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

suggested by Dr Aristodemou, it may be the subject of a future study.

References
1 Jonas JB, Harder B. Central retinal artery and vein pressure estimation in giant cell arteritis. *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,

Optical coherence tomography in retinal cavernous haemangioma may explain the mechanism of vitreous haemorrhage

Cavernous haemangioma is a rare cause of vitreous haemorrhage. Optical coherence tomography (OCT) helps explain the mechanism.

Case report

An asymptomatic 12-year-old girl with no relevant ophthalmic, medical, or drug history was referred with a lesion in the retina of the right eye. Best-corrected visual acuities were 6/6 OD, 6/5 OS. Anterior segment examination was unremarkable. There was a cluster of discrete dark red vascular saccules with an overlying grey–white epiretinal membrane in the right nasal retina, consistent with a retinal cavernous haemangioma.

Fluorescein angiography demonstrated slow flow through the saccules. Areas of masking within the saccules were due to thrombosis and hyperfluorescent areas within the saccule were due to pooling of fluorescein. Typical fluid levels (see arrow, Figure 1) within the saccules were seen where thrombus and fluid were present.

OCT elegantly images the lesion in cross section (Figure 2) and may explain why spontaneous vitreous haemorrhage can occur in the absence of a vitreous detachment. An overlying epiretinal membrane is imaged as a continuous hyper-reflective signal attached to the saccules and forming bridges between them (see arrow, Figure 2).

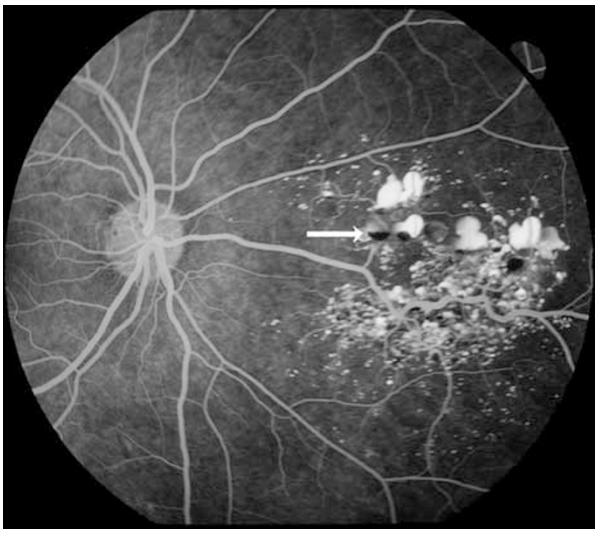

Figure 1 Venous phase of the fluorescein angiogram of the retinal cavernous haemangioma.

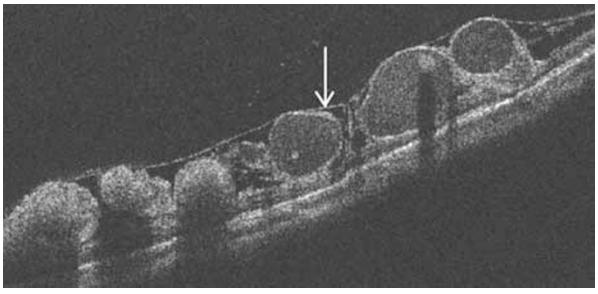

Figure 2 Optical coherence tomography of the retinal cavernous haemangioma.

Comment

Retinal cavernous haemangioma is a rare vascular malformation, it is often unilateral and may present either as an incidental finding or as a cause of vitreous haemorrhage. There may be associated cutaneous lesions and intracranial vascular malformations.[1] An autosomal dominant inheritance has been reported, but cases are usually sporadic.[2]

Histology of cavernous haemangiomas has been reported.[3] The ultrastructurally normal vessel wall maintains the blood retinal barrier, which explains the absence of fluorescein leak. The epiretinal membrane is formed by retinal glial cells, which proliferate on the inner retinal surface after gaining access through breaks in the internal limiting membrane.

Vitreous haemorrhage in retinal cavernous haemangioma is recognised in the absence of trauma and vitreous detachment. Histological studies suggested that these cases of vitreous haemorrhage may be due to epiretinal membrane contraction. The OCT identifies points of attachment to the saccule where a contracting epiretinal membrane could exert traction and cause vitreous haemorrhage.

References
1 Gass JDM. Cavernous hemangioma of the retina: a neuro-oculo- cutaneous syndrome. *Am J Ophthalmol* 1971; **71**: 799–814.