Correspondence

Behaviour of the fellow eye in acute angle-closure glaucoma

SIR, In his article Mr R. S. Edwards¹ is mistaken in assuming that because he had a sample of fellow eyes the results derived from a nonconcurrent prospective study have general application. It all depends on what happened to the sample.

The study started with 220 fellow eyes (the 25 patients who presented with bilateral closed-angle glaucoma cannot be included for the reason that there was no fellow eye to study) of which 23 (10%) developed closed-angle glaucoma within a few days of presentation. During that interval, too, 168 fellow eyes had a peripheral iridectomy. From the information given in the paper there is no reason to suppose, nor evidence to suggest, that the decision to do an iridectomy was made randomly. Therefore while the sample of eyes at the start may have been random, the sample of eyes left for study after discharge from hospital was at best haphazard and at worst biased. The samples remaining for study at 1–12 months and at 12–48 months became even more haphazard (or biased) as time passed. Furthermore the pre- and postoperative treatment regimens were not randomly allocated, and each eye had at least one of 8 different treatments, viz:

(1) Carbonic anhydrase inhibitors plus pilocarpine; (2) pilocarpine 2% or stronger four times daily; (3) other pilocarpine regimen; (4) no treatment; (5) pilocarpine fellow eye, no mydriatic operated eye; (6) pilocarpine 2% or stronger 4 times a day fellow eye, mydriatics operated eye; (7) other pilocarpine regimen fellow eye, mydriatic operated eye; (8) no pilocarpine fellow eye, mydriatic operated eye. An additional complication was introduced by using 4 different mydriatics. To suppose that a sample manipulated in this fashion can yield useful information is totally to ignore the methodology of science.

Even if it is assumed that treatments had been in some way randomly allocated, the author's conclusions are not supported by the data he presents. On page 578 he writes: 'Once unoperated eyes had been followed-up for more than one month, and mydriatic drops to the operated eye had been stopped, the liability to an acute ACG attack became less.' In fact if the proportion of eyes getting closed-angle glaucoma before discharge from hospital (23 out of 220), during the interval 0–1 month after discharge (7 out of 53) and thereafter (3 out of 35) is compared, then there is no significant difference between the 3 groups ($\chi^2=0.53$, $p>0.7$). Such differences as there are can be ascribed to sampling variation and not to different susceptibilities. The probability of any one fellow eye developing closed-angle glaucoma in each of the 3 intervals was therefore the same; what was different was the time lapse before the event occurred. Again, in the summary it is stated that pilocarpine and carbonic anhydrase inhibitors confer the best protection on the fellow eye. From the data recorded in Table 3 the author has produced no evidence to show that there is any statistically significant difference in the number of eyes getting glaucoma between any of the treatment

groups. Because of this there is no reason not to ascribe the results to sampling variation.

This article has nothing to say about fellow eyes in general. It is relevant only to 220 fellow eyes allocated one or more of a dozen or so treatments, in a nonrandom way, in one hospital, between January 1972 and December, 1978. St Paul's Eye Hospital,
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R. MAPSTONE

Reference

- 1 Edwards RS. Behaviour of the fellow eye in acute angle-closure glaucoma. *Br J Ophthalmol* 1982; **66**: 576–9.

SIR, My paper¹ is a retrospective study of the fellow eyes of 245 patients admitted as an emergency with primary acute closed-angle glaucoma (ACG).

The 25 patients with bilateral acute ACG do merit inclusion in this series. They help to illustrate points I make in my paper concerning the early involvement of the fellow eye and the role of mydriatics. Of these 25 patients 7 had bilateral acute ACG after receiving mydriatics, though of these 7, 6 had been dilated with Mydrilate (cyclopentolate HCL) 1% and phenylephrine 10% and constricted with pilocarpine 2%—a situation similar to the provocative test described by Mapstone.² Eight patients gave a history of one eye being involved before the other prior to admission. Five gave a history of simultaneous onset of symptoms, and in 5 others no reliable history was obtained.

As regards the decision to perform surgery during the time studied, 2 consultants routinely performed bilateral simultaneous surgery on all patients admitted with acute ACG. The other consultants operated on the fellow eye on their next available operating list (hence the mean time interval of 6.7 days between acute and fellow eye surgery). A decision to discharge a patient with acute ACG rather than operating on the fellow eye was based on a variety of reasons, but the chief one was unsuitability for further surgery on the grounds of the patient's general condition. None of these fellow eyes was deemed safe from the risk of subsequently developing acute ACG. In view of these facts I do not consider my samples of unoperated fellow eyes to be biased.

Mr Mapstone's list of 8 different treatments is misleading. Preoperatively and postoperatively treatment regimens for fellow eyes fell into 4 distinct groups as outlined in Tables 2 and 3 of my paper. No mydriatics were given preoperatively and no carbonic anhydrase inhibitors postoperatively. Individual consultants varied in their preferences for different medicaments, but these were generally consistent. In as much as the consultant the patient was admitted under was determined randomly, so also was the treatment randomly allocated. Although it would have been preferable for only one type of mydriatic to have been used postoperatively for the acute eye, this was obviously not possible in what was, I emphasise, a retrospective study.

Mr Mapstone's statistical analysis of my figures only affects my conclusions if the time intervals for the various periods studied (as cited in table 2) are the same. This is not the case. By expressing my figures in terms of attack rates I

have emphasised the greater susceptibility of the fellow eye to ACG in hospital and immediately after discharge.

On the subject of medication, it is notable that no fellow eyes developed acute ACG when mydriatics were withheld from the operated eye. Although numbers are small (16 out of 187 eyes in hospital and 14 out of 52 eyes at initial follow-up), I think they are sufficiently large to warrant further study. As regards pilocarpine treatment, this possibly has a protective effect preoperatively (Table 3: $\chi^2=3.17$ $p>0.05$ but less than 0.1) and certainly has a protective effect postoperatively (Table 4: $\chi^2=16.7$, $p<0.001$) (Yates's correction applied for both calculations) consistent with Bain's observations in 1957.³ Although my data suggest that carbonic anhydrase inhibitors may have a protective effect, I concede that this cannot be established with certainty from my study.

I would like to emphasise that no medical regimen entirely prevented acute ACG in the fellow eye. In this observation I am supported by Bain,³ Lowe,⁴ and Mapstone.² In Mapstone's study of 202 fellow eyes² 9 developed acute ACG spontaneously in spite of having a negative pilocarpine phenylephrine provocative test.

In answering Mr Mapstone's letter I therefore still maintain that my large retrospective study does provide additional useful information on the behaviour of the fellow eye.

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References

- 1 Edwards RS. Behaviour of the fellow eye in acute angle-closure glaucoma. *Br J Ophthalmol* 1982; **66**: 576-9.
- 2 Mapstone R. The fellow eye. *Br J Ophthalmol* 1981; **65**: 410-3.
- 3 Bain WES. The fellow eye in acute closed-angle glaucoma. *Br J Ophthalmol* 1957; **41**: 193-9.
- 4 Lower RF. Acute angle-closure glaucoma: the second eye—an analysis of 200 cases. *Br J Ophthalmol* 1962; **46**: 641-50.

Book reviews

Limbusprobleme. By WILHELM DODEN. Pp. 115. DM 44. Ferdinand Enke Verlag: Stuttgart. 1982.

The 88th volume in the series 'Bucherei des Augenarztes' consists of a collection of articles on the limbus. These include a description of the anatomy and physiology and discussions on inflammations, injuries, degenerations, and tumours of this region. The contributions derive from a symposium held in Frankfurt in 1977, and the authors have been requested to bring the text up to date and to expand the illustrations. The result is an interesting, well-illustrated portrayal of a specific area of the eye in health and disease which makes a welcome change from the more conventional textbook approach. The high standards of this on-going series are maintained, and the book may be recommended for ophthalmologists of all levels.

T. J. FFYTCH

Diagnosis and Management of Uveitis: An Atlas Approach. By ALAN H. FRIEDMAN, MAURICE LUNTZ, WALTER L. HENLEY. Pp. 116. US\$95.00. Williams and Wilkins: Baltimore. 1982.

In the field of uveitis most ophthalmologists still despair of ever seeing any sort of order in the confusion of clinical syndromes ranged under this heading. Despite there being no dramatic new advances in either diagnosis or treatment a general text presenting a thorough review of current concepts would be welcome. I received this book hoping it might fill this purpose, but unfortunately it does not. It is a slender volume of 116 pages, more than one-third of which bear colour illustrations. The type is clear and there are very few errors in the text. Although the style is easy to read, the text is frequently interrupted by tables which could be more smoothly related to the text. One often has to turn forward several pages to take up the text line, and there are many irritating half empty pages. There is a useful chapter of basic immunological concepts and a brief discussion of most types of uveitis and their treatment. The imbalances in the book presumably reflect the particular interests of the distinguished authors.

It is not, however, to be judged as a comprehensive text but in the light of its illustrations, since these justify its title and undoubtedly account for its high cost. Many are excellent, especially in those cases where pathological sections are related to the clinical photographs. On the other hand there are many which fail to make their point clearly, if at all, and one or two which seem to be unrelated to their captions. Many of the illustrations seem superfluous, and one wonders what criteria were used in selection.

W. J. DINNING

Pediatric Optometry. Ed. JEROME ROSNER. Pp. 458. £34.00. Butterworths: Sevenoaks, Kent. 1982.

It is of course perfectly sensible that there should be a movement towards specialisation in the optometry of children and that there should be a book on paediatric optometry to help those who are less familiar with the needs of children. Professor Rosner's book is logically laid out, being divided into 2 basic parts: examination and diagnosis, and the postexamination process. The description of history taking and defining the child's problem is rather overburdened with detail, and while the author warns the reader not to overstep his professional skills he then goes into detail about the relevance of genetic, prenatal, perinatal, and postnatal factors, augmenting the text with turgid lists of names of conditions which may affect children, some so rare as to be unfamiliar to most paediatric ophthalmologists. The section on examination of the vision is relevant, and there are good descriptions on the examination of binocular vision and refraction.

The chapter on examination of the eye and adnexae is a perfect demonstration of the need for closer co-operation between ophthalmologists and optometrists and an end of the dreary cold war between the 2 professions. In this chapter the author lists a series of conditions that could affect the eye of the child. There are no illustrations, and asking the reader to stay awake while reading lists of names such as euryblepharon, posterior lenticonus, Argyll