skin crease by passing the adjustable sutures into the tarsus at the level of the intended skin crease. This is a different technique from that described by Small, and allows the upper lid retractor complex not only to be removed for the treatment of lid retraction but also to be resected and advanced for the correction of ptosis. We prefer our new technique, but should have given Small credit for his very real and original contribution to the use of adjustable sutures in lid retraction.

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Effects of morphine on corneal sensitivity and epithelial wound healing

EDITOR,—Recently Peyman et al reported a fascinating study, demonstrating an analgesic effect of topically applied morphine formulation in patients with post-surgical corneal abrasions.1 Their results are in accordance with numerous clinical experiences within the past 10 years in different fields of acute and chronic pain therapy showing that a local application of opiates is useful in clinical practice. It might be of interest to Peyman and colleagues that the use of opiates as topical ophthalmic analgesics has been reported previously.

Remarking, as seen from a communication of Keil,2 ophthalmic surgery is obviously used opiates in this way more than 500 years ago. In the late 14th century the most important old German ophthalmic monograph was published. The author (probably named ’meister Johannes’), of whom little is known, cited Arabian and Middle Ages authorities such as Arnald of Villanova. In this little book there are many prescriptions for ’kräncke augen’ (sick eyes), including some local—e.g. for corneal application of opium (popppy) for surgery and pain relief. Therefore, the indisputable merit of Peyman et al’s paper is the rediscovery and scientific proving of an old medical technique.

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Selective cell death in glaucoma

EDITOR,—In his recent article J E Morgan raises various interesting ophthalmal issues. However, I would like to draw attention to several facts that I believe were not completely addressed in this article. We had previously analysed the lateral geniculate nucleus from patients with glaucoma, as well as a control population. We identified a selective diminution of cell density in the magnocellular system of glaucomatous patients. In describing our data on lateral geniculate changes in human glaucoma, Morgan suggests, first, that our technique incorrectly assessed cells per unit area rather than accurately considering volume. This methodological comment points out the tremendous difficulty in properly designing a valid stereological study. Carefully when he examines cell bodies. However, we were able to avoid this pitfall. Rather than counting the cell body per se, we only included cells in which the nucleolus could be detected. This allowed for an accurate assessment of density, given that microscopical thickness could easily be determined. The conservation of nucleolar size between magnocellular and parvocellular cells, as well as the relatively small size of the nucleolus compared with the overall thickness of the sections, minimise the technical errors he suggests. Since the microscopic section has a fixed thickness, our measurements are indeed per unit volume, and not per unit area. More importantly, Morgan suggests that we have not adequately considered the possibility that lateral geniculate cell density might actually go up in the face of ganglion cell loss. We should point out that the seminal point of our work was that we saw a differential effect on magnocellular and parvocellular tissue. No matter how you slice it (pun intended) this difference suggests that glaucoma is doing different things to the magnocellular and parvocellular systems.

The simplest explanation (supported by the majority of papers cited by Morgan) would be that if glaucoma causes some parvocellular loss (as is most certainly the case), earlier damage is done at the magnocellular level.

This brings me to a more significant comment. It is certainly possible we are wrong, and that some as yet undetermined flaw in our study (or those of other groups) has confounded the issue. However, Morgan does not cite any referred work supporting the hypothesis that glaucoma does not first damage large retinal ganglion cells, while there are many publications supporting this hypothesis. We certainly recognise that these data contradict the fondly held hypothesis that glaucomatous damage is not preferential to the magnocellular system. I would be the last to suggest our data are incriminatory, and we know that glaucoma beyond all doubt damages larger cells and therefore the magnocellular system first. But the weight of published data does support this statement. I am presented, the current answer to Morgan’s question—does selective cell damage in glaucoma occur—must be yes.

Nevertheless, we would like to reinforce one corollary of our work that was alluded to obliquely in our article. The anatomical and functional elegance of the magnocellular and parvocellular layering of the lateral geniculate nucleus has led to the seductive but unfortunately incorrect assumption that a similar simple distinction of magnocellular and parvocellular cells exist at the level of the retina. The retinal ganglion cell layer contains a plethora of cell types, and we have as yet only a limited knowledge of how these cell types function in the normal as well as the glaucomatous retina. Future psychophysical and histopathological studies will hopefully shed light on what is a most compelling question.

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Reply

EDITOR,—Dreyer discusses some important aspects of his work on the lateral geniculate nucleus in glaucoma.1 In my review I referred specifically to the volume of the geniculate laminae and not to the calculation of cell density. The finding of a differential effect on the density of geniculate cells in magnocellular and parvocellular laminae is interesting and is not what is at issue here. The point is that without reference to the lamina volume, density measurements such as those in Dreyer’s figure and changes in the number of cells in a given population.

A decrease in cell density certainly reflects cell loss (assuming that expansion of the geniculate laminae had not occurred). However, changes in cell density are the product of changes in the total cell population and laminar volume. In macaques, for example, the cell density is deafferented laminae can increase by as much as 53% but when the lamina volume is taken into account the estimated decrease in the cell population for that lamina is of the order of 22%.2 In the human, monocular enucleation results in marked geniculate cell loss3 but the change in cell density in the deafferented laminae is minimal because of laminar shrinkage. A similar process may explain why the parvocellular cell densities in Dreyer’s study did not change significantly even with the inclusion of subjects with extensive visual field damage. The differential effects of glaucoma on cell densities could reflect selectively greater cell death in the magnocellular laminae. However, caution must be exercised in drawing this conclusion without knowing the degree of laminar shrinkage.

The aim of the review was to emphasise alternative explanations of the published data on selective cell death. It is arguable whether to decide in favour of selective or non-selective mechanisms. I referred to one important paper that certainly raises questions about the role of selective cell death. Casson et al4 have detailed the mechanisms that defects occurred in both temporal modulation and blue yellow sensitivity at a similar stage in the disease, arguing against selective M or P pathway damage. Further work needs to be done to characterise the anatomical changes that occur early in

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Paecilomyces keratitis

EDITOR,—The successful treatment of a Paecilomyces keratitis described by Mizunoya and Watanabe1 raises a number of important issues. Paecilomyces as a cause of deep keratitis has been reported in a series2 of cases to which now a further two cases have been added. As described in this article, medical treatment alone is rarely successful and the only cases in which the patients were able to maintain an eye was when surgical extirpation of the corneal infection was undertaken. The lesion as depicted in Figure 1C, would have lent itself to a large eccentric corneal patch graft despite the authors’ concerns about approaching the limbus at the 6 o’clock position. Although there was an apparent beneficial effect of pulling a conjunctival flap over a perforated cornea, this procedure risks secondary glaucoma from loss of the angle result. This sequela is far less acceptable than a possible corneal graft rejection from an eccentric graft. I believe the authors were exceptionally lucky to maintain an eye with some vision. The suggestion that a conjunctival flap is an appropriate method of treatment in a perforated cornea can lead readers to emulate this treatment which will result in many disasters of corneal perforation and secondary glaucoma. The use of large corneoscleral grafts despite their many associated problems, would be a preferable method of treatment, both in eradicating the disease and maintaining a relatively normal angle anatomy, than covering an active infection and a large perforation with conjunctiva.

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Reply

EDITOR,—We appreciate the comment of Hirst concerning our case report. We agree that treating corneal defect by sclerokeratoplasty improved by Cobo et al1 will prevent secondary angle closure glaucoma. However, the sclero keratoplasty has the following problems. Firstly, because in this method a 1 to 2 mm peripheral corneal lip is left in the recipient’s eye, when the lens is close to the limbus as our case, fungi may remain in the flap. Secondly, a very thin layer of graft rejection necessitates repeated keratoplasty, which is difficult to do in Japan where donor corneal grafts are few in number. We regret that in our case anterior segment reconstruction2 after secondary keratoplasty was not enough. If it had been done thoroughly, trabeculocentrone might have been unnecessary.

Keratocoryne with perforation is a very severe condition, and there is no reliable treatment to salvage an eye. We would select primary keratoplasty, sclerokeratoplasty or the method we reported according to the position of the perforation, the size of it, and the activity of the lesion.

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Could colour vision tests predict or find retinopathy in diabetic schoolchildren?

EDITOR,—Only a few studies concerning colour vision of diabetic children are available.1-3 In them, colour vision has been found to be normal, and a follow-up study of diabetic children and young adults showed that colour vision (examined with the Farnsworth-Munsell 100 hue test) deteriorated with the development of retinopathy.4 No significant difference was found between diabetic children and young adults with and without retinopathy. 4 Distinguishing retinopathic eyes from those without retinopathy has not been studied with colour vision tests in this age group.

We have observed colour vision of diabetic schoolchildren, aged 4-19 years, from 1987 to 1993. Answers for two questions were sought: (1) Would the results of colour vision tests in diabetic children without retinopathy in 1987 predict the development of retinopathy during the next 6 years? (2) Would the results of colour vision tests in 1993 distinguish the eyes with retinopathy from those without retinopathy?

(1) The follow up study 1987-1993. In 1987 colour vision of 54 diabetic schoolchildren with healthy eyes was studied (29 girls, 25 boys; aged 9-19 years (mean 14 (2 SD) years); duration of diabetes from 1 month to 15 years, mean 6 (4) years. Colour vision was examined with the Farnsworth panel D 15, Lanthony desaturated panel, and Nagel anomaloscope. The panel D 15 was correctly interpreted by all of the children. In the desaturated panel, seven children did not pass the test showing 1-3 mistakes on the Farnsworth lines. In the Nagel anomaloscope examination, normal anomalous quotients (AQ, from 0-7 to 1-2) and normal matching ranges (MR, from 0 to 6) were observed in all children. Fifty five eyes had an AQ completely on the red side of the Rayleigh equation. (The predictive value of the red side MR for the appearance of retinopathy has been suggested.)

In 1993, 23 of the children (35 eyes) had retinopathy, which in 33 eyes was background retinopathy. None of these eyes had macular involvement. Only one patient had proliferative retinopathy with macular oedema in both eyes. Of those seven children who did not pass the desaturated panel test in 1987, only two developed background retinopathy in one eye. However, all these seven children passed the desaturated panel in 1993. The patient with proliferative retinopathy passed the test normally in 1987. Of the 56 eyes with a red side MR in 1987, 16 (29%) had developed background retinopathy, 40 (71%) had intact retinas. Of the 52 eyes with a green side or mixed red-green side MR, 19 (37%) had developed retinopathy, 33 eyes had intact retinas.

(2) The cross sectional study 1993. In addition to the above mentioned colour vision tests, the blue equation of another anomaloscope: colour vision meter (CVM) 712 was taken on the three of the panel. Fifty of the eyes had the panel D 15 test and the desaturated panel test. Only the 23-year-old man with proliferative retinopathy and macular oedema in both eyes could not correct interpret the panel D 15 test. Four of the eyes had AQs within normal limits. The Nagel anomaloscope results, AQs and MRs, were all within normal limits. In the CVM, four diabetic patients (four eyes) had an abnormal AQ, >1-4. One of the eyes had no retinopathy, two eyes had background retinopathy, and one eye had proliferative retinopathy. All the other 31 eyes with retinopathy (one of them with proliferative retinopathy) had AQs within normal limits. Ten of the retinopathic eyes showed an anormal MR in the CVM, from 11 to 26, mean 18 (5). Also 10 non-retinopathic eyes had an abnormal MR, from 11 to 21, mean 15 (6). No significant difference was found between these means.

Conclusion. The 6 year follow up study in colour vision of diabetic schoolchildren showed that no predictive signs for retinopathy could be found with the desaturated panel or Nagel I anomaloscope test results. In the cross sectional study, it was observed that the eyes with and without retinopathy in diabetic schoolchildren and young adults could not be distinguished from each other with the results of four colour vision tests: panel D 15, desaturated panel, Nagel I anomaloscope, and colour vision meter 712 (blue equation). Only the two eyes with non-proliferative retinopathy with macular oedema could be found with the panel D 15 test.

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Selective cell death in glaucoma.

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