differential affection of muller cell segment in XLR are possible explanations. This suggests that if adequate numbers of K^+ channels are intact, the reflex is absent. As greater numbers of K^+ channels are affected, Mizuo phenomenon becomes demonstrable wherein excessive K^+ ions are cleared during dark adaptation. When most of the K^+ channels are affected, the tapetal reflex becomes constant, as there is persistent K^+ ion backlog. Previous reports of increased tapetal reflex with time[1] suggest progressive K^+ channel damage in XLR. Tapetal reflex disappeared following vitrectomy and posterior hyaloid peeling[5] in XLR. The disruption of K^+ channels at muller cell foot plate during the surgical procedure could have increased the inward conductance of K^+ ions trapped within dysfunctional muller cells causing disappearance of the reflex. The selective reduction of 'b' wave of combined maximal ERG and foveal schisis on OCT confirmed diagnosis of XLR in the absence of peripheral retinoschisis and presence of Mizuo phenomenon.

References

1 Jong PTVM, Zrenner E, van Meel GJ, Keunen JE, van Norren D. Mizuo phenomenon in X-linked Retinoschisis. *Arch Ophthalmol* 1991; **109**: 1104–1108.
2 Mizuo G. A new discovery in dark adaptation in Oguchi's disease. *Acta Soc Ophthalmol Jpn* 1913; **17**: 1148–1150.
3 Newman EA. Membrane physiology of retinal glial (Muller) cells. *J Neurosci* 1985; **5**: 2225–2239.
4 Newman EA. Distribution of potassium conductance in mammalian Muller (glial) cells: A comparative study. *J Neurosci* 1987; **7**: 2423–2432.
5 Miyake Y, Terasaki H. Golden tapetal-like fundus reflex and posterior hyaloid in a patient with X-linked juvenile retinoschisis. *Retina* 1999; **19**: 84–86.

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Sir,

Comment on central retinal artery and vein collapse pressure in giant cell arteritis *vs* non-arteritic anterior ischaemic optic neuropathy

Jonas and Harder[1] measured the central retinal artery and vein collapse pressure in two groups of patients with anterior ischaemic optic neuropathy (AION). The aim of the study was to evaluate the role of these ophthalmodynamometric measures in differentiating an arteritic from a non-arteritic aetiology.

The giant cell AION group entry criteria need some clarification. Did all patients exhibit the classic symptoms of giant cell arteritis (GCA) as well as elevated inflammatory markers and a positive temporal artery biopsy? When a patient presents with an AION and the classical features of GCA, the diagnosis is not usually challenging.

The issue is whether this change in central retinal artery collapse pressure has the same sensitivity in patients where there are no other clinical features of GCA. In occult GCA, the underlying diagnosis of vasculitis of an AION can be challenging[2] and it is in these circumstances where the clinical value of this ophthalmodynamometric test would be useful.

References

1 Jonas JB, Harder B. Central retinal artery and vein collapse pressure in giant cell arteritis versus nonarteritic anterior ischaemic optic neuropathy. *Eye* 2008; **22**: 556–558.
2 Hayreh SS, Podhajsky PA, Zimmerman B. Occult giant cell arteritis: ocular manifestations. *Am J Ophthalmol* 1998; **125**: 521–526.

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Sir,

Reply to Dr Aristodemou

The authors would like to thank Dr Aristodemou for his interest in our study.[1] All patients with giant cell arteritis who were included into the study fulfilled the inclusion criteria for acute arteritic anterior ischaemic optic neuropathy, which were a positive temporal artery biopsy showing the typical granulomatous inflammation with giant cells, an erythrocyte sedimentation rate markedly above the age-related normal value, an elevated C-reactive protein level in the serum, clinical symptoms with a sudden and marked loss in vision, headache often persisting for weeks and months, an acute optic disc swelling with an unsharp and prominent optic disc border, few haemorrhages, and often a rather whitish ischaemic appearance of the swollen neuroretinal rim. The authors agree with Dr Aristodemou that when a patient presents with these typical features of arteritic anterior ischaemic optic neuropathy, the diagnosis is usually not very challenging.

Since the study included only patients with acute arteritic anterior ischaemic optic neuropathy exhibiting the typical ophthalmoscopic and laboratory findings, the authors have unfortunately no ophthalmodynamometric data of patients with occult giant cell arteritis, although without doubt, it would clinically be helpful.[2] As