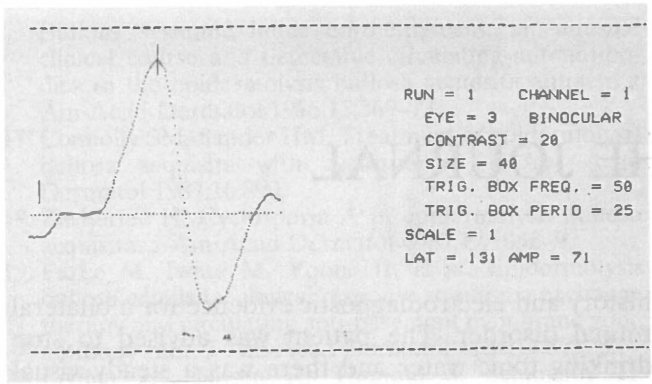

Fig. 2. *Pattern ERG showing abnormally delayed transient visual evoked responses.*

recovery following its withdrawal. There were abnormal electrodiagnostic features possibly indicating outer retinal disease, but no evidence for alcoholic or nutritional amblyopia. The macular abnormalities, possibly at the level of the retinal pigment epithelium (RPE), may represent deposition of quinine. Like chloroquine, quinine binds to uveal tissue *in vitro*[4] and Dekking[5] describes pigmentary abnormalities in a case of acute quinine poisoning.

A carefully documented case[1] of acute quinine toxicity found evidence for an acute direct effect upon the photoreceptor cell layer and outer retina. The decreased light EOG rise in acute poisoning has suggested involvement at the RPE/photoreceptor level, while the initially normal flash ERG implied damage to the ganglion cell layer. Recent case reports[6,7] which review the literature have suggested electrophysiological evidence for damage in all retinal layers. Quinine appears to have deleterious effects at both the RPE/photoreceptor interface (low EOG) and inner retina, possibly at the level of bipolar cells (decreased or absent ERG b wave). In chronic toxicity there appears to be a similar although reversible sequence of effects. We are presuming the macula to be the most sensitive to chronic toxicity as indicated by the abnormal pattern ERG N95 wave and presence of pigmentary macular features. Peripheral retina appears unaffected as shown by the normal flash ERG and EOG (whole retinal responses).

Other causes for visual loss were considered. Tobacco–alcohol amblyopia was unlikely in the absence of measurable plasma alcohol and with normal liver function. Demyelination is unusual in this age group and bilateral optic neuritis extremely rare. Additionally, we felt the time course of blindness was unusually protracted for demyelination and to date there have been no further episodes of neurological deficit.

We conclude that an investigation of blindness must include a careful dietary history and that idiosyncratic sensitivity may occur to commonly well-tolerated substances including quinine.

The authors acknowledge the expert advice provided by Mr John Kelsey, FRCS, Electrodiagnostic Department, Moorfields Eye Hospital.

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Sir,

Argon and YAG Lasers in the Treatment of Ophthalmia Nodosa

We recently published a report on the management of intraocular caterpillar hairs (setae).[1] It has long been concluded that hair removal is of great importance,[2] but this may be difficult and is not without its own complications. We wished to investigate an alternative theory aimed at immobilising the hairs. Samples of the setae removed from the patient were disrupted *in vitro* with argon and YAG lasers and then examined under the electron microscope. Major alteration of the hair structure, likely to impair their continued migration, was seen after treatment with both lasers.

Method

As previously reported, a 15-year-old boy had a live

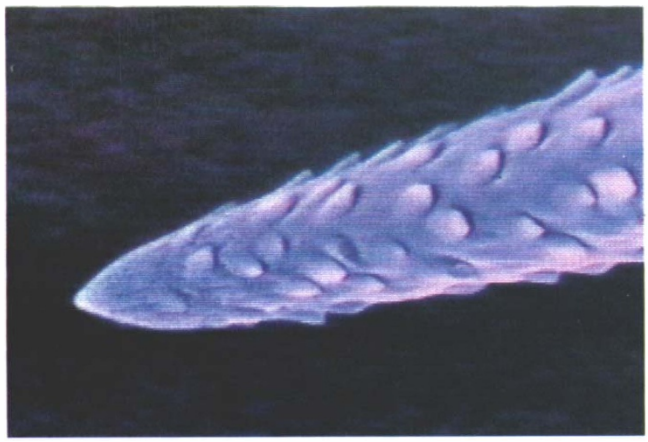

Fig. 1. *Electron micrograph of seta removed from the eye.*

caterpillar rubbed in his eye.[1] Initial treatment consisted of removal of superficial hairs, but later he required anterior chamber setae removal and subsequently underwent a vitrectomy after relentless penetration of the hairs into the posterior segment.

A number of the initial hairs that were removed undamaged were retained and divided into two groups. The first group were processed and examined under the electron microscope (Fig. 1). The remaining complete hairs were fixed to a microscope slide with clear tape and suspended from the forehead guard on the YAG and argon lasers. The hairs were then lasered, with the power being increased until a break in continuity of the hair was witnessed. The hairs were examined under the electron microscope on the same day. The resulting pictures are shown in Fig. 2 (argon laser) and Fig. 3 (YAG laser).

Discussion

In a previous paper we discussed the role of vitrectomy in the management of intraocular caterpillar hairs (setae).[1] It would, however, be preferable to avoid such invasive treatment. Once the setae have penetrated the cornea or sclera they

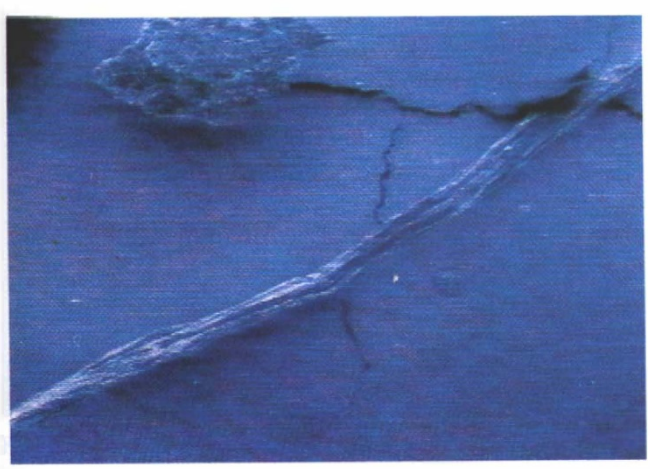

Fig. 2. *Electron micrograph of seta after argon laser.*

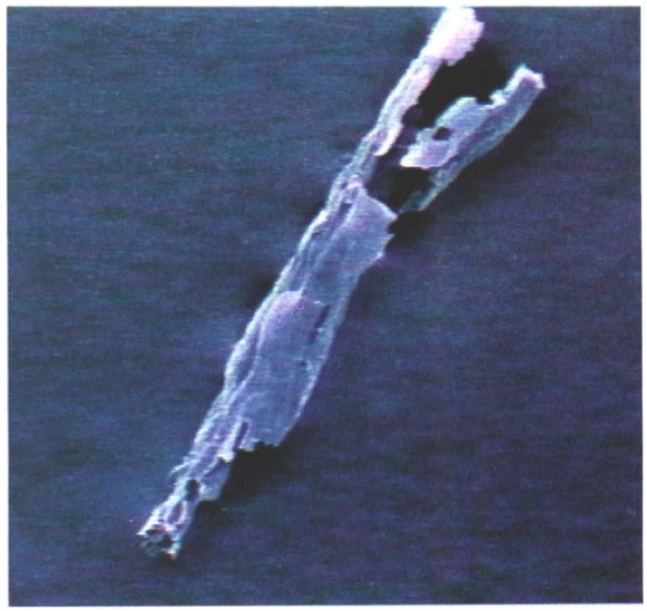

Fig. 3. *Electron micrograph of seta after YAG laser.*

have the potential to damage any intraocular structure; the great problem with the hairs is their remarkable ability to penetrate through the eye. Almost any serious ocular complication is possible including vitreal hairs,[2] retinal hairs,[1] subretinal hairs,[2] choroidal hairs,[3] optic nerve hairs,[4] iridectomy,[5] endophthalmitis[6,7] and enucleation.[7–9]

Because of these potential problems meticulous hair removal is the essence of treatment, but of course has its own hazards. Removing the hairs increases the trauma to an already insulted eye and anterior segment manipulation increases the risk of corneal endothelial loss, iris damage and cataract. Posterior segment surgery has the risk of retinal damage/detachment and cystoid macular oedema. Any surgery exposes the eye to the risk of infection and of course there is the risk from the general anaesthetic.

Both action and observation can put the eye at risk, but if the setae could be altered to retard their penetration, then this risk may be reduced. There have been attempts at this by severing the tip of the hair from its shaft with a sharp blade while in the cornea.[2] Similarly our *in vitro* pictures show very marked disruption of the hairs by the lasers and, most importantly, show loss of the tip and reverse barbs – which are vital factors in continued penetration of hairs.[8] In theory the setae could be lasered anywhere in the eye: YAG laser could be used for iris or anterior vitreous hairs, while argon laser could be used on the sclera, iris or retina. As far as the cornea is concerned, collateral damage from the laser would be likely and direct rémoval may be safer in this site.

It is of course a big step from our experiments to

actual lasering of setae *in vivo*. We are aware of this being attempted only once, and this involved vitreal hairs being treated with the argon laser.[10] It was noted that there was a subsequent reduction in vitreous activity immediately after treatment.

When the setae have entered the eye with some force, the risks of penetration are high[1,2,8] and subsequent damage so potentially serious that prophylactic lasering may be justified.

We are grateful to Dr H. K. McClelland for help with the translation of the references.

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Sir,

The Eye and Adenocarcinoma of the Breast: Metastases and Meningiomas

There have now been a number of reports highlighting the association between adenocarcinoma of the breast and meningioma.[1–3] Breast cancer is the commonest fatal malignant neoplasm of women,[4] and meningioma is one of the commoner intracranial tumours, accounting for 19% of all central nervous system tumours.[2] Carcinoma of the breast is also the second commonest source of intracranial metastases (after carcinoma of the bronchus)[3] and the most frequent source of choroidal metastases.[5,6] We present a patient with advanced carcinoma of the breast whose signs of intracranial mass with cranial nerve involvement were initially ascribed to a metastasis but subsequently found to be due to a meningioma. Detailed ophthalmic examination revealed the presence of an associated choroidal metastasis.

Case Report

A 71-year-old woman with known disseminated adenocarcinoma of the breast was referred to the eye unit by the radiotherapy department with a short history of diplopia. She had initially presented 5 years earlier with a large fungating tumour of the right breast and had subsequently received several courses of chemotherapy and radiotherapy for her metastatic disease, which included multiple bony secondaries and malignant pleural and pericardial effusions.

Her initial symptoms consisted of migrainous headaches in the morning, 'trouble focusing' and photopsia in the left eye. Two weeks later she developed diplopia on upgaze and was referred for ophthalmic assessment. Her vision was 6/6 in both eyes but she had a mild left ptosis, an enlarged and unresponsive left pupil and a small left hypotropia (7Δ) and exotropia (5Δ). Elevation of the left eye was limited, particularly in abduction. A Hess chart confirmed this and also revealed a small limitation of adduction supporting the clinical diagnosis of a partial third nerve palsy (Fig. 1). On dilated funduscopy, a pale elevated lesion approximately 3 disc diameters in size with indistinct borders was noted inferonasal to the optic disc in the left eye (Fig. 2). Fluorescein angiography (Fig. 3) showed a diffuse leak and this lesion was thought to be a choroidal metastatic deposit with an overlying serous retinal detachment.

An initial computed tomography (CT) scan of the head revealed a suspicious area adjacent to the pituitary which was thought to represent a secondary deposit. However, whilst the bone scan showed secondaries in the vault, there were no 'hot-spots' in the orbit or base of the skull. Therefore, a repeat CT scan with contrast was performed which showed