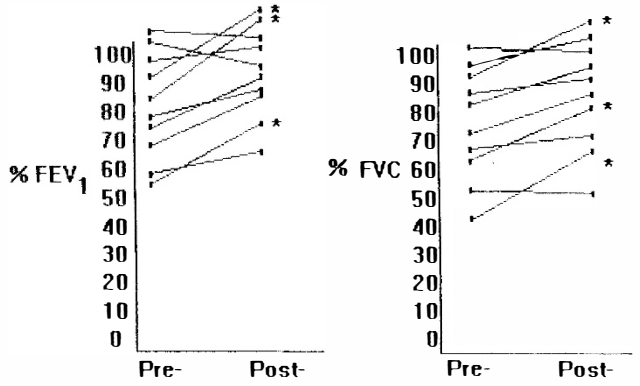

Fig. 1. *Percentage change from predicted values of FEV_1 and FVC in 10 patients before and after nebulised ipratroprium bromide. *>15% change.*

atory clinic at the Royal Hallamshire Hospital showed an increased FVC but no change in FEV_1 30 minutes after nebulised ipratropium bromide (Fig. 1; unpublished data).

Diggory *et al.*[1] have noted changes in respiratory function which warrant further investigation. Their strong claim regarding possible detrimental effects to patients using timolol, and the suggestion that timolol is no longer acceptable to use in the elderly, cannot be sustained from this study.

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Sir,

In their article 'Outpatient management of small traumatic hyphaemas: is it safe?', Williams *et al.*[1] concluded that outpatient management did not increase the rate of complications and was therefore safe. Whilst the authors quite correctly stated that their conclusion applied to Caucasian patients, they did not elaborate on the reasons for this. We think it is important to stress that any black patient with hyphaema, microscopic or otherwise, must have haemoglobin electrophoresis to exclude sickle cell disease or trait. Outpatient management is not appropriate in such patients.

A 13-year-old Ghanaian boy was recently admitted to this unit with a small hyphaema in the left eye following blunt trauma. Funduscopy was normal with no evidence of commotio retinae and the intraocular pressure (IOP) was 18 mmHg. The following day his IOP was markedly raised and the visual acuity had fallen to 6/18. The IOP failed to respond to topical ocular antihypertensives and acetazolamide and eventually required osmotic therapy with oral glycerol. His IOP ranged from 38 to 53 mmHg for 2 days before returning to normal levels. He was found to have sickle cell trait (Hb AS) on haemoglobin electrophoresis with a percentage of Hb S of 38%. Six days later he was noted to have scattered retinal haemorrhages in the mid-periphery of the left eye, and white 'ghost' vessels extending peripherally from the equator. The optic disc was normal, in particular showing no signs of pallor or glaucomatous cupping. The haemorrhages resolved but extensive occlusion of the peripheral retinal vessels remained, and this was confirmed by fluorescein angiography (Fig. 1). The visual acuity was 6/5 in both eyes 8 weeks after the episode and his other eye was normal on angiography.

Although complications such as raised IOP following blunt trauma have been well described in patients with sickle cell disease, there have been few reports of such problems in sickle cell trait, which is traditionally considered a benign condition.[2,3] In addition, most patients with sickle cell or Hb SC disease are aware of their diagnosis, whereas many patients with sickle cell trait are not.

There have been four reports of central retinal artery occlusion in patients with sickle cell trait with traumatic hyphaema and raised IOP.[3–6] In one case the occlusion immediately followed mannitol administration.[6] Three of

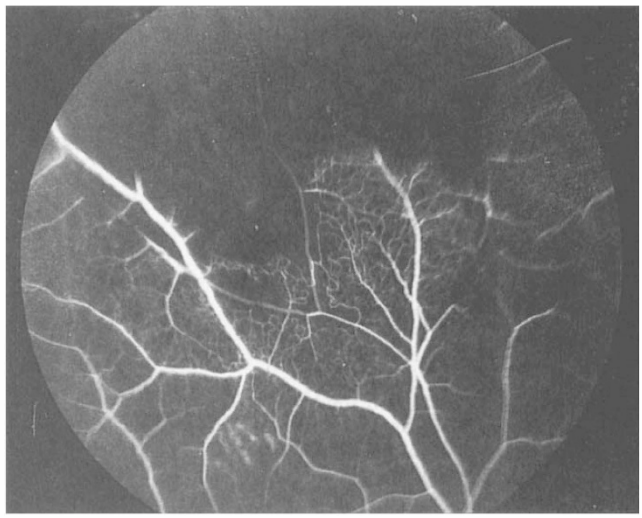

Fig. 1. *Fluorescein angiogram demonstrating occluded vessels in the superotemporal retina of the left eye.*

these patients underwent prompt anterior chamber paracentesis with reversal of the occlusion and some visual recovery.[3,4,6] Crouch and Frenkel[7] observed 27 patients with traumatic hyphaemas. Two patients developed optic atrophy although their final visual acuity is not reported; both had sickle cell trait. In one the IOP had ranged from 35 to 39 mmHg for 2 days, and in the other it varied between 35 and 40 mmHg for 4 days.

It has been suggested that flow in the central retinal artery may be seriously impaired at IOP levels of over 40 mmHg in patients with a sickle haemoglobinopathy.[6] However, the level of IOP deemed 'safe' in the sickle haemoglobinopathies has been suggested to be as low as 27 mmHg, as the microvasculature of the optic nerve head and macula may be susceptible to much lower pressures.[2,7] In the case described here, it seems likely that occlusion of the peripheral retinal vessels was due to the effect of a raised IOP on a susceptible vasculature. It has been suggested that treatment with acetazolamide and osmotic agents must be approached with caution as they may produce conditions likely to increase sickling by increasing haemoconcentration and viscosity in an already compromised vasculature.[2,6] Acetazolamide may also cause systemic acidosis, further encouraging the sickling process.[2] Because of this, anterior chamber paracentesis has been recommended as the safest method of lowering the IOP in these patients.[2–6]

It is essential to exclude sickle cell disease or trait in any black patient with traumatic hyphaema, particularly as the IOP may not be raised at presentation, and the patient may be unaware of his haemoglobinopathy. A modest increase in IOP may cause damage to the retinal vasculature in such cases. If haemoglobin electrophoresis reveals any abnormality then outpatient management is not appropriate.

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Sir

Hay *et al.*[1] reported the successful treatment of two patients with *Acanthamoeba* keratitis using a combination of topical chlorhexidine and propamidine isethionate (Brolene, M&B). We wish to report the successful treatment of a further patient with the same therapeutic regime.

The patient, a 31-year-old woman, first presented to an ophthalmologist in November 1993 with a 5 day history of a sore left eye. The patient was an intermittent soft contact lens wearer (Igel daily wear soft contact lens, 37% water content), with a wearing time of approximately 4 hours once or twice a week for 5 years. The patient had initially been using a hydrogen peroxide based cleaning system (Allergan, Oxysept), but 3 months prior to the onset of symptoms had changed to a chlorine based system (Alcon, Softabs), used in accordance with the manufacturer's instructions.

At presentation corrected visual acuity was 6/5 and a localised area of superficial stromal infiltrate was noted with initially no overlying epithelial defect. Initial treatment with a variety of topical gentamicin, chloramphenicol, acyclovir and prednisolone (Predsol, Glaxo, 0.5% t.d.s. for 3 weeks) was unsuccessful with an enlarging stromal infiltrate and overlying epithelial defect.

The patient was referred to the External Eye Disease Clinic at St James's University Hospital 9 weeks following the onset of symptoms. Examination revealed limbitis, subepithelial and anterior stromal infiltration, overlying frank epithelial ulceration, perineural infiltrates, and a mild anterior chamber reaction. The patient was experiencing severe pain from the involved eye. A clinical diagnosis of *Acanthamoeba* keratitis was made and all treatment stopped prior to an epithelial scrape and stromal biopsy. Trophozoites with vacuoles were identified on binocular microscopy of the epithelial sheet and subsequently culture-confirmed as *Acanthamoeba* sp. The isolates grew well at 35 °C. The drug sensitivities of the isolates are listed in Table I. No evidence of *Acanthamoeba* was found from the stromal biopsy. In accordance with the classification suggested by Bacon *et al.*[2] this case would be categorised as a late diagnosis.

Table I. Drug sensitivities of the patient's corneal isolate

Drug (μg/ml)	MTAC[a]	MCC[a]
Chlorhexidine	3.2	6.3
PHMB	3.2	12.5
Neomycin	12.5	50.0
Brolene	6.3	12.5
Pentamidine	12.5	25.0
Acyclovir	100.0	100.0

MTAC, minimum trophozoite amoebacidal concentration; MCC, minimum cysticidal concentration; PHMB, Polyhexamethylene biguanide.
Courtesy of the Tennent Institute of Ophthalmology, Glasgow.