CORRESPONDENCE

Natural history of recurrent erosion syndrome

EDITOR,—I read with interest the recent article by Heyworth and coworkers.¹

In their paper there is no specific mention of, or discussion of, the role of oral tetracycline in the treatment of recurrent corneal erosions. In their results they state that 55% of patients who were symptomatic were taking some form of treatment, which included topical drops or ointment. They do not mention whether any patients were using systemic treatment in the form of oral tetracycline or any other systemic treatment. In the discussion there is no reference to the possible role of oral tetracycline in the management of these patients. They refer to topical treatment and possible surgical treatments. They also do not indicate whether any of the patients in the review are using a bandage contact lens to manage their symptoms. In their introduction they state there have been no randomised controlled trials of treatment in the management of recurrent corneal erosion. This in fact is incorrect and our paper in 1994 on the role of oral tetracycline in the treatment of recurrent corneal erosions reported the results of a prospective randomised controlled trial.² Continued clinical experience since then strongly supports the use of oral tetracycline in the treatment of recurrent corneal erosions, where the symptoms are not controlled by simple lubricating treatment.

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- 1 Heyworth P, Morlet N, Rayner S, *et al*. Natural history of recurrent erosion syndrome—a 4 year review of 117 patients. *Br J Ophthalmol* 1998;82:26-8.
- 2 Hope-Ross MW, Chell PB, Kervick GN, *et al*. Oral tetracycline in the treatment of recurrent corneal erosions. *Eye* 1994;8 Part 4:384-8.

Reply

EDITOR,—McDonnell is correct in stating that no mention was made regarding the role of systemic tetracycline in the management of recurrent corneal erosions. Our study¹ looked at a group of patients who were initially treated for recurrent corneal erosions using topical lubricants. This group of patients was recruited over 5 years ago before the results of Hope-Ross and McDonnell's valuable study were known.² Our study was a telephone questionnaire which was not investigating different treatment modalities in the management of recurrent erosions but merely reporting the symptomatology of a well documented group 4 years after the original study. We can say with some certainty that among those who were using some form of treatment those who were self medicating (67%) were not taking systemic tetracycline, but with regard to the remainder it is unlikely that those attending their general practitioner (26%) were taking systemic tetracycline. We do not have the data available for the remaining two patients who were continuing to attend an ophthalmologist. Although we accept Hope-Ross and McDonnell's study findings our continued clinical

experience with tetracycline has been less encouraging than theirs.

Only two patients from this study continue to attend an ophthalmologist. One had certainly used a bandage contact lens in the past but had subsequently undergone phototherapeutic keratectomy and was still symptomatic, although to a lesser degree. Bandage contact lenses certainly have a role in the management of recurrent erosions in patients who are sufficiently troubled by symptoms to seek medical advice—something that the vast majority of these patients were not doing.

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- 1 Heyworth P, Morlet N, Rayner S, *et al*. Natural history of recurrent erosion syndrome—a 4 year review of 117 patients. *Br J Ophthalmol* 1998;82:26-8.
- 2 Hope-Ross MW, Chell PB, Kervick GN, *et al*. Oral tetracycline in the treatment of recurrent corneal erosions. *Eye* 1994;8:384-8.

Topical ointment does not prevent recurrent symptoms following traumatic corneal abrasion

EDITOR,—We would like to expand on comments by Heyworth *et al*¹ regarding the use of topical lubricating ointment for prophylaxis of symptoms of recurrent corneal erosion.

The authors make the point that there are very few available data as to the prevalence of symptoms following traumatic corneal abrasion. We have addressed this specific issue in an ongoing prospective study. The study was designed to assess symptoms following uncomplicated traumatic corneal abrasion, and to assess the effect on symptoms of using a topical lubricant ointment. The project has the approval of our local research ethics committee.

All patients presenting with traumatic corneal abrasion in a previously healthy eye were treated with our standard regimen of cyclopentolate ointment 1% immediately and chloramphenicol eye drops four times daily for 5 days; eyepads were not used.² Injuries caused by a fingernail are felt to be at higher risk of progressing to recurrent corneal erosion,³ so these patients were randomised to one of two treatment groups. One group received our "standard regimen" alone, the other group continued with a "prophylactic regimen" of lubricating ointment⁴ (Lacrilube, Allergan) at night for 2 months. Patients were followed up after 3 months by telephone, using a symptom based questionnaire. Recurrent symptoms were graded as: (i) none or minimal, (ii) mild, (iii) moderate (difficulty with some daily activities, or sought further

advice from a health professional), and (iv) severe (macroform⁵ recurrent erosion). Case notes were reviewed at 2 years. Three year follow up, again by telephone questionnaire, is due to take place later this year.

Seventy four patients completed the 3 month follow up questionnaire (Table 1). Symptoms were considered to be due to recurrent corneal erosion if they were reported as frequent and significant pain, grittiness, photophobia, or watering³ of the injured eye only. A total of 21 patients (28%) reported such recurrent symptoms at 3 month follow up. We found no significant difference in symptom prevalence between "fingernail" and "non-fingernail" injuries which had been managed with our standard regimen (χ^2 test: $p = 0.54$). Treatment of fingernail injuries with additional nightly ointment was associated with a higher prevalence of symptoms at 3 months (significant to χ^2 test, $p = 0.016$). Despite the high prevalence of symptoms in all groups at 3 months, only two patients had re-presented with macroform recurrent corneal erosion by 2 years. Both had been injured by a fingernail: one presented 3 months after treatment with our standard regimen, the other presented 7 months after a similar injury which was treated with additional nightly ointment.

These early results suggest that nightly lubricating ointment does not prevent recurrent symptoms when used following uncomplicated traumatic corneal abrasion. Instead, the reverse appears to be true, in that the group receiving additional ointment had significantly more symptoms at 3 month follow up. We were surprised at the high prevalence of recurrent symptoms in all groups. These early results imply that we should reconsider the place of both eyepad and ointment in our initial management. Future studies of treatment for corneal abrasion or erosion should pay close attention to patient symptoms.

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Correspondence to: Dr Danny Morrison, Department of Clinical Genetics, Molecular Medicine Centre, Western General Hospital, Edinburgh EH4 2XU.

- 1 Heyworth P, Morlet N, Rayner S, *et al*. Natural history of recurrent erosion syndrome—a 4 year review of 117 patients. *Br J Ophthalmol* 1998;82:26-8.
- 2 Kirkpatrick JNP, Hoh HB, Cook SD. No eye pad for corneal abrasion. *Eye* 1993;7:468-71.
- 3 Brown N, Bron A. Recurrent erosion of the cornea. *Br J Ophthalmol* 1976;60:84-95.
- 4 Hykin PG, Foss AK, Pavesio C, *et al*. The natural history and management of recurrent corneal erosion: a prospective randomised trial. *Eye* 1994;8:35-40.
- 5 Chandler PA. Recurrent erosion of the cornea. *Am J Ophthalmol* 1945;28:355-63.

Table 1 Prevalence and severity of recurrent symptoms, 3 months after treatment for traumatic corneal abrasion. Symptoms were assumed to be due to recurrent corneal erosion if they were described as frequent and significant pain, grittiness, photophobia, or watering of the injured eye only³

Cause of injury and treatment group	No symptoms or minimal symptoms	Mild symptoms (non-disabling)	Moderate symptoms (difficulty with daily activities, or sought further opinion)	Severe symptoms (confirmed macroform recurrent corneal erosion)
Fingernail: standard regimen (see text) (n=20)	17 (85%)	2 (10%)	0	1 (5%)
Fingernail: additional nightly ointment (see text) (n=22)	11 (50%)	7 (32%)	4 (18%)	0
Other causes: standard regimen (n=32)	25 (78%)	3 (10%)	4 (12%)	0

Reply

EDITOR,—Eke *et al* have addressed the issue of symptoms following an acute traumatic abrasion and demonstrate that 28% of patients were still symptomatic 3 months after the event. In our discussion¹ we suggest that what is required is a study examining the symptomatology of patients with established recurrent erosion syndrome before deciding what treatment modalities would be appropriate. Eke *et al*'s series represents a small group of patients (21) who are still symptomatic 3 months following an acute corneal abrasion, not long after the initial healing phase. This surely does not represent the group of patients that we are interested in—namely, those who have recurrent erosions some time following the initial injury. When Eke's group report the results regarding symptomatology at 3 years (due later this year) this will better represent a group of patients with recurrent erosion syndrome albeit limited to a small subgroup of these patients with a traumatic aetiology and whose numbers may be considerably less than those reporting symptoms at 3 months.

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- 1 Heyworth P, Morlet N, Rayner S, *et al*. Natural history of recurrent erosion syndrome—a 4 year review of 117 patients. *Br J Ophthalmol* 1998;82:26–8.

Horner's syndrome in infancy

EDITOR,—George *et al*¹ recently undertook a review of 23 cases of Horner's syndrome presenting in the first year of life. They remind us of the difficulty in differentiating between congenital and acquired Horner's syndrome in this age group. In their introduction they state that, in both congenital and acquired cases, "heterochromia may appear to be progressive as the child develops normal pigmentation in the fellow iris". Iris heterochromia would therefore appear to be of little value in distinguishing between congenital and acquired cases, the latter potentially being the group with underlying pathology. The authors conclude with a management protocol for the investigation of Horner's syndrome in infancy—namely, a full general examination and urinary vanillylmandelic acid levels.

However, their inclusion of the recommendation that further investigation is indicated if increasing iris heterochromia is noted would appear to be contradictory to their earlier statement. Iris pigmentation depends predominantly on the distribution of pigment within melanocytic cells on the anterior border and within the stroma. These cells migrate into the human iris late in gestation and postnatally, modulated by the sympathetic nervous system.² In 1915, Sym³ noted that iris colour is not fully developed until the 12th to 16th month, an observation continued by Giles *et al*⁴ in 1958 who reported the development of normal iris pigmentation in children up to 2 years of age. Thus, patients with Horner's syndrome, either congenital or acquired in the first year of life, might be noted to have progressive heterochromia as the fellow eye pigments. On this advice, both groups of children would be subject to further investigation which the authors are suggesting we should try to avoid.

In order to further clarify this excellent protocol, we would suggest the omission of

progressive iris pigmentation among the list of signs meriting radiological investigation.

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- 1 George NDL, Gonzalez G, Hoyt CS. Does Horner's syndrome in infancy require investigation? *Br J Ophthalmol* 1998;82:51–4.
- 2 McCartney ACE, Riordan-Eva P, Howes RC, *et al*. Horner's syndrome: an electron microscopic study of a human iris. *Br J Ophthalmol* 1992;76:746–9.
- 3 Sym WG. Review of Scalinci's article. *Ophthalmic Rev* 1915;34:167.
- 4 Giles CL, Henderson DA. Horner's syndrome: an analysis of 216 cases. *Am J Ophthalmol* 1958;46:289–301.

Reply

EDITOR,—Thank you for the interest shown in our paper.¹ We agree that in a congenital Horner's syndrome, an apparent increase in iris heterochromia may be observed as a result of an increase in the iris melanocyte pigment of the unaffected eye during the first 18 months of life. However, progressive heterochromia has been reported following acquired Horner's syndrome in both children and adults.^{2,3} In our patient with progressive heterochromia a Horner's syndrome had been diagnosed in infancy. When she presented to us the child's mother was concerned that the affected eye had originally had some pigment but was becoming progressively depigmented. A computed tomograph of the thorax was subsequently performed and showed a ganglioglioma. We have therefore included increasing heterochromia in our series as an index of suspicion because this had prompted further investigation in this one patient.

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- 1 George NDL, Gonzalez G, Hoyt CS. Does Horner's syndrome in infancy require investigation? *Br J Ophthalmol* 1998;82:51–4.
- 2 Makley LB, Abbott K. Neurogenic heterochromia: a report of an interesting case. *Am J Ophthalmol* 1965;59:297–9.
- 3 Miller NR. *Walsh and Hoyt's clinical neuro-ophthalmology*. 4th ed. Vol 2. Baltimore: Williams & Wilkins, 1985:503.

Improved impression cytology techniques for the immunopathological diagnosis of superficial viral infections

EDITOR,—I read with great interest the paper by Thiel *et al*¹ who applied immunoperoxidase and immunofluorescence techniques to diagnose viral infections of the ocular surface. Eleven years ago, we described a method of immunocytological staining for viral antigens on ocular surface cells collected on cellulose acetate strips.² Thiel and coworkers should be congratulated for improving this methodology by the use of a Biopore membrane device that facilitates specimen collection and processing. Another advantage common to these diagnostic techniques is that sample collection simultaneously accomplishes debridement, which may be beneficial in herpes keratitis and other viral infections of the ocular surface.

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- 1 Thiel MA, Bossart W, Bernauer W. Improved impression cytology techniques for the immunopathological diagnosis of superficial viral infections. *Br J Ophthalmol* 1997;81:984–8.
- 2 Wittmann JR, Pepose JS. Impression debridement of herpes simplex dendritic keratitis. *Cornea* 1987;5:245–8.

Reply

EDITOR,—We thank Dr Pepose for his comments on our improved impression cytology technique. He does point out the potential of this method for therapeutic debridement in superficial herpetic keratitis. As shown in Figures 3A and B of our recent article¹ this technique does indeed allow a gentle and effective debridement of infected epithelial cells. It is also our experience that debridement by this method is beneficial for the healing process, and none of our patients followed so far developed complications after impression. The therapeutic effect of sample collection, however, was beyond the scope of this study and further investigations are required to clarify this aspect.

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- 1 Thiel MA, Bossart W, Bernauer W. Improved impression cytology techniques for the immunopathological diagnosis of superficial viral infections. *Br J Ophthalmol* 1997;81:984–8.

Visual field progression in glaucoma

EDITOR,—We read with interest the article by Viswanathan *et al*.¹

We were surprised by the authors' progression criteria for the PROGRESSOR method; their criteria are extremely sensitive and very non-specific: only one point needs to show significantly negative slope when tested on the 5% level, together with an observed slope of <–1 dB/year and <–2 dB/year for inner and outer points, respectively. With 74 test points, it goes without saying that one here faces a problem with mass significance testing, which explains the very low specificity. It is obvious that almost any stable glaucoma patient would be detected as progressing with the reported method.

To illustrate the effect, we studied 10 patients with manifest glaucoma which, after at least 5 year follow up studies and at least eight visual fields, did not show visual field progression on the same clinical grounds as those used by Viswanathan *et al*. We had used same type of visual field test as the authors—that is, Humphrey 30-2 full threshold. In order to eliminate the possibility of any significant subclinical progression, the order of field tests were randomised before tests were analysed by the PROGRESSOR method. This randomisation secures that all significant progression would be the results of pure chance. Using the progression criteria of Viswanathan *et al*, stated above, five visual fields showed progression at the fourth visit, and already at the sixth visit eight eyes (80%) had shown false progression at one or more visits.

Further, we performed a Monte Carlo simulation study with the same criteria: 10 000 fields were simulated and tested 10 times and the regression slope was tested at each point starting at the third test occasion. The field was divided into two regions with 50 inner points and 24 outer points, respectively, as stated by Viswanathan *et al*, and the field

generated by Gaussian variables with one homogeneous random component common to all points in the region (SD 1.1 dB), and with an independent inhomogeneous variation (SD 0.9 dB) between points.² All fields were assumed to be stable, with constant mean at all test occasions. We assumed three tests to be performed each year. With the criteria of Viswanathan *et al*, 60% of the simulated fields showed significant progression in at least one inner point and 29% showed progression in at least one outer point. This means that at least 72% showed false significant progression on at least one occasion. In fact, 35% of the simulated fields showed progression on two consecutive test occasions. If we had included the short term fluctuation, the number of false significances would have been even larger. This finding of 72% false significances is in agreement with our experience with the 10 glaucoma patients.

Viswanathan *et al* must have been fooled by their own selection of progressing patients, and the use of remaining statistical significance at the last visit as a criterion for reliability. Naturally, this is erroneous. It is not surprising that many points in subjectively progressing visual field tests show significant progression at the end of the follow up period, and this yields no information as to the specificity of the analyses employed.

It is generally and correctly considered that, because of the large random variability of glaucomatous visual fields, real progression can seldom be demonstrated in glaucomatous visual fields in only 1 year. In view of this we are quite satisfied that the mean time until progression was demonstrated with STATPAC 2 was more than 2 years. In fact, we recommend stricter progression criteria for STATPAC 2 than those used by the authors, again in the interest of specificity.

Linear regression analysis has advantages in visual field follow up; one is that all available data are used. Such analyses can be applied both properly and improperly. Analyses that lack specificity are only misleading, however, and may even result in improper clinical management. A more thorough analysis of linear regression analyses for visual field follow up is needed, and we will address this subject in a future article.

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1 Viswanathan AC, Fitzke FW, Hitchings RA. Early detection of visual field progression in glaucoma: a comparison of PROGRESSOR and STATPAC 2. *Br J Ophthalmol* 1997;81:1037-42.

2 Flammar J. Fluctuations in computerized perimetry. In: Whalen WR, Spaeth GL, eds. *Computerized visual fields. What they are and how to use them*. Thorofare, NJ: Slack, 1985:45-58.

Reply

EDITOR.—We thank Heijl *et al* for their interest in our paper.¹ Their letter raises a number of issues, all of which are worthy of discussion.

Our paper was a continuation of previous work (ref 25 in our paper) comparing a new analysis program (PROGRESSOR) with the widely available visual field analysis program STATPAC 2. In the earlier paper we showed that the two programs identified the same locations in the visual field as "progressing". In this paper we showed that "progression" as we

defined it occurred much earlier with PROGRESSOR than STATPAC 2. As an unwelcome outcome of glaucoma management is (further) visual field loss, any approach looking for earlier detection of visual field loss seemed to have merit.

Drs Heijl, Bengtsson, and Lindgren criticise us on our methodology and approach. Specifically they express concern at the use of a significance level of $p < 0.05$, suggest that our patient selection would bias the results, and express satisfaction that the mean time before STATPAC 2 can detect progression is more than 2 years.

We set the significance level for our study at the 5% level because this is the only significance level available with STATPAC 2.² With this in mind, we set out to compare PROGRESSOR and STATPAC 2 in as fair a trial as possible. Since there is no external "gold standard" for glaucomatous visual field progression it is impossible to measure the true specificity of any analysis.³⁻⁵ Our patient selection, which would have a bearing on the results, was from a cohort of patients with normal pressure glaucoma who had progression identified on clinical examination of a large series of visual fields. This group may well be unrepresentative of glaucoma patients as a whole but as examples of undoubted progression they allowed us to perform the study. We agree with Heijl *et al* that the 5% significance level is likely to result in overdiagnosis of progression. In fact, even using the 5% level PROGRESSOR would be expected to produce less overdiagnosis than STATPAC 2 since PROGRESSOR contains a rate (that is, slope) criterion for progression which STATPAC 2 does not: this also mitigates against the "problem with mass significance testing" which "goes without saying". When we first compared PROGRESSOR with STATPAC 2 (in terms of numbers of progressing points detected rather than speed of detection) we expected the 5% level to be an overly lax criterion for PROGRESSOR: we were surprised to find that this criterion had to be relaxed still further to the 10% level in order for PROGRESSOR to emulate STATPAC 2.⁶

It is not surprising that Heijl *et al* found a high rate of false progression using a rudimentary numerical simulation. This is to be expected when the 5% significance level is specified, and their simulation echoes research on true clinical data.⁷ The authors have seemingly fallen into the trap of equating statistical significance with clinical significance. Any change in the visual field must be placed in the context of the overall management of the patient (which will include consideration of intraocular pressure, optic disc features, current and previous therapy, together with the patient's age and general medical condition) before the clinical implications may be assessed. Statistical significance in visual field change, no matter how it is obtained, does not itself equate to clinical significance. The subject of which progression criteria correspond most closely to "true" clinically observed worsening of glaucoma is currently under investigation.

Because setting the progression criteria at the $p < 0.05$ level will provide a large number of false positive responses, we do not recommend the use of this level for clinical use. As PROGRESSOR (unlike STATPAC 2) is not widely available the chance of "improper clinical management" is therefore negligible. This cannot be said to be the case for the widely available STATPAC 2 for the original criteria established by the authors (ref 20 in our

paper) are considered by many as the gold standard for progression. We are interested that Heijl *et al* now set stricter criteria for progression than is currently available on STATPAC 2 and would be interested to learn what these might be as they have not, to our knowledge, appeared in print.

We are surprised that Heijl *et al* are "quite satisfied" with a detection time of more than 2 years for STATPAC 2. This seems a nihilistic approach when a more sensitive analysis, such as PROGRESSOR, coupled with clinical perception, offers the hope of earlier detection and treatment (if required) of an irreversibly blinding condition.

Finally, we are delighted that Heijl *et al* are engaged in "a more thorough analysis" of linear regression, particularly as they dismissed linear regression as "the best way to diagnose cataracts" at the open glaucoma meeting of the ICO in 1990. We await their results with interest.

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1 Viswanathan AC, Fitzke FW, Hitchings RA. Early detection of visual field progression in glaucoma: a comparison of PROGRESSOR and STATPAC 2. *Br J Ophthalmol* 1997;81:1037-42.

2 Heijl A, Lindgren G, Lindgren A, *et al*. Extended empirical statistical package for evaluation of single and multiple fields in glaucoma: Statpac 2. In: Mills RP, Heijl A, eds. *Perimetry update 1990/1991*. Amsterdam: Kugler & Ghedini, 1991:303-15.

3 Birch MK, Wishart PK, O'Donnell N. Determining progressive field loss. In: Mills RP, Wall M, eds. *Perimetry update 1994/1995*. Amsterdam: Kugler & Ghedini, 1995:31-6.

4 Nouri-Mahdavi K, Brigatti L, Weitzmann M, *et al*. Comparison of methods to detect visual field progression in glaucoma. *Ophthalmology* 1997; 104:1228-36.

5 Smith SD, Katz J, Quigley HA. Analysis of progressive change in automated visual fields in glaucoma. *Invest Ophthalmol Vis Sci* 1996;37: 1419-28.

6 McNaught AL, Crabb DP, Fitzke FW, *et al*. Visual field progression: comparison of Humphrey Statpac 2 and pointwise linear regression analysis. *Graefes Arch Clin Exp Ophthalmol* 1996;234: 411-18.

7 Schulzer M. Errors in the diagnosis of visual field progression in normal-tension glaucoma. *Ophthalmology* 1994;101:1589-94.

Topical steroids and alkali burns

EDITOR.—In their recent report attesting to the safety of topical prednisolone in the treatment of alkali burns¹ Davis and colleagues do not specify the type of prednisolone which was prescribed. Prednisolone acetate (Pred Forte) is absorbed into the human eye and reaches aqueous concentrations many fold higher than prednisolone phosphate (Predsol)² perhaps on account of an acetylating enzyme in the cornea.

It is therefore important to know which form of prednisolone was employed in this study.

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1 Davis AR, Ali QH, Aclimados WA, *et al*. Topical steroid use in the treatment of alkali burns. *Br J Ophthalmol* 1997;81:732-4.

2 McGhee CNJ, Watson DG, Midgley JM, *et al*. Penetration of synthetic corticosteroids into human aqueous humour. *Eye* 1990;4:526-30.

Reply

EDITOR,—In answer to Mr Dutton's question, I have rechecked our data and the paragraph detailing treatment should now read:

"All patients with a grade 1 injury or greater were treated with a standard regimen which included . . . topical steroid (dexamethasone alcohol 0.1% or prednisolone sodium phosphate 0.5%, or betamethasone sodium phosphate 0.1%; in addition two patients had betamethasone sodium phosphate ointment at night)"

Of those who had prednisolone drops, none had prednisolone forte eye drops. The aim of our paper was to emphasise the safety of topical steroids when used in conjunction with vitamin C in the treatment of alkali burns. To compare the use of different types of steroids would require a prospective study.

ALISON DAVIS

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senator of foreign affairs, Gareth Evans, and Mrs Gabi Hollows with her children. This laboratory is an expression of his admiration, understanding, and love of the people of Eritrea and their struggle to achieve independence and self reliance. The factory produces an estimated 60 000 intraocular lenses per year; in Eritrea there are 30 000 cataract patients per year.

His desire to help and identify himself with the suffering of the people of Eritrea did not end with the eye programme. He undertook the task of finding a solution to the removal of the millions of landmines scattered in Eritrea and other countries.

Fred Hollows was no dry, dusty academic but had considerable style and joie de vivre. He maintained an active family life with his wife and their five young children.

Fred Hollows will remain for a long time in our hearts and memories. We share deepest sympathy with his family.

BINIAM GHEBREMEDHIN

IN MEMORIAM

Fred Hollows

Fred Hollows died on 10 February 1993 in the age of 63 years after a short and valiant battle with kidney, lung, and brain cancer. In his remarkable career he had quickly risen to being a major international figure in the world of ophthalmic surgery.

In the 1950s Fred Hollows decided as a young medical student to become an ophthalmologist because this would give him a useful skill to work in Africa. As a postgraduate ophthalmology student in Wales, Hollows studied epidemiology so that he could link eye doctoring with wide ranging community health programmes. In 1968 at Wattie Creek in northern Australia Hollows identified in an aboriginal community "dahlak blindness", a hazy corneal condition first found in Eritrea. He began to lobby for funds and between the years 1976 and 1979 a "better vision for all" programme called the National Trachoma and Eye Health Programme treated the eyes of 100 732 people; 62 116 of these were indigenous Australians.

Eventually this led to series of eye health programmes in Eritrea, Nepal, and Vietnam, which have restored the sight of thousands, and to a struggle to lessen disparity in health and treatment between the "haves" and "have nots".

When he went to Eritrea in 1988 during the hardest struggle for independence he taught the medical doctors in ophthalmological surgery. There were many patients with cataract who could not be treated in Eritrea at that time because of lack in experience and materials; many of the patients came from Danakel (south eastern Eritrea), said to be the hottest place on the earth.

From 1990 to 1992 Professor Hollows gathered the necessary capital to build the intraocular lens factory in Asmara; such a factory has been put to the test before in Australia.

His dream came true: The Foundation of Fred Hollows was officially opened on 20 January 1994 in Asmara by the president of the state of Eritrea, I Afeworki, the Australian

NOTICES

Avoidable blindness

The latest issue of the *Community Eye Health* (no 25) discusses the elimination of avoidable blindness. With an editorial by Bjorn Thylfors, the director of the WHO Programme for the Prevention of Blindness and Deafness, the issue covers treatment of cataract in regions of India and the role of patient counsellors in increasing the uptake of cataract surgery and IOLs. For further information please contact *Community Eye Health*, International Centre for Eye Health, Institute of Ophthalmology, 11-43 Bath Street, London EC1V 9EL. (Tel: (+44) 171 608 6910; fax: (+44) 171 250 3207; email: eyeresource@ucl.ac.uk) Annual subscription £25. Free to workers in developing countries.

Residents' Foreign Exchange Programme

Any resident interested in spending a period of up to one month in departments of ophthalmology in the Netherlands, Finland, Ireland, Germany, Denmark, France, Austria, or Portugal should apply to: Mr Robert Acheson, Secretary of the Foreign Exchange Committee, European Board of Ophthalmology, Institute of Ophthalmology, University College Dublin, 60 Eccles Street, Dublin 7, Ireland.

XVI Tuebingen Detachment Course

The XVI Tuebingen Detachment Course in retinal and vitreous surgery will be held 4-5 September 1998 in Odessa, Ukraine. Further details: Professor I M Logai, Director, The Filatov Institute, 49/51 Boulevard Francais, Odessa, 270061, Ukraine. (Tel: +38-0482-22 20 35; fax: +38-0482-68 48 51.)

International Agency for the Prevention of Blindness (IAPB)

The International Agency for the Prevention of Blindness (IAPB) will hold its next general assembly in Beijing, China on 5-10 September

1998. Further details: Gullapalli N Rao, Secretary General, IAPB Secretariat, LV Prasad Eye Institute, LV Prasad Marg, Banjara Hills, Hyderabad 500 034, India. (Tel: 091-40-215389; fax: 091-40-248267; email: IAPB@lvpeye.stph.net)

XI Tuebingen Angiography Course

The XI Tuebingen Angiography Course will be held 12 September 1998 at the auditorium university dental clinic, Tuebingen. Further details: Dr W Inhoffen, University Eye Clinic Department of Ophthalmology III, Schleierstrasse 12, D-72076 Tuebingen, Germany. (Tel: +49-(0) 7071-292968; fax: +49-(0)-7071-293746; email: ingrid.kreissig@uni-tuebingen.de; <http://www.medicin.uni-tuebingen.de/~webaugen/index.html>)

ICOP 98

The next International Conference in Ophthalmic Photography (ICOP) will be held on 19-21 September 1998. Further details: Mrs Gillian Bennerson, Senior Ophthalmic Photographer, Bristol Eye Hospital, Lower Maudlin Street, Bristol BS1 2LX. (Tel: 0117-928-4677.)

IV meeting of the European Society for Out-Patient Eye Surgery (ESOPES)

The IV meeting of the European Society for Out-Patient Eye Surgery (ESOPES) will be held in Vittel, France on 9-11 October 1998. Further details: Mrs Nicole Charron, Director, Palais des Congrès, Av Bouloumie, BP 57, 8802 Vittel, France. (Tel: +33 329 08 18 30; fax: +33 329 08 6601.)

VIth International Symposium on Graves' Ophthalmology

The VIth International Symposium on Graves' Ophthalmology will be held on 27-28 November 1998 in Amsterdam. Further details: Amsterdam Thyroid Club, Department of Endocrinology, F5-171, Academisch Medisch Centrum, Meibergdreef 9, 1105 AZ Amsterdam, Netherlands.

Hong Kong Ophthalmological Symposium 98

The Hong Kong Ophthalmological Symposium 98 on myopia will be held on 28-29 November 1998 at the Hong Kong Convention and Exhibition Centre. Further details: Dr Woon-ming Chan, Secretary, Organising Committee, Hong Kong Ophthalmological Symposium 98, University Eye Centre, 3/F, Hong Kong Eye Hospital, 147K Argyle Street, Kowloon. (Tel: (852) 2761 9128; fax: (852) 2715 0089; email: cohk@netvigator.com)

Singapore National Eye Centre

The 3rd SNEC international meeting and 11th international meeting on cataract, implant, microsurgery and refractive keratoplasty (ICIMRK) will be held at the Shangri-La Hotel, Singapore on 28-30 November 1998. Further details: Organising Secretariat, 3rd SNEC International Meeting and 11th ICIMRK, Singapore National Eye Centre Pte Ltd, 11 Third Hospital Avenue, Singapore 168751. (Tel: (65) 2277-255; fax: (65) 2277-290/1)