What’s new at the back of the eye?

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The changing demographic pattern of visual impairment
VISION 2020 and the prevention of visual impairment are moving into the political and public spotlight. Recent WHO discussions on the growing incidence of chronic, age-related, non-communicable diseases indicate that visual health and its preservation are now receiving attention. Recent publications illustrate the changing demographic pattern of visual impairment, implications for public health and possible interventions to control sight-threatening conditions. Both The State of the World’s Sight, VISION 2020: the Right to Sight, 1999-2005,1 published jointly by WHO and the International Agency for the Prevention of Blindness (IAPB), and an article in the World Health Bulletin, November 2004,2 illustrate these trends.

The recent WHO data on blindness give clear evidence: among the causes of blindness, the share of chronic, age-related, non-communicable potentially blinding eye conditions is dramatically increasing (Figure 1, over page). The State of the World’s Sight provides an insight into what has thus far been achieved in the prevention of visual impairment through international alliances and collaboration since the launch of VISION 2020 in 1999. Attempts to modernise the attitude of health care providers towards the preservation of visual health have been further cultivated by the representatives of many WHO member states when adopting, in 2003, the Resolution on the Elimination of Avoidable Blindness, and when preparing a new resolution for the World Health Assembly in 2006.

What does all this mean for eye health care professionals – new challenges, new opportunities?

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Meeting the challenge through specialised eye care services

One challenging aspect of the latest WHO data on the changing pattern of blindness prevalence in the world is that the demand for high-quality eye care services is expected to increase. Predicted population growth, along with the increase in life expectancy, will substantially alter the picture of future recipients of eye care. The numbers of people affected by sight-threatening eye conditions will likely increase.

One such condition is diabetes. Diabetic retinopathy accounts for about 5 per cent of the global pattern of blindness. WHO is expecting a significant increase in the incidence of diabetes in all geographic regions. For instance, in many highly populated countries in Asia, the number of individuals suffering from diabetes will double by 2030.

Glaucoma is another example which, from the public health perspective, may be even more challenging. Controlling blindness from glaucoma requires early detection, life-long treatment and the compliance of patients. It is estimated that glaucoma is responsible for 12 per cent of global blindness. However, numerous unresolved issues in glaucoma control deprive many populations from effectively planned and delivered specialised eye care services.

The WHO data also indicate the growing threat of age-related macular degeneration. The increasing share of this eye condition is progressively reshaping the ratio of avoidable sight-threatening eye conditions to those in which conventional eye care is thus far failing. Cataract is another age-related eye condition causing avoidable blindness. Given that it has been one of the major stimuli for technological research and development in eye care, the failure of modern society to adequately control
The recent WHO data on blindness give clear evidence: among the causes of blindness, the share of chronic, age-related, non-communicable potentially blinding eye conditions is dramatically increasing.

cataract blindness is particularly shameful. The last WHO data indicate that cataract is still responsible for almost half of global blindness. Its cure is known, and several options for its surgical treatment have been tested and broadly implemented to reasonably fit healthcare budgets, even in the poorest societies. Demographic changes and longevity, along with the lack, for the foreseeable future, of knowledge about cataract prevention, will further increase the demand for cataract surgical services. Age-related macular degeneration poses a different set of challenges, as the condition cannot be prevented, and only some forms are amenable to very expensive, time-consuming interventions which are of limited benefit. The mainstay will need to be rapid expansion of low vision services, along with raising awareness among the general public and eye care professionals.

The implications of demographic changes and the ageing of populations are a growing concern. However, preventable and treatable sight-threatening eye conditions occurring in children must also remain among the top priorities. The World Health Bulletin of November 2004 also sent a strong warning signal emphasising the need to address uncorrected refractive errors, an often overlooked public health issue relevant to all age groups. The importance of refractive errors in the pattern of visual impairment is likely to be substantially greater than generally assumed.

The need for targeted action and an interdisciplinary approach

The current situation presents us with a new opportunity for targeted action. The preservation of visual health requires, more than ever, a coordinated approach by health care providers, who will develop comprehensive, integrated eye care services. An effectively designed eye care component in primary, secondary and tertiary health care is an essential prerequisite to respond adequately to the changing pattern of avoidable visual impairment. Over the long term, vertical interventions and projects oriented to a single eye disease may no longer bring the desired achievements.

The nature of the causes of blindness which WHO considers to be preventable and/or treatable, and therefore avoidable, requires an interdisciplinary approach for long-term control. For example, controlling diabetic retinopathy is complex, requiring: health education to prevent diabetes; early diagnosis of diabetes; comprehensive, cost-effective screening programmes to detect diabetics with treatable retinopathy; laser treatment and follow-up; and patient education and compliance. In many countries, attitudes about diabetes control are being reshaped by constituting national diabetes control programmes, rather than by pursuing the control of this disease through uncoordinated efforts within individual medical sub-specialties. In those countries where the number of diabetic patients is rapidly increasing, introducing control programmes on the scale needed will be extremely challenging. We need to educate more ophthalmologists to manage diabetic retinopathy, make appropriate technology available, develop new programmes, establish new partnerships, and develop a major public health education strategy.

Integrating eye care into national health plans

Health care providers in the most affluent societies and in many other countries with modern health care systems are becoming increasingly interested in comprehensive, integrated care. In this context, eye care providers have three responsibilities: to be ready to explain the known and tested options in blindness control to national health care planners and providers; to be proactive in health care policy development; and to take part in updating national health care strategies where eye care should be addressed. It would be disappointing to lose the opportunity for integrating eye care into national health care plans because of a failure to provide health care planners with an adequate briefing on the management of sight-threatening conditions. Several national health care areas should be explored, especially neonatal care, health care programmes for mothers and children, preventive health care programmes for working-age populations as well as for the elderly. As the world copes more adequately with the challenges of blindness control, eye care professionals will play a vital role in advocating for comprehensive health care which integrates and adequately addresses visual health.

References


Introduction
Age-related macular degeneration (AMD) was regarded as unimportant in global blindness, of relevance only to the minority of the world’s population that live in wealthy countries. However, increasing life expectancy, particularly in Asia, has challenged this view. The latest WHO estimates of global blindness suggest that there are over 3 million people blinded by AMD, representing 9 per cent of global blindness. Only cataract and glaucoma cause more blindness.

What is AMD?
AMD is a disorder affecting people over the age of 55. It affects the macula in the centre of the retina. The macula is essential for detailed fine vision tasks, such as reading and recognising faces. There are two types of AMD, dry and wet. Dry macular degeneration is often asymptomatic. However, patients may progress to vision loss through progressive atrophy of the macular tissue (an advanced dry form termed geographic atrophy). Wet, or exudative, AMD is characterised by a more rapid change in vision due to haemorrhage and fluid leakage (see Figures 1a & 1b).

Clinical features
Wet AMD is due to a fibrovascular membrane growing under the retina. These abnormal choroidal new vessels (CNV) arise in the choriocapillaris, therefore, unlike retinal vessels, they leak fluid. This causes a localised retinal detachment at the fovea (central macula), which may be surrounded by oedema and exudates. In addition, the blood vessels may rupture causing sub-retinal haemorrhage and hard exudates as well. These are much easier to detect using a biomicroscopic lens, such as a 78 or 66 dioptre lens. This gives a stereoscopic view of the macula, which is not possible with a direct ophthalmoscope.

The definitive investigation is fluorescein angiography. Fluorescein dye is injected intravenously, and as it circulates around the eye, a series of photographs are taken of the retina using a blue light to illuminate the retina. The fluorescein then emits green light, which is detected by the camera. Unlike normal retinal blood vessels, the CNV leak fluorescein, and this can be seen as a bright area in the angiogram photos (see Figure 3). The fibrovascular membranes are classified as either ‘classic’ (well-defined on fluorescein angiography), or ‘occult’, if leakage is seen but the membrane is not well-defined. New digital fundus cameras, which are expensive to buy but have very low running costs, are making fluorescein angiography more accessible.

Dry AMD causes a very gradual loss of vision, and is visible as an area of atrophy of the retinal pigment epithelium under the fovea.

Risk factors
No one knows exactly what causes AMD, but there have been some important developments recently. Firstly, we know that age is the most important risk factor. The condition is much more common in people aged 80 or over. Unfortunately, we cannot modify this risk factor as we are all ageing. Several epidemiological surveys have demonstrated that smoking is a risk factor for AMD. The link between smoking and blindness is now well known, and this is an important public health message. Smoking one pack of cigarettes per day for 40 years is associated with a threefold increase in the risk of AMD. Even passive smoking is associated with an increased risk, but stopping smoking appears to reduce the danger. Possibly 15 per cent of AMD cases are caused by smoking. Genetic influences are also important. Several recent studies have shown that a variant of the gene that encodes complement factor H (a protein that helps to regulate the body’s response to inflammation) and a second gene, less characterised and located on chromosome 10, are independently associated with AMD. As many as 40-50 per cent of all cases of AMD may be caused by these gene variations.

Prevention
There is no perfect method of preventing AMD. Stopping smoking reduces the risk, but does not abolish it entirely. A large study in the USA showed that taking high doses of vitamins A, C, and E, combined with zinc supplements, significantly reduced the risk of progression in AMD. The treatment was protective against CNV, but had no clear effect on geographic atrophy. The supplements are most beneficial when used in patients at greater risk of developing wet
AMD, particularly patients who already have one eye affected by wet AMD. In these patients, the supplements reduce the risk of developing wet AMD in the second eye by about 25 per cent. Smokers should not use this treatment, as it may increase their risk of lung cancer. It is estimated that in the USA there are 8 million people over 55 who are at risk and that 1.3 million of them are likely to lose vision as a result of AMD over the next five years. Use of vitamin and zinc supplements would reduce this by 300,000, and stopping smoking might reduce it even further. We should ensure that patients at risk have access to vitamin supplements, and should take every opportunity to stress the strong link between smoking and blindness.

**Treatment**

No treatment has been shown to be effective against geographic atrophy. For many years the only treatment for CNV was laser photocoagulation. This is effective, but, as the treatment destroys the overlying retina, it also results in irreversible loss of central vision; therefore laser photocoagulation is only useful in the 10 per cent of patients with wet AMD in whom the CNV are located away from the fovea.

Recently some vision-sparing treatments have become available. The first of these is photodynamic therapy (PDT) with verteporfin. Verteporfin is a drug that only becomes active when exposed to light of a particular wavelength. It is injected intravenously, and the light is delivered to the retina by a laser. Verteporfin absorbs the light, causing it to be converted into a toxic form that kills the abnormal blood vessels. Although PDT may be effective for six months. A recent clinical trial compared sub-Tenon’s injection of anecortave to PDT with verteporfin. Unfortunately, 1.3 per cent of patients given PDT developed endophthalmitis, with 0.6 per cent had a retinal detachment. It is not known for how long the treatment must be continued, but it seems probable that stopping the injections would lead to reactivation of the new vessels. A second drug that is not yet available, called ranibizumab, also works by blocking the action of VEGF. It has shown even more promising results, with less than 10 per cent of patients losing three lines of vision and over 30 per cent gaining at least two lines.

Anecortave is an angiostatic steroid which means it does not possess any glucocorticoid action and has none of the usual ocular side-effects of steroids, such as elevated IOP or reduced immunity. It is being evaluated both for the treatment and prevention of wet AMD. Anecortave can be given by sub-Tenon’s injection and a treatment may be effective for six months. A recent clinical trial compared sub-Tenon’s injection of anecortave to PDT with verteporfin. In both treatment groups, about 45 per cent of patients lost less than three lines of vision. The injections must be repeated every six months, but appear to be very safe. Because PDT was used in the control group, all patients had classic membranes, so there is no information on the effectiveness of anecortave in occult membranes. We are awaiting results from more clinical trials.

Although PDT, pegaptanib and anecortave are all effective at reducing the rate of visual loss, they are not a complete cure. They significantly reduce the risk of blindness, but do not restore normal vision. In most trials, success was defined as loss of less than three lines of vision, so an eye that dropped from 6/36 to 6/60 after treatment would be regarded as a success, although the patient would still have poor vision.

The alternative to medical treatment is surgery. Although no large trials have been carried out, retinal translocation appears to offer some hope to patients who develop CNV in their second eye. The principle of this operation is that the CNV are a response to abnormalities in the retinal pigment epithelium and choroid, and the overlying macula is initially healthy. If the macula could be moved to another part of the retina, with healthy pigment epithelium, it would regain its function. This is achieved by pars plana vitrectomy, after which the retina is then completely detached by injecting saline under the retina through a 40 guage needle. Once the retina is detached, a 360 degree retinotomy is carried out just behind the ora serrata. The CNV membrane can then be picked up and removed with fine intraocular forceps. The retina is then re-attached at the optic disc, and it can be rotated by about 45 degrees, so that the macula now lies over healthy choroid. The retina is re-attached by filling the vitreous cavity with silicone oil. After three months, the silicone oil is removed and the superior and inferior oblique muscles are adjusted so that the eyeball is rotated in the opposite direction and the macula is once again at the centre of the visual axis. This is very complex and costly surgery. However, in the largest series to date, the median distance visual acuity improved from 6/36 to 6/24, and reading vision improved even more, with the median reading speed increasing from 71 to 105 words per minute.

Despite our best efforts, many patients will be left with profound visual loss. Although this may not be curable, we can still help by providing low vision aids, and social support. Peripheral navigational vision is usually preserved, so mobility training is not usually required. However, poor reading vision may be very disabling, and although magnifying low vision aids are unlikely to improve vision enough to read a book, they allow the patient to read prices in the market or headlines in a newspaper.

**Conclusion**

As the world’s population ages, we will face an increasing challenge from AMD. Although there have been some very significant advances in our understanding of the causes and treatment of AMD, we are still a very long way short of a cure or an effective prevention.
Can we grow new retina?

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Introduction
Retinal degenerations and dystrophies, the major causes of genetically inherited blindness, are characterised by the death or degeneration of photoreceptors (rods and/or cones).1 Approaches to treating this disease include: a) replacing the defective gene; b) introducing a drug or agent that either slows down or stops the premature death of photoreceptors; c) introducing electronic chips; or d) replacing the damaged cells by cellular therapy. Gene therapy is aimed at counteracting the defective gene by substituting it with the normal gene in the target tissues. Though successful visual recovery has been reported with gene therapy in dog models, 2,3 it remains a challenge to identify a safe and reliable way of introducing the corrective gene in humans, given that the genes need to act for the lifetime of the patient. Introduction of ‘a’ factors (such as growth factors) into the eye, directly or through implants, is another novel approach to preventing or slowing premature cell death.2,4 The challenge lies in delivering the drug to the appropriate site in a safe and sustained manner. Electronic chips, similar to the ones used for audio aids, have shown exciting results in some studies, but the technology is still in its infancy.5-7

As knowledge relating to stem cells has increased over the last two decades, attempts have been made to translate this research into clinical practice, particularly for ocular surface reconstruction. Certain ocular surface disorders, like chemical burns, cause damage to the corneal epithelial stem cells. The consequence of this is that the normal corneal epithelium is replaced by conjunctival epithelium, which leads to corneal opacity and vascularisation, with loss of vision. Many centres across the world,8-10 including our centre,11-13 have grown sheets of epithelial cells from stem cells, supported on amniotic membrane. These sheets of cells have then been successfully transplanted to cover the entire corneal surface in individuals with ocular surface disorders, leading to less inflammation and scarring. Though the clinical outcome of this new technique is well established, there are still many unanswered questions, particularly in relation to the long-term survival of the transplanted cells.

What are stem cells?
Stem cells are defined as undifferentiated (‘primitive’) cells that are capable of self-renewal (dividing) and differentiation (changing into cells which have different structural characteristics and function). There are basically two types of stem cells, embryonic and adult stem cells. The embryonic cells are totipotent and pluripotent, with a potential to generate all the types of cells. The adult stem cells are few in number and are located in different parts of the body, like bone marrow, skin, and intestinal mucosa, and serve the purpose of regenerating that particular tissue/cells of the body throughout the life span. It is now known that some of these adult stem cells, in addition to generating cells of their own lineage, can also generate cells of other lineages by a principle called ‘transdifferentiation’ or ‘plasticity’.14 Bone marrow stromal cells, also called mesenchymal stem cells, are the best example of such cells, as they have the potential to form bone, cartilage, neurons and muscle cells.

What is cell therapy?
Conceptually, cell therapy can be broadly classified into four types:
(a) autologous and homogeneous – i.e. the use of the patient’s own adult stem cells to regenerate cells of the same kind, e.g. skin cells, limbal stem cells;
(b) autologous and non homogeneous – i.e. the use of the patient’s own cells, but to make cells of a different kind, for example regeneration and remodelling of myocardium after injecting autologous bone marrow-derived stem cells;
(c) allogenic cell therapy – i.e. the use of cells of the same kind, but from a different donor. This requires the use of immunosuppressive drugs to prevent the rejection of cells, e.g. bone marrow transplantation;
(d) embryonic cell therapy – i.e. the use of embryonic cells that have been characterised, isolated and shown to differentiate along the desired cell type only.

This article presents a conceptual approach to cell therapy and a brief review of progress in this field.

Which cells to choose?
Referring to the above classification of cell therapy, in principle, retina could be grown from: a) stem cells within the retina, retinal stem cells (Figure 1); b) stem cells within the eye, but outside the retina, e.g. ciliary body stem cells or retinal pigment epithelial cells; c) from the patient’s own tissues, but using non-ocular sources, e.g. bone marrow stromal cells or neural stem cells; d) non-self stem cells, e.g. embryonic stem cells. The use of these cells would depend on their availability/accessibility, the techniques available to grow them, the risks involved in harvesting them without the depletion of the donor cells, success in growing them without altering their nature and transplanting them back into the retina.

The progenitor cells of the retina (neural precursor cells15-16) do have the potential to constitutively replace the different cells of retinal-like neurons, photoreceptors, and glial cells. This approach could be useful in developing and studying the pathobiology of the stem cells in health and disease, but the technical difficulty in obtaining these cells and their limited availability could be an issue. The ciliary and iris pigment progenitor cells contain a mitotically quiescent population of neural progenitors that proliferate to make neural stem cells, with a potential for self-renewal.17-18 Experiments have documented the incorporation of these cells into injured retina, but not normal retina, suggesting that functional integration is possible in damaged tissues. Neural progenitor cells from the brain can restore and survive in the damaged retina,19 but the major limitation is the source of these cells, which is as rare as, or even rarer than, the retinal cells themselves. It has been shown that bone marrow stromal cells can differentiate into retinal cells in injured rat retina, show functional recovery, and also promote or inhibit retinal angiogenesis.20-22 Similarly, embryonic stem cells (ESCs) were shown to survive, migrate and integrate into the host retina,24-25 but they do pose a potential risk of tumour-induction after engraftment.

Do we need cells or three-dimensional structures?
The question that now arises is: should we deliver the cells into the target site and hope for the damaged tissue to help in the final integration of the transplanted cells, or should we attempt to organise the cells into tissues before transplantation? A 3D tissue architecture not only provides anatomic integration of cells but also improves the functional outcome.26 The cells could also be delivered on polymer substrates to the subretinal space, so as to improve their survival, migration and functional restoration.27

Will the cells integrate?
Now, assuming the cells and the tissues are ready to go into the retina, will they integrate into the adult retina? Unlike the clinical application of cultivated epithelial cells for ocular surface reconstruction, the use of neural cells poses tough challenges. More rigid proof of integration of these cells is warranted. Successful cell therapy should fulfill the following criteria: the desired cells
should multiply/grow in sufficient quantities, organise into functional units, survive and integrate into the host, and ultimately, function appropriately in a safe manner.

Ethical issues
Clinicians and researchers must constantly deal with the question of adequacy of proof in animal experiments, before moving to human clinical trials. Though a few clinical trials were conducted using foetal cells, there are no published trials using any other cells for clinical transplantation. Reviewing the progress in all fields, it appears that the bone marrow-derived cells have the advantage of being autologous, with proven clinical safety. They could therefore be considered for a pilot study after seeking the approval of regulatory bodies, the Institutional Review Board clearance and patients’ informed consent.

Conclusion
In summary, when permanently damaged, the retinal cells which are specialised neurons, cannot be rescued or repaired in the natural process and therefore warrant cell therapy in future.

References
**Cell therapy glossary**

**Adult stem cells**
Undifferentiated cells found in most adult tissues. Adult stem cells can renew themselves and differentiate to yield all the specialised cell types of the tissue from which they originated. Also referred to as 'somatic stem cells'.

**Cell-based therapies**
Treatment in which stem cells are induced to differentiate into the specific cell type required to repair damaged or depleted adult cell populations or tissues.

**Cellular therapy**
A new way to treat disease and injury. It aims to repair damaged and diseased body-parts with healthy new cells provided by stem cell transplants.

**Cones**
A type of specialised light-sensitive cells (photoreceptors) in the retina that provide sharp central vision and colour vision. See also Rods.

**Differentiation**
The process whereby an unspecialised early embryonic cell acquires the features of a specialised cell, such as a heart, liver, or muscle cell.

**Embryonic stem cells**
Primitive (undifferentiated) cells from the embryo that have the potential to become all cell types found in the body (totipotent). Embryonic stem cells (ESCs) are derived from four to five day-old embryos.

**Gene therapy**
Therapy aimed at counteracting the gene defect by substituting normal gene material at the site of the problem.

**Mesenchymal stem cells**
Stem cells found primarily in the bone marrow that can transform into bone, cartilage, fat, and connective tissue. These cells are also referred to as bone marrow stromal cells.

**Multipotent stem cells**
Stem cells that can give rise to several other cell types, but those types are limited in number. An example of multipotent cells is haematopoietic cells – blood stem cells that can develop into several types of blood cells.

**Photoreceptors**
Cells that are sensitive to light.

**Plasticity**
The ability of stem cells from one adult tissue to generate the differentiated cell type of another.

**Progenitor cells**
Cells that can produce only one cell. They can differentiate into a limited number of cell types, but cannot make more stem cells (or renew themselves).

**Proliferation**
Expansion of a population of cells by the continuous division of single cells.

**Regenerative medicine**
A treatment in which stem cells are induced to differentiate into the specific cell type required to repair damaged or depleted adult cell populations or tissues.

**Retina**
The light-sensitive layer of tissue that lines the back of the eyeball; sends visual messages through the optic nerve to the brain.

**Retinal pigment epithelium**
The pigment cell layer that nourishes the retinal cells; located just outside the retina and attached to the choroid.

**Rods**
A type of specialised light-sensitive cells (photoreceptors) in the retina that provide side vision and the ability to see objects in dim light (night vision). Also see Cones.

**Stem cells**
Unspecialised cells that serve as the source, or ‘stem’, for specialised cells like heart, brain, or blood cells. They have two important characteristics that distinguish them from other cells in the body. Firstly, they can replenish their numbers for long periods through cell division. Secondly, after receiving certain chemical signals, they can differentiate, or transform into specialised cells with specific functions, such as a heart cell or nerve cell. Found in days-old embryos and a few adult organs.

**Subfoveal**
Beneath the fovea, the central pit in the macula that produces the sharpest vision.

**Undifferentiated cells**
Cells that have not changed to become a specialised type of cell.

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**What will be new and different in the future, specifically in the year 2020?**

Shaheen Shah reports from the World Ophthalmology Congress

**Diabetic retinopathy**

Dr Alexander Brucker, Professor of Ophthalmology at the University of Pennsylvania and Editor of the journal Retina, suggests that by the year 2020, decisions about treatment will be based on diagnosis using high definition optical coherence tomography (OCT) visualisation of the retina, in conjunction with fluorescein angiography (FA). Although the interpretation of the clinical findings may be similar, management will be more pharmacologically directed. He anticipates change will also be effected through alteration of the patient’s individual risk factor profile. For proliferative disease, the treatment will probably continue to be with panretinal laser photocoagulation, but the addition of new pharmacologic agents (e.g. anti-Vascular Endothelial Growth Factor or anti-VEGF) could reduce the requirement for this destructive treatment.

According to Dr Alistair Laidlaw, Consultant Vitreoretinal Specialist at St Thomas’ Hospital, London, UK, the prevention of diabetic retinopathy through effective screening will take priority. He foresees an increased use of non-mydriatic, wide-field, low-light systems, which will make screening comfortable and effective. Management will be through improved medical care of diabetes overall, and use of newer agents (e.g. protein kinase C inhibitors) as well as further developments in non-destructive laser systems.

**Retinopathy of prematurity (ROP)**

Dr Rajivardhan Azad, Professor of Ophthalmology and Head of Vitreo-Retinal and ROP unit at the Dr R.P. Centre for Ophthalmic Sciences, New Delhi, predicts that by 2020, there will be increased awareness of the condition amongst ophthalmologists and neonatologists through better, easier and more cost-effective imaging of the retina (e.g. RetCam).
Retinoblastoma

Dr Carol Shields, Professor of Ophthalmology and Co-Director of the Oncology Service at the Wills Eye Hospital, Philadelphia, foresees earlier detection of cases through increased awareness (e.g. routine screening of the red reflex) which will potentially identify the sporadic cases. A change in chemotherapy treatment from systemic to local delivery will reduce overall side-effects. New developments in slow-release mechanisms (e.g. a reservoir system inserted into the sub-Tenon space which can then be regularly filled with chemotherapeutic agent) and increased use of adjunctive treatments (e.g. locally placed anti-proliferative agents like combretastatin) will further improve treatment success.

Dr Alejandro A. Valenzuela of the Royal Children’s Hospital, University of Queensland, Australia, considers that by 2020, better education and increased surveillance by the health community will be fundamental to earlier diagnosis and successful outcomes. Multimodal therapeutic advances will save not only the life of the patient, but also preserve the eye and, in some cases, preserve the vision. The addition of gene therapy to the particular Rb1 mutation affecting some children may provide a further avenue in management.

Retinal detachments

Dr Yasuo Tano, Professor of Ophthalmology, University of Osaka, President of Asia-Pacific Academy of Ophthalmology, envisages that pars plana vitrectomy (PPV) will take over as the primary choice for detachment repair. Improved imaging with 3-dimensional OCT high resolution imaging will improve visualisation of the posterior segment. Non-vitrecomising macular surgery may offer the hope of non-accelerated progression of nuclear cataract that currently occurs following PPV. Bimanual techniques for surgery are also likely to be more widespread.

Dr G W Aylward, Medical Director at Moorfields Eye Hospital, London, foresees no significant change in the diagnostic and management techniques for routine retinal detachments, as reattachment rates currently reach 90 per cent. He suggests that the main thrust by the year 2020 will be focused at the public health level, such as alerting the public to early symptoms and signs in order to ‘catch’ detachments before the macula is affected.

Dr Borja Corcostegui, President of España de Retina y Vitreo (SERV) and Director of Instituto de Microcirugía Ocular (IMO), Barcelona, added that, as the posterior segment diseases are better understood (particularly the vascular disorders), the range of conditions that require surgery and indications for intervention would be quite different in 2020.

Age-related macular degeneration (AMD)

Dr Rosario Brancato, Professor of Ophthalmology, University San Raffaele, Milan, Italy and Editor of the European Journal of Ophthalmology, predicts that diagnosis for AMD will be directed at three levels:

- Understanding pathological angiogenic mechanisms
- Understanding these effects in the local tissue
- Epidemiological and genetic research regarding predispositions to AMD.

Retinal dystrophies, e.g. retinitis pigmentosa

Dr Ian Constand, Professor of Ophthalmology, University of Western Australia and Director of Lions Eye Institute, Perth, believes that by the year 2020, the range of specific gene defects will have been documented for the various clinical phenotypes. Gene function (e.g. enzymatic, cell signalling) for most dystrophies will also be understood, and animal models in place. Gene therapies will predominantly be available for large families or populations, however there will be some scope for developing customised treatments. In general, the strategy will be:

- Autosomal recessive – replace the defunct gene
- Autosomal dominant – insert a separate gene.

Dr Richard Gisbert, Professor of Ophthalmology, University of Hamburg and co-founder of the European Society of Retinal Specialists (EURETINA), foresees potential treatment options for retinal dystrophies in the future to include cell replacement strategies (i.e. transplantation of stem cells, progenitor cells, primary retinal cells or retinal tissue), gene therapy, and, for advanced cases, electronic retinal prostheses.

These imaging systems will also improve diagnosis and therefore subsequent management. Therapies will include increased use of angiostatic agents, and more focal, less destructive laser treatments. The current belief that surgical techniques do not work will change as advances in techniques develop (e.g. use of plasminogen to liquefy the vitreous to reduce the traction). Dr Clare Gilbert, Reader in International Eye Health at the London School of Hygiene and Tropical Medicine, believes that clinical trials currently underway (e.g. optimum oxygen concentrations for premature babies) will help to reduce the incidence of sight-threatening disease. Until now, detection of ROP has been performed by ophthalmologists using indirect ophthalmoscopy, but in the future digital imagery (taken by non-ophthalmologists) with automated image analysis, or remote (teledicine) expert reading, will offer the possibility of screening in the true sense of the word. Regarding management, she believes that, as with other vasoproliferative eye conditions, there will be medical treatments that block the disease from progressing. The current use of ablative techniques will be kept to a minimum.

The entrance to the World Ophthalmology Congress

The entrance to the World Ophthalmology Congress
Diabetic retinopathy (DR)
Diabetic retinopathy can be subdivided into two basic forms:

1. Non-proliferative
Non-proliferative disease can be identified by a number of clinical findings: microaneurysms, ‘dot and blot’ haemorrhages, cotton wool spots. Clinically significant macular oedema (CSME) is defined as:
- Retinal thickening within 500 microns of the centre of the fovea
- Hard exudation within 500 microns of the centre of the fovea if associated with retinal thickening
- Retinal thickening of one disc area, any part of which is located within one disc diameter (1,500 microns) from the centre of the fovea.
Eyes with CSME benefit from focal/grid laser photocoagulation to the macula.

2. Proliferative
In proliferative diabetic retinopathy, the eyes demonstrate, singularly or in combination, new vessel formation from the disc (NVD), new vessel formation elsewhere (NVE), or neovascularisation of the iris (NVI) capillaries and veins. High-risk characteristics include:
- NVD greater than or equal to one fourth to one third of a disc area (one quarter of a disc area in eyes with large optic discs and one third of a disc area in eyes with small optic discs)
- NVD of any size associated with preretinal or vitreous bleeding
- NVE at least 0.5 disc area and associated with preretinal or vitreous bleeding.
Eyes of high-risk characteristics benefit from panretinal laser photocoagulation.

Age-related macular degeneration (AMD)
Age-related macular degeneration (AMD) is a progressive deterioration of Bruch’s membrane, retinal pigment epithelium, choriocapillaris, and outer retina in the macular area. There are two variants:

1. ‘Dry type’: Drusen and associated retinal pigment epithelial changes (atrophy and clumping). The majority of these eyes have moderate visual disturbance. Extensive or ‘geographic’ atrophy of the retinal pigment epithelium, however, can result in marked visual acuity loss.

2. ‘Wet or Exudative type’: Choroidal neovascularisation (or ‘membrane’) with associated fluid, lipid exude, and haemorrhage under either the retinal pigment epithelium or neurosensory retina. This typically causes moderate to severe loss of central vision. The natural history is poor, often leading to subretinal fibrosis and scarring. This type can be further divided into the ‘classic’ membrane and the ‘occult’ membrane using fluorescein angiography.

Retinopathy of prematurity (ROP)
The condition was initially referred to as retrolental fibroplasia. There are five stages in classification:
- Stage 1: defined as a thin structure within the plane of the retina that separates vascularised from avascular retina.
- Stage 2: represents an elevated ridge that has extended beyond the plane of the retina.
- Stage 3: there is extraretinal fibrovascular proliferation or neovascularisation at the ridge.
- Stage 4: there is a partial traction-like retinal detachment.
- Stage 5: is defined as a total retinal detachment in an open or closed funnel configuration.

The term ‘plus disease’ denotes significantly dilated and tortuous retinal vessels in the posterior pole. It indicates extensive vascular incompetence, and can be associated with vitreous haze, iris vessel engorgement, and poor pupillary dilation. ‘Plus disease’ is a poor prognostic sign in ROP.

Retinal detachment
A retinal detachment occurs when the retina’s neurosensory layer and pigment epithelial layers separate. There are three types of retinal detachments:

Rhegmatogenous
This is the most common type and occurs when there is a break in the sensory layer of the retina, and liquefied vitreous seeps underneath, causing the two layers of the retina to separate.

Tractional
The second most common type occurs when strands of vitreous or scar tissue create traction on the retina, pulling it loose.

Exudative
This results from an accumulation of fluid under an intact neurosensory retina. This usually occurs in conjunction with another disease, e.g. posterior scleritis, choroidal inflammatory conditions and neoplasms.

Retinoblastoma
Retinoblastoma is a primary malignant intraocular neoplasm that arises from immature retino-
The retina is the most common primary intracranial malignancy of childhood. Most cases occur in children younger than six years of age.

The most common presenting symptoms of retinoblastoma are leukocoria (a white pupil), in the tumour-containing eye or eyes, strabismus or symptomatic or asymptomatic visual loss. Retinoblastoma can be hereditary.

**Retinal dystrophies – Retinitis pigmentosa (RP)**

A group of hereditary retinal conditions that cause degeneration of the retina. Retinal cells are among the most specialised cells in the human body and depend on a number of unique genes to create vision. A disease-causing mutation in any one of these genes can lead to vision loss. RP results from a large and as yet unknown number of gene defects, of which around a hundred have been found so far. RP can be passed to succeeding generations by one of three genetic inheritance patterns: autosomal dominant, autosomal recessive, or X-linked inheritance. RP causes the degeneration of photoreceptor cells from the outer edges of the retina, causing a progressive loss of peripheral vision, night blindness and reduced or absent electroretinogram (ERG) recordings.

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**Cochrane Eyes and Vision Group (CEVG) systematic review activity on posterior segment treatments**

**Working titles**

A working title is a protocol in progress prior to being submitted for publication. Working titles dealing with back of the eye treatments currently registered with CEVG are:

- Acupuncture for age-related macular degeneration
- Blood pressure control for the management of diabetic retinopathy
- Calcium dobesilate for diabetic retinopathy
- Carbonic anhydrase inhibitors for cystoid macular oedema
- Chinese herbal treatment for diabetic retinopathy
- Fish oils and vitamin A for hereditary retinal disease
- Intravitreal steroid for retinal vein occlusion
- Laser photocoagulation for retinal vein occlusion
- Laser treatment for diabetic retinopathy
- Laser treatment of drusen in age-related macular degeneration
- Pars plana vitrectomy for diabetic macular oedema
- Pharmacotherapy for preventing proliferative vitreoretinopathy in retinal detachment surgery
- Statins for age-related macular degeneration
- Sub-threshold laser treatment for diabetic maculopathy
- Surgical interventions for repairing simple rhegmatogenous retinal detachments
- Tamponade in surgery for retinal detachment associated with proliferative vitreoretinopathy
- Traditional Chinese medicine for retinitis pigmentosa.

**Available titles**

CEVG invites people from around the world interested in preparing a review on the following titles that do not have authors. Available titles for CEVG reviews in the area of back of the eye treatments are:

- Interventions for cytomegalovirus retinitis
- Sub-macular surgery for age-related macular degeneration.

**Access to Cochrane systematic reviews**

The Cochrane Library is available by subscription, either on CD-ROM or via the internet. Residents in a number of countries, including Australia, New Zealand and South Africa, can access the *Library* free of charge through a ‘national provision’. Higher Education and Further Education residents can access the *Library* using an Athens password.

**Further information**

You can either visit the CEVG website at [www.cochraneeyes.org](http://www.cochraneeyes.org) or email Anupa Shah, Review Group Co-ordinator at [cevg@lshtm.ac.uk](mailto:cevg@lshtm.ac.uk)
How to prescribe spectacles for presbyopia

What is presbyopia?
As we grow older, the lens loses the ability to focus at close distances. Starting around the age of 40, near vision will slowly become worse, but distance vision will not be affected. Spectacles for near vision can help a person see clearly for tasks such as sewing, carving or reading.

Indications
People with presbyopia usually say that their near vision has slowly become worse.

You will need
• Distance and near vision charts with letters, Es or shapes
• Pinhole (optional)
• A trial set of lenses or a selection of ready-made spectacles (RMS). Most people with presbyopia do not need spectacles with powers of less than +1.00 or more than +3.00. See Table 1 for suggested powers.

Method
The correct power of spectacles for presbyopia depends on the person’s age, the distance at which they want to see for near work, and how well they can see.

1 Take a detailed history. Write down the person’s age and medical history and symptoms. Find out if there is a general medical history of diabetes, hypertension, thyroid disease, rheumatoid arthritis, or other eye disease.

2 Find out the person’s working distance, that is the distance at which they would like to do most of their near work.
• Find out what kind of near work (see Figure 1) the person does
• Ask him or her to hold a near vision chart at the distance they do most near tasks. Around 40 cm is a comfortable distance for most people.

3 Measure near vision
• The person holds the near chart at their working distance with both eyes open. Ask them to read the smallest line or show the smallest shapes they can see clearly. Write this down as their near visual acuity (e.g. N8 or J6).
• If the person already has spectacles for presbyopia, measure their near vision with these being worn. Write this down as ‘near visual acuity with spectacles’
• If the person is able to see N8 or better without any spectacles, they might not need spectacles for presbyopia. If they can see N8 or better with their old spectacles, they might not need new spectacles.

4 Identify the correct lens power
• Look up the person’s age in Table 2 and select the power to try first.

<table>
<thead>
<tr>
<th>Person’s age</th>
<th>Lens power</th>
</tr>
</thead>
<tbody>
<tr>
<td>35 to 45</td>
<td>+1.00</td>
</tr>
<tr>
<td>45 to 50</td>
<td>+1.50</td>
</tr>
<tr>
<td>50 to 55</td>
<td>+2.00</td>
</tr>
<tr>
<td>Over 55</td>
<td>+2.50 or higher</td>
</tr>
</tbody>
</table>

How to do... Checking the range of clear vision

Measure near vision with the selected power spectacles or trial lenses. Give the person the near chart to hold at the distance they would like to see clearly. Ask him or her to show the smallest line they can see. If the person cannot see at least the N8 line, try the next stronger power.

Check the range of clear vision. Many people will have good vision using the approximate power, but some may not. If you want to make sure that the lens power is suitable for that individual, check that the person’s range of clear vision with the lens power is correct. The range of clear vision is the distance between the closest that a person can see clearly and the furthest that they can see clearly. The range is achieved by trying out the testing at various distances.

Ask the person to look at the smallest line they can see on the near chart and then bring the chart closer until the letters become blurred. Hold one hand to mark the closest distance (Figure 2a), then ask the person to move the chart further away until the letters become blurred. Mark the furthest distance (Figure 2b).

Ask the person to hold the chart at the distance they want to see clearly. This is the working distance. If the range is correct, the working distance should be in the middle of this range, for example at about 40 cm (Figure 2c). This means that a person will be able to see clearly for the same distance in front and behind their working distance.

The power is correct if the middle point of the range is the same as the working distance. If the middle point of the range is further away than the person’s preferred working distance, try one stronger (higher)

Figure 1. Woman demonstrates her working distance

Figure 2a. Checking the range of clear vision

Figure 2b.

Figure 2c.
How to prescribe spectacles for presbyopia

Disadvantages

• The person will see clearly at distance but will be blurred when they look up.

5 Before prescribing spectacles, note:

• Approximate lens powers, based on age, will not be suitable for all. A weaker lens power than expected for a person’s age, or no presbyopic lenses, might be needed if a person has myopia (short-sightedness). They should remove their distance spectacles if they want to see at a close distance. A lens power stronger than expected for the person’s age may be needed if the person has hyperopia (far-sightedness), low vision, wants to work at a distance closer than 40 cm, or to see very small objects, for example, a 48-year-old man may like to make jewellery at 25 cm, so might need +2.00.

• Do not prescribe a power that is too high. If there is no difference in the near vision when a person looks through a +0.50 stronger power, do not prescribe the stronger power. This is because if the power is stronger than needed, the person will have to hold things too close to their eyes. Also, most people would like to see at their near working distance as well as a little further away. For example, a woman may mainly want to see her sewing at 40 cm, but holds a book at 50 cm and chops vegetables at 60 cm.

• A change in spectacles is usually only necessary if the person needs at least 0.50 stronger than their old spectacles, has received spectacles for presbyopia about two years ago, or can see better with the new spectacles than their old spectacles.

6 Select the type of lenses that would be best for the person. Table 3 describes the options.

Table 3. Types of lenses

<table>
<thead>
<tr>
<th>Types of lenses</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single vision (Ready-made, near or reading spectacles)</td>
<td>Less expensive</td>
<td>The person will see clearly at near but their distance vision will be blurred when they look up</td>
</tr>
<tr>
<td>Bifocal lenses and Multifocal lenses (varifocal)</td>
<td>Useful if a person has a distance refractive error and presbyopia, or if they need to see clearly at distance and near</td>
<td>Usually more expensive than single vision spectacles for presbyopia, and may take longer to acquire</td>
</tr>
</tbody>
</table>

7 Adjust the spectacles and explain how to use them. Before the person leaves with their new spectacles:

• Adjust spectacles to fit properly and feel comfortable

• Explain the use of spectacles for presbyopia and that it may take a little time to get used to them. Tell them to come back if they experience any problems

• Explain how to look after the spectacles so that the lenses do not become scratched. Advise them to wash the spectacles daily with soap and water and wipe with a clean cloth.

8 Remind them to return in about two years to check if they need new spectacles to see more clearly at close distances.
The importance of shielding the eye in referrals of ocular injuries

Tontu Zik
Ophthalmic Clinical Officer, Mbingo Baptist Hospital Eye Department, Cameroon.
Email: MBHcameroon@aol.com

Ocular injuries are common in the northwest province of Cameroon. Here, the majority of the people are farmers. In 2004, 31 patients with ocular injuries were seen in Mbingo Baptist Hospital Eye Department. As of November 2005, 17 patients have been seen. Injuries to the lids are not included. Causes of these injuries include tree branches and leaves, lacerations from knives and cutlusses, pellets from locally made guns used during traditional ceremonies, stone fragments injuring the eye when cracking stones without wearing protective spectacles, sticks and stones when children play.

The majority of people live far away from an eye hospital. Some villages require eight hours of trekking to reach a motorable road to the hospital. The visual prognosis after management of an ocular injury will not only depend on the extent and location of the injury, but also on what happens during transportation to the hospital. The majority of our roads are rough, untarred and dusty. These expose the eye, especially an open injury, to further trauma and foreign bodies. Further injury to the eye during referral could be reduced by the use of an eye shield.

Making an eye shield is easy and inexpensive. It can be made from cardboard or firm paper. Health centres and health posts found in the villages readily have this, for example, from medication boxes. Where available, used x-ray film can also be used.

Our community outreach programme is in partnership with the community-based rehabilitation programme of the Cameroon Baptist Convention Health board, sponsored by Christoffel-Blindenmission (CBM). Their field workers and volunteers see and refer eye patients in the community. Every year, refresher courses are organised, where they are taught, amongst other things, how to make an eye shield.

Figure 1. How to make an eye shield

2006 themes

The themes planned for 2006 include:
- Outreach: beyond the clinic (June 2006)
- Finding the cataract patient (September 2006)
- Glaucoma (December 2006).

Readers are invited to submit 500 word submissions on any of these themes, or any other topic relevant to community eye health. If you want your submission to be considered for a particular theme, please ensure that I receive it at least one month before the publication date.

Victoria Francis, Editor, Community Eye Health Journal

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EXCHANGE Continued

GP s responded to the tele-survey after they were called more than five times. 33.4 per cent (n = 53) of the general practitioners responded after three to five calls and the remaining 20 per cent responded immediately.

31.3 per cent (n = 50) feel that diabetics should undergo an eye examination every six months and 53.3 per cent (n = 85) feel that diabetics should undergo eye examination every year. 15.4 per cent felt that eye examination every two years is sufficient for diabetics. Ophthalmoscopy was done by 1.3 per cent (2/159) of the GPs. Of the two, one GP performs ophthalmoscopy with dilation while the other performs it without dilation. The reason stated for not dilating was lack of time. Almost all GPs said that they would refer a patient with diabetes to an ophthalmologist. 84 per cent of the practicing physicians were aware of laser photoacoagulation as a treatment modality for diabetic retinopathy.

54 per cent of GPs were aware of annual dilated eye examination referral guidelines for diabetics. Regarding attitudes for screening for diabetic retinopathy, only 1.3 per cent of GPs were using direct ophthalmoscopy. Among them only 50 per cent were practicing dilated direct ophthalmoscopy. Barriers for doing diabetic retinopathy screening by general practitioners were lack of time, lack of ophthalmoscopes and lack of training.

Discussion: This study shows the need for training GPs about diabetic retinopathy and its detection with direct ophthalmoscope. Barriers for dilated eye examination, as perceived by GPs, need to be addressed. McCarty et al.1 reported that lack of dilating drops in the practice, lack of confidence in detecting changes, concern about time taken and fear of precipitation of angle-closure glaucoma with their patients were some of the barriers expressed by GPs.1 Knowledge of the guidelines is another important factor to consider. Residency programmes should focus on providing more exposure to ophthalmoscopy practice among GPs, compared to the current low levels of exposure of only a few hours.

References

Getting over the histopathology barrier

Michael Ekuoba Gyasi and Oscar Debrah
Bawku Presbyterian Hospital, Bawku, Upper East Region, Ghana.

In many developing countries, specialised laboratory services are simply non-existent. Where they exist, they are usually limited to large hospitals. The distance and the cost of accessing such specialised services, place enormous barriers to remote eye care facilities and the patients they serve. In Ghana, getting histopathology tests done could take as long as three months. In our case, the patient has to send it to the nearest facility, located some 600 kilometres away. Such a situation meant that most ophthalmic specimens simply never saw the microscope at all. That is why we felt relieved when a global mail was sent by the Vice President of our Ophthalmological Society, introducing members to a free histopathology service at the Royal Hallamshire Hospital in the UK.

Through such collaboration, we now have a free and reliable ophthalmic histopathology service with results delivered within a few days through email. Packaging materials and guidelines are provided free, with only outward postage paid by the beneficiary institution.

Visiting the centre recently, however, I realised that only a fraction of clinics in Africa have taken advantage of this facility, despite the obvious benefits derived from histopathologic evaluations.

This International Ophthalmic Histopathology Service, is accessible to all developing countries. The centre can be contacted through the following address:
Dr Hardeep Mudhar, Ophthalmic Pathology, Department of Histopathology, E-Floor, Royal Hallamshire Hospital, Glossop Road, Sheffield S10 2JF, UK. Tel: (+44) 0114 2268967.
Email: hardeep@ mudharh.fsnet.co.uk

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COMMUNITY EYE HEALTH JOURNAL | VOL 19 NO. 57 | MARCH 2006
Who responded?
1,704 readers from 84 countries returned the questionnaire within the required time. Figure 1 provides a picture of the regional breakdown of respondents and highlights the fact that the greatest proportion of returns came from Africa, which also provided the best response rate at 13 per cent.

Who are our readers?
Jobs and level of service provision
Sixty per cent of our readers have specialist ophthalmic training, of which 23 per cent are ophthalmologists and 37 per cent are mid-level eye workers. This mid-level group has increased by 17 per cent since the last survey in 1997.

What do readers like about CEHJ?
Ninety seven per cent of readers report finding the journal very useful or useful. The top four types of material described as “very useful” were ranked as: articles on the theme for the issue, Evidence-based Eye Care Series, the Technology Series and original research.

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NOTICES

Erratum
We apologise for a spelling error in the EXCHANGE section of Volume 18 Issue 5. The author of the article “A low-cost, still lamp-based video-documentation system” is Syed Amjad Rizvi, not Syed Amjad Nizvi as published in the paper version. – Editor

Obituary
We have been very sad to hear of the untimely death of Dr Vijay Mehra. He was a member of the first class of diploma students at the International Centre for Eye Health in 1981, just after Professor Barrie Jones had started the new department. He was the first alumnus to become active in population-based research and he was responsible, in collaboration with Dr Darwin Minassian and Dr Angela Reidy, for some landmark observations on cataract aetiology and incidence. We send our deep sympathy to his family and everyone in India who was associated with Dr Vijay Mehra.
– Professor Gordon J Johnson, Director of ICH 1986–2002

Courses and conferences

6th International Glaucoma Symposium (I.G.S)
March 28-31, 2007. Venue: Athens, Greece. Information: Symposium Organisers, Kennes International – Global Congress Organisers and Association Management Services, 17 Rue du Cendrier, PO Box 1726, GH-1211 Geneva 1, Switzerland. Tel: (+41) 22 908 0488, Fax: (+41) 22 732 2850. Email: glaucoma@kennes.com Website: www.kennes.com/glaucoma

Planning for VISION 2020 short course
July 3–7, 2006. Venue: London School of Hygiene and Tropical Medicine, London. Cost: £630. Information: Adrienne.Burrough@lshtm.ac.uk or shortcourses@lshtm.ac.uk

Certificate course in Community Eye Health
October 16 – December 8, 2006. Venue: Kiliimarjaro Centre for Community Ophthalmology. Course aims and objectives: To equip eye health professionals with the skills necessary to develop, implement, and manage A VISION 2020 programme. The eight-week course will focus on disease control, planning, human resource development, management, bridging strategies, and budgeting. The course is suitable for ophthalmologists, policy managers, or other eye care professionals who are responsible for developing and implementing district VISION 2020 plans. Information and admission procedures: Dr Paul Courtright – Email: pcourtright@kcco.net

New resources available

Marsden J (editor). Ophthalmic Care. A new comprehensive textbook written by an international panel of ophthalmic nurses and other contributors. Wiley, 2006. Available from the International Resource Centre, ICHE. Discount price: UK £25 plus post and packaging (for developing countries only). Enquiries – Email: sue.stevens@lshtm.ac.uk

Kanellis G J. Clinical Ophthalmology – a synopsis
Butterworth Heinemann, 2004. Available from the International Resource Centre, ICH. Discount price: UK £30 plus post and packaging (for developing countries only). Enquiries – Email: sue.stevens@lshtm.ac.uk

Useful resources: Back of the eye

Johnston R L. Retina, Vitreous & Choroid Clinical Procedures


Adis B. Detached Retina

Wu Gloria. Retina: The Fundamentals
Saunders (W B) 1995. Available from Waterstones at £33.

Packer A J. Manual of Retinal Surgery

MSc Community Eye Health

Cost: £13,750 (Overseas) or £4,370 (Home)
Venue: The London School of Hygiene & Tropical Medicine

The MSc in Community Eye Health is designed to equip eye care professionals and planners with the knowledge and skills to reduce blindness and visual disability. Training in community eye health extends the training in clinical ophthalmology applied to individual patients, to a consideration of the eye health of whole populations - how these can be assessed, resourced and evaluated.

The course is designed in keeping with the aims, priorities and strategies of VISION 2020. VISION 2020 is a World Health Organisation programme designed to eliminate avoidable and treatable blindness, globally, by 2020. Applicants are expected to be health care professionals involved in eye care and have relevant work experience.

Course duration is one year (full-time) but it is available on a part-time basis over two years.

Application forms are available from the Registry, 50 Bedford Square, London WC1B 3DP, UK. Telephone: +44 (0)20 7299 4646 fax: +44(0)20 7233 0638, e-mail: registry@lshtm.ac.uk or MScCEH@lshtm.ac.uk

Website: www.lshtm.ac.uk/prospectus/masters/mseh.html

Please quote ref: JCEH.

Liesegang T J et al. Retina and Vitreous – Section 12 – Basic Clinical Science Course

Updated annually. Produced by the American Academy of Ophthalmology. Enquiries – Email: wovait@aao.org

Advances in Vitreo-Retinal Disease Management CD
Arawin, 2002. Produced by LAICO. Enquiries – Email: communications@aravind.org

Retinopathy of Prematurity Study
This technical paper reports the results of a retrospective study ofROPat the National Institute of Paediatric Care, Vietnam. The study is part of the first diagnosis, prevention and treatment project in the country. Available from ORBIS International.

Retinopathy of Prematurity Screening
A 28-minute instructional video for ophthalmologists presenting a model developed by the LV Prasad Eye Institute in India for the screening, diagnosis and management ofROPAvailable fromLVPEI.

Community Eye Health Journal back issues

Volume 9, Issue 20, 1996 – Diabetes and Diabetic Retinopathy
Volume 10, Issue 22, 1997 – Retinopathy of Prematurity
Volume 16, Issue 46, 2003 – The Retina

Suppliers

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Article competition
Thank you to readers who submitted an article for the article competition. The final selection of articles will take place at the next editorial meeting in April.

Next issue

The next issue of the Community Eye Health Journal will be on the theme Outreach: beyond the clinic.