necessary in order to have sustained enhanced activation of NMDA receptors leading to the start of neurodegenerative processes.

Therefore, looking for increases in vitreal or retinal glutamate levels in glaucoma or glaucoma models may not be the correct question. What will be more telling and significant is to see whether antagonists of NMDA receptors are effective treatments in such conditions. Experimental work shows some promise in this regard,[5] and the forthcoming clinical trials data with memantine will be of great significance here.

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TE Salt and MF Cordeiro

Institute of Ophthalmology, University College London, London, UK

Correspondence: TE Salt,
Institute of Ophthalmology,
University College London,
11-43 Bath Street,
London EC1V 9EL, UK
Tel: +44 20 7608 6843;
Fax: +44 20 7608 6912.
E-mail: t.salt@ucl.ac.uk

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Sir,

Reply to: Glutamate excitotoxicity in glaucoma: throwing the baby out with the bathwater?

We appreciate the opportunity to respond to the letter by Salt and Cordeiro. We agree that there are less invasive means of continuously measuring the vitreous glutamate level in an experimental model of retinal ischaemia (eg, microdialysis). As discussed in our report,[1] several studies have used such technique to measure the vitreous glutamate level in various animal models of ocular ischaemia. Briefly, one study found gradual elevation of vitreous glutamate following ocular ischaemia to the peak of 6.7 times the preischaemia level in the cat.[2] Another study found only transient elevation of vitreous glutamate up to seven times the preischaemia level in the rabbit.[3] However, a third study failed to show any increase in the vitreous glutamate in a rabbit ocular ischaemia model.[4] Another study not cited in our report monitored retinal glutamate levels continuously in real time using a dialysis electrode in a rat model of ocular ischaemia, and also failed to find glutamate elevation.[5] In fact, the retinal glutamate level decreased during ischaemia, which is consistent with our results (see Table 1[1]). Interestingly, in the same experiment, the authors induced brain ischaemia simultaneously and measured almost five-fold elevation in the brain glutamate level.[5] The authors concluded that slower depletion of ATP in the retina compared to the brain allowed the retina to maintain the physiologic glutamate level and a longer tolerance to ischaemia.

How do we reconcile these disparate results? We have outlined some of the possible reasons in the report.[1] One plausible explanation lies in the ability of the retinal cells' reuptake of released glutamate through glutamate transporter. If there is sufficient reuptake into the neurons and glia during ischaemia, one may not observe elevated glutamate levels. On the other hand, if the ischaemia overwhelms the reuptake mechanism through ATP depletion and depolarization, one may see a rise in glutamate levels. In our primate model of central retinal artery occlusion, ischaemia affects only the inner retina and spares the outer retina and choroid. Such partial retinal ischaemia may allow sufficient reuptake of glutamate through intact functioning of glutamate transporter system in the retina. Indeed, there is evidence that retinal glutamate transporter activity can persist in mild ischaemic conditions *in vitro*.[6]

In our report, we limited the scope of discussion to glutamate excitotoxicity and acute retinal ischaemia. The accompanying editorial by Lotery[7] and the letter by Salt and Cordeiro extend the discussion into glutamate excitotoxicity and *glaucoma*. Unlike the large body of literature supporting the role of glutamate excitotoxicity in acute ischaemia, the role of glutamate excitotoxicity in glaucoma was based on a handful of reports that showed elevation of vitreous glutamate levels in human and animal models of glaucoma.[8,9]

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.

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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

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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