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.

Treatment was initiated following the biopsy with topical chlorhexidine 0.02% and propamidine isethionate at half-hourly intervals. In addition topical neomycin was used for a period of 4 days and subsequently discontinued.

A response to treatment was noted within 72 hours of commencing treatment with relief of pain, complete epithelial cover and break-up of the perineural infiltrates. Treatment was reduced to hourly drops after 4 days and to drops 6 times per day after 3 weeks. Two weeks following the commencement of treatment the patient developed an area of stromal oedema with underlying keratic precipitates in the inferior third of the cornea and a mild anterior chamber reaction. Topical antiamoebal therapy was continued and the use of topical steroids avoided. The stromal reaction resolved completely over 3 weeks. Currently the patient's corrected visual acuity is 6/4, there has been complete resolution of pain and inflammation, and the cornea is clear with a faint nebular opacity at the site of the peripheral stromal biopsy only. The patient is currently on treatment with 6 times per day chlorhexidine and propamidine.

We therefore suggest that this case report provides supportive evidence that chlorhexidine combined with propamidine isethionate offers an effective and well-tolerated first-line treatment for *Acanthamoeba* keratitis.

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Sir,

We are pleased that Richardson and Waterhouse[1] have shown interest in the report of our study,[2] but feel their comments confuse what is a simple message. We studied a group of 52 asymptomatic patients over 65 years of age, with no history of obstructive airways disease, who were using topical timolol. We found an increase in mean peak flow from 278 l/min to 328 l/min ($p<0.001$), in mean FEV_1 from 1.66 l to 1.85 l ($p<0.001$) and in mean FVC from 2.40 l to 2.64 l ($p<0.001$) when topical timolol therapy was changed to betaxolol or pilocarpine. Twenty controls showed no significant change in spirometry.

The most important finding was that 19 of 47 patients who changed therapy and completed the study, improved peak flow and FEV_1 by more than 15%. These patients demonstrated reversible airflow tract obstruction.[3] Non-selective beta antagonists are contraindicated in such patients, even if they are asymptomatic, because they risk developing severe bronchospasm, especially if a chest infection supervenes.

The suggestion that only those with a FVC over 2 litres can be included implies that elderly people with poor lung function should not be the subject of study.

Spirometry values depend upon age, height and sex as well as respiratory disease. There are well-validated predicted norms available for people up to 65 years but there is controversy as to reliability and reproducibility of norms in very elderly people. The American Thoracic Society advises caution when using predicted norms in the elderly.[4] Studies have demonstrated differences in predicted values as great as 20% for elderly people using different reference equations for spirometry.[5] We recorded the patients' heights, but decided not to express the results as a percentage of a predicted value because so many of the subjects enrolled in the study were very old [42 (58%) were over 75 and 11 (15%) over 85 years].

We tested for significance of changes in mean values between enrolment and review in treatment change and control groups separately and found significant change only in the treatment change group. Age and height are not important as we are looking at differences within groups.

Having a larger proportion of males in the treatment change group does mean slightly larger absolute values of spirometry values. This may exaggerate the magnitude of changes when the treatment change group is compared with the control. We feel that since spirometry of the controls hardly changed at all and the change in the treatment group was so large we can stand by our figures.

In patients with airways obstruction the amount of air that can be expelled in the first second (FEV_1) falls, but the total volume that can be expelled (FVC) is relatively preserved, and a low FEV_1/FVC ratio results. FVC is the most commonly used measure of vital capacity but may not be appropriate in those with emphysema. Emphysematous people have air-trapping which reduces FVC. However, if they are asked to expel air gently, to give the so-called relaxed vital capacity (RVC), there is less air-trapping and a more accurate measure of vital capacity is obtained. We regarded FEV_1 and peak flow as the most important measurements. Our spirometer recorded peak flow, FEV_1 and FVC on a single breath; a RVC would have required a separate recording.

We were surprised by the increase in mean FVC that we found. The most obvious explanation is a learning effect with the spirometer, but one would then expect the same change in FVC to be seen in the control group – which it was not. Perhaps when the airways obstruction is reduced, by changing therapy, the intrathoracic pressures generated in a forced expiration may be lower, resulting in less air-trapping and an increase in FVC. We can only report the results we obtained.

Our spirometer was calibrated to an accuracy of ±2%. We note the tolerance of the Wright Mini Peak Flow Meter of 2–12%, but we did not use such an instrument.