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Editorials

Who's afraid of prevention of blindness?

In this new millennium, the post-genomic era is likely to herald significant advances and changes in the field of ophthalmology research. Wave front deviation guided LASIK to enhance visual acuity, gene knockout therapy for AMD, and ex vivo clonal expansion of limbal stem cells for ocular surface reconstruction are just some of the exciting developments on our horizon. Where then lies any interest in prevention of blindness (POB) programmes? Can research in ocular epidemiology have an impact any longer with young ophthalmic residents, and who attends the POB session at ophthalmic meetings?

Think of cataract, and one usually thinks of the latest chop, or flip flop technique of nucleus removal within an elegant clear corneal, topical phacoemulsification procedure, not cataract camp surgery in India. And yet, in this new age of technological success and innovation, more people than ever before will continue to go blind from cataract by simply not having access to surgery. Hugh Taylor estimated in 1995 that five out of six people blind from cataract die before they receive cataract surgery.¹ Age related blindness from cataract now accounts for 50% of blindness in the world, and because only 20% of those blind from cataract currently have access to cataract surgery, mass blindness will double in number to 80 million people within 10 years.² The impact of world blindness unfortunately is usually not felt in developed countries. Of the 37.9 million blind in the world, reported by the World Health Organization in 1995, 75% live in the developing countries of Asia (21.4 million) and Africa (7.1 million), and at least 50-70% of adult blindness are either preventable or curable with currently available medical or surgical technology.³

As doctors trained to alleviate suffering and disease within an increasingly global society, we need to step back from our busy clinical workloads and clinical trials, and focus on these startling statistics. After all, Lim stated that "the movement to alleviate cataract related blindness should excite everyone, for it is about humanity: the willingness to help the less fortunate. It is about human organization. It is about international cooperation."² Faal and coworkers in this issue of the *BjO* (p 948) describe the profound impact of a national eyecare programme (NECP) on the prevalence of blindness and low vision in the Gambia. In a return survey 10 years after the initial survey, they report a 40% reduction in the crude prevalence of blindness from 0.70% to 0.42%, and show that this reduction is mainly due to a significant drop in the prevalence of cataract blindness in the western health

region, where the NECP was started. This POB programme clearly illustrates how a carefully planned intervention in preventive healthcare policy specifically targeted at reducing the burden of blinding cataract can result in significant reduction of blindness on a national scale.

The success of the NECP hinges on two important factors, the training of village health workers in primary eye care, and the training of a paramedic cadre of ophthalmic medical assistants to perform cataract surgery. Establishment of an effective training programme for local ophthalmic personnel to perform cataract surgery is of paramount importance for long term success, and this was also clearly illustrated by the formation of training centres for cataract surgery in the People's Republic of China. Ten years ago, the concept of a training centre in China to teach extracapsular cataract extraction and posterior chamber implant was developed, and in 1989, the International Intraocular Implant Training Centre was established in Tianjin. With the cooperation of regional hospitals, it has been the training centre for 1500 ophthalmologists, and its programmes have restored normal vision to 20 000 cataract patients in Tianjin and in the affiliated regional hospitals whose ophthalmologists were trained at this centre.²

In India, too, significant changes are afoot. In 1996, the World Bank announced a historic programme to solve the problem of cataract blindness in India. The goal, to restore vision to 11 million cataract victims in 5 years, involved a loan exceeding \$100 million to the Indian government, to be spent on training seven medical colleges and 1500 government surgeons to perform extracapsular cataract extraction.⁴

Wormald, in a recent *BjO* editorial, commented on quantity and quality of cataract surgery, and stated that while output may be increased and costs are reduced, quality must be maintained or improved.⁵ Within this epoch of phacoemulsification, the change in less developed countries from intracapsular cataract extraction (ICCE) to extracapsular cataract extraction (ECCE) with intraocular lens implantation in Africa, China, and India finally signifies the close of an era in which uncorrected aphakia was responsible for a significant cause of visual disability even after surgery. Alfred Sommer reported a decade ago that "at least half of the people who have undergone allegedly successful surgery are blind, because they do not have aphakic glasses",⁴ and Faal's study also suggests that the highest proportion of uncorrected aphakia occurring in the Western region (19% of blindness) is directly related to the

fact that ICCE was the dominant procedure during the first 5 years of the NECP.

Let us then remind ourselves of prevention of blindness programmes which have the potential to do the greatest good for the most people. Sir John Wilson, recipient of the 1997 Jose Rizal Gold Medal, the highest award of the Asia-Pacific Academy of Ophthalmology said:

"Increasingly, it is likely that the prevention of blindness will be seen, not as an exclusive initiative, but as the model for more comprehensive action against all causes of avoidable disability which affect massive populations."⁶

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Amblyopia

Amblyopia is a fairly common disease affecting between 1% and 2% of the population of most developed countries.¹ Amblyopia is the unilateral or, less commonly, bilateral loss of vision caused by abnormal visual inputs during a critical period of visual development. The critical period is seen as the period of time during which abnormal visual inputs can result in amblyopia, but it is also the time during which amblyopia can be reversed by eliminating the abnormal visual inputs and, usually, occluding the normal eye for some periods of time.

However, there are now reasons to believe that these critical periods for development and treatment of amblyopia cannot be rigidly defined. Firstly, it is clear that visual acuity of the amblyopic eye is not always firmly established even after amblyopia therapy has been terminated, because the age of the patient is beyond what is generally considered to be the critical period.^{2–4} Scott and Dickey reported a short term follow up study of amblyopic patients after patching therapy was stopped. Seventeen per cent of patients lost a line of visual acuity and 8% lost two lines or more.³ In a study of patients 10 years after amblyopia therapy was stopped, Gregersen and Rindziunski reported that 14% of patients lost all of the previously improved visual acuity in the amblyopic eye⁵; 67% of patients lost at least one line of visual acuity in the amblyopic eye.

Secondly, improvement in visual acuity and changes in the neurophysiological substrates of amblyopia can be demonstrated in some adult experimental animal models.^{6–8} This is most dramatic when the non-amblyopic eye is enucleated in the adult animal. In the case of amblyopic cats, a sixfold increase in cells in the visual cortex responding to the amblyopic eye can be demonstrated after enucleation of the non-amblyopic eye.^{6–7} Similar but less dramatic effects have been reported in adult primate amblyopes.⁸

Finally, several clinical reports have demonstrated improved visual acuity in the amblyopic eye of human adults following loss of central vision in the non-amblyopic eye.^{9–14} In a retrospective multicentre report, Vereecken and Brabant reported at least three lines of visual acuity improvement in 28.5% of patients with visual loss in the good eye.⁹ Typically, improvement occurred in the first few weeks after visual loss in the non-amblyopic eye. Api and coworkers reported eight adult strabismic amblyopes with visual loss in the non-amblyopic eye.¹¹ There were more than two lines of visual acuity improvement in the amblyopic eye in all patients. It is noteworthy that in three cases enucleation of the non-amblyopic eye was performed. Klaeger-

Manzanell and coworkers documented a case of a man with strabismic amblyopia who experienced a two step recovery of vision in the amblyopic eye after loss of vision and then subsequent enucleation of the non-amblyopic eye.¹²

In this issue of the *BJO* (p 952) El Mallah and coworkers detail amblyopic patients who exhibited increased visual acuity following loss of vision in the non-amblyopic eye due to macular degeneration. It is not clear if, in this 465 patient study group with age related macular degeneration, there were any amblyopic patients who lost vision in the non-amblyopic eye but did not experience visual acuity improvement in the amblyopic eye. This information would be of interest. By 12 months after the vision loss in the non-amblyopic eye, visual acuity had improved by an average of three lines in the non-amblyopic patients. Improvement of visual acuity seems to have occurred primarily in the first 6 months following visual loss in the non-amblyopic eye. So, if visual acuity can improve in the adult amblyope following visual loss in the non-amblyopic eye, why should we spend so much time trying to treat amblyopia in children with the purpose of creating a "spare tyre" in case the non-amblyopic eye is lost in adulthood?

In the case of strabismic amblyopia, improving visual acuity in the amblyopic eye is critical since the long term stability of the ocular motor alignment is directly affected by the degree to which the amblyopic eye is effectively treated in childhood.

Not all adult amblyopes experience visual acuity improvement in the amblyopic eye after visual loss in the non-amblyopic eye. We do not yet know what percentage of amblyopes can anticipate improvement if they are unfortunate enough to lose visual function in the non-amblyopic eye as an adult. Certainly, there is no evidence that form deprivation will improve in an adult who loses vision in a non-amblyopic eye. Moreover, there is good reason to believe from primate studies that this would not be expected.¹⁵

In most cases, the improvement in the adult amblyopic eye following loss of vision in the non-amblyopic eye is reasonably modest. In addition, studies to date have limited their assessment of these patients to measuring resolution visual acuity. No reports have studied the other many abnormalities that occur in amblyopia, in particular, motion processing, ocular motor stability and efficiency, or orientation discrimination. In my experience, these adult amblyopic patients with improved visual acuity are still

much more visually impaired than the resolution acuity measurement would suggest. In particular, their motion processing seems to be still severely affected even when the recognition acuity is by all measures quite good.

Finally, it is not yet clear how much of the improved visual acuity reported in adult amblyopes with visual loss in the non-amblyopic eye is nothing more than recapturing the "slipped" visual acuity that occurs in so many "successfully" treated amblyopes.^{3,4}

Nevertheless, this group of patients is of obvious interest to ophthalmologists. What are the biochemical and cellular correlates for changes in visual acuity that occur in adult amblyopes? If these were better understood, would they lead us to pharmacological forms of amblyopia therapy? We look forward to more detailed studies of adult amblyopes with improvement of visual acuity following loss of vision in the non-amblyopic eye.

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Age related macular degeneration: could we improve the services we offer?

The letter written by Dr Margaret Ewart (p 1083), a retired general practitioner who has age related macular degeneration (AMD), coincides with the publication of the document "Age Related Macular Degeneration, Best Clinical Practice Guidelines" by the Royal College of Ophthalmologists.¹ Both the letter and the guidelines highlight a number of common issues.

A recognised scenario in the United Kingdom is the elderly patient with loss of vision in one eye due to AMD who presents to her general practitioner with symptoms of distortion of vision in her other eye. The referral letter from the general practitioner seeks a routine appointment and by the time the patient is seen she has a subfoveal neovascular membrane with a visual acuity of 6/24. A verbal description of the disorder is given and she is told that "there is nothing we can do". The vision is not deemed poor enough to recommend registration and the busy ophthalmologist who sees her omits to arrange for her to be assessed for a low vision aid. She goes home where she lives alone. Six months later she returns with a visual acuity of 3/60. In the interim she has fallen and broken her hip and is now receiving long term care. Could she have been seen more promptly? Could she have received effective laser treatment? Could she have received low vision aids? Could she have received training in how to use them? Could she have been given practical advice concerning day to day living which would have diminished her risk of injury? Could the cost to society have been less? The answers to all these questions are yes, but why is this a situation which we all recognise?

In the first place, general practitioners and high street optometrists need to be well informed which patients with macular degeneration warrant referral and with what urgency. Whether or not the eye pathology can be treated, each patient requires a positive, informative, and struc-

tured approach. It can be very helpful to write to our patients (in an appropriate print size)² enclosing information about such organisations as the Macular Disease Society, and about CCTV equipment and other resources. The provision of low vision aids throughout the United Kingdom is inconsistent. There is evidence that training in eccentric viewing improves rehabilitation,³ but education in the use of low vision aids is commonly limited to brief instructions. However, the guidelines stress that "it is not adequate to issue a patient with a magnifying glass and not provide sufficient aftercare".¹ Eccentric fixation entails shifting the focus of one's attention to a perifoveal site. Those trained in this technique of reading can greatly improve their reading ability particularly in conditions of optimal lighting.³ In addition, an information and advisory service can be provided by trained staff who can spend more time identifying specific problems and addressing them.

A significant problem is that of registration as being blind or partially sighted. Such registration is often carried out at a late stage in the evolution of the disease and the promptness and quality of service delivery which results can be variable. The visual acuity is a measure of visual function at maximum contrast. It does not necessarily provide an index of disability. Metamorphopsia, impaired colour vision, and impaired contrast sensitivity combined with paracentral scotoma formation can conspire to render the patient "substantially and permanently handicapped by defective vision . . ." (the criterion for partial sighted registration in the United Kingdom) despite a visual acuity of 6/18 or better. When it is clear that the retinal pathology is progressive, partial sighted registration can lead to the provision of statutory social services at an appropriate time.

It is now recognised that “the early provision of advice and support will encourage independence and minimise the socioeconomic isolation that AMD causes”.¹ Dr Ewart sets a challenge to our profession. A challenge of leadership and a challenge of improving the service we offer. The best clinical practice guidelines for age related macular degeneration address the issues which she raises. The question is whether we can bring about their implementation for this vulnerable group of patients and extend their application to other patients with progressive loss of vision.

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Cover illustration: Your eyes are bigger than your stomach

The new world screech owl, as typified by this eastern screech owl (*Otus asio*) seen on the cover, is an endearing species widely distributed throughout North America and with close relatives throughout the world (approximately 40 related species). This is the most abundant and perhaps most familiar owl in North America. About the size of a soda can and weighing much less, this attractive little bird (20–23 cm) is a fierce defender of its nest and has the nickname “feathered wild cat”. Other species will be vigorously attacked near the nest if the bird fears intrusion. It will often kill animals larger than itself in the name of nest defence, and yet it can be gentle and playful with humans in other situations. The bird is also a fearsome nocturnal hunter. Although the principal foodstuff is insects, this magnificent bird can and will take rodents and even small birds, especially in winter when insects can be scarce.

These birds have tubular eyes as can be seen on the cover. Evolution has eliminated almost all extraocular musculature, to maximising the size of the globes. In essence these birds have sacrificed extraocular motility for improved visual acuity, especially at night. To compensate, most owls can turn their heads with great speed, nearly 270° atop a thin, lightweight spinal column; so quickly that myths of complete 360° cranial revolution have sprung up adding to the mythology of owls. As can be seen in the cranial slice taken at the midline of the cornea above the upper mandible, the eyes are large, both on an absolute and relative basis. They are frontally placed, providing as much binocular stereopsis as possible, as is typical for avian predators. The stereoscopic binocular visual field is approximately 60–70°, although the visual field is larger. The eyes together outweigh the brain as often occurs in raptorial species, especially those that are nocturnal wing feeders. This species is an “eye minded” one with an evolutionary concentration on visual abilities.

The tubular eyes (longer anterior-posterior than equatorial diameter) are asymmetric and structurally not as stable as our own. Consequently, they contain bony plates at the mid-portion of the globes much like girdles to help stabilise the ocular contents. These scleral ossicles are found in the region occupied by the ciliary body and pars plana, and are not continuous, since a solid ring of bone could not grow with the eye. The retinal area is surprisingly small with the anterior edge of the retina corresponding to the widest equatorial diameter. The entire eye is outsized with a large lens, a deep anterior chamber, and a long anterior-posterior diameter. This longer “throw” of the image allows for sharper acuity with a larger image spread across the retina. The single fovea in each eye is deep walled, and the retina is duplex, but rod-rich. The pecten is small with fewer pleats when compared with the pecten of other birds and, similarly, accommodation is limited when compared with other avian species.

These owls are usually non-migratory, but may change feeding habits as prey species change with the seasons, as mentioned above. Their prey species have been documented to include flying squirrels, bats, birds, frogs, reptiles, and various invertebrates. Even ducks and poultry have been found in their nests. In essence, this bird is an indiscriminate carnivore. In more northern climates, they tend to remain in their nest cavities relying on cached food in the worst of the weather. Imagine how such a bird with a small crop and stomach must struggle to kill and eat prey species such as a duck (!), which may be nearly its own size and with a bill no less.

Screech owls are heard far more frequently than they are seen. Their melodic whistle beginning in early February in North America, is a sure sign that, despite cold and wintry weather, spring cannot be far behind.—IVAN R SCHWAB, *University of California, Davis, Medical School Department of Ophthalmology*