Oncology Unit, University of California, San Francisco. Retrospective chart review identified 20 diabetics (4.8%). All had both fundus photography and fluorescein angiography. Three patients were insulin dependent, 11 were on oral hypoglycaemics, and six used diet management with their diabetes. Prior to the operation 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 retinoblast 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

<table>
<thead>
<tr>
<th>Variable</th>
<th>Diabetic</th>
<th>Non-diabetic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Greatest base diameter</td>
<td>11-3 (5-2)</td>
<td>10.0 (3-9)</td>
</tr>
<tr>
<td>Shortest base diameter</td>
<td>9.7 (5-6)</td>
<td>9.0 (5-1)</td>
</tr>
<tr>
<td>Height</td>
<td>5.9 (2-1)</td>
<td>6.2 (2-6)</td>
</tr>
<tr>
<td>Distance to optic nerve</td>
<td>4.9 (2-6)</td>
<td>4.9 (2-6)</td>
</tr>
<tr>
<td>Distance to fovea</td>
<td>5.7 (4-9)</td>
<td>4.6 (4-6)</td>
</tr>
<tr>
<td>Neovascular glioma</td>
<td>25%</td>
<td>21%</td>
</tr>
<tr>
<td>Optic neuropathy</td>
<td>12%</td>
<td>12%</td>
</tr>
<tr>
<td>Radiation maculopathy</td>
<td>20%</td>
<td>12%</td>
</tr>
</tbody>
</table>

Standard deviations are in parentheses.

The incidence of complications (Table 1) was higher in diabetics for neovascular glioma 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: tumor 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 retinopathy, complications, and visual outcome.

Accidental reports in the literature suggest that the diabetic eye is more likely to develop radiation morbidity.1-4 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 other increased morbidity in diabetics. A definitive statement on diabetic radiation morbidity awaits additional data.


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

Sr,-I read with interest the articles on excimer laser treatment of superficial corneal disorders by Gartry et al5 and Sher et al6 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 fluids). Firstly in addition to the four potential mechanisms postulated to be responsible for the observed hyperopic shift in some patients' three further mechanisms are probably in play: (1) with multi-zonal ablation using a 4 mm ablation diameter it is almost impossible not to expose the axial cornea to more pulses of ablation than the more peripheral parts of the cornea (Fig la). 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 this 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. 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 ablative process is completed the zone 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 after the sutures were removed, of 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).7 It would be of interest to know whether the refractive results quoted in each paper were compensated for by the addition of the amount of subjective refraction, or 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, thus the 2-5% methylcellulose used by Sher et al was clearly too thick. Furthermore Sher et al were also complicated by the use of an effluent fan causing ripples on the surface of the masking agent. 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 ablative 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.
and biological tissues and more research is needed. When a masking agent is used, a fan causing ripples on the surface is undoubtedly undesirable.

Masking agents provide the key to producing a regular surface and must not be dismissed. Gartry et al obtained superior results because great care was taken continuously to refill and uniformly to redistribute the masking agent over the ablated area and constantly to monitor the changes in tissue fluorescence. This is a surgical art rather than worshipping at the altar of high technology.

CHRISTOPHER LIU
5 Fleming Brook Close, London NW1 1XS


Reply

SIR,—Following on the interesting comments made by Dr Liu in his letter reported that superficial keratotomy with the excimer laser results in a hyperopic shift of 2-85 dioptres on average in our series. This was an unexpected finding since we had assumed that photoablation of an even layer of tissue across the entire cross-section of the beam (4 mm in diameter in most cases) would produce no change in corneal curvature. This is in contrast to the planned change in corneal curvature attempted when a motorised iris diaphragm is used during photorefractive keratectomy.

We put forward four possible mechanisms to explain this phenomenon. (1) The distribution of calcium within the band keratopathy might mask the beam allowing greater ablation of tissue centrally. (2) A centrifugal contraction of the lamellae at the ablated surface. (3) Shielding of the periphery of the beam by debris ejected from the surface. (4) Increased obliteration of incident radiation at the edge of the beam (due to the curvature of the cornea) resulting in a slightly less photoablation in the periphery of the ablation zone.

We described the treatment of two main categories of corneal pathology, namely rough surfaces and smooth surfaces. The background and principles of superficial keratotomy with the excimer laser to smooth a rough surface were documented in detail and in particular the importance of masking areas of stroma between surface peaks to avoid reproducing the surface irregularity deeper into the cornea. We found 1% methyl cellulose optimal for this purpose. When treating the second category (example smooth band keratopathy) since we started with a smooth surface it was unnecessary to apply any masking agent. As we explained in our paper in general the eyes with the best visual potential fell into this last category and it was in these cases that subjective refraction revealed a hyperopic shift (cases 18, 19, 21, 22 and 23).

Dr Liu suggests that when overlapping ablative zones more tissue will be removed from the central cornea due to intersection of treated areas. This is not the explanation since we found considerable hyperopic shift in patients treated with a single axial zone (for example cases 22 and 23). In addition, in those patients with good visual potential treated with multiple zones a 4 mm central zone was usually cleared first followed by continuous areas with minimal overlap. He also states that 'it is conceivable that both regular and irregular astigmatism could be created in the same way.' We agree with this of course since we documented the effect in case 19. As stated in our paper it was this potential for irregular astigmatism when using multiple continuous zones that led us to conclude that where good visual potential exists a single central zone is the most effective treatment method.

Dr Liu's second suggestion to explain the hyperopic shift relates to the use of methyl cellulose and since this was only employed (as a single application) in cases with rough corneal surfaces (where the aim was smoothing rather than glare reduction and improvement in acuity) this cannot provide a basis for the effect.

The third mechanism proposed however—that of epithelial hyperplasia—warrants further consideration. We have found that regenerating epithelium accounts for a large part of the regression in refraction occurring in our photo- refractive keratectomy patients. We have hypothesised that epithelial hyperplasia over the ablated zone accounts for the first part of an asymptomatic regression towards a plateau with stromal remodelling providing the basis for subsequent less change. It is difficult to envisage that entirely the reverse could occur when treating smooth band keratopathy. We also suggest that any 'relative concavity' due to hyperplasia at the edge of the ablated zone would be minimal since it is necessary only to ablate between 10 and 15 μm of stroma (virtually only Bowman's layer) to clear smooth band keratopathy and this is a shallow ablation compared with the thickness of the overlying regenerating epithelium. Of the four mechanisms proposed in our paper to explain the hyperopic shift we favour centrifugal contraction of lamellae resulting in a flattening of central cornea. This concept will be clarified further and evidence in its support provided when we report our photorefractive keratectomy series.

DAVID GARTRY
MALCOLM KERR MUIR
JOWAN MARSHALL
Lambeth Wing Eye Dept,
St Thomas's Hospital,
London SE1 YEH


SIR,—I read with interest the recently reported case.1 The author reported dramatic visual recovery in the second episode of anterior ischaemic optic neuropathy (AION) when pulsed IV steroid therapy (methylprednisolone, 500 mg daily for 2 days) was used, and recommends this treatment for visual loss due to AION caused by giant cell arteritis (GCA) despite its attendant mortality and morbidity.2 Clearly this recommendation can only be accepted if a clear benefit of this treatment can be demonstrated over the more conventional steroid treatment of 80-100 mg methylprednisolone orally. I would suggest there is no evidence of such benefit particularly as during the first episode of AION this patient suffered, there was an equally impressive recovery of vision on 60 mg of prednisolone orally daily. It seems reasonable to suppose that this may have again occurred had conventional therapy been used. As the author points out where there is severe visual loss from AION secondary to GCA marked improvement is rare although several other cases of substantial visual improvement using 80 mg prednisolone orally daily are reported.

Pulsed IV steroids are recommended by the standard reference textbook of ocularrpharmacology which states 'with acute visual loss of less than 36 hours duration significant visual improvement may be obtained with 'pulsed' steroid therapy using 1 gm of methylprednisolone intravenously every 12 hours for 5 days.' Only two case reports are cited as evidence to support this statement, and both are discussed by Mr Diamond. However neither is particularly persuasive.

In the case reported by Modelov vision had at no stage been formally tested. In addition prior treatment had been inadequate and of short duration (a single intravenous dose of 100 mg of hydrocortisone and no more than 20 mg of methylprednisolone orally (10 mg four times daily for 12 h). It is therefore uncertain what the degree of alteration in vision was and whether pulsed IV steroid was essential to accomplish it.

In the other case3 vision in the eye for which pulsed IV steroids were given was 2/20 before the patient was referred for a second opinion. When assessed immediately prior to pulsed IV steroids, vision was 20/40 ('slowly'), with an inferior alititudinal scotoma to 14e on Goldmann perimetry and ability to recognize only seven of 15 colour vision plates. Following pulsed IV steroids the field loss recovered and visual acuity improved to 20/25. No details are given of how quickly she read or of colour appreciation. The author claims to have 'restored vision' seems to overstate the case. More accurately they seem to have arrested moderate fluctuations of vision. It is worth emphasising that, judging from the recorded changes in visual acuity, the majority of improvement (20/70 to 20/40) had occurred before pulsed IV treatment was used (visual acuity improved from 20/40 to 20/25 following this treatment) and it may be argued that recovery was inevitable in this case.

Recovery of vision on AION due to GCA is by no means irreversible with pulsed IV steroids and progressive visual loss during therapy has been reported.3

Although appropriateness of corticosteroids in the treatment of GCA with starting doses of 80-100 mg orally is well established,1 it is apparent that the evidence to support the use of pulsed IV treatment in order to restore visual function lost from AION due to GCA is deficient. In view of the potential for adverse effects (in the age group most susceptible to adverse effects of steroid) formal critical evaluation of any therapeutic efficacy is required before recommendations for use of this treatment can be accepted.

LOUIS CLEARKIN
Barnsley District General Hospital, Gainsborugh Road, Barnsley S75 7EB


4 Clearkin LG, Caballero JC. Recovery of visual loss in giant cell arteritis (in press).