Community Eye Health

Volume 16 Issue No. 46 2003

AN INTERNATIONAL JOURNAL TO PROMOTE EYE HEALTH WORLDWIDE

SUPPORTING VISION 2020: THE RIGHT TO SIGHT

The Resolution of the World Health Assembly on the Elimination of Avoidable Blindness

FIFTY-SIXTH WORLD HEALTH ASSEMBLY

WHA 56.26

Agenda Item 14.17: 28 May 2003

Elimination of Avoidable Blindness

The Fifty-sixth World Health Assembly,

Having considered the report on elimination of avoidable blindness;[1]

Recalling resolutions WHA22.29, WHA25.55 and WHA28.54 on prevention of blindness, WHA45.10 on disability prevention and rehabilitation, and WHA51.11 on the global elimination of blinding trachoma;

Recognizing that 45 million people in the world today are blind and that a further 135 million people are visually impaired;

Acknowledging that 90% of the world's blind and visually impaired people live in the poorest countries of the world;

Noting the significant economic impact of this situation on both communities and countries;

[1]Document A56/26

Aware that most of the causes of blindness are avoidable and that the treatments available are among the most successful and cost-effective of all health interventions;

Recalling that, in order to tackle avoidable blindness and avoid further increase in numbers of blind and visually impaired people, the Global Initiative for the Elimination of Avoidable Blindness, known as VISION 2020: The Right to Sight, was launched in 1999 to eliminate avoidable blindness;

Appreciating the efforts made by Member States in recent years to prevent avoidable blindness, but mindful of the need for further action,

1. URGES Member States:

(1) to commit themselves to supporting the Global Initiative for the Elimination of Avoidable Blindness by setting up, not later than 2005, a national VISION2020 plan, in partnership with WHO and in collaboration with nongovernmental organizations and the private sector;

(2) to establish a national coordinating committee for VISION 2020, or a national blindness prevention committee, which may include representative(s) from consumer or patient groups, to help develop and implement the plan;

(3) to commence implementation of such plans by 2007 at the latest;

(4) to include in such plans effective information systems with standardized indicators and periodic monitoring and evaluation, with the aim of showing a reduction in the magnitude of avoidable blindness by 2010;

(5) to support the mobilization of resources for eliminating avoidable blindness;

2. REQUESTS the Director-General:

(1) to maintain and strengthen WHO's collaboration with Member States and the partners of the Global Initiative for the Elimination of Avoidable Blindness;

(2) to ensure coordination of the implementation of the Global Initiative, in particular by setting up a monitoring committee grouping all those involved, including representatives of Member States;

(3) to provide support for strengthening national capability, especially through development of human resources, to coordinate, assess and prevent avoidable blindness;

(4) to document, from countries with successful blindness prevention programmes, good practices and blindness prevention systems or models that could be modified or applied in other developing countries;

(5) to report to the Fifty-ninth World Health Assembly on the progress of the Global Initiative.

Tenth Plenary Meeting, 28 May 2003
A56/VR/10

Community Eye Health 2003; 16: 17–32 Issue No. 46

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Volume 16 Issue No. 46 2003

Supporting VISION 2020:
The Right to Sight

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Typeset by
Regent Typesetting, London

Printed by
The Heyford Press Ltd.

On-line edition by
OASIS/Xalt www.jceh.co.uk

ISSN 0953-6833

The Resolution of the World Health Assembly on the Elimination of Avoidable Blindness

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The word 'resolution', is a derivative of the French word, 'soluere', meaning 'to solve', and is defined by The Oxford Dictionary as:

'The formal expression of opinion or intention by a legislative body or public meeting'.

The Resolution on the Elimination of Avoidable Blindness adopted by the Fifty-sixth World Health Assembly meeting in Geneva on the 28 May 2003, therefore, has special significance.

The Resolution was adopted by the member states unanimously and testifies first and foremost to the fact that the Right to Sight is not a contentious issue.

The Resolution is a statement of intent based on an assessment of the prevailing situations with regard to avoidable blindness and its consequences. The Resolution first urges member states to take note of the magnitude, and far reaching consequences of needless blindness and visual impairment in their own countries, generally among the poorest of their poor citizens. Next, it urges a course of action for the World Health Organization, to be carried out in collaboration with her various partners. This takes the form of supportive actions to member states, to make their stated intent a reality.

Thus, there is political awareness and commitment, The Resolution also calls for enhanced support from WHO, the International Agency for the Prevention of Blindness, its constituent members, including professional bodies, civil society organisations and the private sector – to assist member states. This provides a new opportunity to stem the rising tide of avoidable blindness through the synergy derived from working in Partnership. This Partnership is seen as an unique strength of the Global Initiative. The international aid community emphasises national capacity development as a critical factor in poverty alleviation. This is equally applicable to VISION 2020. Such an effort at capacity development, will promote self-reliance and increasing sustainability.

The stated objective of VISION 2020: The Right to Sight is the global elimination of avoidable blindness by the year 2020. In the process of achieving this objective it is hoped that each member state will develop a sustainable, comprehensive eye care system as an integral part of the national health system. This will ensure that avoidable blindness is eliminated as a public health problem in all countries, and within any community.

The Resolution deals with plans of action, implementation, targets, monitoring and evaluation. These are all important elements as we move the Global Initiative forward.

However, plans in themselves mean little unless these are implemented. Enhanced implementation will require better utilisation of existing resources, in the first instance, and new additional resources where appropriate. Acquisition of new resources must go hand in hand with learning how to deploy these resources to accomplish often complex tasks. Implementation without monitoring what is being done, and evaluating their outcomes, is to live in the complacent world of 'presumed merit'.

The WHA Resolution is a wake–up call, based on the realisation of a major escalating public health crisis in the field of eye health – a crisis with far reaching socio-economic, developmental and quality of life implications. It is also a crisis against which we can act, using the knowledge, skills, and cost effective interventions already at our disposal.

The Global Initiative provides us all with an opportunity to translate our resolve and plans, into action. History will prove whether we seized and acted on that opportunity to ensure the right to sight for all persons.

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Retinal Diseases and VISION 2020

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Historically, retinal disease has had a low priority in prevention of blindness programmes in developing countries. There are several reasons for this. Firstly, it was thought that retinal disease was an uncommon cause of blindness in the developing world; secondly, that the results of treating retinal disease did not justify the effort and expense involved; and, thirdly, that the equipment required was too costly and unreliable for use in a developing country environment. Finally, there is a lack of skilled personnel with sub-speciality training in retinal disease.

Retinal Disease Worldwide

As countries become wealthier, and per capita income increases, the prevalence of blindness decreases, and the causes of blindness change. In a poor African country, the major blinding conditions are likely to be cataract and corneal scar. In a middle income country in Latin America, the leading causes of blindness will be glaucoma and diabetic retinopathy. Because cataract surgery is more readily available, fewer people become blind from cataract. In a wealthy country, glaucoma and cataract will continue to be very common and important conditions, but most of the blindness will be due to retinal disease.

Retinal diseases are already the most common cause of childhood blindness worldwide.[1] Some of these children are blinded by inherited retinal conditions, such as retinitis pigmentosa, which can neither be treated nor prevented at present. However, many of them have retinopathy of prematurity, which can be prevented, and is treatable in its early stages. The excellent and very detailed Andhra Pradesh Eye Disease Study (APEDS) found that retinal diseases were a much more common cause of adult blindness in India than had previously been thought.[2] The APEDS study dilated the pupils of every subject, and all those with reduced vision had their fundus checked by an ophthalmologist. Blindness surveys in which the fundus is not routinely examined may underestimate the prevalence of blindness caused by retinal disease.

Diabetes is a growing problem in developing countries. In India, it is estimated that 8–10% of the population is diabetic, and the prevalence is increasing.[3] Although population-based studies suggest that diabetic retinopathy is not a major cause of blindness in India at present,[4] this is likely to change in the future.

Our own efforts will increase the incidence of retinal disorders. At present, there are about 10 million cataract operations per year. By 2020, it is intended to increase this to over 30 million. Almost all this growth will take place in poorer, developing countries. More cataract surgery will lead to more posterior segment complications of cataract surgery, such as retinal detachment, and retained lens material. These complications are very treatable, provided a skilled and well-equipped vitreo-retinal surgeon is available.

In view of these trends, it is likely that retinal diseases are already a significant and increasing problem in every part of the world.

Treatment of Retinal Disease

The second reason for the low priority of retinal disease is the belief that little can be done to treat these conditions. It is true that there are many retinal degenerations for which no cure is available. However, patients can benefit greatly from receiving an accurate diagnosis, with a detailed explanation and clear prognosis; provision of low vision aids; and genetic counselling. Even where the disease is untreatable in one eye, prevention may be possible in the other eye. For example, the Age Related Eye Disease Study showed that in patients with age-related macular degeneration in one eye, daily vitamin and zinc supplements reduced the risk of macular degeneration in their other eye.[5] Recent studies have shown that surgery for retinal detachment in India and East Africa can be very effective. Retinas were successfully re-attached in 70–80% of patients, and even in 'macula-off' detachments, over 60% of eyes could see 6/60 or better.[6] Approximately 25% of retinal detachment operations restored sight to a blind person.

Equipment

The third issue is the availability and reliability of equipment. Newer technology offers significant improvements. For example, conventional fundus cameras rely on film to record the images. This is expensive, and developing the films is also costly. Digital fundus cameras are more expensive to buy, but record the image directly to a computer, and do not need film, which substantially reduces the running costs. The first Argon lasers were bulky, expensive, and fragile. However, newer diode lasers are robust and portable. While they are not cheap, the running costs are very low. Assuming a cost of $40,000, and a five year life expectancy (which is pessimistic), if 400 treatments are carried out each year, the cost per treatment is $20. The key to cost effectiveness is volume – if the number of treatments is reduced, the cost per treatment increases. Vitrectomy equipment is also expensive, and can be more difficult to maintain. However, it is useful not only for retinal surgery, but also

for managing congenital cataract, trauma, and complicated cataract surgery. Because the capital cost of lasers and vitrectomy machines is so high, they should only be used in centres that have sufficient volume of patients to justify the expense.

Training in Retinal Disease

To summarise, retinal disease is likely to become more common in the developing world. Treatment of retinal conditions is improving, and may be cost effective, even in a developing world eye clinic. Owing to advances in technology, equipment to treat retinal disease, although still expensive, is now much more suitable for use in a developing country. However, a significant limitation remains the shortage of skilled personnel. Ophthalmic education should prepare eye workers not only for the challenges they will face today, but also for future developments. This means that we need more developing world ophthalmologists with sub-speciality training in retinal disease who can train future generations of eye workers.

Questions:

1. Is your training programme orientated towards diseases that are becoming less common, or is it aimed at preparing eye workers to manage the conditions that are going to be most important over the next twenty years?
2. Is retinal disease an increasing problem in your country – and if so, how is your training programme planning to address this challenge?

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Diabetic Retinopathy: Clinical Findings and Management

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Diabetes Mellitus

Diabetes mellitus is a metabolic abnormality in which there is a failure to utilise glucose and hence a state of hyperglycaemia can occur. If hyperglycaemia continues uncontrolled over time, it will lead to significant and widespread pathological changes, including involvement of the retina, brain and kidney.

In industrialised countries, approximately 1% of the population is diabetic, and at least another 1% are undiagnosed diabetics. Insulin dependent diabetes (IDDM), accounts for approximately 10–15% of cases, the remainder being maturity onset or non-insulin dependent diabetics (NIDDM). Diabetes mellitus is an international public health problem with estimated prevalences ranging from 2.0% to 11.7% in studied populations across the world.[1]

Prevalence of Diabetic Retinopathy

Diabetic retinopathy is increasingly becoming a major cause of blindness throughout the world in the age group of 20–60 years.[2,3,4] Loss of productivity and quality of life for the patient with diabetic retinopathy will lead to additional socio-economic burdens on the community.

Diabetic retinopathy is the cause of blindness in approximately 2.5 million of the estimated 50 million blind people in the world. However, diabetic retinopathy, as a cause of blindness, is less common in India according to population-based studies.[5,6] A recent study of diabetic patients in Pakistan indicated that cataract and uncorrected refractive error were more common causes of visual impairment than retinopathy.[7]

The VISION 2020 protocol projects diabetic retinopathy and the glaucomas as the 'emerging' causes of blindness in developing countries.

Epidemiological Studies in Diabetic Retinopathy

Epidemiological studies such as the Wisconsin Epidemiological Study on Diabetic Retinopathy,[8] the Diabetic Retinopathy Study (DRS),[9,10,11] the Early Treatment of Diabetic Retinopathy Study (ETDRS),[12,13,14,15,16,17] the Diabetes Control and Complications Trial (DCCT),[18] the Diabetic Retinopathy Vitrectomy Study (DRVS), [19,20] and the UK Prospective Diabetic Survey [21] have established the various risk factors and provided guidelines for the management of diabetic retinopathy.

General Risk Factors for Diabetic Retinopathy

1. ***Type of Diabetes and its Duration***. Tables 1 and 2 show the relationships between the type, duration of diabetes mellitus and retinopathy.
2. ***Control of Diabetes Mellitus***. The Diabetes Control and Complications Trial (DCCT)[18] has shown that in Type 1 insulin dependent diabetes mellitus (IDDM), good control of metabolic status will reduce the risk of progression of diabetic retinopathy and delays the onset of retinopathy in patients who do not have retinal changes at the time of presentation. The United Kingdom Prospective Diabetes Study (UKPDS)[21] has confirmed that good glycaemic control in Type 2 non-insulin dependent diabetes mellitus is also beneficial and delays the onset of retinopathy.
3. ***Hypertension***. Reports have indicated that high diastolic blood pressure in young individuals[8] and higher systolic blood pressures in older individuals[21] can worsen the retinopathy.
4. ***Pregnancy*** in women can be associated with worsening of the retinopathy.
5. ***Hyperlipidaemia***. Some studies have indicated that high levels of serum cholesterol and/or triglycerides are significant risk factors for retinopathy. However, it is yet to be clearly proved that therapy to reduce serum lipids affects any retinopathy. Cigarette smoking may or may not be an additional risk factor as its effect on cardiovascular disease is well documented.
6. ***Age***. In younger onset diabetes, diabetic retinopathy is uncommon before the age of 13 years. The onset of puberty may influence retinopathy – although the duration of diabetes is a significant factor. In those with older onset diabetes there is an increased frequency of retinopathy in those younger than 50 years.
7. ***Ethnicity***. The variety of study designs make comparisons difficult, but Afro-American blacks do seem to have more retinopathy than whites.

Table 1: Any Retinopathy
(Viswanath K, unpublished data)

	< 2 years	> 15 years
IDDM	2%	95%
NIDDM	10%	58%

Table 2: Proliferative Retinopathy
(Viswanath K, unpublished data)

	< 4 years	> 15 years
IDDM	0%	26%
NIDDM	3%	4%

Clinical Types of Diabetic Retinopathy

Clinical classification is as follows:

- ***Non-proliferative diabetic retinopathy***
- ***Proliferative diabetic retinopathy***.

Non-Proliferative Diabetic Retinopathy (NPDR)

The lesions in the retina at this stage are within the retina and include microaneurysms, small 'dot and blot' haemorrhages, 'splinter' haemorrhages, intraretinal microvascular abnormalities (IRMA) and 'cotton wool' spots.

The presence of these lesions in various degrees determines whether the NPDR is 'mild', 'moderate', 'severe' and 'very severe'.

1. *Mild Non-Proliferative Diabetic Retinopathy*

At least one microaneurysm, and also dot, blot or flame-shaped haemorrhages in all four fundus quadrants.

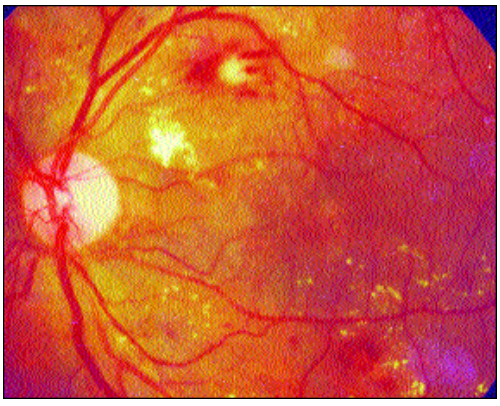

Fig. 1: Moderate non-proliferative diabetic retinopathy

Photo: K Viswanath

2. *Moderate Non-Proliferative Diabetic Retinopathy*

Intraretinal microaneurysms and dot and blot haemorrhages of greater severity, in one to three quadrants. Cotton wool spots, venous calibre changes including venous beading, and intraretinal microvascular abnormalities are present but mild.

3. *Severe Non-Proliferative Diabetic Retinopathy*

At least one of the following should be present: a) 'severe' haemorrhages and microaneurysms in all four quadrants of the fundus, b) venous beading, which is more marked in at least two quadrants, and c) intraretinal microvascular abnormalities, which are more severe in at least one quadrant.

4. *Very Severe Non-Proliferative Diabetic Retinopathy*

Two or more of the criteria for severe non-proliferative diabetic retinopathy, but without any proliferative diabetic retinopathy.

Diabetic Maculopathy

Diabetic retinopathy situated in and around the macula is described as ***diabetic maculopathy***, which can result in significant visual impairment. All these background diabetic retinal changes are due to pathology occurring at the microvascular level of the retina, including dilatation of the capillaries, destruction of the capillary walls and closure of the capillaries resulting in hypoxia and micro-infarcts.

The Early Treatment of Diabetic Retinopathy Study (ETDRS) classified patients who were given macular focal laser therapy, based on whether 'clinically significant macular oedema' was present or not.[12,13,15] This was classified as:

- Retinal thickening at or within 500µ (one third of the diameter of the optic disc) at the centre of the macula
- Hard exudates at or within 500µ of the centre of the macula, if there is thickening of the adjacent retina
- An area of retinal thickening greater than one optic disc area in size, at least a part of which is within one disc diameter of the centre of the macula.

The following photographs show non-proliferative and macular diabetic retinopathy.

1. Moderate non-proliferative diabetic retinopathy (Figure 1).
2. Diabetic maculopathy (Figure 2).

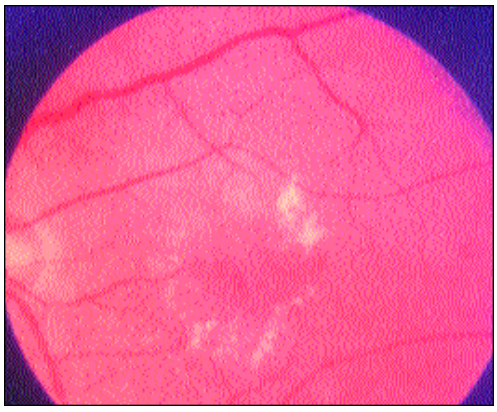

Fig. 2: Diabetic maculopathy

Photo: K Viswanath

Proliferative Diabetic Retinopathy (PDR)

Micro-vascular pathology with capillary closure in the retina leads to hypoxia of tissue. The hypoxia leads to release of vaso-proliferative factors which stimulate new blood vessel formation to provide better oxygenation of retinal tissue. These new vessels growing on the retina are called neovascularisation elsewhere (NVE) and those on the optic disc are called neovascularisation of the disc (NVD). These new vessels can bleed and produce haemorrhage into the vitreous.

The following photographs show examples of proliferative diabetic retinopathy (PDR).

1. PDR with NVE (Figure 3).
2. PDR with NVD (Figure 4).

Advanced Proliferative Diabetic Retinopathy

The unchecked progression of proliferative diabetic retinopathy can lead ultimately to tractional retinal detachment, which may or may not involve the macula. Vitreous haemorrhage may require B-scan ultrasonography to determine if a tractional or rhegmatogenous (retinal break or hole) retinal detachment is present. Neovascularisation of the anterior segment of the eye may cause intractable painful blindness due to neovascular glaucoma.

Screening for Diabetic Retinopathy

Diabetic retinopathy does not reduce vision in its early stages, when treatment is most effective. Preventing blindness from retinopathy relies on early detection of asymptomatic disease by fundus examination. The fundus may be examined by ophthalmoscopy, using a slit lamp and either a contact lens or a 78D lens, or by retinal photography, which may use conventional film or a digital camera. It has been shown that fundus photography is the most accurate means of screening for retinopathy. The photos allow an ophthalmologist to examine a large number of eyes very quickly. Digital fundus photography is expensive initially, but has very low running costs as it does not require film or developing the pictures. The images are available instantly. The quality of digital photos is not as good as conventional film,

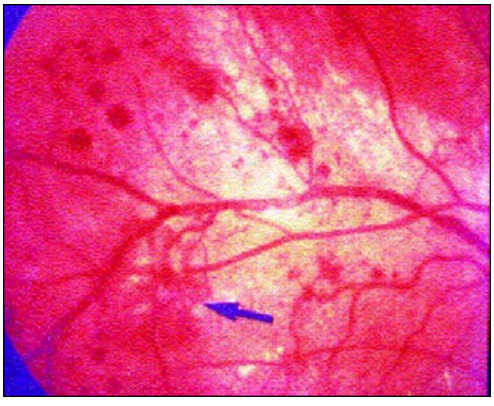

Fig. 3: Proliferative diabetic retinopathy with neovascularisation elsewhere

Photo: K Viswanath

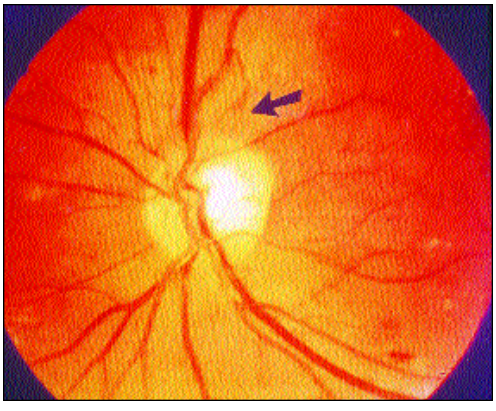

Fig. 4: Proliferative diabetic retinopathy with neovascularisation on the disc

Photo: K Viswanath

however, they are quite adequate for retinopathy screening.

In most developing countries there are too few ophthalmologists for every diabetic to be examined annually by an ophthalmologist. If retinal photography is not possible, then the fundus may be examined by the diabetic physician, an optometrist, or an ophthalmic assistant.

Screening for diabetic retinopathy is only effective if it achieves high coverage (at least 80% of known diabetics). It is essential that the screening process should be made as convenient as possible for diabetic patients. It should also be free.

Insulin-dependent/juvenile-onset diabetes

- Dilated fundus examination every year beginning 5 years after diagnosis, from puberty onwards
- Examinations more frequently once diabetic retinopathy is diagnosed.

Non insulin-dependent/maturity-onset diabetes

- Dilated fundus examination every year once diabetes diagnosed
- Examination more frequently once diabetic retinopathy diagnosed.

Diabetics are at significantly increased risk of cataract. All diabetics should have an annual measurement of visual acuity, and those with vision of less than 6/18 in either eye should have a full eye examination, as they may have cataract, refractive error, or glaucoma.

Treatment

Diabetic Control

As previously mentioned, good glycaemic control significantly reduces the risk of diabetic retinopathy developing and subsequently progressing. The importance of good control should be emphasised.

Laser Photocoagulation

The advent of laser photocoagulation of the retina has dramatically changed the management of diabetic retinopathy. The photocoagulation of non-proliferative diabetic retinopathy with clinically significant macular oedema is called macular photocoagulation, and widespread photocoagulation for proliferative diabetic retinopathy is called pan-retinal photocoagulation.

Macular Photocoagulation

Photocoagulation for diffuse leakage around the macula may be applied in a 'grid' fashion to prevent leakage – grid macular photocoagulation. Diffuse or focal leakage can be identified by fundus fluorescein angiography (FFA). FFA is done with black and white retinal photography using the contrast dye, sodium fluorescein, injected into the blood.

If 'clinically significant macular oedema' is present this may include:

- Focal leaks greater than 500µ from the centre of the macula, causing retinal thickening or hard exudates
- Focal leaks 300µ–500µ from the centre of the fovea, without significant damage to the perifoveal capillary network
- Areas of diffuse leakage on fluorescein angiography within the macular area
- Avascular areas within the macular area.

Pan-retinal Photocoagulation (PRPC)

Photocoagulating the posterior 45°–60° of the retina, away from the vascular arcades of the macula, with graded burns – to reduce the oxygen demand of the hypoxic retina in diabetic retinopathy – converts the hypoxic zones of the retina into anoxic zones, thereby reducing the release of vasoproliferative factors (Figure 5). PRPC, therefore, prevents new vessels appearing and can result in the regression of already existing new vessels on the retina or optic disc.

PRPC is indicated for the following clinical findings:

- Proliferative retinopathy
- New vessels of the iris.

Follow-up Management

Patients with diabetic retinopathy, whether treated or untreated, need periodic follow-up. Patients with diabetic maculopathy should be reviewed 3 to 4 months after treatment to check if the macular oedema has subsided. Patients who receive PRPC should be reviewed in 3 months to check for the regression or closure of new vessels and for the presence of any new vessels.

Surgery in Diabetic Retinopathy

Non-resolving vitreous haemorrhages and tractional retinal detachment, due to fibrovascular proliferation involving the macular region, require surgical procedures such as vitrectomy, pealing of epi-retinal membranes, endo-laser photocoagulation during surgery and vitreous replacement with silicone oil or perfluorocarbons.

Vitrectomy

Vitrectomy is indicated for vitreous haemorrhage – performed early for insulin dependent diabetics and after six months in non-insulin dependent diabetics if the haemorrhage does not clear.

Prevention of Blindness due to Diabetic Retinopathy

Prevention of blindness due to diabetic retinopathy requires information on the prevalence of diabetic retinopathy in the general population, identifying the high risk groups amongst diabetics, using cost effective screening methods such as ophthalmoscopy or fundus photography. Treatment facility centres require photocoagulators. Continuing medical education for diabetic care physicians, training ophthalmologists in photocoagulation and health education amongst diabetic patients should be established. It should be kept in mind that diabetic patients in certain populations may have visual impairment or blindness due to other causes, such as refractive error or cataract.

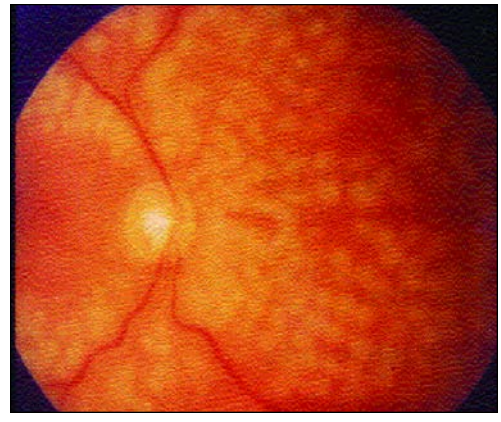

Fig. 5: Laser pan-retinal photocoagulation
Photo: K Viswanath

Acknowledgements and Further Reading

The authors are very grateful to Ian Murdoch FRCS FRCOphth and David Yorston FRCS FRCOphth who kindly reviewed this article.

Our particular thanks to Philip Hykin FRCS FRCOphth who has written previously for this Journal and whose articles were a source of both guidance and content for this article.

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Letters

Monitoring Cataract Surgical Outcomes
Moses C Chirambo (J Comm Eye Health 2002; 15: 58–59)

Dear Editor

Dr Chirambo paints a realistic picture of African cataract surgery where the result of the average cataract operation is not reaching the WHO recommended visual outcome.

Suppose I am a cataract surgeon working in an isolated hospital. Suppose I have the intellectual honesty and humility to begin the process of auditing my results. Suppose I find that 50% or more of my post-operative cataract patients fail to attain 6/18 or better vision. Who will help me to improve?

Suppose my surgical skills need to be updated. Suppose my selection of patients needs to be bettered. Where do I turn for help? Improvement is not going to happen simply by doing an audit. lf my results are mediocre then I need a non-threatening helping hand. But from where and from whom?

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MRCOphth
H pital deBOKO
Parakou
Benin Republic

Monitoring Cataract Surgical Outcomes
Hans Limburg (J Comm Eye Health 2002; 15: 56–57)
Monitoring Cataract Surgical Outcomes: Computerised Systems
David Yorston (J Comm Eye Health 2002; 15: 51–53)

Dear Editor

I read with interest the articles by Hans Limburg and David Yorston. Both mention using software to help with monitoring and the production of reports. David Yorston goes on to say '. . . the design of the database and the reports do need input from someone with the necessary expertise.' This expertise was obviously available at Kikuyu (Kenya) but will not necessarily be available to everyone.

I would therefore like to suggest that, if possible, the relevant files are made available to others who perhaps already have the hardware and software necessary, but lack the technical expertise to adapt the software for this purpose. This would also have the advantage that information could be readily shared between Eye Units and that, at a National, or Regional level, reports could be easily produced. Perhaps one means of disseminating these files would be by making them available to download from the JCEH website. In the future, perhaps other resources (power-point presentations, photographs from the teaching slide sets, leaflets, etc.) might be made avail-able in this way. This would help to avoid already hard pressed personnel 're-inventing the wheel' on a regular basis.

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Retinal Detachment

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Detachment of the retina is a serious event, which may result in complete blindness. The outer segments of the photoreceptors receive oxygen and nutrition from the choroid. If the retina is detached from the choroid, the photoreceptors will fail. The fovea has no retinal blood vessels and depends wholly on the choroid for its oxygen, so detachment of the macula leads to permanent damage to the cones and rods at the posterior pole, and loss of vision. If the macula is not detached, then good vision can be retained if the retina is re-attached promptly.

Types of Retinal Detachment

Retinal detachment (RD) is broadly classified into three types based on the clinical appearance and underlying aetiology.

1. ***Rhegmatogenous retinal detachment*** (RRD) where the RD develops due to a retinal break ('rhegma', meaning a rent or a fissure) (Figure 1). Fluid, from the vitreous cavity, passes through the retinal break into the potential space under the retina, leading to separation of the retina from the underlying choroid. This requires surgical treatment.
2. ***Tractional retinal detachment*** (TRD) which occurs due to pre-retinal membrane formation and scarring that pulls the retina from its attachment. This may require surgery depending on the extent of the RD. The commonest causes of TRD are diabetes, Eales's disease, sickle cell retinopathy and trauma.
3. ***Exudative and serous retinal detachments*** occur due to abnormalities in water transport across the bed of the retina (retinal pigment epithelium) or in its blood supply.

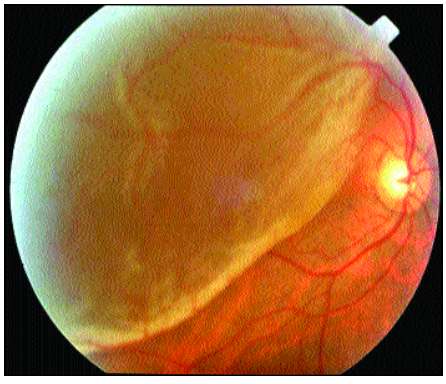

Fig 1: Recent subtotal rhegmatogenous retinal detachment

Photo: Subhadra Jalali

Tractional and exudative/serous retinal detachments are less common and will not be discussed in this paper.

Symptoms and Signs

The commonest presenting symptom of RD is sudden, painless loss of vision or blurring of vision in the affected eye. Some patients with partial RD notice field loss, i.e., loss of vision in only one part of the visual field and describe this as a veil or shadow in one area of their vision. Flashes and floaters may occur in the affected eye a few days or weeks before the loss of vision. This is due to vitreous degeneration and its traction on the retina. Inferior retinal detachments can often be silent and slowly progressive so that the onset of RD goes unnoticed until it reaches the posterior pole. Sometimes RD is accompanied by mild discomfort and redness due to associated uveitis and hypotony, and this may be mistakenly diagnosed as idiopathic anterior uveitis. In children and young adults, RD may be asymptomatic initially and is diagnosed only after the affected eye develops squint, or redness, or a white pupillary reflex due to rapid progression of cataract.

In developing countries, retinal detachment frequently presents late, and this means that the macula is detached in approximately 90% of eyes at presentation. Patients are more likely to have scarring and fibrosis of the retina, and other problems associated with long-standing retinal detachment. Because the abnormalities that caused the detachment are often bilateral, up to a third of patients may be blind in their other eye at presentation – often because of untreated retinal detachment.[1]

Diagnosis of Retinal Detachment

The best method of diagnosing RD is by binocular indirect ophthalmoscopy with scleral indentation. An obvious RD is recognised by loss of the red fundus reflex and marked elevation of the retina (Figure 1). The retina appears grey, and shows folds and undulations. Shallow detachments are difficult to diagnose but can be seen with stereoscopic visualisation of the retinal vessels that cast a shadow on the underlying retinal pigment epithelium (Figure 2).

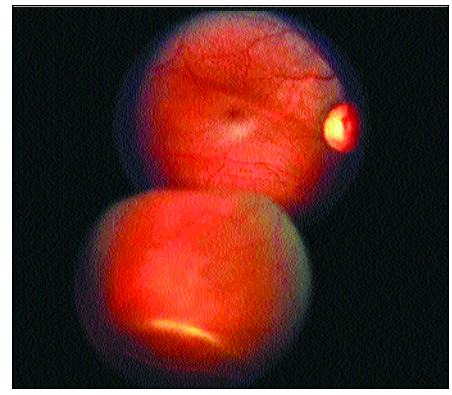

Fig. 2 Shallow retinal detachment with traumatic dialysis misdiagnosed as serous macular detachment due to central serous retinopathy – can be managed by simple scleral buckling

Photo: Subhadra Jalali

It is important to assess the state of the macula. If the macula is still attached, this is a medical emergency, and the patient should have surgery within 24 hours in order to prevent macular detachment and permanent loss of vision. If the macula is already detached, then surgery should be carried out within a week or two.

In eyes with opaque media, ocular B-scan ultrasonography is useful for diagnosing RD and associated pathology, like proliferative vitreoretinopathy (PVR), intraocular foreign bodies, etc. Ultrasonography also rules out many lesions associated with exudative retinal detachments such as tumours, posterior scleritis, etc.

Predisposing Causes

Although RD can occur in any eye, certain eyes are predisposed to develop detachment. The risk factors are given in Table 1. All eyes that are predisposed to RD should undergo periodical, dilated retinal examination (including the retinal periphery by scleral depression), to detect any retinal breaks/areas of lattice degeneration, that can predispose to RD. Early detection of some of these conditions can give an opportunity for prophylactic treatment.

Management

Most retinal detachments progress to total retinal detachments and complete loss of vision. If the retina is not re-attached promptly (usually less than a week after macular detachment), then visual recovery is progressively affected. Also, long-standing retinal detachments start to develop scarring, called 'proliferative vitreoretinopathy' (PVR) that can prevent re-

Table 1: Risk Factors for Rhegmatogenous Retinal Detachment*

1. Axial myopia.
2. Post cataract surgery (aphakia/pseudophakia) especially if the posterior capsule is ruptured during surgery and/or there is vitreous loss.
3. Yag laser capsulotomy.
4. Lattice degeneration of the retina.
5. Symptomatic (flashes/floaters) retinal tears.
6. Ocular trauma.
7. RD in one eye.
8. Family history of RD.
9. Certain genetic disorders such as Marfan's syndrome, Stickler's syndrome.
10. Pre-existing retinal diseases like coloboma choroid, retinoschisis.
11. Following acute retinal infections as in acute retinal necrosis syndrome (ARN) or CMV retinitis.

*** Excludes causes that result in combined rhegmatogenous and tractional retinal detachment**

attachment. Besides PVR changes, chronic retinal detachments can develop other complications such as hypotony, pigmentary glaucoma, new iris vessels, cataract and uveitis, which can compromise visual outcome. Rarely, the detachment does not progress, either due to spontaneous closure of the retinal break or by development of demarcation lines.

The principle of retinal re-attachment surgery is to close all the retinal breaks and create strong chorioretinal adhesions so that these breaks do not open and new breaks do not occur.

Two approaches are established to achieve this objective. One is an external approach using scleral indentation with silicone material called 'scleral buckling'. This approach needs minimal instrumentation and materials, and is widely available. It is suitable for uncomplicated forms of retinal detachment, with a high success rate. However, this surgery is not appropriate for complicated retinal detachments such as those with PVR (Figures 3a, 3b), giant retinal tears, coloboma choroid, penetrating ocular trauma, etc.

In these situations, an internal approach called `vitrectomy' is used. This requires expensive and complex equipment and is available in few centres in developing countries. However, vitrectomy techniques have revolutionised retinal detachment surgery, giving a higher rate of successful re-attachment than previously.

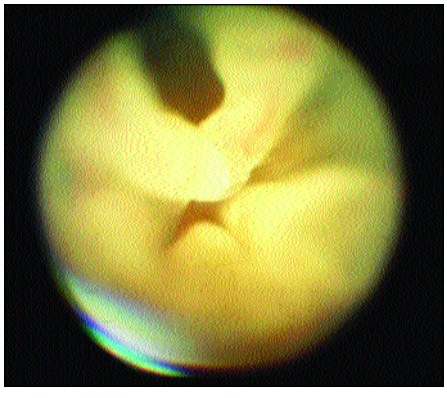

Fig. 3a: Chronic retinal detachment with advanced PVR and large horse-shoe tear

Photo: Subhadra Jalali

Results of Treatment

RD is no longer an incurable condition. Surgical results have improved considerably in the last two decades.[2,3] In developing countries, the final re-attachment rates vary from 77–87% with the use of modern technology.[1] The anatomical success depends on a variety of factors including the type of retinal detachment, age of patient and surgical expertise. Unfortunately, visual results do not always match the anatomical success. If the macula has been detached for a long time, central vision will not be regained, however, the patient will usually obtain useful navigational vision. In India, 80% of successfully re-attached retinas obtained a vision of 6/60 or better.[1]

Prophylaxis

It is important to prevent RD, since 5–15% of retinal re-attachment operations are unsuccessful and only 55–60% eyes with re-attached retinas get good visual outcomes.[3,4] Also RD surgery is more expensive than prophylactic treatment and can be associated with serious complications. Most rhegmatogenous RDs are due to retinal tears that occur from vitreoretinal traction in areas of abnormally firm vitreoretinal adhesions. Exceptions are post-traumatic tears and round holes in areas of lattice degeneration in myopic eyes of young patients. Prophylactic treatment aims to create strong chorioretinal adhesions in areas of retinal tears or areas of strong vitreoretinal traction. Visible lesions that could be considered for prophylactic treatment include:[4,5]

1. Horseshoe tears (high risk of progression to RD without treatment).

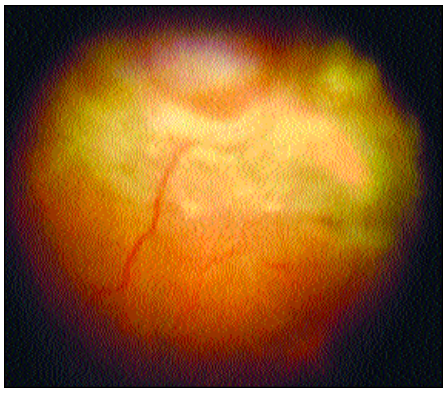

Fig. 3b: Same eye after re-attachment surgery with vitrectomy and silicone oil injection

Photo: Subhadra Jalali

2. Lattice degeneration with or without holes and with or without vitreous traction (risk of progression uncertain).

To 'treat or not to treat' depends on other factors that predispose to a high risk of retinal detachment (Table 1) and on theknown complications of prophylactic treatment. Methods of prophylactic treatment include cryotherapy, laser photocoagulation and, very rarely, prophylactic scleral buckling.

Conclusion

Retinal detachment is a vision threatening condition that requires early surgery. It can be diagnosed best by retinal examination using indirect ophthalmoscopy. Treatment outcomes have improved with modern surgical techniques, but the key to successful re-attachment is early detection and prompt referral by primary eye care workers. More widespread availability of trained human resources and equipment is essential to manage and prevent retinal detachments that can cause unilateral and, not uncommonly, bilateral permanent blindness.

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Retina Picture Quiz: Questions (Figs. 1 to 6)

David Yorston

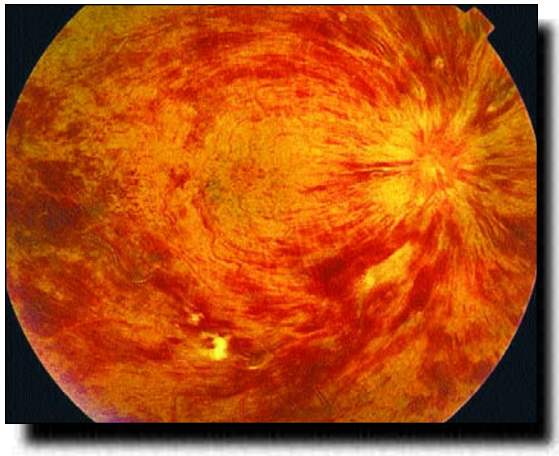

Fig 1: 55 year old man. Open angle glaucoma in left eye. Noticed loss of vision in right eye on waking this morning. VA right eye 2/60.

Fig 2: 23 year old woman. Headache for two months. Worse on waking. VA 6/6 in both eyes.

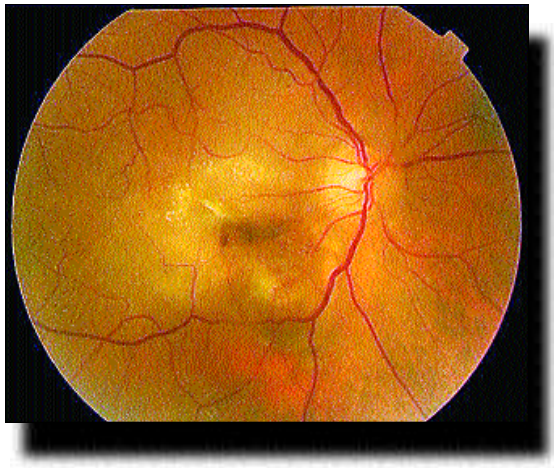

Fig 3: 75 year old man. Noticed distortion of vision in right eye, then loss of central vision. VA right eye 2/60.

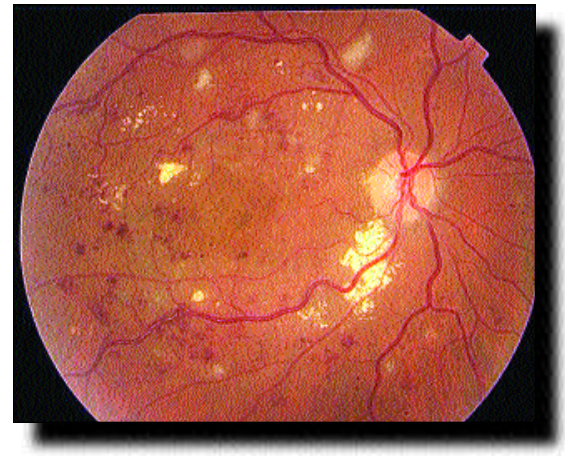

Fig 4: 65 year old man. Gradual loss of vision right eye for one year. Also polydipsia and polyuria. VA right eye 6/18.

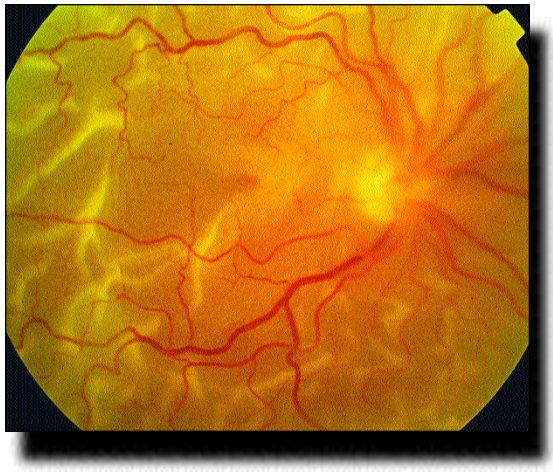

Fig 5: 58 year old woman. Sudden loss of vision in right eye one week ago. Floaters in right eye two weeks previously. VA right eye HM.

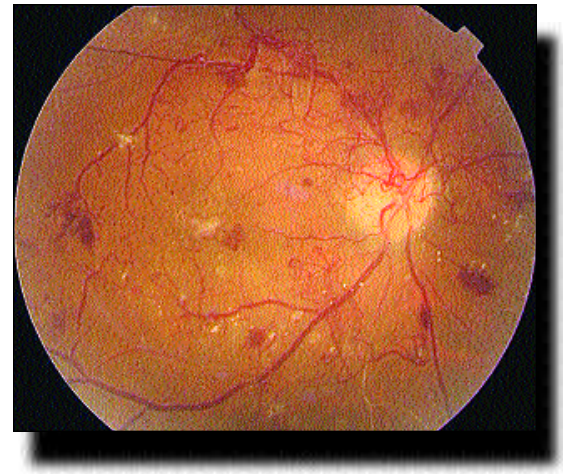

Fig 6: 28 year old woman. Type 1 diabetic for 13 years. VA right eye 6/12.

Retina Picture Quiz: Answers (Figs. 1 to 6)

David Yorston

Fig. 1: Central retinal vein occlusion

The large number of haemorrhages, the white cotton wool spot, and the poor vision all suggest that this is probably an ischaemic CRVO. There is a high risk that this will progress to rubeotic glaucoma within the next three months. If iris new vessels are detected, then pan-retinal laser can prevent secondary glaucoma. A high IOP greatly increases the risk of CRVO, so it is important to treat the glaucoma in the other eye.

Fig. 2: Papilloedema

There is a swollen optic disc. As the vision is normal it is unlikely to be optic neuritis, so the most likely diagnosis is papilloedema. Possible causes include raised blood pressure, and benign intra-cranial hypertension as well as intra-cranial space occupying lesions.

Fig. 3: Age-relatedmacular degeneration

There is a sub-retinal scar (retinal blood vessels pass in front of the paler scar tissue) under the macula. The dark area is due to haemorrhage. Fibrous and vascular tissue has grown from the choroid under the retina at the macula, destroying the photoreceptors at the fovea, and causing irreversible blindness. This is the commonest cause of blindness in Europe and North America.

Fig. 4: Diabetic maculopathy

Diabetic retinopathy may occur before the patient knows he has diabetes. This patient has multiple haemorrhages and cotton-wool spots, due to capillary closure, as well as hard exudates, which indicate leaking capillaries. Laser treatment at this stage reduces the risk of further loss of vision over the next five years.Diabetes is becoming a problem in developing countries and health education programmes must raise awareness of the loss of vision due to diabetes.

Fig. 5: Retinal detachment

The wrinkled surface of the retina, and the loss of the normal red reflex are characteristic of a retinal detachment. The flashes and floaters are caused by a vitreous detachment, which caused the retinal break that led to the retinal detachment. The macula is already detached, but surgery to re-attach the retina will at least restore navigational vision.

Fig. 6: Proliferative diabetic retinopathy

There are active new vessels arising from the optic disc and from the retina. Untreated, there is a high risk of blindness within five years. This can be greatly reduced by urgent pan-retinal laser treatment. Screening for diabetic retinopathy and offering appropriate treatment is essential to reduce loss of vision.

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Technology for VISION 2020

Mould In Optical Instruments

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Mould can damage optical instruments beyond repair within only a few weeks. There is a good deal of information available on the treatment of mould in buildings, because common respiratory problems and allergic reactions can be caused by mould. Knowledge is also available in the field of conservation of books and fine art because of the high cost of mould damage. However, very little information is available on mould in optical instruments and the management of mould is often ignored by equipment manufacturers and users.

Moulds are plant organisms which form cobweb-like branching arms, from which spores project into the air (see Glossary). Moulds are very common and very widely dispersed. There are 250,000 species of mould, many of which can damage optical instruments. Among the moulds commonly found in instruments are members of *aspergillus, penicillium* and *trichoderma* species.

Conditions of Growth

Although moulds grow in almost every environmental condition on the planet, most prefer temperatures of 20–30°C and relative humidity in excess of 90%. Moulds can germinate from nutrients stored in the spore, but, for growth, they need an additional source of nutrients such as protein, carbohydrate and cellulose. The mould network produces a microclimate close to the supporting surface which can trap dust particles containing nutrients, and can maintain the conditions of temperature and humidity needed for growth. In conditions of high humidity and moisture, many of the nutrients come directly from water vapour in the air.

According to the International Organisation for Standardisation,[1] moulds cannot grow on the glass optical surfaces of lenses, prisms, mirrors or filters without access to other sources of nutrient – such as textile fibres and dust, grease and fingerprints, or varnish. This usually comes from the edges of the optical surface, from contamination left in the joint between the lens and the mounting cell during cleaning, or from varnish or other material in the mounting cell. Figure 1 shows the typical cobweb growth of a mould mycelium from the edge to the centre of a glass surface.

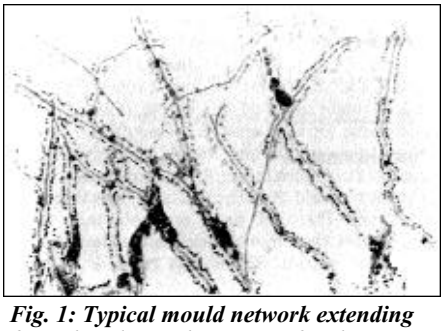

Fig. 1: Typical mould network extending from the edge to the centre of a glass surface (from Kaneko[2])

Mould can grow very quickly. It takes only a few days for mould spores to germinate, and only a few weeks to extend hyphae and grow extensively. Many regions of Africa, South-East Asia and Latin America provide ideal conditions of temperature and humidity for rapid mould growth. Even so, within these regions, the individual risk of damage to instruments varies widely. Some optical instruments are kept in operating rooms, clinics or laboratories which are continually air conditioned and so the humidity never reaches the level needed for mould growth, while others are not. Some instruments have internal fungicidal protection, while others do not. Each instrument must be individually assessed for risk, based on its environment and on the importance of mould damage to it.

In countries where the conditions for mould growth are optimum, mould is often

seen on the outside surfaces of optical instruments such as the eyepiece and objective lens surfaces. Mould on internal surfaces may be seen through the instrument if it is close to a focal plane, but usually it is only evident by reduced light transmission or reduced image quality caused by scattering or absorption of light in the mould mycelia. If there is a rapid loss of light transmission or image quality, the possibility of mould should always be considered.

Mould can also damage instrument electronics through short circuits and corrosion, but this can usually be repaired. Damage to optical surfaces is rarely cost effective to repair. A growing mould mycelium produces organic acids which etch the glass surface with minute grooves, leaving behind a print of the mould network (Figure 2) and, as optical components cannot be resurfaced economically, the instrument is then destroyed.

Some glass types are attacked by mould much more readily than others.

Anti-reflection coatings seem to have little effect on the susceptibility of glass surfaces to mould attack, and these coatings are etched along with the glass substrate.

Inhibition of Mould Growth

Two methods are commonly used to inhibit mould growth in instruments.

1. **Environmental control**. Some military optical systems are filled with a dry gas and then sealed, but this method is not used in commercial instruments. Storing the instrument at a relative humidity of less than 65% will prevent the growth of most moulds. This can be achieved either by storing in an air conditioned room, or in a sealed container (a sealed plastic bag may be enough) with a drying agent. If a drying agent is used, it is essential to use one which changes colour when it is saturated and dry or replace it when this becomes necessary.
2. **Fungicides**. Fungicides have been added to instrument varnishes, waxes and cements, to surface coatings of lenses, and to replaceable strips and pellets. The fungicide must provide a concentration throughout the instrument sufficient to prevent mould growth, but, at the same time, not sufficient to condense on optical surfaces or corrode components. In Australia, defence optical instruments have had the fungicide, ethyl mercury thiosalicylate incorporated into paints, cements and waxes to inhibit mould growth. Some optical instruments have also used radioactive or fungicidal surface coatings on optical components, but this is not common and, according to the International Organisation for Standardisation,[1] is not effective. The useful life of a fungicide is necessarily limited by evaporation of the active ingredients unless the instrument is completely sealed, and so attempts to incorporate a permanent fungicide have largely been replaced by the use of replaceable fungicidal pellets. These can be obtained from many instrument manufacturers, and have a useful life of about three years.

Cleaning Mould Contamination

Moulds do not have roots attached to the optical surfaces, and so can be wiped away easily. A mixture of alcohol and ether is often used to clean optical surfaces. Care must be taken in choosing the cleaning agent, as many solvents such as acetone may damage antireflection coatings, paint work and plastics.

Ordinary facial tissues should not be used to clean optics. The paper often contains grit particles that scratch, and lint that is electrostatically charged and is hard to remove. A commercial lens cloth, or a cotton cloth that has been washed several times, should be used. Cotton buds are suitable and can reach some of the internal optical surfaces that are difficult to clean.

It is hard to remove mould spores completely once they have become established, and optical instruments that have been affected by mould should be cleaned regularly to prevent regrowth.

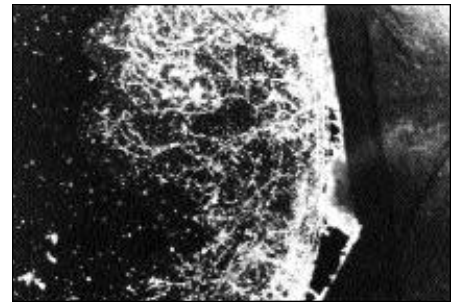

Fig. 2: Grooves etched into a glass surface by organic acids from a mould mycelium (from Kaneko[2])

Rules for Managing Mould in Optical Instruments:

1. Do not wait until mould appears on the outside surfaces of an instrument. By then it may be too late.
2. Inhibit mould growth, if possible by installing a fungicide in the instrument and changing it at recommended intervals, and by storing the instrument in a relative humidity of less than 65%.
3. Inspect the instrument regularly, and clean the accessible surfaces with a disinfectant.

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GLOSSARY

Fungus:	A sub-division of the *Thallophyta* division of the plant kingdom. Fungi are simply organised plants, either single celled or made of cellular filaments, and lacking in green colouring matter (chlorophyll).
Fungicide:	Any substance which destroys fungus.
Germination:	The sprouting or budding of a mould; production of the initial shoot of a hypha.
Hypha:	A filament of a fungus, composed of one or more cylindrical cells. Hyphae increase in length by growth at the tips and give rise to new hyphae by lateral branching.
Mould:	Any superficial growth of a fungus mycelium.
Mycelium:	The collective term for the mass of hyphae that constitutes the growing part of a fungus.
Nutrient:	Any substance that provides nourishment to the mould.
Spore:	Single – or multi-celled reproductive body that becomes detached from the mould and gives rise to a new individual. Spores are usually microscopic, and are produced in a variety of ways. They are often produced in enormous numbers and are distributed widely, serving for a very rapid increase in the population of the mould. Spores may be able to survive over long periods that are unfavourable to growth.

The Ocular Application of Povidone-Iodine

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The use of antiseptic agents to prevent blindness was not a self-evident development. It evolved over a number of years to the point that one antiseptic agent – povidone-iodine – is now used throughout the world, every day, to prevent blindness.

The possible effect of iodine on the eye was first appreciated in 1951 when a reduction in ocular flora was reported following the application of iodine solution to the skin.[1] Iodophors were reported to reduce skin flora around the eye in 1970,[2] and only later was the specific combination of povidone and iodine utilised for direct ophthalmic use.

Why was povidone-iodine chosen as an antimicrobial agent?

- *In the appropriate concentration*, it is not toxic to the eye as are other iodine bearing compounds
- It has a very broad antimicrobial spectrum, including bacteria, viruses, and fungi, given enough contact time *in vitro*
- Resistance by bacteria is rare
- The medication turns the eye brown for a few minutes proving that it has been applied
- It is widely available as a solution or powder; and so it is available throughout the world in some form
- Finally, especially important for use in developing areas, it is not expensive.

Pre-operative Use

Ophthalmic surgeons have come to appreciate the possibility of reducing post-operative infections, including endophthalmitis, by effective pre-operative preparation. However, even by the 1980s, pre-operative preparation of the eye had not been scientifically validated. A series of investigations were then conducted to evaluate various aspects of this preparation – to reduce the bacterial flora of the eye. By far the most effective measure was to place a drop of 5% povidone-iodine ophthalmic solution on the eye before surgery. Bacterial colonies were reduced by 91% and species by 50%. This decrease was significantly better than in the control group. This 1984 study was the first controlled trial of the ophthalmic use of povidone-iodine conducted.[3]

We then investigated the possible benefit of using povidone-iodine 5% solution placed on the eye just before the operation, in addition to a combination ophthalmic antibiotic solution (neomycin, polymyxin B, and gramicidin) used as an outpatient medication for three days before surgery.[4]

The combination reduced the mean number of bacterial colonies per eye from 1745 to 2 after use of both medications ($p < 0.01$). For species, the corresponding reduction was from 2.4 to 0.2 after both medications were used ($p < 0.01$). After use of both agents, 83% of eyes became sterile. This effect was the greatest reduction of bacteria ever produced in a controlled study to that time. A subsequent study showed that the synergistic effect of the antibiotic used three days pre-operatively, combined with povidone-iodine applied on the operating table before surgery, proved better than using povidone-iodine alone as an outpatient and before surgery.[5]

Is this technique effective in preventing endophthalmitis?

A 1991 study in New York of 8000 patients undergoing cataract surgery showed that the use of povidone-iodine solution placed on the eye just before surgery reduced the endophthalmitis rate from 0.24% to 0.06% ($p < 0.03$).[6] The resulting potential reduction in blindness in the United States alone can be estimated, assuming 1.8 million cataract surgeries per year and a 50% frequency of legal blindness after endophthalmitis. This would mean about 1600 American patients annually who would have been blind in the operated eye were it not for the pre-operative application of povidone-iodine. The actual figure would be much larger if one were to include all intraocular surgeries and extend the patient base to the entire world.

Post-operative Use

We then considered the effect that povidone-iodine would have if used after ocular surgery. The effect of 2.5% povidone-iodine solution placed on the eye immediately at the completion of the operation was compared with the similar use of a combination antibiotic (neomycin, polymyxin B, and gramicidin).[7] At 24 hours after surgery, povidone-iodine significantly reduced the colony forming units ($p=0.035$), but the combination of neomycin, polymyxin B, and gramicidin did not ($p=0.17$). The colony counts increased in the first 24 hours in the antibiotic treated eyes ($p=0.013$), but not in the povidone-iodine treated eyes. At 24 hours, the povidone-iodine treated eyes had a lower species count ($p=0.034$) than the antibiotic treated eyes.

Subsequently, patients were studied who used povidone-iodine 2.5% or 1.25% solution compared with a similar group using the antibiotic combination eyedrop three times a day for a week after ophthalmic surgery.[8] No ocular infections occurred. While the species counts increased in both groups over the post-operative week, they increased less in the povidone-iodine treated eyes ($p = 0.011$) and were lower than the untreated control group ($p=0.01$). However, in the antibiotic treated group the species count was not less than the controls ($p=0.29$).

Prevention of Ophthalmia Neonatorum

It has been estimated that ophthalmia neonatorum blinds more than 10,000 babies per year throughout the world. While prophylaxis using silver nitrate or other agents can be effective, it often is not utilised in areas where the prevalence of ophthalmia neonatorum is highest, primarily due to expense. Developing countries often cannot afford the cost of neonatal prophylaxis. Since povidone-iodine solutions can be prepared locally from stock solutions or powders, it is very inexpensive (about US$0.10 for a 5 ml bottle) and available worldwide.

A pilot study in California showed that povidone-iodine significantly reduced the bacterial ocular flora in normal neonates.[9] In 1995, a multi-year trial of povidone-iodine 2.5 % ophthalmic solution, used for ophthalmia neonatorum prophylaxis in more than 3000 neonates in Kenya was reported.[10] The study compared the effectiveness of povidone-iodine with silver nitrate and erythromycin. In Kenya, where prophylaxis had not been generally utilised because of expense, ophthalmia neonatorum occurred in as many as 23.2% of newborns. The trial found povidone-iodine to be more effective than the other two agents and was less toxic. Based on this report, povidone-iodine is now being used increasingly for neonatal prophylaxis in much of the world.

Treatment of Infections

All the studies mentioned have been concerned with prevention.

Would povidone-iodine be effective in treating an ongoing ocular infection?

There had never been a controlled randomised trial of the use of povidone-iodine to treat ocular infections. While conjunctivitis often is innocuous in the developed world, in developing areas, the infection can lead to corneal scarring and blindness from a number of causes, including a lack of appropriate antibiotics, malnutrition, vitamin A deficiency, trachoma, rubeola, and trauma.

To investigate the use of povidone-iodine in the treatment of paediatric conjunctivitis, 459 children were studied in Manila, Philippines.[11] This trial is believed to be the largest controlled investigation of conjunctivitis treatment ever reported. Povidone-iodine 1.25% ophthalmic solution, applied four times daily, was compared with the effect of an antibiotic combination (neomycin-polymyxin-B-gramicidin). As determined by 'time to cure', povidone-iodine was as effective in the treatment of bacterial conjunctivitis, marginally more effective against chlamydial conjunctivitis ($p = 0.057$), but equally ineffective against viral conjunctivitis. Povidone-iodine 1.25% ophthalmic solution can, therefore, be used to treat bacterial and chlamydial conjunctivitis, especially in emerging countries where topical antibiotics are unavailable or costly.

New investigations are underway to evaluate the effectiveness of povidone-iodine to treat corneal infections. These studies have the potential of decreasing the frequency of blindness from corneal infections and subsequent corneal scarring – the most frequent cause of preventable paediatric blindness in developing countries.

Practical Application

Povidone-iodine is readily available worldwide, either as a powder or as a 10% solution. Depending on the type of application, for ophthalmic use, the solution must be diluted to the appropriate strength. The diluent may be a balanced salt solution or other appropriate diluent.

It is important to avoid the detergent version of povidone-iodine generally used as a skin antiseptic, since this solution will adversely affect the cornea.

In summary, povidone-iodine ophthalmic solution has been proven effective before (5% solution) and after ocular surgery (1.25%), at birth (2.5%), and for some forms of conjunctivitis (1.25%). Investigations of its use in treating other types of ophthalmic infections are continuing. The use of povidone-iodine in ophthalmic practice continues to reduce the incidence of blindness in children and adults throughout the world.

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(See also page 32)

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