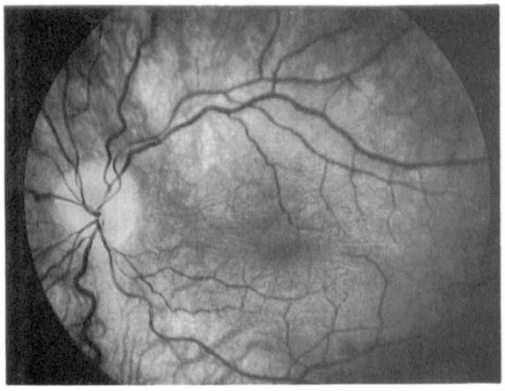

Fig. 3. *The optic disc is now pale.*

tomogram of the orbits and brain. All of these investigations were within normal limits. Visual fields were inconsistent but generally constricted in the left eye with a central scotoma. A pattern reversal visual evoked response (VER) was reduced in amplitude in the left eye as compared to the right (7.2 uv, 12.5 uv). The P100 of the whole field VER from the left eye was spuriously delayed at 132 ms. A half field pattern reversal VER showed that this delay was predominantly due to a dominant contralateral P135, with a reduced ipsilateral P100 (3 uv) occurring at 118 msec. This is a recognised entity in optic neuritis.[2,3] Auditory evoked potentials were normal. Two weeks post presentation her visual acuity had improved to 6/6 right and 6/12 left but with no improvement in colour discrimination. There was resolution of the disc swelling but persistence of the area of retinal whitening. A fluorescein angiogram showed a now perfused retinal arteriole corresponding to the area of retinal whitening (Fig. 2). There was no autofluorescence of the optic disc.

Six weeks post presentation the retinal whitening had completely cleared. Visual acuity was 6/6 right and 6/12 left and there was some improvement in colour discrimination. A relative afferent pupillary defect was still present however, along with pallor of the optic disc (Fig. 3).

Comment

The occurrence of a uveitis and or vasculitis in patients with optic neuritis is well recognised, but as far as we are aware there are no reports of an occlusion of a retinal arteriole in childhood optic neuritis. Retinal artery occlusion is uncommon below the age of 30 years. The cause is often multifactorial and multiple organ systems may be involved.[4] Pre-retinal arterial loops have been associated with retinal artery occlusion in young people[5] and although not a cause by itself, raised intraocular pressure has been found to be a predisposing factor in retinal artery occlusion.[4] The rise in intraocular pressure in this case was presumed to be part of a glaucomatocyclitic crisis and together with the small arteriolar loop may have contributed to the occlusion of the vessel.

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Sir,

Herpes Virus in a Corneal Donor

Although donor to host transmission of infection by corneal transplantation is rare,[1] the number of potentially transmissible agents is large. The Creutzfeldt-Jakob agent, and Hepatitis B and rabies viruses, have all been reported to be transmitted by this surgical procedure.[2] All donors are now screened for antibodies to Hepatitis B and HIV as both viruses have been demonstrated in the cornea and tear film.[3,4] Despite being a common

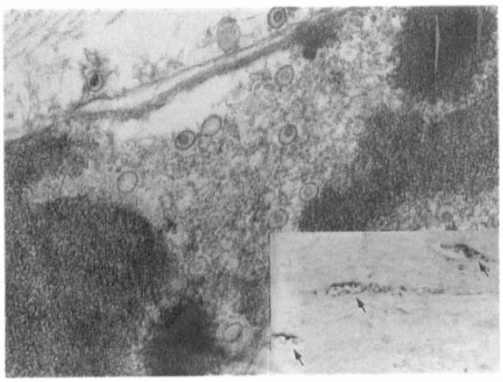

Fig. 1. *Electron micrograph showing herpes simplex viral particles within the nucleus and adjacent to cytoplasm of a keratocyte (×54,000). Inset. HSV 1 nucleic acid revealed by* in situ *hybridisation (×200), arrows. The peripheral localisation of the probe on the inner aspect of nuclear membrane is consistent with active transcription.*

ocular pathogen, we are unaware of any firm clinical evidence to suggest that herpes simplex virus (HSV) has been transmitted in this way. We report the identification of this agent in the keratocytes of a corneal donor.

The donor was a single, fit 42 year old male who was ventilated for eight days after a head injury resulting from a road traffic accident. The eyes were macroscopically normal and showed no obvious abnormality on slit-lamp examination after removal and before placement of the corneo-scleral discs in organ culture medium. Routine bacteriology at eight days in culture failed to reveal any organisms. However, assessment prior to transplantation revealed that both endothelial layers were necrotic or absent. A sample of the storage medium from the left cornea was cultured for virus and both corneas were fixed for microscopic examination. Culture in three separate cell lines revealed typical cytopathic effect typical of herpes simplex type I. Light microscopy of both corneas revealed intranuclear inclusions in a large number of keratocytes. Electron microscopy revealed large numbers of herpes virus particles in and around keratocytes (Fig. 1). *In situ* DNA hybridisation using a probe to herpes simplex type I thymidine kinase revealed widespread localisation of the probe in the nuclei of many stromal cells (Fig. 1, inset). The donor had complement fixation titres against HSV of less than 1/10, but was weakly positive by radioimmunosorbent assay. This report has highlighted the usefulness of organ culture in screening donor tissue for infection. The observation of unexpected endothelial cell loss has been made by several workers in different organ culture eye banks (personal communications) but hitherto HSV type I has not been incriminated. We are currently seeking further evidence to demonstrate that this pathogen is transmissible by corneal transplantation.

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Sir,

Extensive chorioretinopathy associated with very low dose Thioridazine

A case is reported in which a 57 year old woman presented with an extensive form of chorioretinal dystrophy following a short course of low dose thioridazine.

Pigmentary retinopathy is a well recognized side effect of high daily dose thioridazine treatment (above 800 mg/day)[1] and less frequently with lower doses.[1,2] Most cases reported with low daily dose toxicity present with rather mild pigmentary disturbances and some of these patients seem to have been taking the drug for relatively long periods.