High prevalence of lactase absorbers in patients with presenile cataract from northern Sardinia

EDITOR,—An elevated incidence of cataract has been observed by Simoons et al. in high milk drinking populations. Other workers have found that the percentage of lactase absorbers among subjects with cataract was higher than in the normal population. We studied 40 adult male subjects with cataract (they were aged less than 56 years and more than 44 years), and 50 healthy similarly aged control males from northern Sardinia. Milk drinking habits were similar for the two groups. None of the patients was affected by cataract due to congenital, inflammatory, iatrogenic, diabetic, traumatic factors, or other causes. Thirty five of the 40 cataractous subjects had bilateral cataract, 23 of whom had nuclear, seven cortical posterior, six corona, two posterior polar, and two zonular cataract. All the subjects were given a 50 g oral load of lactose at a 15% water solution; the hydrogen breath concentration in the expired air was measured in each subject before the lactose oral load and at 15 minute intervals for the next 4 hours using a Quintron 121 gas chromatograph. Lactose malabsorption was diagnosed if the maximum increase in hydrogen in the expired air was more than 20 parts per million. Only seven (14%) of the 50 normal subjects were lactose absorbers compared with 16 (40%) of the 40 cataractous subjects. The χ² test revealed a significant difference (p=0.01) in the frequencies of the two groups. One fact that must be emphasized is that all the lactose absorber cataractous patients had bilateral nuclear cataract; the difference between the frequency of this subgroup and the control group is highly statistically significant. These results confirm that in our region (northern Sardinia), characterized by high milk drinking habits, adult lactase absorbers are more prone to develop presenile cataract than non-lactose absorbing individuals.

GIANFRANCO MELONI AUGUSTO OGANA MARIA CRISTINA MANNI TULLIO MELONI Clinica Pediatrica FRANCO CARTA ADOLFO CARTA Clinica Oculistica, Università di Sassari, Viale San Pietro 12, 07100 Sassari, Italy


In defence of goniometry

EDITOR,—O’Connor’s editorial states that ‘primary trabeculotomy has replaced goniometry as the preferred surgical treatment for congenital glaucoma.’ To support this statement he cites Burke and Bowell’s report1 of an 87% success rate for primary trabeculotomy based on 13 eyes with primary infantile glaucoma and a mean follow up of 3.9 years.2 Rice has reported an 86% success rate (211/246 eyes) for goniometry for eyes with primary infantile glaucoma which in the majority (73%) of cases only required a single procedure.3 We reported a large long term follow up study of Rice and Lester’s patients with trabeculodysgenesis (339 eyes) in which we showed that 92% of eyes controlled in infancy by goniometry were still controlled after 5 years of follow up; however, Kaplan- Meier actuarial survival curves demonstrate that there is a risk of relapse throughout life.4 The risk of relapse in eyes having undergone trabeculotomy in childhood is significant especially if antiglaucoma agents are not used.5 The advantage of a primary goniometry is that the conjunctiva is preserved for any future drainage surgery for individual children, if they are to have a drainage operation, it will be when they are older and young age is a significant negative factor in bleb survival. Also there will hopefully have been advances in both surgery and in agents used to improve bleb survival.

O’Connor also cites Miller and Rice6 as advocating primary trabeculotomy. This appears to be a misquote as their paper reports trabeculotomy being performed in eyes with relapse or failure of control of congenital glaucoma after goniometry or trabeculotomy.

My remarks refer entirely to eyes with typical primary congenital glaucoma (trabeculodysgenesis in the Hoyos classification), which is the commonest form of congenital glaucoma seen in the UK and to the results for a surgeon experienced in the technique of goniotomy. Examination of the angle is crucial when planning the surgical approach. Goniometry should never be performed without a view of the angle. However, a view of the angle can be usually achieved by removal of the corneal epithelium. O’Connor states that goniometry cannot be performed in approximately 50% of cases of congenital glaucoma because of corneal opacity. However, we found that in addition to the 211 patients reported in our study to have been treated by goniometry, only a further 20 patients (<10%) with trabeculodysgenesis were seen during the same time but were excluded from our paper because the initial surgery was not goniotomy. In most of these cases a primary trabeculotomy was performed as an adequate view of the angle could not have been achieved because of corneal clouding. Trabeculotomy can be performed at a temporal, or even superior fornix, to preserve the superior bulbar conjunctiva. The congenital glaucoma which occurs in Middle Eastern countries is more often familial than it is in the West7 and may respond less well to goniotomy. O’Connor makes no reference to the eye angle anomaly (personal observation). I note that Elder’s paper in the same issue of the BJ OJO relates to his experience in Jerusalem.8 It may be that in this population and in eyes with iridotrabeculodysgenesis with infantile onset glaucoma, combined trabeculotomy-trabeculotomy may be a useful operation. I, therefore, read his paper with interest and look forward to him reporting a longer follow up.

ISABELLE RUSSELL-EGGITT Great Ormond Street Hospital, London WC1N 3JH


Adjustable sutures in eyelid surgery

EDITOR,—I would like to comment on the article by J R O Collin and E A O’Donnell.1 In that article, they state that their technique of postoperative adjustment of lid height is new. Work I have done shows that the adjustable suture technique was introduced in 1982 and has been used with modifications for 12 years.2

ROBERT G SMALL
Dean A McGee Eye Institute, 608 Stanton I Young Drive, Oklahoma City, OK 73104, USA


Reply

EDITOR,—In 1982, Small used a posterior approach technique to excise Muller’s muscle and recess the aponeurosis with two monofilament polypropylene mattress sutures passed through the muscle. In 1988, he modified the technique to an anterior approach, cut the levator muscle above Whitnall’s ligament and held it resected with a 6/0 monofilament polypropylene mattress suture brought through the skin. The suture is adjusted if necessary immediately after recovery from anaesthesia but by bringing the suture through a rubber cylinder cut from the edge of a surgical glove, the adjustment can be modified during the first postoperative week before finally tying the suture.

His paper shows some very impressive results and our recent paper confirms that adjustable sutures are very effective in treating lid retraction. We recess Muller’s muscle with the aponeurosis and the levator muscle via an anterior approach and maintain the.
Selective cell death in glaucoma

EDITOR,—In his recent article J E Morgan raises various interesting medical 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. 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 up the tremendous difficulty in properly designing a valid stereological study, 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 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 way to show 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 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 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, based on these data, that retina is not definitively, 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 stance. When data are as 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 Infrmary,
Department of Ophthalmology,
Glaucoma Consultation Service,
243 Charles Street, Boston,
MA 02114-3996, USA

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 may lead to inaccurate 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 lamina volume. In macaques, for example, the cell density is deaffiliated 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 loss 3 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 and shrinkage. 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 and I would hope that it should encourage researchers 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 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