Journal of the South East Asia Glaucoma Interest Group

Volume 7, Number 4, 2005

Asian Journal of OPHTHALMOLOGY

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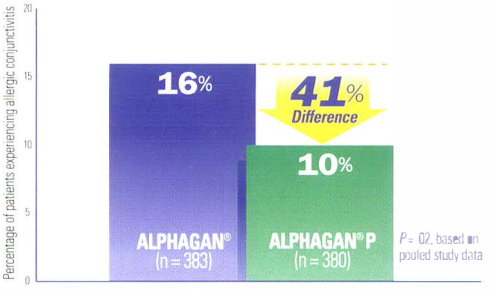

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1. ALPHAGAN® P Prescribing Information. 2. Katz LJ. Twelve-month evaluation of brimonidine-purite versus brimonidine in patients with glaucoma or ocular hypertension. *J Glaucoma* 2002;11(2):119-126

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Asian Journal of
OPHTHALMOLOGY
Volume 7, Number 4, 2005

SEAGIG
South East Asia Glaucoma Interest Group

Asian Journal of OPHTHALMOLOGY is the official peer-reviewed journal of the South East Asia Glaucoma Interest Group (SEAGIG) and is indexed in EMBASE/Excerpta Medica. The website of Asian Journal of OPHTHALMOLOGY and SEAGIG membership details can be found at **www.seagig.org**.

As new technologies and therapeutic interventions are continually being developed, ophthalmology has become a field of rapid change, particularly in the Asia-Pacific region, where disease patterns and health care delivery differ greatly from those seen in the West. Asian Journal of OPHTHALMOLOGY was established in 1998 and became the official journal of SEAGIG in 2003, with the aim of disseminating information relevant to ophthalmology and glaucoma throughout Asia and to interested groups worldwide. The objectives of Asian Journal of OPHTHALMOLOGY are as follows:

- to provide a platform for the publication of information with a focus on ophthalmology in Asia
- to disseminate information that will improve the care of patients with all types of ophthalmological disorders, with a special focus on glaucoma
- to increase the understanding of such disorders through reporting of educational activities
- to publish the results of research programmes to expand knowledge about the causes, prevention, and treatment of ophthalmological disorders
- to work closely with Asian and international researchers to achieve these aims
- to provide a forum for young and relatively inexperienced researchers to present their research results as Original Articles via an international platform
- to maintain and promote relationships with any organisation with similar goals.

Although the focus of Asian Journal of OPHTHALMOLOGY is on glaucoma, other topics relevent to the region will not be ignored, and submissions on all aspects of ophthalmology are welcome.

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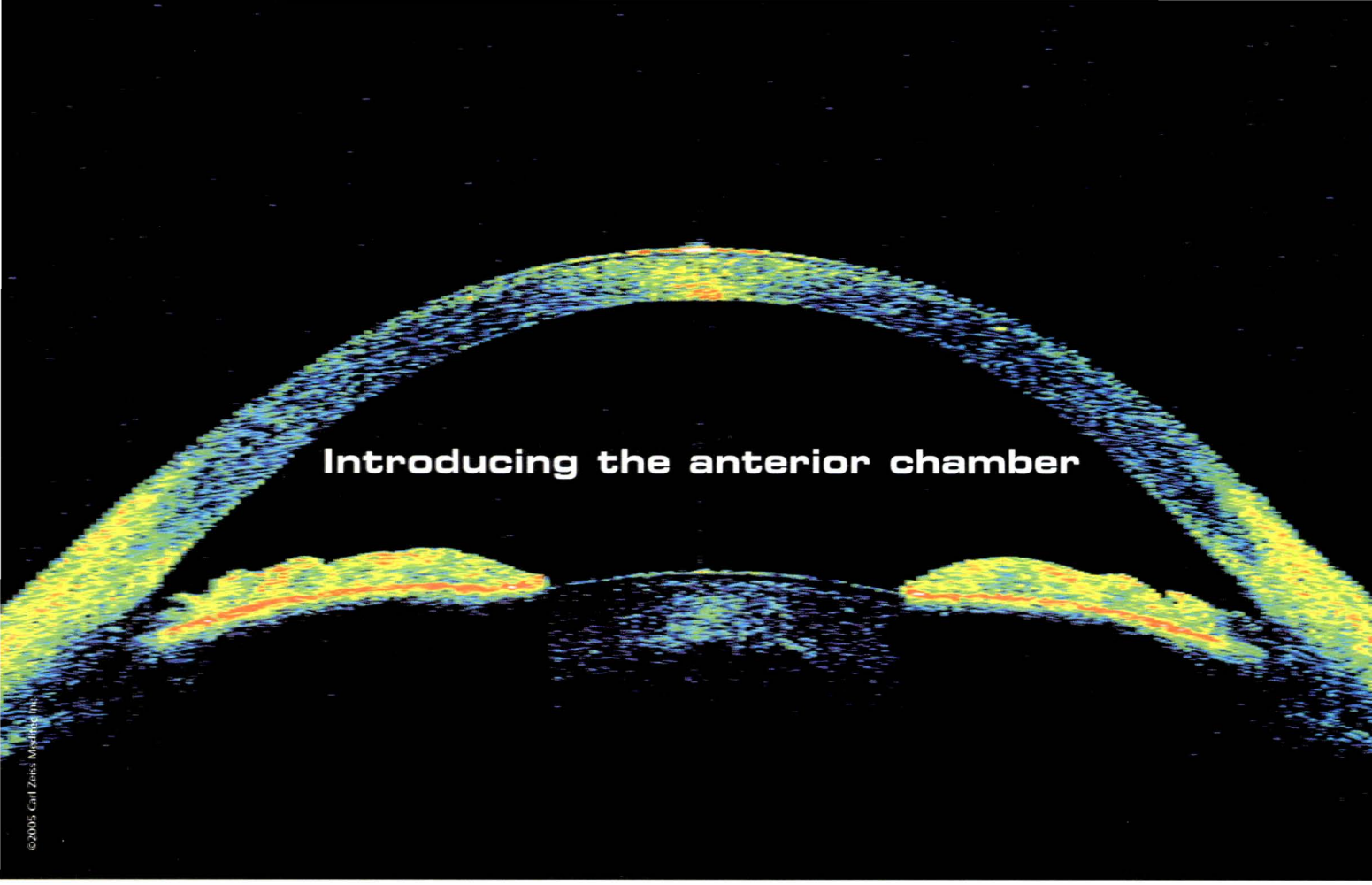
Volume 7, Number 4, 2005

TABLE OF CONTENTS



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An Asian Journey

I am a part of all that I have met;
Yet all experience is an arch wherethro'?
Gleams that untravell'd world, whose margin fades?
For ever and for ever when I move.

Sir Alfred Tennyson, *Ulysses*

The August 2005 issue of *The Times* bears the statement: "Today, it is clear that the Asian Century has begun. What remains unclear are the factors that caused that enormous change." The answer to our query is not the destructive world wars and the consolidation of national sentiments, but the ability of man to open his eyes to widen his horizons and make his mind borderless — to make his eyes, heart, and mind work together.

The Asia Pacific Glaucoma Guidelines are a fine example of that human gesture to enhance humanity, irrespective of race, colour, or creed. It is a Ulyssean effort to reach Ithaca, his home, and that of his father.

The guidelines have been produced by the South East Asia Glaucoma Interest Group (SEAGIG) to assist ophthalmologists throughout the region to optimally manage patients with glaucoma using the world's best current knowledge and standards. The aim of the guidelines is to improve the mutual understanding of glaucoma and to provide a rational approach to the diagnosis and management of this disease, with specific emphasis on the problems associated with glaucoma among Asian races. The work is intended to complement the existing scientific literature and textbooks, and serves as an aid to dealing with glaucoma in a rapidly changing medical and socio-economic environment.

East Asians have the highest rates of blindness in the world, caused by a relatively common form of glaucoma, primary angle closure glaucoma (PACG). The new guidelines launched by SEAGIG at the 19th Congress of the Asia Pacific Academy of Ophthalmology (APAO) in Bangkok aim to help clarify issues surrounding glaucoma treatment and to reduce the rate of blindness in the region. The first ever Asia Pacific Glaucoma Guidelines describe the pathological and epidemiological differences in glaucoma, particularly PACG, between Asians and Caucasians. The guidelines also give doctors in Asia guidance on how to employ new developments in glaucoma treatment, including highly effective medicines — prostaglandins — and laser surgery.

The guidelines are easy reading because the format is divided into 3 sections in the usual way of approaching patients — assessment, treatment, and follow-up.

We are grateful for the constructive criticism and novel ideas put forward by many colleagues who are experts in their field. Let us look into our own techniques set against the format of these guidelines; this way, we can learn or profit from it. This way too, we might find out that our strategies and techniques may contribute to the ongoing research into glaucoma.

Do it now — read the guidelines. Test them, use them, and comment on them. Although they are Asian, involving all the experience of our Asian practitioners, the guidelines are a resource for anyone involved in eye care anywhere in the world. They certainly can show you an unknowing assessment of your own practices and strategies in the professions.

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Asia Pacific Glaucoma Guidelines

Ivan Goldberg
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During the past 20 years, increasing data from across the Asia Pacific region have highlighted the varying prevalence and incidence rates, diagnostic types, and behaviours of different glaucoma disease patterns encountered by the ophthalmological community. What is relevant, timely, and applicable in one part of the world may not be in another, and it was felt that there was a need for guidelines relevant to the Asia Pacific region. In 2003 to 2004, the South East Asia Glaucoma Interest Group, with the support of the Asian Oceanic Glaucoma Society, published the Asia Pacific Glaucoma Guidelines. By increasing awareness and knowledge of the glaucomas and their detection and management across the Asia Pacific region, the Asia Pacific Glaucoma Guidelines aim to reduce glaucomatous visual disability and to provide a rational basis for glaucoma management in a cost-effective manner.

Key Words: *Asia, Glaucoma, Guidelines*

Asian J Ophthalmol 2005;7(4):126-130.

In 2003 to 2004, the South East Asia Glaucoma Interest Group (SEAGIG), with the support of the Asian Oceanic Glaucoma Society (AOGS), published the Asia Pacific Glaucoma Guidelines.[1] Why was this done? Why was it done just now?

With the support of the American Glaucoma Society, the American Academy of Ophthalmology produced its *Preferred Practice Pattern* for primary open angle glaucoma in 1996 (with a limited revision in 2003),[2] with one for primary angle closure following 4 years later,[3] and a third on primary open angle glaucoma suspect in 2002.[4] These guidelines identify characteristics and components of quality eye care "commensurate with present knowledge and resources". This translates to guidance for state-of-the-art patterns of practice, which should be helpful for the care of most patients, although not for that of a particular individual. Where data permit, these guidelines are firmly evidence-based; otherwise, they rely on collective wisdom.

Similarly, the European Glaucoma Society published its *Terminology and Guidelines for Glaucoma* in 1998,[5] with the aim of "improving the mutual understanding of this disease in addition to providing a rational approach to the diagnosis and management of glaucoma". A second edition was published in 2003.[6] These projects have set out to complement the existing scientific literature, and involved input from many glaucoma subspecialists. The resulting publications have proven useful for, and thus popular with, comprehensive ophthalmologists around the world; they have also been useful for communication with allied eye health care professionals, and with governmental agencies and non-governmental organisations.

During the past 20 years, increasing epidemiological data from across the Asia Pacific region have highlighted the varying prevalence and incidence rates, diagnostic types, and behaviours of different glaucoma disease patterns encountered by the ophthalmological community. With the availability of this information, the need could be met for similar guidelines relevant to the Asia Pacific region. Such guidelines needed to be sensitive to the wide range of human, structural, and equipment resources available throughout the region. What is relevant, timely, and applicable in one country or location may not be in another.

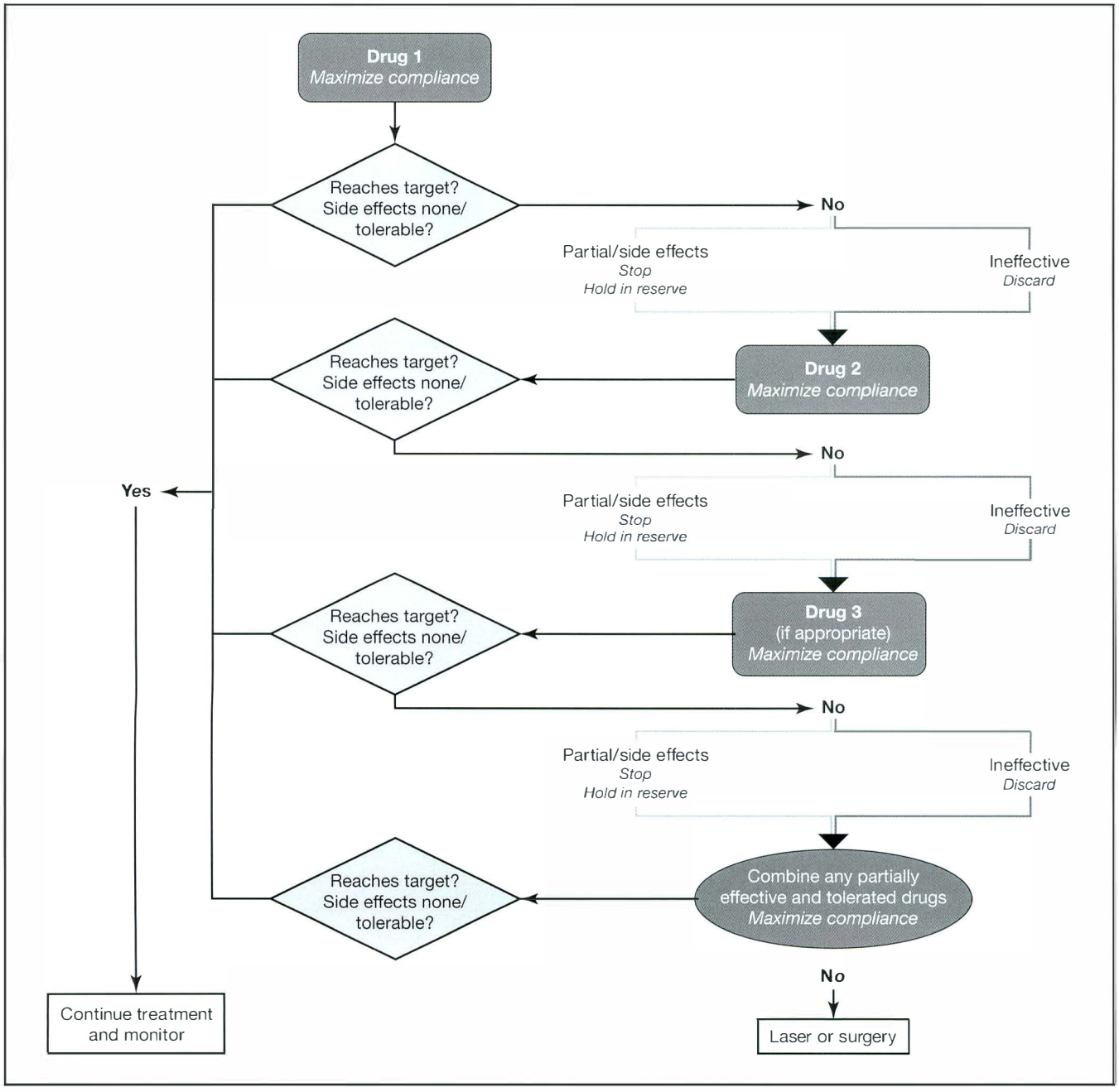
The Asia Pacific Glaucoma Guidelines have been developed during a time of rapidly expanding medical technology, knowledge, and skills in a changing environment. As public awareness and expectations increase, and as the population ages (even in less developed societies), there is an increasing demand for high-level care for a greater number of people over extended periods of time.

However, the available resources have been unable to expand proportionally. As cost containment is an inescapable reality, every treatment or investigation that is undertaken reduces the capacity to implement another intervention that could benefit another patient for the same or a different condition. What we as clinicians do for our patients needs to be demonstrated to be effective. If not, it must at least be recognised to be only partly proven or as yet unproven. Guided by this knowledge, our lines of enquiry can be channelled appropriately. By identifying missing areas of knowledge, the very act of developing guidelines influences research efforts and funding priorities to support such research.

In the development of these guidelines, the SEAGIG Working Party tried to use underlying evidence and to stratify the recommendations according to their strength. This allowed the guidelines to suggest areas for further research. It is hoped this will facilitate our growth as a mutually supportive ophthalmic community.

Dr Ivan Goldberg is President of the South East Asia Glaucoma Interest Group.

Figure 1. Medical treatment algorithm.



What Is the Structure of the Guidelines?

How big is the problem? What are the risk factors? The section on Epidemiology tries to answer these questions. How do we take a focused history, perform an appropriate examination, and what additional tests might we consider for the patient before us with respect to glaucoma presence or absence, the risk of developing the condition, and the degree of damage if present? The section on Patient Assessment tackles this, for both initiation of therapy and its follow-up.

The Diagnostic Category and Targets section helps the reader to tailor treatment goals to the severity of glaucoma. Treatment of glaucoma has been divided into medical, laser, and surgical therapy (Figure 1 shows the medical treatment algorithm). Case detection is considered from both population screening and opportunistic points of view.

These guidelines have been developed in an easy-to-read format for the benefit, in the first instance, of comprehensive ophthalmologists. The format answers questions of 'Why?', 'What?', 'When?', and 'How?', as shown in the example in Figure 2.

Table 1. Appendix 4: How to optimize patient performance in subjective perimetry.

1. **Choose the most appropriate investigation**
   - Test pattern — 24-2: early/moderate damage and glaucoma suspects; 10-2: advanced damage or paracentral scotomas.
   - Test strategy — e.g., SITA (Humphrey field analyzer): most patients and suspects.
2. **Patient set-up at the perimeter**
   - Use near lens power based on current refraction.
   - Support the patient's feet comfortably so that the thighs are horizontal.
   - Support the patient's back.
   - Adjust chin rest height so the forehead touches its holding band easily.
   - Cover other eye fully — some patients prefer it open, some closed.
   - Support the arms so shoulders and neck do not tire.
3. **Instructions to the patient before starting the test**
   - "We are getting you to do this test to give us information. We want to see how full and perfect your vision is, or if it isn't, we want to know where the damage is, and what sort of damage it is."
   - "The test is not difficult, but to get the best information for your care, it needs to be done in a particular way."
   - "The key to success is to look straight ahead all the time. (Point where you want them to look.) Let the light come to you — don't go looking for it."
   - "You won't see the light a good deal of the time, so don't worry if time seems to be passing without a light appearing. The machine makes the light very dim so that it can tell when you can just see it."
   - "Press the button when you think you see the light. All the lights you see count — they can be fuzzy, dim, bright, it doesn't matter."
   - "Blink whenever you need to, but do so when you press the button. That will stop your eyes drying out and hurting, and you won't miss any lights."
   - "Hold the button down when you want to rest. That will pause the machine. Release the button when you want to continue. Remember you can rest as often as you like. You're the one controlling the machine."
   - "Let's have a practice run now so you can get a feel for the whole thing." (This is essential for perimetric novices, but may be important for many others as well. Run the demonstration program.)
4. **Patient support during the test**
   - Do not abandon the patient during the test — have your technician return regularly and frequently to supervise.
   - Reassure and encourage the patient during the test.
   - Restart the test if the performance is proving unreliable. Try to identify and to rectify the cause of the problem. Do not disparage or "blame" the patient.
   - Consider rescheduling the test if the patient cannot cope.
   - Be patient, more patient, and then even more patient.

Table 2. Appendix 1: How to test calibration of a Goldmann tonometer.

1. Set the tonometer in position on its slit lamp stand, with its Perspex biprism head in place and the tension on the circular dial on its right side (from the examiner's side of the slit lamp) set at 5 mmHg. The head should lean slightly forwards (away from the examiner).
2. Slowly twirl the circular dial counter-clockwise until the head rocks back towards you. The tension should read 0-2 mmHg below zero (Figure A).
3. Slowly twirl the dial clockwise until the head rocks forward again. The tension should read 0-2 mmHg (Figure B).
4. Remove the calibration rod from its box. Firmly screw into position the holding bracket that slides along the rod so that the closest mark in front of the center one (i.e., on the other side of the center from you) is aligned as exactly as you can (Figure C).
5. Slip the rod and its holder into the receptacle on the right side of the tonometer. The head will rock back towards you.
6. Slowly twirl the circular dial clockwise until the head rocks forwards. Note the tension reading on the dial: it should be 20-23 mmHg.
7. Slowly twirl the circular dial counter-clockwise until the head rocks backwards. The tension on the dial should read 17-20 mmHg.
8. Remove the rod and holding bracket from the tonometer and reposition the bracket so that it is aligned exactly with the most forward mark on the rod — furthest away from you (Figure D).
9. Replace the rod in its bracket in the tonometer receptacle. The tonometer head should rock backwards, towards you.
10. Slowly twirl the dial clockwise until the head rocks forward. The tension should read 60-64 mmHg.
11. Slowly twirl the dial counter-clockwise until the head rocks backwards — the tension should read 56-60 mmHg.

Comments:
The three threshold tension levels being used to test the tonometer's calibration are at 0, 20 and 60 mmHg. At each of these thresholds, you can gently twirl the dial backwards and forwards, reading the tension as the head responds: these points should bracket the threshold level evenly — the higher the level being tested, the greater the interval is likely to be.

(Figures A, B and D — Courtesy of Haag-Streit AG and Mandarin Opto-Medic Co Pte Ltd)

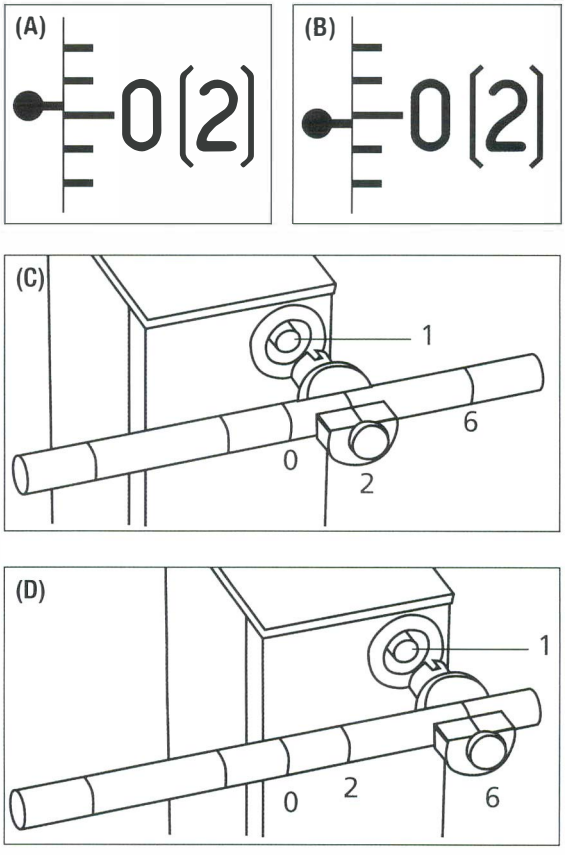


Figure 2. Example showing the comprehensive format of the Asia Pacific Glaucoma Guidelines.

Slit lamp examination — tonometry

Why?	Intraocular pressure (IOP) is the only modifiable risk factor for glaucoma.
What?	Goldmann-style Applanation Tonometry (GAT). (Tonopen, if GAT not available).
When?	Every visit.
How to perform Goldmann tonometry?	• Ensure tonometer is calibrated (see Appendix 1: How to test calibration of a Goldmann tonometer). • The prism tip must be disinfected and then disinfectant removed. • The eyelashes must be kept out of the way (avoid pressure on the eye). • The cornea must be anesthetized. • The tip must touch the central cornea gently with the observer looking through the slit lamp eyepiece just prior to the tip making contact *(tip: look for the white split ring that will fluoresce when the tip touches the cornea).* • Adjust the gauge until the split tear meniscus just touches on the inside.

Table 3. Appendix 3b: Goniogram/gonioscopic chart.

Grading system for gonioscopic findings (without indentation):

A. van Herick method uses corneal thickness as a unit of measure

Grade 0	Iridocorneal contact
Grade I	Peripheral anterior chamber depth between iris and corneal endothelium is less than 1/4 corneal thickness (occludable)
Grade II	Greater than 1/4 but less than 1/2 of corneal thickness
Grade III	Greater than or equal to 1/2 of corneal thickness (non-occludable)

B.

Grade	0	I	II	III	IV
Shaffer	Closed	10°	20°	30°	40°
Modified Shaffer	Schwalbe's line is not visible	Schwalbe's line is visible	Anterior TM is visible	Scleral spur is visible	Ciliary band is visible

C. Spaeth

1. Iris insertion
   Anterior to Schwalbe's line or TM
   Behind Schwalbe's line
   Centered at scleral spur
   Deep to scleral spur
   Extremely deep/on ciliary band
2. Angular width
   slit
   10°
   20°
   30°
   40°
3. Peripheral iris configuration
   queerly concave
   regular
   steep
4. TM pigment
   0 (none) to **4** (maximal)

Reference

Stamper RL, Lieberman MF, Drake MV. Clinical Interpretation of Gonioscopic Findings. In: Becker-Shaffer's Diagnosis and Therapy of the Glaucomas, 1999, 7th Ed, p101-113, St. Louis: Mosby.
Spaeth GL. The normal development of the human anterior chamber angle: a new system of descriptive grading. Trans Ophthalmol Soc UK 1971;91:709-739.

As with all treatment guidelines, the Asia Pacific Glaucoma Guidelines are not a prescription for automated care. By adapting the information to the individual patient, relevant to each individual's needs plus the socio-economic environment and medical facilities available, and involving the reader's own experience, the SEAGIG Working Party responsible for the guidelines hopes that the hallmark of excellent care will be more easily and effectively achieved.

How Can the Reader Derive Maximal Benefit from these Guidelines?

The guidelines contain a wealth of information in a condensed and approachable format. At first they need to be read comprehensively in their entirety so that their scope and depth are familiar. Then they can be available in the clinic where they can be used as required, one section or part of a section at a time. Ophthalmic assistants or technicians, for example, could benefit greatly from advice on how to set up a patient to obtain perimetric data most effectively (Appendix 4; [Appendix 4 is shown in Table 1]); registrars could use Appendix 1 to understand exactly how to check the calibration of a Goldmann tonometer (Appendix 1 is shown in Table 2) or Appendices 3a to 3c to enhance their understanding of gonioscopy (Appendix 3b is shown in Table 3).

In contrast to the format of most glaucoma textbooks, which deal with the presentation, recognition, and treatment of each glaucoma subtype, the guidelines address the management of glaucoma in the setting of a clinician confronted with a patient. Detection of the presence of any type of glaucoma requires certain skills and knowledge in history taking, and then focused examination with ordering of relevant investigations. Interpretation of the results of the investigations needs to be appropriate and then integrated with the

Table 4. Table 3.1: SEAGIG decision square for GON.

- The table below illustrates how various combinations of risk factor profiles and levels of disease stability/progression would influence the aggressiveness of medical, surgical or laser intervention.
- Intervention is graded +, ++, +++, with the last indicating the most active level of intervention.
- A +++ grade may be associated with a rapid, stepwise progression through medical to surgical management.
- A — indicates no addition to therapy.

Risk	Disease status		
	Stable	Uncertain	Progressing
Increased	+	++	+++
Uncertain	Reassess risk	Reassess both	++
Stable	—	Reassess disease	+

history and clinical examination. The SEAGIG decision square for glaucomatous optic neuropathy summarises this (Table 4). Treatment strategies then flow. Assessment of the success of these strategies, with modifications as needed, is also addressed for all types of glaucoma, again from the perspective of a clinician helping a single patient. The more the guidelines are used, the more they will benefit a clinician.

By increasing awareness and knowledge of the glaucomas, their detection and management across the Asia Pacific region, these guidelines aim to reduce glaucomatous visual disability and to provide a rational basis for glaucoma management in a cost-effective manner. These are worthy goals. You, the reader, will have to determine whether or not these goals have been achieved. SEAGIG and AOGS welcome constructive criticism so that the second edition will be even better.

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Barriers to Learning in Teaching Programmes

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For the past 10 years, I have had the privilege of teaching 2-week glaucoma workshops in ophthalmology training centres worldwide; the majority have been in the various regions of Asia. The condition that we call glaucoma is one of the more complex that ophthalmologists encounter. The correct diagnosis and treatment of any given patient with glaucoma requires skilful history taking combined with a thorough ocular exam. I often tell those whom I teach that, when presented with a patient with, or suspected of having, glaucoma, we must be expert detectives, paying great attention to even the smallest detail.

There are well-recognised centres of excellent tertiary eye care in Asia such as those in Singapore and Beijing, but these programmes are very much the exception. Unfortunately, the majority of ophthalmologists in developing countries have not received adequate training in the pathophysiology, diagnosis, and treatment of this very complex disease process. Too often this results in misdiagnosis and/or inappropriate treatment

The ophthalmologists I encounter in my workshops, with only rare exceptions, are intelligent, motivated, and caring individuals, but have received substandard training for a variety of reasons. They have been trained almost exclusively by didactic lectures and may not have been allowed to ask questions. To successfully diagnose and treat patients with glaucoma, thinking in the decision-tree algorithmic manner is essential. Not to do so leads to haphazard and dangerous conclusions. But what if this pattern of thinking is missing in an ophthalmologist who has completed the training available at the local level? What would be a suitable solution for filling in the gaps in essential knowledge?

Supplemental education in the form of an intensive 2-week workshop can correct much of the knowledge gap and result in a substantial improvement in the quality of care and, hopefully, reduction in the chances of glaucoma blindness. The format of the workshop involves selection of 5 ophthalmologists from 5 different teaching centres within a country or adjoining countries. They become the core group and must have completed a residency, have a sincere interest in glaucoma, and agree to teach others what they have learned from the workshop once they return to their home institutions. In addition, the workshop comprises 20 hours of didactic lectures, with no limit on the numbers who attend.

With worrying frequency, I experience situations that are serious barriers not only to learning, but also to acceptable clinical care. I have come to expect, but am still surprised by, very serious gaps in basic knowledge that are obvious barriers to good clinical practice. The inability to elicit a meaningful history from the patient is almost always present and is accompanied by a failure to perform a thoughtful and systematic ocular examination, making it impossible to present a patient in the usual and universal manner. Other examples include not knowing the significance of, or how to test for, a relative afferent pupillary defect, not understanding the clinical importance of pinhole vision and not knowing how to write a meaningful clinical record. In fact, it is not unusual to discover that there has been a total absence of any documentation of clinical findings. Very few trainees have ever been taught to perform gonioscopy.

Clinical Practice Guidelines

Elsewhere in this issue, Ivan Goldberg describes the recent publication by the South East Asia Glaucoma Interest Group of the Asia Pacific Glaucoma Guidelines. This 100-page booklet was produced by leading international glaucoma experts from the Asia Pacific region to establish best practice methodologies throughout Asia. It is a remarkably concise, comprehensive, and easy-to-follow set of instructions about how to diagnose and treat the glaucomas. I am constantly looking for teaching aids; the guidelines eloquently describe the best and most practical clinical recipe for glaucoma diagnosis and treatment that I have encountered. The information is appropriate for all ophthalmologists who treat patients with glaucoma, regardless of their educational level.

It is apparent to me that these guidelines can be a major part of the solution to the problem of the many undereducated ophthalmologists in developing countries who, through no fault of their own, were not taught the essentials of basic clinical care — not only for glaucoma, but also for general medicine. The guidelines offer a straightforward approach to the problems noted above, such as history taking, a thorough clinical examination, and performance of appropriate clinical tests. The goals of therapy are clearly defined and treatment options are presented in an algorithmic format. I plan to incorporate the guidelines into my future workshops and leave a copy with all of the core members.

Although the guidelines are not a substitute for personal, over-the-shoulder clinical teaching by knowledgeable ophthalmologists, they are a most valuable resource for appropriate clinical care of patients worldwide, especially in Asia where the most common form of glaucoma is that of chronic angle closure. Accordingly, the guidelines emphasise gonioscopy, which is essential for chronic angle closure evaluation and treatment, although greater emphasis could be given to indention/pressure gonioscopy with the 4-mirror gonioprism in future editions. This is a handbook of glaucoma diagnosis and treatment that can supplement personal clinical training or be referred to, along with other resources, until personal training is available.

Train the Trainers

I cannot over-emphasise the need for clinical training by well-educated volunteers. You do not have to be a subspecialty professor to teach residents how to think and do gonioscopy. The AAO International Volunteer Registry will match potential volunteers with those who are looking for help. Use this resource, pick a destination, and become a Trainer of Trainers. You will make a huge contribution to the reduction of global blindness by spending 1 to 2 weeks in a general eye clinic of a teaching institution by teaching basic clinical care. Of course, subspecialty teaching is also in demand, but be prepared to incorporate the basics as well.

The computer and the digital age have enormously expanded the amount of readily available quality ophthalmological teaching material. A partial list of resources follows:

1. The AAO Basic and Clinical Science Course, Section 10, 2004-2005. Glaucoma. www.orbis.org
2. AAO International Registry. www.aao.org
3. ORBIS Cyber Sight. telemedicine.orbis.org/bins/home.asp
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Cross-sectional Differences in Axial Length of Young Adults Living in Urban and Rural Communities in Mongolia

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Aim: *To assess differences in axial length of the eye between young adults aged 20 to 39 years living in urban and rural environments in central Mongolia.*

Participants and Methods: *Multi-stage, clustered, simple random sampling was used to identify 375 people living in the capital city (Ulaanbaatar) and 375 in an adjacent rural district (Selenge aimag). Axial length was measured using A-mode ultrasonography. Height and weight were recorded. Socio-economic data were obtained using a questionnaire.*

Results: *568 participants (75.7%) were examined. Mean axial length was 23.35 mm (95% confidence interval, 23.27-23.43 mm). Participants in the urban area were 2 cm taller and 3 kg heavier than the rural population, and 5 times as likely to have been educated to college level ($p < 0.001$). The ratio of participants in the rural group reporting a very low income to those in the urban group was more than 3:1 ($p < 0.001$). Axial length was significantly longer in the urban participants (23.53 mm; SD, 1.03 mm) than in the rural participants (23.19 mm; SD, 0.90 mm) [$p < 0.001$]. This difference was apparent in the age groups of 20 to 29 years and 30 to 39 years. Mean axial length was significantly longer in people aged 20 to 29 years (23.66 mm; SD, 1.13 mm) compared with those aged 30 to 39 years (23.37 mm; SD, 0.85 mm) [$p = 0.03$] in the urban group, but the same difference was not present in the rural group. Multivariate analysis identified height ($p < 0.001$) and educational achievement ($p < 0.001$) as being significantly associated with axial length. Urban or rural residence was of borderline significance ($p = 0.054$).*

Conclusions: *These data indicate that axial length is significantly longer in city-dwelling young adults in Mongolia. In part, this is attributable to their greater height. The link with educational achievement may reflect the recognised association with near-work and myopia. It seems likely that other important associations explaining axial length differences between urban and rural populations remain to be identified.*

Key Words: *Age groups, Mongolia, Myopia, Rural population, Urban population*

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Introduction

Myopia is increasingly being recognised as an endemic eye disease in industrialised regions in Asia. Moreover, the prevalence appears to be increasing, particularly in East Asia.[1] The pattern is not only discernable in children but also in older people. For example, the prevalence among Chinese Singaporeans aged 40 years and older is one of the highest worldwide.[2]

The explanation for this higher prevalence of myopia in East Asians is unclear. In the Singapore study, the prevalence of myopia was bimodal, with the highest rates being seen among those aged 40 to 49 years and 70 to 79 years.[2] This has been attributed to a higher prevalence of axial myopia in younger people (a cohort effect), and more index myopia (associated with nuclear sclerosis) in older people.[3] This latter observation highlights a major potential confounding factor in the study of epidemiology of refractive errors in adults. Grouping axial and index myopia in risk factor studies may risk obscuring important associations for 2 conditions with separate causal pathways. Increases in the prevalence of cataract with age, at least in part, drives the association between index myopia in the population and increasing age, whereas increasing axial length is linked with increasing rates of myopia in the younger population prior to the onset of cataract.[3] Hence, such studies in adults must separately address index and axial myopia. A clear understanding of axial biometry in populations being studied is therefore essential. A cross-sectional study showed that mean axial length is approximately 23.00 mm in Chinese Singaporean people older than 55 years. However, in people in their 40s, it is approximately 23.50 mm ($p < 0.001$).[3] This suggests either a significant decrease in axial length with advancing age, or a pronounced shift in the distribution of refractive errors in the population is

underway. The former seems biologically implausible. The latter is supported by the observation that, in a cross-sectional study of Mongolian people aged 40 years and older, axial length was longer in older individuals by an average of 0.05 mm/decade ($p = 0.03$).[4]

Both environmental and genetic aetiologies of myopia have been suggested. In Singapore, questionnaire-based research in children linked current near-work activity to the presence of myopia.[5] Among Singaporean military conscripts, intensity of previous education was associated with myopia.[6] It has also been suggested that dietary factors may play a role in the causation of myopia.[7,8] Twin studies indicate heritability of refractive error to be between 24% and 85%,[9,10] and genetic loci associated with familial high myopia have been identified.[11,12] However, to date, the relative role of environment and genotype in the aetiology of myopia remains unclear.[13]

In the context of the authors' previous biometric studies, the hypotheses for this study was that axial length is increasing in younger people, and that this trend is especially pronounced in urban areas. The aim of the study was to characterise axial length distribution in a young adult population in Mongolia and compare differences between the urban and rural populations.

Participants and Methods

This study was approved by the Ethics Committees of The Health Sciences University of Mongolia, Ulaanbaatar, Mongolia, and London School of Hygiene and Tropical Medicine, London, UK, and performed in accordance with the tenets of the World Medical Association's Declaration of Helsinki.

Sample Size

Sample size calculations used data from Singapore, where axial length was found to be substantially longer in younger people than in older people (age 40 to 49 years — 23.58 mm; SD, 1.31 mm; age 70 to 79 years — 23.00 mm; SD, 0.88 mm).[3] This age-related difference in Singapore of 0.6 mm over 4 decades is assumed to be a secular trend reflecting a cohort effect associated with environmental/lifestyle changes. From these data, it was assumed that a difference of approximately 50% (0.3 mm; SD, 1.3 mm) might exist between urban and rural Mongolians. To achieve 80% power to detect such a difference with a type I error rate of 5% would require 295 participants in each group. Anticipating 25% absenteeism and non-participation rates, the calculated sample size was 750 participants. Non-permanent residents and those who had moved to an area of permanent residence elsewhere were specifically excluded.

Sampling

Mongolia has a population of 2.5 million, with 28% residing in the capital city, Ulaanbaatar. Nearly 90% of the population is ethnic Mongolian with smaller numbers of Buriads, Darhads, Uzbeks, Uighurs, Russians, and Chinese. Administratively, Mongolia is divided into 21 provinces (aimags), the populations of which range from 46,000 to 122,000. Aimags are in turn divided into towns (aimag centres) and surrounding rural districts (sums). Each sum has an average population of 3000, and is comprised of sum centre (village) and bags (areas roamed by a group of semi-nomadic families), the lowest rural administrative unit. Ulaanbaatar city is divided into 6 city districts (duureg). These in turn contain smaller administrative units (horoo). Ulaanbaatar city was selected as the urban study site (population, 707,200). Population records maintained by the central government are used to allocate people living in specific areas of the city to a family doctor. The doctors maintain these records with current addresses of their patients. Also included are age, sex, and national identity number. There is an incentive to register, as failure to do so means the individual cannot receive government-subsidised healthcare. From the 6 duureg, 3 were selected at random: Chingeltei, Sukhbaatar, and Songinokhairkhan. Within each of these, one horoo was randomly identified. The family doctors' records were then used to draw a simple random sample within each of the 3 selected horoo. From the 3 aimags adjacent to Ulaanbaatar city, Selenge aimag (population, 105,500; area, 41,200 km^2) was chosen at random. Selenge has 17 sums, from which Shaamar was randomly selected. The neighbouring sums (Dulaanhaan and Zuunburen) were chosen on the basis of convenience. The electoral register was used to draw a clustered, simple random sample from the population of these 3 sums forming the rural study group. The electoral register is updated every 4 years. This had been done recently, as a parliamentary election was held on 27 June 2004. The record of urban and rural residents obtained was then used to stratify the population by age and sex. Approximately equal numbers of participants were drawn from the age strata 20 to 29 years and 30 to 39 years.

Examinations

Examinations were carried out between 1 June and 1 August 2004. The axial length, anterior chamber depth, and lens thickness were measured in all participants using the A-mode corneal contact ultrasound device (model 820; Humphrey Instruments Inc., San Leandro, USA), recording the mean of 5 separate readings. Benoxinate drops (Chauvin Pharmaceuticals, Kingston-on-Thames, UK) were instilled into both eyes before biometric assessment. Self-reported highest educational achievement was recorded as primary (to age 11 years), secondary (to age 16 years), or college/university. Height and weight of the participants were measured without boots. Body

mass index (BMI) was calculated as weight/(height)^2.

Data Analysis

Biometric data from the right and left eyes were highly correlated (Pearson correlation of right and left axial length, r = 0.91; p < 0.001), and consequently data from the right eyes were arbitrarily selected for presentation. Differences between groups were analysed using the Student *t* test (continuous variables) and the Chi squared test (categorical variables). Significance was assumed at the 5% level. The relationships between axial length, socio-demographic, and anthropometric data were analysed using linear regression. Differences between axial length in urban and rural areas were assessed using multiple logistic regression to adjust for multiple effects. Results are presented as mean and SD.

Results

750 eligible participants were enumerated, of whom 568 (75.7%) were examined. The response rate in the 6 clusters ranged from 68% to 86%. 267 participants (47%) were men and 301 (53%) were women. Men were significantly heavier (66.4 kg; SD, 12.2 kg) and taller (169.1 cm; SD, 6.8 cm) than women (57.6 kg; SD, 9.9 kg; and 158.2 m; SD, 6.0 cm) [both p < 0.001]. There was no significant difference in BMI between men (23.2) and women (23.0) [p = 0.165]. In the 3 sums of Selenge aimag (rural) 298 participants (79.5%) were examined. In the 3 districts of Ulaanbaatar city (urban) 270 participants (72.0%) were examined. Urban residents were taller (164.4 cm) and heavier (63.3 kg) than people in the rural area (162.3 cm and 60.2 kg, respectively) [both p = 0.002]. Table 1 summarises the numbers of participants selected and examined. Table 2 gives the numbers of participants examined in rural and urban areas by age and sex. There was no difference in the proportion of people in the age groups 20 to 29 years and 30 to 39 years in rural versus urban cohorts.

Table 1. Summary of participants selected and examined by cluster.

Cluster	Number examined	Number enumerated	Response rate (%)
Selenge (rural)			
Shaamar	172	200	86.0
Zuunburen	51	75	68.0
Dulaankhaan	75	100	75.0
Subtotal (rural)	298	375	79.5
Ulaanbaatar (urban)			
Chingeltei	102	145	70.3
Sukhbaatar	38	55	69.1
Songinokhairkhan	130	175	74.3
Subtotal (urban)	270	375	71.7
Total (urban and rural)	568	750	75.7

Table 2. Age and sex distribution of participants examined in rural and urban locations.

	Rural		Urban	
Age (years)	Men	Women	Men	Women
20-29	69	75	76	76
30-39	70	84	52	66
Subtotal	139	159	128	142
Total	298		270	

Ocular biometric data were available for 566 of the 568 participants (99.6%). There was no significant difference in axial length between right and left eyes in rural or urban areas. For right eye data, mean axial length was 23.35 mm (95% confidence interval [CI], 23.27-23.43 mm), mean lens thickness was 3.89 mm (95% CI, 3.87-3.92 mm), and mean anterior chamber depth was 3.24 mm (95% CI, 3.21-3.27 mm). Figure 1 shows that axial length distribution closely approximated a Gaussian distribution, but was leptokurtotic

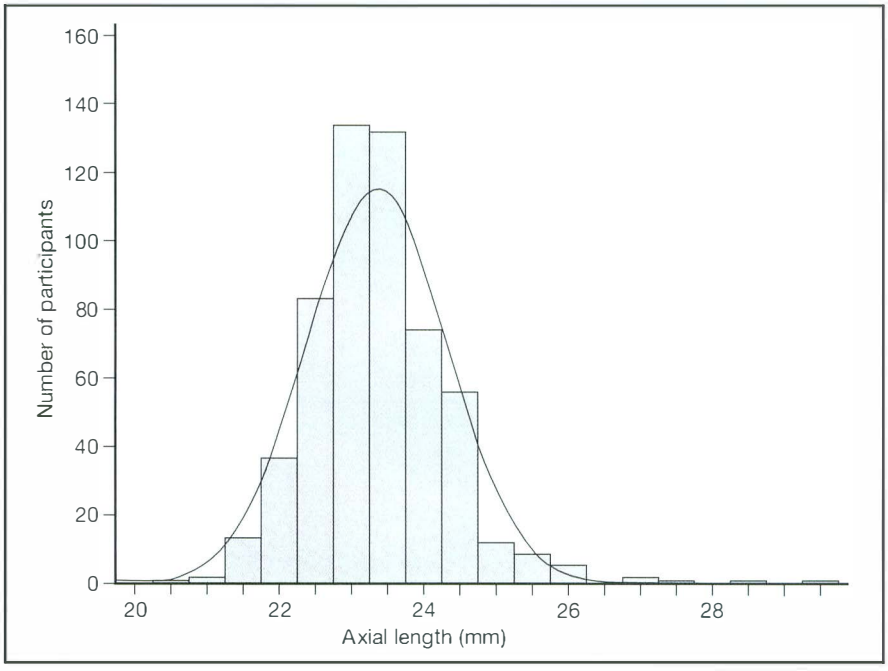

Figure 1. Histogram of axial lengths among the study participants.*

* The distribution approximates to a Gaussian distribution (superimposed curve) but is leptokurtotic (an exaggerated central peak) with a modest right-hand skew.

(exaggeration of the height of the central peak) with a modest right-hand skew. Table 3 compares sociodemographic and ocular biometric variables between participants in urban and rural areas. There was no difference in the mean age or proportion of women. Similarly, there was no difference in BMI, anterior chamber depth, or lens thickness. There was a trend towards lower intraocular pressure (IOP) in the urban group, but this was not significant ($p = 0.095$). Participants in the urban area were, on average, 2 cm taller and 3 kg heavier than the rural population ($p = 0.02$). Five times more urban people had been educated to college level than those from the rural environment ($p < 0.001$). The ratio of those receiving a very low income in rural to urban groups was more than 3:1 ($p < 0.001$). Table 4 shows the mean age-specific mean axial length in rural and urban groups. Axial length was significantly longer in people living in Ulaanbaatar city compared with those in the country. This difference was apparent in the age groups 20 to 29 years and 30 to 39 years. Mean axial length was significantly longer in people aged 20 to 29 years compared with those aged 30 to 39 years in the city. This difference was not seen in the rural group.

Figure 2 shows the variation in axial length of the right eye with height. Inspection shows an increase in axial length with height that appears linear until height exceeds 180 cm, at the extreme upper end of the distribution in this population. In a univariate model, a 10 cm increase in height was associated with a 0.27 mm (95% CI, 0.17-0.36 mm) increase in axial length. As shown in Table 4, height, weight, income, and education all differed significantly between urban and rural participants. A multiple linear regression model was constructed to explore the relationships between axial length (dependent) and these other variables. The model was built using a forward stepwise method requiring $p < 0.05$ for entry. Neither weight nor income were significant and were excluded. Area of residence (urban/rural) was not significant in this model. The regression coefficients suggest that a 10 cm increase in height was associated with a 0.27 mm ($p < 0.001$; 95% CI, 0.18-0.37 mm) increase in axial length. With a unit increase in educational achievement (primary [3], secondary [2], and college [1]) there was a 0.36 mm ($p < 0.001$; 95% CI, 0.19-0.52 mm) increase in axial length. The process was repeated for age and sex, neither of which were significant. This suggests that height and educational achievement are significantly associated with axial length. If urban/rural residence was included in the model, in conjunction with height and educational achievement, axial length was found to be 0.17 mm ($p = 0.061$; 95% CI, -0.008 to 0.35 mm) longer in city residents, after adjusting for differences in height and education. Using logistic regression to compare the characteristics of city and country-dwellers (dependent variable), education ($p < 0.001$) and height ($p = 0.002$) were again significant in the model. Axial length was found to be of borderline significance ($p = 0.062$).

Table 3. A comparison of sociodemographic and ocular biometric variables between urban and rural Mongolian people aged 20 to 39 years.

	Urban	Rural	p Value
Age (SD) [years]	29.0 (5.4)	29.7 (5.8)	0.11
Women (%)	142 (52.6)	159 (53.4)	0.86
Height (SD) [cm]	164.4 (8.6)	162.3 (8.1)	0.02*
Weight (SD) [kg]	63.3 (13.5)	60.2 (10.0)	0.02*
Body mass index (SD) [kg/m^2]	23.3 (4.0)	22.9 (3.5)	0.17
College education (%)	156 (57.8)	34 (11.5)	<0.001*
Income below US$50 (%)	60 (22.2)	211 (70.1)	<0.001*
Right axial length (SD) [mm]	23.53 (1.03)	23.19 (0.90)	<0.001*
Right anterior chamber depth (SD) [mm]	3.26 (0.34)	3.22 (0.33)	0.23
Right lens thickness (SD) [mm]	3.89 (0.29)	3.90 (0.29)	0.85
Intraocular pressure (SD) [mm Hg]	14.9 (2.3)	15.2 (2.5)	0.095

Differences between groups were analysed using the Student *t* test (continuous variables) and Chi squared test (categorical variables).

* Probability of the null hypothesis <5%.

Discussion

The data presented here show a significant difference in axial length of the eye between

Table 4. Age-specific axial length for urban and rural groups.

	20 to 29 years	30 to 39 years	Difference 95% confidence interval of difference	All ages
Rural (SD)	23.23 (0.81)	23.15 (0.98)	0.08 ($p = 0.46$)	23.19 (0.90)
95% confidence interval	23.09-23.36	22.99-23.31	-0.13 to 0.28	23.08-23.29
Range	21.05-26.76	20.13-29.46		20.13-29.46
Urban (SD)	23.66 (1.13)	23.37 (0.85)	0.28 ($p = 0.03$)	23.53 (1.02)
95% confidence interval	23.47-23.84	23.22-23.53	0.04-0.52	23.41-23.66
Range	20.25-28.33	21.62-27.20		20.25-23.53
Difference	0.43 ($p < 0.001$)	0.23 ($p = 0.048$)		0.35 ($p < 0.001$)
95% confidence interval of difference	0.20-0.65	0.002-0.45		0.19-0.51

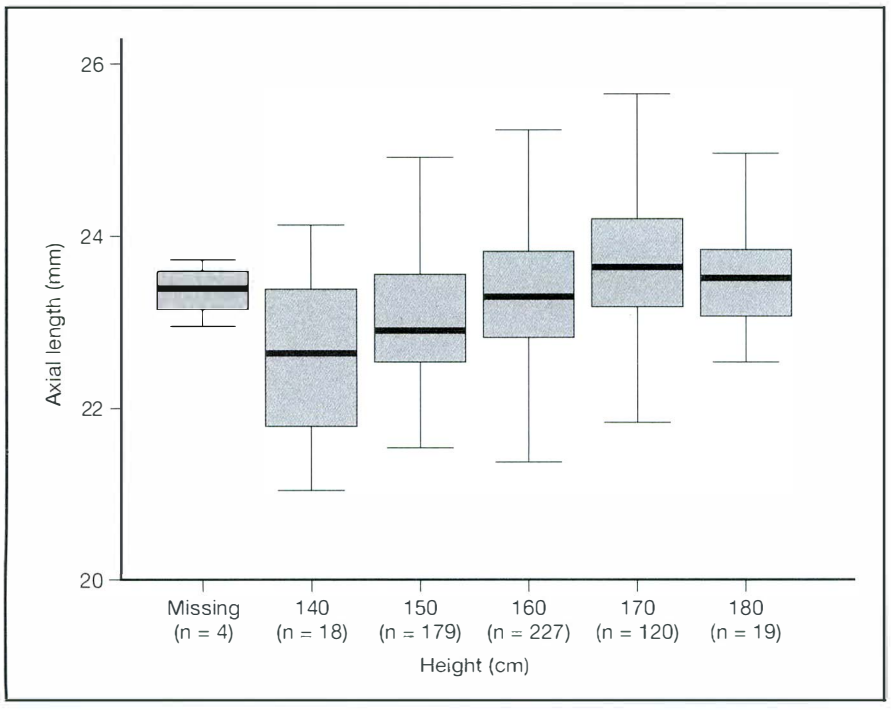

Figure 2. Box and whisker plots of right axial length against height category.*

* The central dark band inside the box represents the median value, with quartiles being indicated by the dimensions of the box. Extreme values are indicated by the whiskers.

young adult people living in urban and rural areas in Mongolia. Furthermore, there is a significant difference in axial length between people aged 20 to 29 years and those aged 30 to 39 years living in Ulaanbaatar city.

These findings may provide insights into the epidemiology of myopia in East Asia. In a previous report, the authors described axial length distribution in people aged 40 years and older living in Hövsgöl and Ömnögobi, areas populated by nomadic herders with some small permanent settlements. In these people, a small increase in mean axial length with age (0.05 mm/decade; $p = 0.03$) was found.[4] When compared with data from Singapore, there is a striking difference. In people aged 40 years and older, mean axial length is much shorter in people aged 70 years and older, compared with those aged 40 to 49 years. Although these findings must be interpreted with some caution if longitudinal trends are inferred, it appears that ocular dimensions in Chinese Singaporean people are changing in a dramatic fashion. These changes are consistent with the well-documented high rates of myopia in younger Taiwanese and Chinese Singaporean people.[1,14] The relative stability of axial length distribution in Mongolian people living in rural areas suggests that they were either not susceptible to the factors responsible for the change observed in Chinese Singaporean people, or had not yet been exposed to these factors.

Genetic and linguistic data suggest that Mongolian and Inuit (Eskimo) people have a common genetic heritage.[15,16] The finding of a pronounced cross-section difference in refractive error and axial length in Alaskan Inuit (longer axial length and more myopic refraction in the young),[17] indicates that people in Mongolia should be susceptible to a similar change, if exposed to the causative factor. A graphical illustration of the data from the present study, a previous survey in rural Mongolia (from 1995 and 1997), and Chinese people in Singapore, illustrates clearly that mean axial length in urban and rural Mongolians appears to be rapidly diverging (Figure 3). City-dwelling Mongolians now have a mean axial length significantly longer than their rural counterparts. It appears that the rate of this divergence of axial length in these 2 groups is approximately similar in magnitude to the cross-sectional changes seen in Chinese Singaporean people.

However, these observations must be interpreted with the understanding that axial length is longer in taller people.[18] This observation has subsequently been made in children.[19] These data support the concept that there is a highly significant relationship between height and axial length. In the multivariate model, differences in height and educational achievement were the 2 identifiable factors that were associated with differences in axial length between people in urban and rural areas. The observed differences are, at least in part, attributable to the urban population being taller. In turn, this is probably a result of improved living standards and nutrition in childhood for city residents.[20] However, the trend is subject to short, medium, and long-term fluctuation in business cycles, attributed to variations in food quality and quantity.[21] In Mongolia, the transition from Soviet-sponsored leadership to democratic independence in 1994 was a time of considerable economic change. The previous decade had seen a gradual winding down of financial and logistic support from the Soviet Union. People undergoing the pubertal growth spurt (age 12 to 15 years) when the country opened its doors to external trade would now be aged 22 to 25 years. The effect of an increase in food quality and quantity at this sensitive period is likely to have had more pronounced effects in the capital city (Ulaanbaatar). The large size of the nation (similar to France, Spain, and Germany combined) and its poor internal transport infrastructure means this influence is unlikely to have had a meaningful effect in areas outside the city.

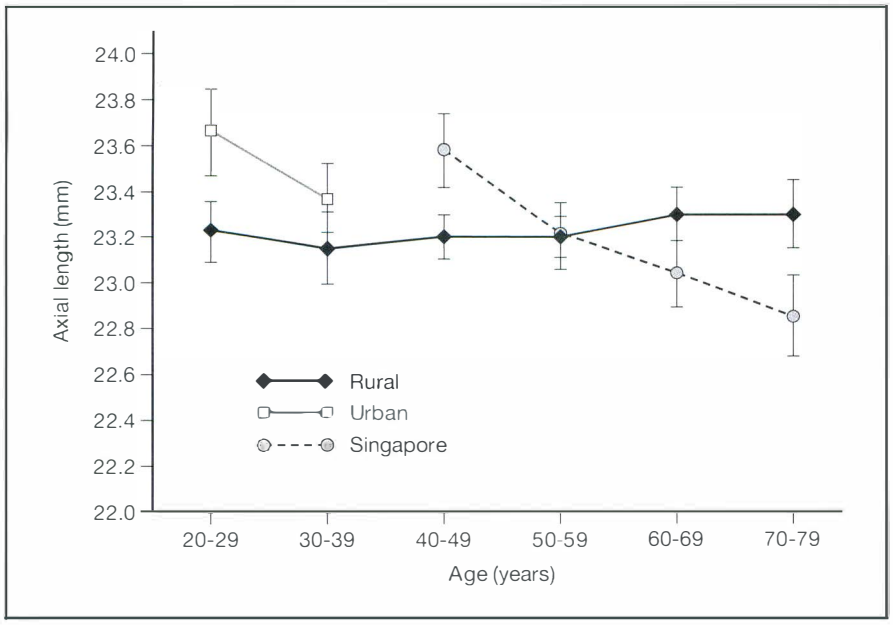

Figure 3. Axial length in urban and rural Mongolian people compared with figures for Chinese Singaporean people.*

* The data for a rural population aged 40 years and older are drawn from previous surveys in 1995 and 1997. From these cross-sectional data, it appears that while the axial length in rural Mongolia remains relatively stable, the young people living in an urban environment are experiencing a significant increase in their mean axial length. The change in urban Mongolian people resembles that seen in the Chinese population of Singapore.

High educational level is probably a surrogate for exposure to near work, which in turn has been linked with increased incidence and progression of myopia in children and adults.[5,22] In a study of Asian people, educational achievement was found to be strongly associated with prevalence and severity of myopia. After adjustment for relative differences in educational achievement, myopia rates were higher in Chinese people than in Indian and Malay people.[23] The Mongolian education system is less intensive than in other East Asian countries, although adult literacy rates are 98% for men and 95% for women.[24] Mongolia has a unique system of residential schools for children of nomadic herders. Currently, all children begin compulsory full-time education between the ages of 6 and 8 years, for a minimum period of 8 years. The association between near-work exposure and myopia in children appears to be greater in younger children than in older children.[5] The relatively late age at induction into formal education may, in part, explain why the prevalence of myopia has been shown to be lower in adult Mongolians than in other East Asian people.[4] It has been observed previously that there may be a socio-economic gradient in myopia prevalence among Chinese children aged 6 and 7 years, with the highest rates found in the affluent Chinese population of Singapore (12.3%) and the lowest rates in people living in rural southern China adjacent to Xiamen City (3.9%). Rates in Xiamen City itself were intermediate between the 2 (9.1%).[25] These data did not identify an independent association between current income and myopia after adjustment for height, previous education, and urban/rural residence.

Previous research in Inuit communities found an association between refractive error and age. Among Alaskan Inuit, the prevalence in people older than 40 years was 1.5%. However, among the 11- to 40-year-old people, 51% had a myopic refraction.[26] This difference was attributed to compulsory, western-style education for the younger members of the community. A study of Canadian Inuit gave similar results.[7] The existing body of data supports the concept that a transition from a subsistence, hunter-gatherer lifestyle to a settled existence with exposure to products and practices from the industrialised world is linked to an increase in the prevalence of myopia. This appears to be predominantly axial myopia. Myopia has a heritable component, as demonstrated by both molecular genetic and twin studies. While it appears that East Asian people have the potential to develop myopia to a greater degree than Europeans, presumably as a consequence of genetic differences, these data further underline the importance of environment and lifestyle in precipitating this change.

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Visual Impairment and Disc Cupping Among Newly Diagnosed Patients with Glaucoma

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Aim: To determine the presenting vision, cup-disc ratio, and referring diagnosis among newly diagnosed patients with glaucoma.
Patients and Methods: *In this cross-sectional observational study, 161 records of patients were selected from the archives at the Eye Clinic, Hospital Pulau Pinang, using systematic sampling. Patients were classified into 3 categories of good vision, low vision, or blind according to best-corrected visual acuity and visual field of the better eye. Cup-disc ratios of patients' worst eye at presentation were classified into severe cupping, moderate cupping, and mild cupping. The referral diagnosis and duration at presentation were analysed.*
Results: *Of the 161 newly diagnosed patients with glaucoma, 8.1% were blind and 14.9% had low vision. One-third of the patients had severe cupping, one-third had moderate cupping, and one-third had mild cupping. Among the patients with severe cupping, 60.5% still had functionally good vision at presentation. Only 5.6% of patients were correctly referred as either having glaucoma or to rule out glaucoma.*
Conclusions: *Detection of glaucoma is difficult and the diagnosis is often missed or delayed. A large number of newly diagnosed patients with glaucoma had severe disc cupping and were blind or had low vision at presentation. Screening of the optic disc by ophthalmoscopy for selected high-risk patients by primary care physicians could increase early detection and control of the disease.*

Key Words: *Glaucoma, Vision, low*

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Introduction

In 2000, the number of people with glaucoma was estimated to be 67 million, of whom 7 million were bilaterally blind.[1] Glaucoma is the second leading cause of blindness worldwide.[1] Despite this, more than 50% of people with glaucoma in developing countries are unaware of the disease.[1] Even in developed countries, 75% to 78% of the population have heard of glaucoma but fewer than 10% are able to correctly define glaucoma.[2,3]

According to the National Eye Survey in Malaysia, glaucoma is the fifth major cause of blindness and low vision.[4] However, it is the third most common cause of irreversible and progressive visual impairment. In the Tanjong Pagar Study, involving a population aged 40 years and older, glaucoma was the largest contributory cause of blindness (60%), followed by cataract and age-related macular degeneration.[5]

Late presentation has been shown to be the major contributory factor for blindness, occurring in 29% to 41% of patients with glaucoma registered as blind.[6] Greater disease severity at the time of diagnosis is associated with subsequent progression to blindness despite treatment.[6] The challenge for managing the disease is to detect glaucoma early in its course, even though the early changes associated with the disease may be difficult to detect. This hospital-based study was performed to determine the presenting vision and cup-disc ratio, and referring diagnosis among newly diagnosed patients with glaucoma.

Patients and Methods

This was a cross-sectional study for which eligible patients were selected according to their ophthalmic records using systematic sampling. The ophthalmic records of patients attending the Eye Clinic, Hospital Pulau Pinang, Pulau Pinang, Malaysia, were arranged in 5 racks in the archive room, with each rack consisting of 10 rows for a total of 50 rows. The records in the first row and every subsequent fifth row were screened. The inclusion criterion was newly diagnosed glaucoma at initial visit to the clinic.

Primary open angle glaucoma (POAG) was characterised by the following features in at least 1 eye: evidence of glaucomatous optic nerve damage; adult onset; normal appearing, open anterior chamber angle; and absence of other known causes of glaucoma.[7] Primary angle closure glaucoma (PACG) was characterised by appositional or synechial closure of the anterior chamber angle caused by pupillary block in the absence of other causes of angle closure.[8] Secondary glaucoma includes lens-induced glaucoma, steroid-induced glaucoma, angle recession glaucoma,uveitic glaucoma, pseudoexfoliative glaucoma, neovascular glaucoma, and iridocorneal endothelium syndrome. Patients with an indistinct cause of glaucoma, including juvenile glaucoma, were classified as 'other'.

The demographic data, visual acuity, cup-disc ratio, gonioscopic findings, intraocular pressure (IOP), and visual field tests were obtained. The visual acuity was based on the baseline best-corrected visual acuity (BCVA) using a standard Snellen chart. Slit lamp examination, including gonioscopy and funduscopy, was performed to measure the cup-disc ratio and glaucomatous optic disc changes and confirmed by 1 of the authors. Applanation tonometry was used to measure the IOP between 9 am and 1 pm. The visual field findings were based on 2 reliable Humphrey perimetry (24-2) tests taken within the first year of follow-up.

The definition of visual impairment was based on the International Classification of Diseases established by the World Health Organization (Tables 1 and 2).[9] In this classification, BCVA was used instead of habitual visual acuity to eliminate any confounding refractive error. Visual acuity and visual fields of the better eyes were emphasised because this reflects the true social, occupational, and daily functional status of the patients.

The vertical cup-disc ratios were determined by slit lamp examination of the fundus with a dilated pupil during the first visit. Severity of the disc cupping was classified as mild when the cup-disc ratio was <0.6, moderate when the cup-disc ratio was 0.6 to <0.8, and severe when the cup-disc ratio was ≥0.8. Patients without documented cup-disc ratios were classified as having missing data.

Results

4318 ocular records were screened and 161 patients with a new diagnosis of glaucoma were included in the study. Their ages ranged from 12 to 87 years (mean age, 60 years; SD, 14 years); 94.4% of patients were older than 40 years at presentation. The majority of the study population were Chinese, followed by Malays and Indians (Table 3), which is representative of the ethnic ratio in the area. The most common type of glaucoma was POAG, followed by secondary glaucoma, PACG, normal tension glaucoma (NTG), and others. There was no sex predilection in the study population or among the various types of glaucoma.

Thirteen patients (8.1%) presented with bilateral blindness and 24 (14.9%) presented with low vision (Figure 1). Of the 161 patients, 75.8% had near-normal vision in at least 1 eye at diagnosis. However, 8 of 92 patients (8.7%) with open angle glaucoma were bilaterally blind. Of the 13 patients who were blind, 7 had POAG (53.4%), 4 (30.7%) had secondary glaucoma, 1 had NTG, and 1 had PACG. Three of the patients with bilateral blindness had concomitant significant cataract, 2 had macular disease, 1 had bilateral aniridia, and 1 had unilateral phthisical bulbi (Figure 2). The remaining patients had no concomitant ocular disease. The blindness rate according to type of glaucoma was 9.9% for POAG, with 4.7% having NTG, 10.3% having secondary glaucoma, and 3.4% having PACG.

Among the 24 patients presenting with low vision, 41.7% had POAG, 29.2% had secondary glaucoma, 16.7% had PACG, and 12.4% had NTG. Twelve of the patients with low vision had no concomitant ocular

Table 1. Visual impairment according to the International Classification of Diseases.[9]

Category of visual impairment	Visual acuity with best possible correction	
	Maximum less than	Minimum equal to or better than
1	6/18	6/60
2	6/60	3/60
3	3/60	1/60 (counting fingers 1 m)
4	1/60 (counting fingers 1 m)	Light perception
5	No light perception	
9	Undetermined or unspecified	

Table 2. Definition of vision adapted from the International Classification of Diseases.

Vision	Definition
Good/near normal vision	Visual acuity of 6/18 or better at least in 1 of the eyes (No visual impairment in either eye) Or ICD-10 classification: H54.4 (visual impairment category 3,4, or 5 in 1 eye with normal vision in other eye) H54.5 (visual impairment category 1 or 2 in 1 eye with normal vision in other eye) H54.6 (visual impairment category 9 in 1 eye with normal vision in other eye)
Low vision	Better eye's best-corrected visual acuity worse than 6/18 but better than or equal to 3/60 or better eye's visual field less than 10° around the fixation Or ICD-10 classification: H54.1 (visual impairment category 3,4, or 5 in 1 eye with category 1 or 2 in other eye) H54.2 (visual impairment category 1or 2 in both eyes)
Blind	Better eye's best corrected visual acuity worse than 3/60 or better eye's visual field less than 5° around central fixation Or ICD-10 classification: H54.0 (visual impairment category 3,4, or 5 in both eyes)
Unqualified or unspecified vision	Undetermined or unspecified Or ICD-10 classification: H54.3 (visual impairment category 9 in both eyes) H54.7 (visual impairment category 9 NOS)

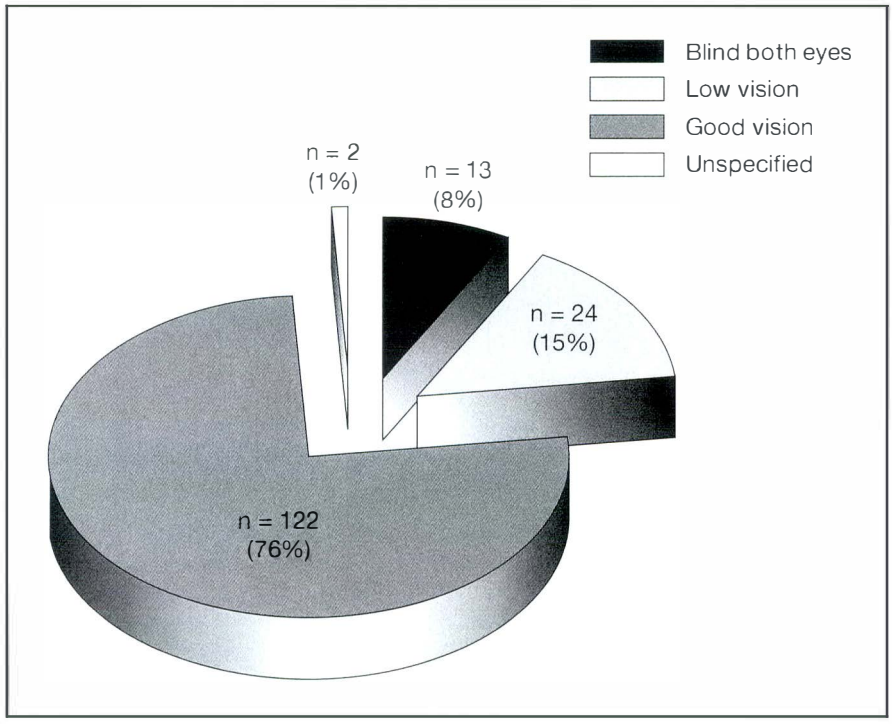
Table 3. Distribution of vision at presentation based on sex, race, glaucoma type, and severity of cup-disc ratio

	Number of patients (%)				
	Good vision	Low vision in better eye	Bilateral blindness	Unspecified vision	Total (% of total)
Sex					
Male	65 (78.3)	11 (13.3)	7 (8.4)	—	83 (51.6)
Female	57 (73.1)	13 (16.7)	6 (7.7)	2 (2.5)	78 (48.4)
Race					
Chinese	75 (72.8)	18 (17.5)	9 (8.7)	1 (1.0)	103 (64.0)
Malay	31 (79.5)	5 (12.8)	2 (5.1)	1 (2.6)	39 (24.2)
Indian	13 (81.2)	1 (6.3)	2 (12.5)	—	16 (9.9)
Other	3 (100)	—	—	—	3 (1.9)
Glaucoma					
Primary open angle glaucoma	54 (76.0)	10 (14.1)	7 (9.9)	—	71 (44.2)
Normal tension glaucoma	17 (81.0)	3 (14.3)	1 (4.7)	—	21 (13.0)
Secondary glaucoma	27 (69.2)	7 (17.9)	4 (10.3)	1 (2.6)	39 (24.2)
Primary angle closure glaucoma	23 (79.4)	4 (13.8)	1 (3.4)	1 (3.4)	29 (18.0)
Other	1 (100)	—	—	—	1 (0.6)
Disc cupping					
Mild	41 (87.2)	6 (12.8)	—	—	47 (29.2)
Moderate	32 (84.2)	5 (13.2)	1 (2.6)	—	38 (23.6)
Severe	26 (60.5)	8 (18.6)	9 (20.9)	—	43 (26.7)
Undetermined	23 (69.7)	5 (15.1)	3 (9.1)	2 (6.1)	33 (20.5)
Total	122 (75.8)	24 (14.9)	13 (8.1)	2 (1.2)	161 (100)

conditions, 7 had significant cataract, and 1 each had macular disease, chronic uveitis, amblyopic eye, subluxated lens, and optic atrophy (Figure 2). The low vision rate according to type of glaucoma was 14.1% for POAG, 14.2% for NTG, 17.9% for secondary glaucoma, and 13.7% for PACG.

The distribution of the severity of disc cupping was evenly divided among the mild, moderate, and severe disc-cupping classification. The cup-disc ratios of 128 patients were analysed (Table 4). Of the 95 patients for whom data was documented, 36.7% had mild cupping, 29.7% had moderate cupping, and 33.5% had severe cupping. The cup-disc ratio was not available for 33 patients and this was categorised as missing data.

Figure 1. Visual impairment among newly diagnosed patients with glaucoma.

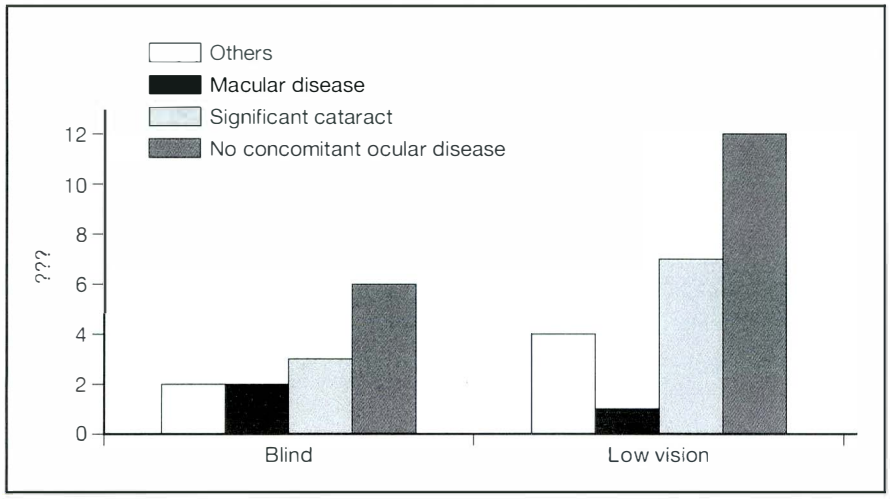


There was no significant correlation between race, age, and sex with the severity of cupping. Most patients were referred from outpatient departments (65.0%) and hospital wards (10.5%). The mean IOP at presentation was highest among patients with PACG (43.7 mm Hg; SD, 15.0 mm Hg) followed by those with secondary glaucoma (38.0 mm Hg; SD, 14.0 mm Hg), POAG (29.7 mm Hg; SD, 10.0 mm Hg), and NTG (17.2 mm Hg; SD, 2.9 mm Hg).

Only 14.9% of the patients were originally referred with a diagnosis of glaucoma (Table 5). For the majority of the patients, glaucoma was detected during routine eye examination such as for screening for diabetic retinopathy. Only 3 patients had glaucoma detected by an optometrist.

Of the 43 patients with severe cupping at presentation, most were not detected by primary care physicians and only 4 patients (9.3%) were initially referred as having glaucoma or to rule out glaucoma. Nearly one-third of the patients (27.3%) were referred to the clinic without a provisional diagnosis.

Figure 2. Concomitant ocular disease in newly diagnosed patients with glaucoma.



Discussion

This study demonstrated that glaucoma is mainly a disease of the elderly population (mean age, 60 years). Glaucoma is a serious irreversible optic neuropathy that is commonly overlooked as a diagnosis. Even though most of the patients in this study had relatively good vision at presentation, a substantial percentage were irreversibly blind as a result of late diagnosis.

The majority of patients with glaucoma remain active at the time of presentation. Patients are asymptomatic during the early phase of the disease due to the gradual onset, peripheral visual field loss instead of central visual field loss, and frequently asymmetrical involvement. The visual field loss in the glaucomatous eye is compensated for by the fellow eye. Such patients would have severely limited vision should the better eye start to deteriorate due to glaucoma or other causes.

The rate of blindness at first presentation among the patients with POAG in this study was high at approximately 10% compared with the prevalence of blindness due to glaucoma reported in western countries. The rate of bilateral blindness due to POAG in western countries is reported to be 3% to 5%.[6] Nevertheless, the incidence of blindness among the newly diagnosed patients with glaucoma in this study is considerably lower than that in China; 18.1% of the 5.2 million patients with glaucoma in China are bilaterally blind.[10] The higher prevalence of PACG among the Chinese population is believed to be the most important contributory factor. The percentage of PACG and blindness due to PACG in the Malaysian population is not as high as in China. The ratio of PACG to POAG in this study was 1:2.3, which is the reverse of the ratio in China of 2:1. This figure is supported by a hospital-based study in Malaysia in which the ratio was found to be 1:1.5.[11]

This study found a small but significant number of patients (9 of 161) with severe disc cupping resulting in blindness at the initial visit. However, there was no correlation for the severity of cup-disc ratio with the extent of the visual impairment. Even among patients with severe disc cupping, more than 60% had good vision at presentation. A more alarming finding is that one-third of the patients had severe disc cupping at presentation. Furthermore, disc cupping had only been detected by the referring doctor in less than 10% of these patients, suggesting that glaucoma is under-diagnosed.

These findings demonstrate that the detection of glaucoma is mostly accidental. Primary care doctors play a role in the detection of glaucoma in Malaysia, unlike in the UK where optometrists play a role in the early detection of glaucoma.[12] However, regardless of who is responsible for the detection of glaucoma, the challenge is to diagnose the disease as early as possible to prevent blindness in the community.

Optic disc changes precede visual field changes by several years.[13] There may be nerve fibre loss of up to 40% before conventional perimetry is able to demonstrate visual field defects.[14] Thus, the morphology

Table 4. Severity of cup-disc ratio in patients with newly diagnosed glaucoma based on sex, race, and types of glaucoma.

	Number of patients (%)			Total (n = 128)
	Mild cupping [n = 47]	Moderate Cupping [n = 38]	Severe Cupping [n = 43]	
Sex				
Male	23 (33.8)	17 (25.0)	28 (41.2)	68
Female	24 (40.0)	21 (35.0)	15 (25.0)	60
Race				
Chinese	34 (39.5)	23 (26.8)	29 (33.7)	86
Malay	9 (33.4)	9 (33.3)	9 (33.3)	27
Indian	3 (23.0)	5 (38.5)	5 (38.5)	13
Other	1 (50.0)	1 (50.0)	—	2
Glaucoma				
Primary open angle glaucoma	23 (34.3)	21 (31.4)	23 (34.3)	67
Normal tension glaucoma	3 (14.3)	11 (52.4)	7 (33.3)	21
Secondary glaucoma	12 (60.0)	3 (15.0)	5 (25.0)	20
Primary angle closure glaucoma	9 (47.4)	2 (10.5)	8 (42.1)	19
Other		1 (100)		1

Table 5. Referring diagnoses and severity of disc cupping.

Referring diagnosis	Mild cupping	Moderate cupping	Severe cupping	Missing	Total
Cataract	12	18	20	9	59 (36.7)
Glaucoma	11	4	4	5	24 (14.9)
No diagnosis	10	10	8	16	44 (27.3)
Screening for diabetic retinopathy	4	2	3	—	25 (15.5)
Other	10	4	8	3	9 (5.6)
Total	47	38	43	33	161 (100)

of the optic disc is the gold standard of glaucoma screening as well as in staging the disease.[15] At least two-thirds of patients with glaucoma in this study had already developed moderate to severe cupping of ≥0.6 at presentation. Primary care physicians need to acquire the ability to detect optic disc changes using direct ophthalmoscopy and to refer appropriately.

Widespread glaucoma screening is not cost-effective because the predictive value of a positive test is unacceptably low.[16] Some policymakers recommend that glaucoma screening should be limited to high-risk groups.[17] With the recent knowledge gained from large population-based studies,[18-26] more precise identification of high-risk populations would invariably increase the cost-effectiveness of screening. Populations at high risk for glaucoma are those with a family history of glaucoma; thyroid disease, diabetes, or hypertension; high myopia; older age; and difference in disc cupping between both eyes of >0.2.[6,18-28]

Glaucoma has a higher prevalence in the older population.[21] As the ageing population increases in Malaysia, the increasing prevalence of blindness and advanced glaucoma is inevitable. Appropriate measures to facilitate early detection and referral of high-risk patients are timely to ensure a healthier ageing population.

In conclusion, the detection and diagnosis of glaucoma is difficult and the disease often remains undetected. Significant numbers of patients with glaucoma are diagnosed late in the course of the disease because of the quiescent presentation of the disease and a low index of suspicion among health care providers. Screening of the optic disc for high-risk patients at primary care facilities could result in early detection and intervention.

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Relationship between Retinal Vein Occlusion and Axial Length of the Eye

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Aim: To determine the association between the axial length of the eye and retinal vein occlusion (branch retinal vein occlusion and central retinal vein occlusion).

Patients and Methods: Eighteen patients with central retinal vein occlusion and hemi-retinal vein occlusion and 18 patients with branch retinal vein occlusion were enrolled in this study. Eighteen patients who were referred for cataract extraction were enrolled as a control group. Axial length was measured in both eyes using A-scan biometry. The data were analysed using the Student t test.

Results: The axial length differed significantly between affected and fellow eyes in the central retinal vein occlusion group. This difference was less in the branch retinal vein occlusion group but was still significant. The axial length of the non-affected eyes of both the central retinal vein occlusion and the branch retinal vein occlusion groups was less than the axial length of eyes of the control group. In the branch retinal vein occlusion group, the mean axial length of the affected eyes was 22.52 mm and that of the fellow eyes was 22.77 mm. In the central retinal vein occlusion group, the mean axial length of the affected eyes was 22.71 mm and that of the fellow eyes was 23.23 mm. In the control group, the mean axial length was 23.77 mm.

Conclusions: The affected eyes in both groups of retinal vein occlusion had a smaller mean axial length than the fellow eyes, and the fellow eyes of patients with retinal vein occlusion had a smaller mean axial length than the control eyes. Short axial length may be a local risk factor for retinal vein occlusion.

Key Word: Retinal vein occlusion

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Introduction

Retinal vein occlusion (RVO) is the most common form of retinal vascular disease after diabetic retinopathy.[1] Hyperopia is a risk factor for retinal vein occlusion.[2,3] In previous studies, hyperopia was measured in terms of refractive error. Refractive error can be affected by age-related changes such as the effect of nuclear sclerosis on the power of the crystalline lens and therefore may not accurately represent hyperopia.

The aim of this study was to determine whether there is a difference between the axial length of eyes with RVO and control eyes with no RVO, and whether the axial length is a local risk factor in the pathogenesis of RVO.

Patients and Methods

From December 2000 to May 2001, thirty six patients who were referred to the Shiraz Poustchi Eye Clinic, Shiraz, Iran, with clinical and fluorescein angiographic evidence of RVO were enrolled in the study. In addition to routine history taking and eye examination, the axial lengths of both eyes were measured by a person who was unaware of the patients' diagnosis. The measurement was similar to the intraocular lens (IOL) measurement for the control group and was performed by means of Biovision Class 1 Type B A-Scan ultrasonography (Biovision International, Clermont-Serand, France).

The control group comprised 18 patients who had undergone biometry for IOL calculation who were age- and sex-matched with the study patients.

All monocular patients and those with previously operated eyes were excluded from the study. The data were analysed first by descriptive statistics for the study and control groups. Axial length difference between the 2 eyes in patients with central retinal vein occlusion (CRVO) and branch retinal vein occlusion (BRVO) was assessed using the Student paired *t* test. The axial length of the non-affected eyes in each group was compared with the control group using independent *t* test. Statistical analysis was conducted using the Statistical Package for the Social Sciences software. Significance was considered to be $p < 0.05$.

Results

There were 18 patients in the control group (9 men and 9 women; mean age, 64 years; range, 40 to 78 years). Ten patients had hypertension and 1 had diabetes; no patients had glaucoma.

Eighteen patients had BRVO (9 men and 9 women; mean age, 61 years; range, 29 to 76 years). In 15 patients, the artery crossed over the vein and in the remaining 3 patients, the status was unclear. Eleven patients had supratemporal branch occlusion and 7 had infratemporal branch occlusion. Fourteen patients had hypertension and 2 patients had diabetes; 1 patient had glaucoma. The mean axial length of the affected eyes was 22.52 mm (SD, 0.72 mm) and the mean axial length of the healthy fellow eyes was 22.77 mm (SD, 0.64 mm);

this difference was statistically significant (t = -2.13; p = 0.048) [Figure 1]. In 3 patients, the axial length of the affected eye was longer than that of the fellow eye; one of these patients was the youngest patient at 29 years. The mean axial length of the control eyes was 23.77 mm (SD, 1.02 mm) and the mean axial length of the healthy fellow eyes in the patients with BRVO group was 22.77 mm (SD, 0.64 mm); this difference was statistically significant (p = 0.017). The affected eyes had a shorter mean axial length than the non-affected fellow eyes.

Fifteen patients had CRVO and 3 had hemi-RVO (10 men and 8 women). In the CRVO group, the mean age was 60 years (range, 35 to 75 years). Thirteen patients had hypertension and 3 patients had diabetes; no patients had glaucoma. The mean axial length of the affected eyes was 22.71 mm (SD, 0.85 mm) and the mean axial length of the healthy eyes was 23.23 mm (SD, 0.71 mm) [Figure 2]; this difference was statistically significant (t = -3.10; p = 0.007). Only 1 patient had a longer mean axial length in the affected eye. The axial length of the healthy fellow eyes in the CRVO group was less than the axial length of the control group (p = 0.04).

Discussion

Many risk factors that may predispose patients to RVO, including systemic hypertension, diabetes, atherosclerosis, and hyperopia, have been reported. BRVO usually occurs at an arteriovenous crossing, so the possibility of an underlying inflammatory condition should be considered when it does not occur at an arteriovenous crossing. On the other hand, CRVO may be associated with the use of oral contraceptives and diuretics, blood dyscrasias, dysproteinaemia, and inflammation.[2-4] The association between axial length of the eye and BRVO has been investigated in affected eyes, using the fellow as a control group.[5,6] The results were inconclusive. This study compared axial

Figure 1. Axial length in eyes with branch retinal vein occlusion, fellow eyes, and controls.

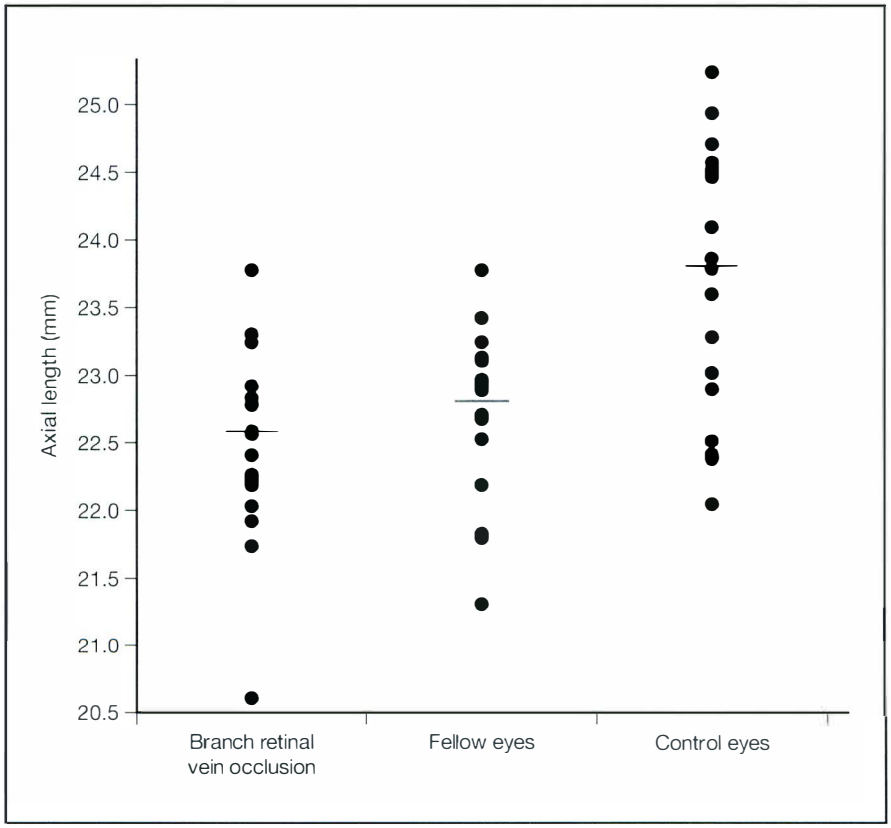
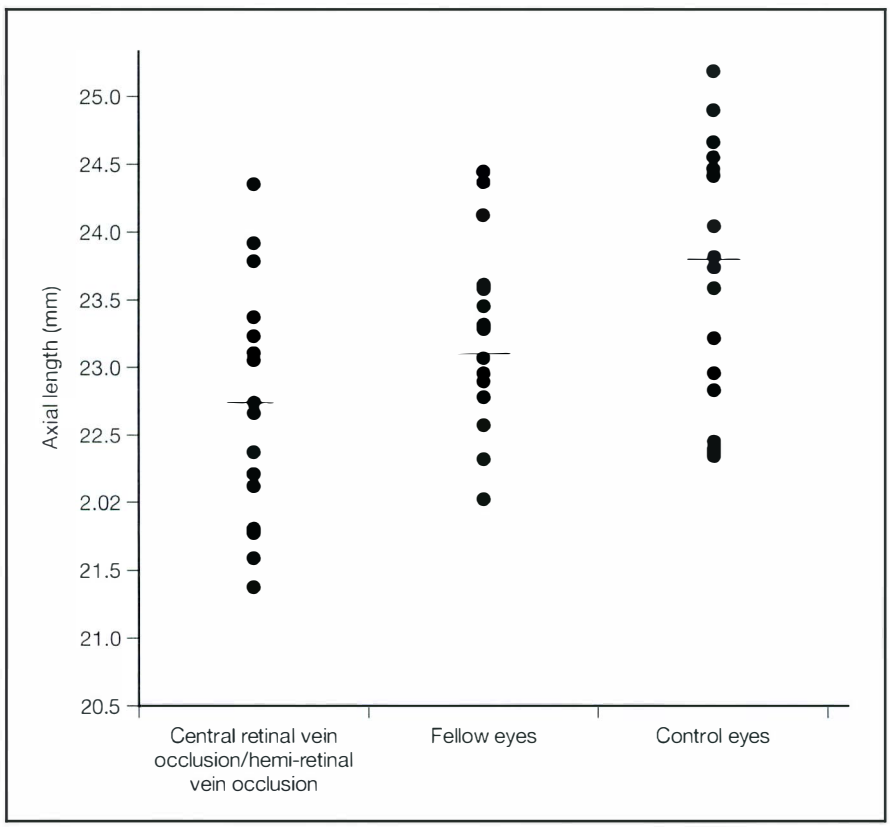


Figure 2. Axial length in eyes with central retinal vein occlusion, fellow eyes, and controls.



length instead of the amount of hyperopia because the axial length does not change with age. The axial length of affected and fellow eyes of patients with RVO were compared with the axial length of healthy eyes, thus avoiding the probable effect of macular oedema and duration of disease on axial length.

In this study, the mean difference in axial length between the affected and fellow eyes was 0.52 mm in the CRVO group and 0.25 mm in the BRVO group. The larger difference in the CRVO group may be associated with macular oedema, which is more pronounced in CRVO. For this reason, the healthy eyes of each group were compared with the control eyes. The mean axial length of the control eyes was 23.77 mm, which is close to the mean axial length of 23.65 mm reported for the general population.[7]

In the fellow eyes without macular oedema in the BRVO group, the mean axial length was 22.77 mm, which was 1 mm shorter than the mean axial length in the control group, suggesting that smaller eyes are at high risk for BRVO. This result was similar to that of a case control study that revealed more hyperopia in eyes with BRVO.[8] However, in this study, hyperopia, which can be induced by lens ageing, was replaced with axial length, which is more stable during the ageing process.

Arteriovenous crossing sheatotomy for surgical decompression of BRVO addresses the theoretic pathogenic mechanism of BRVO and is a technically feasible procedure that can re-establish retinal blood flow.[9] It is possible that, in a small eye, a vein that is compressed between the artery and the retina is prone to focal narrowing, turbulence, and venous stasis.[10,11] Short axial length is a risk factor for BRVO and the possibility of involvement of the other eye is approximately 12% over 4 years.[12]

A high incidence of hyperopia has been reported to be associated with CRVO.[13] In this study, the mean axial length of the healthy fellow eyes in the CRVO group was 23.23 mm — only 0.5 mm shorter than the mean axial length of the control eyes. Thus, there is less correlation with axial length for CRVO than for BRVO. This may be due to a greater association of CRVO with systemic risk factors. Biochemical abnormalities and hyperviscosity states have been described as systemic risk factors for CRVO. CRVO occurring in patients younger than 50 years may also be related to resistance to activated protein C (deficiency of factor V Leiden), an important factor in the anticoagulation system.[14]

In this study, 2 patients in the CRVO group and 1 in the BRVO group were younger than 40 years. Their mean axial length was 23.4 mm. Interestingly, in 2 patients, the affected eyes were longer than the fellow eyes. It is possible that axial length had an insignificant role, if any, in these patients.

Small hyperopic eyes may have small optic discs, so they are susceptible to CRVO because of neurovascular compression within the confined space at the optic disc. Surgical decompression of CRVO via radial optic neurotomy, as described by Opremcak et al, may be based on this hypothesis.[15]

Previous studies only compared the 2 eyes of the same patient or investigated the amount of hyperopia as possible risk factors, so some controversial issues do exist.[5,6] This study was designed to resolve these issues and also to cancel the effect of macular oedema by comparison of healthy eyes in patients with RVO with healthy eyes of a control population. However, this study is limited by its small sample size.

It appears that short axial length is a local risk factor for RVO, especially BRVO, in conjunction with other systemic risk factors.

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Central Retinal Vein Occlusion: Role of Axial Length

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Aim: To evaluate role of axial length in patients with central retinal vein occlusion.
Patients and Methods: *Thirty patients with unilateral central retinal vein occlusion (19 ischaemic and 11 non-ischaemic) were enrolled in the study. Twenty nine individuals who matched the study group for age, sex, hypertension, and diabetes mellitus status were enrolled as a control group. Patients with macular oedema were excluded. The axial lengths of the affected and fellow eyes of patients with central retinal vein occlusion and the controls were measured using A-scan ultrasonography.*
Results: *The mean axial length of the affected eyes was not statistically significantly shorter than the mean axial length of the unaffected fellow eyes in the group with central retinal vein occlusion (22.88 mm and 22.90 mm, respectively). There was no statistically significant difference in mean axial length between the ischaemic and non-ischaemic subgroups and the unaffected eyes. There was no statistically significant difference in mean axial length between the eyes with central retinal vein occlusion and the control eyes (22.88 mm and 23.11 mm, respectively). There was no difference in mean axial length between the ischaemic and non-ischaemic subgroups and the control eyes.*
Conclusions: *This study did not demonstrate a significantly shorter axial length in eyes with central retinal vein occlusion. Axial length as a measurement of hyperopia may not be a risk factor for central retinal vein occlusion if patients with macular oedema are excluded.*

Key Words: *Hyperopia, Ischemia, Retinal vein occlusion*

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Introduction

Retinal vein occlusion (RVO) is the second most common retinal vasculopathy, after diabetic retinopathy.[1] Many systemic and local factors that contribute to thrombus formation can predispose to the development of RVO, including hypertension, diabetes mellitus, hyperviscosity, hyperlipidaemia, primary open angle glaucoma (POAG), and hyperopia.[2,3]

The association between hyperopic refractive error and RVO has been demonstrated.[4-8] In most of these studies, hyperopia was measured in terms of refractive errors. As age-related lens changes may interfere with refractive error, recent studies have focused on the relation of axial length as a measurement of hyperopia with RVO.[4,9-15] However, there is no general agreement on the role of axial length as a predisposing factor for RVO.[4] It has been proposed that the significant difference between the affected eyes and contralateral unaffected eyes in some studies may be due to the effect of macular oedema on the ocular axial length measurements.[13]

The primary goal of this case-control study was to determine the association of axial length as a measurement of hyperopia and central retinal vein occlusion (CRVO) rather than refractive error and CRVO in patients without macular oedema.

Patients and Methods

Thirty patients with CRVO who were admitted to Farabi Eye Hospital, Tehran, Iran, between July 2002 and July 2003 were enrolled in the study. The mean duration of symptoms was 3.6 months (range, 1 to 6 months).

All patients underwent systemic and ocular examination. Systemic examination included fasting blood glucose level and systemic blood pressure measurement with cardiovascular examination. Complete ophthalmic examination included intraocular pressure (IOP) measurement, indirect ophthalmoscopy, gonioscopy, and fundus fluorescein angiography. Exclusion criteria were persistent macular oedema, retinal detachment, eye trauma, intraocular inflammation, tumour, or previous ocular surgery. Based on ocular examination and fluorescein angiography, the patients in the CRVO group were further divided into 2 subgroups of ischaemic CRVO (19 patients) and non-ischaemic CRVO (11 patients). Ischaemic CRVO was defined as: visual acuity <20/200, afferent papillary defect ≥2+, and more than 10 disc diameter capillary non-perfusion in fluorescein angiography.

Ocular axial lengths were measured by A-scan ultrasonography (Nidek Echoscan US-2500, Tokyo, Japan) with a sound velocity of 1550 m/second; 6 consecutive measurements were taken by the manual direct contact technique. The same person, who was not aware of the eye condition, did all the measurements.

The control group consisted of 29 patients from the outpatient clinic matched for age, sex, hypertension, and diabetes. Exclusion criteria were the same for the control group except for the presence or absence of CRVO. Right eyes were used for statistical analyses.

Differences between the demographic data of the patient and control groups were assessed by the Chi squared test. The axial length and refraction of the affected eyes of patients with CRVO were compared with the unaffected healthy eyes and with the control eyes. Statistical analyses were performed by paired *t* and Student *t* tests. Statistical significance was considered to be $p < 0.05$.

Results

The characteristics of patients in the CRVO and control groups are shown in Table 1. No significant differences were found between the groups for age, sex, and risk factors, including diabetes, hypertension, and mean IOP. There was no significant difference in mean refractive error (spherical equivalent) between the affected eyes (1.56 D; SD, 2.66 D) and the unaffected eyes (1.52 D; SD, 2.56 D), and between the affected eyes and control eyes (1.11 D; SD, 2.73 D).

Table 2 shows the mean axial length measurements for eyes with CRVO, including the ischaemic and non-ischaemic subgroups, and the control eyes. Although the mean axial length of the eyes with CRVO was shorter than the mean axial length of unaffected fellow and control eyes, the difference was not statistically significant between the groups. The mean axial length did not differ between the ischaemic and non-ischaemic subgroups and the unaffected and control eyes (Table 2).

Discussion

There are many risk factors for RVO, including hypertension, diabetes mellitus, arteriosclerosis, POAG, hypermetropia, hyperlipidaemia, hyperviscosity, increase in fibrinogen and coagulation factors, and deficiencies of proteins C and S.[12,16] In this study, 56.7% of the patients had hypertension and 23.3% had diabetes mellitus, which corresponds with reports in the literature.[17,18]

In this study, although the mean axial length of the eyes with CRVO was shorter than the mean axial length of the unaffected fellow eyes and the control eyes, the difference was not significant between the groups. The difference between the mean axial length of the affected eyes in the ischaemic and non-ischaemic subgroups and the unaffected fellow eyes and the control eyes was not significant.

Thrombus formation has been observed at or near the lamina cribrosa in eyes with CRVO in histopathological studies.[18,19] Theoretically, eyes with shorter axial length as a measure of axial hypermetropia may be predisposed to greater crowding of the central retinal vein and artery at the lamina cribrosa, and are therefore more likely to develop CRVO.[4] Although hypermetropia has been reported to be an ocular risk factor for RVO in some studies,[5-8] it is more accurate to look at axial length as a measurement of hyperopia.[4,9-11,14,15] Refractive error resulting from age-related lens changes may cause myopic shift and may not accurately reflect true hyperopia.[14] Brown et al,[12] Cekic et al,[4] Tsai et al,[10] and Shi and Chen[9] found significantly shorter axial length in affected eyes of patients with CRVO compared with control eyes. However, this difference was not observed between affected and unaffected fellow eyes. Ariturk et al reported significantly shorter axial length in affected eyes of patients with CRVO compared with both unaffected fellow eyes and control eyes.[13] The axial length of unaffected fellow eyes was also found to be significantly shorter than the axial length of control eyes. In addition, some studies have found significantly shorter axial length in eyes with RVO compared with control eyes,[5,13,15] although others have not found a difference.[14]

It is possible that differences between the studies may play a part and depends on the selection of the control group and the methods of statistical analysis. Differences in demographic characteristics of patients with CRVO in these studies may be a contributing factor. For example, this study did not include any patients with glaucoma, which may have affected the results. Significant differences between the axial length in affected eyes and unaffected fellow eyes in some studies may be due to

Table 1. Characteristics of patients with central retinal vein occlusion and control patients.

	Central retinal vein occlusion	Control	p Value
Number of patients	30	29	
Mean age (SD) [years]	61.9 (9.7)	64.8 (9.6)	0.28*
Sex (female/male)	14/16	14/15	0.92†
Hypertension (number of patients)	16	17	0.94†
Diabetes (number of patients)	6	7	0.81†
Mean intraocular pressure (SD) [mm Hg]	16.90 (3.44)	16.75 (3.76)	0.52*

* Student *t* test.
† Chi squared test.

Table 2. Comparison of axial length in eyes with central retinal vein occlusion, by ischaemic and non-ischaemic subgroups, unaffected fellow eyes, and control eyes.

	Affected eyes Mean (SD) [mm]	Unaffected eyes Mean (SD) [mm]	p Value*	Control eyes Mean (SD) [mm]	p Value†
Central retinal vein occlusion	22.88 (1.02)	22.90 (1.06)	0.97	23.11 (0.86)	0.35
Ischaemic central retinal vein occlusion	22.26 (1.12)	22.69 (1.16)	0.69	23.11 (0.86)	0.12
Non-ischaemic central retinal vein occlusion	23.26 (0.72)	23.21 (0.79)	0.55	23.11 (0.86)	0.61

* Paired *t* test
† Student *t* test

the effect of macular oedema on the axial length measurements.[13] In this study, patients with macular oedema were excluded. Nevertheless, the lack of statistical significance may be due to the small sample size.

Cekic et al suggested that the ratio between axial length and area of the lamina cribrosa might explain variations in the results of the previous studies and recommended quantitative comparison of both the axial length and the area of the lamina cribrosa and comparing the differences between eyes with RVO and unaffected fellow eyes.[4]

This study did not evaluate other possible systemic risk factors such as hyperlipidaemia, blood hyperviscosity, increase in erythrocyte sedimentation rate, and coagulation factors.[20] Moreover, as patients and controls were matched for age, sex, hypertension, and diabetes, the association of these variables with CRVO could not be studied.

It has been demonstrated that POAG or elevated IOP is a significant risk factor for CRVO in some studies, but not in others.[4-7] As there were no patients with POAG or elevated IOP in this study, the association of POAG or high IOP with retinal vein occlusion could not be examined.

There is no uniformity of risk factors in the studies of CRVO. In this study, although the mean axial length of the eyes with CRVO was shorter than the mean axial length of unaffected fellow eyes and control eyes, the differences were not significant. This was also the case for comparison of the ischaemic and non-ischaemic subgroups with the unaffected fellow eyes and control eyes. Thus, this study shows that hyperopia as measured by axial length may not be a risk factor for CRVO if patients with macular oedema are excluded. Further studies with a larger number of patients are needed to establish this hypothesis.

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A Discussion of Central Retinal Vein Occlusion and Axial Length

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In this issue of Asian Journal of OPHTHALMOLOGY, there are 2 fascinating studies of the relationship between retinal venous occlusion and axial length. The article by Mehdizadeh et al concluded that the axial length in branch and central retinal vein occlusion (BRVO and CRVO) was shorter than that in a control group.[1] However, the work by Mirshahi et al was case-control in design and suggested that the axial length in the CRVO group was no different from that in a control group.[2] We are thus placed in the interesting position of making some sense of these differing conclusions.

CRVO is a cause of devastating visual loss with a 5-year incidence of 0.2% in the older age group.[3] The landmark study of risk factors for CRVO was a case-control study examining 258 patients with CRVO and 1142 carefully matched controls.[4] The authors found controls who matched the patients for point of entry into the specialty or general clinic, as well as roughly matching the age, race, and sex. The major risk factors identified were elevated intraocular pressure (IOP), glaucoma history, and elevated blood pressure.

The selection of controls for a case-control study is never easy. Finding the controls can be more difficult than the patients because one needs to select controls matched for characteristics that may be associated with the variable(s) under investigation. Mehdizadeh's group used 18 controls taken from a cataract assessment clinic having measurements for intraocular lens calculation. The mean age was 64 years and the mean axial length was 23.77 mm.[1] A recent study examining the relationship between age and axial length in American patients undergoing cataract surgery demonstrated a longer axial length in younger patients, with a mean axial length of 24.1 mm in the 60- to 70-year-old patients.[5] Additionally, patients with any degree of myopia are more likely to undergo cataract surgery than those with emmetropia or hyperopia.[6] Hence, it is possible that the inclusion of patients undergoing cataract surgery induced an axial length bias, which may also account for some of the axial length difference in the control groups from the 2 articles (23.11 mm vs 23.77 mm).

As both authors say, there is some evidence of a relationship between refractive error and CRVO, but this is also contentious and the results of studies are inconsistent.[4] If we suppose that a relationship between axial length and CRVO does exist, then we need to ask how this could be so. There is a relationship between optic disc size and axial length.[7] However, there is no proven relationship between optic disc size and CRVO.[8] There is a relationship between glaucoma, IOP, and myopia,[9] which could explain some of any relationship between axial length and CRVO.

Of all the putative factors possibly implicated with CRVO, glaucoma and elevated IOP have the greatest odds ratio.[4] Spontaneous venous pulsation is less frequent in glaucoma, and a greater ophthalmo-dynametric force is required for its induction with more severe field loss.[10,11] The latter is a likely index of venous resistance along the hemi- and central retinal veins in the optic nerve head region,[12] the implication being that this segment of retinal vein may narrow in glaucoma. Why this should occur is not clear. However, we do know that the pressure gradient along the central retinal vein in the lamina cribrosa region may be high,[13,14] and with lamina thinning from glaucoma,[15] will rise further. This may lead to elevated shear stress within the vein and result in endothelial cell proliferation and change,[16] with resultant luminal narrowing. This is supported by histological evidence of endothelial cell proliferation from patients with CRVO.[17] Histopathological studies of CRVO have usually examined eyes removed due to neovascular glaucoma, and so one cannot be certain what changes occurred leading up to the venous occlusion, at the time of the occlusion, or as a neovascular sequela.

Sadly, our ability to treat CRVO and BRVO is very weak. It is difficult to know the place for direct optic disc surgery at present.[18] Laser anastomotic techniques are currently being trialled and may prove useful for certain forms of CRVO.[19] Unfortunately, current laser photocoagulation therapy for prevention of neovascular glaucoma often does not work.[20] Further investigation of risk factors predictive of CRVO and BRVO is certainly warranted. The links between CRVO, retinal vascular changes, and glaucoma, including the other vascular features of glaucoma such as optic disc rim haemorrhages and venous collaterals, deserve more study.[17]

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Unilateral Proptosis and Bilateral Uveitis in a Child with Idiopathic Orbital Inflammation

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A 14-year-old child presented with unilateral right-sided acute onset proptosis. The diagnosis of idiopathic orbital inflammation was made after the clinical and laboratory investigations revealed no local or systemic identifiable causes. Moderate anterior chamber activity was present on the same side as the proptosis as well as in the contralateral asymptomatic eye. There was bilateral disc oedema. The presence of a bilateral idiopathic orbital inflammatory process was suggested radiologically by a computed tomography scan showing evidence of extensive diffuse anterior inflammatory infiltrate in the right orbit and periscleral infiltrate in the left orbit. However, the only clinical manifestation of idiopathic orbital inflammation in the eye with no proptosis was anterior uveitis. Idiopathic orbital inflammation should be considered in the differential diagnosis of paediatric uveitis, even in the absence of proptosis.

Key Words: *Inflammation, Uveitis, anterior*

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Introduction

Idiopathic orbital inflammation has highly variable clinical presentations ranging from a diffuse to a highly focal process targeting specific orbital tissues. It is predominantly a disorder of adults, but is also known to occur in children.[1] Paediatric orbital inflammation may be associated with uveitis, unlike the adult form of disease. However, orbital inflammatory syndrome is rarely considered in the differential diagnosis of childhood uveitis. This report is of a child with unilateral acute proptosis who had radiological evidence of bilateral idiopathic orbital inflammation with anterior uveitis presenting as the only clinical manifestation on the side with no proptosis.

Case Report

A 14-year-old Saudi Arabian boy presented with bulging of the right eye of 5 days' duration associated with periorbital pain, diplopia, photophobia, and reduced vision (Figure 1). The patient had mild malaise but no fever, and no history of preceding trauma. He had had an upper respiratory tract infection 10 days earlier, which had successfully resolved with treatment with antibiotics. His past medical and ocular history was otherwise unremarkable. The family history was negative for any relevant ophthalmic and systemic disorders.

His best-corrected visual acuity was 6/12 in the right eye and 6/9 in the left eye. The intraocular pressure in both eyes was normal. There was no afferent papillary defect and colour vision was unaffected. Confrontation visual fields were normal. He had proptosis of 4 mm on the right side with Hertel measurements of 21 mm for the right eye and 17 mm for the left eye, with bitemporal measurement of 112 mm.

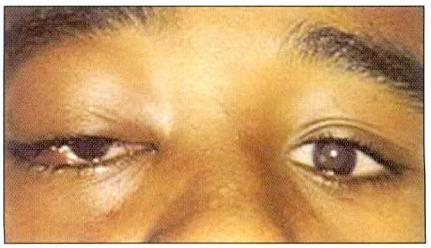

Figure 1. Right proptosis at presentation with acute periorbital congestive signs.

Right eye movements were restricted in all directions except laevo-version. Marked lid and conjunctival oedema and congestion were present in the right eye. Bilaterally, there was moderate anterior chamber activity with no keratic precipitates. Bilateral disc congestion and disc oedema were noted. The left eye was not congested and showed no other clinical abnormality except moderate anterior chamber activity and disc oedema.

Computed tomography (CT) revealed the presence of lacrimal gland enlargement, uveoscleral thickening, and diffuse anterior orbital infiltrate on the right side and periscleral thickening with ragged edges on the left side. The sinuses were clear. Magnetic resonance imaging examination confirmed the above findings and revealed no involvement of the orbital apex or cavernous sinus.

Microbiological examinations of specimens from the conjunctiva and throat, and blood cultures gave negative results. X-ray of the chest was unremarkable. Otorhinological and medical evaluation was negative for any systemic disorder. Laboratory investigations revealed mild leukocytosis with eosinophilia. Serum glucose, calcium, angiotensin-converting enzyme, immunoproteins, autoantibodies, C-antineutrophilic cytoplasmic antibody, and thyroid profile were all within normal limits. With the exclusion of any local and systemic identifiable cause, the diagnosis of idiopathic orbital inflammation was made and the patient started systemic oral prednisolone 60 mg per day. There was

a dramatic improvement in the proptosis within 48 hours. The steroid was tapered gradually over a 3-month period. Topical cycloplegic and steroid agents were discontinued after 10 days. The repeat CT scan performed after 10 days showed near complete resolution of orbital infiltrates. There was no recurrence of the disease reported in the 2-year follow-up period.

Discussion

Orbital pseudotumour or idiopathic orbital inflammation is a clinical diagnosis of exclusion. It is defined as a benign non-infective clinical syndrome characterised by features of non-specific inflammatory conditions of the orbit without identifiable local or systemic causes. Although idiopathic orbital inflammation is a more common disorder in adults than in children, it can occur very early in childhood.[1] It often presents acutely, as in this patient. It is known to be associated with upper respiratory tract infections, but the exact nature of association is not known.[2] Constitutional symptoms such as malaise and nausea are associated with the condition more frequently in children than in adults.[3] However, fever and gross leukocytosis are usually absent. This is helpful for differentiating this condition from infective orbital cellulitis, which is additionally associated with sinusitis in 96% of paediatric patients. Eosinophilia is a known accompaniment of idiopathic orbital inflammation in children.[2] Clinical and laboratory investigations are necessary to rule out specific causes of inflammation before the diagnosis of idiopathic orbital inflammation can be confirmed. In endemic and epidemic areas, the differential diagnosis of Lyme borreliosis, which could cause orbital, optic nerve, and uveal inflammation, should be ruled out.

Unlike in adults, idiopathic orbital inflammation in children is commonly bilateral. In adults, bilateral occurrence should alert the physician to the possibility of underlying systemic disorders such as Wegner's granulomatosis and lymphoma. In this patient, the test results for vasculitis and sarcoidosis were negative. Both these entities can present with uveitis as well as an orbital disease. Thyroid orbitopathy is an exceedingly rare cause for acute proptosis in children and is not associated with uveitis. The rapid dramatic response to steroids within 48 to 72 hours supports the diagnosis of idiopathic orbital inflammation. Biopsy is required only for patients who are not responsive to steroids or for recurrence.

Idiopathic orbital inflammation in children is commonly associated with uveitis. This patient is interesting because proptosis manifested unilaterally, while radiological evidence of orbital inflammation was present bilaterally. The only clinical manifestation of orbital inflammation on the side with no proptosis was the presence of anterior uveitis. This supports the suggestion that idiopathic orbital pseudotumour should be considered in the differential diagnosis of any child with uveitis with negative diagnostic test results, even when proptosis is absent.[2] Clues such as pain on eye movement, periorbital oedema, optic disc oedema, persistence or recurrence of uveitis, or a lack of response to topical steroids should specifically alert the ophthalmologist to the possibility of idiopathic orbital inflammation.[4] Patients may eventually develop proptosis and other orbital manifestations at serial follow-up.[3] B-mode ultrasound and magnetic resonance imaging may be the preferred modalities for the follow-up of paediatric patients with suspected orbital disease because of their freedom from the hazard of radiation.

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Correction

In the author list of the Original Article *Efficacy and Safety of Bimatoprost for Patients with Open Angle Glaucoma or Ocular Hypertension in the Asia Pacific Region* by Julian L Rait, Cze Hong Low, Edgar U Leuenberger, Pall Singh, Michael T O'Rourke, Rossana Romani, Boonsong Wanichwecha-rungruang in Asian J Ophthalmol 2005;7:82-90, Pall Singh was erroneously listed as Paul Singh. The online version of the article has been corrected.

Localised Graft-host Disparity: a Late Manifestation Following Deep Lamellar Keratoplasty

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Deep lamellar keratoplasty has proved to be a safe and effective surgical alternative for the management of anterior corneal disorders; very few complications have been reported. This report is of a 57-year-old woman who presented with diminution of vision following suture removal after deep lamellar keratoplasty. At examination, there was graft-host disparity with steepening of the inferotemporal axis of the cornea.

Key Words: *Corneal transplantation, Graft survival*

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Introduction

Deep lamellar keratoplasty (DLK) is a surgical alternative to penetrating keratoplasty for the management of anterior corneal disorders in which the endothelium and Descemet's membrane is normal.[1] The advantages of DLK over penetrating keratoplasty have been well established in the literature.[2] Only a few complications associated with DLK have been reported.[3] The biggest challenge in performing DLK is the removal of full-thickness stromal tissue while preserving an intact Descemet's membrane along with the endothelium. Various techniques have been described for removal of the full thickness stroma while preserving an intact Descemet's membrane.[2] However, all these techniques are associated with the risk for inadvertent perforation. Full thickness removal of the stroma is not always possible and surgeons can leave behind some stromal lamellae on the recipient bed. This report is of a patient who underwent DLK and developed localised graft-host disparity following suture removal after 5 months.

Case Report

A 57-year-old woman presented to the Cornea and Anterior Segment Services, LV Prasad Eye Institute, Hyderabad, India, in May 2002 with complaints of white spots over the cornea associated with decreased vision in both eyes for the previous 3 to 4 years. At presentation, visual acuity was 20/125 in the right eye and 20/400 in the left eye. B-scan ultrasonography of the posterior segment in both eyes was anechoic. A diagnosis of granular dystrophy with bilateral immature senile cataract was made, with the left eye being more affected than the right eye. Her keratometry readings in the right and left eyes before cataract surgery were 49.5 D at 110° and 48.0 D at 20°, and 47.4 D at 90° and 45.8 D at 180°, respectively.

She underwent penetrating keratoplasty with extracapsular cataract extraction with intraocular lens (IOL) implantation in the left eye in November 2002 and had an uneventful postoperative course, achieving a final visual acuity of 20/25. DLK was performed in the right eye in September 2004. Host trephination of 8 mm was followed by removal of stromal tissue in 4 layers, leaving intact Descemet's membrane with a few stromal lamellae. A graft of 8.5 mm was taken, from which Descemet's membrane was removed and the remaining tissue was sutured to the recipient bed with 16 interrupted 10-0 nylon sutures with knots buried on the host side. The patient had an uneventful postoperative course with good visual improvement.

At the 5-month follow-up visit, the graft-host junction was apposed with significant cataract with vision of 20/80; nine sutures were removed. At 8 months postoperatively, there was graft-host disparity in the temporal quadrant with a visual acuity of 20/100; the cataract had not progressed sufficiently to explain the visual deficit. Keratometry readings were 53.7 D at 100° and 46.5 D at 10° and there was severe steepening along the inferotemporal axis due to graft-host disparity (Figure 1). Throughout the follow-up period, the intra-ocular pressure remained within normal limits.

The patient underwent cataract extraction with IOL implantation. Three weeks after the cataract extraction, the visual acuity in the right eye was 20/40 with +6 diopter sphere and -13 diopter cylinder at 20°. Two months after the cataract surgery, the patient underwent right eye orbscan for keratometry and pachymetry, which revealed the following findings: 57.8 D at 113°

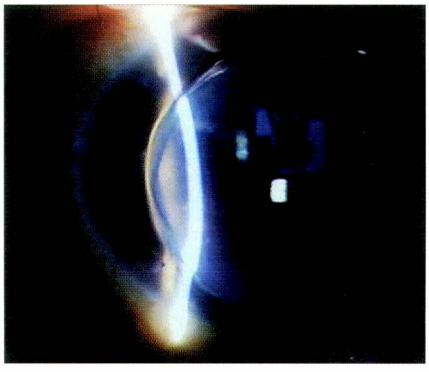

Figure 1. Slit lamp photograph of the right eye showing graft-host disparity in the temporal quadrant.

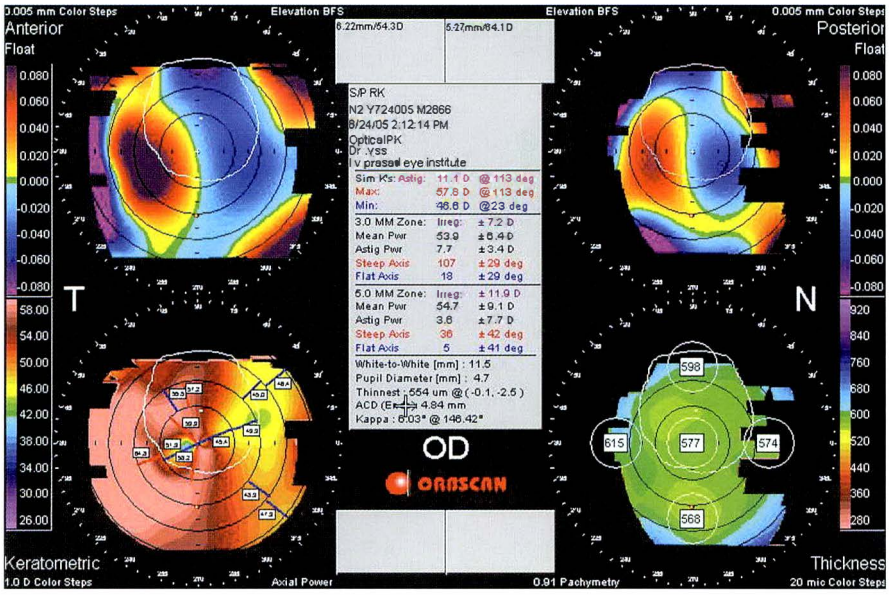

Figure 2. Orbscan image of the right eye after cataract surgery, showing the keratometry and pachymetry results.

and 46.6 D at 23° with corneal thickness of 615 μm temporally, 598 μm superiorly, 524 μm nasally, 568 μm inferiorly, and 577 μm centrally, as shown in Figure 2.

Discussion

In contrast to penetrating keratoplasty that requires the sutures to remain in situ for at least 18 to 24 months, the sutures may be removed after 6 to 12 months following DLK.[4] Graft-host junction stability after penetrating keratoplasty is poor and will not reach the original tensile strength of the tissue. This is mainly due to the avascular nature of the cornea, prolonged use of steroids, and the use of non-inflammatory suture materials.[5] Animal studies have shown that recovery of the wound after penetrating keratoplasty is slow in the first week, with up to 50% recovery after 6 months, and complete recovery is never achieved regardless of whether the wound is full thickness, partial thickness, or has 10-0 sutures in place.[6]

Graft-host junction disparity can be due to either disproportionately thick donor tissue or insufficient removal of stroma from the recipient bed. Melles et al have shown that when the donor tissue thickness exceeds the depth of the recipient bed, the donor button still fits because the peripheral recipient cornea is split while the dissection is made, and the excess thickness of the button only causes little separation of the recipient posterior stromal layers.[2] In normal wound healing after keratoplasty, the average keratometry reading can be up to 45 D. If the keratometry reading is ≥46 D, there is likely to be wound dehiscence in that meridian.[7]

In this patient, graft-host disparity was limited to a particular zone, which could have been due to insufficient removal of the stroma from the recipient bed in that zone. This was also suggested by the pachymetry readings. This disparity was masked by the compressive effect of sutures and later became apparent when the sutures were removed.

Acknowledgements

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Angle Closure Glaucoma — Diagnosis and Treatment

From the World Glaucoma Congress held in Vienna, Austria, 6-9 July 2005

GONIOSCOPY

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Gonioscopy is an essential component of glaucoma management as treatment depends on understanding whether angle closure is present. Gonioscopy is also necessary to identify individuals at risk for acute angle closure (AAC) as well as those with evidence of chronic angle closure (CAC). Furthermore, gonioscopy can clarify the exact mechanism at play, including pupil block, peripheral anterior synechiae (PAS), lens-induced angle changes, plateau iris, and angle recession.

The prevalence of narrow angles is estimated to be 1.5% among Chinese people and 0.6% among Caucasians older than 40 years. However, the actual prevalence may be higher than this as the use of excessive lighting during gonioscopy can open the angle, leading to an underestimate of the true rate of angle closure. Lighting needs to be controlled for accurate results by using a small (1 mm) bright beam and ensuring that no light falls on the pupil during examination. Angles that appear open in bright illumination may be closed when the lights are off. This has been well illustrated with ultrasound biomicroscopy (UBM) and optical coherence tomography (OCT).

An alternative to gonioscopy for angle assessment is the van Herrick test to measure the limbal anterior chamber depth (ACD). Although gonioscopy and limbal ACD do not always match, the finding of a very shallow van Herrick may be as important as seeing angle closure on gonioscopy. More research is needed to clarify the predictive value of a narrow van Herrick.

The Spaeth system for gonioscopy grading assesses the amount of angle opening, iris configuration, and iris insertion. Iris configuration is important for determining different mechanisms of angle closure, possibly leading to more appropriate treatments. By recording the level of iris insertion, the observer is forced to consider whether the posterior trabecular meshwork is visible. When it is not, the angle may be at higher risk of developing problems related to angle closure.

A Goldmann gonioscopy lens gives the best view of the angle, although compression gonioscopy using a different lens may be required to rule out PAS; such a lens should also be available. Newer technologies such as OCT and other imaging techniques may increase knowledge about angle closure mechanisms and may act as an adjunct to gonioscopy.

ULTRASOUND BIOMICROSCOPY

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UBM obtains a high-resolution image of the anterior segment structures and their relationships by using a 50 MHz high-frequency transducer that yields axial resolution up to 25 μm with lateral resolution of 50 μm. UBM is the current best imaging technology for viewing the posterior chamber and the ciliary process. Pupillary block can be easily identified using UBM and iris-lens contact, anterior rotation and elongation of the ciliary process, anterior chamber depth, iris thickness, and posterior chamber area can all be visualised.

The advantages of UBM are that it is non-invasive and is superior to gonioscopy, which requires a high skill level, needs light, produces variable results, and is not able to visualise the posterior chamber. UBM can produce dynamic study of the angle in light versus dark conditions, and may reveal more information about occludable angles. Disadvantages of UBM include the limited field, resolution, and position.

UBM reveals previously unseen information on the relationships of anterior segment structures. The technology enhances understanding of the pathophysiology of angle closure. UBM can identify angle closure mechanisms and guide clinical decision making, for example lens removal, and is useful for monitoring progressive change. It is the only non-invasive technology for visualising the posterior chamber and ciliary body. Importantly, UBM provides both qualitative and quantitative assessment.

OPTICAL COHERENCE TOMOGRAPHY

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The currently available methods of angle assessment are gonioscopy and UBM. However, recent advances in imaging techniques have led to more objective ways to define the angle. Anterior segment OCT (AS-OCT) is a new high-resolution anterior segment imaging modality that offers an

alternative method of assessment using low-coherence interferometry. This is a non-contact procedure that is performed with the patient sitting upright, so is more physiological than some methods of assessment. Image acquisition is rapid.

The technology is easy to operate with low interobserver variability. Both angles may be viewed simultaneously and iris-lens interaction can be assessed. AS-OCT enables dynamic in vivo high-resolution imaging of the anterior segment, study of the relationship between the tissues, study of the architecture of the tissues, and quantitative and qualitative analysis. AS-OCT has been used to assess light-dark changes, pupil block, and plateau iris, and to check the efficacy of treatment post-laser peripheral iridotomy, as well as measuring corneal thickness, anterior chamber depth, angle opening distance, and angle recess area. Other uses for AS-OCT may include monitoring of accommodative intraocular lenses, corneal refractive surgery, and anterior segment tumours.

The disadvantages of AS-OCT include difficulty in visualising the ciliary body and the technology is expensive. However, AS-OCT is a rapid non-contact high-resolution method of imaging the angle, with the potential for use as a diagnostic/screening tool for angle closure.

LASER PERIPHERAL IRIDOTOMY

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In patients with acute angle closure glaucoma (AACG), laser peripheral iridotomy (LPI) protects the fellow eye from developing an acute attack. However, in the affected eye, further treatment such as iridoplasty may be required to break an acute attack. LPI alone is probably not effective for control of IOP in eyes with acute angle closure (AAC) or AACG, with a 40% success rate if performed early in the attack and only 6% success for late treatment. Most eyes with AAC will require surgery following LPI, and surgical options vary.

For patients with primary angle closure suspect (PACS), primary angle closure (PAC), or primary angle closure glaucoma (PACG), the number needed to treat (NNT) to prevent 1 person from progressing to glaucoma or blindness is a useful indicator for targeting high-risk groups.

For patients with PACS, of whom 22% will progress to angle closure, assuming that LPI is 100% effective, the NNT is only 5. However, this is for preventing raised IOP and PAS. The NNT to prevent early glaucoma is 21 over 10 years and this number will be much greater to prevent blindness. A randomised controlled trial is currently underway to investigate the efficacy of LPI as an intervention following screening, which should provide data of the benefits versus side effects. This information will help in the decision making process for PACS.

For patients with angle closure, of whom 28% will progress to PACG over 5 years, assuming an 80% efficacy rate for LPI, the number needed to treat is 5. While the NNT to prevent blindness would be greater, this intervention does prevent early glaucoma and potential blindness. LPI is the standard of care for this indication. A small population-based study of patients with PAC found that of 9 patients who underwent LPI, 1 progressed to glaucoma, while 9 of 19 patients who did not undergo LPI progressed.

LPI is less effective for PACG than for PACS or PAC, with the majority of patients requiring further medical or laser treatment and up to 50% of patients require surgery. A study of the efficacy of LPI for 233 eyes of 158 patients with PAC and early PACG, found a 74% success rate, with 11% of patients requiring further medications for IOP control. Twenty six percent of angles did not open due to plateau iris, but iridoplasty was effective for these eyes and 13% required further medication for IOP control.

LPI is effective for the fellow eyes of those with AAC, and is the mainstay of treatment for the affected eye. It is the standard treatment for PAC and PACG but the success seems to decrease with increasing disease severity. LPI is effective for PACS, but should be selected on an individual basis.

AFTER LASER PERIPHERAL IRIDOTOMY

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Patients with angle closure frequently have episodic high IOP after LPI. Frequent problems associated with LPI include iridotomy patency, inflammation, residual angle closure, and IOP level with or without the presence of glaucomatous optic neuropathy (GON).

To check the patency of the iridotomy, it is necessary to be able to see the lens through the iridotomy. The iridotomy should move over the lens surface as the pupil constricts in the presence of light. Movement indicates that the PI is not stuck to the lens and blocked by anterior synechiae. Vitreous in the PI is also undesirable.

Inflammation is a common problem post-LPI. The trabecular meshwork is exposed to debris from the laser photodisruption and from the acute episode irself. Adequate suppression of inflammation is ensured with topical steroids, which should be tapered gradually as the iris surface is usually disrupted after corneal contact.

Table 1. Managing high intraocular pressure after laser peripheral iridotomy.

- Ensure peripheral iridotomy patency
- Suppress inflammation
- Perform gonioscopy post-peripheral iridotomy to determine the angle status
- Add on iridoplasty and intraocular pressure–lowering medication as needed
- Monitor for intraocular pressure rise and occurrence of glaucomatous optic neuropathy

Residual angle closure may persist despite a functional iridotomy. This may occur because of multiple angle closure mechanisms — the angle will remain closed if there is more than one mechanism of angle closure present, despite initial treatment. PAS may be present if there is established PAC, and pupil block or angle crowding (plateau iris) may cause residual angle closure. It is therefore crucial to perform gonioscopy after LPI to evaluate the angle status once the view is clear.

A study of long-term outcomes for patients with acute angle closure show that only 41.8% were successfully treated with LPI alone, with 58.1% developing raised IOP after treatment. Long-term outcomes after LPI for chronic angle closure glaucoma (CACG) show that 94% of patients developed raised IOP, with 53% requiring filtering surgery.

Approximately half of all patients with symptomatic PAC develop raised IOP by 6 months despite a patent PI, and only 10% of patients with PACG with GON achieve adequate IOP control with LPI alone. Gonioscopy should therefore be performed after LPI to check the angle status (Table 1).

TREATMENT OPTIONS

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The first-line treatment for AAC is usually medical, based on the mechanism of angle closure. Medications include α-agonists, β-blockers, or carbonic anhydrase inhibitors; osmotics may be required if the attack persists or the IOP increases. LPI should be attempted as early as possible as the mainstay of treatment.

Newer interventions include acute laser gonioplasty plus medication, which leads to a more rapid decrease in IOP than medication alone (mean IOP reduction, 66 mm Hg to 18 mm Hg in 1 hour). This technique is useful if an attack is difficult to treat. Another approach that works quickly is paracentesis (mean IOP reduction, 66 mm Hg to 15 mm Hg immediately).

AAC is a bilateral disease, with 10% of patients presenting with bilateral angle closure and 50% having an acute attack in the contralateral eye if left untreated. Contralateral involvement can occur within days, so LPI should be performed in the fellow eye as soon as possible.

Pilocarpine is still widely used as prophylaxis for the contralateral eye. Pilocarpine can widen the angle, but it can also precipitate an attack, so early LPI is preferable.

Medications that substantially lower IOP are now available for the treatment of PACG and latanoprost is effective for this indication (Table 2). Lens extraction can result in a large increase in angle opening for patients with PACG, and it has been shown that this technique lowers the IOP. Lens extraction may also be combined with other procedures such as goniosynechialysis and diode gonioplasty.

In summary, narrow angles are common. LPI is effective for the treatment of acute attacks, but may be less effective for other indications. Acute attacks are typically managed with medications unless LPI can be performed. Gonioplasty may be a reasonable alternative. The contralateral eye should be treated as soon as possible. The management of PACG is evolving and medications have been found to be effective for this condition. There are many surgical options available, but more research is needed to resolve the question of which approach is the most favourable.

CATARACT EXTRACTION

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Lens extraction by the phaco technique may be a useful treatment for ACG, particularly if some cataract is already present. Lens extraction may be performed after aborting an acute attack, to prevent progression to CACG, and for management of established CACG.

Lens extraction has been reported to be safe and effective when performed for patients with an 'uncontrolled' acute attack of angle closure. However, the procedure is technically challenging and is skill-dependent in this situation, so may not be advisable for all patients. An alternative procedure recommended by Dr Lam for patients with an uncontrolled acute attack is to use argon laser peripheral iridoplasty (ALPI).

Up to 58% of eyes develop a rise in IOP after AAC, so prevention of progression to CACG is important. After the acute attack

Table 2. Efficacy of latanoprost versus timolol for primary angle closure glaucoma after 12 weeks of therapy.

	Latanoprost	Timolol
Mean decrease in intraocular pressure	8.2 mm Hg	5.2 mm Hg

has been controlled, early lens removal is likely to open the angle sufficiently to prevent progression to chronic disease.

A randomised controlled study is currently ongoing in Hong Kong to compare the effects of early phaco with LPI for acute angle closure. Early results suggest that phaco is much more effective than LPI (4.4% vs 22.2% of patients had IOP >21 mm Hg). It is thought that widening the drainage angle may prevent PAS formation and reduce the chance of progression to CACG. Lens extraction may therefore become a good second-stage treatment for AAC. For established CACG, lens extraction has been shown to be successful for IOP control. However, the usual method of lens extraction in the reports was extracapsular cataract extraction, and the patient groups were not homogeneous so the results are difficult to interpret. An ongoing study is underway to assess the effect of phaco for patients with CACG with and without cataract. Of 26 eyes treated to date, 19 (73%) have reduced IOPs and need fewer medications to control IOP. ALPI is preferable to lens extraction at the time of an acute attack uncontrolled by standard treatments. Lens extraction is promising for preventing progression to CACG, but more data are required, particularly with respect to the timing of the procedure. Encouraging data are available for the use of lens extraction for established CACG, but further research is required.

Text prepared by a staff medical writer.

Considerations for Adjunctive Therapies

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For many patients with glaucoma, monotherapy is not sufficient to control intraocular pressure (IOP) and adjunctive therapy may be required. The criteria for the choice of adjunctive therapy include incremental efficacy, compliance, tolerability, and safety. There are a number of possible adjunctive combinations for lowering IOP, as shown in Table 1.

Fluctuation in IOP is a consideration when selecting adjunctive therapy, as the risk for disease progression is associated with IOP fluctuation rather than high IOP alone. Compliance issues should be considered when a new drug is added to a treatment regimen, as compliance is thought to be better with once-daily dosing. A further consideration when administering multiple medications is the washout effect as some of the efficacy of a drug may be lost with only a short interval between administration of 2 different eye drops. The focus for adjunctive therapy has moved to fixed combination medications to avoid compliance issues and the washout effect.

Clinical Practice

Konstas et al evaluated the 24-hour efficacy of brimonidine versus dorzolamide added to latanoprost.[1] The additional mean IOP-lowering between the 2 regimens was not significantly different at approximately 2.0 mm Hg for brimonidine and 2.1 mm Hg for dorzolamide. Brimonidine and dorzolamide had a similar additive effect to latanoprost, and no unexpected side effects occurred with the addition of these drugs.

A study performed to investigate administration of latanoprost given as adjunctive therapy to timolol found that the addition of latanoprost significantly lowered IOP whether given in the morning or evening ($p < 0.0001$ compared with baseline) [Table 2].[2] Addition of latanoprost also reduced diurnal fluctuation in this group of patients.

A crossover trial examined the administration of latanoprost/timolol fixed combination compared with latanoprost alone for 8 weeks.[3] Latanoprost/timolol produced additional IOP-lowering of 2.5 to 3.0 mm Hg over latanoprost alone, suggesting greater efficacy for the combination therapy.

Latanoprost/timolol has been compared with latanoprost plus timolol. While both regimens effectively and safely controlled IOP, there was a 1.1 mm Hg greater IOP reduction with the unfixed combination.[4] Further study has found that the IOP-lowering efficacy of latanoprost/timolol and the unfixed components were not significantly different.

In a comparison of latanoprost/timolol with timolol/dorzolamide, there was no difference between the 2 treatments after 1 month, and 1 mm Hg difference in IOP in favour of latanoprost/timolol after 3 months.[5] Both treatments were well tolerated.

Latanoprost/timolol has been compared with brimonidine plus timolol.[6] Both treatments effectively lowered IOP, but latanoprost/timolol produced slightly better IOP lowering than brimonidine plus timolol at certain time points. In addition, there were a greater number of ocular adverse events in the patients receiving brimonidine plus timolol.

Table 1. Drug combinations for lowering intraocular pressure.

Type of therapy	Drug class		
First-line therapy	Prostaglandin	β-Blocker	α-Agonist
Adjunctive therapy	β-Blocker α-Agonist Carbonic anhydrase inhibitor	α-Agonist Carbonic anhydrase inhibitor	Carbonic anhydrase inhibitor

Table 2. Additive effect of latanoprost to timolol with morning or evening dosing.

Time	Intraocular pressure (mm Hg)
Baseline	21.1 (SD, 3.3)
Latanoprost morning	17.3 (SD, 3.1)
Latanoprost evening	17.1 (SD, 2.7)

Conclusion

Given the currently available data, latanoprost/timolol fixed combination is more efficacious than brimonidine/timolol and than timolol/dorzolamide. Latanoprost/timolol has similar efficacy to the unfixed combination of latanoprost and timolol. However, fixed combination therapies are likely to be advantageous for compliance.

References

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Current Treatment for Chronic Angle Closure Glaucoma

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Glaucoma Service and International Affairs
Department of Ophthalmology
Chulalongkorn University
Bangkok
Thailand

Chronic angle closure glaucoma (CACG) comprises 2 clinical entities of angle closure and glaucomatous optic neuropathy (GON). Angle closure and GON involve structural and functional changes to the angle and optic disc, respectively. CACG has several stages in its development, as shown in Figure 1.

The structural damage associated with angle closure involves synechial closure of the trabecular meshwork and the functional damage occurs in the form of an acute, intermittent, or chronic rise in intraocular pressure (IOP). Early removal of a mechanical blockage can restore normal drainage in otherwise healthy eyes. The aims of treatment for CACG are to preserve visual function and avoid end organ damage.

Treatment Concepts

The basic principles of management of CACG are as follows:

- removal of the angle closure by modification and correction of the angle configuration
- control of IOP and other risk factors
- monitoring and maintaining the structural and functional integrity of the optic disc and nerve fibre layer.

Figure 1. Angle closure glaucoma cascade.

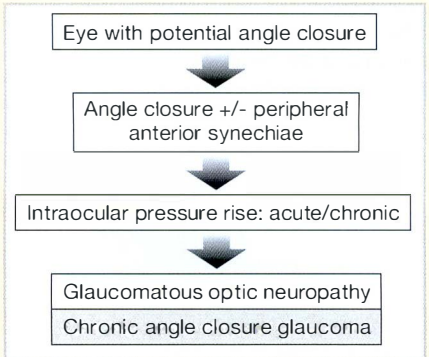

Table 1. Steps for the management of chronic angle closure glaucoma.

1. Document angle closure and glaucoma (presence or evidence of peripheral anterior synechiae, previous attack, intraocular pressure rise, glaucomatous optic disc changes, and visual function change).
2. Identify and correct angle closure mechanisms.
3. For chronic angle closure glaucoma, start medication and consider laser peripheral iridotomy.
4. Reassess after laser peripheral iridotomy for need for iridoplasty, medication, and/or surgical intervention.
5. Monitor intraocular pressure, optic disc, and visual field.

The steps for the management of CACG are shown in Table 1.

Examination of the angle structures involves ascertaining whether the trabecular meshwork is open or closed, and the extent of the closure, if present, using slit lamp examination or gonioscopy. The most common mechanism for angle closure is pupillary block, which may be corrected by laser peripheral iridotomy (LPI). Other mechanisms include plateau iris, thick peripheral iris, lens-related closure, and ciliary block. However, angle closure is often caused by multiple mechanisms.

LPI is an effective treatment for occludable/angle closure suspects proving successful in 97% of eyes. The procedure is less effective when performed in the fellow eyes of patients with acute angle closure (91%), eyes with acute angle closure (68%), and eyes with GON (6%).

Medication is the usual first-line therapy for CACG, with an option to perform LPI in the presence of pupillary block. However, even after successful LPI, most eyes presenting with raised IOP and optic disc and visual field damage require further treatment to control IOP. This is thought to be due to the presence of multiple mechanisms of angle closure or because the angle structure has been irreversibly damaged.

Iridoplasty to thin the peripheral iris is successful for treating acute angle closure. While the procedure has no proven efficacy for treating CACG, there is suggestive evidence that it is highly effective for plateau iris syndrome and CACG with a positive provocative test. While the lens should only be removed if it is a mechanism of angle closure, glaucoma filtering surgery can be considered as the next step for IOP control.

Medication is often required for IOP control, even after other treatment options have been performed. The first large-scale randomised controlled trial of medication for treatment of CACG investigated the efficacy of latanoprost or timolol for this indication.[1] Latanoprost reduced the IOP by 8.2 mm Hg compared with 5.2 mm Hg for timolol ($p < 0.001$), and achieved up to 30% IOP reduction from baseline compared with 20% for timolol. More patients receiving latanoprost achieved a lower target pressure. Latanoprost acts by increasing uveoscleral outflow, so it may be that the mechanism of action of latanoprost in CACG is to facilitate flow through the iris and ciliary body.

Conclusion

Understanding the fundamental concepts of angle closure is crucial to developing a treatment plan for CACG. LPI remains the most important step in the treatment plan for pupillary block, but its efficacy remains unreliable, and other mechanisms of angle closure need to be identified and corrected. Most eyes with CACG need medical therapy to control IOP.

Reference

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From the Pfizer Ophthalmics satellite symposium Therapies in Focus: Current and Emerging Perspectives in Glaucoma *held at the Asian Oceanic Glaucoma Society/South East Asia Glaucoma Interest Group and Australia and New Zealand Glaucoma Club Congress, Cairns, Australia, 3 September 2005.*

FEBRUARY 2006

9-12
64th Annual All India Ophthalmological Conference (AIOS)
Jalna, India
Contact: Dr Salil Kumar
Tel: (91 075) 52466557/5277862
E-mail: dr_salilkumar@yahoo.co.in

19-24
World Ophthalmology Congress
XXX International Congress of Ophthalmology
XXVI Panamerican Congress of Ophthalmology
XVII Brazilian Congress of Prevention of Blindness
São Paulo, Brazil
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MARCH 2006

18-22
American Society of Cataract and Refractive Surgery
San Francisco, CA, USA
Tel: (1 703) 591 2220
Fax: (1 703) 591 0614
E-mail: ascrs@ascrs.org
Website: www.ascrs.org

29-2 April
6th International Symposium on Ocular Pharmacology & Therapeutics — ISOPT
Berlin, Germany
Contact: Congress Secretariat, Kenes Building, Airport City 70151, P.O.Box 56, Ben-Gurion Airport 70100, Israel.
Tel: (97 239) 727 510
Fax: (97 239) 727 555
E-mail: isopt@kenes.com

APRIL 2006

14-16
6th Congress of Ophthalmology and Optometry China (COOC 2006) Trade Exhibition
Shanghai, China
Contact: Prakash M Kamath
Tel: (91 8621) 6351 4526
E-mail: pmk_shanghai@yahoo.com
Website: www.realexpo.net/cooc

30-5 May
Association For Research In Vision & Ophthalmology (ARVO)
Fort Lauderdale, FL, USA
Website: www.arvo.org

Future ARVO Meetings
Fort Lauderdale, FL, USA
2007 — 6-10 May
2008 — 27 April-1 May
2009 — 3-7 May
2010 — 2-6 May

MAY 2006

18-22
Annual Symposium and Congress of the American Society of Cataract and Refractive Surgery
San Francisco, CA, USA
Contact: ASCRS-ASOA, 4000 Legato Road, Suite 850, Fairfax, Virginia 22033, USA
Tel: (1 703) 591 2220
Fax: (1 703) 591 0614

25-28
International Congress on Glaucoma Surgery
Toronto, Canada
Contact: Continuing Medical Education Office
Tel: (1 416) 978 2719
Fax: (1 416) 946 7028

JUNE 2006

9-12
2007 Congress of the European Society of Ophthalmology
Vienna, Austria
Contact: Britta Sjöblom
Tel: (46 84) 596 650
Fax: (46 86) 619 125

10-14
21st Congress of the Asia-Pacific Academy of Ophthalmology
19th Asia Pacific Association of Cataract & Refractive Surgeons Annual Meeting
4th Asia Pacific Society of Ophthalmic Plastic & Reconstructive Surgery Annual Meeting
Singapore
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24-25
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Local contact: Prof Xiaoguang Dong
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Fax: (86 532) 8589 1110
E-mail: seih@public.qd.sd.cn
or
ingrid.kreissig@augen.ma.uni-heidelberg.de
Website: http://kreissig.uni-hd.de/

AUGUST 2006

16-18
11th International Myopia Conference
Singapore
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Fax: (65) 6438 9757
E-mail: colleen_koh@hpb.gov.sg

NOVEMBER 2006

11-14
2006 American Academy of Ophthalmology's Joint Meeting with the Asia Pacific Academy of Ophthalmology
Las Vegas, NV, USA
Contact: American Academy of Ophthalmology, PO Box 7424, San Francisco, CA 94120-7424, USA
Tel: (1 415) 561 8500
Fax: (1 415) 561 8533
E-mail: meetings@aao.org

DECEMBER 2006

1-3
SEAGIG 2006
Meeting of the South East Asia Glaucoma Interest Group/Asian Oceanian Glaucoma Society and the Glaucoma Society of India
Chennai, India
Contact: Dr L Vijaya, Director, Sankara Nethralaya, No.18, College Road, Chennai 600 006, Tamil Nadu, India
Tel: (91 44) 2827 1616
Fax: (91 44) 2825 4180
E-mail: seagig_2006@yahoo.com or drlv@snmail.org

21st Congress of the Asia-Pacific Academy of Ophthalmology

The 21st Congress of the Asia-Pacific Academy of Ophthalmology (APAO) will be held in Singapore on 10-14 June 2006 at Suntec Singapore International Exhibition and Convention Centre.

The APAO Congress is one of the most important ophthalmological meetings in the Asia Pacific region. It has a superb scientific programme reviewed by an international panel, a renowned faculty of lecturers, high-quality free paper sessions and poster presentations, and an interactive exhibition showcase. Not only will this meeting provide updates on professional ophthalmic practices and advances, a distinguished faculty of the top specialists in ophthalmology from around the world has also been invited to share their expertise, experience, and vision for the future of ophthalmology with clinicians, eye surgeons, and researchers from the Asia Pacific region and beyond.

The Organising Committee has strived to design an interesting, balanced, and comprehensive programme with special highlights such as named lectures, plenary lectures, subspecialty symposia, live surgery, instruction courses, skilled transfer workshops, wet labs, satellite symposia and meetings, high-quality free paper and poster scientific presentations, video presentations, welcome reception, congress banquet, awards, and exciting social programme. Whether your interests are focused or broad, you will find a rewarding session at all times.

The scientific programme is devoted to various subspecialty topics, which include cataract, cornea, glaucoma, international ophthalmology, medical retina, neuro-ophthalmology, oculoplastics, paediatric ophthalmology, refractive ophthalmology, surgical retina, uveitis, and paramedical topics such as orthoptics, nursing/technical, optometry, contact lens, low vision, and ophthalmic photography.

At the same time, APAO welcomes the participation of the 19th Asia Pacific Association of Cataract & Refractive Surgeons and the 4th Asia Pacific Society of Ophthalmic Plastic & Reconstructive Surgery annual meetings in conjunction with the APAO Congress in 2006, which will significantly enhance the overall scientific content of the Congress.

The Congress also boasts an interactive exhibition showcase, taking up exhibition hall space of more than 6000 square metres, that offers a unique platform and opportunity to sponsors and exhibitors to reach out to and interact with an international group of ophthalmologists and eye surgeons.

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Come join us as we welcome you with the wonderful culture and hospitality that is uniquely Singapore and sharing "Our Vision for the Future". See you in Singapore in June 2006!

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Online Registration Starts NOW!	30 November 2005
Closing of Early Bird Registration	31 March 2006
Online Hotel Booking Starts NOW!	30 November 2005
Closing of Hotel Booking	15 April 2006

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The manuscript must be accompanied by a title page, an abstract, and key words. The title page must contain the title of the manuscript; a short running title (no more than 40 characters, including spaces); the full names of all authors and their 2 highest qualifications; the department(s) and institution(s) to which each author is affiliated; and the full name, address, telephone and fax numbers, and e-mail address of the corresponding author. The abstract for original articles must be structured with the following subheadings: Aim(s), Patients and Methods, Results, and Conclusion(s). Abstracts for all other articles must be unstructured. Abstracts should be no longer than 250 words. The key words must be Medical Subject Headings taken from Medline/*Index Medicus*.

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Supplement

Taylor A, Jacques PF, Epstein EM. Relations among aging, antioxidant status, and cataract. Am J Clin Nutr 1995;62 (6 Suppl): 1439-1447.

Books and other monographs

Kupfer C, Underwood B, Gillen T. Leading causes of visual impairment worldwide. In: Albert DM, Jakobiec FA, editors. Principles and practice of ophthalmology. Philadelphia: WB Saunders Company; 1994: 1250-1251.

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Reference

1. International Committee of Medical Journal Editors (ICMJE). Uniform requirements for manuscripts submitted to biomedical journals: writing and editing for biomedical publication. ICMJE; 2004. www.icmje.org

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References:
1. Higginbotham EJ, Feldman R, Stiles M, Dubiner H, for the Fixed Combination Investigative Group. Latanoprost and timolol combination therapy vs monotherapy: one-year randomized trial. *Arch Ophthalmol.* 2002;120:915-922. **2.** Data on file. Pfizer Inc, New York, NY.

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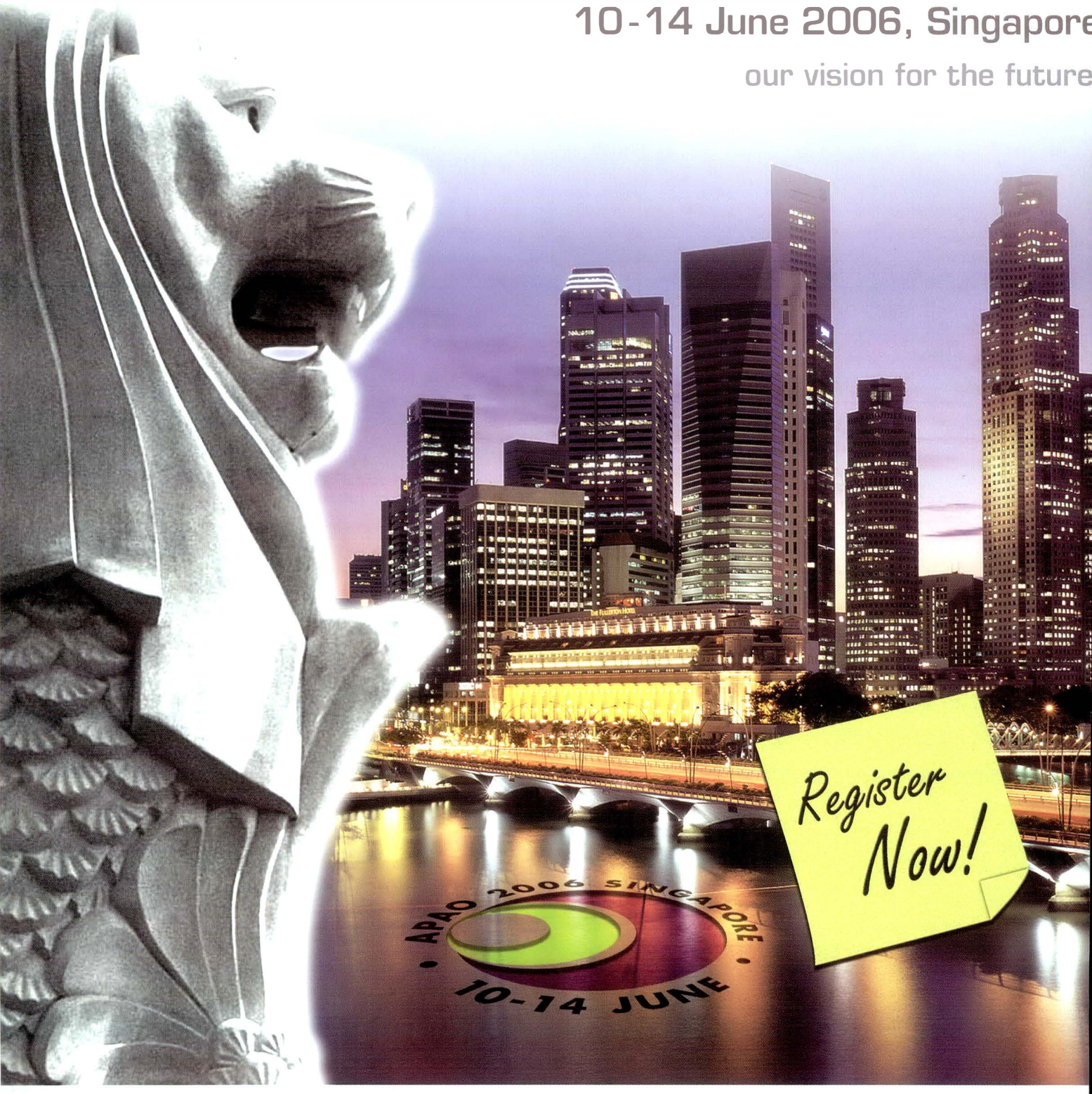

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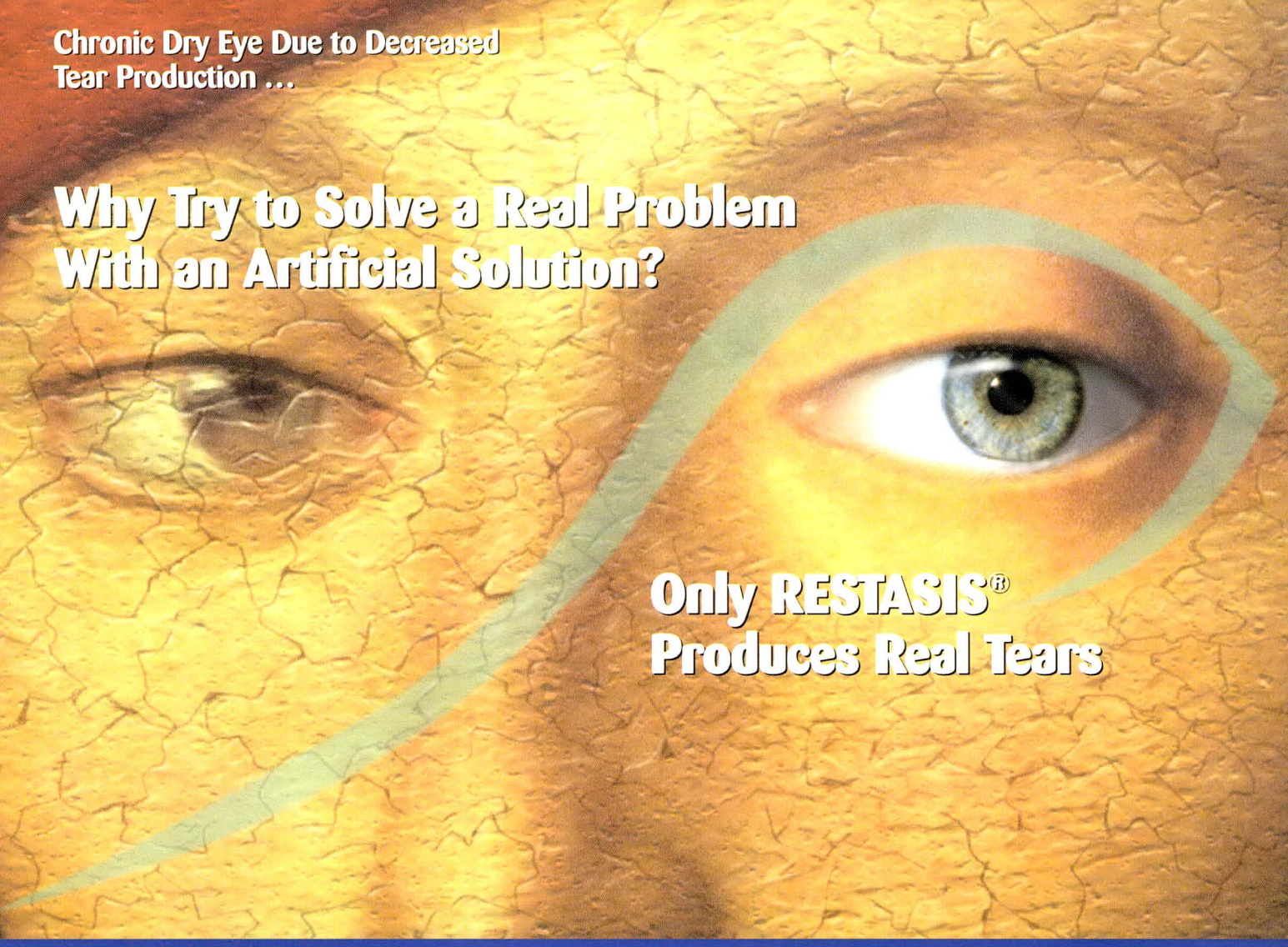

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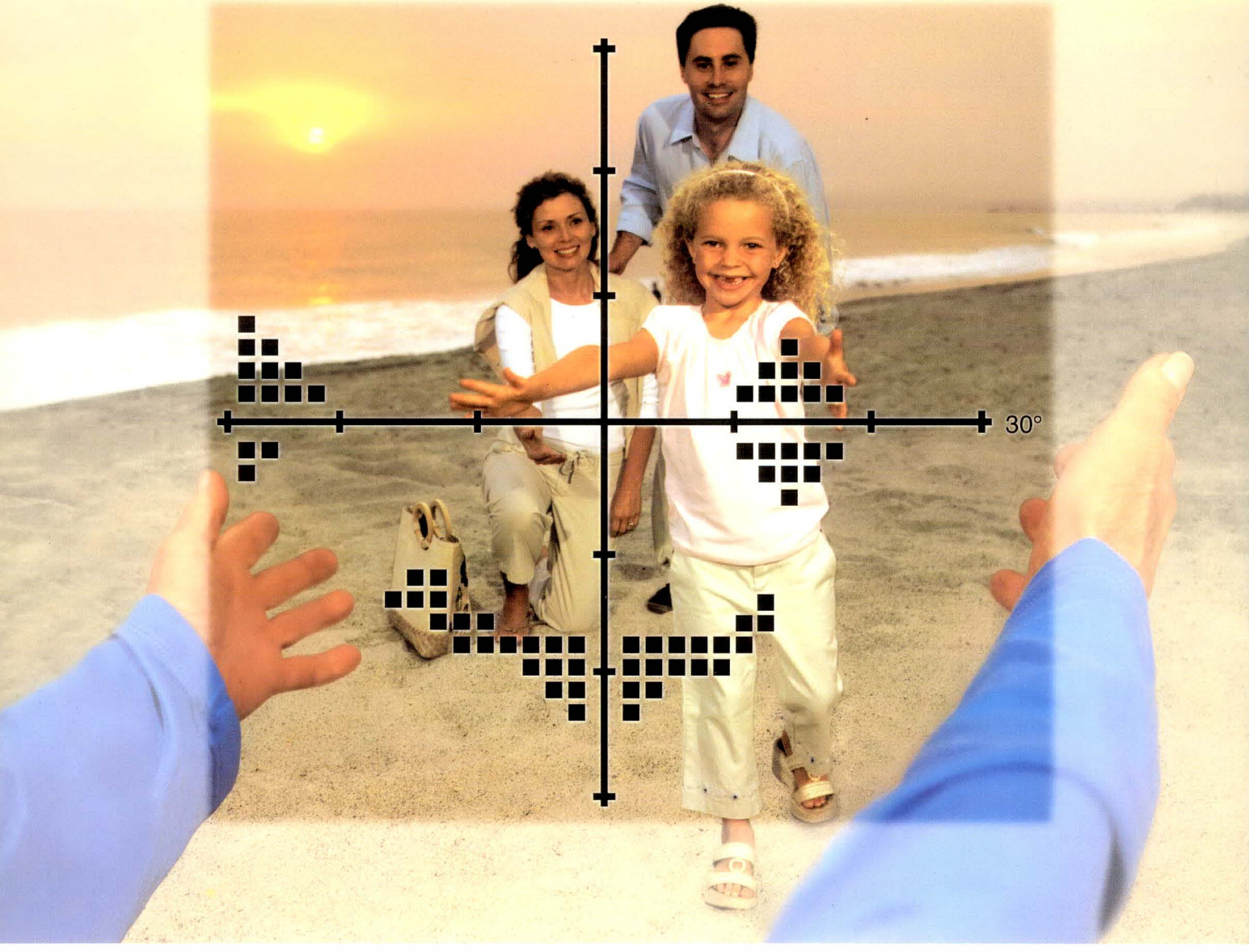

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