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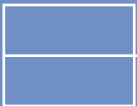
SEA  IG

South East Asia Glaucoma Interest Group

SECOND EDITION

ASIA PACIFIC

Glaucoma Guidelines



Scientific Communications



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**ASIA PACIFIC**

**Glaucoma Guidelines**

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## **CONTENTS**

|   |            |
|---|------------|
| Members of the Working Party/Contributors | <i>ii</i>  |
| Members of the Review Committee           | <i>iii</i> |
| Acknowledgement                           | <i>iv</i>  |
| Introduction                              | 1 ■        |
| Epidemiology of Glaucoma in Asia          | 3 ■        |

### **SECTION 1 ASSESSMENT**

|   |      |
|---|------|
| 1.1 Patient Assessment                    | 5 ■  |
| 1.2 Risk Categories and Treatment Targets | 17 ■ |

### **SECTION 2 TREATMENT**

|                             |      |
|-----------------------------|------|
| 2.1 Initiation of Treatment | 23 ■ |
| 2.2 Medical Treatment       | 25 ■ |
| 2.3 Laser Treatment         | 29 ■ |
| 2.4 Surgery                 | 41 ■ |

### **SECTION 3 FOLLOW-UP**

|                                |      |
|--------------------------------|------|
| 3.1 Follow-up                  | 47 ■ |
| 3.2 Screening                  | 53 ■ |
| 3.3 Frequently Asked Questions | 59 ■ |

### **SECTION 4 APPENDICES**

|                                      |       |
|--------------------------------------|-------|
| 4.1 Appendices                       | 73 ■  |
| Further Reading                      | 99 ■  |
| Suggested Areas for Further Research | 101 ■ |
| Definition of Terms                  | 103 ■ |
| Abbreviations                        | 105 ■ |
| Index                                | 107 ■ |

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## **APPENDICES**

|     |   |    |
|-----|---|----|
| 1   | Treatment of Childhood Glaucoma                             | 73 |
| 2   | Treatment in Pregnancy and Lactation                        | 74 |
| 3   | Systemic Medications that May Induce Angle Closure          | 76 |
| 4   | How to Test Calibration of a Goldmann Tonometer             | 77 |
| 5   | Tonometry Mires   | 78 |
| 6A  | Gonioscopy  | 79 |
| 6B  | Goniogram/Gonioscopic Chart                                 | 80 |
| 6C  | Van Herick Grading  | 81 |
| 6D  | Corneal Wedge Diagram                                       | 82 |
| 7A  | How to Optimise Patient Performance in Subjective Perimetry | 83 |
| 7B  | Common Artifacts for Visual Field Measurements              | 84 |
| 8   | Secondary Glaucomas — Principles of Management              | 85 |
| 9   | Angle Closure Mechanisms                                    | 86 |
| 10  | Side Effects of Glaucoma Medications                        | 87 |
| 11A | Laser Trabeculoplasty                                       | 89 |
| 11B | Contact Trans-scleral Diode Laser                           | 90 |
| 12  | Glaucomatous Optic Neuropathy                               | 91 |
| 13A | Imaging Devices   | 92 |
| 13B | Field Progression   | 95 |
| 14  | The Glaucoma Quality of Life–15 Questionnaire               | 97 |

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## **INTRODUCTION**

In November 2003, the South East Asia Glaucoma Interest Group (SEAGIG) launched the Asia Pacific Glaucoma Guidelines (APGG) in Bangkok. These Guidelines have been distributed widely in print and CD-ROM format and are available at no cost on the SEAGIG website, at: [www.seagig.org](http://www.seagig.org). The APGG have formed the basis of the curriculum for SEAGIG's development of the vast IMAGE educational resource, which, when launched progressively through 2007 with the help of SEAGIG's official publication the Asian Journal of Ophthalmology, attracted over 200,000 visits to the SEAGIG website monthly.

This success suggests that the Guidelines have achieved their goals and met the needs of ophthalmologists, ancillary eye care workers, and educators, as well as government agencies and non-governmental organisations. Five years later, as promised in 2003, this 2nd Edition of the APGG will be released, this time in Seoul, Korea, bringing new evidence and advances in glaucoma management to the same audiences, in the same user-friendly format.

By increasing awareness and updating clinical knowledge about glaucoma across the Asia Pacific region, the Guidelines aim to reduce visual disability caused by this group of eye diseases, and to provide a rational basis for glaucoma management in a cost-effective manner. Once again, we hope the Guidelines will facilitate SEAGIG's growth as a mutually supportive ophthalmic community.

The establishment of best-practice methodologies throughout the Asia Pacific region continues to represent a unique challenge, given the diverse health care service systems and the wide range of available resources. The APGG Working Party has collaborated closely and widely, with invaluable assistance from the Review Group, to compile information and recommendations to assist comprehensive ophthalmologists, general health care and eye care professionals, and health care policy makers to deliver effective glaucoma management to their communities.

Relying on published evidence wherever possible, and on expert consensus when definitive evidence was not available, the 2nd Edition of the Guidelines are as up-to-date as possible.

Critical to the development of the 2nd Edition of the APGG has been the strong support of the many glaucoma subspecialists in the Working Party, the extended Working Party, and the Review Group, as well as the generous educational grants from industry: Pfizer Inc, Allergan Inc, Alcon Laboratories Inc, and Merck Inc. This sponsorship permitted the Working Party to meet face-to-face, as well as the subsequent publication, and distribution of the 2nd Edition of the APGG.

The Guidelines aim to be sensitive to the wide variations in human, structural, and equipment resources available throughout the Asia Pacific region, as well as the ethnic diversity of the local communities. Even though what is applicable in one country or location may not be in another, there is an optimal standard of care deserved by all our patients and communities; the Guidelines try to identify this standard.

Developed during a time of rapidly expanding medical technology, knowledge, and skills in a changing environment, the 2nd Edition of the APGG aims to meet the increasing public awareness and expectations in all our communities and, as the population ages (even in less developed societies), the increasing demand for high-level care for a greater

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number of people over extended periods of time. Available resources have been unable to expand proportionally to meet these demands; cost containment is an inescapable reality. Every treatment or investigation that is undertaken reduces the capacity to implement an intervention that could benefit another patient. As clinicians, what we do for our patients needs to be demonstrated to be effective. If not, we must recognise it to be only partly proven or as yet unproven. Guided by this knowledge, our lines of enquiry and research can be channelled appropriately.

The easy-to-read format of the 1st Edition of the APGG has been retained; each section answers questions of 'Why?', 'What?', 'When?', and 'How?'.

As with all treatment guidelines, this publication is not a prescription for automated care. By adapting the Guidelines to the patient before you, bearing in mind individual needs, and the socio-economic environment and medical facilities available, plus your own experience, we hope the 2nd Edition of the APGG helps you to achieve the hallmark of excellent care.

***Ivan Goldberg***

Chair, Asia Pacific Glaucoma Guidelines 2nd Edition Working Party

On behalf of the South East Asia Glaucoma Interest Group

## EPIDEMIOLOGY OF GLAUCOMA IN ASIA<sup>1,2</sup>

The age-specific prevalence rate of GON is the highest amongst people of African origin, and is probably the lowest among Caucasians of European origin, such as the populations of Australia and New Zealand. Asian populations seem to have rates of GON that are intermediate between these 2 groups. European- and African-derived people predominantly have POAG, whereas rates of PACG are higher amongst East Asians. Although a direct and exact comparison of POAG rates is difficult, it is likely that POAG has a similar prevalence in Asian people to that seen in European populations. The higher rate of GON in Asians is probably attributable to the excess of PACG.

Incidence rates of symptomatic 'acute' AC (given as cases/100,000 persons/year for the population aged 30 years and older) range from 4.7 in Europe (Finland)<sup>3</sup> to 15.5 in Chinese Singaporeans.<sup>4</sup> Malay and Indian people in Singapore have lower rates than do Chinese Singaporeans (6.0 and 6.3, respectively).<sup>5</sup>

Detailed population surveys in Mongolia found glaucoma to be the cause of blindness in 35% of affected adults (compared with cataract in 36%).<sup>6</sup> Among Chinese Singaporeans, a population survey found that 60% of adult blindness was caused by glaucoma.<sup>7</sup> Cautious extrapolation of these data suggests that glaucoma probably causes blindness in approximately 1.7 million people in China. PACG is responsible for the vast majority (91%) of these cases. Secondary glaucoma is the most frequent cause of unocular blindness.<sup>8</sup> Glaucoma is the leading cause of registered, permanent blindness in Hong Kong (16%).<sup>9</sup> In Japan, diabetic retinopathy (18%), cataract (16%), and glaucoma (15%) are the leading causes of blindness.<sup>9</sup> In Hyderabad, India, a population survey found the leading causes of blindness to be cataract (30%) and retinal disease (17%). Glaucoma was the cause of blindness in 12% of cases.<sup>10</sup>

Advancing age is the single most consistent risk factor for glaucoma, whether it is POAG, PACG, or secondary glaucoma.<sup>4,5,7,10-15</sup> Female gender is recognised as a major predisposing factor for the development of PACG.<sup>7,15,16</sup> There appears to be no gender difference for POAG. Chinese ethnic origin confers a higher risk of AC compared with Malay and South Indian people.<sup>4,5</sup> Studies in urban centres generally find POAG prevalence exceeds PACG,<sup>7,15</sup> whereas in rural areas, the reverse is true.<sup>11,17</sup> Due to lack of care, blindness rates are higher in rural than urban centres.

AC is associated with a hypermetropic refractive state, although this association is not consistent, as cases do occur in people with myopia. A shallow anterior chamber predisposes toward AC.<sup>18</sup> The depth of the anterior chamber reduces with age, tends to be shallower in women than in men, and is highly heritable.<sup>19-21</sup> There may also be an association between myopia and POAG.<sup>22</sup> In Japan, 92% of POAG occurs with 'normal' IOP (<21 mm Hg).<sup>23</sup>

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## 1.1 PATIENT ASSESSMENT

The purpose of this section is to describe the initial assessment of a patient in whom glaucoma is suspected, from the perspective of clinicians in both developed and developing countries. Inevitably, some sections will have more relevance to one or other setting. However, time taken to examine a patient is seldom wasted. The initial consultation lays the foundations for successful management of the patient.

Assessment of a child with suspected glaucoma raises specific and distinct questions. Such a child should be referred urgently to a specialised centre (Appendix 1).

### Why?

The aims during the initial assessment are:

- To determine whether or not glaucoma is present
- To assess risk factors for glaucoma to determine the likelihood of glaucoma developing or glaucoma progressing
- To exclude or confirm alternative diagnoses
- To identify the underlying mechanism(s) of damage to guide appropriate management
- To plan a management strategy
- To determine whether treatment is appropriate based on risk factors
- To identify suitable forms of treatment, and to exclude those that are inappropriate

### What?

Understand the natural history of the glaucomas in your region. The initial assessment can be divided into 3 phases:

- History
- Examination
- Investigations

## HISTORY

### Key Points

- Most glaucomas are asymptomatic until advanced
- Assess medical and social factors that will affect treatment decisions
- Assess the risk factors for glaucoma (family history of glaucoma/visual problems)
- Younger age means a longer exposure to glaucoma and its treatment

### Past Ophthalmic History

#### Consider

- Previous medications (especially glaucoma medications, steroids), trauma, or previous eye surgery or laser treatment
- Allergies/adverse reactions to medications

### **SYMPTOMS OF ACUTE INTRAOCULAR PRESSURE ELEVATION OR GLAUCOMA**

- Intermittent/episodic blurring, discomfort, frontal headache:<sup>1</sup>
  - may be due to acute IOP elevation in the presence of AC, or Posner-Schlossman, pigment dispersion, or PXF syndromes
- Glare and coloured rings around lights from corneal oedema:
  - may be due to acute IOP elevation
  - needs to be differentiated from migraine
- Poor light/dark adaptation and/or difficulty tracking fast-moving objects (golf/tennis balls):
  - may be due to retinal nerve fibre damage

## Past Medical History

### Consider

- Factors that will affect life expectancy and adherence with treatment
- Exclude past history of systemic conditions that may mimic glaucoma but are not progressive, such as haemodynamic crises (postpartum haemorrhage, blood transfusions, severe trauma), anterior ischaemic optic neuropathy that may cause optic disc pallor and cupping, or intracranial pathology<sup>2-5</sup>
- Systemic conditions relevant to glaucoma history taking (Table 1.1)

**Table 1.1.** Factors to consider when taking a medical history from a patient with suspected or established glaucoma.

| System                  | Condition  |
|-------------------------|--|
| Respiratory             | Asthma and COPD associated with hyper-responsive airways and/or reduced lung capacity will limit the use of topical $\beta$ -blockers  |
| Cardiovascular          | Cardiac arrhythmias (heart block) may preclude the use of topical $\beta$ -blockers or $\alpha$ -agonists<br>Systemic hypotension<br>Systemic hypertension — over-treatment (causing hypotension, particularly at night) may worsen glaucoma risk and progression; systemic $\beta$ -blockers may mask elevated IOP<br>Vasospastic tendency (migraine, Raynaud's phenomenon) may be associated with an increased incidence and severity of glaucoma, especially NTG<br>Previous episodes of low blood pressure, haemodynamic shock, or significant blood loss requiring blood transfusion<br>Possible interaction between systemic and topical medications |
| Endocrine               | Diabetes — increasingly prevalent and associated with OAG and neovascular glaucoma<br>Thyroid eye disease<br>Pituitary tumours   |
| CNS                     | Previous CVA/head injury/pituitary lesions (VF loss)<br>Early dementia — affects adherence, understanding, and insight into the disease  |
| Musculoskeletal         | Arthritis (osteo-, rheumatoid) may severely affect the ability to administer eye drops   |
| Urogenital              | Urinary stones may limit use of systemic CAIs  |
| Pregnancy and lactation | Present or possible, renders all interventions potentially hazardous (Appendix 2)  |

### **MEDICATION**

Use of any current medication needs to be considered, along with certain specific past medications, including:

- Steroids — any route of administration is associated with OH and OAG; sometimes found in traditional medicines
- Glaucoma eye drops (prolonged use may increase the likelihood of trabeculectomy failure)
- Anticholinergics/tricyclic antidepressants — can cause AC
- Anticonvulsants:
  - topiramate: can cause acute AC
  - vigabatrin: linked to nasal peripheral VF loss without disc changes
- Systemic  $\beta$ -blockers/CCBs — may interact with topical  $\beta$ -blockers
- $\alpha$ -Agonists are contraindicated for:
  - patients taking MAOIs (prescribed for depression, migraine prophylaxis, or Parkinson's disease)
  - infants and children
- Check for sulphur allergy prior to using CAIs

**Note:** See Appendix 3 for systemic medications that may induce AC.

### Socioeconomic Factors

**Consider**

- How regularly can the patient attend?
- Can the patient afford and comply with treatment?
- How will having glaucoma affect the patient’s life/work/family (disease and treatment)?

### Family History

**Consider**

- What is the disease type and course in the family? (See Epidemiology).

## EXAMINATION/INVESTIGATIONS

Examination requires appropriate equipment, sufficient training in examination techniques, and accurate and reliable recording of findings. While resources vary widely across the region, there is a minimal acceptable standard of equipment and training.

|  |  |
|--|--|
| <p><b>MINIMAL<br/>ACCEPTABLE<br/>RESOURCES FOR<br/>EXAMINATION</b></p> | <ul style="list-style-type: none"> <li>• A slit lamp with indirect lens (60 to 90 D) and/or direct ophthalmoscope</li> <li>• An automated perimeter</li> <li>• A gonioscope that allows indentation gonioscopy</li> <li>• A Goldmann-style applanation tonometer; Schiötz or Maklakov tonometers are not generally acceptable</li> </ul> |
|--|--|

|  |  |
|--|--|
| <p><b>WHEN THE<br/>PATIENT CANNOT<br/>GET TO A<br/>SLIT LAMP</b></p> | <ul style="list-style-type: none"> <li>• A portable hand-held slit lamp may be very useful</li> <li>• In the absence of a portable slit lamp, a jeweller’s loop or an operating loop with a torchlight will allow a reasonable anterior segment examination</li> <li>• A direct ophthalmoscope set at +10 to +12 D will allow anterior segment examination</li> <li>• Measuring IOP in this setting is best performed with a Tonopen or Perkins tonometer</li> </ul> |
|--|--|

## SLIT-LAMP EXAMINATION — GOLDMANN APPLANATION TONOMETRY

- |              |   |
|--------------|---|
| <b>Why?</b>  | <ul style="list-style-type: none"> <li>• IOP is the only modifiable risk factor for glaucoma</li> </ul>   |
| <b>What?</b> | <ul style="list-style-type: none"> <li>• Goldmann-style applanation tonometry</li> </ul>  |
| <b>When?</b> | <ul style="list-style-type: none"> <li>• Every visit</li> </ul>   |
| <b>How?</b>  | <ul style="list-style-type: none"> <li>• Check calibration of tonometer (Appendix 4)</li> <li>• Disinfect prism tip and remove disinfectant</li> <li>• Keep eyelashes out of the way (avoid pressure on eye)</li> <li>• Anaesthetise the cornea</li> <li>• Instil fluorescein</li> <li>• Gently touch the tip to the central cornea with the observer looking through the slit-lamp eyepiece just prior to the tip making contact</li> <li>• Adjust the gauge until the split tear meniscus just touches on the inside</li> </ul> |

**Note:** Look for the white split ring that fluoresces when the tip touches the cornea.

## Factors Associated with Intraocular Pressure

(Tables 1.2 and 1.3, and Appendix 5)

**Table 1.2.** Factors affecting measured intraocular pressure.

| Factor                   | Mechanism   |
|--------------------------|---|
| Circadian cycle          | IOP follows a circadian cycle, which varies with posture and is often highest when the patient is horizontal at night<br>Diurnal (day time) IOP is often highest in the morning and reduces toward evening; the normal diurnal variation is 3 to 6 mm Hg  |
| CCT                      | Thicker corneas are associated with artificially elevated IOP measurements, and thinner corneas with artificially depressed IOP measurements<br>While correction nomograms based solely on corneal thickness are neither valid nor useful in individual patients, <sup>6</sup> the clinician needs to put the measured IOP into context |
| Blood pressure           | IOP is positively associated with systemic blood pressure <sup>7-9</sup>  |
| Intra-abdominal pressure | Increased intra-abdominal pressure by playing wind instruments or Valsalva manoeuvre increases episcleral venous pressure and IOP   |
| Age                      | For individuals, IOP usually rises with age <sup>10</sup>   |
| Exercise                 | Exercise may increase IOP (head-down yoga positions) or decrease IOP (by dehydration and/or acidosis)   |
| Lifestyle                | Large-volume rapid fluid intake increases IOP, while alcohol and marijuana depress IOP  |
| Posture                  | Head-down position doubles IOP <sup>11</sup><br>Supine or prone position increases IOP  |

**Table 1.3.** Measurement errors associated with Goldmann-style applanation tonometry.<sup>12</sup>

| Error   | Possible cause  |
|---|---|
| IOP reading artificially low                            | Insufficient fluorescein in tear film<br>Microcystic epithelial corneal oedema  |
| IOP reading artificially high                           | Excessive fluorescein in tear film<br>Eyelid pressure on globe from blepharospasm<br>Digital pressure on globe to hold lids apart<br>Obese patient<br>Patient straining to reach chin/forehead rest<br>Patient breath-holding<br>Patient wearing constricting clothing around neck (tight shirt collar ± tie for men)<br>Hair lying across cornea distorting mires<br>Lens-corneal apposition |
| Technical difficulties (interpret results with caution) | Corneal abnormalities (scars, graft, oedema, keratoconus)<br>Marked corneal astigmatism<br>Small palpebral aperture<br>Nystagmus<br>Tremor  |

## Anterior Segment

When examining the anterior segment, pay attention to the following areas:

### ***Globe surface***

- Episcleral blood vessels
- Conjunctival injection (papillae or follicles)

### ***Cornea and anterior chamber***

- Pigment on corneal endothelium (pigment dispersion)

- Peripheral ACD (van Herick technique)
- Central ACD
- Evidence of inflammation (keratic precipitates, anterior chamber flare and cells)
- Descemet's membrane rupture (Haab's striae)
- Corneal diameter and curvature

### ***Iris***

- Mid-dilated poorly reactive (post-AC attack)
- Isolated zones of patch atrophy or spiralling
- Rubeosis iridis
- Synechiae
- Ectropion uvea
- Configuration in relation to lens
  - PXF material on pupil edge
  - pigment deposit on anterior surface (PXF or pigment dispersion)
  - transillumination defect (PXF — peripupillary; pigment dispersion — mid-peripheral), loss of pupillary ruff (early sign of PXF)
- Displaced pupil with iris atrophy and/or hole(s) [ICE syndrome, Axenfeld-Rieger syndrome]
- Pigmented iris nodule (ICE syndrome, Cogan-Reese type)

### ***Lens***

- PXF material
- Lens opacity
- Phacodonesis
- Glaukomflecken (past acute high IOP)
- Early PXF changes on lens after pupillary dilatation

## **GONIOSCOPY**

(Appendix 6)

### ***Why?***

- Detect AC and secondary glaucomas

### ***What?***

- Angle width and characteristics (see below)

### ***When?***

- Initially for all
- Repeated more frequently for patients with AC

### ***How?***

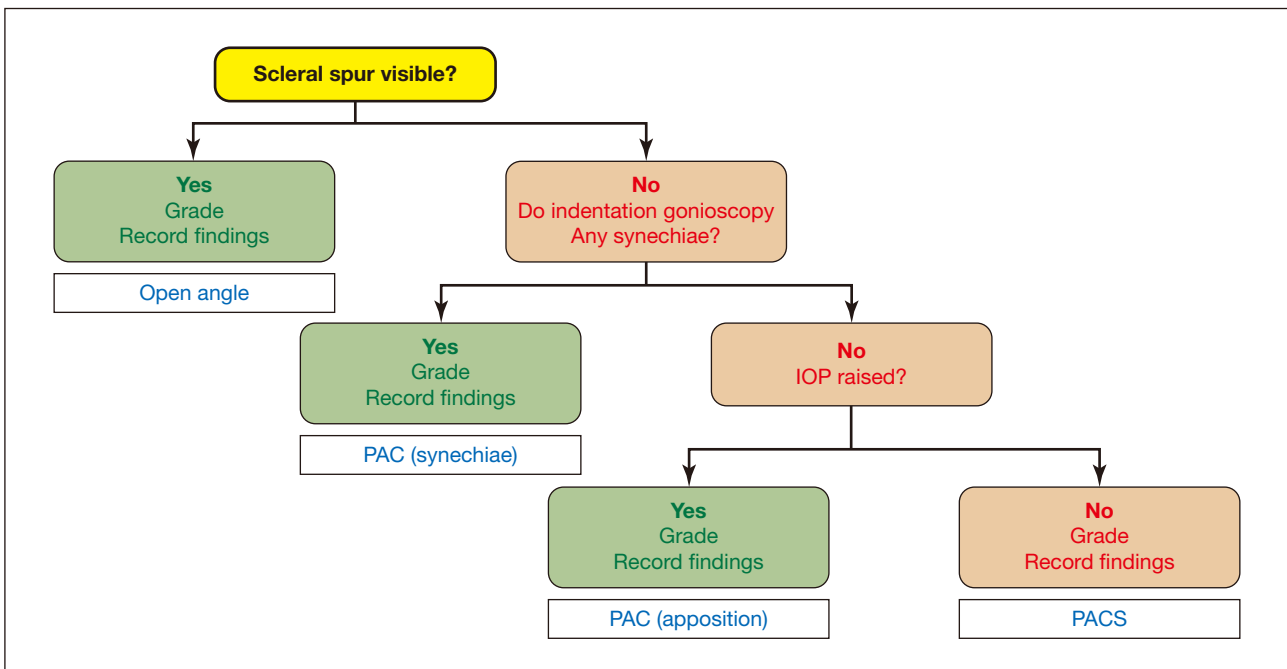
Gonioscopy should be performed to look for iridotrabecular contact. Gonioscopy needs to be performed in a dark room with a small slit-lamp beam:<sup>13</sup>

- Minimal room illumination
- Good anaesthesia
- Shortest slit practicable
- High magnification
- Dim slit illumination
- Set slit lamp on upper cornea, beam off-centre 30° to 45° nasally
- If necessary, elevate upper lid
- Place lens gently on eye while looking through slit lamp (as if you are doing tonometry) — no gel needed with Zeiss-type lenses
- Look through the upper mirror (inferior angle) as you place lens on eye, stop pushing when you can see the iris
- Move slit-lamp beam inferiorly (avoid pupil) to examine superior angle

- Turn beam 90° and move on axis
  - Move to nasal side (temporal angle), then to temporal side (nasal angle)
  - Record findings on gonioscopic gonioscopy
  - In the presence of appositional closure, indentation should be performed to look for PAS
  - It may be necessary to alter the position of the mirror or the position of gaze to look over a convex iris to visualise the angle
- (Figure 1.1 and Appendix 6)

**Tip:** If you cannot find the angle structures, use a bright wide slit (parallel to the mirror) at low magnification. Once you have found the angle structures, turn the illumination down, shorten and narrow the slit, and look for the change in iris/angle configuration. Avoid light entering the pupil. You may need to wait a minute or so. If suspicion of AC is high then, in some cases, you may need to wait for 2 to 3 minutes after reducing the illumination.

**Figure 1.1.** Gonioscopy flow diagram.



### Angle Closure Signs

- PAS
- Pigment patches over TM (evidence of irido-trabecular contact)
- Iris insertion above scleral spur

### Abnormal Open Angles

- TM with pigment, pseudoexfoliative materials, new vessels, precipitates, or abnormal iris processes
- Wide ciliary body band or sclera (angle recession, cyclodialysis cleft)
- Schlemm's canal with blood reflux

## OPTIC NERVE HEAD AND RETINAL NERVE FIBRE LAYER

### Why?

- Defines glaucoma

### What?

- Scleral ring (disc size and shape)
- Neuroretinal rim
- Disc haemorrhage
- RNFL defect

- PPA
- Vascular pattern

**When?**

- Every visit

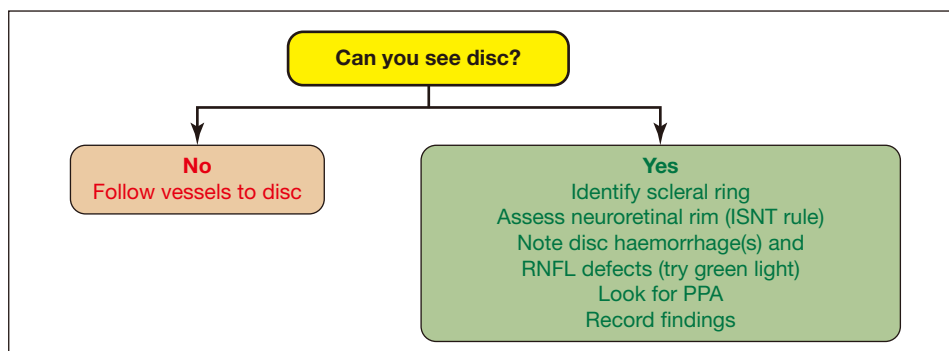
**How?**

Consider non-glaucomatous optic neuropathies. Differentiate compressive optic neuropathy and anterior ischaemic optic neuropathy from glaucoma, especially giant cell arteritis;<sup>14,15</sup> both can cause pale cupped disc and field loss.

- Slit lamp
- Very thin, bright beam for disc measurement
- Dimmer beam for clearer lenses or pseudophakos
- Indirect slit-lamp lens (60 to 90 D)
  - in myopia, optic disc assessment with contact lens (like a 3-mirror lens) would be better than non-contact lens for appreciating glaucomatous changes
- It is important to gain a stereoscopic view (best when dilated — recommended if safe)
- Red-free (green) illumination may help assessment of RNFL
- Direct ophthalmoscopy or slit-lamp view through undimpled Koeppel lens if pupil is small and not able to be dilated

(Figure 1.2 and Table 1.4)

**Figure 1.2.** Optic disc examination flowchart.



**Table 1.4.** Normal vertical cup-disc ratios for vertical disc diameter.<sup>16</sup>

| Disc diameter | Mean CDR | 95% CI    |
|---------------|----------|-----------|
| ≤1.0          | 0.26     | 0.20-0.32 |
| 1.2           | 0.33     | 0.32-0.34 |
| 1.4           | 0.39     | 0.39-0.39 |
| 1.6           | 0.45     | 0.45-0.45 |
| 1.8           | 0.50     | 0.50-0.50 |
| ≥2.0          | 0.55     | 0.53-0.57 |

**Disc Recording**

- Draw optic disc (large), rim, key vessels that define rim, and peripapillary signs
- Document disc size, ie, whether large, average, or small
- Draw notches, shelving, loss to rim-clock hours
- Record whether RNFL is visible and assess for wedge(s) or slit defects
- Record vertical CDR in the narrowest part of the rim — consider recording the rim-disc ratio at key parts of the rim
- Record splinter haemorrhages, PPA (β-zone), bearing of circumlinear blood vessels, blood vessels bayoneting

**Tip:** Disc margin is *INSIDE* the peripapillary scleral ring of Elschnig.

Appropriate lens magnification correction for Volk lenses: Superfield 1.5x, 90 D 1.3x, 78 D 1.1x, Super 66 D 1.0x.

## **DISC SIZE**

- Disc size is variable (Table 1.5) — large discs have large CDRs, although the neuroretinal rim area is normal; while a large CDR may not be pathological, pathological rim loss can be missed in a small disc, especially if generalised
- CDR is related to optic disc size and is not important for diagnosis of glaucoma; asymmetry of CDR of  $>0.2$  between 2 eyes is suspicious unless disc size is similarly asymmetrical
- Disc size can be measured by:
  - using the small size spot ( $5^\circ$ ) of a direct ophthalmoscope, which approximates the average disc size, to estimate whether a disc is large or small
  - adjusting the vertical beam of a slit lamp to the diameter of the optic disc
  - using HRT

**Table 1.5.** Percentiles for vertical cup-disc ratios in non-glaucomatous eyes.\*

| Percentile | Disc size |        |       |
|------------|-----------|--------|-------|
|            | Small     | Medium | Large |
| 50th       | 0.35      | 0.44   | 0.55  |
| 95th       | 0.56      | 0.65   | 0.71  |
| 97.5th     | 0.59      | 0.68   | 0.74  |
| 99th       | 0.62      | 0.72   | 0.80  |

\* The data were derived by planimetric measurement of disc size and CDR.

## **NEURORETINAL RIM**

The rim is more important than the cup. The rim width is defined by the area that extends from the scleral ring to where the rim falls just below the level of the scleral ring.

The cardinal feature of GON is a loss of tissue from the inner edge of the rim.

Features that should raise suspicion that glaucomatous damage has already occurred include:<sup>17</sup>

- Diffuse loss or notching of the rim (especially to the disc margin)
- Haemorrhage crossing the rim
- Undercutting of the rim (also found in many physiological large cups)
- Asymmetry of rim width between the eyes in the absence of asymmetry of disc size
- An abnormally thin rim in 1 or 2 sectors

Check the ISNT rule.

**Tip:** An approximate rule is that a vertical CDR of  $>0.7$  (note importance of disc size) or loss of rim to the disc margin anywhere outside the temporal sector strongly suggests glaucoma. This rule may not apply if the disc is extremely large or very tilted.

## **ISNT RULE — A GUIDE**

Normally, the thickest to thinnest parts of the neuroretinal rim of the optic disc are Inferior  $>$  Superior  $>$  Nasal  $>$  Temporal (ISNT). Any variation from this may help to detect glaucomatous damage. The ISNT rule may not be followed in up to 50% of normal discs in certain populations.<sup>18</sup> The essence of the ISNT rule is the 'T': in almost all normal eyes, independent of ethnicity, the narrowest part of the rim is in the temporal  $60^\circ$ .

**Disc  
HAEMORRHAGE**

- Important risk factor for glaucoma progression
- Suggests ongoing damage to the optic nerve head
- Independent risk factor for development of glaucoma<sup>19</sup>
- Presence of disc haemorrhage in OH increases the risk of conversion to POAG by 6 times (univariate analysis) and 4 times (multivariate analysis)
- Recurrent haemorrhages increase the risk of optic nerve damage by 3 to 4 times compared with single haemorrhage<sup>20,21</sup>

**OPTIC DISC PHOTOGRAPHY AND IMAGING**

The most reliable way to detect glaucomatous optic nerve progression may be with serial optic nerve head photographs or imaging technologies (Table 1.6).

If a fundus camera is available, all glaucoma suspects and patients with glaucoma should have their optic discs photographed (preferably stereoscopically) at the time of diagnosis. These images should be used as an aid in follow-up examinations.

**Table 1.6.** Optic disc/retinal nerve fibre layer assessment.

| Qualitative                                    | Quantitative                                     |
|--|--|
| Direct ophthalmoscopy                          | Disc photography with digitalisation             |
| Slit-lamp indirect ophthalmoscopy              | Stereo disc photography with optic disc analysis |
| Disc photography                               | HRT  |
| Simultaneous stereophotography                 | GDx  |
| RNFL photography (red-free fundus photography) | OCT  |

**Optic Disc and Retinal Nerve Fibre Layer Imaging Technologies**

Currently, sensitivity and specificity for glaucoma diagnosis for 3 technologies (HRT, OCT, and GDx variable corneal compensation) is approximately 80%. Agreement between all 3 technologies is approximately 25% to 40%.

The WGA consensus on structural and functional tests states: “The current literature does not provide the requisite evidence to validate any of these imaging instruments for widespread clinical use. Currently, in the hands of an experienced clinician who understands the strengths and limitations of the instruments, information may be helpful in many clinical situations.”

**VISUAL FIELD EXAMINATION**

**Why?**

- Defines state of optic nerve function
- Defines visual impairment

**What?**

- Automated perimetry with machines having appropriate normative database

**When?**

- When glaucoma is suspected at examination

**How?**

- It is very important to understand the correct procedure for performing VF testing
- Users should read and be familiar with the perimetry manual

## **TIPS FOR BETTER VISUAL FIELDS**

- The patient should be carefully instructed in a language they understand before and during the examination
- During and at the end of the test, the patient should be told how well they have performed and feedback should be given to them about the results of their test so that, in future, they can improve on their test performance
- The technician makes the best assessment of performance
- VF test performance usually improves over the first 2 to 3 tests
- Check pupil size and note any change
- Use appropriate correction for near vision

**Note:** See Appendix 7.

## **Characteristics of Glaucomatous Visual Field Defects**

- Asymmetrical across horizontal midline\*
- Located in mid-periphery\* (5° to 25° from fixation)
- Reproducible
- Not attributable to other pathology
- Clustered in neighbouring test points (localised)
- Defect should correlate with the appearance of the optic disc and neighbourhood

\* *Early/moderate cases.*

## **FUNCTION- SPECIFIC TESTS**

Function-specific tests of the optic nerve may detect glaucomatous defects prior to standard automated perimetry but the increased sensitivity is accompanied by reduced specificity. These tests may be useful for monitoring early glaucoma:

- Short wavelength automated perimetry (blue-on-yellow perimetry): for detecting damage in koniocellular pathway
- Frequency doubling technology perimetry: for detecting damage in the magnocellular pathway

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## 1.2 RISK CATEGORIES AND TREATMENT TARGETS

### Why?

- Tailor treatment to likelihood of future visual disability, depending on disease stage, risk factors for progression, and patient's overall health status and life expectancy

### What?

#### Glaucoma with High 5-year Risk for Progressive Visual Loss or High 5-year Risk for Visual Disability

Moderate to advanced GON with correlating VF loss and:

- Demonstrated progression over a short time
- Higher IOPs
- Bilateral VF loss<sup>1</sup>
- Pigmentary and PXF glaucoma<sup>1</sup>
- Very advanced VF loss, fixation threat, or glaucoma-related visual disability
- Young age with advanced disease
- Secondary glaucoma
- ACG

#### Glaucoma with Moderate 5-year Risk for Visual Loss or Glaucoma Suspect with High Risk for Visual Loss

- Mild GON with correlating early VF loss and higher IOP
- Mild-to-moderate glaucoma with low IOP<sup>2</sup>
- PAC with high IOP and PAS
- Younger age

#### Glaucoma Suspect at Moderate Risk for Visual Loss

- Fellow of eye with established GON (excluding secondary unilateral glaucomas)<sup>3</sup>
- OH with multiple risk factors (thin CCT, high IOP, suspicious disc)<sup>4</sup>
- GLC gene mutations associated with severe glaucoma
- Recurrent optic disc haemorrhages
- PXF syndrome
- Younger age

#### Glaucoma Suspect or Other Condition with Low Risk for Glaucomatous Visual Loss

##### *More important*

- OH<sup>3,4</sup>
- Older age<sup>3,4</sup>
- PACS (anatomically narrow angle with no PAC signs or raised IOP)
- Pigment dispersion syndrome with normal IOP
- Glaucoma suspect disc, including disc asymmetry
- Family history of glaucoma

##### *Less important*

- Steroid responder, steroid user
- Myopia
- $\beta$ -Zone PPA
- Diabetes mellitus
- Uveitis
- Systemic hypertension

Table 1.7. shows the risk factors for OAG incidence and progression.

**Table 1.7.** Risk factors for open angle glaucoma incidence and progression.

| <b>Risk factors for OAG incidence</b>         |  |
|---|--|
| <i>Genetic risk factors</i>                   | <i>Other risk factors</i>              |
| Higher IOP                                    | Optic disc haemorrhage                 |
| Older age                                     | Larger CDR                             |
| Ethnicity                                     | Higher VF sensitivity deviation        |
| Family history                                | Diabetes mellitus                      |
| Lower CCT                                     | Vasospasm                              |
| Contralateral disease                         | Migraine                               |
|   | Raynaud's phenomenon                   |
|   | Sleep apnoea (uncertain)               |
| <b>Risk factors for OAG progression</b>       |  |
| <i>Genetic risk factors</i>                   | <i>Other risk factors</i>              |
| Higher IOP (at baseline and during follow-up) | Optic disc haemorrhage                 |
| Older age                                     | Bilateral OAG                          |
| Family history                                | More advanced glaucomatous vision loss |
| PXF syndrome                                  |  |
| Lower CCT                                     |  |
| <i>Vascular risk factors</i>                  |  |
| Lower ocular perfusion/low blood pressure     |  |

The presence of multiple risk factors proportionally increases glaucoma risk and may elevate a patient into a higher risk category.

**When?**

- Each visit

**How?**

- Modifiable mechanisms for risk factors

**OBJECTIVE**

To maintain functional vision throughout the patient's lifetime with minimal effect on QOL.

**SETTING GOALS**

**Goal of Intervention Is Risk Factor Reduction**

- IOP
- Angle control; elimination of AC
- Treatment of predisposing disease/factors (diabetes mellitus, uveitis, steroids)

**Note:** Improving ocular perfusion is a potentially beneficial, but as yet untested, goal.

**RATE OF NEURAL LOSS AND LIFE EXPECTANCY**

- Treatment sets goals to preserve sight and maintain visual abilities
- This should be a balance of disease stage, progression rate, and life expectancy of the patient (the time course over which the disease is expected to run)
- A slow disease process in an elderly patient results in little progression during his/her life expectancy; a fast disease process in a younger patient tends to result in blindness
- This is the balance that has to be assessed by the clinician before determination of target pressure and all glaucoma treatments

### Stage of Disease

Use the 4 risk categories above.

### Estimate Rate of Neural Loss

Higher → more aggressive risk factor reduction.

### Severity of Risk Factors

Higher or greater number → more aggressive risk factor reduction.

### Modifiers of Goals

- Life expectancy
- Ability to attend follow-up
- Diseases that prevent accurate disc or field assessment
- Treatment morbidity

## INTRAOCULAR PRESSURE CONTROL

### Intraocular Pressure Landmarks

- Presenting (untreated) IOP
- IOP in fellow normal eye in unilateral secondary glaucoma
- Population mean and SD IOP for normal eyes

### **TARGET INTRAOCULAR PRESSURE**

- Target IOP is the pressure estimated to slow or halt disease progression
- Target IOP is determined from the baseline IOP, stage of disease, estimated progression rate, and life expectancy
- When the target is achieved, the patient needs continued monitoring for structural and functional changes
- Target IOP needs to be individualised within a risk category
- The benefits of further pressure reduction need to be weighed against the risks
- Target IOP is dynamic: changing with life expectancy and risks of intervention weighed against risk of visual disability from disease process

## TREATMENT TARGETS

### Glaucoma with High Risk for Progressive Visual Loss or Visual Disability

Target pressure reduction of  $\geq 40\%$ <sup>3,5,6</sup> or 1 to 2 SD below the population mean (9 to 12 mm Hg), if achievable safely.<sup>3,6-8</sup>

### Glaucoma with Moderate Risk for Visual Loss or Glaucoma Suspect with High Risk for Visual Loss

Target pressure reduction of  $>30\%$ <sup>3,6-8</sup> or population mean, whichever is lower.

### Glaucoma Suspect at Moderate Risk for Visual Loss

- Monitor closely for change or treat depending on risk and patient preferences
- Treat if risk(s) increase(s) with target pressure reduction of  $\geq 20\%$ <sup>3,6-8</sup> or 1 SD above the population mean, whichever is lower
- The fellow eye of unilateral glaucoma may require the same target as the affected eye depending on risk and state

### Glaucoma Suspect with Low Risk for Visual Loss

Monitor, no treatment.

**Accounting for central corneal thickness when setting target intraocular pressure**

- Absolute IOP target: increase or decrease target IOP by 1 SD for every 1 SD increase or decrease in CCT
- CCT landmarks\* (Table 1.8):
  - Europeans and Chinese Singaporeans:
    - mean  $\approx$  540  $\mu$ m
    - SD  $\approx$  35  $\mu$ m
  - Japanese and Indians (urban):
    - mean  $\approx$  520  $\mu$ m
    - SD  $\approx$  30  $\mu$ m
  - Indians (rural) and Mongolians:
    - mean  $\approx$  505  $\mu$ m
    - SD  $\approx$  30  $\mu$ m
- \* For all populations: older age  $\rightarrow$  lower CCT.
- Thick cornea  $\rightarrow$  true IOP lower than GAT measurement
- Thin cornea  $\rightarrow$  true IOP higher than GAT measurement
- Percentage IOP decrease unaffected by CCT (but low CCT is risk factor for progression)

**ALTERNATIVE  
APPROACH TO  
SETTING TARGET  
INTRAOCULAR  
PRESSURE**

- Percentage IOP-lowering targets are not appropriate when presenting IOP is very high or low
- Use population IOP data to set targets relative to normal population:
  - for most populations:
    - mean —  $\sim$ 15 mm Hg
    - SD — 3 mm Hg
  - in Japan:
    - mean — 14 mm Hg
    - SD — 2 mm Hg
- Higher target
  - for most populations:
    - mean +1 SD —  $\sim$ 18 mm Hg
  - in Japan:
    - mean +1 SD — 16 mm Hg
- Moderate target
  - for most populations:
    - mean —  $\sim$ 15 mm Hg
  - in Japan:
    - mean — 14 mm Hg
- Lower target
  - for most populations, including Japan:
    - mean -1 SD —  $\sim$ 12 mm Hg
- Very low target:
  - for most populations, including Japan:
    - mean -2 SD —  $\sim$ 9 mm Hg

**Table 1.8.** Examples of central corneal thickness and target intraocular pressure by race.

| Ethnicity                | CCT ( $\mu$ m) | Example of moderate target IOP adjustment (mm Hg) |
|--------------------------|----------------|---|
| European/Chinese         | 540            | 15  |
|                          | 500            | 12  |
|                          | 580            | 18  |
| Japanese/Indian (urban)  | 520            | 14  |
|                          | 490            | 12  |
|                          | 550            | 16  |
| Indian (rural)/Mongolian | 505            | 15  |
|                          | 475            | 12  |
|                          | 535            | 18  |

## ANGLE CONTROL

The goal is to deepen the peripheral anterior chamber by:

- Iridotomy to reduce pupil block
- Peripheral iridoplasty (ALPI) to flatten the peripheral iris
- Lens extraction to reduce pupil block and/or displace the iris posteriorly
- Vitreous surgery to allow lens/iris diaphragm to fall backwards

## TREATING PREDISPOSING DISEASES

The goal is to prevent onset of GON by appropriate management of disease.

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## 2.1 INITIATION OF TREATMENT

**Why?** Glaucoma is a progressive optic neuropathy; if left untreated, the patient may go blind.

**What?** Assess the patient as a whole to individualise treatment. Aim to preserve visual function. IOP is the only known causal risk factor, and the only factor that can be manipulated effectively.<sup>1-8</sup>

Mechanisms that elevate IOP:

- Reduced aqueous outflow (functional abnormality)
- AC ± glaucoma (anatomic disorder)
- Secondary glaucomas (Appendix 8)

### **MULTIPLE MECHANISMS FOR ANGLE CLOSURE**

AC is caused by different sites of blockage (Appendix 9), which can often occur at multiple levels simultaneously and/or sequentially:

- Site 1: pupil block — iris bombé appearance
- Site 2: anteriorly rotated ciliary processes that push the iris forward and/or thick peripheral iris — plateau iris configuration, plateau iris syndrome
- Site 3: lens-induced forward displacement of the iris — volcano configuration
- Site 4: ciliary block and other posterior mechanisms

**When?** In the presence or increased likelihood of developing VF damage that will interfere with QOL during the patient's lifetime.

- Demonstrable functional and/or structural defect
  - higher risk for glaucomatous progression
    - higher IOP<sup>6,7</sup>
    - PXF<sup>6,7</sup>
    - bilateral disease<sup>6,7</sup>
    - worse VF mean deviation<sup>6,7</sup>
    - older age<sup>6,7</sup>
    - presence of disc haemorrhage<sup>6,7</sup>
    - lower diastolic perfusion pressure<sup>7</sup>
    - lower systolic blood pressure<sup>7</sup>
    - history of cardiovascular disease<sup>7</sup>
    - thinner cornea<sup>7</sup>
  - difficulty in monitoring and/or detecting progression from patient, geographic, or resource limitations
    - younger patients (disease progression more likely during lifetime)
    - fixation threatened
    - 'one-eyed' patients
- Progressive structural and/or functional damage
- High risk of developing such damage
  - OH patients at higher risk for conversion to glaucoma:
    - IOP >26 mm Hg<sup>4</sup>
    - thinner corneas (<555 µm)<sup>4</sup>
    - larger vertical CDRs<sup>4</sup>
    - older age<sup>4</sup>
    - poorer follow-up
    - glaucoma in first-degree relative

- Presence of an anatomical disorder that decreases aqueous outflow
  - AC<sup>9</sup>
  - ACG<sup>9</sup>
  - PACS/occludable angle (absolute or relative), especially in patients with:
    - AC in the fellow eye<sup>9</sup>
    - reported or confirmed family history of AC
    - need for repeated dilated examinations
    - poor access to regular ophthalmic care

## How?

### Treat the Mechanism(s)

- Remove precipitating factors
  - any drug that may elevate IOP (steroids; Appendix 3)
- IOP reduction
  - medication(s)
  - laser
  - surgery
- Correct the abnormal anatomy (AC),<sup>9</sup> if present
  - laser
  - surgery
- For secondary glaucoma, treat the underlying pathology (Appendix 8).

**Note:** Once any AC component has been appropriately treated, the management is similar to OAG.<sup>9</sup>

Collaborate with colleague(s) to treat systemic problems.

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## 2.2 MEDICAL TREATMENT

### Why?

- Effective for the majority of patients
- Generally acceptable therapeutic index
- Mostly acceptable to patients
- Widely available

### What?

- The choice depends on the mechanism of glaucoma as well as other risk factors (Section 1)<sup>1,2</sup>
  - For AC, medical treatment is only suitable for patients after PI<sup>3</sup>
- See Tables 2.1 and 2.2 for the drug classes and their mechanisms of action.

**Table 2.1.** Efficacy, safety, and dosing frequency of various drug classes.<sup>1,4-10</sup>

| Drug class                            | Daily dosage | Efficacy      | Side effects |             |
|---------------------------------------|--------------|---------------|--------------|-------------|
|                                       |              |               | Local        | Systemic    |
| PGAs*                                 | 1x           | ++++          | + to ++      | 0           |
| β-Blockers†                           | 1x to 2x     | +++           | +            | + to +++    |
| α <sub>2</sub> -Agonists‡             | 2x to 3x     | ++ to +++     | ++           | + to ++     |
| CAIs                                  |              |               |              |             |
| Topical                               | 2x to 3x     | ++            | ++           | 0 to ++     |
| Systemic                              | 2x to 4x     | ++++          | 0            | ++ to +++++ |
| Cholinergics                          | 3x to 4x     | +++           | ++++         | 0 to ++     |
| Hyperosmotic agents                   | Stat dose(s) | +++++         | 0            | ++ to +++++ |
| Proprietary fixed combinations        |              |               |              |             |
| β-Blocker + CAI                       | 2x           | +++ to +++++  | ++           | + to +++    |
| β-Blocker + PGA                       | 1x           | ++++ to +++++ | + to ++      | + to +++    |
| β-Blocker + pilocarpine               | 2x           | ++++          | ++++         | + to +++    |
| β-Blocker + α <sub>2</sub> -agonist†† | 2x           | +++ to +++++  | + to ++      | + to +++    |

\* Excluding unoprostone.

† If a patient is taking systemic β-blockers, the decrease in IOP with topical β-blockers is likely to be reduced, and the potential for systemic side effects increased: consider other drug classes first.

‡ α<sub>2</sub>-Agonists are absolutely contraindicated for patients taking MAOIs and for children <2 years.

**Table 2.2.** Mechanism of action of different drug classes.<sup>1,4-10</sup>

| Mechanism of action         | Drug class                          | Preparations   |  |
|-----------------------------|-------------------------------------|--|--|
| Increase in aqueous outflow | PGAs                                | Latanoprost<br>Travoprost<br>Bimatoprost<br>Unoprostone                              |  |
|                             | Cholinergics                        | Pilocarpine<br>Carbachol   |  |
| Decrease in aqueous inflow  | β-Blockers                          | β1-Non-selective<br>Timolol<br>Levobunolol<br>Carteolol<br>β1-Selective<br>Betaxolol |  |
|                             | α <sub>2</sub> -Adrenergic agonists | Brimonidine<br>Apraclonidine   |  |
|                             | CAIs                                | Systemic   | Acetazolamide<br>Methazolamide<br>Dichlorphenamide |
|                             |                                     | Topical  | Dorzolamide<br>Brinzolamide                        |

---

**How?****Choose the Most Appropriate Medication**

- Greatest chance of reaching target IOP
- Best safety profiles
- Minimally inconvenient
- Affordable

**Start Low and Slow**

- Minimal concentration
- Minimal frequency

**One-eyed Therapeutic Trial**

- Start treatment in the worse eye
- Check the IOP response after 2 to 4 weeks
- Assess side effects
- If acceptable and effective, make treatment bilateral

If the response is inadequate to achieve the target pressure, switch before adding:

- Switch to a different class of medication (switching within the PGA class may be useful, but adherence and regression to the mean need to be considered)
- If a drug fails to reduce IOP by  $\geq 15\%$  from baseline or produces significant side effects, a unilateral drug trial should be tried for a second drug

Use more than 1 agent only if each has demonstrated efficacy but is insufficient to reach target pressure:

- Apply this principle also to the fixed combinations
- Do not combine 2 drugs with the same pharmacological action
- Do not use 2 fixed combinations containing overlapping categories

See Figure 2.1 for the medical treatment algorithm.

See Appendix 10 for the side effects of glaucoma medications.

**Maximise the Likelihood of Adherence<sup>11</sup>**

- Establish a therapeutic alliance with the patient and their family — they need to view the doctor as an ally against the disease
- Patient and family education
- Least complex regimen
- Least disruption of lifestyle

**Teach the Technique for Eye Drop Instillation<sup>12</sup>**

- Demonstrate the preferred method, including punctal occlusion and eyelid closure for at least 3 minutes (double DOT technique – ‘don’t open the eyelid’ and ‘digital occlusion of the tear duct’)
- Ensure the patient can do it
- If  $\geq 2$  drops are to be instilled, wait at least 5 minutes between drops
- Provide educational material



- 
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## 2.3 LASER TREATMENT

### TYPES OF LASER TREATMENT

#### Open Angle Glaucoma

- Outflow enhancement: laser trabeculoplasty
- Inflow reduction: cyclophotocoagulation (usually for end-stage disease)

#### Angle Closure ( $\pm$ Glaucoma)

- Pupillary block relief: laser iridotomy
- Modification of iris contour: laser peripheral iridoplasty
- Inflow reduction: cyclophotocoagulation (usually for end-stage disease)

#### Post-filtering Surgery

- Outflow enhancement: laser suture lysis

### LASER TRABECULOPLASTY

#### Why?

- Relatively effective
- Relatively non-invasive
- Avoid medical non-adherence
- Easy to perform

#### What?

- Laser treatment to TM to increase outflow

#### When?

- Medical therapy failure or inappropriate
- Adjunct to medical therapy
- Primary treatment if appropriate

#### How?

#### Prelaser Management

- Explain the procedure
- To reduce post-treatment IOP spike or inflammation, consider 1%<sup>1</sup> apraclonidine<sup>2,3</sup> or 0.15% to 0.2% brimonidine<sup>4</sup> and/or 2% to 4% pilocarpine<sup>3</sup> (pilocarpine may decrease the blood-aqueous barrier, which may increase inflammation), and/or  $\beta$ -blocker and/or steroid drops before the procedure
- Topical anaesthesia

#### Laser

- Argon green or blue-green<sup>5</sup>
- Diode<sup>6</sup>
- Frequency-doubled Nd:YAG (532 green) — SLT<sup>7-10</sup>

#### Lens\*

- Goldmann gonioscopy lens
- Ritch trabeculoplasty lens
- CGA<sup>®</sup> LASAG/Meridien CH
- Latina SLT Gonio laser lens
- Magna View Gonio argon/diode laser lens

\* Should be coated to minimise reflection and hazard to observers.

## Placement of Laser Spots

Between pigmented and non-pigmented TM (Table 2.3. and Appendix 11).

### **LASER PARAMETERS**

|                 |   |
|-----------------|---|
| Spot size       | 50 µm (for ALT), 75 µm (for DLT), 400 µm (for SLT)  |
| Exposure time   | 0.1 sec (for ALT and DLT), 3 nsec for SLT   |
| Power           | 300-1200 mW depending on the reaction   |
| Number of spots | 30-50 spots evenly spaced over 180°<br>Treat the remaining 180° sequentially or at the same time, as required |

**Table 2.3.** Selective laser trabeculoplasty versus argon laser trabeculoplasty.

|                               | SLT   | ALT         | Ratio        |
|-------------------------------|---|-------------|--------------|
| Number of spots               | 50  | 50          |              |
| Exposure time (nsec)          | 3   | 100,000,000 | 1:33,000,000 |
| Fluence (mJ/mm <sup>2</sup> ) | 6   | 40,000      | 1:6000       |
| Power                         | 0.4-1.4 mJ                                    | 300-600 mW  | 1:100        |
| Aim                           | To target only pigment-containing cells       |             |              |
| Laser requirements            | Ultrashort pulse duration<br>Low laser energy |             |              |

## Complications

- Temporary blurred vision
- IOP spike with possible VF loss
- Transient iritis
- PAS if placement of burns is too posterior or post-laser inflammation control is not effective
- Endothelial burns if treatment is too anterior
- Chronic increase in IOP
- Suprachoroidal effusion<sup>11</sup>

## Post-laser Management

- Continue any current medical treatment
- Recheck IOP, especially if IOP spike prevention treatment is not available
- Topical steroid qid for 4 to 14 days<sup>12,13</sup> (may omit with SLT)

Closer monitoring is suggested for certain patients:

- Advanced glaucoma with severe VF loss
- One eye
- High pre-laser IOP
- Previous laser trabeculoplasty

## Repeat Treatment

Initial treatment may not be long lasting. Laser trabeculoplasty can be repeated, especially in eyes that have shown a prolonged response to previous treatment.<sup>14</sup> There is suggestive evidence that SLT is relatively safe to repeat.<sup>15</sup>

## IRIDOTOMY

### **Why?**

- Effective
- Relatively non-invasive
- Preferable to surgical iridectomy

**What?**

- Laser treatment to connect the anterior and posterior chambers to relieve pupillary block

**When?**

- PAC — pupil block significant
- PACG — pupil block significant
- PACS (absolute):
  - PAC in the fellow eye
- PACS (relative):
  - need for repeated dilated examinations
  - poor access to regular ophthalmic care
  - confirmed family history of PACG

**How?**

**Prelaser Management**

- Explain the procedure
- Instil 2% or 4% pilocarpine
- To reduce post-treatment IOP spike/inflammation, consider 1% apraclonidine<sup>16</sup> or 0.15% to 0.2% brimonidine,<sup>16</sup> and/or  $\beta$ -blocker, and/or oral CAI, and/or steroid drops before the procedure
- Topical anaesthesia
- Topical glycerine, if the cornea is oedematous
- Superior one-third of peripheral iris (beneath upper lids) desirable

**Laser**

- Nd:YAG<sup>17</sup>
- Argon<sup>17</sup> or krypton
- ‘Sequential’ laser — argon followed by Nd:YAG

**LASER  
PARAMETERS FOR  
ARGON LASER**

**Preparatory stretch burns**

|               |                 |
|---------------|-----------------|
| Spot size     | 200-500 $\mu$ m |
| Exposure time | 0.2-0.5 sec     |
| Power         | 200-600 mW      |

**Penetration laser burns**

|               |             |
|---------------|-------------|
| Spot size     | 50 $\mu$ m  |
| Exposure time | 0.02 sec    |
| Power         | 800-1000 mW |

**For pale blue or hazel iris**

First step, to obtain a gas bubble:

|               |            |
|---------------|------------|
| Spot size     | 50 $\mu$ m |
| Exposure time | 0.5 sec    |
| Power         | 1500 mW    |

Second step, penetration through the gas bubble:

|               |            |
|---------------|------------|
| Spot size     | 50 $\mu$ m |
| Exposure time | 0.05 sec   |
| Power         | 1000 mW    |

**For thick dark brown iris (chipping technique)**

|               |               |
|---------------|---------------|
| Spot size     | 50 $\mu$ m    |
| Exposure time | 0.01-0.02 sec |
| Power         | 1500-2500 mW  |

Choose and modify parameters depending on individual response

## **LASER PARAMETERS FOR Nd:YAG LASER**

- Energy: 2 to 5 mJ; use minimum energy, 1 to 3 pulses per burst (lens damage possible above 2 mJ per pulse)
- Focus the beam within the iris stroma rather than on the surface of the iris
- Choose an iris crypt or an area of thin iris
- Can be effectively combined with argon laser
- To facilitate penetration of a uniformly thick iris, argon laser pretreatment can:
  - coagulate
  - stretch
  - thin the target area

## **LASER PARAMETERS FOR 'SEQUENTIAL' LASER — ARGON FOLLOWED BY Nd:YAG LASER**

### **Preparatory burns — argon laser (chipping technique)**

|               |  |
|---------------|--|
| Spot size     | 50 µm  |
| Exposure time | 0.02-0.05 sec  |
| Power         | 600-900 mW (depends on iris pigmentation. Lower power for darker irides) |

Apply preparatory burns through the iris stroma, until iris pigment epithelium reached (pigment puff)

### **Penetration laser burns — Nd:YAG laser**

|                 |  |
|-----------------|--|
| Power           | 3-8 mJ   |
| Number of spots | As required, until hole of adequate size created <sup>18</sup> (usually 2-5) |

## **Complications**

Many complications are operator dependent and can be avoided with careful and proper technique.

- Temporary blurring of vision
- Corneal epithelial and/or endothelial burns with argon (especially with bubble formation and proximity to endothelium)<sup>19</sup>
- IOP spikes
- Postoperative inflammation
- Posterior synechiae
- Intraoperative bleeding
- Iridotomy closure<sup>20</sup>
- Failure to penetrate
- Localised lens opacities or cataract progression<sup>21</sup>
- Rarely: retinal damage, retinal and subhyaloid haemorrhage,<sup>22</sup> cystoid macular oedema, ciliary block glaucoma,<sup>23</sup> endothelial decompensation,<sup>24-26</sup> decompression retinopathy,<sup>27</sup> Descemet's membrane detachment<sup>28</sup>

## **Post-laser Management**

- Particularly if IOP spike prevention treatment is not available:
  - re-check IOP 1 to 6 hours after laser and again at 24 to 48 hours
  - systemic acetazolamide or mannitol may be indicated if IOP rises rapidly
  - discharge patient only when IOP stable at safe level
- Topical steroid at least 4 to 6 times/day for 4 to 14 days depending on inflammation
- Stop topical pilocarpine, and taper any other topical IOP-lowering drugs as indicated
- Verify the patency of the PI
- Repeat gonioscopy when effect of pilocarpine worn off — if appositional closure remains and IOP high, may consider laser peripheral iridoplasty or early cataract extraction<sup>29</sup> if lens mechanism is identified
- Pupillary dilatation to break posterior synechiae when suspected

## PERIPHERAL IRIDOPLASTY<sup>30</sup>

### Why?

- Reasonably effective
- Relatively non-invasive
- Adjunct to PI<sup>30</sup>

### What?

- Laser treatment to contract the peripheral iris:
- To flatten the peripheral iris
  - To widen the anterior chamber angle inlet
  - To re-open appositionally closed segments of drainage angle

### When?

- Help to break an attack of acute AC as initial treatment,<sup>31-37</sup> or as adjunctive measure when systemic medications fail to control IOP
- Angle remains occludable following PI, eg, plateau iris<sup>38,39</sup>
- To break attack of secondary forms of acute AC (phacomorphic glaucoma)<sup>40-42</sup>
- Minimise the risk of corneal endothelial damage during iridotomy
- Facilitate access to TM for laser trabeculoplasty<sup>31</sup>
- As an adjunct to goniosynechialysis<sup>43,44</sup>
- Contraindicated in area with PAS; will not break PAS, may cause more inflammation

### How?

#### Prelaser Management

- Explain the procedure
- Instil 2% or 4% pilocarpine
- To reduce post-treatment IOP spike/inflammation, consider 1% apraclonidine or 0.15% to 0.2% brimonidine, and/or  $\beta$ -blocker, and/or oral CAI, and/or steroid drops before the procedure
- Topical anaesthesia
- Topical glycerine if the cornea is oedematous

#### Lens

- Any laser iridotomy contact lens
- Goldmann 3-mirror lens

#### Procedure

- Argon green or blue-green
- Diode laser<sup>35</sup>
- Placement of laser spot
  - aim at the most peripheral location
  - aiming beam may have to straddle limbus
- If peripheral anterior chamber too shallow, a mid-peripheral laser spot could be placed first to deepen the anterior chamber, before a more peripheral laser spot is applied
- Charring of iris or ‘pop’ sound or bubble signifies too much power — reduce power accordingly

### LASER PARAMETERS

|                 |  |
|-----------------|--|
| Power           | 150-400 mW depending on the reaction — the smaller the spot size, the lower the power setting  |
| Spot size       | 200-500 $\mu\text{m}$ — both small-spot and big-spot pattern can be used with appropriate adjustment of power setting  |
| Exposure time   | 0.2-0.5 sec  |
| Number of spots | 10 to 40 applications over 360°, leaving at least 1- to 2-spot diameters between spots; 180° treatment may also be effective <sup>32</sup><br>Do not overtreat |

---

## Endpoint

Iris contraction with peripheral anterior chamber deepening.

## Complications<sup>30,35,37</sup>

- Mild iritis
- Iris atrophy
- Mydriasis
- Corneal endothelial burns
- IOP spikes
- PAS and/or PPS
- Rarely: decompression retinopathy<sup>45</sup>
- Rarely: Urrets-Zavalía syndrome<sup>46</sup>

## Postoperative Treatment

- If treatment for prevention of IOP spike is not available, check IOP within 1 to 6 hours and at 24 to 48 hours depending on the status of the patient
- Topical corticosteroids 4 to 6 times/day for 7 days or more depending on the post-laser inflammation
- Repeat gonioscopy to evaluate the anterior chamber angle and identify any other mechanism(s) of AC that might necessitate further intervention
- Pupillary dilatation to break posterior synechiae when suspected

## CYCLOPHOTOCOAGULATION

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### Why?

- Reasonably effective
- Preferable to cyclocryotherapy or cyclodiathermy because less collateral damage and inflammation
- Can be repeated if IOP-lowering effect wears off

---

### What?

- Reduces aqueous production by coagulative destruction of ciliary epithelium

---

### When?

- Failed multiple filtering surgeries
- Primary procedure to alleviate pain in neovascular glaucoma with poor visual potential
- Painful blind eye
- Surgery not appropriate

---

### How?

#### Prelaser Management

- Explain procedure
- For trans-scleral technique, careful slit-lamp examination to identify suitable/unsuitable sites for laser application
- Topical and sub-Tenon's anaesthesia, or retro-/peribulbar anaesthesia
- General anaesthesia when indicated

#### Techniques

- Transpupillary
- Trans-scleral
- Endolaser
- Conservative, incremental applications avoiding 3 and 9 o'clock positions

### Contact Trans-scleral Nd:YAG Laser

Continuous wave Nd:YAG laser with trans-scleral contact probe.

|                             |                 |                        |
|-----------------------------|-----------------|------------------------|
| <b>LASER<br/>PARAMETERS</b> | Exposure time   | 0.5-0.7 sec            |
|                             | Power           | 4-7 J                  |
|                             | Number of burns | 30-40 over 360°        |
|                             | Location        | 1.0-2.0 mm from limbus |

### Contact Trans-scleral Diode Laser

Diode laser with trans-scleral contact probe (Appendix 11).

|                             |                 |                        |
|-----------------------------|-----------------|------------------------|
| <b>LASER<br/>PARAMETERS</b> | Exposure time   | 0.5-2.0 sec            |
|                             | Power           | 1.0-2.5 W              |
|                             | Number of burns | 20-40 over 180-360°    |
|                             | Location        | 1.0-2.0 mm from limbus |

### Endolaser

- Diode endoscopic laser
- Argon or krypton laser

|                             |  |
|-----------------------------|--|
| <b>LASER<br/>PARAMETERS</b> | Depends on laser system used — consult the instruction manual and clinical updates |
|-----------------------------|--|

### Complications

- Pain
- Persistent inflammation
- Loss of visual acuity<sup>47,48</sup>
- Hypotony<sup>49</sup>
- Scleral thinning<sup>50,51</sup> or rupture<sup>52</sup>
- Pupillary distortion<sup>53</sup>
- Macular oedema
- Retinal detachment<sup>54</sup>
- Aqueous misdirection syndrome<sup>55</sup>
- Phthisis<sup>55</sup>
- Sympathetic ophthalmia<sup>56</sup>
- Failure to control IOP — multiple procedures may be needed

### Postoperative Management

- Analgesia
- Continue any current treatment
- Check IOP after 24 to 48 hours
- Topical corticosteroids 4 to 6 times/day for 14 days or more depending on post-laser inflammation
- Cycloplegia 2 to 4 times/day for 7 to 14 days
- Continue any current IOP-lowering treatment; taper as indicated

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## LASER SUTURE LYSIS

### Why?

- Effective
- Non-invasive
- Avoid early bleb failure
- Staged postoperative IOP control

### What?

- Laser treatment to selectively lyse the subconjunctival suture(s), without disturbing the overlying tissues, allows postoperative titration of IOP by increasing outflow<sup>67</sup>

### When?

- Commonly within 14 days of glaucoma filtering surgery
- More than 14 days if adjunctive MMC used

### How?

#### Prelaser Management

- Explain the procedure
- Topical anaesthesia

#### Laser

- Argon green or blue-green
- Diode
- Frequency-doubled Nd:YAG

#### Lens

- Ritch
- Hoskins
- Mandelkorn
- Zeiss 4-mirror
- Glass rod

#### Uses of Lens

- Blanch the conjunctival vessels
- Focus on the suture
- Fix the globe
- Open the lids

#### Placement of Laser Spots

- Subconjunctival scleral flap sutures (nylon)

#### Complications

- Conjunctival burn, leak
- Hypotony
- Shallow anterior chamber

### **LASER** **PARAMETERS**

|                 |  |
|-----------------|--|
| Spot size       | 50 µm  |
| Exposure time   | ≤0.1 sec   |
| Power           | 300-800 mW   |
| Number of spots | 1 or more, as needed   |
| Technique       | Cut 1 suture at a time<br>If blood present under the conjunctiva, choose different suture to cut, or use a longer wavelength laser, or use a short exposure time<br>Cut suture close to one end or the other |

- Bleeding from ostium
- Hyphaema

### Post-laser Management

- Continue current postoperative regimen
- If bleb does not form spontaneously, apply pressure, eg, around trapdoor
- Recheck IOP and outflow 5 minutes after laser and within 1 week

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## 2.4 SURGERY

### TYPES OF SURGERY

#### Open Angle Glaucoma

- Outflow enhancement: penetrating and non-penetrating filtering surgery
- Glaucoma drainage devices

#### Chronic Angle Closure Glaucoma

- Pupillary block relief: iridectomy
- Outflow enhancement: trabeculectomy
- Widening of anterior chamber angle: lens extraction
- Plateau iris syndrome: laser peripheral iridoplasty (or lens extraction)
- Angle surgery: goniosynechialysis
- Glaucoma drainage devices

#### Acute Angle Closure ( $\pm$ Glaucoma)

- Pupillary block relief: iridectomy
- Outflow enhancement: trabeculectomy
- Angle surgery: goniosynechialysis
- Widening of anterior chamber angle inlet: lens extraction occasionally indicated

#### Childhood Glaucoma

- Angle surgery: goniotomy and trabeculotomy
- Outflow enhancement: trabeculectomy  $\pm$  trabeculotomy
- Glaucoma drainage devices

### GLAUCOMA SURGERY

#### Why?

- Reasonably effective<sup>1,2</sup>
- Reasonably safe
- Widely available

#### What?

- Penetrating filtering surgery:
  - trabeculectomy
  - trabeculectomy with antimetabolites
- Non-penetrating surgery (with or without implant):
  - deep sclerectomy
  - viscocanalostomy
- Glaucoma drainage devices
- Surgical iridectomy: largely replaced by laser iridotomy (Section 2.3)
- Lens extraction for lens-induced ACG
- Goniosynechialysis
- Vitrectomy for ciliary block

#### When?

- Failed medical and/or laser treatment<sup>1</sup>
- Anticipated failure of medical and/or laser treatment (very high IOP)<sup>2,3</sup>
- Patient preference
- Other forms of therapy are inappropriate: poor adherence, side effects, socioeconomic problems

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**How?****Preoperative Assessment**

Identify risk factors for failure and treat where applicable.<sup>4</sup>

- Asian, African, Hispanic ethnicity
- Previous ocular surgery
- Young age
- Aphakia
- Pseudophakia
- Active ocular inflammation
- Prolonged use of topical antiglaucoma medications<sup>5</sup>
- Tendency to form keloid scars
- Neovascular glaucoma

**SURGICAL TECHNIQUE: TRABECULECTOMY**

- Select appropriate technique and decide whether or not to use antimetabolites
- Fornix-based or limbus-based conjunctival flaps
- A corneal traction suture is an alternative to a superior rectus traction suture
- If antimetabolites are used, these should be applied to a large surface area of conjunctiva to reduce the risks of bleb infections and cystic blebs<sup>6</sup>
- The scleral flap should encourage posterior flow
- Pre-placing scleral sutures and inserting an infusion cannula reduce periods of hypotony during surgery
- Meticulous Tenon's and conjunctival closure to prevent leaks and their sequelae

**ENHANCEMENT  
OF SURGERY**

- Use of antimetabolites:
  - intraoperative<sup>4</sup>
  - postoperative<sup>4,6-8</sup>
- Use of anti-inflammatory agents such as topical or systemic corticosteroids<sup>9,10</sup>
- Use of laser suture lysis or releasable sutures<sup>11</sup>
  - adjustable or releasable sutures add an extra dimension of flexibility towards a gradual and titratable postoperative modulation of flow<sup>12</sup>

**Postoperative Management**

- First 4 postoperative weeks critical for results
- Examine first postoperative day
- Topical steroids for 6 to 12 weeks
- Topical antibiotics for  $\geq 14$  days
- Cycloplegics for 2 to 6 weeks, especially for those at risk for ciliary block (short axial length)
- Analgesics
- Intensive individualised postoperative care (globe indentation, suture lysis or release, subconjunctival 5-FU)

**USE OF ANTIMETABOLITES IN GLAUCOMA SURGERY**

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**Why?**

- Scarring is the major cause of failure following filtration surgery. Antimetabolites have been shown to inhibit scarring and to increase the success rate<sup>13,14</sup>

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**What?**

- MMC
- 5-FU

**When?**

- For when there is high risk of failure following standard filtering surgery (including repeat surgery, neovascular glaucoma, glaucoma in uveitis, glaucoma in aphakia, younger age, African-derived populations)
- In primary surgery, especially when a lower target pressure is required<sup>15,16</sup>
- To increase the success rates for glaucoma drainage devices
- With needling of a failed filter

In these instances, the enhanced success rates with antimetabolites may make the complications associated with their use (hypotony, bleb-related infections) more acceptable.

**How?****Intraoperative Application****Dose**

- Sponge soaked in MMC (varying doses of 0.2 to 0.5 mg/mL) applied for 1 to 3 minutes
- Sponge soaked in 5-FU (50 mg/mL) for 1 to 5 minutes

**Mode of application**

- Minimise exposure of the conjunctival edge and cornea to antimetabolites
- Sponge placed under the conjunctiva
  - a little extra subconjunctival blunt dissection allows multiple sponges or a large single sponge to be placed<sup>13</sup>
  - count sponges to avoid leaving in wound
- Apply to a large surface area of conjunctiva to reduce the risks of bleb infections and cystic blebs<sup>6</sup>

**Removal of residual drug when applied during surgery**

- Copious irrigation of the treated area with balanced salt solution, normal saline, or Ringer's lactate solution

**Postoperative Application**

5-FU is also used as postoperative injections of 5 mg in 0.1 mL for up to 4 weeks. The injection may be given alongside or behind the bleb, or sometimes 90° to 180° away, preferably using a 30 G needle. The number of injections is titrated according to the appearance of the bleb. Care is taken to avoid spillage on the cornea and the resulting epitheliopathy. The conjunctiva over the area of injection may be tamponaded with a cotton bud for about 1 minute after the injection. Discontinue injections in the presence of corneal epithelial defects.

**Note:** *The use of antimetabolites can be associated with sight-threatening complications and they must be used with caution.<sup>17</sup> Use of an algorithm developed by those experienced in the use of such agents is desirable.<sup>15</sup>*

**SURGICAL TECHNIQUE: NON-PENETRATING SURGERY****Why?**

Non-penetrating surgery reduces IOP less effectively than penetrating surgery, but with lower complication rates (postoperative hypotony, bleb-related infections) compared with trabeculectomy with antimetabolites.

**When?**

Failed medical and/or laser treatment, when there is less need for lower target IOP.

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**How?**

In deep sclerectomy, Schlemm's canal is de-roofed underneath a scleral flap and a deep lamella of corneo-sclera removed to leave a scleral lake. Aqueous percolates through the remaining TM into this area. A very shallow filtration bleb can be seen with imaging. A collagen implant can be used as a spacer to keep the lake patent.<sup>18</sup>

Viscocanalostomy modifies the above procedure by injecting hyaluronic acid into Schlemm's canal. This may increase outflow by widening and/or microrupturing the walls of Schlemm's canal and collector channels.

Non-penetrating surgery requires a steep learning curve.<sup>18</sup> Further management with Nd:YAG goniopuncture may be required in up to 40% of surgeries to lower the IOP in the longer term.

## **SURGICAL TECHNIQUE: GLAUCOMA DRAINAGE DEVICES**

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**Why?**

A glaucoma drainage device allows aqueous to flow from the anterior chamber into a bleb that forms around the plate of these devices. Aqueous diffuses through the capsule and is collected by blood vessels in the surrounding capsule. Implants are typically used for patients for whom there is a high risk of failure with conventional surgery.<sup>19-22</sup>

One-year results comparing glaucoma drainage devices with trabeculectomy + MMC in patients who had previous trabeculectomy and/or cataract extraction with IOL implantation and uncontrolled glaucoma<sup>23</sup> found that glaucoma drainage devices recipients were:

- Less likely to experience postoperative complications
- Less likely to experience failure
- More likely to use more glaucoma medications

Surgical complications and IOP showed statistically similar results between the 2 groups at 1 year.

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**What?**

Glaucoma drainage devices<sup>24</sup>

- Valved
  - Ahmed
  - Krupin disc
- Non-valved
  - Molteno
  - Baerveldt

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**When?**

Where there is a very high risk of failure of trabeculectomy, even with antimetabolites; these eyes invariably have severe, refractory glaucoma:

- Previously failed trabeculectomies with antimetabolites
- Prior multiple ocular surgeries with conjunctival scarring
- Traumatic, inflammatory, or chemically induced surface scarring
- Intraocular membrane formation likely to occlude a non-implant drainage procedure (ICE syndrome, neovascular glaucoma)

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**How?**

Glaucoma drainage device surgery and the postoperative management of such patients can be complicated.

Depending on the surgeon's preference, one of the glaucoma drainage devices is positioned on the scleral surface, usually in the superior temporal or superior nasal quadrant (or both for a 2-plate glaucoma drainage device) and connected to the anterior chamber by a tube. Insertion beneath a scleral flap and/or use of a scleral patch over the

tube reduces risk of tube extrusion, and is recommended. There is minimal evidence for the use of antimetabolites with glaucoma drainage devices.

A tube-occluding suture may be used to avoid immediate postoperative hypotony with a non-valved tube; venting slits may be needed to avoid high IOP until the suture is removed or dissolves. Valved tubes may have a lower rate of immediate hypotony, but postoperative hypotony can occur via leak around the tube. A hypertensive phase is common with any shunt and may require medical therapy to reduce IOP.<sup>19-22</sup>

### Complications of Glaucoma Drainage Devices

- Failure to control IOP
- Hypotony
- Corneal decompensation (long-term complication with anterior chamber tube — reduced with pars plana tube)
- PAS
- Pupillary distortion
- Cataract
- Tube blocked by blood, vitreous, or fibrin
- Erosion of the tube and/or plate(s)
- Globe malposition and/or motility disturbance
- Endophthalmitis (rare)

## CATARACT AND GLAUCOMA SURGERY

Cataract and glaucoma are both common conditions, which often coexist. A recent review has assessed current surgical management and highlights the limited evidence in the literature about the effectiveness of combined cataract and glaucoma surgery compared with separate surgeries (Table 2.4).<sup>25</sup>

**Table 2.4.** Evidence for surgical management of cataract and glaucoma.<sup>20</sup>

| Review comments   | Evidence level |
|---|----------------|
| MMC (but not 5-FU) has a small benefit (2 to 4 mm Hg) for ECCE-trabeculectomy                 | Moderate       |
| Two-site surgery provides slightly lower IOP (1 to 3 mm Hg) than 1-site surgery               | Weak           |
| IOP is lowered more (1 to 3 mm Hg) by phacoemulsification than by ECCE in combined procedures | Weak           |
| Two-stage versus combined procedures  | Insufficient   |
| Alternative glaucoma procedures versus trabeculectomy in combined procedures                  | Insufficient   |

## LENS EXTRACTION FOR ANGLE CLOSURE GLAUCOMA

There is limited evidence about the effectiveness of lens extraction for ACG.<sup>26</sup> The surgery is technically difficult because of frequently coexisting shallow anterior chamber, large bulky lens, iris atrophy secondary to ischaemia, and zonular weakness. Removing the lens may lower, and control, IOP satisfactorily,<sup>27</sup> while deepening the angle may reduce the likelihood of progressive AC and chronic rise in IOP.<sup>28</sup>

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### 3.1 FOLLOW-UP

#### Why?

The aim of follow-up is:

- To implement the glaucoma management plan or revise it if necessary
- To detect progression and rate of change
- To detect effectiveness and any side effects of treatment
- To detect any change in health or systemic medications that may affect the glaucoma management plan
- To monitor/treat ocular comorbidity such as cataract and diabetic retinopathy
- To detect any change in risk profile

#### What?

The follow-up process starts with the management plan made at the initiation of therapy. At the follow-up visits the doctor should:

- Assess the patient's subjective well being, visual function, and QOL
- Reassess risk factors, especially IOP and gonioscopic change(s)
- Reassess structure and function of the optic nerve
- Estimate rate of (any) progression and discuss its significance in relation to patient's age and status of the other eye
- Identify adverse effect(s) of treatment
- Assess adherence to, and persistence with, the treatment plan
- Identify change(s) in medical and ophthalmological problems
- Reinforce appropriate patient information:
  - revise management if necessary
  - plan follow-up

#### When?

- The initial follow-up after diagnosis of manifest glaucoma should be relatively frequent so as to detect the rate of progression reliably<sup>1</sup>
- The more severe the damage, the greater the risk factors, the more frequent should be the follow-up
- The faster the rate of progression, the more frequent should be the follow-up

### PATIENT'S SUBJECTIVE WELL BEING AND VISUAL FUNCTION

- Patients often wish to tell the doctor how they feel their condition has/has not changed
- This discussion helps build a good doctor-patient relationship and therapeutic alliance
- There is a degree of association between patients' self-reported visual function and both VF status and VF progression

Subjective changes in vision with glaucoma are rare but, in advanced disease, changes in the following qualities of vision may indicate a deterioration of GON:

- Night vision (night driving difficulties — vehicles jump into vision, glare from oncoming headlights)
- Dark adaptation (difficulty walking into dark environment)
- Glare
- Stereopsis
- Acuity (high and low contrast — reading small print, especially in dim light; identifying faces while walking; contrast sensitivity problems; poor distance judgements)
- Missing pieces of vision
- Increased chances of falls and motor vehicle accidents

Although subjective changes in vision are rarely reported until late in the disease (see above), even patients with mild to moderate glaucoma may report problems with 'visual mobility'; bumping into things, difficulty with stairs, and difficulty finding things that have been dropped.

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## REASSESS RISK FACTORS

### Intraocular Pressure

- IOP is currently the only major modifiable risk factor for glaucoma
- Assessment at every visit is vital
- Establish whether target IOP has been achieved
- A single measurement of IOP cannot detect fluctuations — IOP should be reassessed at different times of the day
- Repeat unexpected readings at the same visit and soon after

### **CAUSES OF CHANGE IN INTRAOCULAR PRESSURE AT FOLLOW-UP**

- Increased IOP:
  - reduced outflow
  - gradual loss of efficacy of a drug (tachyphylaxis)
  - poor adherence
- Reduced IOP:
  - therapeutic effect
  - LASIK/corneal refractive surgery causing unexpected changes in measured but not actual IOP
  - resolving pathology (regression of pigment dispersion)
- Reduced or increased IOP:
  - regression to the mean
  - variation during the day and between days
  - change in systemic medications
  - poor instrument calibration/function

### Gonioscopic Changes

- Maintain baseline examination conditions
- Perform gonioscopy at least every 3 years for all patients, and more frequently for those with, or at high risk for, AC
- Look for increased appositional and/or synechial closure
- Pupil size changes have dynamic effects on the angle configuration
- Look for change in angle width, synechiae, and pigmentation
- Consider gonioscopic photography or anterior segment imaging if available for more objective comparison between visits

## REASSESS STRUCTURE AND FUNCTION OF THE OPTIC NERVE

### Optic Disc

Progression of GON usually occurs over a long period, which can make change detection difficult. The optic disc of patients with AC should be examined at every visit until opening of the angle is achieved and at least every 3 years thereafter.

The occurrence of the following indicate GON progression (Appendix 12):

- Disc haemorrhage
- Neuroretinal rim notching (incidence or enlargement)
- Change in vessel position on the disc
- Neuroretinal rim thinning (enlargement of CDR)
- Wedge-shaped RNFL defects (incidence or enlargement)

Where baseline and serial optic disc imaging are available, detection of change is substantially enhanced. If imaging is not available, the pupil should be dilated (if it is not possible to do this safely, consider prophylactic iridotomy/iridoplasty) to obtain an adequate view of the disc for diagnosis and if progression is suspected.<sup>2</sup>

### Visual Field

Apparent change is frequent in perimetry: a small proportion is owing to GON progression.

#### **CAUSES OF CHANGE IN VISUAL FIELDS**

- Learning: VF performance usually improves with experience — usually this change is greatest between the first and second tests
- Reliability changes and poor concentration may cause generalised depression
- False-positive errors may reflect poor reliability — fixation losses may indicate improper fixation; assess other signs such as the gaze monitor or technician's notes
- Progression of disease
- Cataract: may cause generalised depression<sup>3,4</sup> and may mask relative scotomas<sup>5</sup>
- Pupil size changes: miosis causes generalised VF depression; minimum 3-mm diameter recommended
- Retinal disease (vein occlusion, macular degeneration, significant diabetic retinopathy)
- Retinal laser
- Miscellaneous artifacts (lens rim, ptosis, deep-set eyes)
- Change in measurement strategy (Fastpac versus Full Threshold versus SITA)
- Decline in general health

### Detecting Progression

Progression is characterised by:

- Widening or deepening of an existing scotoma
- Development of a new glaucomatous scotoma
- Generalised field depression; consider concomitant cataract, miosis, or poor reliability (Appendix 13)

Changes in VF should be confirmed by at least 1 repeat test. VF change is best detected by the use of software that highlights areas of possible change.

There is a close correlation between glaucomatous changes in structure of the optic disc and consequent VF loss.<sup>6,7</sup> However, there may be considerable variations in morphology of a 'normal' disc, and in a patient's ability to perform VF tests adequately.

Changes should be regarded skeptically until the deviation exceeds the SD of serial measurements.<sup>8</sup>

### Retinal Nerve Fibre Layer

Imaging of the RNFL should be considered when available.

## IDENTIFY ADVERSE EFFECTS OF TREATMENT

Adverse effects of treatment should be actively sought. These include:

- General effects — self-rated health, feelings about/attitude towards treatment
- Systemic effects — respiratory, cardiovascular, digestive, neurological, impotence
- Local effects — stinging/burning, blurring, itching, redness

## QUALITY OF LIFE ISSUES

- The patient's QOL should be estimated and the impact of glaucoma and its management on QOL assessed<sup>9</sup> (Glaucoma Quality of Life–15 questionnaire; Appendix 14)
- The emphasis of management should be customised towards improving patient's QOL; this forms part of the assessment of burden of disease and burden of treatment
- Referral to appropriate community health management

## SEAGIG DECISION SQUARE FOR GLAUCOMATOUS OPTIC NEUROPATHY

- Table 3.1 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 +, ++, and +++, with the last indicating the most aggressive level of intervention
- A +++ grade may be associated with a rapid stepwise progression through medical to surgical management
- A – indicates no addition to therapy

**Table 3.1.** SEAGIG decision square for glaucomatous optic neuropathy.

| Risk      | Disease status |                                  |             |
|-----------|----------------|----------------------------------|-------------|
|           | Stable         | Uncertain                        | Progressing |
| Increased | +              | ++                               | +++         |
| Uncertain | Reassess risk  | Reassess risk and disease status | ++          |
| Unchanged | –              | Reassess disease status          | +           |

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### Risk Factors

The following factors confer a high risk for loss of vision from glaucoma:\*

- High or rising IOP
- Any appositional AC
- Any PAS, or an increase in PAS if seen before
- Longer life expectancy
- Ethnicity (African descent)
- PXF
- Late/advanced presentation

\* *The more risk factors there are, the higher the risk. Therefore, add + for every additional risk factor.*

### Disease Progression

- Stable disease: no change in optic disc or VF status
- Uncertain disease: change in VF that is not consistent with the status of the disc or vice versa — check for artifacts
- Progressing disease: changes consistent with glaucoma in the optic disc and/or VF\*

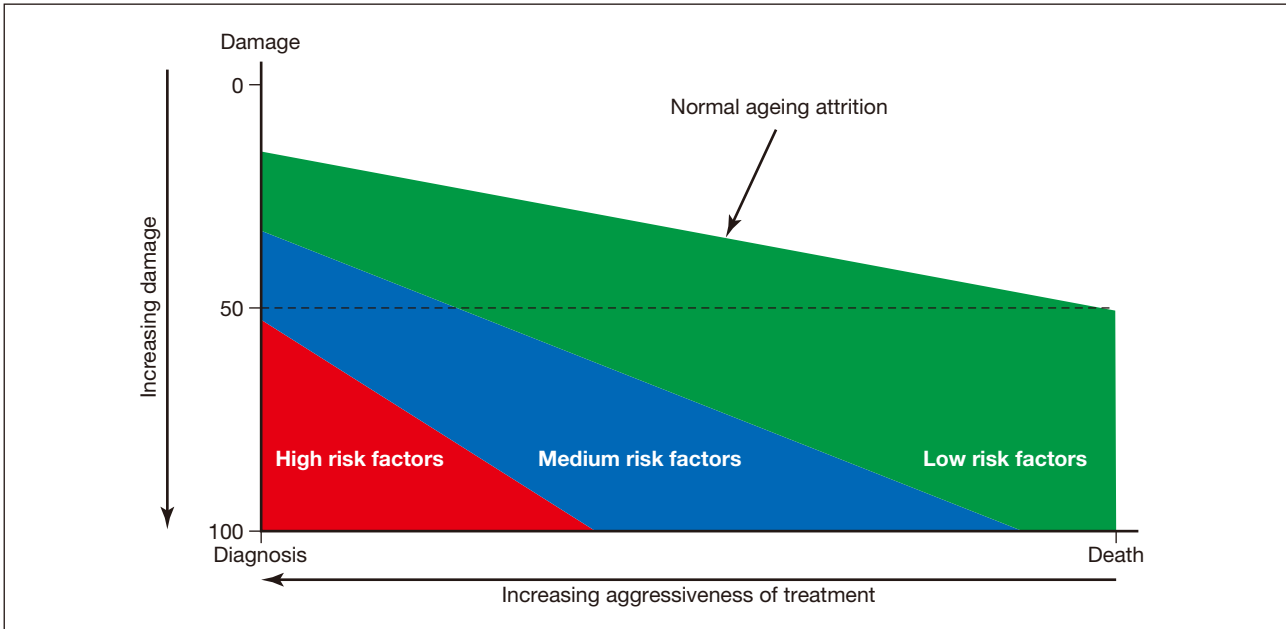
\* *Add + if the rate of progression appears rapid.*

## THE GLAUCOMA LIFE STORY

The determinants of the glaucoma life story (Figure 3.1) are:

- State of damage
- Life expectancy
- Rate of progression

**Figure 3.1.** The glaucoma life story.



The graph plots life expectancy against the extent of glaucomatous damage at diagnosis. The slope of the line is the rate of progression; this is determined by risk factors. Although rate of progression is the key factor for determining success of treatment, it is very difficult to measure accurately or reliably. Generally, risk factors are used to estimate the likely rate of progression, with that estimate then being acted upon. However, knowledge of risk factors is incomplete.

Attempts can be made to reduce the rate of progression by reducing IOP. The minimum slope is the rate of normal ageing of the nerve. The target pressures are based on the slope that it is thought will allow the patient to maintain good vision for his/her life.

The colour in the graph represents the risk of blindness from glaucoma. Green represents low risk and red represents high risk.

### Timing of Follow-up

The target pressure is the IOP at which it is believed the patient can retain vision for the rest of his/her life. However, target pressure must be reassessed and modified depending on the patient's course of disease.

Follow-up timing is determined by the treatment regimen, if this has changed. If the patient has stable disease, the timing is determined mainly by the extent of damage (Table 3.2).

**Table 3.2.** Timing of follow-up.

|                    | Extent of damage |      |          |        |
|--------------------|------------------|------|----------|--------|
|                    | Glaucoma suspect | Mild | Moderate | Severe |
| Follow-up (months) | 6-24             | 6-12 | 4-6      | 1-4    |

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## 3.2 SCREENING

### GENERAL RECOMMENDATIONS

- Opportunistic glaucoma screening for:
  - patients who visit ophthalmologists, using a comprehensive clinical examination and directed investigations
  - patients who visit an optometrist, physician, or other trained health worker using more rapid and specific tests
- Universal glaucoma screening in isolation from other diseases is probably not feasible at present

**Note:** see Tables 3.3 to 3.5 for definitions of terms.

**Table 3.3.** Definitions of screening terms.

| Term   | Definition  |
|--|---|
| Glaucoma screening   | Examination of asymptomatic people to classify them as likely or unlikely to have glaucoma  |
| Glaucoma diagnosis   | Examination of a person by an expert health care practitioner to confirm or exclude the presence of glaucoma  |
| Universal glaucoma screening<br><i>(also known as population-based glaucoma screening)</i>                 | Screening for glaucoma by inviting all people within a group to attend for a screening examination; this involves incorporating glaucoma screening into a universal periodical health examination programme |
| Opportunistic glaucoma screening<br><i>(also known as case-based glaucoma screening or case detection)</i> | Screening for glaucoma when people visit an eye care or medical professional for any reason   |
| Prevalence   | The proportion of people with the target disorder (glaucoma) in the population tested   |
| Sensitivity  | The ability of a test to correctly identify people who have glaucoma (true positive)  |
| Specificity  | The ability of a test to correctly identify people who do not have glaucoma (true negative or normal)   |
| Positive predictive value  | The proportion of people with positive test results who actually have glaucoma  |
| Negative predictive value  | The proportion of people with negative test results who do not have glaucoma  |

**Table 3.4.** Glaucoma screening versus glaucoma diagnosis.

| Screening  | Diagnosis  |
|--|--|
| Aim to classify people as likely or unlikely to have glaucoma  | Aim to confirm or exclude glaucoma with very high certainty  |
| Most people will not have glaucoma, so the screening test needs to be very good at confirming normality                    | A much larger proportion will have glaucoma (although this may be <50%), so the tests need to be very sensitive for detecting glaucoma |
| Many people will be screened, therefore the process must be rapid and inexpensive  | A smaller number of people will be examined, so the process must be thorough   |
| The endpoint is the referral of a person with a positive result to an ophthalmologist; this must occur in a timely fashion | The starting point is a new doctor-patient relationship and lifelong care for people with glaucoma                                     |

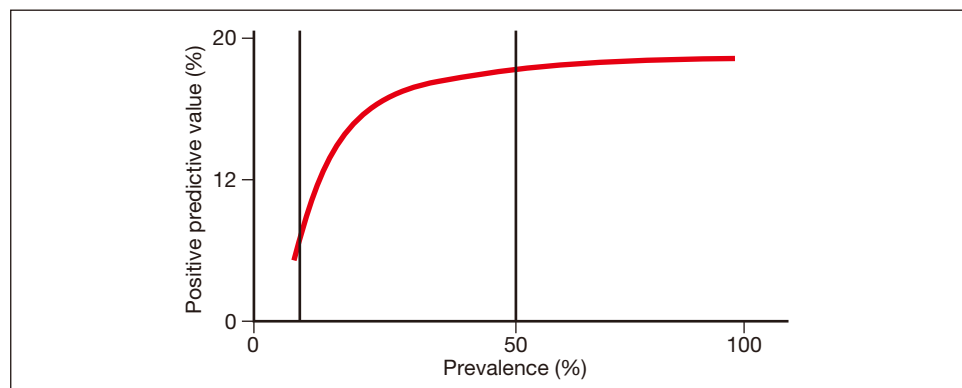
**Table 3.5.** Universal glaucoma screening versus opportunistic glaucoma screening.

| Universal glaucoma screening <sup>1</sup>   | Opportunistic glaucoma screening  |
|---|---|
| Health care professionals seek out patients: there is an implied pledge that people may be cured, but this may not always be true<br>After screening positive, doctors are obliged to establish a diagnosis and treatment regimen using the best techniques<br>Without the requisite equipment, trained personnel, and infrastructure, screening is not justified | Relies on detection of glaucoma in people who present for other reasons<br>Patients seek out health care professionals, who treat them to the best of their ability but without the guarantee of a cure<br>Success or failure of screening is marginal to the reason the patient presents |
| Patients who have a false-positive result carry the burden of being labelled with the disease: the consequences may be severe <sup>2</sup>  | Based on detection of glaucoma in 'at-risk' patients in whom the prevalence of glaucoma is higher; therefore, most of the tests described below have a reasonably high positive predictive value  |
| Patients who have the disease but have a negative test (false-negative result) are told they are healthy, which could lead them to stop participating in the screening programme  |   |
| Many countries in the region may not have the requisite infrastructure to follow-up and categorise people who test positive, or even treat them appropriately; even if possible, it may not be feasible   | The optometrist and general physician can play an important role in screening for glaucoma; the same issues apply to infrastructure for diagnosis and treatment but the numbers may be smaller  |
| Screening cannot be a 1-time event and even developed countries may not be able to afford to screen the general population for glaucoma, as well as the burden of further testing, treatment, and follow-up   | Most elderly people and those with diabetes or myopia (all at risk for glaucoma), often visit the offices of ophthalmologists and optometrists for other eye care needs; follow-up is also easier   |

## PREDICTIVE VALUE

The predictive value of a test is dependent on the prevalence of glaucoma in the population being tested. As shown in Figure 3.2, assuming all other factors remain constant, the positive predictive value will increase with increasing prevalence.

**Figure 3.2.** Positive predictive value.



With a low prevalence of glaucoma, most people who test positive will have a false-positive result.

To increase the effectiveness of the tests, the prevalence of glaucoma in the population to be tested must be reasonably high. The prevalence of glaucoma can be 'increased' by targeting high-risk groups such as elderly people, those with a family history of glaucoma, and people with diabetes or myopia. Most people who are at high risk visit eye care professionals for other reasons, presenting an opportunity to screen them for glaucoma.

**Why?**

- To detect disease in the presymptomatic stage

**How?**

The epidemiology section outlines the current understanding of the magnitude of glaucoma blindness in Asia. This large burden raises the question of how best to detect glaucoma. Universal screening and opportunistic screening are 2 possible strategies.

The World Health Organization recommends that certain defined criteria be fulfilled before any universal screening is undertaken:<sup>2</sup>

- The disease must be an important public health problem
- There must be a recognisable latent or early stage, during which people with the disease can be identified before symptoms develop
- There must be an appropriate, acceptable, and reasonably accurate screening test
- There must be an accepted and effective treatment for people with the disease, that must be more effective at preventing morbidity when initiated in the early asymptomatic stage than when begun in the later symptomatic stages
- The cost of case finding must be economically balanced with possible expenditure on medical care
- Opportunistic screening should be a continuous process and not a once-only project

Other questions that need to be asked before embarking on any screening programme are listed below:<sup>3</sup>

- Does early diagnosis lead to improved clinical outcomes in terms of visual function and QOL?
- Can the health system cope with the additional clinical time and resources required to confirm the diagnosis and provide long-term care for people who screen positive for a chronic disease such as glaucoma?
- Will the patients in whom early diagnosis is achieved comply with subsequent recommendations and treatment regimens?
- Are the cost, accuracy, and acceptability of the screening tests adequate for the purpose?

Glaucoma fits many of the criteria required for universal screening, but other diseases are more problematic. It is likely that the health systems of only the most developed countries in the region may have the ability to cope with the additional clinical time and resources required for universal screening.

Opportunistic screening is simply an extension of the usual clinical routine. Every patient who visits an eye care/health care professional can be considered a glaucoma suspect and undergo a screening examination. Every patient who visits an ophthalmologist can be considered a glaucoma suspect and undergo a comprehensive eye examination.

**What?**

Opportunistic screening is recommended rather than universal screening.

**How to Perform Screening**

For non-ophthalmic professionals, screening consists of a comprehensive risk factor assessment (include IOP measurement) and optic disc examination. If a slit lamp is available, assessment of the limbal ACD predicts PAC when the IOP is above the normal range. It is critical that all people who are screened have an optic disc examination, as a large proportion of people with OAG have IOP within the normal range. In some centres, VF testing is used; if so, it must not be used indiscriminately as false-positive functional test results are common.

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Opportunistic glaucoma screening in an ophthalmologist's clinic relies on a comprehensive clinical examination (slit-lamp examination, IOP measurement, gonioscopy, dilated optic disc examination), followed by investigations that are directed by the results. Such an approach increases the positive predictive value of the tests used. Detecting early glaucoma is ideal, but requires a sensitive test, which leads to false-positive results.

For population-based screening, a test should have a reasonably high sensitivity with a very high specificity. Prevent Blindness America has suggested a sensitivity of 85% and a specificity of 95% to 98% to detect moderate-to-severe glaucoma.<sup>4</sup> For most populations older than 50 years, a specificity of 97% to 98% would mean that approximately half the people who have positive results would have glaucoma. A sensitivity of 85% would detect approximately two-thirds of currently undiagnosed cases in developed countries and 88% of undiagnosed cases in the developing world and Japan.

### Screening for Primary Angle Closure Glaucoma

Population-based studies from western countries have shown that the prevalence of OAG is 5 times that of PACG.<sup>5</sup> However, it has been estimated that half the glaucoma in the world is caused by PAC.<sup>6</sup> As approximately 75% of patients with PACG in Asia have optic nerve damage, screening strategies that detect functional damage in OAG may also be suitable for PACG.<sup>7</sup> Such tests will not detect eyes without functional damage, or eyes at risk for PAC:

- Tonometry will only detect PAC in the presence of raised IOP
- Optic disc examination and perimetry will only detect PAC in the presence of a damaged optic disc or VF

The ideal way to identify PAC and eyes at risk for PAC is to examine the angle using a gonioscope. The required clinical expertise and instrumentation render gonioscopy inappropriate for screening.

Methods to identify eyes at risk for PAC include ACD measurement and ACD-axial length ratio. The sensitivity and specificity of these techniques do not make them appropriate for screening.

Easier techniques include the torchlight test and the van Herick test. In the torchlight test, a light is shone from the temporal side onto the cornea, parallel but anterior to the iris. A shadow on the nasal limbus identifies an eye with a shallow anterior chamber at risk for PAC. The sensitivity of the torchlight test is 80% to 86% and the specificity is 69% to 70%.<sup>7,8</sup>

The van Herick test uses a slit beam to compare the peripheral ACD with the corneal thickness. The sensitivity and specificity of the test are 61.9% and 89.3%, respectively.<sup>8</sup> Expressing the test in decimals yields similar results.<sup>9</sup>

If the van Herick test is positive and the IOP is increased, the specificity improves to 99%. Therefore, as a screening strategy, if the IOP is raised and the van Herick test is positive, the specificity is sufficiently high to diagnose PAC. However, this strategy is not appropriate for diagnosis or screening in an ophthalmology clinic; the latter requires the use of a gonioscope.

## SPECIFIC RECOMMENDATIONS

### Universal Glaucoma Screening

This is not recommended as a strategy:

- Universal screening is not feasible for developing countries without adequate infrastructure
- Adequate infrastructure implies:
  - the availability of the expertise (trained ophthalmologists), time, and instrumentation required to confirm the diagnosis among people with positive test results in an appropriately modern manner
  - the availability of expertise (trained surgeons) and instrumentation to appropriately treat those for whom the diagnosis is confirmed

The requirements for the diagnosis and management are covered in Section 1.

Individual countries need to decide on universal screening based on an assessment of the costs, benefits, and societal preferences.

### Opportunistic Glaucoma Screening

Any person older than 35 years who seeks ophthalmic attention for any reason should have a comprehensive ophthalmic examination. This includes tests used to opportunistically screen for glaucoma (Tables 3.6 and 3.7).

Currently, the optimal method for detection of individuals with glaucoma is periodic routine comprehensive eye examinations. The feasibility of this depends on the health care system in an individual country. In lieu of an ideal screening method, opportunistic screening is recommended.

**Table 3.6.** Additional requirements for glaucoma diagnosis in an ophthalmology clinic.

| Test   | Ideal  | Acceptable   | Less than ideal       | Comments  |
|--|--|--|-----------------------|---|
| Gonioscopy   | Indentation gonioscopy using a Sussman, Zeiss, or Posner lens  | Goldmann single or 2-mirror lens with 'manipulation'       | NA                    | NA  |
| VF examination (if the IOP is >21 mm Hg and/or the disc is suspicious) | A full threshold test using a calibrated white-on-white automated perimeter or Goldmann perimetry  | Frequency doubling Hensons VF screen<br>Bjerrums screen    | NA                    | A trained technician must perform Goldmann perimetry, automated perimetry, frequency doubling perimetry, Hensons screen, and Bjerrums screen  |
| Optic disc photography/imaging (if optic disc suspicious)              | Dilated stereoscopic evaluation by slit-lamp biomicroscopy <b>and</b> stereoscopic optic disc photography<br>One of the optic disc imaging technologies (OCT, GDx, or HRT) for follow-up for early and moderate glaucoma | Dilated stereoscopic evaluation by slit-lamp biomicroscopy | Direct ophthalmoscopy | Imaging has a limited role in the diagnosis of glaucoma<br>In the hands of an experienced clinician, can provide additional information for borderline cases<br>May have larger role to play in identifying early progression |

**Table 3.7.** Tests appropriate for screening for glaucoma.

| Test   | Ideal   | Acceptable   | Less than ideal                      | Comments  |
|--|---|--|--------------------------------------|---|
| Tonometry  | Goldmann applanation tonometry  | Perkins applanation tonometry, Tonopen, or similar   | Pneumotonometer<br>Schiotz tonometer | New tonometry methods that take into account corneal hysteresis may have an important role in the future  |
| Dilated evaluation of the optic disc                         | Dilated stereoscopic evaluation by slit-lamp biomicroscopy<br>Stereoscopic optic disc photography | Direct ophthalmoscopy  | NA                                   | Monoscopic optic disc assessment will underestimate CDR and will increase false negative results  |
| Slit-lamp biomicroscopy and van Herick test                  | NA  | NA   | NA                                   | The torchlight and/or van Herick tests are not appropriate tools to screen for AC<br>A positive torchlight or van Herick test requires confirmation by gonioscopy<br>A negative test does not exclude PAC   |
| Gonioscopy — for all screening performed by ophthalmologists | Indentation gonioscopy using a Sussman, Zeiss, or Posner lens                                     | Goldmann single- or 2-mirror lens with 'manipulation'                                      | NA                                   | Mandatory for every glaucoma suspect, irrespective of whether the suspicion is based on raised IOP, optic disc, or VF findings<br>In view of the high prevalence of AC in the region, routine gonioscopy for all clinic patients is recommended<br>Ideal to have both types of gonioscope |
| VF examination   | NA  | Full threshold<br>Suprathreshold white-on-white<br>Frequency doubling<br>Hensons VF screen | NA                                   | Has limited value unless comprehensive examination suggests glaucoma <b>and</b> tests are performed reliably<br>Should not be performed universally   |

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## 3.3 FREQUENTLY ASKED QUESTIONS

### EPIDEMIOLOGY OF GLAUCOMA IN ASIA

**Q:** Is PACG more common than POAG in South East Asian countries?

**A:** PACG is not more common than POAG in South and East Asian countries, but it is more common than in western populations. Population-based studies have reported the prevalence of PACG to vary from 0.5% to 2.2% in South East Asian countries. Population-based studies of Caucasian and African populations have reported a prevalence of PACG of 0.4% to 0.7%, mostly among people older than 40 years.

**Q:** Does PACG cause more blindness than POAG?

**A:** PACG causes 3 times more blindness than POAG.

**Q:** Does the clinical presentation of AC vary in different parts of Asia?

**A:** Yes, acute AC is more common in China than in India. Compared with CAC, acute AC is rare in the Indian subcontinent.

**Q:** What is the natural history of PAC?

**A:** There is little information available: a population-based study from South India reported 22% progression of PACS to PAC and 29% of PAC to PACG over 5 years. Recently, a study in a high-risk Mongolian population, with a central ACD of <2.53 mm, reported a 20.4% incidence of PACS over 6 years. A study in Eskimos reported a 35% progression rate for PACS after 10 years.

### SECTION 1.1 PATIENT ASSESSMENT

**Q:** Is it necessary to take a full medical history as part of the glaucoma evaluation?

**A:** It is essential; various systemic diseases and treatments can affect IOP and glaucoma. For example, a  $\beta$ -blocker may be contraindicated for a patient with heart block or asthma. If the patient is taking a systemic  $\beta$ -blocker, a topical  $\beta$ -blocker will be less effective for lowering IOP and may cause systemic side effects. Some antiglaucoma medications may be contraindicated with certain systemic drugs, for example, MAOIs such as isocarboxazid and phenelzine contraindicate  $\alpha_2$ -agonists.

**Q:** Why is a family history of glaucoma important?

**A:** Risk for glaucoma increases 5-fold for a patient with a positive family history. First-degree blood relatives are at highest risk.

**Q:** Can steroid ointment used for skin lesions increase IOP?

**A:** Steroids in any form can increase IOP. A detailed drug history is necessary, especially if the response to treatment changes, for example, loss of IOP control in a previously stable patient.

**Q:** Is migraine associated with NTG? Are there other clinically important associations to look for?

**A:** Most population- and clinic-based studies have found migraine to be a risk factor for NTG. A migrainous patient may be approximately 2½ times more likely to progress than a patient without migraine. All glaucoma patients and suspects should be asked about a history of systemic vasospastic diseases (Raynaud's phenomenon), blood loss, severe hypotension (haemodynamic crisis), and stroke.

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**Q:** Do we need to check CCT for all glaucoma patients and suspects?

**A:** Ideally, yes. IOP measurement is not precise, and there is no 'correction' factor to make it accurate. CCT should be checked in suspected OH and NTG before diagnosis, and especially before a patient undergoes expensive or invasive investigations such as imaging or angiography.

**Q:** What is the effect of corneal oedema on IOP measurement?

**A:** Corneal oedema can cause the tonometer to read falsely low. With mild corneal oedema secondary to contact lens wear, the IOP measurement is falsely high.

**Q:** Is the type of musical instrument a patient plays important for the management of glaucoma?

**A:** Playing wind instruments increases IOP considerably by the Valsalva manoeuvre increasing episcleral venous pressure. Similarly, a person who performs head-down Asanas (Sirsasana) as part of Yoga can have a 2- to 3-fold increase in IOP.

**Q:** What is the current role of the Schiøtz tonometer?

**A:** The Schiøtz tonometer has no role to play in modern glaucoma diagnosis and management.

**Q:** As the air puff non-contact tonometer works on the applanation principle, can it be used instead of the GAT?

**A:** The air puff tonometer has reasonable agreement with GAT in the physiological range of IOP. However, its variability is higher and GAT remains the gold standard.

**Q:** What is the role of the diurnal variation test? Do all patients require it?

**A:** IOP fluctuates over 24 hours — the size of the fluctuations is different for each patient. Knowing the baseline before starting medication is important, as is knowing the effect of the medication during the day. As a full 24-hour diurnal variation test is logistically difficult for patients and practitioners, it is best to obtain several IOP readings during the day, or at different times for clinic visits. A '24-hour' or full diurnal variation test is recommended before subjecting a patient with 'NTG' to invasive or expensive investigations, and for patients who progress despite acceptable IOP readings during office hours.

**Q:** How is applanation IOP performed for a patient with high astigmatism (>4 D)?

**A:** In this scenario, the usual method gives an inaccurate IOP reading. The Goldmann or Holladay methods can provide an accurate IOP measurement, but alterations to the standard method are required. Clinically, the Holladay method is easier: measure the IOP with the tonometer prism at 90° and 180°, then take the mean of these 2 readings to derive the IOP. For the Goldmann method, the red line on the applanation prism (set at 43°) is adjusted to the flat axis of the corneal curvature and the measurement is taken as usual. Alternatively, the Tonopen can be used as it is less influenced by corneal shape.

**Q:** Can the van Herick method be used instead of gonioscopy for angle assessment in the clinic?

**A:** No, all clinic patients need gonioscopy. If it is impossible to do gonioscopy due to patient factors, as long as the van Herick test result is at least one-quarter corneal thickness and the torchlight test is negative, it is almost 98% certain that the angle is not closed.

**Q:** What is the ideal gonioscope? Can the Goldmann 3-mirror lens be used for gonioscopy?

**A:** It would be ideal to have an indentation gonioscope (Susmann, Zeiss, or Posner

4-mirror) as well as a Goldmann single or 2-mirror lens. The indentation gonioscope uses the patient's tear film as coupling fluid, and allows easier indentation to look for anatomical landmarks and to distinguish appositional from synechial closure, and to perform routine gonioscopy. However, it is easier to put pressure on the eye and artificially open the angle with this type of lens. The disadvantages of the 3-mirror gonioscope are that it does not have the right optics (mirror height and distance from the centre of the lens) for gonioscopy, it is harder to use for indentation gonioscopy, it is bulky, and it needs coupling fluid. The latter disadvantage also applies to the single and 2-mirror lenses, making routine gonioscopy difficult.

**Q:** How often should patients with glaucoma undergo gonioscopy? What if the patient is known to have POAG?

**A:** Gonioscopy is mandatory at the initial evaluation to assess whether the angle is closed or open and, in the presence of AC, to distinguish the amount of synechial versus appositional closure. How much the angle opens at indentation predicts how much it will open after LPI. In an open angle, gonioscopy identifies other findings in the angle, for example, PXF material or irregular pigmentation. After LPI, repeat gonioscopy identifies the response to the procedure (when the effect of pilocarpine has worn off). Subsequently, gonioscopy can be performed if there is a suspicion that something has changed. Patients with POAG can develop angle narrowing and require regular gonioscopy, especially if anything changes. Changes in angle configuration and other findings (pigment, new vessels) provide information about secondary risk factors within the eye.

**Q:** What is the best clinical method of optic disc assessment? Can a direct ophthalmoscope be used?

**A:** Ideally, dilated stereo-biomicroscopic disc evaluation with a contact or non-contact lens is best. In experienced hands, direct ophthalmoscopy can be excellent.

**Q:** Should the optic disc size always be measured?

**A:** CDR depends on disc size. While it is unnecessary to measure accurately the size of every disc, it is important to know whether the disc is 'small', 'medium', or 'large'. A small cup may be abnormal for a small disc. A large cup may be normal for a large disc. The disc can be measured using the slit lamp and a correction made for the lens being used. Charts are available for this correction.

**Q:** How can the RNFL be assessed clinically?

**A:** By using dilated stereo-biomicroscopic evaluation with a contact or non-contact lens with the red-free filter (green light). An indirect ophthalmoscope can also reveal defects. However, these techniques need experience, and the RNFL cannot be visualised in all patients, for example, those with lens opacities or very light-coloured fundi.

**Q:** Is it necessary to examine the anterior segment again after dilatation?

**A:** Post-dilatation flare and pigment release can help diagnosis. Look for early signs of PXF.

**Q:** Is it necessary to dilate the eye for routine optic disc assessment?

**A:** Dilated optic disc examination is ideal. In addition to glaucoma evaluation, the rest of the fundus should be examined for related and unrelated pathology, as several retinal disorders can produce glaucoma-like field defects.

**Q:** Can HRT, GDx, OCT or other imaging technologies be used in isolation to diagnose glaucoma?

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**A:** Abnormal HRT, GDx, or OCT results alone are not adequate to diagnose glaucoma. Glaucoma diagnosis is based on both structural (optic disc) and functional (VF) changes, along with a patient's clinical profile.

**Q:** What is the role of imaging technologies for optic disc evaluation in the clinic?

**A:** According to the WGA consensus statement, "The current literature does not provide the requisite evidence to validate any of these imaging instruments for widespread clinical use. Currently, in the hands of an experienced clinician who understands the strengths and limitations of the instruments, information may be helpful in many clinical situations."

**Q:** How do you estimate optic disc size using a high plus lens at the slit lamp?

**A:** Focus a co-axial thin vertical slit beam on the optic disc. Shorten the length of the slit until it covers the longest vertical extent of the optic nerve head. The scale measurements of the slit lamp are recorded and multiplied by the appropriate correction factor for the lens used; multiplication factors depend on lens power and the manufacturer. This technique gives the vertical diameter of the optic disc.

**Q:** Can POAG and ACG coexist?

**A:** A patient with POAG can develop anatomically narrow angles, which needs to be recognised and managed appropriately. This is likely to be related to enlargement of the anteroposterior diameter and a more anterior position of the lens, which occurs over time.

**Q:** Do all PAC eyes with open angles need to be followed up post-LPI?

**A:** PAC is a multi-mechanism disease. An LPI only treats one of the components (pupillary block); these patients must be followed up. Eyes with PAC that have undergone LPI need to be monitored in the usual manner (and have gonioscopy repeated), since PAS or raised IOP in such eyes results from angle damage and outflow restriction; further increases in IOP can develop. A patent LPI is not a cure.

**Q:** Is unilateral POAG common? How should such a patient be assessed?

**A:** Although POAG may be very asymmetric with minimal damage in 1 eye and advanced loss in the other, true unilateral POAG is uncommon. Carefully assess such eyes to rule out AC or a secondary glaucoma, for example, PXF, pigment dispersion, angle recession, or uveitis.

**Q:** How should glaucomatous disc changes be differentiated from non-glaucomatous disc changes?

**A:** If disc pallor is out of proportion with cupping, then it is more likely to be non-glaucomatous. Rim pallor is 94% specific for a non-glaucomatous disc.

**Q:** What is the significance of optic disc (peripapillary nerve fibre layer) haemorrhage?

**A:** This finding is very specific for glaucoma, and suggests an active disease process. Disc haemorrhage in OH increases the risk of conversion to POAG 4- to 6-fold; in POAG, disc haemorrhage increases the risk for progression of VF loss by 4- to 5-fold. NTG has a higher incidence of optic disc haemorrhage.

**Q:** What is the current role of short-wave automated perimetry or blue-on-yellow perimetry in glaucoma management?

**A:** Short-wave automated perimetry may demonstrate VF loss up to 5 years earlier than standard automated white-on-white perimetry in glaucoma suspects. As glaucoma usually occurs in age groups with nuclear sclerosis, short-wavelength automated perimetry results need careful interpretation.

**Q:** What is the relationship of blood pressure to glaucoma?

**A:** Raised blood pressure has been associated with increased IOP, but it is not a simple 1:1 relationship. Systemic hypertension has been associated with glaucoma in hospital-based studies, and some population-based studies show a link. Low perfusion pressure has been linked to glaucoma.

**Q:** What is the value of provocative testing for glaucoma diagnosis?

**A:** Provocative tests (water drinking test for POAG, dark-room and dark-room prone tests for PAC) are rarely performed now. A simpler manoeuvre involves checking IOP and gonioscopy in the mid-dilated position in PACS: elevated IOP and angle narrowing may change the management plan.

**Q:** How should the applanation tonometer tip be sterilised?

**A:** The head should be cleaned with wipes soaked in 70% isopropyl alcohol or 3% hydrogen peroxide. Time should be allowed for the alcohol to evaporate, and sterile saline should be used to wash off the hydrogen peroxide before the next patient to prevent iatrogenic corneal ulcers.

**Q:** What is the effect of laser refractive surgery on IOP measurement?

**A:** Refractive surgery, including LASIK, LASEK, and PRK, causes a falsely low IOP measurement. A similar depth of ablation will result in a greater decline in IOP measurement following LASIK than following surface ablation. Pascal dynamic contour tonometry and ocular response analyser are less sensitive to changes in corneal biomechanics.

## SECTION 1.2 RISK CATEGORIES AND TREATMENT TARGETS

**Q:** Should the target IOP be calculated for every patient?

**A:** In some ways, this is done for every patient, but is not always formally written in the notes. IOP-lowering should be individualised for each patient: an individualised (target) IOP tends to prevent over- or under-treatment and minimises treatment effects on QOL. There is no definitive evidence for the concept or the methods used to determine a target IOP.

**Q:** There are various tables and formulae available to calculate target IOP? Which one should be used in clinical practice?

**A:** Most formulae and tables provide similar values. A general rule is to reduce the IOP by the following:

- Mild glaucoma —  $\geq 20\%$
- Moderate glaucoma — 30%
- Severe glaucoma —  $\geq 40\%$

Percentage IOP reductions are not appropriate targets when the pretreatment IOP is very high or very low, so the following targets should be aimed for:

- High risk for progressive damage — 1 to 2 SDs less than the mean IOP for the local patient population
- Moderate risk for progressive damage — mean IOP for the local patient population
- Low risk for progressive damage — 1 to 2 SDs above the mean IOP for the local patient population

**Q:** Does the target IOP need to be calculated only at the beginning of management?

**A:** Target IOP is not a static or magic number, but a range that should change depending on the results of long-term monitoring. If a patient progresses at the target IOP, lower it further. If a patient is stable at the target IOP, perhaps the target could be reset to a higher level (with ongoing careful monitoring), allowing withdrawal of some treatment.

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**Q:** A healthy 75-year-old Indian patient has moderate glaucoma. However, in India, the average life expectancy is 66 years. How should this patient be treated?

**A:** The average life expectancy is calculated from birth. A person who has reached the age of 75 years is a survivor and, if healthy, could live for many more years, so should be treated accordingly. Calculators on the Internet facilitate determination of a patient's life expectancy. This might enable a more specific management plan.

**Q:** Do all patients with NTG require systemic evaluation such as brain scan or carotid Doppler test before diagnosing them with NTG?

**A:** No. A small proportion of patients with NTG, especially those who are younger, have unilateral disease, disc pallor out of proportion with cupping, atypical VF defects, and colour deficiency, require appropriate cardiovascular and/or neurological investigation.

**Q:** What is the goal of glaucoma management? Is it IOP reduction, prevention of VF progression, or prevention of progression of optic disc damage?

**A:** Glaucoma management aims to preserve visual function and QOL for individual patients for their lifetime. The aim is not to treat only the IOP, optic disc, or VF, but to treat the patient as a whole to provide maximum benefit with minimal side effects.

**Q:** Should target IOP be similar for POAG and PACG?

**A:** Target IOP calculation depends on structural and functional damage, baseline IOP at which the damage occurred, age, and presence of additional risk factors. Even though PACG causes more blindness than POAG, this has not been incorporated into current target IOP calculations.

**Q:** What is the importance of knowing a patient's age and presence of systemic diseases while deciding the management options?

**A:** These factors are important. For a 90-year-old patient with ischaemic heart disease, history of myocardial infarction, diabetes, hypertension, COPD, and early glaucoma in both eyes, the treatment should not be too aggressive.

**Q:** If a patient who is a glaucoma suspect has an IOP of 24 to 28 mm Hg, CCT of 540  $\mu\text{m}$  in both eyes, optic disc showing inferior wedge-shaped RNFL defects, and normal white-on-white perimetry, and short-wave automated perimetry shows defects, how should the target IOP be decided?

**A:** RNFL defects with a suspicious disc suggest a diagnosis of preperimetric glaucoma. The short-wave automated perimetry defects, if repeatable, would reinforce this diagnosis. Treatment depends on other factors such as age, general health, and family history.

## SECTION 2.1 INITIATION OF TREATMENT

**Q:** With so many drugs available, which one should be used as first-line treatment?

**A:** The ideal drug should be effective, easy to use, affordable, with no systemic side effects, and minimal or no ocular side effects. PGAs are popular as first-line agents.  $\beta$ -Blockers and  $\alpha_2$ -agonists might be appropriate, especially in countries where costs prohibit expensive drugs.

**Q:** The AGIS suggested that an IOP of 12 mm Hg minimises glaucoma progression. Should every patient's IOP be reduced to this level?

**A:** The IOP level of 12 mm Hg arose from a post-hoc analysis of AGIS data, which should be interpreted with care. Lowering IOP is beneficial and, overall, the lower the better. Aiming for an IOP of 12 mm Hg is not necessary for all patients, and could provoke needless side effects.

**Q:** The EMGT, AGIS, and OHTS studies show that if IOP is lowered by 1 mm Hg then the risk for progression is lowered by 10%. How should these data be used in clinical practice?

**A:** Such statements are based on statistical manipulation of the data, an explanation of which is beyond the scope of FAQs. The statement does not necessarily reflect real life and should not lead to needless aggressive treatment. Use information from trials but treat patients as individuals, not as study populations.

**Q:** A patient is using a topical  $\beta$ -blocker, but needs better IOP control. Should another aqueous suppressant such as an  $\alpha_2$ -agonist be added, or is it better to add a PGA?

**A:**  $\beta$ -Blockers act by suppressing aqueous, while PGAs act on the uveoscleral outflow pathway, which might be more effective. A unilateral trial of a PGA would be appropriate. As the aim is to switch medications where possible, rather than add, if IOP-lowering with a PGA is sufficient, withdrawal of the  $\beta$ -blocker could be attempted.

**Q:** The OHTS shows that IOP reduction by 20% reduces risk for progression by 50%. Should all patients with OH be treated?

**A:** The OHTS reported reduction of progression by 50% for the treatment group. Not all patients with OHTS will progress. As 9.5% of patients in the control group progressed, 90.5% did NOT; this figure was reduced to 4.4% in the treatment group over 5 years. Most of the endpoints were disc related, not functional. Accordingly, not all patients with OH require treatment. Using OHTS data and other literature, if patients with high IOP (>26 mm Hg), thin corneas (<550  $\mu$ m), large vertical CDR, and high PSD on VF tests are treated, the maximum benefit could be obtained. Other risk factors such as family history of glaucoma need to be considered.

**Q:** If a patient with PAC has an IOP in the mid-twenties several weeks after LPI, how should he be treated?

**A:** Management depends on the gonioscopy post-LPI and the state of the discs/fields. The main objective of LPI is to open the angle, to prevent anterior segment damage (PAS) and subsequent IOP elevation. If the angle still shows a PACS configuration, determine the underlying mechanism of residual AC and treat accordingly. Plateau iris may need ALPI (or long-term low-dose miotic, if tolerated). Once the angle is open, management is similar to POAG; some patients may need an IOP-lowering agent.

**Q:** If a patient with pigment dispersion syndrome without disc and VF changes has an IOP between 22 mm Hg and 24 mm Hg, should LPI be performed or is observation sufficient?

**A:** In the stage of active pigment dispersion, early in the disease, LPI might correct the reverse pupillary block.

**Q:** Most studies have reported that topical CAIs reduce IOP by approximately 20%. However, whenever this has been started as a third-line agent after a  $\beta$ -blocker and PGA in the clinic, a 20% IOP reduction is never obtained. Why is this?

**A:** IOP cannot be lowered below episcleral venous pressure medically. IOP reduction achieved by first-, second-, and third-line drugs becomes progressively less. IOP reduction of 20% is more likely if the CAI is used first.

**Q:** Several glaucoma specialists advocate a unilateral drug trial, but the recent literature shows this is ineffective. Should a unilateral drug trial still be used to prove that a drug is working?

**A:** A recent retrospective study concluded that unilateral drug trials might not be helpful. Like any technique, a unilateral trial does not always work. Another study showed that a

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unilateral drug trial was effective approximately 80% of the time. If a patient is asked to use a medication that is expensive and may have side effects indefinitely, there should be little doubt that it works. Despite its weaknesses, the unilateral drug trial is probably the best way to demonstrate effectiveness.

**Q:** A patient has moderate glaucoma and the IOP is 28 mm Hg in both eyes. If a unilateral drug trial is used, 1 eye will remain unprotected for a period of 4 to 6 weeks. Is this safe?

**A:** Glaucoma is a chronic progressive disease, so has usually been present for a long time. Nothing significant is likely to happen during the short period of a unilateral trial.

**Q:** Can a relatively selective  $\beta_1$ -blocker (Betaxolol) be prescribed to a patient with asthma?

**A:** Even though they are selective and have fewer respiratory side effects, relatively selective  $\beta$ -blockers can worsen asthma; they should be used with caution, if at all. The ‘double-DOT’ instillation technique should always be used to minimise systemic absorption.

**Q:** What should be remembered when starting a unilateral trial with a  $\beta$ -blocker?

**A:** Used unilaterally,  $\beta$ -blockers have a contralateral effect of approximately 1.5 mm Hg; this correlates with the IOP fall in the treated eye and with the baseline IOP in the contralateral eye. Take this into account when assessing the response to the unilateral trial.

## SECTION 2.2 MEDICAL TREATMENT

**Q:** If a patient has an IOP that is always maintained in the low teens, but non-adherence is suspected, with the drops used only before visiting the clinic, how is adherence checked?

**A:** Non-adherence (non-adherence) is serious, and there is no foolproof method to check. Filling of prescriptions and the amount that remains can be monitored. Patients can be asked whether ANY medication has been missed since the last consultation. The specificity of a “Yes” answer is very high — if a patient admits to missing a single dose, there is likely to be significant non-adherence. However, if a patient’s glaucoma is stable, should adherence be increased, which may be over-treating? This can be a difficult area and one that potentially leads to conflict in the doctor-patient relationship. The best method of ensuring adherence (or concordance) is to build a trusting relationship with the patient so that they understand the need for treatment.

**Q:** What is maximal tolerable medical therapy? If a patient who is a business executive and travels a lot can instil only 1 drop per day (he is using a combination of a PGA and a  $\beta$ -blocker), can this be considered as maximal tolerable therapy for him?

**A:** Theoretically, maximal tolerable medical therapy is the minimum number and concentration of drugs (within the combination of different classes of medications) that provides maximum IOP-lowering for that patient. Practically, it is the greatest burden of drop instillation and side effects a patient can manage, and is very different for different individuals. If a patient cannot use medications reliably more than once per day then, for this patient, such a regimen might be considered maximal tolerable therapy. The patient must be helped to understand the options and risks, and participate in planning realistic treatment strategies.

**Q:** Should topical CAIs be avoided for patients with poor corneal endothelial function? What are the guidelines?

**A:** In a compromised cornea with poor endothelial function, topical CAIs can precipitate

corneal oedema. Topical CAI use should be minimised for patients with poor corneal endothelial function (Fuchs' dystrophy, post-surgical corneal oedema).

**Q:** A patient developed choroidal effusion and AC after starting a systemic CAI. He has used this before with no side effects. How does this happen?

**A:** Choroidal effusion and secondary AC is a rare idiosyncratic complication of systemic CAIs and other drugs. AC follows forward rotation of the ciliary body, pushing the iridolenticular diaphragm forwards. This mechanism requires a sensitising dose and an inciting dose. The effusion is typically not seen at first use.

**Q:** What is the current role of neuroprotection in glaucoma?

**A:** Currently, there is no clinical evidence for neuroprotection as an isolated strategy. CCBs might help in the presence of marked vasospastic disease (migraine and/or Raynaud's phenomenon).  $\alpha_2$ -Agonists are prescribed primarily for their IOP-lowering effects. Phase III trials of the NMDA receptor blocker memantine yielded mixed results.

**Q:** For a patient with hypertension, should the physician change the antihypertensive treatment to a CCB?

**A:** If a glaucoma patient with progressive damage and marked vasospastic disease is also taking antihypertensive agents, systemic CCBs could be considered (although there is no firm evidence of their efficacy for glaucoma). Systemic CCBs can have significant side effects and, given with topical  $\beta$ -blockers, can potentiate negative inotropic and chronotropic cardiac effects. Ensure a patient taking systemic antihypertensive agents is not being over-treated. Where feasible, check 24-hour blood pressure and pulse measurements to detect and rectify nocturnal blood pressure dipping.

**Q:** There are numerous generic PGAs available locally. Are they as good as the original molecule?

**A:** Most generic PGAs have not been compared with the original molecules. When one latanoprost generic was compared with Xalatan™, the IOP reduction was less than for the original drug. If a generic PGA is used, a unilateral trial should be started.

**Q:** Do fixed combination therapies reduce IOP equally to the individual components?

**A:** Medications used individually tend to lower IOP more than if used in fixed combinations. However, fixed combinations are more convenient, encourage adherence, reduce preservative load to the eye, and may be cost-effective.

**Q:** Which antiglaucoma medications can be used during pregnancy?

**A:** Direct research into the use of antiglaucoma medications during pregnancy is lacking, limiting the safety evidence. The USA FDA has classified drugs for use during pregnancy based on animal or human studies. Brimonidine is classified as a class B medication (presumed safety based on animal studies). All other glaucoma medications are classified as class C (uncertain safety; no human or animal studies show an adverse effect). SLT can be considered to try to reduce drug use. For advanced glaucoma, cyclophotocoagulation is an option. Use of MMC is contraindicated and surgery is also associated with a high risk after the first trimester. IOP can spontaneously drop later in the pregnancy, so sometimes watching and waiting may be the best strategy.

**Q:** If a patient does not respond to one PGA, should other PGAs be used?

**A:** Switching between PGAs might be effective, so consider this before switching to another group of medications.

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## SECTION 2.3 LASER TREATMENT

**Q:** Which is more effective: ALT or SLT?

**A:** Most reports suggest that they have similar IOP-lowering effects.

**Q:** Does ALT/SLT alter the success rates of future glaucoma filtering surgery?

**A:** Eyes that have undergone ALT have been reported to have poorer success rates for trabeculectomy. This information is not yet available for SLT.

**Q:** Is there a significant risk of cataract progression following LPI?

**A:** There are a few reports of cataract progression following LPI. However, there are no definite figures for the increased risk.

**Q:** Is it possible for a patent LPI to become blocked with time? Should gonioscopy be performed at regular intervals, even with a patent iridotomy?

**A:** Blockage of a patent iridotomy is uncommon with the YAG laser. Blockage of the LPI or changes in lens thickness can alter angle anatomy. Regular gonioscopy is important.

**Q:** What is the role of iridoplasty in plateau iris?

**A:** Iridoplasty can open the angle in patients with plateau iris and should be considered. Pilocarpine does the same, and can be used instead, in some instances. Iridoplasty seems to have a role in acute ACG resistant to conventional treatment.

**Q:** Can SLT be used as primary therapy?

**A:** SLT can be used as primary therapy, or to decrease the number of medications required for patients whose IOPs are well controlled but for whom the drug regimen is a burden.

## SECTION 2.4 SURGERY

**Q:** Should intravenous hyperosmotics be used routinely prior to glaucoma filtering surgery or only in certain situations?

**A:** Routine use is not necessary. To avoid suprachoroidal haemorrhages intraoperatively, preoperative IOP should not be too 'high'. Use standard medications, including hyperosmotics. Some surgeons gradually decompress the eye intraoperatively with a controlled paracentesis.

**Q:** In what situations could filtering surgery be considered as first-line treatment?

**A:** CIGTS demonstrated that primary filtering surgery was effective to control IOP. Socio-economic factors or poor access to care necessitates primary filtering surgery. A patient presenting with visually significant cataract and glaucoma could warrant primary combined surgery, especially in a primary care setting.

**Q:** Which is preferred, a 1- or 2-site phacotrabeculectomy?

**A:** No evidence exists that one technique is better than another.

**Q:** In a single-site trabeculectomy, is creation of a trabeculectomy flap mandatory or can the cataract incision be used to create the trabeculectomy?

**A:** Either technique may be used, depending on the surgeon's preference.

**Q:** Does the size of the ostium matter?

**A:** Filtration is dependent on a number of factors, including the relative sizes of the scleral

flap and ostium. A critical factor is the extent of overlap between the anterior scleral flap and the wound bed posterior to the trabeculectomy ostium. This is particularly relevant in the immediate postoperative period; long-term results depend more on wound healing processes. In AC, anterior block excision should be used to facilitate aqueous access to the fistula.

**Q:** Should a trabeculectomy punch or surgical scissors or knives be used to create the ostium?

**A:** Either technique could be used depending on the experience of the surgeon.

**Q:** Which is preferable — ALS or releasable sutures?

**A:** Both techniques could be used to modulate filtration postoperatively. ALS requires additional equipment (the laser and the lens), while a releasable suture carries the risk of damaging the flap (if the first attempt at placing the releasable suture is not successful), windshield wiper keratopathy, or inadvertent release. If the Tenon's capsule is thick, the suture might not be visible for ALS. The releasable technique can be modified by passing the suture intrastromally from the cornea to reduce the risk of keratopathy.

**Q:** Performing fornix-based trabeculectomy for a number of years has always obtained good success rates. Should limbus-based trabeculectomy be used instead?

**A:** Both techniques work well depending on the surgeon's expertise. The fornix-based technique with a large treatment area was devised to minimise the localised, thin, overhanging blebs more common with limbus-based surgery. Meticulous closure of the conjunctival edge is very important. The formation of localised, thin, overhanging blebs can be minimised by incorporating principles of large treatment area into limbus-based surgery.

**Q:** Is it justified to suggest early cataract surgery to a patient with PAC and early visually significant lens changes instead of LPI?

**A:** If the cataract is interfering with the patient's daily activities then early cataract surgery is an acceptable alternative to a YAG LPI. The type of surgery undertaken will also depend on the extent of closure. Theoretically, operating on an eye with an open angle (after LPI) may decrease the risk of ciliary block glaucoma.

**Q:** If a patient with glaucoma who is stable and well controlled with a single medication or fixed combination develops visually significant cataract, should cataract surgery alone be planned, or should it be combined with filtering surgery?

**A:** Either option is acceptable; consult with the patient. Take into account the greater risks with the addition of glaucoma surgery, whether the patient had significant QOL issues with medication, could afford therapy, and was willing to continue to use medication indefinitely.

**Q:** Do glaucoma medications need to be stopped prior to cataract surgery? What about PGAs?

**A:** Miotics and PGAs disrupt the blood-aqueous barrier. They could be withdrawn approximately 1 week preoperatively and resumed 4 to 6 weeks later (once signs of inflammation have disappeared). Alternative agents may need to be considered for this period. Evidence for which agents to stop and for how long is lacking.

**Q:** Is it mandatory to perform a YAG LPI before cataract/glaucoma surgery in a patient with PACS, PAC, acute PAC, or PACG?

**A:** No. Be aware of the increased risk for ciliary block glaucoma in such eyes.

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**Q:** What are the indications for glaucoma drainage device surgery?

**A:** Consider the following situations for a glaucoma drainage device:

- Failed trabeculectomy with MMC
- Some secondary glaucomas:
  - neovascular glaucoma
  - uveitic glaucoma
  - aphakic or pseudophakic glaucoma
- Scarred anterior conjunctiva:
  - conjunctival scarring due to multiple surgeries
  - ocular conditions: allergies, inflammation
  - chemical injury
- Marked limbal thinning

**Q:** Does the Tube versus Trabeculectomy study justify primary implant surgery for glaucoma?

**A:** No. The Tube versus Trabeculectomy study was designed to assess the efficacy and safety of an implant in patients who had already undergone either glaucoma or cataract surgery; therefore, the results cannot be extrapolated to patients who have not had surgery. The study follow-up period was only 12 months.

**Q:** How does non-penetrating glaucoma drainage surgery compare with trabeculectomy?

**A:** Trabeculectomy seems to achieve greater IOP-lowering than either viscocanalostomy or deep sclerectomy. The latter are not suitable for ACG. On the other hand, non-penetrating glaucoma drainage surgery seems to have fewer complications. Failed non-penetrating glaucoma drainage surgery or failed trabeculectomy compromises the results of subsequent glaucoma surgery.

**Q:** When should the conjunctival suture be removed after trabeculectomy?

**A:** Absorbable sutures do not need removal, but may increase scarring. Non-absorbable sutures could be removed after 1 to 2 weeks, or longer if MMC was used intraoperatively.

**Q:** What is the effect of cataract surgery in eyes with a functioning glaucoma filter?

**A:** A functioning filter has an increased risk of failure after cataract surgery — with ECCE more than with clear corneal phacoemulsification. If the interval between trabeculectomy and cataract extraction is less than 6 months, the failure rate is highest.

## SECTION 3.1 FOLLOW-UP

**Q:** Do the follow-up protocols for primary and secondary glaucomas differ?

**A:** Secondary glaucoma tends to present with higher IOP, which is often more difficult to control with an active primary process (steroid responsiveness, trauma, neovascularisation, inflammation). These patients may require closer follow-up. This decision needs to be made on an individual patient basis not on a protocol.

**Q:** Is juvenile-onset POAG more likely to progress than adult-onset POAG? Do these patients need closer follow-up?

**A:** Juvenile-onset POAG tends to have a higher presenting IOP and often has a worse response to medical treatment than adult-onset disease. These patients require close follow-up to assess response to medication and to detect progression.

**Q:** Is a sudden deterioration in VF in a patient with well-controlled IOP, whose VFs have remained stable in the past, an indication for further investigation?

**A:** If there is no other significant history (steroid use, trauma, acute systemic hypotension) or clinical finding (new retinal pathology) that can explain the progression, imaging of the visual pathway should be considered.

**Q:** Do patients who have undergone filtering surgery and are stable need to undergo regular follow-up examinations?

**A:** Any patient with glaucoma requires regular monitoring. Glaucoma surgery may not adequately control IOP for a person's lifetime. Periodic follow-up is required for detection of both progression and long-term complications of surgery.

**Q:** Should first-degree relatives of patients with POAG and PACG be advised to undergo an eye examination? If the examination is normal, how often should follow-up be advised?

**A:** A family history of glaucoma is an important predictive risk factor. Depending on the clinical findings, the frequency of regular follow-up ranges from annually to every 2 to 5 years after the age of 35 years.

**Q:** What is the best method to confirm glaucoma progression? What is the role of imaging technologies to detect progression?

**A:** Optic disc photography probably detects progression at the earliest stage. Available statistical programmes such as the GPA and newer metrics (Visual Field Index) for the Humphrey field analyser help detect VF progression. New programmes are also being developed. In early glaucoma (or preperimetric glaucoma), imaging technologies can be helpful to detect progression. Initiation or change of treatment should be made in combination with the clinical picture. Imaging technologies should not be used in isolation, but as part of the assessment.

**Q:** If a patient always has an IOP of 12 to 14 mm Hg, but the VF shows typical progression confirmed by repeat perimetry, should the patient be investigated for systemic diseases?

**A:** IOP control should be assessed at as many different times as possible — are IOPs fluctuating widely at different times of the day? Other risk factors such as nocturnal hypotension, systemic or topical steroid use, recent major surgery, or haemodynamic crises and an abnormally thin central cornea should be assessed. The patient's family physician should be liaised with regarding systemic hypertension and its control (avoid night-time dips), and to exclude sleep apnoea. The patient should be asked about practicing yoga, especially 'asanas' in the inverted position, use of wind instruments, and rapid consumption of large quantities of water or beer. An 'alternative medicine' programme of a litre of water in the morning mimics a water drinking provocative test and may contribute to optic nerve damage.

**Q:** Are at least 5 to 6 VFs needed to confirm progression?

**A:** While this is true in a research setting, clinically, other information such as achieving target IOP, peak IOP with treatment, IOP fluctuation, and changes in the optic disc are used. With 'corroborative' clinical signs, a single repeat VF may confirm progression.

**Q:** What is the role of patient education in the treatment of glaucoma?

**A:** Patient education is most important for glaucoma management. The patient should be an informed participant in the treatment programme. The clinician should explain the disease and its severity, present realistic expectations of treatment outcomes, possible effects of both medical and surgical treatment, and note the patient's preferences. This is an ongoing process; the patient should be kept informed about the course of the disease throughout follow-up.

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## SECTION 3.2 SCREENING

**Q:** There is a lot of undiagnosed glaucoma in the region around the clinic, therefore screening the local population may be beneficial. Is there a single screening test that can be used to diagnose glaucoma?

**A:** There is no single screening test to diagnose glaucoma, which is one of the reasons why 'screening' for glaucoma is not really possible. It is often difficult to be sure about the diagnosis, and the reliability of each test is variable.

**Q:** Who should undergo gonioscopy in a general ophthalmology clinic?

**A:** Ideally, every person older than 40 years who reports for eye examination, especially everyone with glaucoma, family history of glaucoma, eye trauma, uveitis, and/or diabetes.

**Q:** Should LPI be advised for all patients with a van Herick test less than one-quarter the peripheral corneal thickness?

**A:** The van Herick test is only a screening test to detect a narrow angle. The decision to perform LPI is based on gonioscopic and anterior segment imaging findings.

## APPENDIX 1. TREATMENT OF CHILDHOOD GLAUCOMA

- A child with glaucoma should be referred to a specialised centre

### ANTIGLAUCOMA THERAPY

Childhood glaucoma (developmental glaucoma and various secondary glaucomas) usually needs surgical therapy. Safety of antiglaucoma medications in young children has not been established. Systemic side effects may be more common due to small distribution volume and reduced metabolism.

#### $\beta$ -Blockers

Must be used at lower concentrations and with caution. Severe cardiorespiratory side effects, including apnoea attack, are possible.<sup>1</sup>

#### Prostaglandin Analogues

Little IOP effect has been reported in children, but they may be effective in older children or those with juvenile OAG.<sup>2</sup> Side effects in children are uncommon (sleep disturbance, sweating, ocular hyperaemia, irritation, increased iris pigmentation, eyelash growth).

#### $\alpha_2$ -Agonists

Avoid in neonates, infants, and children younger than 7 years. Apnoea and cyanosis, hypothermia, and hypotony related to CNS depression (from blood-brain barrier immaturity) have been reported.<sup>3-5</sup>

#### Topical Steroids

Must be used at lower concentrations and with caution. Ocular hypertensive response is common.<sup>6</sup>

### References

1. Williams T, Ginther WH. Hazard of ophthalmic timolol. *N Engl J Med.* 1982;306:1485-6.
2. Enyedi LB, Freedman SF. Latanoprost for the treatment of pediatric glaucoma. *Surv Ophthalmol.* 2002;47(Suppl 1):S129-32.
3. Bowman RJ, Cope J, Nischal KK. Ocular and systemic side effects of brimonidine 0.2% eye drops (Alphagan) in children. *Eye.* 2004;18:24-6.
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5. Carlsen JO, Zabriskie NA, Kwon YH, Barbe ME, Scott WE. Apparent central nervous system depression in infants after the use of topical brimonidine. *Am J Ophthalmol.* 1999;128:255-6.
6. Ng JS, Fan DS, Young AL, et al. Ocular hypertensive response to topical dexamethasone in children: a dose-dependent phenomenon. *Ophthalmology.* 2000;107:2097-100.

## APPENDIX 2. TREATMENT IN PREGNANCY AND LACTATION

### PREGNANCY AND INTRAOCULAR PRESSURE

Pregnancy often alters IOP, which tends to be lower in mid to late term,<sup>1</sup> possibly from hormonal changes or decreased episcleral venous pressure.

### ANTIGLAUCOMA MEDICATIONS AND PREGNANCY

The FDA has classified drugs for use in pregnancy as follows:

- Class A: safety established using human studies
- Class B: presumed safety based on animal studies
- Class C: uncertain safety; no human studies and animal studies show an adverse effect
- Class D: unsafe; evidence of risk that, in certain clinical circumstances, may be justifiable
- Class X: highly unsafe — risk of use outweighs any possible benefit

Most ophthalmic medications ( $\beta$ -blockers, topical and systemic CAIs, PGAs, cholinergic agents, anticholinesterases, and apraclonidine hydrochloride) are FDA pregnancy class C. Brimonidine and dipivefrin are pregnancy class B. Carefully balance the patient's risk of functional visual loss with the potential risk to the foetus or neonate.<sup>2</sup>

No antiglaucoma medication has been proved completely safe in pregnant patients with glaucoma or OH. When circumstances permit, discontinuation of the drug(s) or reduction of the dose is recommended.

For all medications, decrease systemic absorption by teaching the double DOT technique — 'don't open the eyelid' and 'digital occlusion of the tear duct' after instilling any eye drops.

Closely collaborate with the obstetrician.

Be proactive — discuss with female patients of childbearing age the available options for glaucoma management before pregnancy. Laser or surgical treatments may be offered in advance, to decrease or stop medication use.<sup>3-5</sup>

#### Non-selective $\alpha_1$ -, $\alpha_2$ -, and $\beta$ -Agonists, $\beta$ -Antagonists, and Pilocarpine

May be used with caution.  $\beta$ -Receptors have been found in human placental tissue and placental transfer of  $\beta$ -antagonists have been reported.<sup>6</sup> Monitor the foetus regularly for arrhythmia and bradycardia for patients using  $\beta$ -antagonists.<sup>7,8</sup>

#### Prostaglandin Analogues

Should be avoided in pregnancy. Prostaglandin  $F_{2\alpha}$  can cause uterine contractions and influence foetal circulation.<sup>9,10</sup>

#### Systemic or Topical Carbonic Anhydrase Inhibitors

Should be avoided in pregnancy. CAI teratogenicity has been reported in animals.<sup>11</sup>

#### Antiglaucoma Medications and Lactation

##### $\beta$ -Blockers

Are used systemically during pregnancy for blood pressure control. Use with care during lactation.  $\beta$ -Blockers are excreted in breast milk at concentrations several times higher than those in plasma, potentially resulting in systemic cardiorespiratory side effects in infants.<sup>12,13</sup> Timolol and dorzolamide are approved by the American Academy of Pediatrics for use during lactation and should be used with punctal occlusion.<sup>14</sup>

After topical instillation of an antiglaucoma medication, the drug level in the plasma usually reaches a pharmacologically active level.

**FOR FURTHER  
INFORMATION**

See the Organization of Teratology Information Specialists website at:  
[www.otispregnancy.org/](http://www.otispregnancy.org/)  
or the European Network of Teratology Information Service website at:  
[www.entis-org.com/?section=home&lang=UK](http://www.entis-org.com/?section=home&lang=UK)

## References

1. Sunness JS. The pregnant woman's eye. *Surv Ophthalmol.* 1988;32:219-28.
2. Kooner KS, Zimmerman TJ. Antiglaucoma therapy during pregnancy — part I. *Ann Ophthalmol.* 1988;20:166-9.
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4. Brauner SC, Chen TC, Hutchinson BT, Chang MA, Pasquale LR, Grosskreutz CL. The course of glaucoma during pregnancy: a retrospective case series. *Arch Ophthalmol.* 2006;124:1089-94.
5. Johnson SM, Martinez M, Freedman S. Management of glaucoma in pregnancy and lactation. *Surv Ophthalmol.* 2001;45:449-54.
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7. Berlin RL, Lee UT, Samples JR, et al. Ophthalmic drops causing coma in an infant. *J Pediatr.* 2001;138:441-3.
8. Wagenvoort AM, van Vugt JM, Sobotka M, van Geijn HP. Topical timolol therapy in pregnancy: is it safe for the fetus? *Teratology.* 1998;58:258-62.
9. Karim SM. Physiological role of prostaglandins in the control of parturition and menstruation. *J Reprod Fertil Suppl.* 1972;16(Suppl 16):105-1.
10. Sideris EB, Yokochi K, Coceani F, Olley PM. Prostaglandins and fetal cardiac output distribution in the lamb. *Am J Physiol.* 1985;248(6 Pt 2):H853-8.
11. Scott WJ, Hirsch KS, DeSesso JM, Wilson JG. Comparative studies on acetazolamide teratogenesis in pregnant rats, rabbits, and rhesus monkeys. *Teratology.* 1981;24:37-42.
12. Williams T, Ginther WH. Hazard of ophthalmic timolol. *N Engl J Med.* 1982;306:1485-6.
13. Fidler J, Smith V, De Swiet M. Excretion of oxprenolol and timolol in breast milk. *Br J Obstet Gynaecol.* 1983;90:961-5.
14. American Academy of Pediatrics Committee on Drugs: the transfer of drugs and other chemicals into human milk. *Pediatrics.* 1994;93:137-50.

## APPENDIX 3. SYSTEMIC MEDICATIONS THAT MAY INDUCE ANGLE CLOSURE

| Preparations by class   | Probable mechanism  | Management   |
|---|---|--|
| <b>Sulphur-based agents</b>   |   |  |
| <i>Anticonvulsants</i><br>Topiramate  | Ciliary body oedema with anterior rotation of the lens-iris diaphragm | Cessation of therapy, cycloplegia, and corticosteroid therapy<br>Laser iridotomy is ineffective    |
| <i>CAIs</i><br>Acetazolamide  |   |  |
| <i>Thiazide diuretics</i><br>Hydrochlorothiazide  |   |  |
| <i>Sulphonamides</i><br>Cotrimoxazole   |   |  |
|   |   |  |
| <b>Agents producing pharmacological mydriasis</b>   |   |  |
| <i>Adrenergic agents</i><br>Topical agents (phenylephrine)<br>Nasal sprays (ephedrine)<br>Inhaled nebulised solutions (salbutamol, albuterol, terbutaline)  | Induced pupillary mydriasis<br>Relative papillary block               | Cessation of therapy and/or laser iridotomy  |
| <i>Anticholinergic agents</i><br>Tropicamide<br>Atropine<br>Homatropine<br>Cyclopentolate<br>Ipratropium bromide<br>Antidepressants/antianxiety agents (tri- and tetracyclic antidepressants, paroxetine, venlafaxine, fluvoxamine) |   |  |
| <i>Histamine receptor antagonists</i>   |   |  |
| <b>Agents associated with haemorrhagic or serous choroidal effusion</b>   |   |  |
| <i>Anticoagulants</i><br>Warfarin   | Anterior rotation of the lens-iris diaphragm                          | Cessation of therapy, cycloplegia, and corticosteroid therapy                                      |
| <i>ACE inhibitors</i><br>Candesartan  |   |  |
| <b>Agents associated with ciliary block glaucoma</b>  |   |  |
| <i>Cholinergic agents</i><br>Pilocarpine<br>Acetylcholine<br>Carbachol  | Ciliary block   | Cycloplegia, aqueous suppressants, and/or laser hyaloidotomy, pars plana vitrectomy, or lensectomy |

## APPENDIX 4. HOW TO TEST CALIBRATION OF A GOLDMANN TONOMETER

- Standard method for measuring IOP<sup>1</sup>
- Periodic calibration check recommended: at least twice yearly

1. Set the tonometer in position on the slit-lamp stand, with the perspex biprism head in place and the tension on the circular dial on the right side (from the examiner's side of the slit lamp) set at 5 mm Hg. 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 to 2 mm Hg below zero (Figure 1)
3. Slowly twirl the dial clockwise until the head rocks forwards again. The tension should read 0 to 2 mm Hg (Figure 2).
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 centre one (on the other side of the centre from you) is aligned as exactly as you can (Figure 3).
5. Slip the rod and its holder into the receptacle on the right side of the tonometer. The head will rock backwards 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 to 23 mm Hg.
7. Slowly twirl the circular dial counter-clockwise until the head rocks backwards. The tension on the dial should read 17 to 20 mm Hg.
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 4).
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 forwards. The tension should read 60 to 64 mm Hg.
11. Slowly twirl the dial counter-clockwise until the head rocks backwards — the tension should read 56 to 60 mm Hg.

- The 3 threshold tension levels being used to test the tonometer's calibration are at 0, 20, and 60 mm Hg
- 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

### Reference

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Figures 1, 2, 3, and 4 reproduced courtesy of Haag-Streit AG and Mandarin Opto-Medic Co Pte Ltd.

Figure 1.

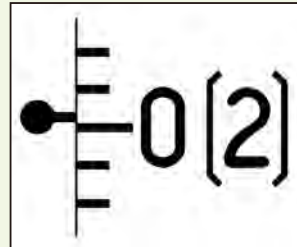


Figure 2.

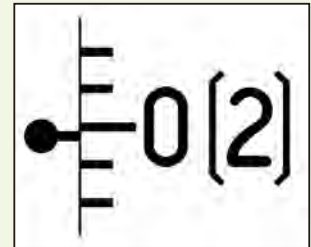


Figure 3.

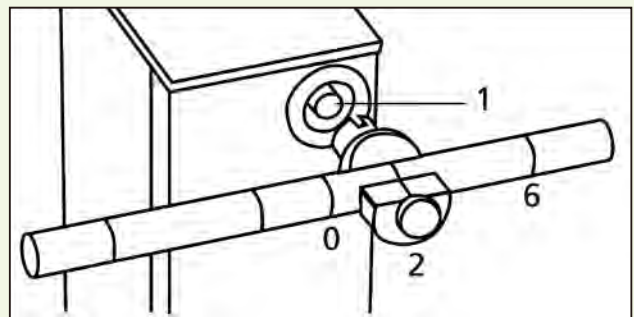
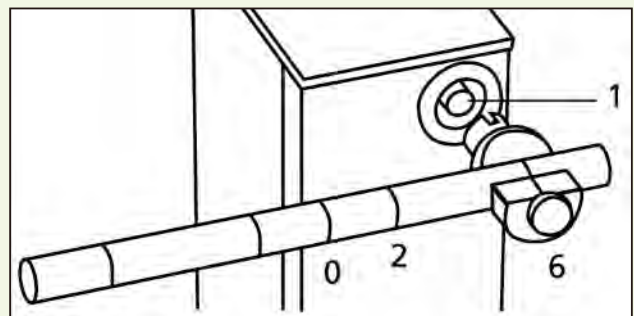


Figure 4.

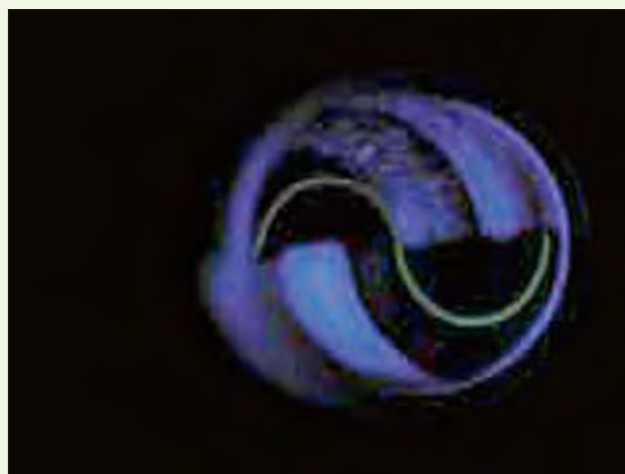


## APPENDIX 5. TONOMOMETRY MIRES

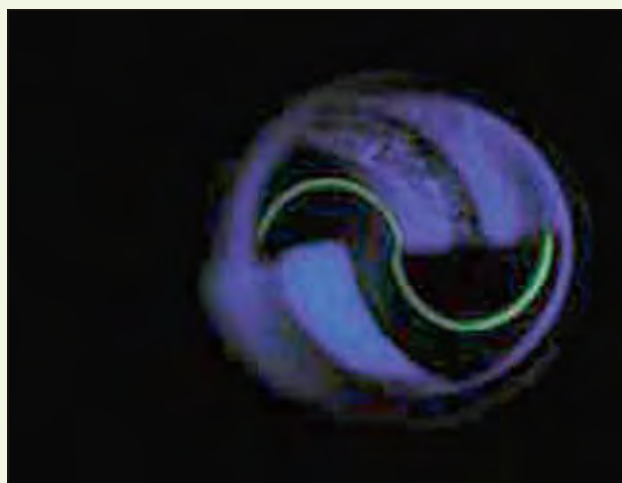
Excess corneal applanation (IOP lower than tonometer reading)



Insufficient corneal applanation (IOP higher than tonometer reading)



Correct endpoint corneal applanation (IOP equals tonometer reading)



*Photographs reproduced courtesy of Ivan Goldberg, Australia.*



## APPENDIX 6B. GONIOGRAM/GONIOSCOPIC CHART

Grading system for gonioscopic findings (without indentation):<sup>1,2</sup>

### 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 <1/4 corneal thickness (occludable) |
| Grade II  | >1/4 but <1/2 of corneal thickness  |
| Grade III | ≥1/2 of corneal thickness (non-occludable)  |

### Schafer method

| Grade            | 0                              | I                          | II                     | III                     | IV                      |
|------------------|--------------------------------|----------------------------|------------------------|-------------------------|-------------------------|
| Method           |                                |                            |                        |                         |                         |
| 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 |

### Spaeth method

#### 1. Iris insertion

- Anterior to Schwalbe's line or TM
- Behind Schwalbe's lines
- Centred 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

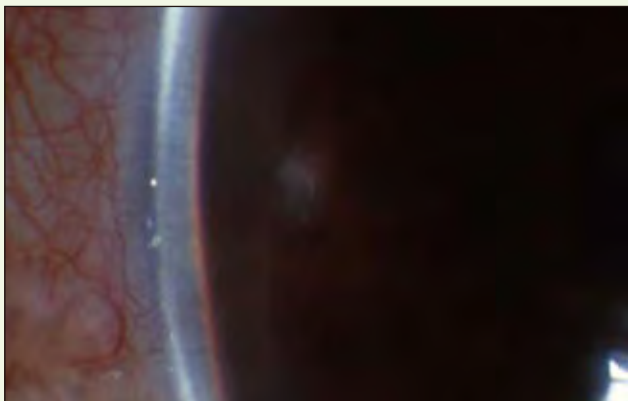
- 0 (none) to 4 (maximal)

### References

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2. Spaeth GL. The normal development of the human anterior chamber angle: a new system of descriptive grading. Trans Ophthalmol Soc UK. 1971;91:709-39.

## APPENDIX 6C. VAN HERICK GRADING

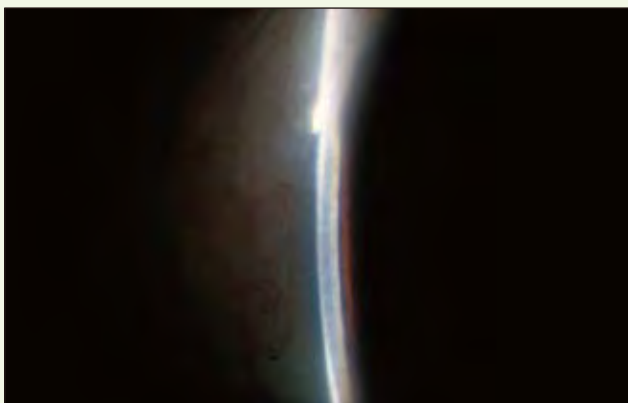
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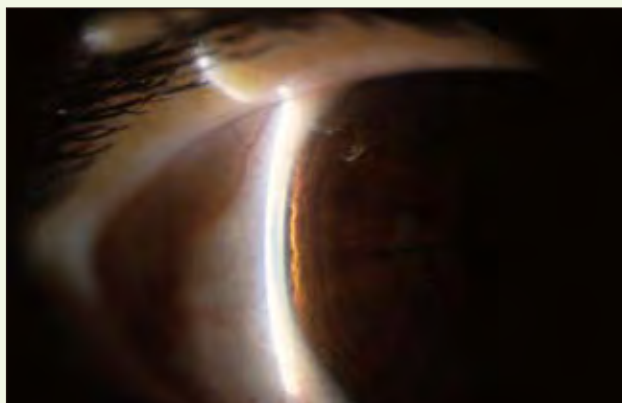
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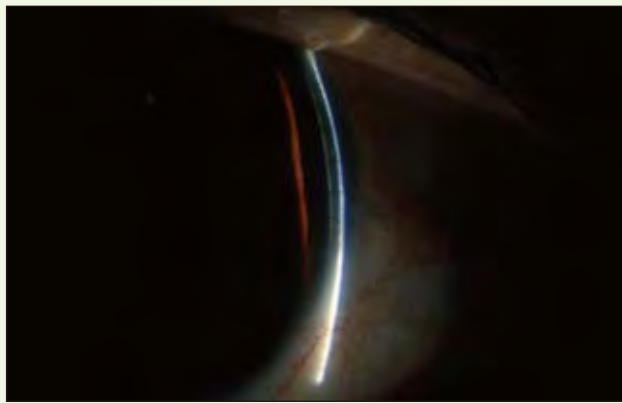
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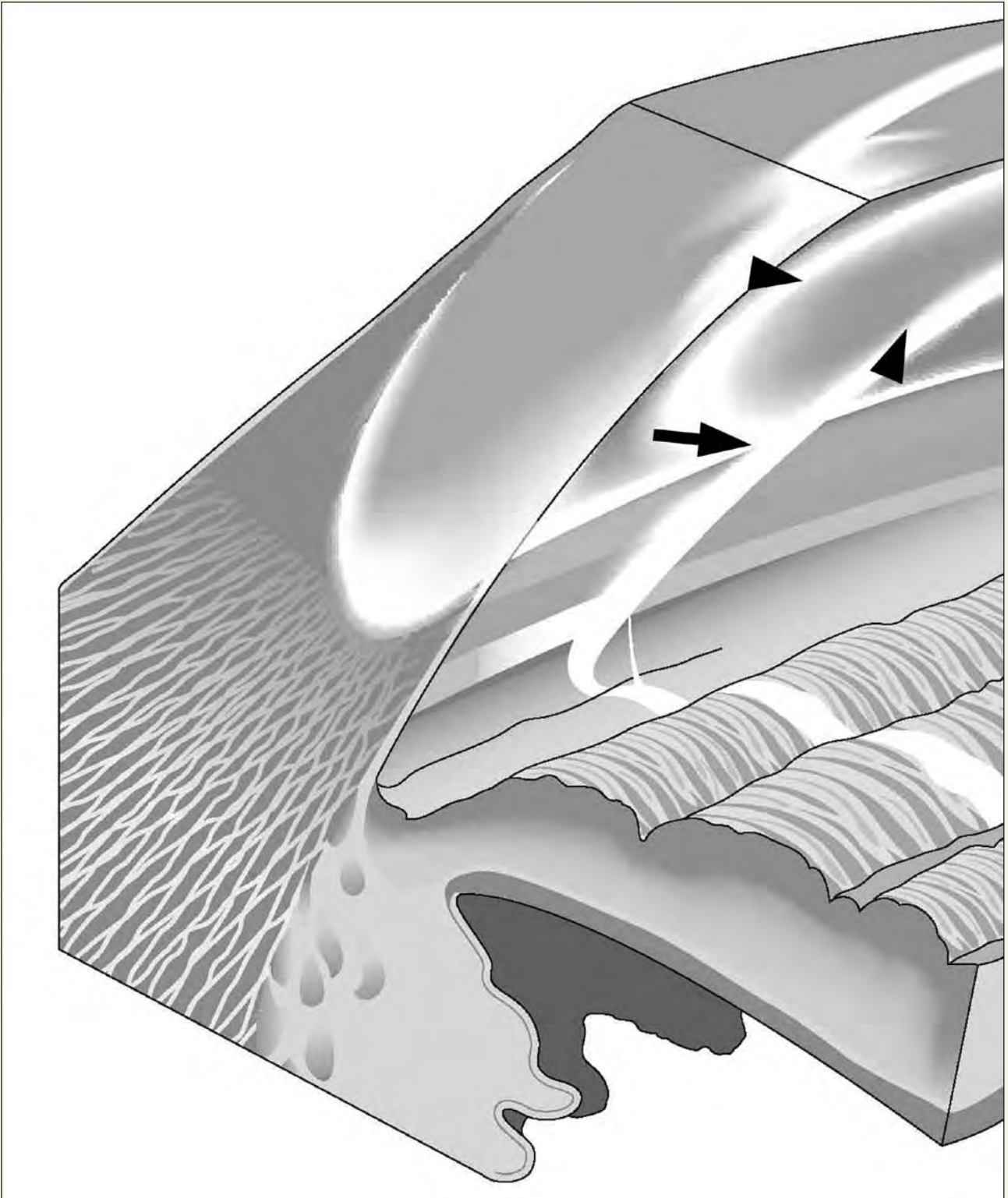
75%



100%



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**APPENDIX 6D. CORNEAL WEDGE DIAGRAM**

- A gonioscopic view of the drainage angle at high magnification (x 16 or x 25)
- The thin slit beam illuminates the angle region and splits to form the 'corneal wedge' (arrow heads)
- The boundaries of the wedge meet at Schwalbe's line (arrow).

**Reference**

1. Foster PJ, Johnson GJ. Classification and clinical features. In: Hitchings E, editor. Fundamentals of clinical ophthalmology: glaucoma. BMJ Books; London: 2000. p 148.

*Schematic reproduced with permission from BMJ Books; Copyright © 2000.<sup>1</sup>*

## **APPENDIX 7A. HOW TO OPTIMISE 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: SITA (Humphrey field analyser) — 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 the holding band easily
- Cover other eye fully — some patients prefer it open, some prefer 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 programme.

### **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

### **5. Maintain quiet environment to support concentration**

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## **APPENDIX 7B. COMMON ARTIFACTS FOR VISUAL FIELD MEASUREMENTS**

Common artifacts may be caused by:

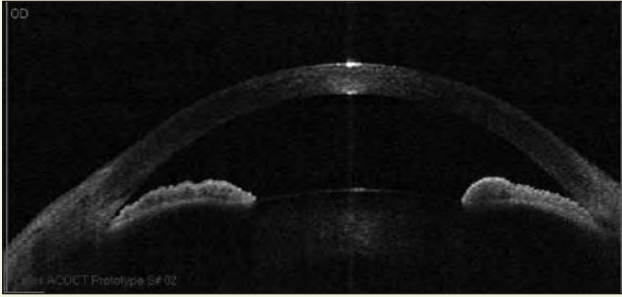
- Refractive error, especially hyperopia, may influence the central field and threshold sensitivities
  - in patients with high myopia ‘refraction scotoma’ may be confused with glaucomatous changes
- Cataract and other opacities in intermediate ocular media (corneal opacity, after cataract, vitreous haemorrhage/opacity) can influence VF
- Small pupil may exaggerate VF abnormalities, especially in eyes with cataract
  - the pupil size at the time of examination should be recorded
- Patient’s experience with and concentration for the test can influence the perimetric results
- Lens rim artifact
- Drooping eyelids, deep-set eyes, prominent brows or nose

## APPENDIX 8. SECONDARY GLAUCOMAS — PRINCIPLES OF MANAGEMENT

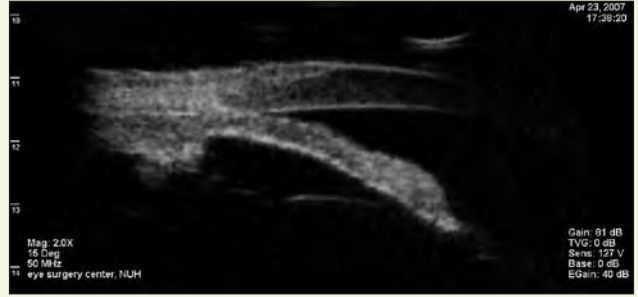
| Strategy  | An example of the approach to management — uveitic glaucoma |
|---|---|
| Diagnose the underlying cause(s)  | Diagnose uveitis and its cause(s)                           |
| Treat the underlying cause(s)   | Anti-inflammatory agents                                    |
| Identify the mechanism(s)   | Posterior synechiae with pupil block                        |
| Treat the mechanism(s) — they may change over the course of the disease | LPI   |
| Medical therapy<br>First-line agents are aqueous inflow inhibitors      | $\beta$ -Blockers, $\alpha_2$ -agonists, CAls               |

## APPENDIX 9. ANGLE CLOSURE MECHANISMS

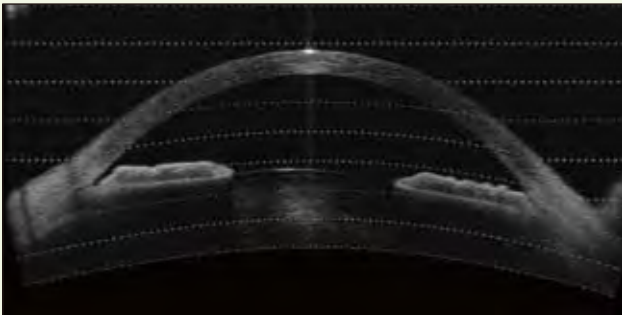
Pupil block (OCT)



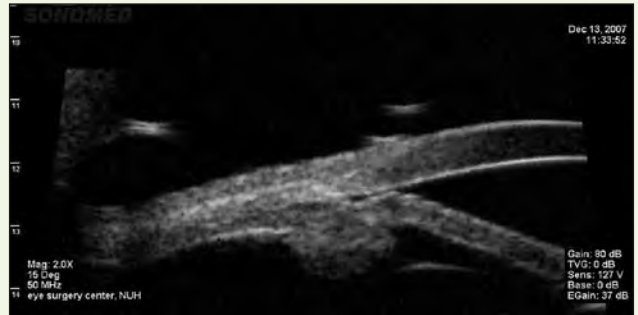
Pupil block RLE (UBM)



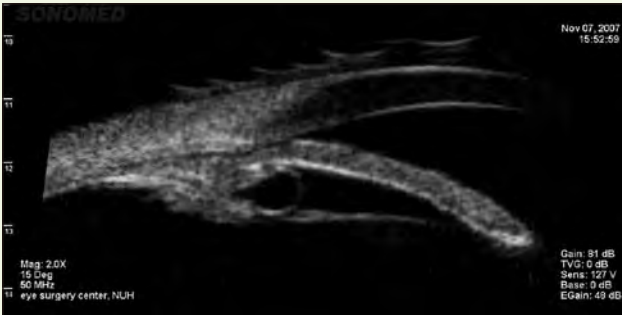
Plateau iris (OCT)



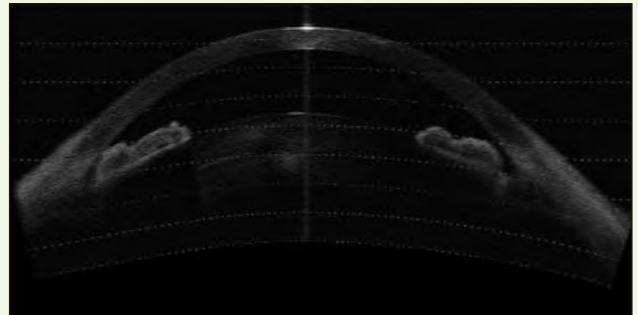
Plateau iris (UBM)



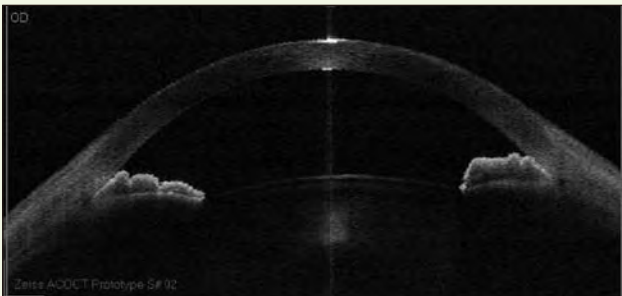
Pseudoplateau iris — ciliary body cyst (UBM)



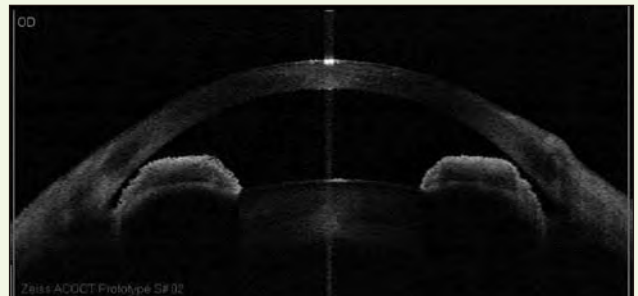
Lens — thick forward position (OCT)



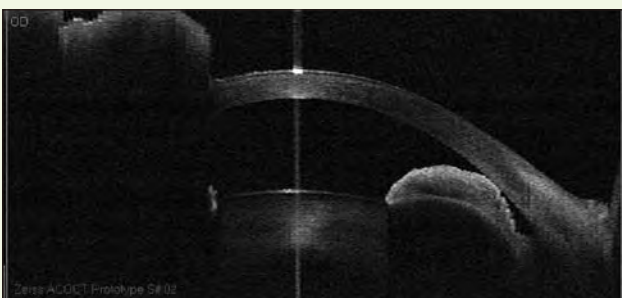
Angle crowding — peripheral iris roll in dark (OCT)



Very steep nasal temporal angle in dark (OCT)



Creeping very steep inferior angle in dark (OCT)



Photographs reproduced courtesy of Paul Chew, Singapore.

## APPENDIX 10. SIDE EFFECTS OF GLAUCOMA MEDICATIONS

| Preparations by class  | Contraindications*   | Drug interactions   | Local side effects  | Systemic side effects  |
|--|--|---|---|--|
| <b><math>\alpha_2</math>-Adrenergic agonists</b><br><i>Selective <math>\alpha_2</math>-adrenergic agonists</i><br>Brimonidine 0.2%, 0.15%, 0.1%<br>Apraclonidine 1%, 0.5%<br><i>Non-selective <math>\alpha_2</math>-adrenergic agonists</i><br>Epinephrine 2%<br>Dipivefrin 0.1% | MAOI therapy<br>Age younger than 2 years<br>Caution is recommended for children younger than 7 years   | CNS depressants:<br>Alcohol<br>Barbiturates<br>Opiates<br>Sedatives<br>Anaesthetics<br>Tricyclic antidepressants          | Ocular allergy<br>Burning<br>Stinging<br>Blurring<br>Foreign-body sensation<br>Itching<br>Hyperaemia<br>Follicular conjunctivitis   | CNS depression<br>Oral dryness<br>Headache<br>Fatigue<br>Drowsiness<br>Bradycardia<br>Hypotension<br>Hypothermia<br>Apnoea   |
| <b><math>\beta</math>-Blockers</b><br><i>Non-selective agents</i><br>Timolol 0.25%, 0.5%, 0.1%<br>Laevobunolol 0.25%, 0.5%<br>Carteolol 1%<br>Metipranolol 0.3%<br><br><i>Selective agents</i><br>Betaxolol 0.25%, 0.5%  | Absolutely contraindicated in:<br>Bronchial asthma<br>COPD<br>Bradycardia<br>Heart block<br>To be used cautiously in cardiac failure<br>Punctate epithelial keratopathy<br><br>Relatively contraindicated in:<br>Bronchial asthma<br>COPD<br>Bradycardia<br>Heart block<br>Cardiac failure | Systemic $\beta$ -blockers<br>CCBs<br><br>As for non-selective $\beta$ -blockers with wider safety margin                 | Burning<br>Stinging<br>Photophobia<br>Itching<br>Tearing<br>Decreased corneal sensitivity<br>Hyperaemia<br><br>As for non-selective $\beta$ -blockers, with wider safety margin | Bronchospasm<br>Hypotension<br>Bradycardia<br>Heart block<br>Mask hypoglycaemia<br>Adversely affects lipid profile (except carteolol)<br>Loss of libido<br>Fatigue<br>Aggravation of myasthenia gravis<br>Depression<br>Memory impairment<br>Reduced exercise tolerance<br>Increased falls<br>Hair loss<br><br>As for non-selective $\beta$ -blockers with wider safety margin |
| <b>CAIs</b><br><i>Topical</i><br>Dorzolamide 2%<br>Brinzolamide 1%<br><br><i>Systemic</i><br>Acetazolamide 125 mg, 250 mg<br>Methazolamide 25 mg, 50 mg<br>Dichlorphenamide 50 mg  | Relatively contraindicated in compromised corneal endothelium and sulfonamide allergy<br><br>Sulfonamide allergy<br>Renal stones/failure<br>Respiratory/metabolic acidosis<br>Hypokalaemia   | None reported, but potential exists for similar interactions as for systemic CAIs<br><br>Steroids<br>Diuretics<br>Digoxin | Burning<br>Stinging<br>Itching<br>Punctate epithelial keratopathy<br>Blepharoconjunctivitis<br>Corneal endothelial cell decompensation<br>Transient myopia                      | Bitter taste<br><br>Fatigue/lethargy<br>Anorexia/weight loss<br>GI upset<br>Paraesthesia<br>Taste disturbance<br>Stevens-Johnson syndrome<br>Blood dyscrasias<br>Renal stones/failure<br>Hypokalaemia<br>Acute leukopenia<br>Agranulocytosis<br>Aplastic anaemia<br>Haemolytic anaemia<br>Neutropenia<br>Pancytopenia<br>Thrombocytopenia                                      |

Continued on page 88

| Preparations by class  | Contraindications*  | Drug interactions  | Local side effects   | Systemic side effects  |
|--|---|--|--|--|
| <b><i>Cholinergics</i></b><br>Pilocarpine 1%, 2%, 3%, 4%, 6%<br>Carbachol 1.5%, 3%<br>Phospholine iodide 0.03%, 0.06%, 0.125%, 0.25%<br>Acetylcholine chloride 20 mg/2 mL  | Uveitic, neovascular, and lens-induced glaucomas<br>Post-drainage surgery<br>Aqueous misdirection syndrome<br>Phospholine iodide in phakic patients   | Many for phospholine iodide, see: <a href="http://www.drugs.com/drug-interactions/phospholine-iodide_d01195.html">www.drugs.com/drug-interactions/phospholine-iodide_d01195.html</a> or consult full prescribing information | Cataract<br>Pain<br>Dimness of vision<br>Blurring<br>Myopic shift<br>Retinal detachment<br>Aggravation of pupillary block  | Headache<br>Salivation<br>Lacrimation<br>Urinary frequency<br>Diarrhoea<br>Abdominal cramps<br>Diaphoresis<br>Headache<br>Tremor<br>Salivation<br>Bronchospasm<br>Pulmonary oedema<br>Hypotension<br>Bradycardia<br>Nausea<br>Vomiting |
| <b><i>Hyperosmotic agents</i></b><br>Mannitol 5%, 10%, 15%, 20%, 25%<br>Glycerol<br>Isosorbide   | Heart failure<br>Pulmonary oedema<br>Renal failure<br>Caution in hypertension   | NA   | NA   | Headaches<br>Unpleasant taste<br>Heart failure<br>Pulmonary oedema<br>Diuresis<br>Death  |
| <b><i>PGAs</i></b><br>Latanoprost 0.005%<br>Travoprost 0.004%<br>Bimatoprost 0.03%<br>Unoprostone 0.15%  | Cataract surgery complicated by posterior capsular rupture and vitreous loss<br>Herpes simplex keratitis (active or quiescent)<br>Relatively contraindicated in:<br>Active inflammatory ocular conditions<br>Cystoid macular oedema | Chronic pilocarpine use may reduce efficacy  | Blurred vision<br>Burning<br>Stinging<br>Conjunctival hyperaemia<br>Foreign-body sensation<br>Itching<br>Increased pigmentation of the iris/periorbital skin<br>Longer, darker, and thicker lashes<br>Punctate epithelial keratopathy<br>Cystoid macular oedema<br>Reactivation of herpetic infection<br>Facial rash | Unlikely, but possible — consult full prescribing information  |
| <b><i>Proprietary fixed combinations†</i></b><br>Combigan (brimonidine/timolol 0.2%/0.5%)<br>Cosopt (dorzolamide/timolol 2%/0.5%)<br>DuoTrav (travoprost/timolol 0.004%/0.5%)<br>Ganfort (bimatoprost/timolol 0.03%/0.5%)<br>Xalacom (latanoprost/timolol 0.005%/0.5%) | As for individual components  | As for individual components   | As for individual components   | As for individual components   |

**Note:** Please refer to manufacturer's summary of product characteristics before prescribing.

\* Known hypersensitivity to any component of the product or pregnancy.

† Differences may exist between fixed combinations and the individual components:

- The incidence of ocular allergy with Combiganbd (26%) is significantly lower than with brimonidine 0.2% tds used as monotherapy (40%)
- There is less conjunctival injection with Xalacom and DuoTrav than with latanoprost and travoprost, respectively

## APPENDIX 11A. LASER TRABECULOPLASTY

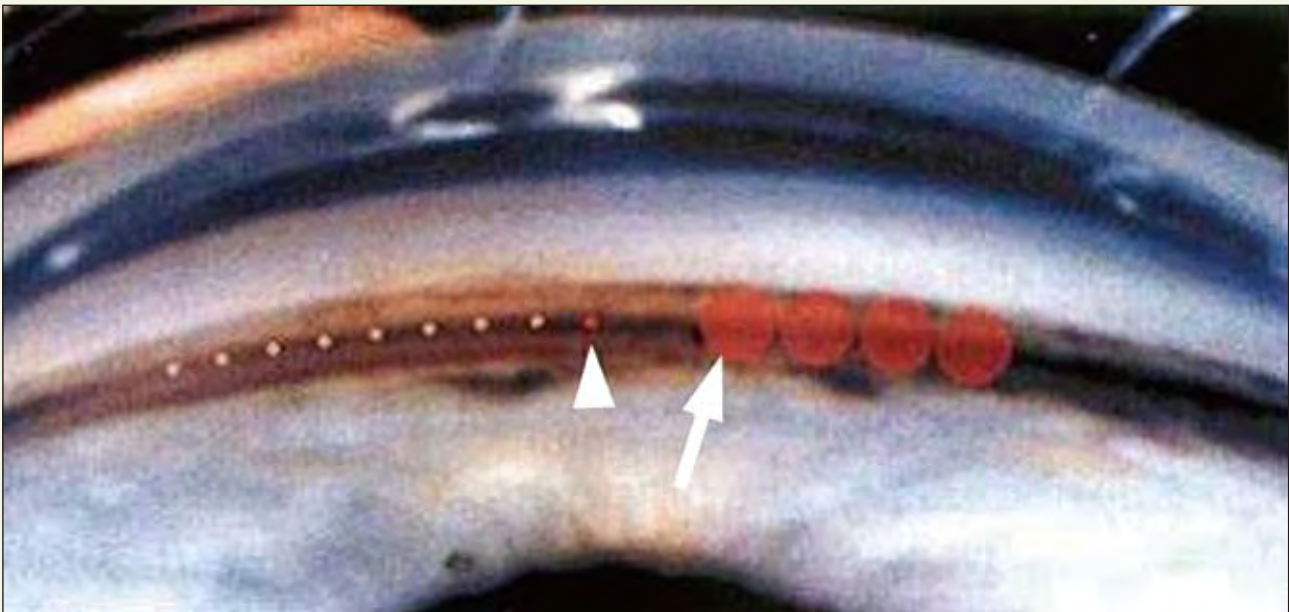
### ARGON LASER TRABECULOPLASTY

- Approximately 100 equally spaced laser spots (diameter 50  $\mu$ ; arrow head) each for 0.1 seconds are applied over 360° of TM, often in 2 sessions of 180°, separated by 1 to 2 weeks
- Ideally, the spots should be applied over Schlemm's canal, avoiding the iris root, at the junction of the anterior one-third and posterior two-thirds of the TM
- The energy level should be set to induce a reaction from a slight transient blanching of the treated area, to small bubble formation

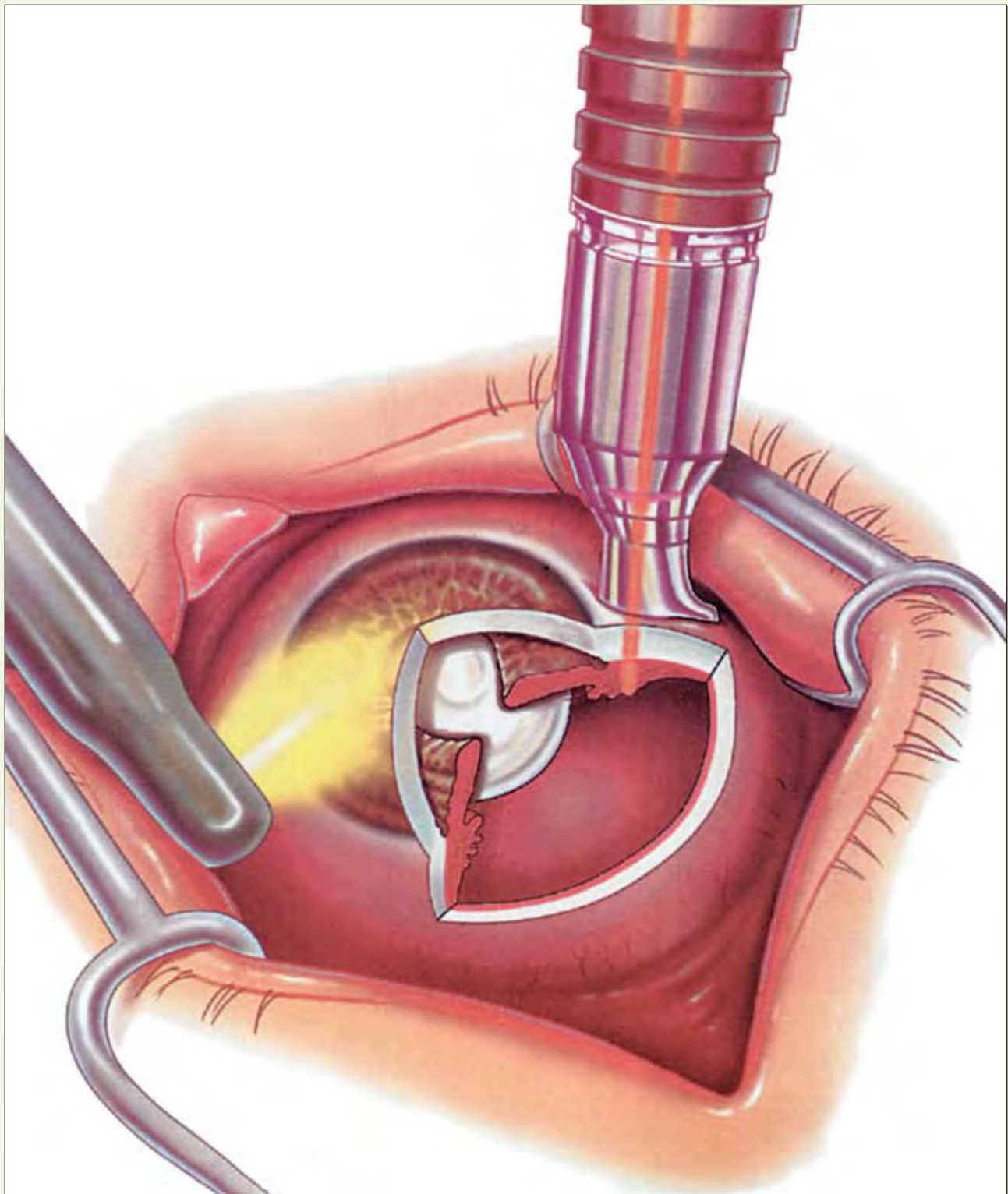
### SELECTIVE LASER TRABECULOPLASTY

- Recently, SLT has been regarded as a safe and effective procedure for patients with POAG, that is equivalent to ALT in terms of IOP reduction
- The settings are: spot size 0.4 mm, duration 0.3 nsec, laser power 0.8 mJ depending upon the tissue reaction
- Laser spots are applied on the TM over 180° in 1 session, with each spot being in contact with the adjacent one (arrow)
- The laser power should be adjusted to induce minimum reaction at the irradiated area
- Bubble formation should be avoided

Argon laser trabeculoplasty and selective laser trabeculoplasty



## APPENDIX 11B. CONTACT TRANS-SCLERAL DIODE LASER

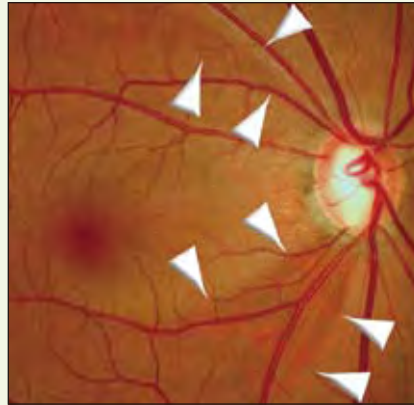
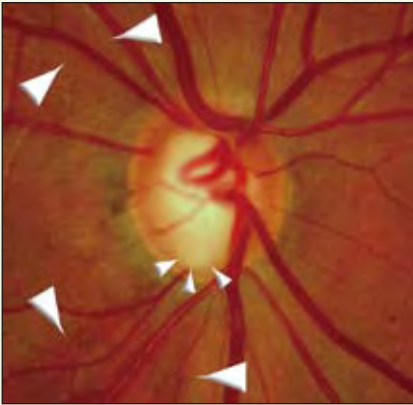


- A cold light source transilluminates the anterior segment, allowing identification of the ciliary body behind the lucent cornea and limbus
- With the G-probe, the fibre-optic laser tip is 1.5 mm behind the anterior edge of the footplate and protrudes 0.7 mm
- The laser tip should be placed over the ciliary body
- Indentation improves energy delivery and blanches the conjunctival blood vessels
- The schematic shows a relatively posterior ciliary body treatment, which may improve pressure reduction

*Schematic Copyright © 2003-2004 SEAGIG.*

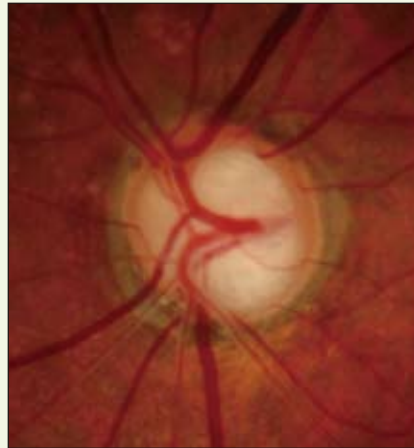
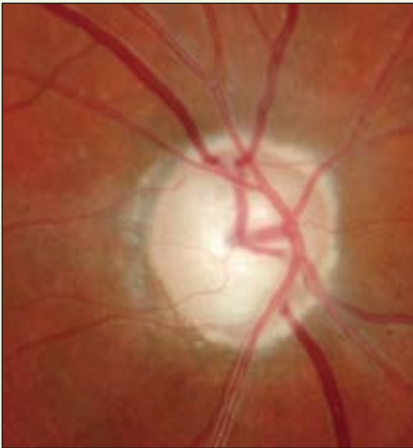
ASIA PACIFIC GLAUCOMA GUIDELINES

## APPENDIX 12. GLAUCOMATOUS OPTIC NEUROPATHY



### MODERATE GLAUCOMATOUS OPTIC NEUROPATHY

- Localised loss of both inferior and superior neuroretinal rim
- A classic inferior notch (small arrow heads)
- Nerve fibre layer defect in both superior and inferior arcuate area (large arrow heads)



### ADVANCED GLAUCOMATOUS OPTIC NEUROPATHY

- Neuroretinal rim thinning
- The cup extends to the disc rim
- Circumlinear blood vessel barring
- Bayoneting of the blood vessels
- PPA



### DISC HAEMORRHAGE

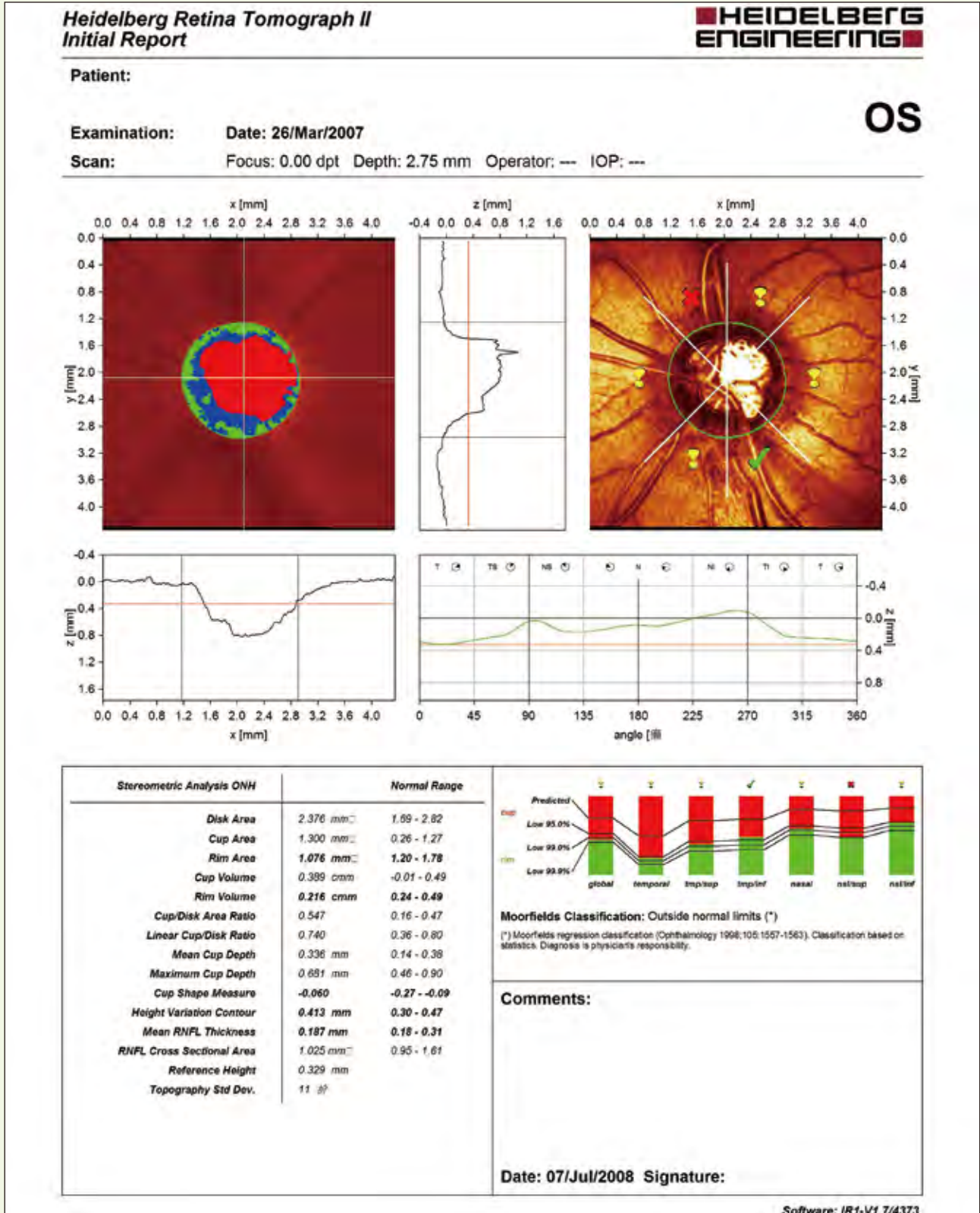
- Splinter, superficial flame-shaped, haemorrhage at disc margin (large arrow head)
- Localised nerve fibre defect at corresponding area (small arrow heads)
- Laminar dots are visible
- A deep notch at the infero-temporal neuroretinal rim with broad nerve fibre defect (dark arrow heads)

Photographs reproduced courtesy of Prin RojanaPongpun, Thailand.

# APPENDIX 13A. IMAGING DEVICES

- Modern technology for optical imaging can enable us to quantitatively and objectively estimate glaucomatous structural changes in the optic nerve head and retina:
  - Heidelberg retina tomography (HRT-II, -III)
  - optical coherence tomography (OCT)
  - scanning laser polarimetry (GDx VCC, GDx ECC)

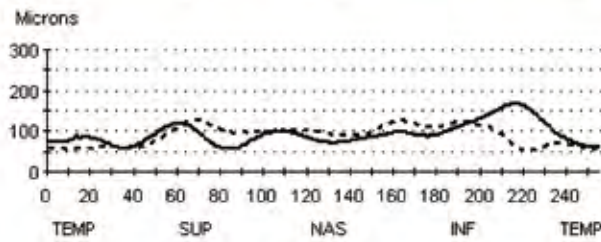
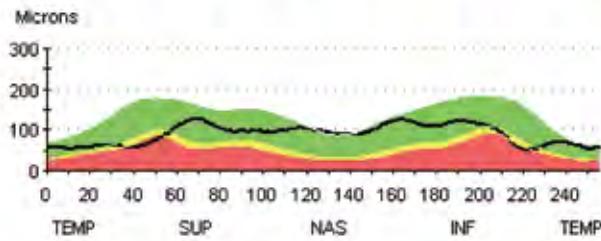
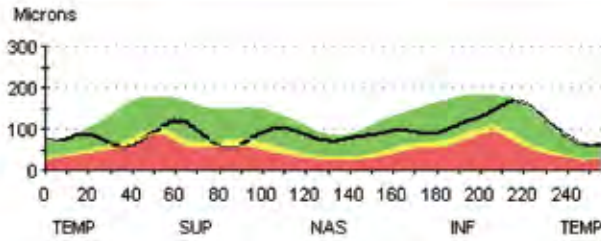
## Heidelberg retina tomography-II



**STRATUS OCT**  
**RNFL Thickness Average Analysis Report - 4.0.7 (0132)**

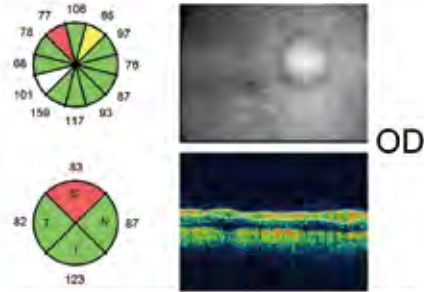
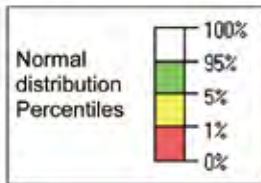


Scan Type: Fast RNFL Thickness (3.4)  
 Scan Date: 3/26/2007  
 Scan Length: 10.87 mm

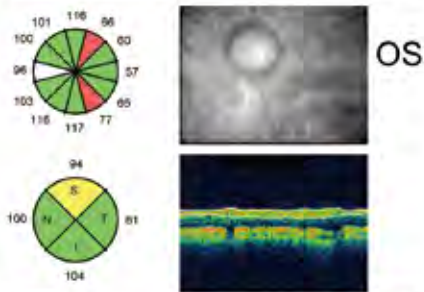


— OD - - - - OS

|           |            |         |
|-----------|------------|---------|
| <b>OD</b> | Scans used | 1, 2, 3 |
| <b>OS</b> | Scans used | 1, 2, 3 |



Signal Strength (Max 10) **6**



Signal Strength (Max 10) **7**

|           | OD (N=3) | OS (N=3) | OD-OS  |
|-----------|----------|----------|--------|
| Imax/Smax | 1.43     | 0.99     | 0.44   |
| Smax/Imax | 0.70     | 1.01     | -0.31  |
| Smax/Tavg | 1.44     | 2.09     | -0.66  |
| Imax/Tavg | 2.05     | 2.06     | -0.01  |
| Smax/Navg | 1.37     | 1.27     | 0.09   |
| Max-Min   | 113.00   | 75.00    | 38.00  |
| Smax      | 118.00   | 127.00   | -9.00  |
| Imax      | 169.00   | 125.00   | 44.00  |
| Savg      | 83.00    | 94.00    | -11.00 |
| Iavg      | 123.00   | 104.00   | 19.00  |
| Avg.Thick | 93.66    | 89.41    | 4.25   |

Signature: \_\_\_\_\_  
 \_\_\_\_\_

Physician: Beijing Tongren Hospital



# Nerve Fiber Analysis

With Variable Corneal Compensation

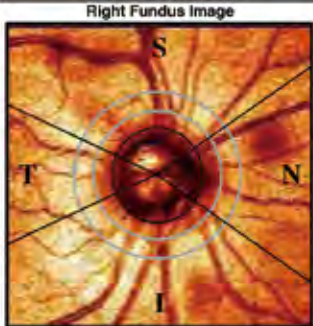
zhudan - handan eye study

ID: A0014

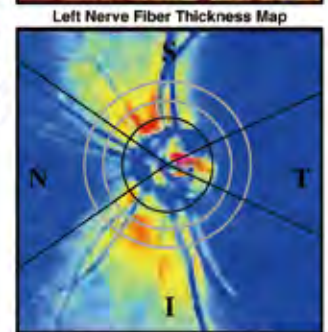
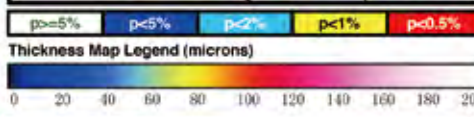
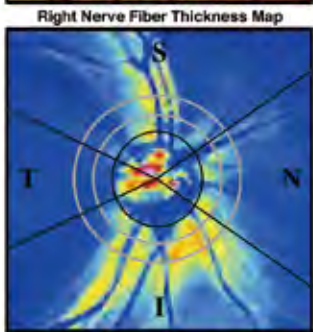
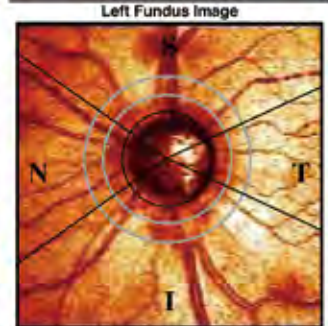
Print Date: 2008-7-7 19:02

**OD Right** Q: 9 Operator: H: 1768 V: 1861 Date: 2007-9-8 09:56

**OS Left** Q: 9 Operator: H: 1768 V: 1861 Date: 2007-9-8 09:56

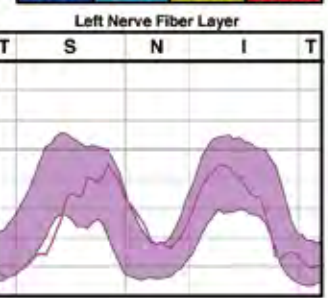
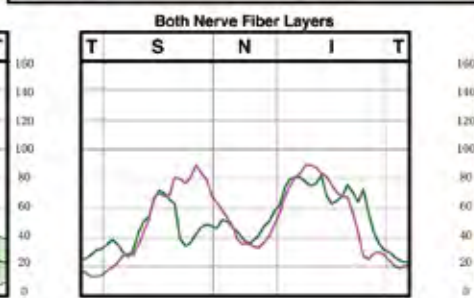
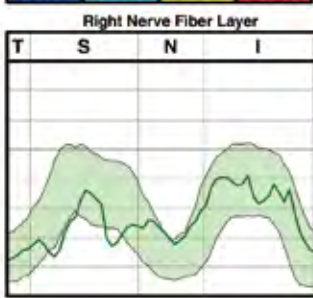
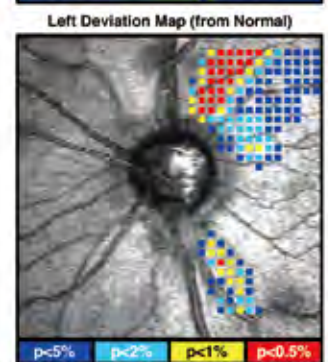


| TSNIT Parameters   | OD Actual Val. | OS Actual Val. |
|--------------------|----------------|----------------|
| TSNIT Average      | 51.5           | 51.6           |
| Superior Average   | 49.6           | 59.5           |
| Inferior Average   | 66.4           | 61.7           |
| TSNIT Std. Dev.    | 17.8           | 24.9           |
| Inter-Eye Symmetry | 0.72           |                |
| NFI                | 26             | 23             |



Impression / Plan:

Signature: \_\_\_\_\_ Date: \_\_\_\_\_



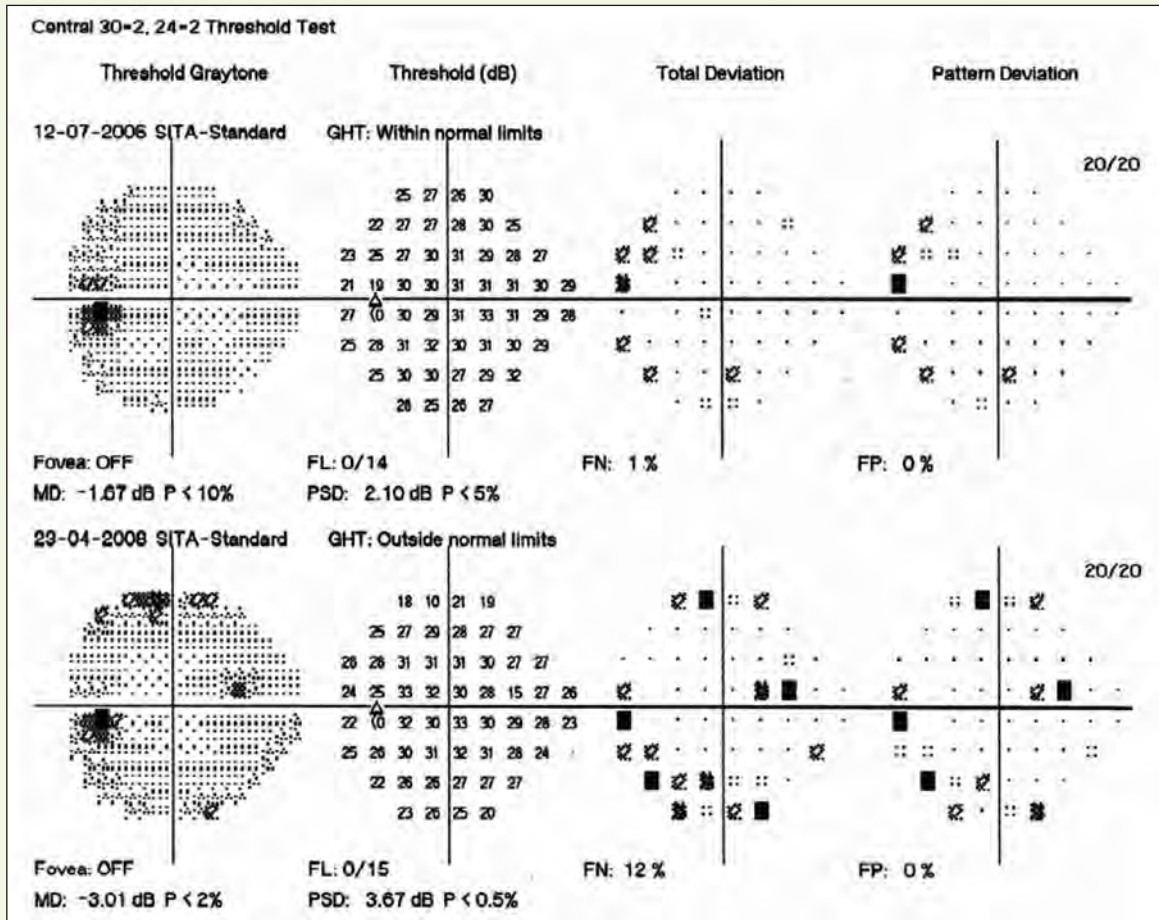
©1992-2004 Laser Spectro-Technologies, Inc., All Rights Reserved. QW 402 5.1.1-E, Spinal D3.0000000000000000, 508 Version, 1.00.00

19801 Townsend Road, San Diego, CA 92121 (858) 473-7000 Ext: 1880 473-7000 www.laser-spectro.com

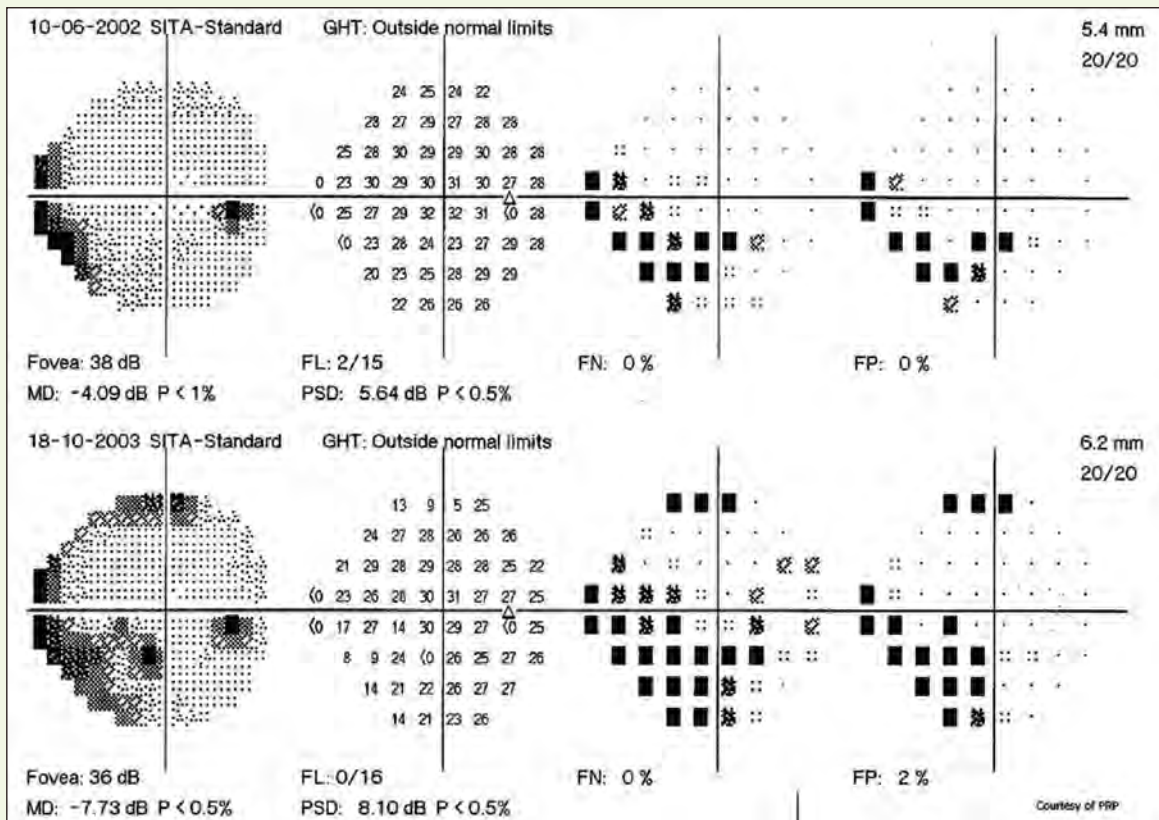
Photographs reproduced courtesy of Yuanbo Liang, China.

# APPENDIX 13B. FIELD PROGRESSION

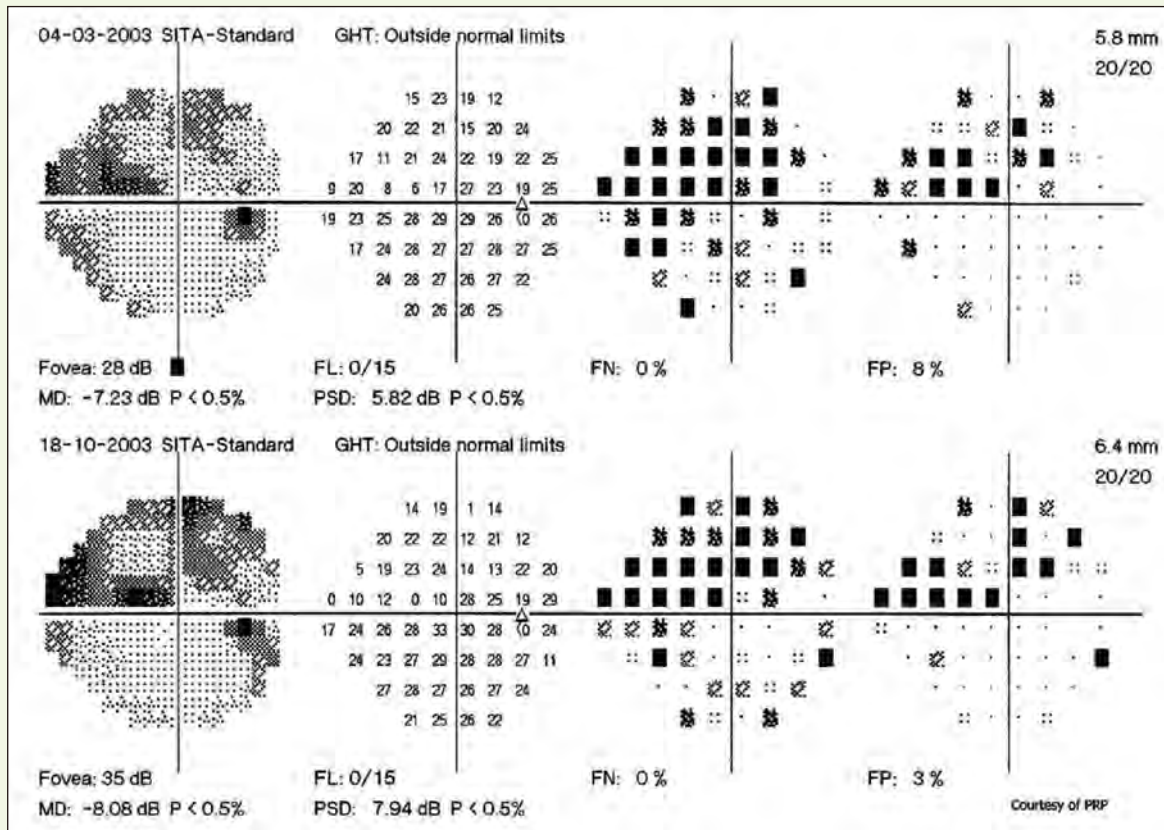
## New scotoma



## Deepening scotoma



## Deepening and enlarging scotoma



- Modern technology for VF measurements enables us to detect early glaucomatous changes in VF, and to evaluate structure-function relationships:
  - Matrix frequency-doubling technology
  - short-wavelength automated perimetry

*Photographs reproduced courtesy of Prin RojanaPongpun, Thailand.*

## APPENDIX 14. THE GLAUCOMA QUALITY OF LIFE–15 QUESTIONNAIRE

The Glaucoma Quality of Life–15 questionnaire: list of daily activities with the strongest relationship with VF loss in glaucoma.\*

Patient instructions: please circle the correct answer on the scale ranging from 1 to 5 where (1) stands for no difficulty, (2) for a little bit of difficulty, (3) for some difficulty, (4) for quite a lot of difficulty, and (5) for severe difficulty. If you do not perform any of the activities for other than visual reasons, please circle (0).

Does your vision give you any difficulty, even with glasses, with the following activities?

|   | None | A little bit | Some | Quite a lot | Severe | Do not perform for non-visual reasons |
|---|------|--------------|------|-------------|--------|---------------------------------------|
| Reading newspapers                          | 1    | 2            | 3    | 4           | 5      | 0                                     |
| Walking after dark                          | 1    | 2            | 3    | 4           | 5      | 0                                     |
| Seeing at night                             | 1    | 2            | 3    | 4           | 5      | 0                                     |
| Walking on uneven ground                    | 1    | 2            | 3    | 4           | 5      | 0                                     |
| Adjusting to bright lights                  | 1    | 2            | 3    | 4           | 5      | 0                                     |
| Adjusting to dim lights                     | 1    | 2            | 3    | 4           | 5      | 0                                     |
| Going from light to dark room or vice versa | 1    | 2            | 3    | 4           | 5      | 0                                     |
| Tripping over objects                       | 1    | 2            | 3    | 4           | 5      | 0                                     |
| Seeing objects coming from the side         | 1    | 2            | 3    | 4           | 5      | 0                                     |
| Crossing the road                           | 1    | 2            | 3    | 4           | 5      | 0                                     |
| Walking on steps/stairs                     | 1    | 2            | 3    | 4           | 5      | 0                                     |
| Bumping into objects                        | 1    | 2            | 3    | 4           | 5      | 0                                     |
| Judging distance of foot to step/curb       | 1    | 2            | 3    | 4           | 5      | 0                                     |
| Finding dropped objects                     | 1    | 2            | 3    | 4           | 5      | 0                                     |
| Recognising faces                           | 1    | 2            | 3    | 4           | 5      | 0                                     |

\* Based on the results of this study.<sup>1</sup>

### Reference

1. Nelson P, Aspinall P, Papasouliotis O, Worton B, O'Brien C. Quality of life in glaucoma and its relationship with visual function. J Glaucoma. 2003;12:139-50.



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## ***FURTHER READING***

1. American Academy of Ophthalmology. Preferred Practice Pattern: Primary Open-Angle Glaucoma. 2005. Available at: [www.aao.org/ppp](http://www.aao.org/ppp)
2. American Academy of Ophthalmology. Preferred Practice Pattern: Primary Angle Closure. 2005. Available at: [www.aao.org/ppp](http://www.aao.org/ppp)
3. American Academy of Ophthalmology. Preferred Practice Pattern: Primary Open-Angle Glaucoma Suspect. 2006. Available at: [www.aao.org/ppp](http://www.aao.org/ppp)
4. European Glaucoma Society. Terminology and Guidelines for Glaucoma. 2nd Ed. Savona: Dogma; 2003.



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## ***SUGGESTED AREAS FOR FURTHER RESEARCH***

- Prevalence and incidence of primary open angle glaucoma and primary angle closure glaucoma in the Asia Pacific region
- Natural history of glaucoma in the Asia Pacific region
- Natural history of angle closure
- Risk factors: ranking risk factors among Asian populations according to importance
- Normal values of central corneal thickness and optic disc parameters in Asian populations
- Applicability of the 'ISNT rule' to Asian eyes
- Target pressure reduction: evidence for extent of reduction required
- Clinical classification of angle closure glaucoma based on visual outcomes
- Clarification of mechanisms responsible for angle closure in Asian populations
- Structural and functional change patterns, rates, and extent in angle closure glaucoma versus open angle glaucoma
- Randomised controlled trials of all aspects of management of angle closure, particularly the roles of laser iridotomy, laser iridoplasty, lens extraction, and filtering surgery
- Treatment outcomes for angle closure glaucoma versus open angle glaucoma for medical, laser, and surgical treatments
- Efficacy of screening and prophylaxis for angle closure
- Cost-efficient glaucoma screening



---

## **DEFINITION OF TERMS**

|  |   |
|--|---|
| <b>Adherence</b>                           | Alternative and identical word for compliance.  |
| <b>Angle neovascularisation</b>            | New vessel formation within or on the surface of angle structures with or without formation of a fibrovascular membrane.  |
| <b>Anterior ischaemic optic neuropathy</b> | Optic nerve head ischaemia resulting from disturbance in the short posterior ciliary artery circulation.  |
| <b>Ciliary block</b>                       | An anatomical/functional abnormality at the level of the lens, zonule/anterior vitreous, and ciliary processes preventing normal aqueous circulation. May also be known as 'malignant glaucoma'.          |
| <b>Cup-disc ratio</b>                      | The fractional decimal value obtained by dividing the cup diameter with the disc diameter. The closer the value is to 1, the worse the damage.  |
| <b>Double DOT</b>                          | Technique for instilling eye drops: combination of 'don't open the eyelid' and 'digital occlusion of the tear duct'.  |
| <b>Glaucoma suspect disc</b>               | Optic nerve head appearance suggestive of glaucomatous damage.  |
| <b>Glaucomatous optic neuropathy</b>       | Characteristic pattern of damage to the optic nerve head caused by glaucoma.  |
| <b>Neovascular glaucoma</b>                | Glaucoma resulting from a fibrovascular membrane across the angle in response to ischaemia.   |
| <b>Normal tension glaucoma</b>             | Characteristic glaucomatous optic neuropathy in the presence of statistically normal intraocular pressure.  |
| <b>Occludable angle</b>                    | Clinical term for an angle that is gonioscopically open but narrow enough to be considered at risk of closure.  |
| <b>Ocular hypertension</b>                 | Intraocular pressure more than 2 standard deviations above the population mean with open angles, normal central corneal thickness, and no evidence of glaucomatous optic neuropathy or visual field loss. |
| <b>Peripapillary atrophy</b>               | Zone of chorioretinal atrophy abutting the optic nerve head.  |
| <b>Peripheral anterior synechiae</b>       | Permanent adhesions between the peripheral iris and other angle structures.   |
| <b>Pigment dispersion syndrome</b>         | Abnormal scattering of iris pigment into the anterior segment of the eye.   |
| <b>Plateau iris configuration</b>          | An occludable angle in the absence of pupil block.  |
| <b>Plateau iris syndrome</b>               | Angle closure in the presence of a patent iridectomy/iridotomy.   |
| <b>Posner-Schlossman syndrome</b>          | Episodic anterior uveitis and presumed trabeculitis with secondary elevation of intraocular pressure.   |

---

|  |   |
|--|---|
| <b>Primary angle closure</b>               | Primary angle closure suspect with either statistically raised intraocular pressure, and/or peripheral anterior synechiae, or signs of trabecular damage.   |
| <b>Primary angle closure glaucoma</b>      | Primary angle closure with glaucomatous optic neuropathy.   |
| <b>Primary angle closure suspect</b>       | An eye in which appositional contact between the peripheral iris and posterior trabecular meshwork is present or considered possible.<br>Epidemiologically: “an angle in which 180° to 270° of the posterior trabecular meshwork cannot be seen gonioscopically.” |
| <b>Primary open angle glaucoma</b>         | Chronic progressive optic neuropathy with characteristic changes in the optic nerve head and/or visual field in the absence of primary causes.  |
| <b>Primary open angle glaucoma suspect</b> | Significant risk factors for glaucoma (ocular hypertension, family history) and/or glaucoma suspect disc in the absence of frank glaucomatous optic neuropathy or visual field loss.  |
| <b>Pseudoexfoliation syndrome</b>          | Deposition of an abnormal fibrillo-granular protein predominantly in the anterior segment of the eye.   |
| <b>Secondary angle closure glaucoma</b>    | Glaucomatous optic neuropathy with angle closure and an identifiable cause.   |
| <b>Secondary open angle glaucoma</b>       | Raised intraocular pressure in the presence of identifiable cause(s). Without treatment, it is presumed this will cause glaucomatous optic neuropathy.  |

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## **ABBREVIATIONS**

|        |  |
|--------|--|
| 5-FU   | 5-Fluorouracil                                 |
| AC     | Angle closure                                  |
| ACD    | Anterior chamber depth                         |
| ACE    | Angiotensin-converting enzyme                  |
| ACG    | Angle closure glaucoma                         |
| AGIS   | Advanced Glaucoma Intervention Study           |
| ALPI   | Argon laser peripheral iridoplasty             |
| ALS    | Argon laser suture lysis                       |
| ALT    | Argon laser trabeculoplasty                    |
| bd     | Twice daily                                    |
| CAC    | Chronic angle closure                          |
| CAI    | Carbonic anhydrase inhibitor                   |
| CCB    | Calcium channel blocker                        |
| CCT    | Central corneal thickness                      |
| CDR    | Cup-disc ratio                                 |
| CI     | Confidence interval                            |
| CIGTS  | Collaborative Initial Glaucoma Treatment Study |
| CNS    | Central nervous system                         |
| COPD   | Chronic obstructive pulmonary disease          |
| CVA    | Cerebrovascular accident                       |
| DLT    | Diode laser trabeculoplasty                    |
| ECCE   | Extracapsular cataract extraction              |
| EMGT   | Early Manifest Glaucoma Trial                  |
| FAQs   | Frequently asked questions                     |
| FDA    | Food and Drug Administration                   |
| GAT    | Goldmann applanation tonometry                 |
| GDx    | Scanning laser polarimetry                     |
| GI     | Gastrointestinal                               |
| GON    | Glaucomatous optic neuropathy                  |
| GPA    | Glaucoma progression analysis                  |
| HRT    | Confocal scanning laser ophthalmoscopy         |
| ICE    | Irido-corneal endothelial                      |
| IOL    | Intraocular lens                               |
| IOP    | Intraocular pressure                           |
| LASEK  | Laser-assisted subepithelial keratectomy       |
| LASIK  | Laser in situ keratomileusis                   |
| LPI    | Laser peripheral iridotomy                     |
| MAOI   | Monoamine oxidase inhibitors                   |
| MMC    | Mitomycin C                                    |
| NA     | Not applicable                                 |
| Nd:YAG | Neodymium yttrium aluminum garnet              |
| NMDA   | N-methyl-D-aspartate                           |
| NTG    | Normal tension glaucoma                        |
| OAG    | Open angle glaucoma                            |
| OCT    | Optical coherence tomography                   |
| OH     | Ocular hypertension                            |
| OHTS   | Ocular Hypertension Treatment Study            |
| PAC    | Primary angle closure                          |
| PACG   | Primary angle closure glaucoma                 |
| PACS   | Primary angle closure suspect                  |

---

|        |  |
|--------|--|
| PAS    | Peripheral anterior synechiae              |
| PGA    | Prostaglandin analogue                     |
| PI     | Peripheral iridotomy                       |
| POAG   | Primary open angle glaucoma                |
| PPA    | Peripapillary atrophy                      |
| PPS    | Peripheral posterior synechiae             |
| PRK    | Photorefractive keratectomy                |
| PSD    | Pattern standard deviation                 |
| PXF    | Pseudoexfoliation                          |
| qid    | 4 times daily                              |
| QOL    | Quality of life                            |
| RNFL   | Retinal nerve fibre layer                  |
| SD     | Standard deviation                         |
| SEAGIG | South East Asia Glaucoma Interest Group    |
| SITA   | Swedish Interactive Thresholding Algorithm |
| SLT    | Selective laser trabeculoplasty            |
| tds    | 3 times daily                              |
| TM     | Trabecular meshwork                        |
| VF     | Visual field                               |
| WGA    | World Glaucoma Association                 |
| YAG    | Yttrium aluminum garnet                    |

## INDEX

### A

|                                      |  |
|--------------------------------------|--|
| Advanced Glaucoma Intervention Study | 64, 65   |
| $\alpha$ -Agonists                   | 6, 25, 59, 64, 67, 73, 85, 87  |
| Angle closure                        | 3, 5, 6, 9, 10, 18, 23, 24, 25, 31, 33, 45, 48, 50, 58, 59, 61, 62, 65, 67, 69 |
| Primary                              | 10, 17, 31, 55, 56, 58, 59, 62, 63, 65, 69                                     |
| Glaucoma                             | 3, 17, 24, 31, 41, 45, 56, 59, 62, 64, 68, 69, 70, 71                          |
| Suspect                              | 10, 17, 24, 59, 63, 65, 69   |
| Anterior Chamber                     | 3, 8, 9, 21, 33, 34, 36, 41, 44, 45, 56, 79, 80                                |
| Depth                                | 9, 55, 56, 59, 105   |
| Antimetabolites                      | 41, 42, 43, 44, 45, 46   |
| 5-FU                                 | 42, 43, 45   |
| MMC                                  | 36, 42, 43, 44, 45, 67, 70   |
| Artifact                             | 84   |

### B

|                   |                                       |
|-------------------|---------------------------------------|
| $\beta$ -Blockers | 6, 25, 64, 65, 66, 67, 73, 74, 85, 87 |
|-------------------|---------------------------------------|

### C

|  |   |
|--|---|
| Carbonic anhydrase inhibitors                  | 6, 25, 31, 33, 65, 66, 67, 74, 76, 85, 87 |
| Cataract                                       | 3, 32, 44, 45, 47, 49, 68, 69, 70, 84, 88 |
| Central corneal thickness                      | 8, 17, 18, 20, 60, 64                     |
| Child  | 5, 6, 25, 41, 73, 87                      |
| Cholinergics                                   | 25, 88                                    |
| Collaborative Initial Glaucoma Treatment Study | 68  |
| Cup-disc ratio                                 | 12, 18, 23, 48, 58, 61, 65                |
| Cyclophotocoagulation                          | 29, 34, 67                                |

### D

|                  |                                    |
|------------------|------------------------------------|
| Disc haemorrhage | 10, 11, 13, 17, 18, 23, 48, 62, 91 |
| Double DOT       | 26, 66, 74                         |

### E

|                               |               |
|-------------------------------|---------------|
| Early Manifest Glaucoma Trial | 65            |
| Episcleral venous pressure    | 8, 60, 65, 74 |

### G

|                          |                    |
|--------------------------|--------------------|
| Glaucoma drainage device | 41, 43, 44, 45, 70 |
|--------------------------|--------------------|

|                               |   |
|-------------------------------|---|
| Glaucoma life story           | 51  |
| Glaucomatous optic neuropathy | 17, 21, 47, 48, 49  |
| Gonioscopy                    | 9, 10, 32, 34, 48, 56, 58, 60, 61, 62, 63, 65, 68, 72, 79, 82 |
| Dynamic                       | 79  |
| Gonioscopic changes           | 47, 48, 80  |
| Indentation                   | 7, 10, 57, 58, 61   |
| Lens                          | 29  |

### H

|                     |        |
|---------------------|--------|
| Hyperosmotic agents | 25, 88 |
|---------------------|--------|

### I

|                             |  |
|-----------------------------|--|
| Intraocular pressure        | 3, 5, 6, 7, 8, 9, 10, 17, 18, 19, 20, 23, 24, 25, 26, 29, 30, 31, 32, 33, 34, 35, 36, 37, 41, 43, 44, 45, 47, 48, 50, 51, 55, 56, 57, 58, 59, 60, 62, 63, 64, 65, 66, 67, 68, 70, 71, 73, 74, 77, 78, 89 |
| Diurnal                     | 8, 60, 71  |
| Spike                       | 30, 31, 32, 33, 34   |
| Target                      | 19, 20, 26, 43, 48, 63, 64, 71   |
| Iridoplasty                 | 29, 32, 33, 41, 49, 68   |
| Iridotomy, laser peripheral | 21, 61, 62, 65, 68, 69, 72, 85   |
| ISNT rule                   | 11, 12   |

### L

|                       |   |
|-----------------------|---|
| Laser                 | 5, 24, 27, 29, 31, 32, 33, 34, 35, 37, 41, 42, 43, 49, 50, 63, 68, 69, 74, 76, 89, 90               |
| Diode                 | 90  |
| Argon                 | 29, 30, 31, 32, 36, 89  |
| Scanning, polarimetry | 92, 94  |
| Suture lysis          | 36, 42  |
| YAG                   | 29, 31, 32, 35, 36, 44, 68, 69  |
| Lens                  | 7, 8, 9, 11, 21, 23, 29, 32, 33, 36, 41, 45, 49, 57, 58, 60, 61, 62, 68, 69, 76, 79, 83, 84, 86, 88 |
| Extraction            | 21, 41, 45  |
| Goldmann              | 23  |
| Goniolens             | 7, 79   |
| Laser                 | 29  |
| Mirror                | 11, 33, 36, 57, 58, 61, 79  |

|                                     |  |  |                           |   |
|-------------------------------------|--|--|---------------------------|---|
| <b>N</b>                            |  |  | <b>R</b>                  |   |
| Neuroretinal rim                    | 10, 11, 12, 48, 91   |  | Retinal nerve fibre layer | 10, 11, 13, 48, 49, 61, 64  |
| <b>O</b>                            |  |  | Risk factor               | 3, 5, 7, 13, 17, 18, 19, 20, 23, 25, 42, 47, 48, 50, 51, 55, 59, 61, 64, 65, 71 |
| Occludable angle                    | 24, 33, 80   |  |                           |   |
| Ocular hypertension                 | 6, 13, 17, 23, 60, 62, 65, 74                                    |  |                           |   |
| Ocular Hypertension Treatment Study | 65   |  | <b>S</b>                  |   |
| One-eyed therapeutic trial          | 26   |  | Schaffer                  | 80  |
| Open angle                          | 10, 61, 62, 69   |  | Scotoma                   | 49, 83, 84, 95, 96  |
| Glaucoma                            | 3, 6, 13, 18, 24, 55, 56, 59, 61, 62, 63, 64, 65, 70, 71, 73, 89 |  | Screening                 | 53, 54, 55, 56, 57, 58, 72  |
| Suspect                             | 13, 17, 19, 51, 55, 58, 59, 60, 62, 64, 83                       |  | SEAGIG decision square    | 50  |
| Optic disc                          | 11, 13, 14, 48, 49, 50, 56, 57, 58, 61, 62, 64, 71               |  | Secondary glaucoma        | 3, 9, 17, 19, 23, 24, 62, 70, 73, 85  |
| Examination                         | 11, 55, 56, 61, 62   |  | Slit lamp                 | 7, 9, 11, 12, 13, 34, 55, 56, 57, 58, 61, 62, 77, 79                            |
| Photography and imaging             | 13, 49, 71   |  | Spaeth                    | 80  |
| Optic nerve                         | 13, 14, 47, 48   |  | Surgery                   | 5, 21, 24, 27, 29, 34, 36, 41, 42, 43, 44, 45, 67, 68, 69, 70, 71, 88           |
| Damage                              | 13, 56, 71   |  | Cataract                  | 69, 70, 88  |
| Head                                | 10, 13, 62, 92   |  | Combined                  | 68  |
| <b>P</b>                            |  |  | Filtration                | 29, 36, 41, 42, 43, 68, 69, 71  |
| Perimetry                           | 13, 14, 49, 56, 57, 71, 83                                       |  | Refractive                | 48, 63  |
| Automated                           | 13, 14, 62, 64   |  | <b>T</b>                  |   |
| Blue-on-yellow                      | 14, 62   |  | Tonometry                 | 7, 8, 9, 56, 58, 63, 78   |
| White-on-white                      | 62, 64   |  | Torchlight test           | 56, 58, 60  |
| Peripapillary atrophy               | 91   |  | Trabecular meshwork       | 9, 10, 29, 30, 33, 44, 80   |
| Peripheral anterior synechiae       | 10, 17, 30, 33, 34, 45, 50, 62, 65, 79                           |  | Trabeculectomy            | 6, 41, 42, 43, 44, 45, 68, 69, 70   |
| Pigment dispersion                  | 5, 8, 9, 17, 48, 62, 65  |  | Trabecuoplasty            | 27, 29, 30, 33, 89  |
| Posner-Schlossman                   | 5  |  |                           |   |
| Pregnancy                           | 6, 67, 74  |  | <b>V</b>                  |   |
| Prostaglandin analogues             | 25, 26, 64, 65, 66, 67, 69, 74, 88                               |  | van Herick                | 9, 56, 58, 60, 72, 80, 81   |
| Pseudoexfoliation                   | 5, 9, 17, 18, 23, 50, 61, 62                                     |  | Visual field              | 18, 23, 47, 49, 50, 56, 57, 58, 64, 70, 71                                      |
| <b>Q</b>                            |  |  | Damage                    | 6, 17, 23, 30, 47, 49, 62, 64, 65, 71,  |
| Quality of life                     | 18, 23, 47, 50, 55, 63, 64, 69                                   |  | Test                      | 13, 14, 49, 55, 57, 58, 65  |



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