LETTERS TO THE EDITOR

Keratitis in Reiter's syndrome

SIR,—We describe a patient with Reiter's syndrome who presented with a bilateral anterior stromal keratitis with a ragged corneal epithelial defect in one eye. These findings are a characteristic but rare and poorly recognised complication of Reiter's syndrome.

Case report

A 28-year-old man presented with 1 week's

Figure 1 Anterior stromal infiltrates and punctate epithelial erosions in the patient's right eye.

Figure 2 Anterior stromal infiltrates in the left eye.

Figure 3 Ragged epithelial defect in the left eye.

history of redness, pain, and blurring of the vision of both eyes. One month previously the eyes had been red with a mucopurulent discharge but these symptoms had resolved spontaneously over a period of 7 days.

Two months prior to the onset of his ocular symptoms he had had a diarrhoeal illness, followed shortly by polyarthralgia, and a diagnosis of Reiter's syndrome was made by a rheumatologist.

At the time of his initial ophthalmic examination he had coarse anterior stromal and epithelial infiltrates in both corneas (Figs 1 and 2), fine punctate erosions in the right eye, and a large, ragged epithelial defect in the left (Fig 3). He had fine conjunctival papillae in both eyes.

He was treated with guttae chloramphenicol 0.5% and guttae prednisolone 0.3% each four times daily. Within 1 month of presentation the keratitis had resolved completely without scarring.

Comment

Common ocular manifestations of Reiter's syndrome include conjunctivitis and iridocyclitis but, although it is mentioned by Duke-Elder,¹ keratitis is not well recognised as a complication by most ophthalmologists.

Keratitis, similar to that described here, was reported in 1972 by Mills and Kalina,² and more recently by Wiggins *et al.*³ Lee and coworkers reported superficial punctate keratitis and pleomorphic anterior stromal infiltrates in 4% of patients with Reiter's syndrome in their study.⁴

Our case demonstrates all the clinical features of an established case of keratitis in Reiter's syndrome.

Anterior stromal keratitis, affecting mainly the peripheral cornea, and associated with ragged epithelial erosions, is a rare but characteristic finding in Reiter's syndrome. A prodromal conjunctivitis is usual as is spontaneous resolution.

NEIL J ROWSON
JOHN K G DART
Moorfields Eye Hospital,
City Road,
London EC1V 2PD

- 1 Duke-Elder, Sir Stewart. *System of ophthalmology*, Volume VII, Part 1. London: Kimpton, 1965: 530.
- 2 Mills RP, Kalina RE. Reiter's keratitis. *Arch Ophthalmol* 1972; 87: 447-9.
- 3 Wiggins RE, Steinhuller PG, Hamill MB. Reiter's keratoconjunctivitis. *Arch Ophthalmol* 1990; 108: 280-1.
- 4 Lee DA, Barker SM, Su WPD, *et al.* The clinical diagnosis of Reiter's syndrome. *Ophthalmology* 1986; 93: 350-6.

Statistical standards

SIR,—I was recently invited to give a talk to the Department of Ophthalmology in Oxford on statistical methods in eye research. I chose to structure my talk around a review of the standard of statistics in articles published in the *BJO* and I thought that your readers might be interested in my findings.

I reviewed all of the articles published in the *BJO* during the first 6 months of 1990 concentrating on their statistical content including design, presentation, analysis, and interpretation. I identified 42 articles that had employed a statistical analysis and a further seven where I thought that a statistical analysis would have been helpful but was not given. The methods used were typically very simple and often just consisted of tests such as the *t* test or χ^2 test.

Any assessment of quality is bound to contain a large subjective element but even allowing for this the standard was a little

disappointing. I judged that 26 of the papers were either acceptable as they stood or needed only very minor changes perhaps of clarification. The remaining 23 contained what I would consider to be statistical errors. These errors ranged from the omission of important details to faulty design and the misuse of statistical tests. In very few cases were the errors so bad that the paper could not have been corrected, sometimes quite simply. However the errors do detract from the quality of the work and leave the reader in doubt about the conclusions.

These findings might at first sight seem surprisingly poor but they are very much in line with the results of similar reviews of published articles from journals covering other branches of medicine. The problem is well recognised and widespread, a solution is more difficult to find.

It is worth emphasising that the problem does not lie in the misuse of complex statistical techniques but rather in weak scientific method and a haziness about the underlying principles of statistical analysis. It is easy to point to poor medical education as the root of the problem, but medical students often do not see the need for courses in statistics and so pay comparatively little attention to them.

Perhaps it is up to the medical journals to take a lead by insisting on higher standards and by using referees who are trained in statistics.

JOHN THOMPSON
University of Leicester,
School of Medicine,
Clinical Sciences Building,
Leicester Royal Infirmary,
PO Box 65,
Leicester LE2 7LX

** Many thanks to Dr Thompson for his interesting letter which contains much food for thought. It emphasises the gap which exists between clinicians and professional statisticians, a gap which it would be good to narrow even perhaps to the extent of rendering redundant some of the old well-worn jokes about statistics. One is aware however from many conversations with responsible and experienced clinicians that suspicion about statistics dies hard and there are still numerous ophthalmologists who believe that complicated statistics are sometimes used to attempt to prove theories which, if they were really true, would be obvious by commonsense alone.

From my point of view as editor I am, although suitably chastened, at least glad to see that we at the *BJO* are not alone in publishing some papers with statistics which do not satisfy the professionals try as we may to keep authors on the straight and narrow. Looking around at some of our sister publications I should not be surprised if some of them were even worse than us but unfortunately these figures are not vouchsafed to us in the current letter.

On a positive note: we should very much welcome an article from Dr Thompson on the subject of good statistical practice in ophthalmic papers. The last we published, admittedly on a restricted aspect of the subject, the confusion of 'eyes' versus 'patients' in ophthalmic statistics, was back in 1987.¹ — ED, *BJO*.

- 1 Newcombe RG, Duff GR. Eyes or patients? Traps for the unwary in the statistical analysis of ophthalmological studies. *Br J Ophthalmol* 1987; 71: 645-6.

Amblyopia

SIR,—I congratulate Drs Lithander and Sjöstrand on their excellent study of amblyopia and the results of treatment.

Having been involved with the treatment of amblyopia for more than 30 years my views have altered very little.

My work reported in 1964 still stands that a monitored period of total occlusion overcomes amblyopia quickest and best. As improvement occurs one can modify the techniques to part time, or partial, to achieve the final result.

However in the past 10 years or more I found the best answer was prevention of amblyopia and this was achieved by early peripatetic orthoptic screening in postnatal, infant, and preschool clinics.

This meant that squints and amblyopia were detected very early and, with prompt referral, required less specialist treatment; and amblyopia was only relative in depth and required only minimal occlusion to obtain a cure. Indeed most children were detected before amblyopia (as defined) had had time to occur.

I would urge the need to continue to strive for early screening and public education before the age of 4 years contrary to the Hall report.

G V CATFORD
11 Devonshire Place,
London W1N 1PB

1 Hall DMB. *Health for all children*. Oxford: Oxford Medical Publication, 1989.

Cataract surgery

SIR,—The problem of the enormous backlog of cataract surgery in the third world will not I fear be solved by the methods advocated by Mr Arthur Steele.¹

I was fortunate to get my surgical training in Pakistan where my colleagues routinely did 150 cataract operations single-handedly in a 6 h session. Their technique was a Graefe knife incision, peripheral iridectomy, intracapsular lens extraction with forceps, or cryosurgery and one to three corneal sutures with 8/0 silk. The surgery was of superb quality and the results were excellent.

If we had performed the surgery with microscopes, three-stage incisions, IOLs, running 10/0 sutures, etc, then I suspect the rate would have fallen from 20–30 operations an hour to two to three an hour. The resulting cost would be so high that only the wealthy could afford treatment.

No-one would try to ignore the brilliant advances in ophthalmic surgery developed in the west. However I would urge my colleagues there to accept that for huge numbers of cataract blind in Africa and Asia such techniques may not be the most appropriate and that simple methods in expert hands can give excellent results at low cost.

IAN THOMSON
Agogo Hospital,
PO Box 27,
Agogo, Ghana

1 Steele A. Cataract surgery. (Editorial.) *Br J Ophthalmol* 1990; 74: 130–1.

Dominant cone dystrophy starting with blue cone involvement

SIR,—The authors of the paper¹ in vain looked for a blue gene defect. The blue-yellow colour vision defect they observed however finds its origin in the physiology of the retina. Pathology of the macular region, if fixation is preserved, gives rise to blue-yellow defectiveness regardless of the cause: X-linked or dominant inherited cone dystrophy, pigmentary dystrophy, intoxication by synthetocal antimalarial agents, and so on.²

Cone dystrophies start either foveolar or perifoveolar. The latter type is known as peripheral cone dysfunction and is characterised by a disturbed full field cone ERG, a relatively well preserved visual acuity and normal colour vision or an acquired type III blue-yellow colour vision defect.^{3,4} Static perimetry reveals a perifoveolar dip.² The blue-yellow defect, often dichromatic, is an exaggerated small field tritanopia. Case VI.6 is a good illustration. This patient could not read the 8° plate tests but made a classic tritan response with the 2° Panel D-15 test.

The dystrophic process affects the perifoveolar cones, including the blue cones which have their maximum density at 1.5° eccentricity. The blue cone-free foveolar region becomes enlarged resulting in a strong decrease in blue sensitivity.

A PINCKERS
Department of Ophthalmology,
St Radboud Ziekenhuis,
Philips van Leydenlaan 15,
6500 HB Nijmegen, The Netherlands

- 1 Van Schooneveld MJ, Went LN, Oosterhuis JA. Dominant cone dystrophy starting with blue cone involvement. *Br J Ophthalmol* 1991; 75: 332–6.
- 2 Pinckers A, Marré M. Basic phenomena in acquired colour vision deficiency. *Docum Ophthalmol* 1983; 55: 251–71.
- 3 Pinckers A, Deutman AF. Peripheral cone disease. *Ophthalmologica* 1977; 174: 145–50.
- 4 Noble K, Siegel IM, Carr RE. Progressive peripheral cone dysfunction. *Am J Ophthalmol* 1988; 106: 557–60.

Reply

SIR,—In an effort to be concise we have apparently not been sufficiently clear. The main conclusion of our article – concerning a progressive cone dystrophy and not a cone dysfunction – was that our family represents a distinct, as yet undescribed entity (last sentence of the abstract and last sentence of the last paragraph but one of the discussion).

It is now sufficiently well documented that genes coding for visual pigments may if subject to mutation, lead to ocular disease: X-linked cone dystrophy in the case of a deletion of the red cone pigment gene,¹ autosomal dominant retinitis pigmentosa in the case of a mutation of the rhodopsin gene.^{2,3} Sufficient reason for us to ask for an analysis of the tritan gene (thus not as Dr Pinckers states: 'The authors . . . looked in vain . . .').

L N WENT
M J VAN SCHOONEVELD
J A OOSTERHUIS

- 1 Reichel E, Bruce AM, Sandberg MA, Berson EL. An electroretinographic and molecular genetic study of X-linked cone degeneration. *Am J Ophthalmol* 1989; 108: 540–7.
- 2 Drya TP, McGee TL, Reichel E, et al. A point mutation of the rhodopsin gene in one form of retinitis pigmentosa. *Nature* 1990; 343: 364–6.
- 3 Drya TP, McGee TL, Hahn LB, et al. Mutations with the rhodopsin gene in patients with autosomal dominant retinitis pigmentosa. *N Engl J Med* 1990; 323: 1302–7.

Cause of blindness in the Central African Republic

SIR,—It was with much interest I read the recent paper by Dr Andrew Potter.¹

One of the important early steps in the development of a national prevention of blindness programme is the assessment of the dimensions of the problem of blindness and a characterisation of the leading causes of blindness in a particular country or region. The findings in the Central African Republic show that cataract and uncorrected aphakia

accounted for more than half the blindness, a finding in common with many countries.

Quite rightly Dr Potter suggests that one solution to the problem of cataract blindness would be to train eye workers to perform intracapsular cataract extractions (ICCE). One argument made in support of the more expensive operation of extracapsular cataract extraction (ECCE) with intraocular lens implantation has been the permanence of the correction of the surgically-induced aphakia. The aphakic glasses used after intracapsular extraction can become broken or lost and frequently are not replaced.

Few data are available on the proportion of people who have had intracapsular surgery and who continue to wear their corrective glasses. I was wondering whether Dr Potter had information on the proportion of people who were blind because of uncorrected surgical aphakia, and also the proportion of aphakics who in fact remain blind after surgery either because of surgical complications or loss of their aphakic glasses.

HUGH R TAYLOR
University of Melbourne,
Department of Ophthalmology,
Australia

1 Potter A. Causes of blindness and visual handicap in the Central African Republic. *Br J Ophthalmol* 1991; 75: 326–8.

Reply

SIR,—Those of us working in Africa are well aware of the problem of uncorrected aphakia. There are a number of reasons for this problem. Spectacles may not have been provided at the time of the original cataract extraction. Patients may not have found the money for their purchase. The glasses may well have been broken or lost at a later date. Other patients are unable to make the effort to adapt to the larger image and visual distortions. Still others were given a prescription for a pair of glasses which when they presented it to a private optician found the cost prohibitive. A depressing litany of excuses that I regularly encounter.

From my 4 years' work in the Central African Republic I found that uncorrected aphakia accounted for 8% of bilateral blindness. This included a majority who had one eye blind from unoperated cataract and the other aphakic eye without spectacle correction. Of 26 patients with bilateral aphakia without spectacle correction eight had other problems that could not be improved with glasses (optic atrophy, maculopathy, pupillary membrane, retinal detachment, and phthisis.)

Now the alluring prospect of extracapsular cataract extraction (ECCE) with intraocular lens implant is suggested for Africa. This could solve the problem of uncorrected surgical aphakia. But is it realistic for rural Africa at the present time?

ECCE is a more complex surgical procedure requiring sophisticated equipment (needing maintenance) and a longer training for the surgeon involved who would have to be a fully trained medical doctor. Intraocular lenses are more expensive than aphakic spectacles. In the Central African Republic there is only one permanent centre for cataract surgery. It is in the capital Bangui. The whole of the rest of the country and the majority of the population are very infrequently served by visiting surgeons who may spend up to a week in any one place. Such provincial hospitals and dispensaries may well not have mains electricity.

The patients who attend for surgery are elderly and poor. Some may have travelled for two or three days from their homes on foot.