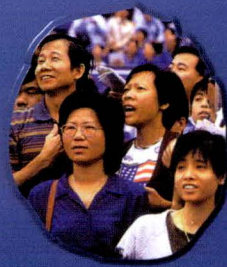


**The Optic Nerve Head – Office Interpretation**

**Glucose Intolerance and Vogt-Koyanagi-Harada Syndrome**

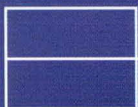
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# The Optic Nerve Head — Office Interpretation

GR Douglas

Professor Emeritus, Victoria, British Columbia, Canada

## Introduction

Examination of the optic nerve head (ONH) is one of the most neglected skills in ophthalmology and general medicine, mainly due to time constraints in the clinic and poor acquaintance with up to date knowledge regarding this familiar bit of anatomy. Epidemiology has taught us that ONH assessment is superior to tonometry and perimetry for diagnosis and follow-up of chronic glaucoma when all 3 methods are used individually. That is not to say that any of these techniques should be eliminated, rather to emphasise the importance of a detailed but efficient ONH examination. The purpose of this article is to present an examination paradigm to clinicians so that early pathology, as well as progressive changes, may be recognised using available tools and requiring only slightly more time.

Since most practitioners in the world will not have reasonable access to the new ONH analysers, it remains the responsibility of each of us to become acquainted with simple, cost-effective, diagnostic methods that are readily available. Even with the inevitable decrease in price of these machines over time, costs will remain too high for all but the most affluent situations. With practice, the concepts and suggestions found in this article can be applied with minimum equipment and time expenditure.

As mentioned, if only a single examination modality is used, most glaucomatologists would choose ONH assessment rather than the other 2 options. Intraocular pressure (IOP) monitoring and perimetry both have a role when available. However, in many settings, this is not possible — thus the need for an article such as this. Tonometry and ophthalmoscopy are often the only practical means of detecting and monitoring chronic glaucoma and, when properly used, can be most efficient for the clinic setting.

The challenge presented here is to acquire and apply new understanding and correlations amongst various parameters of the ONH. This increase in diagnostic acuity will help clinical knowledge to evolve beyond the simplistic and inadequate cup/disc (C/D) ratio approach, which often prevails today.

## Methods of Examination

### Direct Ophthalmoscopy

Ophthalmoscopy remains the instrument of choice in most countries, if for no other reason than economy. With this instrument, we can vary the illumination, aperture size, image shape, and access red-free light. For our purposes, the small light is used for undilated pupils and the large one for dilated pupils. These, plus the red-free light that enhances our view of the retinal nerve fibre layer (RNFL) are the most useful options. An overly bright light may be

disadvantageous both for the patient (photosensitivity) and the examiner (miosis), therefore, this should be adjusted for optimal viewing. The ophthalmoscope offers portability, magnification, and economy but is marred by a narrow field of view and a marginal appreciation of any topographical change in the neuroretinal rim (NRR).

### Indirect Ophthalmoscopy

Indirect ophthalmoscopy offers a wide-angle stereoscopic image to the examiner but, depending on the condensing lens used, magnification may not be adequate to see sufficient detail on the ONH. The main problems with this apparatus are magnification, expense, and the need to dilate all but a few eyes.

### Condensing Lens/Slit Lamp

A 60 to 90 D lens in front of the eye, used with the slit lamp, also gives good quality stereoscopic images of the ONH. This method requires dilation and, of course, an additional lens.

### Contact Lens

Examination of the ONH with the central lens of an indirect Goldmann-style gonioscope provides magnification, stereopsis and, when coupled with the scale on the adjustable vertical light beam of a slit lamp, a means of quantitating the size of the ONH and other fundal findings. Although not absolute units, they may be noted for future changes of cup and NRR width if the same equipment is used. This method requires dilation, a slit lamp, and the lens.

### Summary

If ease of use, economy, and availability are important factors, the ophthalmoscope is the clear winner for method. A Goldmann-style gonioscope, with its central lens, also has a definite advantage, especially in Asia where angle closure glaucoma is so prevalent. In larger or better-equipped



clinics, alternatives may be present and should be used to supplement the ophthalmoscope.

## Optic Nerve Head Parameters



### Optic Nerve Head Size

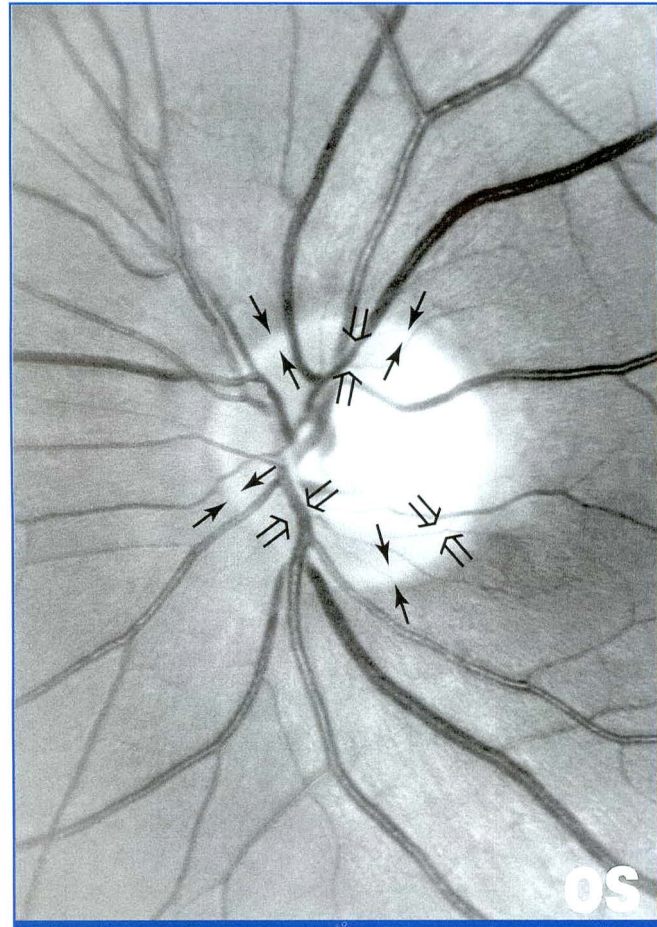
An important but often confusing point is where the outer limits of the disc are located. The scleral rim is commonly seen as a partial or total white circle peripheral to the normally pink NRR. Outside this rim is the peripapillary zone and inside is the ONH (Figure 1). It is now well established that the ONH (disc) area can vary by up to 7-fold amongst individuals.<sup>1</sup> Due to this factor, if the C/D ratio is used and the disc area is not properly interpreted, an examiner

may easily over- or under-interpret the status of the NRR. Since the neuroretinal rim area (NRA) appears to be the most important clinical parameter for clinicians when looking for and monitoring glaucoma, estimation of the disc size becomes an essential part of ONH examination (Figures 1 and 2).<sup>2-4</sup> Of all the parameters of the ONH, NRA appears to be most highly correlated with damage.<sup>5</sup> Even on the Heidelberg Retinal Tomograph (HRT), NRA is second in importance only to a computer-derived term called cup-shape measure.<sup>6</sup> It has also been determined that the size of the cup is directly related to that of the disc.<sup>3</sup> Therefore, small discs with small cups may hide damage whereas large discs with large cups may cause concern and lead to initiation of unnecessary treatment.

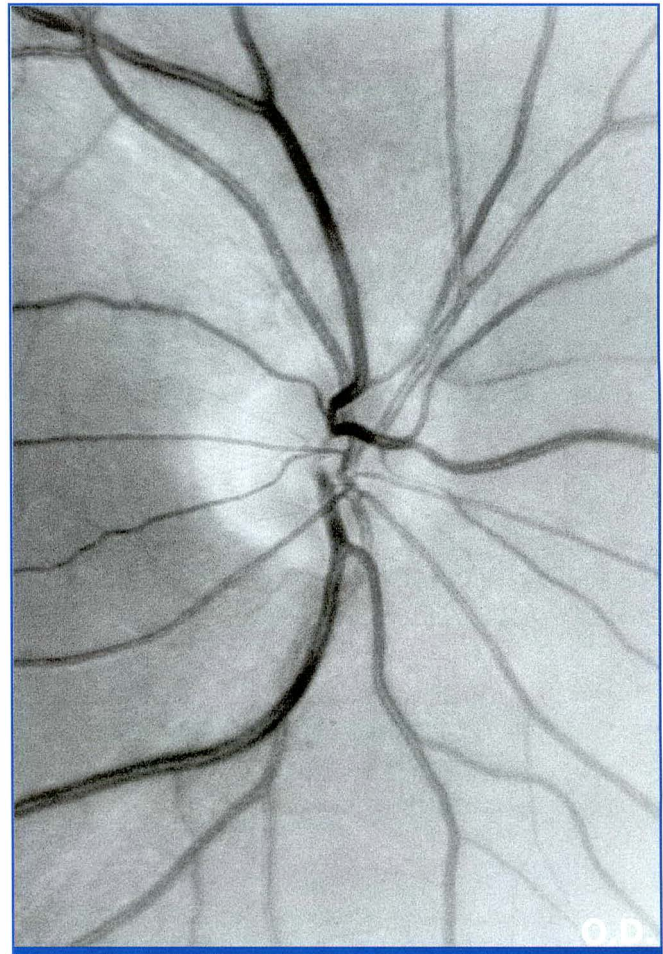
Having shown the importance of disc size, how is it estimated? Clinicians may misinterpret the size of an ONH because of the optics of the eye. Littmann provided a formula by which we can compensate for the effects of optics,<sup>7</sup> although the next section helps to retain this article's concept of simplicity. The image on many ophthalmoscopes subtends an angle of approximately 5°, which is also the size of the 'average' disc. To confirm the size of a particular ophthalmoscopic image, shine the image onto a wall from 1 meter away.<sup>8</sup> The diameter should be approximately 85 to 90 mm. Now, by observing the relative size between this image positioned beside the ONH and that of the ONH disc, the size can be estimated. A large disc will be much larger than the image while a small



**Figure 1.** Normal optic nerve head — scleral rim (arrows), neuroretinal rim thickness (open arrows), retinal nerve fibre layer seen as radiating lines against the darker choroid, and zone alpha is seen temporally.

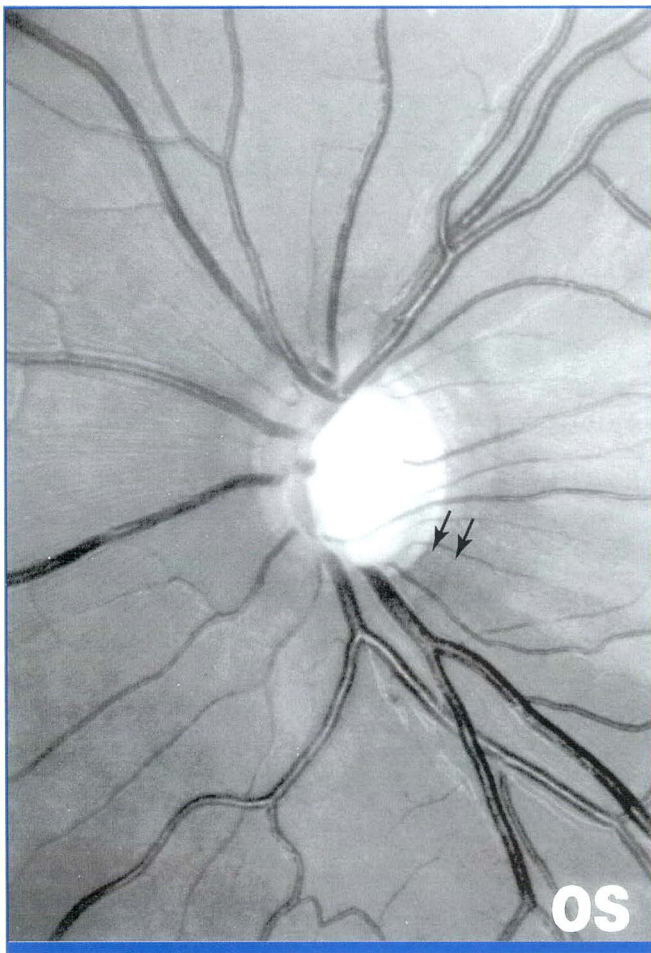


**Figure 2.** Small optic nerve head — compared with Figure 1 for relative size. The blood vessels appear larger relative to the disc.

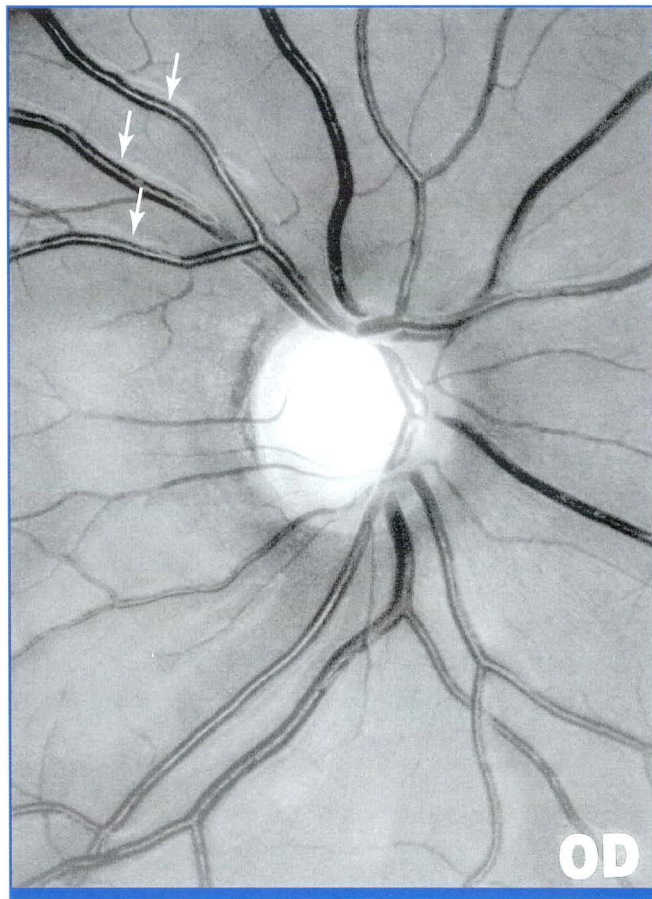




**Figure 3.** Pallor versus neuroretinal rim change — the inferior rim is damaged as shown by a change in blood vessel direction close to the scleral rim — if only pallor had been used to judge the cup size, the true size would have been missed. Note also the retinal nerve fibre layer between 4.30 and 5.30 o'clock on the retina as a darker area (arrows).



**Figure 4.** Generalised enlargement and notch formation — blood vessels change direction directly over the scleral rim from 5 to 6 o'clock, indicating an absolute loss of neuroretinal rim. There is also a narrowing of the superior neuroretinal rim. Note the loss of retinal nerve fibre layer superiorly and inferiorly. Reflections of light, parallel to some blood vessels in these 2 areas, show the inner limiting membrane which is now draped over them rather than being flat on a healthy retinal nerve fibre layer.



disc will be much smaller. Recorded notes of discs can then be more meaningful, as in small, average, or large, especially if the C/D ratio is also noted. Using this method, disc size asymmetry can also be discovered to explain cup asymmetry. This estimating technique is useful for all eyes except those in which extreme ametropia exists.

### Neuroretinal Rim

As for the disc, rim margins may be difficult to identify. Colour change is often used to distinguish between the cup and the rim but this works only for normal ONHs, where contours (topography) and colour are often coincident. In looking for disease, it is important not to utilise a criterion that is

typical only for the normal state. A better method is to observe the change in direction of blood vessels as they cross the NRR (Figures 1 and 3). This allows the clinician to detect a change in NRR topography when 'atrophy' is not yet apparent. In more obvious cases, the 'laminar dots' of the lamina cribrosa or extension of the central cup pallor may help as they are seen to be closer to the disc margin in one area rather than another (NRR thinning).

### Relative Thickness

Relative thickness of the NRR varies in a fairly predictable pattern amongst healthy individuals.<sup>9</sup> Due to the macular fibres, the inferior rim is the widest, followed by the

superior rim. These are followed by the nasal and, finally, the temporal rim, which is the thinnest (Figure 1). In subtle cases, for example, even equality of the rim widths superiorly and inferiorly may indicate pathology. As already mentioned, in glaucoma, the NRR is the most important part of the ONH. Clinicians would gain more information looking at the NRR than what is not there — the cup! Although it is not possible to routinely estimate the rim area, it can be observed and the relative thickness or width of it recorded. Generalised or localised thinning should be noted and correlated with perimetric findings whenever possible (Figures 3 and 4). If a correlation cannot be found, other aetiologies for abnormalities



(retinal, neurological) must be considered. The crucial point in NRR examination is to concentrate on the topography of the NRR and disc and not the central pale area.

Careful observers are already aware of slopes of the NRR rather than the sharply-demarcated rim edges implied above. These are more commonly seen in highly myopic discs but, a gradual slope away from the neuroretinal surface can be found in many eyes, both normal and diseased. A problem arises in trying to identify any margin under these circumstances. This is where stereoscopy (contact lens) can be of help. The term 'saucerisation' has been coined for this situation, to cover an inability to quantify or to adequately describe this appearance. In the absence of a contact lens or a condensing lens, parallax may be used to estimate relative depths, that is,

horizontal or vertical movements within the pupil observing the relative 'with and against' movements of the retina and cup depths.

### Pallor

Pallor of the NRR is difficult to identify until it is well-established, is asymmetrical, or is found only in a localised segment of the rim. There have been attempts to quantitate pallor but clinicians must use their judgement when making this finding. However, segmental pallor is often found following branch vascular occlusions or chorioretinal breaks following trauma, both of which can be the cause of nerve fibre bundle defects typical of glaucoma. This is not to be confused with glaucomatous loss of rim in which the pale deep tissue of the ONH is now exposed.

### Translucency

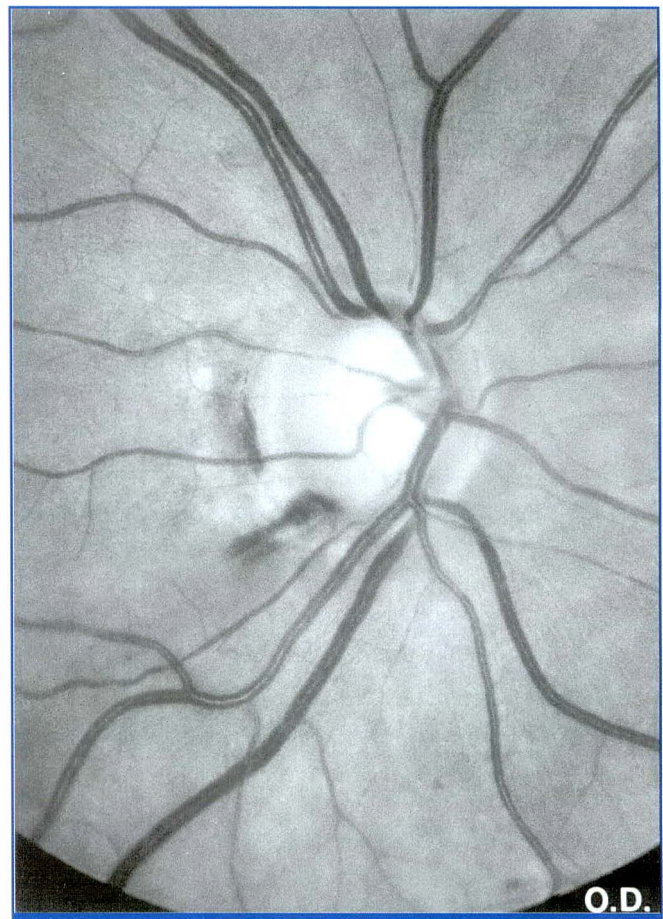
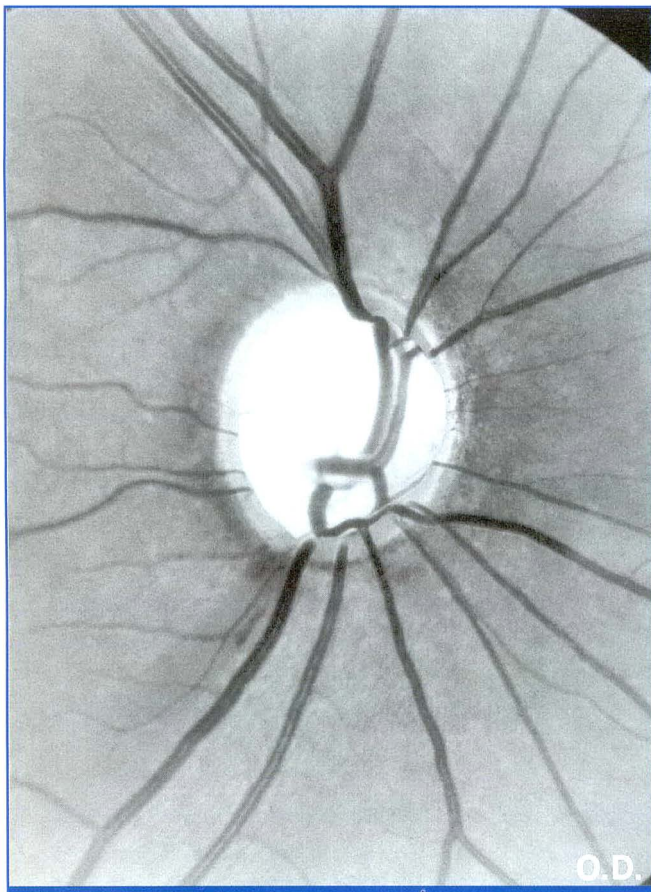
Translucency of the NRR has also been described in glaucoma. This term is often used to describe transparent tissue below blood vessels that has not been displaced backward into a notch or thinning of the rim. Translucency may be suspected but its validity lies mostly in correlation with perimetric findings.

### Haemorrhages

Haemorrhages (Figure 5) are most commonly found crossing the NRR (splinter) but they may also be found in the rim or disc substance (splinter or diffuse). They are elusive and are rarely found unless sought. First recognised by Donders in the 1860s, they were 'lost' by generations of ophthalmologists until rediscovered by Drance and Begg in 1970.<sup>10</sup> Their typical

**Figure 5.** Optic nerve head haemorrhages — this large disc has a large, pathological cup plus a small haemorrhage on the neuroretinal rim at 7 o'clock. A retinal nerve fibre layer defect is also present peripheral to the rim.

**Figure 6.** Optic nerve head haemorrhage — a more obvious haemorrhage than that in Figure 5 is seen at 7:30 on this disc.



flame shape is easily recognised, although many have been missed because of their resemblance to small blood vessels. They may also be large and have to be distinguished from vascular events in the retina. They resorb within 6 to 8 weeks and, in a significant number of patients, leave a perimetric nerve fibre bundle defect or a retinal nerve fibre layer defect.<sup>11</sup> For this reason, finding one or even multiple haemorrhages is considered by many to represent glaucomatous progression.<sup>11</sup> The ophthalmoscope is ideal for finding a haemorrhage because of its magnification. It is now known that, although more commonly found in low tension glaucoma, haemorrhages may be found in any chronic form of the disease.<sup>12</sup>

### Parameters Outside the Optic Nerve Head



#### Chorioretinal Atrophy

Chorioretinal atrophy has now been firmly associated with changes in the ONH as well as age and the refractive state of the eye. Jonas et al have written extensively on this finding, calling the outer area zone alpha (irregular pigmentation derived from the retinal pigment cells) and the inner area zone beta (choroidal vessels and sclera).<sup>13</sup> Zone alpha is often seen in healthy eyes, whereas zone beta is correlated with glaucoma. Studies have shown that localised atrophy may be associated with localised changes at the NRR — a good clue when clinicians are concerned about an area of thinning adjacent to the atrophy. This can also be found in older, sclerotic ONHs and in high myopia separate from any glaucomatous process. The main value is found in cases of localised atrophy in eyes with a suspicious notch or loss of NRR. There are also some theories on why such findings occur in glaucoma. Observations made by Rader et al suggest that vasoconstriction of retinal arterioles as they cross the NRR

may indicate a potential aetiology for glaucomatous chorioretinal atrophy.<sup>14</sup>

#### Retinal Nerve Fibre Layer Defects

Retinal nerve fibre layer defects (Figures 1, 3, 4, and 5) have been recognised for many years but have only been associated with glaucoma since the early 1980s.<sup>15,16</sup> Red-free light is useful for detection and dilation aids the clinician regardless of whether the ophthalmoscope or slit lamp is used. Defects may be discrete or diffuse but the latter are much harder to find. Figures 1 and 6 illustrate the normal pattern in which the fibres associated with superior and inferior vascular bundles meld into the less obvious nasal and temporal quadrants. As with haemorrhages, the observer must appreciate the normal pattern before expecting to 'see' the abnormal. Figures 3, 4, and 5 demonstrate localised defects, all of which are associated with localised NRR defects. When recognising these RNFL defects for the first time they are always seen because of associated notches. With practice, clinicians will be able to recognise some of the RNFL abnormalities first and either verify the presence of a subtle NRR change or recognise glaucomatous pathology prior to recognisable perimetric alteration.

### Developing the Clinical Paradigm



#### Normal Optic Nerve Head

A normal ONH is one in which no changes have taken place over time, that is it remains static. It may be small, average, or large in size and should have rims with thickness as described above. Age and changes associated with extreme refractive errors may be present and should be differentiated from changes associated with glaucoma. 'Normal' for an individual may also include abnormalities such as optic pit(s), coloboma, or a tilted disc, all of which may cause visual field changes consistent with glaucoma.

One method of approach for examination of the ONH is as follows:

- estimation of disc size
- rim — thickness in each quadrant, colour (↓↑pallor/translucency), and an *active* search for haemorrhage(s)
- peripapillary halo pattern
- RNFL defects
- comparison with the other eye
- correlation with visual fields (where possible).

#### Abnormal Optic Nerve Head

An abnormal ONH is one that has changed over time. However, clinicians often lack the all-important earlier photograph or drawing that would indicate the starting point of the eye prior to the onset of the glaucoma. Clear pathological ONHs are not difficult to identify but a large number are 'borderline' and cannot be distinguished from a normal variant. For example, a large disc (with its large cup) may be a concern until disc size is estimated and the rim thickness pattern is found to be normal. A localised thinning of a NRR is highly suspicious, but the presence of a haemorrhage or an adjacent peripapillary halo/RNFL abnormality may help in judging whether or not it is significant. Clinicians can develop stronger opinions about ONHs that have *recognisable* abnormalities prior to development of visual field defects.

At least 4 patterns of ONH abnormality have been found — focal ischaemic, myopic glaucomatous, senile sclerotic, and generalised enlargement.<sup>17</sup> These examples are 'pure' in their appearance but there are many more hybrids of one or more of these categories. Focal ischaemic ONH abnormality has a localised notch whereas myopic glaucomatous ONH is defined by a tilted, cupped disc, temporal crescent, and deficiencies of the superior and inferior rims in a myope. Senile sclerotic ONH has a pale disc, a saucerised cup, and a large halo in an older person.





Patients with generalised enlargement have large, deep cups without peripapillary halo formation.

### Documentation of the Optic Nerve Head

Documentation of the ONH is extremely important, whether the patient has suspected or established glaucoma. Change over time is the hallmark of a progressive disease such as glaucoma, therefore, a baseline must be established for future reference. Many clinicians will write the disc size and the cup/disc ratio on a chart but a freehand drawing is preferred. If at all possible, photography will provide the best record. Often, 2 slides held side-by-side will provide stereopsis, which is the next step of documentation. These can be viewed using 2 single slide viewers hinged together with tape. The more thorough the documentation, the smaller the increment of change at the ONH that can be recognised.

### Conclusion

This article is intended for those ophthalmologists who do not have access to expensive ONH analysis equipment and yet have the responsibility of caring for populations who are at risk of developing chronic glaucoma. Using simple tools and some coordinated knowledge it is possible for reasonable examination of the ONH, even outside of the clinic situation. A paradigm has been presented that should encourage all those interested in the detection and follow-up of patients with chronic glaucoma to do so with greater assurance that these diseases are neither being overlooked nor

overtreated. A 'clinical' study to identify the 4 ONH patterns mentioned above has been reported by some of the authors mentioned. It was encouraging to see that up to 79% were correctly classified.<sup>18</sup> For those who master these guidelines and who, without perimetric backup, can still diagnose chronic glaucoma — congratulations! We can never be *absolutely* certain but we can do better than we are at present.

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# Glucose Intolerance and Vogt-Koyanagi-Harada Syndrome

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 Medical College Hospital, Chandigarh, India



**A** 45-year-old Indian female presented with headache, dimness of vision of both eyes, and alopecia. Systemic and ocular investigations revealed her to have Vogt-Koyanagi-Harada syndrome with glucose intolerance that improved on receiving systemic corticoids.

## Case Report

A 45-year-old Indian female presented with headache and neck stiffness for the previous 3 months, diminution of vision both eyes for the previous 2 months, and hair loss (Figure 1) of 1.5 months duration. The headache was associated with neck stiffness

but was not associated with fever or any kind of motor or sensory weakness. Visual loss was progressive, particularly in the left eye. She complained of progressive scalp sensitivity and loss of scalp hair. There was no history of deafness, tinnitus, vertigo, ocular trauma, or eye surgery.

At clinical examination, the right eye had a visual acuity of 6/36, correctable with pin hole to 6/18. The left eye had a visual acuity of counting fingers at 1 m that did not improve with pin hole. Slit lamp examination of both eyes showed no keratic precipitates, although grade I flare and Grade II cells were present in the anterior chamber. There were 2+ vitreous

cells in both eyes. The intraocular pressure was 12 mm Hg in both eyes and no afferent pupillary defect was detected in the left eye. Fundus examination showed marked disk oedema in the left eye and mild disk oedema in the right eye, with multiple confluent patches of hypopigmentation at the retinal pigment epithelium level in both eyes (Figures 2 and 3) extending to the equator. The hypopigmented lesions in the left eye extended to the inferonasal macula. No evidence of exudative retinal detachment was seen and there were no retinal haemorrhages, vasculitis, or exudates.

Fundus fluorescein angiography (FFA) revealed numerous hypofluorescent spots in early frames becoming hyperfluorescent in later frames. The blood vessels on the disc leaked with marked hyperfluorescence in the left eye compared with the right eye (Figures 4 and 5). B mode ultrasonography showed retinochoroidal thickening ranging from 1.6 to 2.0 mm in both eyes with no evidence of retinal detachment or posterior scleritis. There were no auditory symptoms and pure tone audiometry was normal. Laboratory investigations included complete haemogram, skin testing with purified protein derivative, chest X-ray, syphilis serology, and lupus erythematosus cell phenomenon. Random blood sugar (RBS) was 14.7 mmol/L (normal range, <11.1 mmol/L) at admission to hospital, so a standard oral glucose tolerance test (OGTT) was performed and the patient was found to have impaired glucose tolerance — 5.40 mmol/L at 0 minutes, 11.06 mmol/L at 60 minutes, and 8.00 mmol/L at 120 minutes (normal range, <7.8 mmol/L). Punch biopsy of the scalp from the area of alopecia showed perifollicular lymphocytosis with focal areas of perivascular lymphocytes.

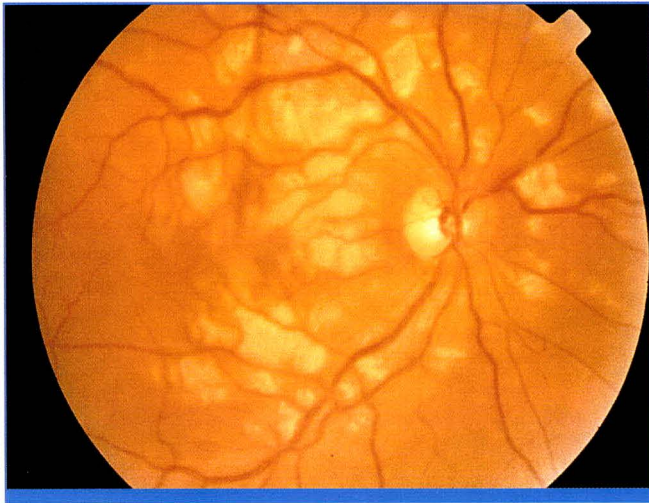
A diagnosis of Vogt-Koyanagi-Harada (VKH) syndrome<sup>2</sup> was made and the patient started intravenous methylprednisolone

**Figure 1.** Alopecia areata in a patient with Vogt-Koyanagi-Harada syndrome.

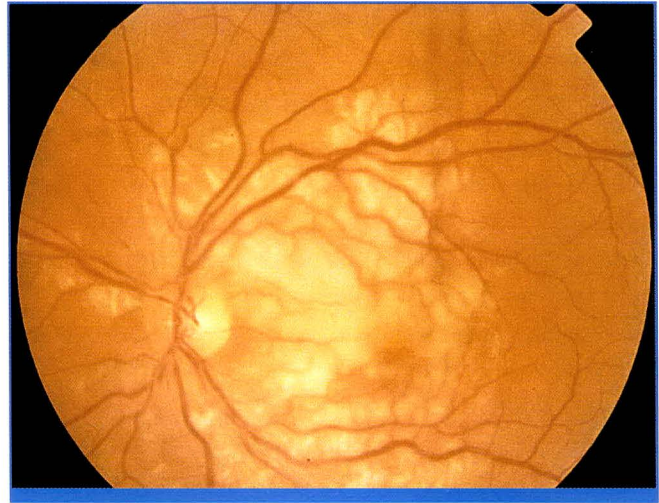




**Figure 2.** Fundus photograph of the right eye showing disk oedema and multiple subretinal confluent patches of hypopigmentation.



**Figure 3.** Fundus photograph of the left eye showing disk oedema and multiple subretinal confluent patches of hypopigmentation.



1 g/100 cc for 30 minutes for 3 days, with topical prednisolone and cycloplegics followed by oral prednisolone 1 mg/kg/day. Within 3 days of starting treatment, the patient's vision improved to 6/9 in the right eye and 6/18 in the left eye.

Post-treatment OGTT showed a normal pattern of 5.40 mmol/L at 0 minutes, 7.44 mmol/L at 60 minutes, and 5.6 mmol/L at 120 minutes. At the last follow-up visit, the posterior uveitis had resolved and no patches of poliosis or vitiligo had appeared. The patient is followed up monthly and takes maintenance prednisolone 7.5 mg once daily. Repeat OGTT was performed and

the patient showed a normal OGTT response (5.40 mmol/L after 2 hours).

**Discussion**

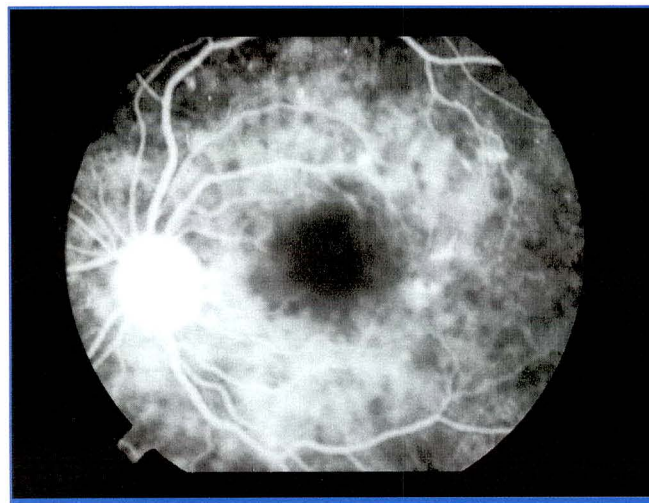


This patient was diagnosed with VKH syndrome, fulfilling the criteria laid down by the American Uveitis Society in 1978<sup>2</sup> and the revised diagnostic criteria for VKH disease.<sup>3</sup> In the literature, retinal oedema has been described as the first retinal sign of retinal involvement in VKH syndrome, followed by development of retinal detachment. This patient, who otherwise showed all the classical features of VKH disease,

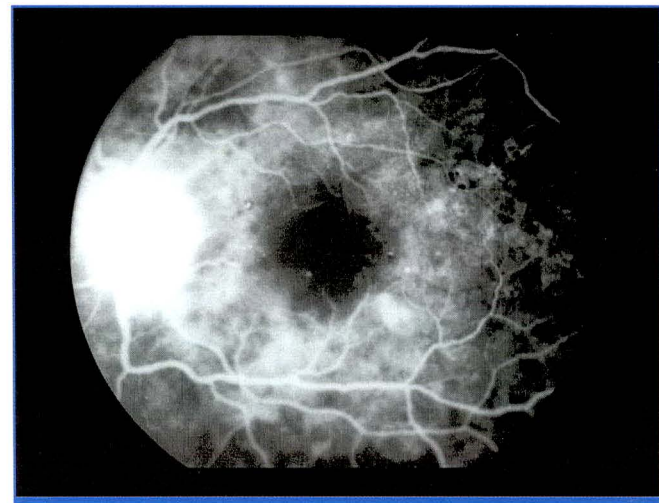
did not have exudative retinal disease and it was concluded that early presentation and prompt treatment prevented the development of exudative retinal detachment. In addition, the occurrence of confluent patches of hypopigmentation as a presenting feature has not been previously reported.

The unusual and clinically important observation was the diagnosis of glucose intolerance following the onset of acute uveitis and alopecia areata. The relationship of non-insulin dependant diabetes mellitus or glucose intolerance has been studied by Yawata et al, who showed that 55%

**Figure 4.** Fundus fluorescein angiography of the right eye with leaking disk capillaries.



**Figure 5.** Fundus fluorescein angiography of the left eye with leaking disk capillaries.





of patients with VKH syndrome (11/20) showed glucose intolerance but no apparent insulin secretion deficiency.<sup>4</sup> None of the control group showed glucose intolerance and these authors showed that glucose intolerance improved for most patients after systemic corticoid therapy, rather than worsening as could be expected. It is possible that glucose intolerance in patients with VKH syndrome may be related to an autoimmune inflammatory process of this disease.

In the patient described in this report, the follow up OGTT had a normal pattern. Jaggarao et al speculated that some processes alter the surface component both in melanocytes and pancreatic  $\beta$  cells, leading to endocrine disturbance.<sup>5</sup> However, there is currently no clear evidence that the cytotoxic cells recognise the same antigens in diabetes and VKH disease. It is now firmly believed that VKH syndrome results from a T cell mediated delayed

hypersensitivity response to an antigen shared by melanocytes in the skin, meninges, inner ear, and the choroid.<sup>1</sup>

Further studies and case reports may be needed to substantiate the mechanisms and association of glucose intolerance in VKH disease, and to understand whether or not the pathogenesis of the disease is autoimmune in nature.

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# Glaucoma — Optic Nerve Head Changes and Treatment

From the American Academy of Ophthalmology  
2001 Annual Meeting, New Orleans, USA,  
11-14 November 2001

## Morphologic Risk Factors for Progressive Glaucomatous Optic Nerve Head Changes

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Erlangen, Germany

A prospective clinical observational study was performed to evaluate which morphologic features of the optic disc are risk factors or indicators for progressive neuroretinal rim loss in chronic open angle glaucoma (COAG). Progression of glaucoma was defined as loss of neuroretinal rim.

The study included 394 eyes of 257 patients with COAG and the mean follow up was 31.8 months. All patients underwent repeated qualitative and morphometric evaluation of colour stereo optic disc photographs.

Progression of optic nerve changes was detected in 42 eyes (11%). At baseline, the neuroretinal rim was significantly smaller ( $p = 0.03$ ) and the beta zone of parapapillary atrophy was significantly greater ( $p = 0.04$ ) in the patients who progressed than in the non-progressing group. However, there were no significant differences between the groups in size and shape of the optic disc, optic cup depth, alpha zone of the parapapillary atrophy, and diameter of the retinal blood vessels. Multiple Cox-regression analysis revealed that glaucoma progression was significantly dependent on the area of the neuroretinal rim and the beta zone of parapapillary atrophy.

## Conclusion

Important morphological risk factors or indicators of progression of the glaucomatous appearance of the optic nerve head are small size of the neuroretinal rim and large area of the beta zone of parapapillary atrophy.

## Blue Arc Entoptic Phenomenon for Detecting Glaucomatous Visual Field Loss

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The blue arc phenomenon is an entoptic response from the nerve fibre layer. Although patients with advanced monocular glaucoma and good visual acuities who are able to see blue arcs in their healthy eyes but not in their glaucomatous eyes have been reported, the potential utility of the blue arc phenomenon in testing for nerve fibre layer function has been largely ignored. The

purpose of this study was to determine the specificity and sensitivity of the blue arc test for detecting perimetric visual field loss due to glaucoma in a clinical setting.

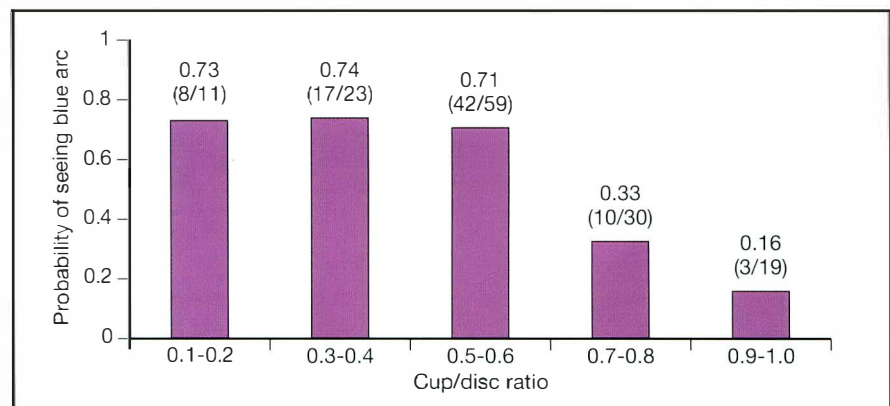
Seventy eight non-consecutive patients from a tertiary care glaucoma clinic who met the inclusion criteria were selected. Glaucomatous visual field loss was defined as one or both eyes demonstrating an abnormal hemifield test on the Humphrey visual field analyser.

The stimulus for the blue arc test was presented on a standard computer video display. A demonstration program was run first, showing the stimulus and simulated blue arcs. Each patient was then light-adapted for 2 minutes and dark adapted for 1 minute. Patients were then asked to fixate on an X while the stimulus was presented. The stimulus was a vertical red slit on a black background, located  $2.3^\circ$  nasal to fixation,  $0.86^\circ$  wide, and extending  $5^\circ$  vertically above and below the horizontal. The stimulus intensity was  $5 \text{ Cd/m}^2$ . The stimulus was presented 10 times for 0.5 seconds at 2-second intervals. The patient was again light- and dark-adapted, and the fellow eye tested in the same manner.

## Results

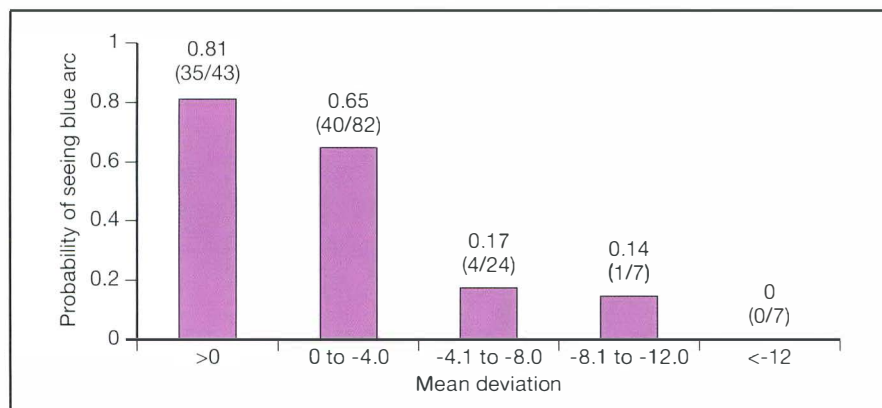
The sensitivity and specificity of the blue arc test for detecting glaucomatous visual field loss were 78% and 73%, respectively. The probability of seeing the blue arcs decreased with increasing cup/disc ratio (Figure 1),

Figure 1. Effect on the blue arc test of cup/disc ratio.

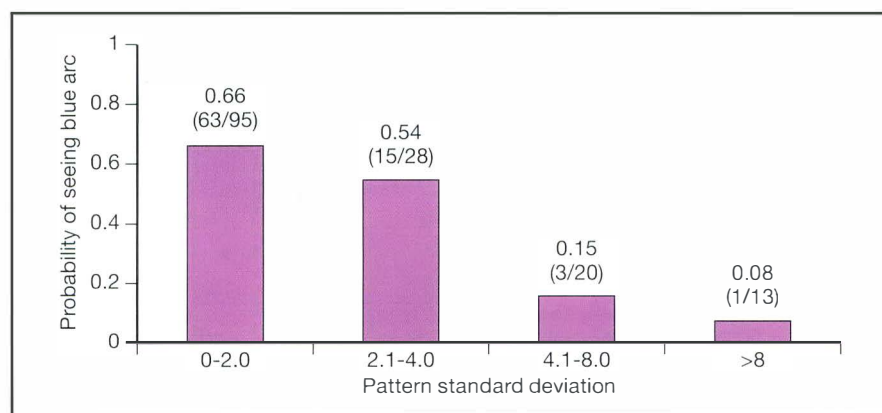




**Figure 2.** Effect on the blue arc test of mean deviation.



**Figure 3.** Effect on the blue arc test of pattern standard deviation.



12

increasing mean deviation (Figure 2), and increasing pattern standard deviation (Figure 3). Patients with pupils of 1 to 2 mm were less likely to see the blue arcs than those with larger pupils. However, this may be confounded by the higher proportion of patients with glaucoma in this subset of patients. Patients with cataract were also less likely to see the blue arcs.

The effect on the blue arc test of cup/disc ratio, mean deviation, and pattern standard deviation, as well as its good sensitivity and specificity in detecting an abnormal glaucoma hemifield test suggests that this test correlates with nerve fibre layer function. Indeed, this test may be uniquely suited to early detection of central glaucomatous nerve fibre layer damage since blue signals travel in larger diameter axons that are more susceptible to damage from high IOPs than small axons. The fact that the entoptic arcs are blue suggests

that this test may be selectively testing for the function of these more susceptible axons enabling the detection of early dysfunction.

In addition, the blue arc test stimulates nerve fibre layer and retinal cells within the central 3° of the visual field. Although central glaucomatous visual field defects tend to occur in the later stages, it is known that ganglion cell loss occurs centrally even early in the disease. Central visual field defects are likely to occur later in the course of the disease than peripheral defects because of the higher density of ganglion cells in this area, suggesting that a higher proportion of ganglion cell loss is required centrally compared with peripherally for equal field losses. The fact that some patients with glaucomatous visual field defects not involving the central 5° are unable to see the blue arcs suggests that this test may be more sensitive for detecting

central nerve fibre layer dysfunction than Humphrey visual field testing.

## Conclusion

The blue arc entoptic phenomenon is suppressed in eyes with nerve fibre layer dysfunction secondary to glaucoma, showing good sensitivity and specificity for detecting glaucomatous visual field defects. The test is easy and rapid for patients to perform, taking less than 30 seconds for each eye.

## Latanoprost for Normal Tension Glaucoma



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Norfolk and Norwich University Hospital  
UK

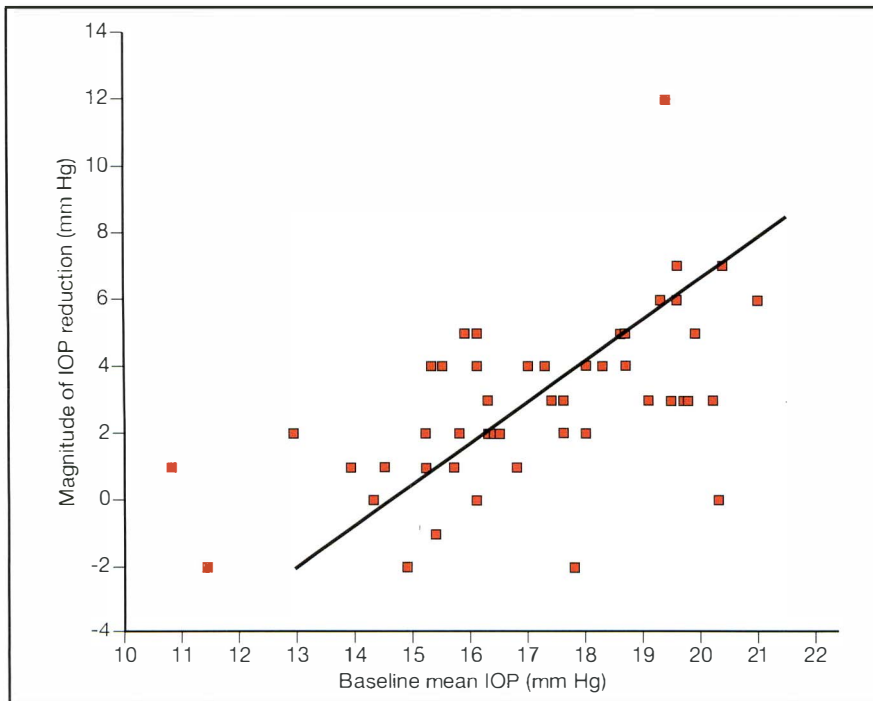
Normal tension glaucoma (NTG) accounts for approximately 25 to 30% of open angle glaucoma. While the pathogenesis of NTG remains an enigma, a reduction in intraocular pressure (IOP) by 20 to 30% from baseline has been shown to slow the rate of progression. Fistulising surgery, with or without antimetabolites is an effective method of lowering IOP. However, potential complications, particularly cataract, may mask the overall visual benefit of IOP reduction and medical treatment represents a potentially safer alternative without the adverse visual effects.

Treatment with pilocarpine, timolol, or betaxolol has had limited success due to side effects and/or a poor ocular hypotensive effect in NTG. Latanoprost has been found to be effective for the treatment of high pressure open angle glaucoma and studies in NTG show that latanoprost 0.005% daily reduces IOP by 18 to 21.3% in the short-term. This study was performed to determine the long-term effect of latanoprost on diurnal IOP in NTG.

In this trial, 81 patients with the following features were randomised to receive latanoprost 0.05% (n = 55) once daily or no treatment (n = 26):



**Figure 1.** Decrease in mean diurnal intraocular pressure (IOP) following treatment with latanoprost for normal tension glaucoma.



- glaucomatous optic disc changes and visual field defects characteristic of glaucoma
- no recorded IOP >24 mm Hg in either eye during a period of routine baseline daytime IOP phasing.

After a minimum of 6 months, each patient underwent a second IOP phasing period. Phasing consisted of hourly IOP measurements between 08.00 and 17.00 hours using an electronic hand held tonometer.

### Results

The mean duration of treatment was 10.5 months (range, 6 to 29 months). A 17.4% decrease in mean diurnal IOP and a 19.5% decrease in maximum diurnal IOP was found in the group receiving latanoprost. Forty percent of treated patients achieved at least a 20% decrease in mean diurnal IOP and the magnitude of reduction in mean IOP correlated with the pretreatment baseline mean IOP (Figure 1). Interestingly, treated patients with a higher initial mean diurnal IOP tended to achieve a

greater magnitude of IOP reduction. No statistically significant IOP changes were found in the control group.

### In Conclusion

Latanoprost appears to have a sustained IOP lowering effect in patients with NTG, with 40% of treated patients achieving a satisfactory decrease in mean diurnal IOP of at least 20%. This result, together with its favourable side effect profile, suggests that latanoprost represents a suitable treatment option for NTG.

### Concomitant Antiglaucoma Medications and Oral Non-steroidal Anti-inflammatory Drugs



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Prostaglandin  $F_2\alpha$  potentiates uveoscleral outflow. However, endogenous production of prostaglandins can be interrupted by

non-steroidal anti-inflammatory drugs (NSAIDs) via cyclo-oxygenase inhibition. Indeed, indomethacin has been shown to block the ocular hypotensive effect of epinephrine.

NSAID use is widespread among elderly populations, who may also be taking anti-glaucoma medications. For this reason, a trial was performed to ascertain whether any clinically relevant changes in intraocular pressure (IOP) lowering, visual function, or ocular perfusion occur in patients taking latanoprost or brimonidine plus NSAID therapy.

All ocular hypotensive medication was stopped 3 weeks prior to baseline, at which point 20 patients were randomised to receive either latanoprost 0.005% once daily or brimonidine 0.2% twice daily in the right eye. After 1 week, the left eye was given the other treatment (i.e. that not used for the right eye), while the right eye continued with the original regimen. After 1 week of bilateral topical treatment, oral indomethacin 25 mg 4 times daily was introduced and coadministered with the eye drops for 2 weeks.

### Results

After 1 week of treatment, the IOPs in the treated eyes had decreased by 14% with brimonidine ( $p = 0.004$ ) and by 25% with latanoprost ( $p < 0.0001$ ) [Table 1]. After 2 weeks of co-therapy with indomethacin, brimonidine-treated eyes had a slightly raised IOP, not significantly different to the baseline value ( $p = 0.3$ ), while latanoprost-treated eyes retained an IOP similar to that prior to the start of indomethacin therapy ( $p = 0.02$ ) [Table 1].

Pulsatile ocular blood flow (POBF) increased significantly with latanoprost by  $296 \pm 82 \mu\text{l}/\text{min}$  ( $p = 0.002$ ), while there was no significant change with brimonidine ( $72 \pm 57 \mu\text{l}/\text{min}$ ) after 1 week.

After 2 weeks coadministration with indomethacin, POBF had further increased



**Table 1. Intraocular pressure lowering effect of latanoprost and brimonidine with concomitant indomethacin.**

	Latanoprost (mm Hg)	Brimonidine (mm Hg)
Baseline	18.5 ± 1.3	19.1 ± 0.9
Week 1	13.8 ± 1.1	16.5 ± 1.3
Week 4	13.9 ± 1.3	16.9 ± 1.3

in the latanoprost-treated eyes, to 1164 ± 94 µl/min, but not in the brimonidine treated eyes, resulting in a difference

of 303 µl/min between the 2 groups (p = 0.004).

Retinal microcirculation increased

significantly with latanoprost alone, but was not significantly elevated with either drug during co-therapy with NSAIDs.

### Conclusion

Indomethacin therapy coincided with a loss of significance of the ocular hypotensive effect of brimonidine, but did not appear to alter that of latanoprost.

## SEAGIG 2002 — Glaucoma: Global & Southeast Asian Perspectives 2nd Biennial Meeting of the South East Asian Glaucoma Interest Group (SEAGIG)

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# Atlas of Oculoplastic Surgery

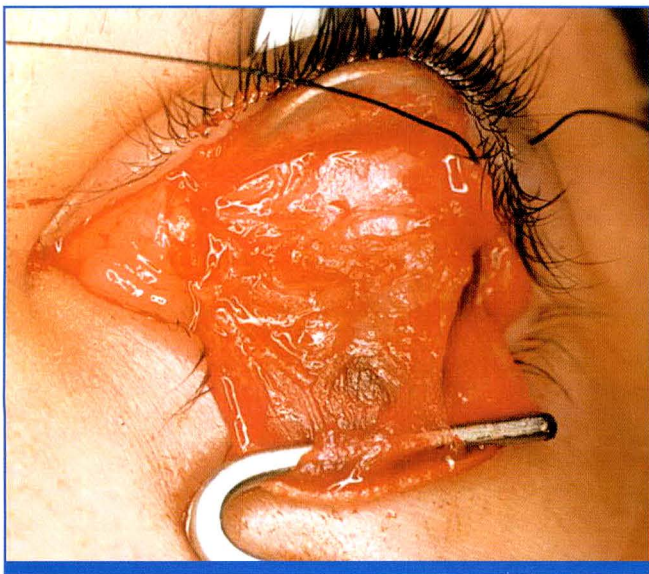
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Department of Ophthalmology, Hospital Valle del Nalón, Asturias, Spain

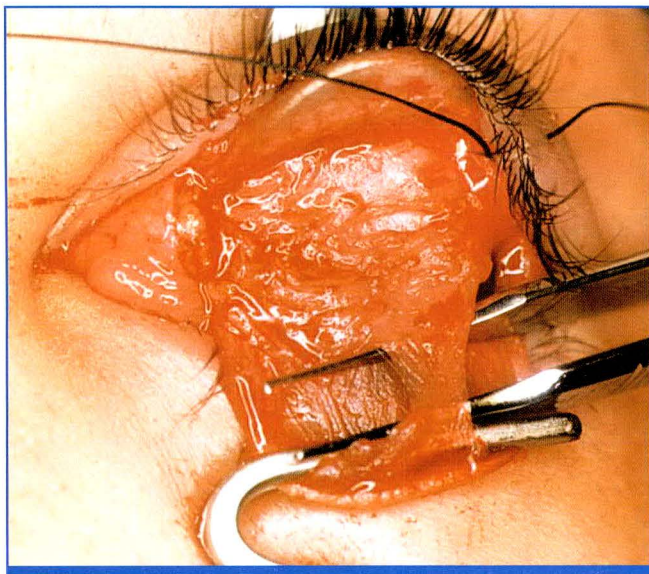
## Correction of Palpebral Ptosis via a Conjunctival Approach



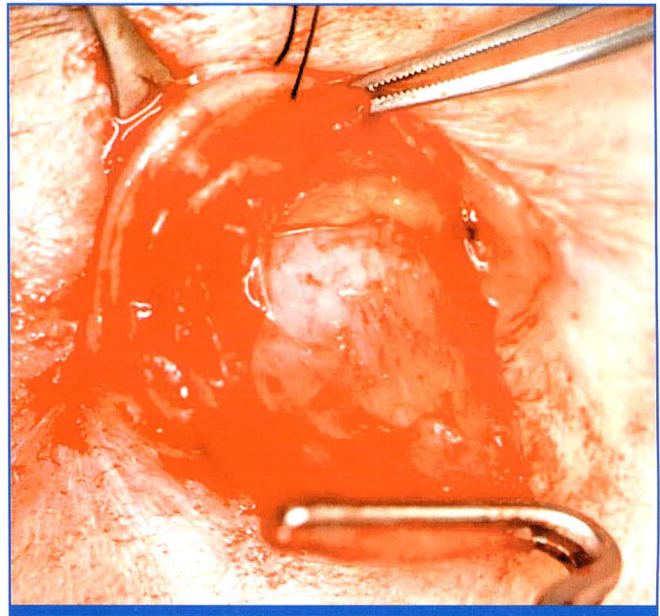
**Figure 1.** Palpebral conjunctiva and tarsus transected. The orbicular muscle is grasped by Berke forceps.



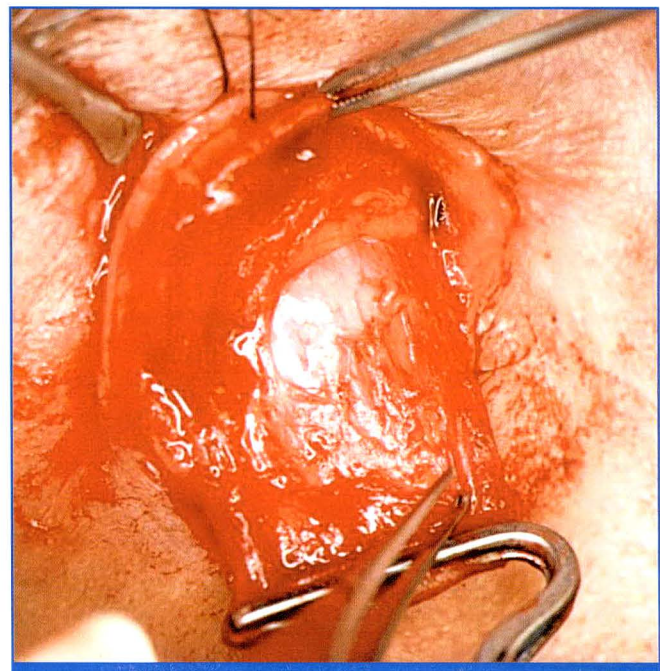
**Figure 2.** The orbicular is freed from the forceps.



**Figure 3.** The anterior surface of the elevator fascia is dissected.



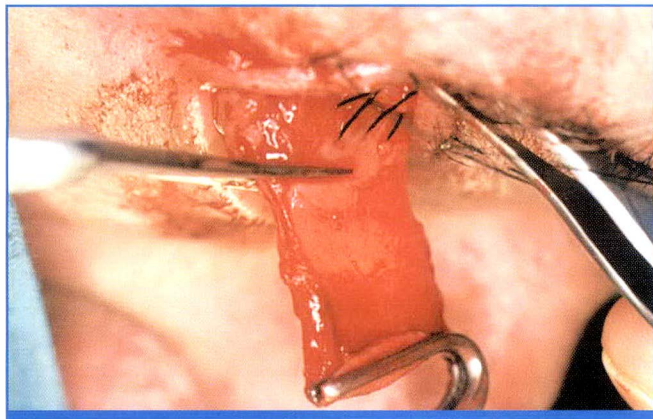
**Figure 4.** The captured tissues are assessed and the amount of fascia to be resected is measured.





# PICTORIAL OPHTHALMOLOGY

**Figure 5.** The selected tissue is transected.



**Figure 6.** Samples for pathology examination.



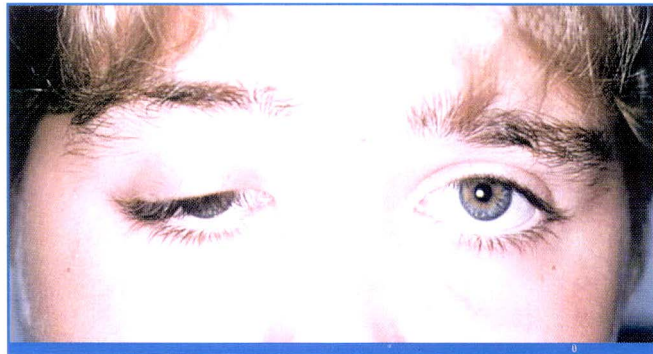
**Figure 7.** Mild congenital palpebral ptosis seen preoperatively in the right eye.



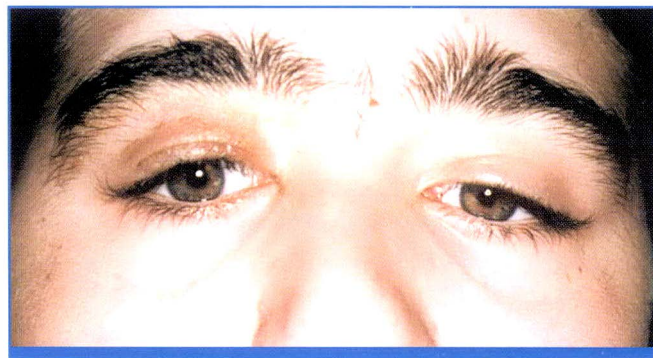
**Figure 8.** The same patient after surgery.



**Figure 9.** Moderate to severe congenital palpebral ptosis seen preoperatively.



**Figure 10.** The same patient 1 month after surgery.



**Figure 11.** The same patient 6 months after surgery.



This Pictorial Oncology was submitted by Dr Juan Junceda and is based on a CD-ROM of 15 years experience of oculoplastic surgery. The CD-ROM contains more than 2 and a half hours of in vivo surgery and 32 films distributed in 5 sections of anatomy, palpebral surgery, conjunctival surgery, lacrimal pathway surgery, and orbital surgery.

The CD-ROM is currently distributed only in Spanish-speaking countries, although it will soon be edited by an International Publisher, which will make it more readily available.



## Abstracts of Asian research published in the international literature

### **Glaucoma in China: How Big Is the Problem?**



This study was performed to derive preliminary estimates for the number of adults in China suffering from glaucoma, and project the burden of visual morbidity attributable to primary and secondary glaucoma. Age- and sex-specific data from 2 population surveys were applied to USA Census Bureau population estimates for urban and rural China. It was assumed that data from Singapore were representative of urban China, and those from Mongolia were representative of rural China.

It was estimated that 9.4 million people aged  $\geq 40$  years in China have glaucomatous optic neuropathy. Of this number, 5.2 million (55%) are blind in at least 1 eye and 1.7 million (18.1%) are blind in both eyes. Primary angle closure glaucoma (PACG) is responsible for the vast majority (91%) of bilateral glaucoma blindness in China. The number of people with the anatomical trait predisposing to PACG (an 'occludable' drainage angle) is in the region of 28.2 million, and of these 9.1 million have significant angle closure, indicated by peripheral anterior synechiae or raised intraocular pressure.

This extrapolation of data from 2 East Asian countries gives an approximate number of people in China suffering from glaucoma. It is unlikely that this crude statistical model is entirely accurate. However, the authors believe that the visual morbidity from glaucoma in China is considerable. PACG is probably the leading cause of glaucoma blindness in both eyes, and warrants detailed investigation of strategies for prevention.

Foster PJ, Johnson GJ. Glaucoma in China: how big is the problem? *Br J Ophthalmol* 2001;**85**:1277-1282.

### **Visual Acuity and Quality of Life After Cataract Surgery in Hong Kong**



Visual acuity, visual functioning, and vision-related quality of life outcomes after cataract surgery were assessed in a population based study in a suburban area of Hong Kong. A cluster sampling design was used to select apartment buildings within housing estates for enumeration. All enumerated residents aged  $\geq 60$  years were invited for an eye examination and visual acuity measurement at a site within each estate. Visual functioning and vision related quality of life questionnaires were administered to subjects who had undergone cataract surgery, to unoperated people with presenting visual acuity less than 6/60 in either eye, and to a sample of people with normal visual acuity.

36.6% of the 310 cataract operated individuals had presenting visual acuity 6/18 or better in both eyes, and 40.0% when measured by pinhole. 4.5% were blind, with presenting visual acuity of less than 6/60 in both eyes. Of the operated eyes, 59.6% presented with visual acuity 6/18 or better. 11.2% of the operated eyes were blind with vision less than 6/60. Visual acuity outcomes of 6/18 or better were marginally associated with surgery in private versus public hospitals. Lens status (pseudophakic versus aphakic) and surgical period (within 3 years versus earlier) were not significantly related to vision outcomes. Mean visual functioning and quality of life scores decreased consistently with decreasing vision status. Spearman correlation with vision status was 0.420 for visual functioning scores and 0.313 for quality of life scores. Among visual functioning/quality of life subscales, correlation was strongest for visual perception ( $r = 0.447$ ) among visual

functioning subscales and weakest for self care ( $r = 0.171$ ) among quality of life subscales. Regression adjusted visual functioning and quality of life total scores for cataract operated individuals were slightly lower than for those of visually comparable unoperated individuals ( $p < 0.05$ ).

Cataract operations in Hong Kong did not consistently produce good presenting visual acuity outcomes, suggesting that postoperative monitoring would be useful to minimise visual impairment in this population. Although vision outcomes were consistently correlated with all visual functioning/quality of life subscale scores, there was a differential impact with visual functioning subscales usually being affected more by reduced acuity than the more general quality of life subscales.

Lau J, Michon JJ, Chan WS, Ellwein LB. Visual acuity and quality of life outcomes in cataract surgery patients in Hong Kong. *Br J Ophthalmol* 2002;**86**:12-17.

### **Enucleation in a Tertiary Eye Care Centre in India**



Enucleation is a standard surgical treatment modality for many end-stage eye diseases. Indications for enucleation vary with changing trends in disease management. Few studies have addressed the issue of the frequency and indications of enucleation of eyes in India. This study aimed to determine the frequency and the current clinical indications for enucleation in patients at a tertiary eye care centre in India, and attempted a clinicopathological correlation.

Medical records of patients undergoing enucleation at a tertiary eye care centre from January 1995 to July 1998 were reviewed to obtain patients' demographic data and socio-economic status. The clinical indications and predisposing factors were assessed. The formalin-fixed, paraffin-embedded sections of all enucleated eyes were re-evaluated and histopathological findings were correlated with the clinical diagnosis. The prevalence of enucleation was calculated, and age



**Table 1. Clinical indications for enucleation.**

Indication	No. of cases (%)
Tumours	74 (49%)
Staphyloma	38 (25%)
Acute injury	20 (13%)
Absolute glaucoma	9 (6%)
Painful blind eye	5 (3%)
Phthisis bulbi	1 (1%)
Others	4 (3%)

adjustments were made using the Indian population data from the 1998 mid-year statistics.

Enucleation of the eye was performed in 150 patients (151 eyes) of 88,991 new ophthalmic cases, constituting 0.17% of the cases seen in hospital, and amounting to a prevalence of 0.33% (95% CI, 0.27-0.40). Males outnumbered females in a ratio of 1.85:1 (98 males, 53 females). The median age was 8 years (mean 16.8 ± 18.3 years). Children younger than 15 years old constituted 85.2% (95% CI, 81.2-89.21%) of patients undergoing enucleation. Clinical indications for enucleation are shown in Table 1.

Of the 74 patients with a clinical diagnosis of tumour, histopathology revealed retinoblastoma in 55 patients (74%), melanoma in 6 (8%), and ocular surface tumours in 4 (6%). Clinicopathological correlation was 100% in cases with a definite clinical diagnosis of retinoblastoma and melanoma. Nine blind eyes (6%) in which an intra-ocular tumour was one of the differential diagnoses were negative for a tumour on histopathology. Staphyloma was more prevalent in the low socio-economic group (p = 0.0004), with a history of childhood trauma in 34% cases.

The prevalence of enucleation in the population reporting to this tertiary eye care centre was 33 per 10,000 population during the study period of 3.5 years. Major indications for enucleation were tumours, staphyloma, and trauma (88% of all cases). Increased frequency in the young was due to the high proportion of retinoblastoma and staphyloma. Childhood trauma, inflammation,

and malnutrition may together play a role in the pathogenesis of staphyloma. Awareness at the level of primary health care providers, paediatricians and general practitioners should be promoted to identify the disease process at an early stage and facilitate early intervention measures that could result in eye and vision salvage.

Vemuganti GK, Jalali S, Honavar SG, Shekar GC. Enucleation in a tertiary eye care centre in India: prevalence, current indications and clinicopathological correlation. *Eye* 2001;15 (Pt 6):760-765.

### Ocular Biometry in Subtypes of Primary Angle Closure Glaucoma in Malaysia

Thirty seven consecutive patients (41 eyes) diagnosed with primary angle closure glaucoma (PACG) attending the Glaucoma Clinic in University Malaya Medical Centre, over a period of 6 months were categorised as having acute, subacute, and chronic PACG from the clinical presentation. Each patient underwent automated refraction, A-scan biometry for anterior chamber depth, axial length and lens thickness, keratometry, and corneal diameter measurement. Calculations for the relative lens position and the lens thickness:axial length index were performed. The data collected was analysed by the non-parametric test (Kruskal-Wallis), one-way analysis of variance (ANOVA), chi-square test, Spearman's non-parametric correlations, and regression analysis. For the controls, 15 eyes from 15 healthy subjects matched for age, sex, refractive error, and race were chosen and underwent the same examinations.

Chronic PACG was the predominant glaucoma subtype (53.6% of patients and 58.5% of eyes). The ocular biometric measurements of acute PACG eyes deviated most from controls in having the shallowest anterior chamber depth, shortest axial length,

smallest corneal diameter, steepest corneal radius, thickest and most anteriorly situated lens, and the greatest lens thickness: axial length index. The subacute subtype was closest to normal and the chronic PACG subtype fell in between in most of the biometric characteristics. These findings were not statistically significant. All PACG eyes as a group however showed statistically significant shallower anterior chamber depth (p < 0.05), and a more anterior relative lens position (p < 0.05) compared with controls.

Mimiwati Z, Fathilah J. Ocular biometry in the subtypes of primary angle closure glaucoma in University Malaya Medical Centre. *Med J Malaysia* 2001;56:341-349.

### Estimation of Blindness in India from 2000 to 2020: Implications for Blindness Control

Appropriate national planning based on current and reliable data is necessary to eliminate avoidable blindness in India. A national survey performed from 1986 to 1989 reported that 1.5% of the Indian population (12 million people) was blind with a presenting visual acuity of <6/60 in the better eye. The original goal of the National Programme for Control of Blindness was to reduce this prevalence to 0.3% by 2000. The prevalence of blindness in the population of Andhra Pradesh has recently been reported to be 1.66% with a presenting visual acuity of <6/60 in the better eye as the sole criterion and 1.84% with a presenting visual acuity of <6/60 or central visual field <20° in the better eye. These population-based data were used to estimate blindness in India in 2000 and the possible scenarios of blindness through to 2020 were projected with a different emphasis of the blindness control policy in India.

Recent population-based data on the age-, sex-, and cause-specific blindness rates from the Andhra Pradesh Eye



**Table 2. Projected blindness in India to 2020.**

Year	Number of blind persons (95% confidence interval)
2000	18.7 million (15.2-22.3)
2010	24.1 million (19.7-28.4)
2020	31.6 million (26.4-36.9)

Disease Study were applied to the population distribution of India to estimate the number of blind persons in 2000. The age-, sex-, and cause-specific rates of blindness were then applied to the estimated age, sex, and urban-rural population distribution of India in 2010 and 2020 to project the number of persons blind (from various causes) and the blind person-years that would be suffered under varying degrees of emphasis in the policy to control blindness due to particular diseases. For these projections, blindness was defined as a presenting distance visual acuity of <6/60 or central visual field <200 in the better eye.

The number of blind persons in India in 2000 was estimated to be 18.7 million, of whom 9.5 million had cataract-related and 3 million had refractive error-related blindness (Table 2). If there is no change in the current trend of blindness, the number of blind persons in India would increase to 24.1 million in 2010, and to 31.6 million in 2020 (Table 2). If effective strategies are put in place to eliminate 95% of blindness due to cataract by 2020, blindness in 15.6 million persons would be prevented who would otherwise be blind in 2020 if the current trend continues, and 78 million blind person-years would be prevented. Similarly, if effective strategies are also implemented to eliminate 95% of the refractive error blindness by 2020, another 4.2 million

persons would be prevented from being blind in 2020, and 82 million blind person-years would be prevented. In addition, if strategies to prevent 90% of the preventable blindness due to corneal disease and glaucoma are successful by 2020, blindness in an additional 3.6 million persons and 29 million blind person-years would be prevented.

The planning of blindness control in India should take into account recent population-based data for the entire age range, which suggest that the number of blind persons in India is currently more than 18 million. This estimate is 50% more than the figure of 12 million from a decade ago that is still widely quoted in the blindness control policy documents. If avoidable blindness is to be substantially reduced in India by 2020, effective strategies against blindness due to cataract and refractive error are urgently needed since both these conditions are relatively easy to treat. Also, strategies against preventable corneal and glaucoma blindness need to be strengthened for them to show an impact over the next 2 decades.

Dandona L, Dandona R, John RK. Estimation of blindness in India from 2000 through 2020: implications for the blindness control policy. *Natl Med J India* 2001;14:327-334.

### **Visual Impairment, Blindness, and Cataract Surgery Among Elderly People in Hong Kong**

The prevalence of vision impairment, unilateral/bilateral blindness, and cataract surgery were estimated in a population-based survey among the elderly in a

suburban area of Hong Kong. Fifteen public, private, and home ownership scheme housing estates in the Sha Tin area of Hong Kong were subjected to cluster sampling to randomly select a cross section of people aged  $\geq 60$  years.

Visual acuity measurements and ocular examinations were conducted at a community site within each estate. The principal cause of reduced vision was identified for eyes with presenting visual acuity worse than 6/18.

3441 subjects from an enumerated population of 4487 (76.7%) underwent an eye examination. The prevalence of presenting visual acuity less than 6/18 in at least 1 eye was 41.3%; and 73.1% in those aged  $\geq 80$  years. Unilateral blindness (acuity <6/60) was found in 7.9% of subjects and bilateral blindness in 1.8%.

Refractive error and cataract were the main causes of vision impairment and blindness, respectively. Visual impairment with either eye <6/18 increased with advancing age and was more prevalent in males, the less educated, and those living in public housing estates. The prevalence of cataract surgery was 9.1% and was associated with advancing age and lower educational standard.

Blindness and visual disability were common in this socioeconomically advanced population, with most of it being easily remedied. Because of a rapidly ageing population, healthcare planners in Hong Kong must prepare for an increasing burden of visual disability and blindness.

Michon JJ, Lau J, Chan WS, et al. Prevalence of visual impairment, blindness, and cataract surgery in the Hong Kong elderly. *Br J Ophthalmol* 2002;86:133-139.



## MAY

5-10

### The Association for Research in Vision and Ophthalmology (ARVO) Annual Meeting

**Fort Lauderdale, Florida, USA**

Tel: (1 301) 571 1844

Fax: (1 301) 571 8311

31-1 June

### Age-Related Macular Degeneration: Update 2002

**Baltimore, Maryland, USA**

Contact: Johns Hopkins Continuing Medical Education, 720 Rutland Avenue, Baltimore, MD 21205, USA

Tel: (1 410) 955 2959

Fax: (1 410) 955 0807

## JUNE

1-5

### 2002 Annual Symposium and Congress of the American Society of Cataract and Refractive Surgery

**Philadelphia, Pennsylvania, USA**

Contact: ASCRS-ASOA, 4000 Legato Road, Suite 850, Fairfax, Virginia 22033, USA

Tel: (1 703) 591 2220

Fax: (1 703) 591 061

E-mail: ascrs@ascrs.org/asoa@asoa.org

9-13

### 3rd International Conference on Ocular Infections

**Jerusalem, Israel**

Contact: Stepping into the New Millennium, P.O. BOX 50006, Tel Aviv 61500, Israel

Tel: (97 23) 5 140 014

Fax: (97 23) 5 175 674/5 140 077

E-mail: ocular@kenes.com

13-16

### Canadian Ophthalmological Society Annual Meeting

**Hull, Quebec, Canada**

Contact: Kimberley Ross

Tel: (613) 729 6779

Fax: (613) 729 7209

E-mail: cos@eyesite.ca

27-30

### 4th International Symposium of Ophthalmology

**Shantou, China**

Contact: Symposium Secretariat, Department of Ophthalmology and Visual Sciences, The Chinese University of Hong Kong, 3/F, Hong Kong Eye Hospital, 147K Argyle Street, Kowloon, Hong Kong

Tel: (852) 2762 3123

Fax: (852) 2715 9490

E-mail: alicechoy@cuhk.edu.hk

## JULY

27-28

### UKM Ophthalmology Symposium 2002: Corneal Update

**Kuala Lumpur, Malaysia**

Contact: Dr N Bachik/Dr ML Bastion, Dept of Ophthalmology, Faculty of Medicine, Universiti Kebangsaan Malaysia

Tel: (60 3) 9170 2962/9173 3333 Ext 2160

E-mail: maelynnb@hotmail.com

## AUGUST

3-5

### Singapore National Eye Centre 5th International Meeting Singapore

Contact: Ms Amy Lim, Organising Secretariat, Singapore National Eye Centre, 11 Third Hospital Avenue, Singapore 168751

Tel: (65) 322 8374

Fax: (65) 227 7290

E-mail: Amy\_Lim@snecc.com.sg

## SEPTEMBER

7-11

### 20th Congress of the European Society of Cataract & Refractive Surgeons

**Nice, France**

Contact: European Society of Cataract & Refractive Surgeons, 10 Hagan Court, Lad Lane, Dublin 2, Ireland

Tel: (353) 1 661 8904

Fax: (353) 1 678 5047

E-mail: escrs@agenda-comm.ie

27-28

### SEAGIG 2002 — Glaucoma: Global & Southeast Asian Perspectives 2nd Biennial Meeting of the South East Asian Glaucoma Interest Group Manila, The Philippines

Contact: The SEAGIG Manila Secretariat, c/o OmniEssence Company, Suite 1014 Shaw Tower, Shaw Blvd, Corner St Francis Street, Greenhills East, Mandaluyong City 1550, The Philippines

Tel: (632) 636 7655

Fax: (632) 636 7656

E-mail: OmniEssence@usa.net

## OCTOBER

6-11

### 15th Congress of the International Society for Eye Research Geneva, Switzerland

Contact: Kenes International, 17 Rue du Cendrier, P.O. Box 1726, CH - 2111 Geneva 1, Switzerland

Tel: (41 22) 908 0488

Fax: (44 845) 127 5678

E-mail: icer@kenes.com

20-24

### Annual Meeting of the American Academy of Ophthalmology Orlando, Florida, USA

Contact: American Academy of Ophthalmology

Tel: (1 415) 561 8500

Fax: (1 415) 561 8533

E-mail: meetings@aaoo.org

## MARCH 2003

19-23

### 4th International Glaucoma Symposium Barcelona, Spain

Contact: Kenes International, PO Box 1726, CH-1211 Geneva 1, Switzerland

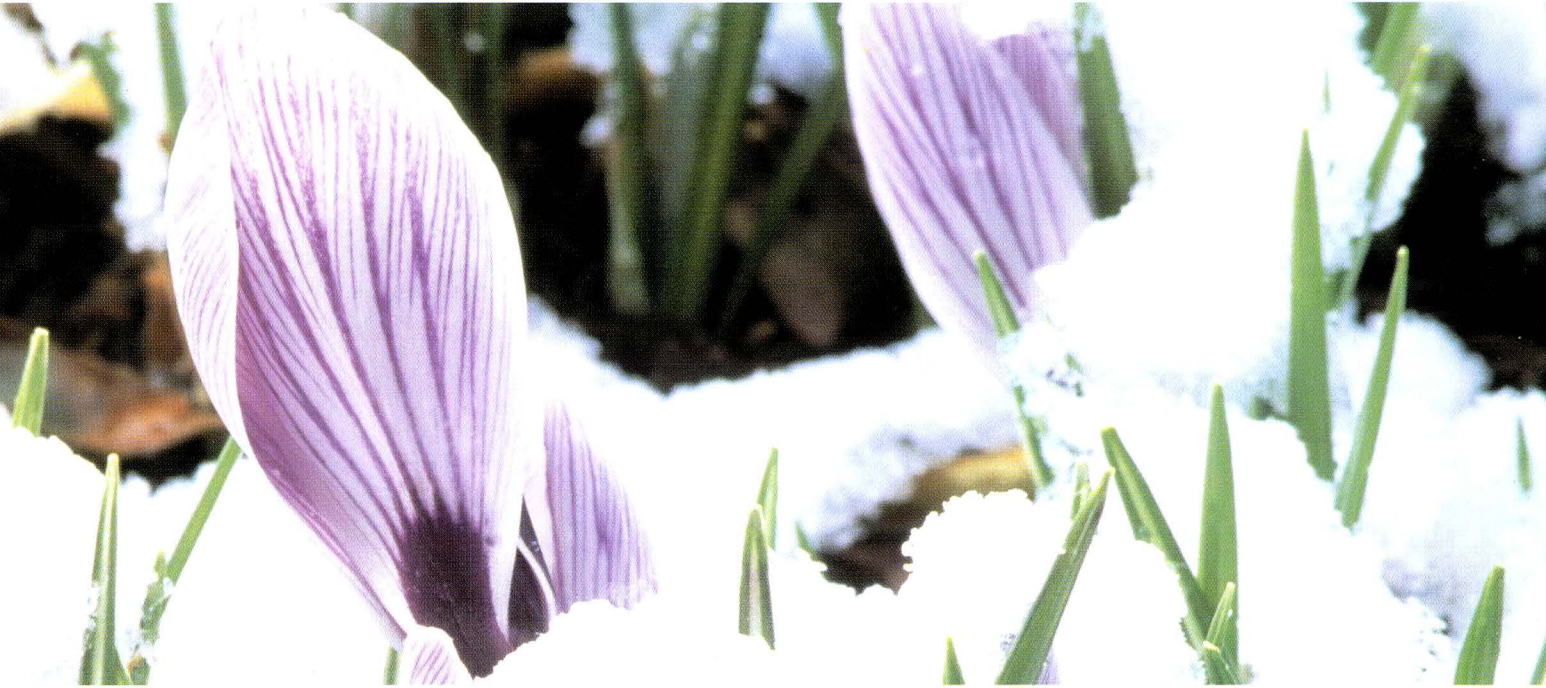
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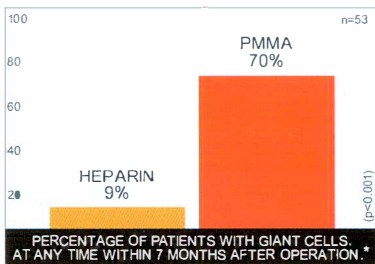
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