

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.

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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 corneoscleral 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.

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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 synthetic 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.

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- 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,³ 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 . . .').

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- 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.

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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.