LETTERS TO THE EDITOR

Trachoma still undefeated

SIR,—Your interesting editorial¹ about problems still outstanding in the study of trachoma was drawn to my attention recently and revived personal memories of this disease. Having spent a substantial part of my working life in various tropical countries I have seen and treated a large number of cases in various stages of the disease.

In August 1939 as the very junior regimental medical officer of an Indian battalion under embarkation orders for the Middle East, in the course of a pre-embarkation medical inspection, I found that some 80% of the Sikh company had trachoma, these men were all from the Punjab in north west India. The disease was relatively inactive but the question of fitness for active service arose. On the eve of war, the whole battalion's move might have been delayed due to the unfitness of virtually one whole company. This was a matter for a more senior opinion than mine and the station ophthalmic specialist was sent to cover me. He confirmed the diagnosis and passed them all 'fit.' The battalion went to North Africa but I was diverted to Burma so I was unable to follow their fortunes. Subsequent experience of trachoma among Indian troops convinced me that the hygienist had been right to pass them fit. Trachoma was fairly common in the Indian Army especially among soldiers recruited from the dry, dusty, northern parts of the country. In the high state of hygiene in which they lived, even on active service, few developed serious complications. It seemed therefore that there must be other factors causing the development of those complications which often cause blindness, notably entropion and gross corneal pathology.

I next met the disease in its endemic form as ophthalmic specialist in the Gold Coast, as it was then called, from 1948–52. From a combined analysis of cases seen personally, in the eye clinic of the Gold Coast Hospital in Accra and on tour in the Northern Territories, I was able to show that the incidence and severity of the complications of trachoma were far greater amongst the inhabitants of the dry dusty Northern Territories than those in the coastal area.² At that time facilities for pathological and bacteriological investigation were limited so findings were based on clinical observation.

From 1961 to 1966 I was working in the Jane Furse Anglican Mission Hospital, a large hospital in Sekukhuniland, a very dry dusty area of the Northern Transvaal. Once again I was struck by the high incidence of trachoma, commonly complicated by entropion and ultimate blindness. Thanks to the interest and help of Dr A Davies, the Medical Superintendent and Pathologist at the hospital, it was possible to demonstrate in some of the more severe cases, an accumulation of silica in the scarred conjunctiva and superior tarsi removed at operation.³ Those Africans who had spent long periods working in the gold mines away from Sekukhuniland rarely went blind from the later complications of trachoma. These men had lived in barracks where hygiene standards were high and water plentiful.

During my last overseas appointment I spent two years as a government ophthalmic special-

ist in the two main government hospitals in the coral island of Mauritius in the Indian Ocean. The population, then about 860 000, consisted of several settled communities, in descent mainly Indian, European, African, Chinese or a mixture of these. I have no figures for the total number of ophthalmic cases seen but some idea may be deduced from the fact that I performed just under 1600 cataract extractions during the two years in the island. Mauritius is based on coral and remarkably free from dust. I did not see a single case of trachoma.

Most of the sociodemographic risk factors in all the rural areas in which I worked were in my opinion similar to those described in a rural Egyptian village.⁴ Proper latrine facilities should reduce the fly density and one mode of transmission during the infective stage of the disease. However, if our findings in the Transvaal, of silica deposits in the tarsal conjunctiva in more advanced cases can be repeated in other areas, then there would be evidence that provision of adequate washing facilities as well as latrines may be equally important.

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- 1 Trachoma still undefeated [Editorial]. *Br J Ophthalmol* 1991; 75: 321.
- 2 Sarkies JWR. Onchocerciasis and trachoma in the Gold Coast. *Ann Trop Med Parasit* 1952; 46: 214–7.
- 3 Sarkies JWR. Dust and the incidence of severe trachoma. *Br J Ophthalmol* 1967; 51: 97–100.
- 4 Courtright P, Sheppard J, Lane S, Sadek A, Schachter J, Dawson CR. Latrine ownership as a protective factor in inflammatory trachoma in Egypt. *Br J Ophthalmol* 1991; 75: 322–5.

Penetrating keratoplasty for keratoconus

SIR,—I would like to congratulate Sharif and Casey on an excellent description of the long-term success of penetrating keratoplasties for keratoconus.¹ However, their study design may have underestimated the probability of graft failure. Figure 4 shows the Kaplan-Meier survival curve of their 100 grafts during the course of the study. They found most failures occurred in the first 500 days (1.37 years). In their materials and methods section, they state only keratoconus patients with a minimum of 4 years' follow up were included in the study. It would therefore appear that the only failures included were those that kept coming back with a failed cornea for some reason, or those that had been regrafted and followed for the minimum of 4 years. Patients having failed corneas without retreatment and being lost to follow up before 4 years would have been excluded in their study design. It has been my experience that some patients with failed corneal grafts do not have a re-graft and are not interested in follow up. Though I suspect their success rate will still be quite high, it would be interesting to know how many total keratoplasties for keratoconus were performed during the study period, and how many of those that did not reach 4 years of follow up had failed corneal grafts or episodes of rejection.

As their data pointed out, a large portion of individuals undergoing corneal transplant surgery for keratoconus are young males. As a group, young males are more prone to be involved in activities that could lead to traumatic rupture of the graft wound with loss of the graft or even the eye. They are also more prone to be lost to follow up, either because of relocating or by being inattentive to the care of their eye. The overall number of these individ-

uals having adverse events may be small but could still significantly change the survival data.

Their study appears to be an important one which provides excellent information on many of the long-term complications and outcomes of eyes having penetrating keratoplasty for keratoconus. However, in order to have an accurate survival analysis all consecutive cases performed during their study period would need to be included.

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- 1 Sharif KW, Casey TA. Penetrating keratoplasty for keratoconus: complications and long-term success. *Br J Ophthalmol* 1991; 75: 142–6.

SIR,—We would like to thank Dr Price for his compliments and thoughtful remarks on our paper.¹

The total number of penetrating keratoplasties for keratoconus performed in our unit during the study period was 148; 100 of these cases were included in the study as they fulfilled our criteria of a minimum follow-up period of 4 years.

The aim of the study was to determine the long-term survival of the grafts, so the remaining 48 cases (with shorter follow-up periods) were excluded from the study. Analysis of this subgroup was carried out and it showed that the average follow-up period was 2 years. Forty five grafts (out of 48) were still clear at the end of the follow-up period (93.7%). Two of the three graft failures occurred secondary to irreversible rejection episodes and the third graft suffered a primary graft failure. Thus, this survival analysis of the unpublished subgroup shows similar findings to those detected in our study with long-term follow-up.

The other point raised by Dr Price was in relation to the male predominance in our study (68%), and the possibility that this could change the survival data.

Further analysis of all the 148 keratoplasties revealed that traumatic wound dehiscence occurred in four cases only; two of which were females (ie male to female ratio was 1:1). Out of the 48 cases, who were lost to follow-up within the first 4 years postoperatively, 25 were females (52%). Contrary to Dr Price's suggestion, all these findings support our initial observations that male to female ratio in our study did not significantly influence the survival data.

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- 1 Sharif KW, Casey TA. Penetrating keratoplasty for keratoconus: complications and long-term success. *Br J Ophthalmol* 1991; 75: 142–6.

Diabetic radiation morbidity

SIR,—Features of ocular radiation vasculopathy were first described in 1933 and have been well characterised.^{1–4} Diabetes and other diseases that affect small vessels (hypertension, collagen vascular diseases, and chemotherapy), possibly increase the risk of radiation induced vasculopathy. However no study has proved this.^{2–4} We reviewed retrospectively 469 uveal melanoma patients all treated with either ¹²⁵I brachytherapy or helium ion irradiation and compared both visual outcome and complications in diabetic and non-diabetic patients.

All patients were examined in the Ocular

Oncology Unit, University of California, San Francisco. Retrospective chart review identified 20 diabetics (4.3%). All had both fundus photography and fluorescein angiography. Three patients were insulin dependent, 11 were on oral hypoglycaemics, and six used diet management of their diabetes. Prior to radiation three patients had proliferative retinopathy, four background, and 13 had no diabetic retinal disease on either ophthalmoscopy or fluorescein angiography. Two patients with proliferative retinopathy were treated with panretinal photocoagulation, one before and one after treatment. Any patient, diabetic and non-diabetic, developing significant proliferative retinopathy and/or rubeosis following irradiation was treated with panretinal photocoagulation. Tumour characteristics (Table 1) were similar in both diabetic and non-diabetic subjects; tumours in diabetics were located slightly more distant from fovea and nerve.

Table 1 Characteristics of tumours

Variable	Diabetic	Non-diabetic
Greatest base diameter	11.3 (3.2)	10.8 (3.9)
Shortest base diameter	9.7 (2.6)	9.0 (3.1)
Height	5.9 (2.1)	6.3 (2.6)
Distance to optic nerve	5.6 (4.5)	4.9 (3.8)
Distance to fovea	5.7 (4.9)	4.6 (4.0)
Neovascular glaucoma	25%	21%
Optic neuropathy	5%	12%
Radiation maculopathy	20%	12%

Standard deviations are in parentheses.

The incidence of complications (Table 1) was higher in diabetics for neovascular glaucoma and radiation maculopathy, but lower for radiation optic neuropathy. No significant difference was found for these three variables on the Cox regression model analysis ($p > 0.05$). Covariates in the analysis were: tumour size and distance from the fovea and nerve, radiation type and dose, age, diabetes status, and presence or absence of hypertension. For those patients who lost vision, the mean time to visual acuity loss (two or more Snellen lines) was the same for diabetics and non-diabetics, 8.1 months. No significant difference was found for ultimate visual outcome. No significant correlation was found between pretreatment severity of diabetic retinal disease, complications, and visual outcome.

Anecdotal reports in the literature suggest that the diabetic eye is more likely to develop radiation morbidity.^{2,4} Our review suggests that the difference in response is probably more subtle than previously thought. Because the number of diabetics in our study is small and incidence of complications low, the probability of detecting correctly a difference in response of the diabetic eye at the $p < 0.05$ level is about one in five. Therefore our analysis may have missed identifying increased morbidity in diabetics. A definitive statement on diabetic radiation morbidity awaits additional data.

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Hyperopic shift and the use of masking agents in excimer laser superficial keratectomy

SIR,—I read with interest the articles on excimer laser treatment of superficial corneal disorders by Gartry *et al*¹ and Sher *et al*,² both of which attracted an editorial. I would like to comment on two aspects on this type of treatment both of which are, I believe, related to the use of masking agents (surface modulator fluids³).

Firstly in addition to the four potential mechanisms postulated to be responsible for the observed hyperopic shift in some patients¹ three further mechanisms are probably in play: (1) with multi-zone ablation using a 4 mm ablation diameter¹ it is almost impossible not to expose the axial cornea to more pulses of ablation than the more peripheral parts of the cornea (Fig 1a). It follows that a relative depression or flattening of the axial cornea would be created — a myopic correction is therefore cut (Fig 1b). A similar mechanism applies to the 'smoothing' technique used in the early cases of site 1 in the American report. It is conceivable that both regular and irregular astigmatism could be created in the same way.

(2) With single zone on-axis ablation, 1% hydroxypropylmethylcellulose (HPMC) creeps up the wall of the circular well created by repeated on-axis ablation (Fig 2a). Gartry *et al* commented that this surface tension effect may be minimised by wiping the walls with a cellulose sponge. These sponges were thought to be suboptimal in their absorptive properties when used in conjunction with 1% HPMC.¹ Capillarity has the effect of forming a relatively

concave meniscus (Fig 2a), and it may be this relative concavity which becomes translated onto the ablated zone (Fig 2b), causing a hyperopic shift.

How can we test whether the latter hypothesis is true? There are two ways. (a) As the diameter of ablation is decreased the effect of the concavity will become more pronounced. The reverse will apply to a larger diameter of ablation. It follows that there should be less hyperopic shift with larger discs of ablation (Fig 3). (b) If on-axis single zone ablation is performed on normal clear cornea without the use of any form of masking agent there should be no hyperopic shift (see also epithelial hyperplasia below). The refractive result of this could be compared with a similar ablation with the use of HPMC.

(3) A third potential mechanism relates to the possibility of epithelial hyperplasia within the ablation zone especially adjacent to the wall of the well. A relative concavity can thus be created decreasing the refractive power of the cornea.

Sher *et al* also noted the phenomenon of hyperopic shift and compensated for it by preprogramming the laser to cut a secondary hyperopic correction ('combined' cut).² It would be of interest to know whether the refractive results quoted in each paper were obtained by retinoscopy, autorefraction, or subjective refraction, and whether there was any keratometric correlation.

Secondly it is intriguing to note that the two groups have come to opposite conclusions regarding the effectiveness of masking agents in creating an optically smooth surface. Gartry *et al* have unequivocally shown that it is possible to produce an optically smooth surface with the help of 1% HPMC. It was also pointed out that 2% HPMC and polyvinyl alcohol (PVA) were less suitable agents as they were too viscous,¹ thus the 2.5% methylcellulose used by Sher *et al* was clearly too thick. Furthermore Sher's results were also complicated by the use of an effluent fan causing ripples on the surface of the masking agent.² The ripples would in turn be etched onto the anterior surface of the cornea causing irregularities. Is an extraction fan necessary?

There are two theoretical advantages of using a fan. Firstly debris created by the ablation process would not be redeposited onto the cornea causing uneven ablation. Secondly both the surgeon and patient would be protected from the 'smoke' created by the ablative process. We do not know the nature of the airborne particles produced by ablating HPMC.

Figure 1a

Figure 1 Multiple ablation zones inevitably remove more corneal tissue axially (a). Profile of the cornea (b) thus created.

Figure 1b

Figure 2a

Figure 2 Cross-sectional diagram of the cornea (a) showing how surface tension could create a relatively concave meniscus. (b) With repeated ablation, this profile is etched onto the cornea.

Figure 2b

Figure 3 The concave meniscus loses its importance as the diameter of ablation enlarges.