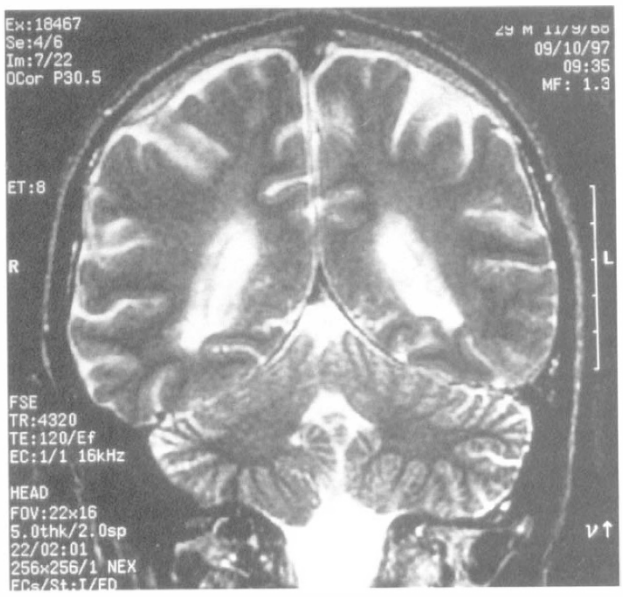

Fig. 2. *Coronal T2-weighted MRI scan of the brain demonstrating extensive plaques in the periventricular white matter.*

Due to the severity of his complaints and his inability to work, the patient was treated with three pulses of intravenous methylprednisolone. Since then his symptoms and visual fields have gradually improved. On examination 1 month after treatment, at rest, the mean deviation of the field in the right eye before exercise was −4.74 dB, and in the left −4.75 dB, falling after 5 minutes' exercise to −12.58 and −10.55 dB respectively.

Comment

Since Uhthoff's original paper in 1889,[1] his symptom has received much attention in the literature. It has been reported to occur in up to half of patients with optic neuritis,[3] and is generally believed to be a direct effect of temperature on conduction in severely demyelinated axons.[4] The symptom has been said to be associated with an increased incidence of recurrent optic neuritis and also of MS.[3] It is also more rarely found in other neurological conditions.[5]

Progressive (non-variable) visual failure is an uncommon but recognised presentation of MS.[6] Reports of MS presenting with intermittent blurring are also very unusual;[2] to our knowledge, however, this is the first reported case where Uhthoff's symptom has occurred as the patient's only problem in such a progressive and debilitating manner. The worsening with time of our patient's symptoms suggests progressive MS (as opposed to relapsing disease); also the occurrence of Uhthoff's symptom has been found to be associated with more extensive demyelination.[2] Progressive disease generally carries a worse prognosis, although in MS sufferers with predominantly sensory disturbance the outlook can be somewhat brighter.[7] Whilst it is possible that the use of high-dose systemic steroids did have some effect on our patient's symptoms, this must be seen in the light of previous evidence that steroids have no influence on the duration or frequency of Uhthoff's phenomenon.[3] Consequently we cannot advocate their routine use in this setting.

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Sir,

Congenital oculomotor palsy associated with brainstem hypoplasia

Oculomotor nerve palsies present at birth are usually thought to be secondary to maldevelopment, intrauterine injury or birth trauma. The association of congenital third nerve palsy with systemic and neurological abnormalities is variously reported in the literature.[1–5] In Miller's series,[1] none of the 13 congenital cases had physical or neurological abnormalities. In the series of Balkan and Hoyt,[2] however, and in the more recent investigations of Hamed[3] and Tsalousmas[4] using modern neuroimaging studies, accompanying neurological or developmental defects are reported to be more frequent and widespread than was once thought. Some cases of congenital oculomotor palsy may therefore be caused by central lesions. We report an 8-month-old female infant with congenital oculomotor nuclear palsy. A magnetic resonance imaging (MRI) scan demonstrated right side brainstem hypoplasia associated with hypoplasia of the corpus callosum.

Case report

A 3200 g term female baby was born to a 38-year-old gravida 2, para 2, woman by uncomplicated spontaneous vaginal delivery. The perinatal history was unremarkable, and there was no drug abuse, teratogen intake or alcohol consumption during pregnancy. The results of amniocentesis were normal, and there was no evidence of trauma during delivery. The other child of the family was quite normal, and the parents are not related.

The baby was brought to the eye clinic at 2 months of age. The ocular examination revealed a large right exotropia of 75 prisms. No blepharoptosis was present in the right eye. The left eye showed a very large angle hypotropia of 50 prism dioptres, and pseudoptosis of the left eye was very prominent (Fig. 1). The infant showed a preferential fixation with her paralytic right eye, with marked turning of her face. The pupils were anisocoric, with the right pupil moderately dilated to 5.5 mm and unreactive to light, and the left pupil 3.5 mm with brisk constriction to light. There was no obvious movement of the right eye, either horizontal nor vertical. The left eye showed only horizontal eye movement, with no vertical movement observed. Since there was no ptosis of the right eye, and given the presence of contralateral hypotropia, it is likely that the entire right oculomotor nucleus was involved, as also were crossing fibres from the right oculomotor subnucleus to the left superior rectus muscle, which caused the contralateral superior rectus paresis.

Neither eye showed signs of aberrant regeneration of the oculomotor nerve; other cranial nerves were intact. However, the infant showed developmental retardation at the following examination. At 8 months of age she could not roll over or sit still, and left limb hemiparesis was found. Cerebral palsy was diagnosed by a paediatric neurologist and she was referred for rehabilitation therapy.

A MRI scan demonstrated striking hypoplasia of the right midbrain. The right-side cerebral peduncle was completely absent. Agenesis of the rostral portion of the corpus callosum was also noted, and the caudal portion of the corpus callosum was found to be rudimentary (Fig. 2).

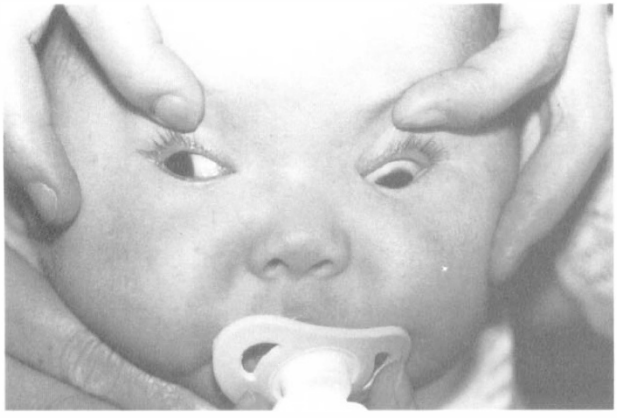

Fig. 1. *A 4-month-old female infant with a large angle right exotropia and elevation of left eyelid disclosing prominent left hypotropia.*

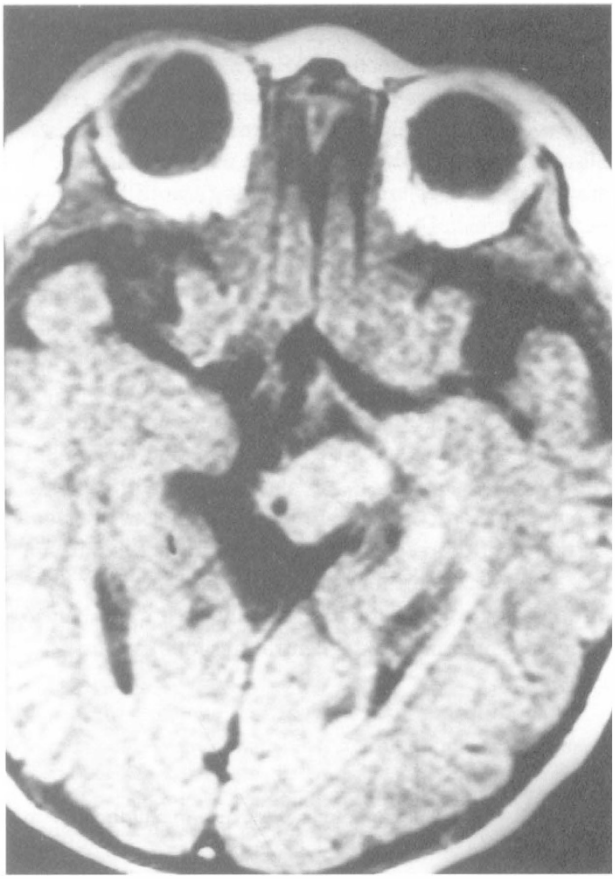

Fig. 2. *T1-weighted axial MRI scan demonstrated striking right midbrain hypoplasia and complete absence of the right cerebral peduncle. Note the extreme exodeviation of the right globe.*

Comment

According to Walsh and Hoyt's hypothesis,[6] the mechanism of congenital oculomotor palsy is birth injury, with traction and tearing of the tentorium near the attachment to the posterior clinoid process. Harley[7] and Miller[1] believe that the congenital lesion is generally indicative of developmental anomaly or birth trauma. The consensus of many early reports is that birth trauma is the main cause of congenital oculomotor palsy, and it was seldom considered to be associated with other neurological or systemic disease. Recent advances in neuroradiology, however, have improved our ability to detect previous occult lesions. In Hamed's series,[3] 10 of 14 patients with congenital oculomotor palsy had associated neurological disorders, including some brainstem lesions. Balkan and Hoyt[2] described 10 patients, 4 of whom had associated focal neurological deficits: three with Weber's syndrome and one with monoplegia. In Tsaloumas's report,[4] 5 of 14 patients had specific neurological abnormalities that indicated brainstem disorders. Imaging studies were abnormal in 5 of 10, including abnormalities of the brainstem or cerebellum, midline midbrain infarctions, etc. Thus a central brainstem lesion was considered to be a

contributing factor in many of the recently reported cases. The high incidence of associated neurological abnormalities with congenital oculomotor palsies suggests a possible role for intrauterine brainstem injury.

Embryologically, the oculomotor nerve, nuclei and extraocular muscles are formed during the fifth gestational week. During the prenatal period the brain is susceptible to a variety of insults, including infections, teratogens and hypoxia. Findings in the present case suggest that the defect was intra-axial and that there was extensive brainstem damage, probably caused by a vascular insult during the early gestational period. As the use of newer neuroimaging techniques has increased, recent investigations have demonstrated that congenital oculomotor nerve palsy is often associated with diverse and sometimes profound neurological abnormalities.

When a child presents with oculomotor nerve palsy at birth, a detailed neurological examination and neuroradiological investigation should be undertaken to look for possible intra-axial central nervous system involvement.

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Sir,

Transient fogging of acrylic (Acrysof) intraocular lenses

The benefits of phacoemulsification cataract surgery are enhanced by the use of foldable intraocular lenses (IOL) and smaller incisions. In terms of their chemical components, the foldable lenses can be divided into two groups, namely acrylate/methacrylate polymers and silicone elastomers. Minor alterations to the side-chain components of the acrylate/methacrylate polymer backbone result in a wide variety of lenses which differ greatly in terms of their physical and biological properties.[1,2]

The AcrySof IOL (MA30BA, Alcon Labs., Texas) is made of phenylethyl acrylate and phenylethyl methacrylate co-polymers cross-bonded with butanediol acrylate. This material is capable of being folded prior to insertion.[3] Although the lens is well tolerated, reported complications associated with its use include transient marks caused by folding,[2] scratches on the lens optic,[4] stress fractures[5] and post-operative glistenings.[6] The lens can be implanted through an incision with an internal diameter exceeding 3.5 mm,[3] although warming the lens may facilitate folding and allow for introduction through a smaller incision.[7] In this unit, our previous policy was to warm the lens in a heating cupboard before use to facilitate implantation. The temperature of the heating cupboard has been recorded at 47°C. We have recently encountered two cases of transient fogging of the AcrySof IOL which we believe were related to this practice. One of these cases is illustrated here.

Case report

Phacoemulsification cataract surgery was performed on the right eye of a 78-year-old woman with a history of primary open angle glaucoma. Capsulorrhexis, hydrodissection, phacoemulsification and irrigation/aspiration were performed through a 3.5 mm corneal incision and a paracentesis without complication using ProVisc (Alcon Labs., Texas) and the Alcon Legacy 20000 Phaco-emulsifier (Alcon Labs., Texas). A 24 D AcrySof lens (Model MA30BA) was warmed prior to use and folded with the manufacturer-recommended forceps. Prior to folding the lens was inspected and noted to be clear. No irrigating solutions were used to rinse the lens before implantation. After unfolding within the capsular bag, the lens optic was noted to be semi-opaque (Fig. 1). This feature failed to clear after a period of 5 min. The lens was then dislocated into the anterior chamber,

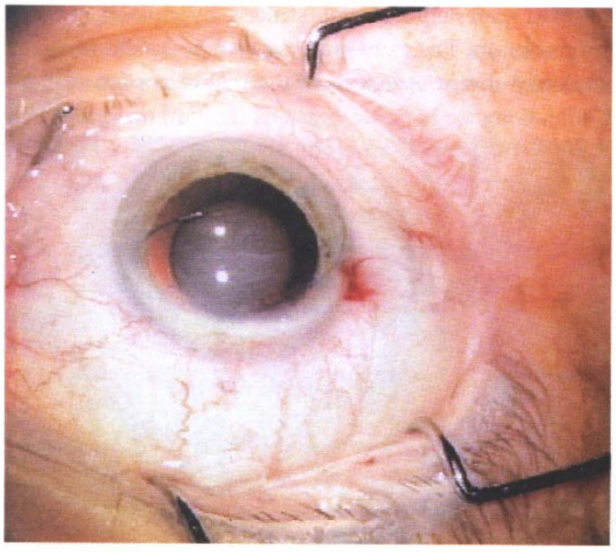

Fig. 1. *The lens has been dislocated into the anterior chamber prior to removal. The fogging of the optic is clearly demonstrated.*