retinal glutamate transporter activity persists under simulated ischemic conditions. *J Neurosci Res* 2004; **78**: 590–599.

7 Lotery AJ. Glutamate excitotoxicity in glaucoma: truth or fiction? *Eye* 2005; **19**: 369–370.

8 Dreyer EB, Zurakowski D, Schumer RA, Podos SM, Lipton SA. Elevated glutamate levels in the vitreous body of humans and monkeys with glaucoma. *Arch Ophthalmol* 1996; **114**: 299–305.

9 Brooks DE, Garcia GA, Dreyer EB, Zurakowski D, Franco-Bourland RE. Vitreous body glutamate concentration in dogs with glaucoma. *Am J Vet Res* 1997; **58**: 864–867.

10 Carter-Dawson L, Crawford ML, Harwerth RS, Smith III EL, Feldman R, Shen FF *et al.* Vitreal glutamate concentration in monkeys with experimental glaucoma. *Invest Ophthalmol Vis Sci* 2002; **43**: 2633–2637.

11 Honkanen RA, Baruah S, Zimmerman MB, Khanna CL, Weaver YK, Narkiewicz J *et al.* Vitreous amino acid concentrations in patients with glaucoma undergoing vitrectomy. *Arch Ophthalmol* 2003; **121**: 183–188.

12 Wamsley S, Gabelt BT, Dahl DB, Case GL, Sherwood RW, May CA *et al.* Vitreous glutamate concentration and axon loss in monkeys with experimental glaucoma. *Arch Ophthalmol* 2005; **123**: 64–70.

YH Kwon, DW Rickman, S Baruah, MB Zimmerman, C-S Kim, HC Boldt, SR Russell and SS Hayreh

Department of Ophthalmology and Visual Sciences, University of Iowa, UIHC, 200 Hawkins Dr., Iowa City, IA 52242, USA

Correspondence: YH Kwon
Tel: +1 319 356 3933;
Fax: +1 319 353 7699.
E-mail: young-kwon@uiowa.edu

Sir,
Monocular complex visual hallucinations and their suppression by eye closure

Following a recent report of monocular Charles Bonnet syndrome (CBS) after enucleation,[1] we wish to present a case of complex monocular visual hallucinations in nonarteritic anterior ischaemic optic neuropathy (NA-AION).

Case report

A 68-year-old hypermetropic gentleman with hypertension, diabetes, and hypercholesterolaemia developed crescendo exertional angina over 6 weeks. He underwent coronary artery bypass grafting (CABG) for triple-vessel disease. Previously in 1998, he suffered a left hemisphere transient ischaemic attack. Carotid Doppler ultrasonography had shown an occluded left internal carotid artery (ICA) with 50% right ICA stenosis. The studies repeated preoperatively showed no significant change.

About 6 days postCABG he awoke in hospital with painless left visual loss. His unaided vision was 6/9 OD and only hand movements inferiorly OS. There was a dense left afferent pupillary defect. His left optic disc was swollen and his right had no cup. NA-AION was diagnosed after giant cell arteritis was excluded.

Later that day he began to experience complex visual hallucinations arising solely from his inferior left visual field. The patient soon realised that these were abolished by left eye closure and gaze aversion and, of his own volition, wore a left spectacle occluding patch. He described two young children in black and white, a boy and a girl, aged 5 to 10 years, dressed in Victorian

clothing. He also described a bearded doctor. These figures were static, smoothly approached him and were almost constantly appearing except when he abolished them. Each apparition lasted 10 s to 1 min. The hallucinations subsided after 8 days.

Ultrasound demonstrated reversed left ophthalmic artery flow and some anomalous arterial flow within his left postero-superior orbit. Intracranial MR angiography and MR orbits excluded an arteriovenous malformation but did show increased vascularity within this region, consistent with collateral vasculature between the extra- and intra-cranial circulations. MRI brain confirmed an old left hemisphere subcortical infarct and demonstrated no occipital abnormalities.

Comment

Our patient's risk factors for NA-AION included vascular comorbidity, hypermetropic discs, and a state of probable low flow within the left collateralized cerebral circulation.

Monocular visual hallucinations are very infrequent. A retrospective study of phantom eye syndrome found 7 of 112 patients experienced complex visual hallucinations after unilateral enucleation, although it is unclear whether these phenomena were subjectively monocular.[2] Uhthoff (1899) probably provided the original description,[3] highlighted by the recent case report in *Eye*.[1]

CBS varies in semiology. Single hallucinations may last from few seconds to hours. Subjects tend to see people, animals, buildings, and scenery that can be static or moving. When moving, the hallucinations tend to do so en masse.[4] Most subjects have reduced vision secondary to ocular disease. Some diagnostic criteria require the absence of central visual or cortical lesions but most require hallucinations with intact sensorium and insight.[4]

Certain pathophysiological mechanisms have been proposed in order to explain CBS. The perceptual release theory[5] postulates the disinhibition of higher cortical activity resulting from a reduction in the afferent stimulus. This releases previously suppressed subconscious perceptual imagery in the form of hallucinations. The phantom vision theory is related to the deafferentation model[6,7] and postulates spontaneous higher visual cortical discharges in response to the loss of visual input.[8] It is unclear how these theories apply to monocular complex hallucinations since a cortically generated process should be represented bilaterally, unless there is a mechanism which suppresses the monocular representation for the normal eye.

Hallucination abolition on eye closure is sometimes explained by secondary normalization of sensory inputs.

In phantom limb syndromes, for example, the painful symptoms of an amputated arm may diminish when a mirror is placed such that the patient views the existing arm on the other side.[9] By covering his affected eye, which had some residual vision, our patient theoretically also normalized his sensory input. This could explain why his hallucinations vanished even with his affected eye open.

References

1 Ross J, Rahman I. Charles Bonnet Syndrome following enucleation. *Eye*, Advance online publication, 17 September 2004, doi:10.1038/sj.eye.6701647.
2 Soros P, Vo O, Husstedt IW, Evers S, Gerding H. Phantom eye syndrome: its prevalence, phenomenology, and putative mechanisms. *Neurology* 2003; **60**: 1542–1543.
3 Duke-Elder S, Scott GI. Disorders of perception: visual hallucinations. In: Duke-Elder S (ed) *System of Ophthalmology*, Vol. **XII**. Henry Kimpton: London, 1971, pp 562–569.
4 Fernandez A, Lichtshein G, Vieweg WV. The Charles Bonnet syndrome: a review. *J Nerv Ment Dis* 1997; **185**: 195–200.
5 Cogan DG. Visual hallucinations as release phenomena. *Albrecht Von Graefes Arch Klin Exp Ophthalmol* 1973; **188**: 139–150.
6 Burke W. The neural basis of Charles Bonnet hallucinations: a hypothesis. *J Neurol Neurosurg Psychiatry* 2002; **73**: 535–541.
7 Berrios GE, Brook P. The Charles Bonnet syndrome and the problem of visual perceptual disorders in the elderly. *Age Ageing* 1982; **11**: 17–23.
8 Bartlett JEA. A case of organised visual hallucinations in an old man with cataract, and their relationship to the phenomena of the phantom limb. *Brain* 1951; **74**: 363–373.
9 Ramachandran VS, Rogers-Ramachandran D. Synaesthesia in phantom limbs induced with mirrors. *Proc R Soc Lond B Biol Sci* 1996; **263**: 377–386.

AT Toosy, BJ Roberton, H Jayaram and GT Plant

Medical Eye Unit, St Thomas' Hospital, Lambeth Palace Road, London SEI 7EH, UK

Correspondence: GT Plant,
Tel: +44 207 1884301;
Fax: +44 207 1884319.
E-mail: gordon.plant@uclh.org

Sir,
Occlusive retinal vasculitis in a patient with ankylosing spondylitis

Ankylosing spondylitis is a seronegative arthropathy which typically involves sacroiliac joints. We described