LETTERS TO THE EDITOR

Sir

8 September 1994 marks the one hundredth anniversary of the death (from a cerebral haemorrhage) of the German physiologist and physicist Hermann Ludwig Ferdinand von Helmholtz (1821–1894). This anniversary has already been marked by the scientific community[1] but also merits the attention of clinicians, and particularly ophthalmologists, for Helmholtz's influence on medical practice is chiefly remembered for his invention of the ophthalmoscope.

Although pre-eminent as a physicist, Helmholtz's first training was in medicine at the army medical school in Berlin, and thereafter he served as a surgeon in the Prussian army. The invention of the ophthalmoscope, and hence the direct method of ophthalmoscopy, occurred during his subsequent tenure of the chair of physiology in Königsberg. Based on principles similar to Galileo's telescope, familiar to Helmholtz from his seminal work on physiological optics, the first report of the *Augenspiegel* ('eye mirror') was presented to the Physikalische Gesellschaft in Berlin on 6 December 1850, and published the following year.[2] This instrument, apparently designed to demonstrate to students how light is reflected from the retina, permitted physicians to observe the ocular media by transillumination through the pupil, investigation of the eye prior to this being limited to visual inspection with or without a magnifying glass. Many changes and improvements were subsequently incorporated in the ophthalmoscope to produce the instrument familiar today.[3]

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Sir,

Whilst interesting changes in respiratory function were reported by Diggory *et al.*[1] in the patients changed from timolol, we wish to express concern about the way in which the study was performed and the conclusions and recommendations drawn, in particular the assumptions made regarding the grouping of the patients, the data obtained and the choice of equipment.

Using the mean values obtained from a group of patients with a wide range of ages and of unspecified distributions of height and smokers between males and females is not appropriate. The results obtained from each patient should be compared with the predicted values available from internationally accepted tables according to the individual's sex, height, age and race and whether they are smokers or not.[2] The minimum volume of expired air during the FVC should be 2 litres to reduce the errors in recording low volumes. The results should be expressed as a percentage change from predicted values and the mean percentage change used to assess any change in therapy.

The treatment group in the paper by Diggory *et al.*[1] had a greater proportion of males than the control group, who are more likely to be taller and to have obstructive airways disease of a more severe nature.[3] In the group changed to betaxolol or pilocarpine, the increase in FVC is difficult to explain in terms of the effect of beta-blockade causing bronchoconstriction of the smaller bronchi. Patients with emphysema have 'air-trapping' on forced expiration which decreases the FVC and which may increase if timolol has an effect other than bronchoconstriction on the more distal airways.

The recommendations regarding the apparatus for respiratory function suggest the spirometer should be calibrated daily to a tolerance of ±2% for both volume and timing measurements.[2] The Wright Mini Peak Flow Meter[4] has a tolerance of between 2% and 12%, and so whilst useful for the clinical situation of monitoring patients' progress, it is not the best choice for basic respiratory function investigations.

If the data are presented as the percentage change in individual patients from predicted values in peak flow, FEV_1 and FVC before and after stopping timolol, a subgroup of patients responding in an unusual manner could be identified. For example, some patients with known chronic obstructive airways disease attending a respir-

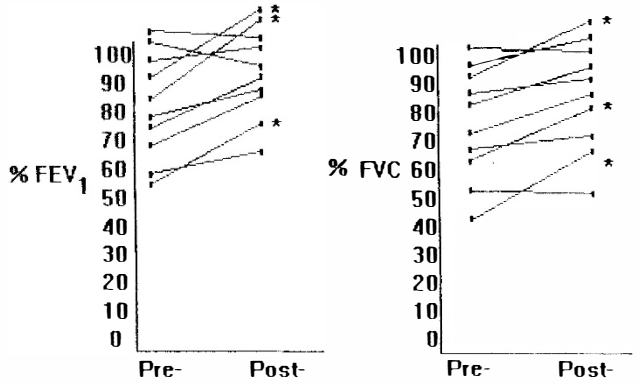

Fig. 1. *Percentage change from predicted values of FEV_1 and FVC in 10 patients before and after nebulised ipratroprium bromide. *>15% change.*

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

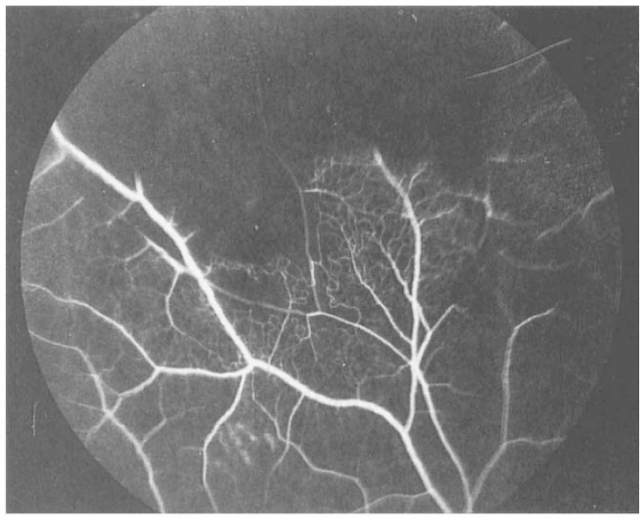

Fig. 1. *Fluorescein angiogram demonstrating occluded vessels in the superotemporal retina of the left eye.*