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 anaesthetics has been reported previously.

Remarkably, as seen from a communication of Keil,2 ophthalmic surgeons 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 Arabic and Middle Ages authorities such as Arnold of Villanova. In this little book there are many prescriptions for 'kräncke augen' (sick eyes), including some local—e.g. corneal application of opium (popp) 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 ophthalmic points. 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, and identified a selective diminution of cell density in the magno-cylindrical cell system of glaucomatous patients. In describing our data on lateral geniculate changes in human glaucoma, Morgan suggests, first, that our technique incorrectly assayed cells per unit area rather than accurately considering volume. This methodological comment points up the tremendous difficulty in properly designing a valid stereological study, and clearly when one 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 microscopic thickness could easily be determined. The conservation of nuclear size between magno-cylindrical and parvocellular cells, as well as the relatively small size of the nucleolus compared with the overall thickness of the sections, minimises 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 paper was that we saw a differential effect on magno-cylindrical and parvocellular tissue. No matter how you slice it (pun intended) this difference suggests that glaucoma is doing different things to the magno-cylindrical and parvocellular systems.

The simplest way to interpret this (supported by the majority of papers cited by Morgan) would be that even if glaucoma causes some parvocellular loss (as is most certainly the case), earlier damage is done at the magno-cylindrical level.

This brings me to a more significant comment. It is certainly possible we are wrong, and that some as yet underdetermined flaw in our study (or those of other groups) has confounded the issue. If 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 glaucomatosus damage is not preferential to the magnocellular system. I would be the last to suggest selective cell death is necessarily definitive, and that we know that glaucoma beyond all doubt damages larger cells and therefore the magno-cylindrical system first. But the weight of published data does support this stance. Therefore, as data are 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 magno-cylindrical and parvocellular layering of the lateral geniculate nucleus has led to the seductive but unfortunately incorrect assumption that a similar simple distinction of magno-cylindrical 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 magno-cylindrical 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 cannot accurately reflect changes in the number of cells in a given population.

A decrease in cell density certainly reflects cell loss (assuming that expansion of the laminae had not occurred). However, changes in cell density are the product of changes in the total cell population and lamina 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 loss.4 The differential effects of glaucoma on cell densities could reflect selectively greater cell death in the magno-cylindrical 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 that oppose the differential death hypothesis and 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 al 5 have described the 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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4 Casset EJ, Johnson CA, Shapiro L. Longitudinal
comparison of temporal modulation perimetry
with white on white and blue on blue for
trying in ocular hypertension and early glance.

Paecilomyces keratitis

EDITOR,—The successful treatment of a
Paecilomyces keratitis described by Mizuno
y 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
is rarely successful and the only cases in which
the patients were able to maintain
an eye was when surgical excision 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 posi-
tion. Although there was an apparent benefi-
cial effect of pulling a conjunctival flap over a
perforated cornea, the probable result of
secondary glaucoma from loss of the angle
resulted. This sequela is far less acceptable
than a possible corneal graft rejection from an
eccentric graft. I believe the authors were
exceptionally fortunate to manage an eye with
such vision. The suggestion that a conjuncti-
val flap is an appropriate method of treatment
in a perforated cornea may lead readers to
emulate this treatment which will result in
many disasters of confusing infection with
secondary glaucoma. The use of large cor-
neoscleral grafts despite their many associ-
ated 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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tival flap and delayed keratoplasty. Br J
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Reply

EDITOR,—We appreciate the comment of
Hirst regarding our case report. We agree
that treating corneal defect by sclerokerato-
plasty improved by Cobo et al1 would prevent
secondary angle closure glaucoma. However,
the sclerokeratoplasty has the following prob-
lems. Firstly, because in this method a 1 to 2
mm peripheral corneal lip is left in the recipi-
ent's eye, when the lesion is close to the
limbus as our case, fungi may remain in the
lip. Secondly, a high incidence of graft
rejection necessitates repeated keratoplasty,
which is difficult to do in Japan where donor
corneal grafts are few in number. We regre-
that in our case anterior segment recon-
struction with secondary keratoplasty
was not enough. It had been done
thoroughly, trabeculectomy might have been
unnecessary.

Keratomycosis 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 posi-
tion 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 avail-
able.1-3 In them, colour vision has been found
to be normal, and a follow up study of dia-
abetic children and young adults showed that
colour vision (examined with the Farnsworth-
Munsell 100 hue test) deteriorated with the
development of retinopathy insignificantly.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 with retinopathy during 6
years, from 1987 to 1993. Answers for two
questions were sought: (1) Would the results
of colour vision tests in diabetic children with-
out retinopathy in 1987 predict the develop-
ment 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? We did this study in
schoolchildren who did not pass the
desaturated panel test in 1987, only two
developed background retinopathy in one
year. 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%) still had
intact retinas. Of the 52 eyes with a green side
or mixed red-green side MR, 19 (37%)
background and two proliferative, 37% had
developed retinopathy, 33 eyes had intact
retinas.

(2) The cross sectional study 1993. In addi-
tion to the above mentioned colour vision
tests, the blue equation of another anamalo-
scope: colour vision meter (CVM) 712 was
available. A 3% of the eyes had an abnor-
mv AQ > 1.4. One of the eyes had no
retinopathy, two eyes had background
retinopathy, and one eye had prolifera-
tive retinopathy. All the other 31 eyes with
retinopathy (one of them with prolifera-
tive retinopathy) had AQs within normal limits.
Ten of the retinopathic eyes showed an abnormal 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 (4). No 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 retino-
pathy could be found with desaturated panel
desaturated panel or Nagel anamaloscope
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 anamaloscope, and colour vision
meter 712 (blue equation). Only the two eyes
with proliferative retinopathy with macular
oedema could be found with the panel D 15 test.

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