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 recessed 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.

J R O COLLIN
Moorfields Eye Hospital,
City Road,
London EC1V 2PD

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.

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 Arnald of Villanova. In this little book there are many prescriptions for 'krankte augen' (sick eyes), including some local – for example, corneal application of opium (poppy) 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.

CHRISTOPH MAIER
Clinic for Anesthesiology and
Operative Intensive Care,
University of Kiel Pain Clinic,
D24105 Kiel,
Germany

1 Peyman GA, Rahimy MH, Fernandes ML. Effects of morphine on corneal sensitivity and epithelial wound healing: implications for topical ophthalmic analgesia. *Br J Ophthalmol* 1994; 78: 138–41.

2 Keil G. Pommersfeldener (schlesisches) Augenbüchlein. In: Ruh K, Keril G, Schröder W, Wachinger B, Worstbrock, eds. *Die deutsche Literatur des Mittelalters. Verfasserlexikon*. Vol 7. Berlin: Dr Gruyter, 1989, 778–9.

Reply

EDITOR,—We are flattered by Christoph Maier's letter regarding our paper. As they say, nothing is new under the sun! However, not having access to the German book that Maier cites, it is difficult to formulate an educated response. I believe, nevertheless, that the old literature has not alluded to the difference in analgesic effect of morphine between denuded and normal cornea and will not have comments regarding normal epithelial wound healing after application of this analgesic agent.

GHOLAM A PEYMAN
LSU Eye Center,
2020 Gravier Street,
Suite B, New Orleans,
LA 70112-2234, USA

Selective cell death in glaucoma

EDITOR,—In his recent article J E Morgan raises various interesting theoretical points.¹ 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.² 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 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, particularly 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 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, 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 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 conclusion (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 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. But Morgan does not cite any refereed 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 that our data are unquestionably definitive, and that 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, and until new 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 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.

EVAN B DREYER
Harvard Medical School,
Massachusetts Eye and Ear Infirmary,
Department of Ophthalmology,
Glaucoma Consultation Service,
243 Charles Street, Boston,
MA 02114-3096, USA

1 Morgan JE. Selective cell death in glaucoma: does it really occur? *Br J Ophthalmol* 1994; 78: 875–80.

2 Chaturvedi N, Hedley-White ET, Dreyer EB. Lateral geniculate nucleus in glaucoma. *Am J Ophthalmol* 1993; 116: 182–8.

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 per se do not indicate 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 glaucomatous 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 in glaucoma and not 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*⁴ found 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