ASIA PACIFIC
Glaucoma Guidelines

SEAGIG
South East Asia Glaucoma Interest Group
Foreword

By increasing awareness and knowledge of glaucoma across the Asia Pacific region, these Guidelines aim to reduce glaucomatous visual disability and to provide a rational basis for glaucoma management.

The establishment of best-practice methodologies throughout the Asia Pacific region represents a unique challenge, given our diverse healthcare service systems and wide range of available resources. To address this need, the South East Asia Glaucoma Interest Group (SEAGIG), with the support of the Asian-Oceanic Glaucoma Society (AOGS), convened an Asia Pacific Glaucoma Guidelines Working Party to develop comprehensive Glaucoma Guidelines for the Asia Pacific region.

The members of the Working Party collaborated during a series of meetings to compile information and recommendations to assist comprehensive ophthalmologists, general healthcare and eye care professionals, as well as healthcare policy makers to deliver effective glaucoma management to their communities. An extensive Review Committee assessed the results and made significant contributions to the final manuscript.

As these Guidelines rely on direct medical experience and, wherever possible, on published evidence, they are as up-to-date as possible. The Asia Pacific Glaucoma Guidelines Working Party is committed to providing updates to the Guidelines on a regular basis.

Critical to the development of the Asia Pacific Glaucoma Guidelines has been the support of the AOGS, and a generous educational grant from then Pharmacia Corporation (now Pfizer Inc.). This sponsorship permitted the Working Party to meet and to produce, publish and distribute these Guidelines.
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ACKNOWLEDGEMENT

This work was made possible by an unrestricted educational grant from Pfizer Inc.

The Asia Pacific Glaucoma Guidelines Working Party would like to thank especially for their support:

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# Contents

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Foreword</td>
<td>i</td>
</tr>
<tr>
<td>Acknowledgement</td>
<td>iv</td>
</tr>
<tr>
<td>Introduction</td>
<td>1</td>
</tr>
<tr>
<td>Epidemiology of Glaucoma in Asia</td>
<td>5</td>
</tr>
</tbody>
</table>

## Section 1

1.1 Patient Assessment   | 11   |
1.2 Treatment Categories and Targets | 25   |

## Section 2

2.1 Initiation of Treatment | 31   |
2.2 Medical Treatment       | 33   |
2.3 Laser Treatment         | 41   |
2.4 Surgery                 | 51   |

## Section 3

3.1 Follow-up              | 59   |

Case Detection             | 67   |

Appendices                 | 75   |

Suggested Areas for Further Research | 88   |

Definition of Terms         | 89   |

Index                      | 91   |
## List of Appendices

<table>
<thead>
<tr>
<th>Appendix</th>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>How to test calibration of a Goldmann tonometer</td>
<td>76</td>
</tr>
<tr>
<td>2</td>
<td>Tonometry mires</td>
<td>77</td>
</tr>
<tr>
<td>3a</td>
<td>Gonioscopy</td>
<td>78</td>
</tr>
<tr>
<td>3b</td>
<td>Goniogram/gonioscopic chart</td>
<td>79</td>
</tr>
<tr>
<td>3c</td>
<td>Corneal wedge diagram</td>
<td>80</td>
</tr>
<tr>
<td>4</td>
<td>How to optimize patient performance in subjective perimetry</td>
<td>81</td>
</tr>
<tr>
<td>5</td>
<td>Secondary glaucomas – principles of management</td>
<td>82</td>
</tr>
<tr>
<td>6</td>
<td>Angle closure mechanisms</td>
<td>83</td>
</tr>
<tr>
<td>7a</td>
<td>Argon laser trabeculoplasty</td>
<td>84</td>
</tr>
<tr>
<td>7b</td>
<td>Contact transscleral diode laser</td>
<td>85</td>
</tr>
<tr>
<td>8</td>
<td>Glaucomatous optic neuropathy</td>
<td>86</td>
</tr>
<tr>
<td>9</td>
<td>Field progression print-outs</td>
<td>87</td>
</tr>
</tbody>
</table>
Introduction

In 1996, with the support of the American Glaucoma Society, the American Academy of Ophthalmology produced its Preferred Practice Pattern for Primary Open-Angle Glaucoma, followed four years later by an update and two additional volumes on Primary Angle Closure and Primary Open-Angle Glaucoma Suspect. These guidelines identify characteristics and components of quality eye care “commensurate with present knowledge and resources” – this translates into guidance for state-of-the-art patterns of practice, which should be helpful in the care of most patients, rather than any particular individual. Where data permits, these guidelines are firmly evidence-based; otherwise they rely on consensus opinion. Similarly, the European Glaucoma Society published its Terminology and Guidelines for Glaucoma in 1998, with the aim of “improving the mutual understanding of this disease in addition to providing a rational approach to the diagnosis and management of glaucoma”. This year a Second Edition has been released. These projects have set out to complement existing scientific literature, and involved input from many glaucoma sub-specialists. The resulting publications have proven useful for, and thus popular with, comprehensive ophthalmologists around the world. They have also been useful in communications with allied eye healthcare professionals, as well as governmental agencies and non-governmental organizations.

Over the past 20 years, increasing epidemiological data from across the Asia Pacific region has highlighted the varying prevalence and incidence rates, diagnostic types and behavior of different glaucoma disease patterns encountered by the ophthalmological community. With the availability of this information, the need for similar guidelines relevant to our region can now be met. Such guidelines must be sensitive to the wide variations in human, structural and equipment resources available throughout the region, as well as the ethnic diversity of our communities. What is applicable in one country or location may not be in another.

These Guidelines have been developed during a time of rapidly expanding medical technology, knowledge and skills in a changing environment. As public awareness and expectations increase, and as the population ages (even in less developed societies), there is an increasing demand for high-level care for a greater number of people over extended periods of time. However, available resources have been unable to expand proportionally. As cost containment is an inescapable reality, every treatment or investigation that is undertaken reduces the capacity to implement another intervention that could benefit another patient. Therefore, what we, as clinicians, do for our patients needs to be demonstrated to be effective. If not, it must at least be recognized to be only partly proven or as yet unproven. Guided by this knowledge, our lines of enquiry can be channeled appropriately.
In the development of these Guidelines, we have tried to use underlying evidence, and to stratify the recommendations according to their strength. This has allowed us to clarify and to append to these Guidelines, suggested areas for further research. It is hoped this will facilitate our growth as a mutually supportive ophthalmic community.

How widespread is the problem? What are the risk factors? How does it differ from elsewhere? The section on Epidemiology tries to answer these questions. How do we take a focused history, perform an appropriate examination? What additional tests might we consider for the patient either in the presence or absence of glaucoma? What is the risk of developing glaucoma? What is the degree of damage if glaucoma is already present? What is the risk of glaucoma progressing? Patient Assessment tackles these, both for initiation of therapy and its follow-up.

The Treatment Categories and Targets section helps us to tailor for the individual patient, therapeutic goals to severity of glaucoma. Treatment of glaucoma has been divided into medical, laser and surgical therapy. Case detection is considered from both population screening and opportunistic points of view.

These guidelines have been developed in an easy-to-read format for the benefit of comprehensive ophthalmologists, other eye care workers and our healthcare colleagues. The format answers questions of ‘why?’, ‘what?’, ‘when?’, ‘how?’ and ‘for whom?’. We strongly recommend that the reader examines the entire Guidelines before applying information from any one section to the care of an individual patient.

As with all treatment guidelines, this publication is not a prescription for automated care. By adapting the guidelines with respect to patients’ individual needs, the socio-economic environment and medical facilities available, as well as your own experience, we hope the hallmark of excellent care will be achieved.

Ivan Goldberg
Chair, Asia Pacific Glaucoma Guidelines Working Party

On behalf of the South East Asia Glaucoma Interest Group and the Asian-Oceanic Glaucoma Society
References


Epidemiology of Glaucoma in Asia

Glaucomatous optic neuropathy (GON) is most prevalent among people of African origin, and least prevalent in full-blooded Australian Aborigines; Asian populations have rates intermediate between these two groups. European- and African-derived peoples suffer predominantly from primary open angle glaucoma (POAG), whereas rates of primary angle closure glaucoma (PACG) are higher among East Asians than in other populations. Although a direct and exact comparison of POAG rates is difficult, POAG likely has a similar prevalence in Asian and European populations. The higher rate of GON in those of Chinese extraction is probably attributable to the excess of PACG. Rates on the Indian subcontinent vary substantially between studies, although these differences are probably methodological. In absolute terms, there are large numbers of people suffering from PACG and POAG in India. Preliminary data suggests that prevalence of PACG is less in Southeast Asian populations than in the Chinese, but more than in Europeans.

Incidence rates of symptomatic acute angle closure (given as cases/100,000 persons/year for the population aged 30 years and over) range from 4.7 in Europe (Finland) to 15.5 in Chinese Singaporeans. Malay and Indian people in Singapore have lower rates than Chinese Singaporeans (6.0 and 6.3, respectively).

Population surveys in Mongolia found glaucoma to be the cause of 35% of blindness in adults (cataract being the cause of 36% of blindness). Among Chinese Singaporeans, 60% of adult blindness was caused by glaucoma. Cautious extrapolation of these data suggests that around 1.7 million people in China suffer blindness caused by glaucoma. PACG is responsible for the vast majority (91%) of these cases. Secondary glaucoma is the most common cause of uniocular blindness.

Glaucoma is the leading cause of registered, permanent blindness in Hong Kong (23%). In Japan, diabetic retinopathy (18%), cataract (16%) and glaucoma (15%) are the leading causes of blindness. 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. This compares with 10.2% in the Aravind survey of rural south India, where 77.5% of blindness was from cataract.

Advancing age is the single most consistent risk factor for all types of glaucoma. A positive family history is also a risk factor for glaucoma. Female gender is recognized as a major predisposing factor toward the development of PACG. There is little clear evidence to support a gender difference in POAG. Those of Chinese ethnic origin are at a higher risk of developing angle closure than those of Malay descent and South Indian people. Raised intraocular pressure (IOP) is a risk factor for glaucomatous optic neuropathy in Chinese people.
Although angle closure is associated with a hypermetropic refractive state, cases do occur in myopes. A shallow anterior chamber has long been recognized to be a factor that predisposes toward angle closure.\(^{20}\) The depth of the anterior chamber reduces with age and tends to be shallower in women than in men.\(^{21,22}\) There may also be an association between myopia and POAG.\(^ {23}\) In India, pseudoexfoliation was found in 26.7% of the cases of POAG.\(^ {24}\)

References:


Section 1
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 the developed and developing worlds. Inevitably, some sections will have more relevance to one or other setting, however, time taken in examining 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 specialized center.

**Why assess?**

The aims during the initial assessment are:

- to determine whether or not glaucoma is present, or likely to develop (i.e., assess risk factors)
- to exclude or confirm alternative diagnosis
- to identify the underlying mechanism of damage, so as to guide the choice of management
- to begin planning a strategy of management
- to identify suitable forms of treatment, and to exclude those which are inappropriate.

**What to assess?**

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

1. History
2. Examination/investigations

**History**

**Key points:**

- Most glaucomas, including angle closure glaucoma (ACG), are asymptomatic until advanced
- Assess medical and social factors that will affect treatment decisions
- Older age and family history of glaucoma are particularly important
- Younger age means a longer exposure to glaucoma and its treatment

**Past ophthalmic history**

**Consider:**

Previous medications (especially glaucoma medications, steroids), trauma, previous eye surgery or laser.
**Visual symptoms of glaucoma**

- Visual blurring and discomfort:
  - This may be due to angle closure, Posner-Schlossman syndrome or pigment dispersion
- Glare and colored rings around lights from corneal edema:
  - This needs to be differentiated from migraine
- Poor light/dark adaptation
- Difficulty tracking fast-moving objects (e.g., golf/tennis balls)

**Past medical history**

**Consider:**

Factors that will affect life expectancy and compliance with treatment. Exclude past hemodynamic crises (postpartum hemorrhage, ruptured abdominal aneurysm, severe trauma) that may cause optic disc pallor and cupping that mimics glaucoma but is not progressive.¹⁻⁴

Specific predisposing diseases are summarized in Table 1.1.

**Table 1.1: Factors to consider when taking a medical history in a patient with suspected glaucoma**

<table>
<thead>
<tr>
<th>Respiratory</th>
<th>Asthma and other chronic obstructive pulmonary diseases associated with hyperresponsive airways and/or reduced lung capacity will limit the use of topical β-blockers</th>
</tr>
</thead>
</table>
| Cardiovascular | • Cardiac arrhythmias, e.g., heart block, may preclude the use of topical β-blockers or α-agonists  
• Systemic hypertension – over treatment may worsen glaucoma risk and progression. Systemic β-blockers may mask elevated intraocular pressure (IOP)  
• Vasospastic tendency (migraine, Raynaud’s syndrome) may be associated with an increased incidence and severity of glaucoma, especially normal pressure  
• Previous episodes of profound hypotension or blood loss |
## Patient Assessment

| Endocrine | • Diabetes – increasingly prevalent and associated with open angle and neovascular glaucoma  
|           | • Thyroid eye disease  
|           | • Pituitary tumors  
| Central nervous system | • Previous cerebrovascular accident (CVA)/head injury/pituitary lesions (field loss)  
|           | • Early dementia – affects compliance, understanding and insight into the disease  
| Musculo-skeletal | Arthritis (osteo-, rheumatoid) may affect severely the ability to administer eye drops  
| Urogenital | Urinary stones may limit systemic carbonic anhydrase inhibitors (CAIs)  
| Ocular trauma | Angle recession, lens dislocation, choroidal/retinal damage  
| Pregnancy and lactation | Present or possible, renders all interventions and potentially hazardous  

### Medication

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

a) Steroids – any route of administration is associated with ocular hypertension, open angle glaucoma. Sometimes found in traditional medicines

b) Glaucoma drops (prolonged use may increase trabeculectomy failure)

c) Anticholinergics/tricyclic antidepressants – can cause angle closure

d) Anticonvulsants (vigabatrin) – linked to nasal peripheral field loss without disc changes

e) Systemic β-blockers/calcium channel blockers – may interact with topical β-blockers
Social history

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

Minimal acceptable resources for examination
- A slit lamp with indirect lens between 60–90D and/or direct ophthalmoscope
- An automated perimeter
- A gonioscope that allows indentation gonioscopy
- A Goldmann-style applanation tonometer (or Tonopen): Schiötz or Maklakov tonometers are not generally acceptable

When the patient cannot get to a slit lamp
- Portable hand held slit lamp may be very useful
- In the absence of a portable slit lamp, a jeweler’s loop or an operating loop with a torchlight will allow a reasonable anterior segment examination
- A direct ophthalmoscope set at +10 to +12 will allow anterior segment examination
- Taking intraocular pressure of these patients is best performed with a Tonopen or Perkin’s tonometer
Intraocular pressure (IOP) is the only modifiable risk factor for glaucoma.

Goldmann-style Applanation Tonometry (GAT). (Tonopen, if GAT not available).

Every visit.

- Ensure tonometer is calibrated (see Appendix 1: How to test calibration of a Goldmann tonometer).
- The prism tip must be disinfected and then disinfectant removed.
- The eyelashes must be kept out of the way (avoid pressure on eye).
- The cornea must be anesthetized.
- The tip must touch the central cornea gently with the observer looking through the slit lamp eyepiece just prior to the tip making contact (tip: look for the white split ring that will fluoresce when the tip touches the cornea).
- Adjust the gauge until the split tear meniscus just touches on the inside.

**Factors associated with IOP**

**Table 1.2: Factors affecting measured IOP**

<table>
<thead>
<tr>
<th>Factor</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Circadian cycle</strong></td>
<td>The IOP follows a circadian cycle, often with a peak in the morning and a trough in the evening. The normal diurnal variation is 3–6 mmHg</td>
</tr>
<tr>
<td><strong>Central corneal thickness</strong></td>
<td>Thicker corneas are associated with artificially elevated IOP measurements, thinner corneas with artificially depressed IOP measurements: apply correction 1–3 mmHg/40µ deviation from 525µ</td>
</tr>
<tr>
<td><strong>Blood pressure</strong></td>
<td>IOP is positively associated with systemic blood pressure, particularly systolic pressure⁵⁻⁷</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td>In Caucasians, each decade after 40 years of age is associated with an approximate increase of 1 mmHg in IOP. Systemic hypertension may confound this relationship</td>
</tr>
<tr>
<td><strong>Exercise</strong></td>
<td>Exercise may increase (e.g., yoga head-down positions) or reduce (by dehydration and/or acidosis) IOP by 2–6 mmHg</td>
</tr>
<tr>
<td><strong>Lifestyle</strong></td>
<td>Large volume rapid fluid intake increases IOP, while alcohol and marijuana depress it</td>
</tr>
<tr>
<td><strong>Posture</strong></td>
<td>Horizontal or head-down position increases IOP</td>
</tr>
</tbody>
</table>
**Table 1.3: Measurement errors associated with GAT**

<table>
<thead>
<tr>
<th>IOP reading artificially low</th>
<th>IOP reading artificially high</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Insufficient fluorescein in tear film</td>
<td>• Excessive fluorescein in tear film</td>
</tr>
<tr>
<td>• Insufficient fluorescein in tear film</td>
<td>• Eyelid pressure on globe from blepharospasm</td>
</tr>
<tr>
<td>• Eyelid pressure on globe from blepharospasm</td>
<td>• Digital pressure on globe to hold lids apart</td>
</tr>
<tr>
<td>• Digital pressure on globe to hold lids apart</td>
<td>• Obese patient</td>
</tr>
<tr>
<td>• Obese patient</td>
<td>• Patient straining to reach chin/forehead rest</td>
</tr>
<tr>
<td>• Patient straining to reach chin/forehead rest</td>
<td>• Patient breath-holding</td>
</tr>
<tr>
<td>• Patient breath-holding</td>
<td>• Patient wearing constricting clothing around neck (e.g., tight shirt collar +/- tie for men)</td>
</tr>
<tr>
<td>• Patient wearing constricting clothing around neck (e.g., tight shirt collar +/- tie for men)</td>
<td>• Hair lying across cornea distorting mires</td>
</tr>
<tr>
<td>• Hair lying across cornea distorting mires</td>
<td>• Lens-corneal apposition</td>
</tr>
<tr>
<td>• Lens-corneal apposition</td>
<td></td>
</tr>
</tbody>
</table>

(Please refer to Appendix 2: Tonometry mires)

**Anterior segment**

When examining the anterior segment, look for:

**Globe surface:**
- episcleral blood vessels
- follicles.
**Anterior chamber:**
- pigment on corneal endothelium (pigment dispersion)
- peripheral anterior chamber depth (van Herick technique)
- central anterior chamber depth
- evidence of inflammation (e.g., keratic precipitates).

**Iris:**
- mid-dilated poorly reactive (post angle closure attack)
- isolated zones of patch atrophy or spiraling
- rubeosis
- synechiae
- configuration in relation to lens
  - pseudoexfoliation material on pupil edge
  - pigment deposit on anterior surface
  - transillumination defect.

**Lens:**
- pseudoexfoliation material
- lens thickness and opacity
- phacodonesis
- glaukomflecken.

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**Gonioscopy**

*(See Appendix 3a: Gonioscopy)*

**Why?**
Detect angle closure, occludable and secondary glaucomas.

**What to look for?**
Angle width and characteristics (see below).

**When?**
- Initially for all.
- Regularly for angle closure patients.

**How to perform gonioscopy?**
- See flow diagram *(figure 1.4).*
  - Low room illumination.
  - Good anesthesia.
  - Shortest slit practicable.
  - High magnification.
  - Dim slit illumination.
  - Set slit lamp on upper cornea, beam off-center 30–45 degrees 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

• Then turn beam 90 degrees and move on axis.

• Move to nasal side (temporal angle) then to temporal side (nasal angle).

• Record findings on goniogram.
  (see Appendix 3b: Goniogram/Gonioscopic chart)

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. You may need to wait a minute or so.

Figure 1.4: Gonioscopy flow diagram

Angle closure signs:
• peripheral anterior synechiae (PAS)

• pigment patches over trabecular meshwork (evidence of irido-trabecular contact)

• iris insertion above scleral spur.

Abnormal open angles:
• trabecular meshwork (pigment, new vessels, precipitates, abnormal iris processes)

• ciliary body (angle recession, cyclodialysis cleft)

• Schlemm’s canal (blood reflux).
Defines glaucoma.

- Disc size.
- Neuroretinal rim.
- Disc hemorrhage.
- Nerve fiber layer defect.
- Peripapillary atrophy (PPA).
- Vascular pattern.

Every visit.

Consider:

Non-glaucomatous optic neuropathies. Differentiate anterior ischemic optic neuropathy (AION) from glaucoma – especially giant cell arteritis.\textsuperscript{9,10} Both cause pale cupped disc and field loss.

- Slit lamp.
- Very thin, bright beam for disc measurement.
- Dimmer beam for clearer/artificial lenses.
- Indirect slit lamp lens (60–90D).
- Stereoscopic view (best when dilated – recommended if safe).
- Red-free (green) illumination may help assessment of nerve fiber layer.
- Direct ophthalmoscopy or slit lamp view through undimpled Koeppe lens if pupil small and not able to be dilated.

\textbf{Figure 1.5: Disc examination flowchart}

\begin{figure}
\centering
\includegraphics[width=\textwidth]{flowchart}
\caption{Disc examination flowchart}
\end{figure}
Table 1.6: Normal vertical cup–disc ratios for vertical disc diameter

<table>
<thead>
<tr>
<th>Disc diameter</th>
<th>Mean cup–disc ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤1.0</td>
<td>0.26</td>
<td>0.20 – 0.32</td>
</tr>
<tr>
<td>1.2</td>
<td>0.33</td>
<td>0.32 – 0.34</td>
</tr>
<tr>
<td>1.4</td>
<td>0.39</td>
<td>0.39 – 0.39</td>
</tr>
<tr>
<td>1.6</td>
<td>0.45</td>
<td>0.45 – 0.45</td>
</tr>
<tr>
<td>1.8</td>
<td>0.50</td>
<td>0.50 – 0.50</td>
</tr>
<tr>
<td>≥2.0</td>
<td>0.55</td>
<td>0.53 – 0.57</td>
</tr>
</tbody>
</table>

Disc recording

- Draw optic disc (large), rim, key vessels that define rim and peripapillary signs.
- Draw notches, shelving, loss to rim–clock hours.
- Record whether nerve fiber layer is visible and assess for wedge or slit defects.
- Record vertical cup–disc ratio in the narrowest part of the rim. Consider recording the rim–disc ratio at key parts of the rim.
- Record disc hemorrhages, baring of circumlinear blood vessels, blood vessels bayoneting.

*Tip: Disc margin is INSIDE the peripapillary scleral ring of Elschnig.*
*Appropriate lens magnification correction: Superfield 1.5x, 90D 1.3x, 78D 1.1x, 66D 1.0x.*

Disc size

- Disc size is extremely variable, large discs have large cup–disc ratios even though the area of the neuroretinal rim is normal. Therefore, a large cup–disc ratio may not necessarily be pathological. Conversely, pathological rim loss can be missed in a small disc especially if generalized.
- Disc size can also be measured by using the small size spot of a direct ophthalmoscope. This spot size can be used to estimate whether a disc is larger or smaller than average.
- Disc size can also be evaluated using more conventional photographic means with an overlay grid, as well as by optic nerve head analysis.
The most reliable way to detect glaucomatous optic nerve progression may be with serial optic nerve head photographs.

Repeated scanning laser imaging is a promising approach to detect change accurately.

These devices are expensive and scanning laser technology is still under development.

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

The neuroretinal rim

The rim is more important than the cup. The cup defines the inner edge of the rim where most signs of glaucoma appear.

The cardinal feature of glaucomatous optic neuropathy is a loss of tissue from the inner edge of the rim.

Features that should raise suspicion that glaucomatous damage has already occurred include:

- notching of the rim (especially to the disc margin)
- hemorrhage crossing the rim
- undercutting of the rim
- asymmetry of rim width between the eyes in the absence of asymmetry of disc size
- an abnormally thin rim in one or two sectors

An approximate rule is that a vertical cup–disc ratio of >0.7 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

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.
Table 1.7: Optic disc/NFL assessment

<table>
<thead>
<tr>
<th>Qualitative</th>
<th>Quantitative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direct ophthalmoscopy</td>
<td>Disc photography with digitalization</td>
</tr>
<tr>
<td>Slit lamp indirect ophthalmoscopy</td>
<td>Stereo disc photography with optic disc analysis</td>
</tr>
<tr>
<td>Disc photograph</td>
<td>Confocal laser scanning ophthalmoscopy (HRT)</td>
</tr>
<tr>
<td>Simultaneous stereophotography</td>
<td>Laser polarimetry (GDx)</td>
</tr>
<tr>
<td>Nerve fiber layer photography (red-free fundus photography)</td>
<td>Optical Coherence Tomography (OCT)</td>
</tr>
</tbody>
</table>

Why?  • Defines state of optic nerve function.  
• Defines visual impairment.  

What?  Automated perimetry.  

When?  When glaucoma is suspected on examination.  

How?  It is very important to understand the correct procedure for performing visual field testing. Users should read and be familiar with the perimeter manual.

Tips for better visual fields (see Appendix 4: How to optimize patient performance in subjective perimetry)

• The patient should be carefully instructed in a language they understand.  
• 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.  
• Field test performance usually improves over the first 2–3 tests.  
• Check pupil size and note any change.
Characteristics of glaucomatous visual field defects

- Asymmetrical across horizontal midline.*
- Located in mid-periphery* (5–25 degrees from fixation).
- Reproducible.
- Not attributable to other pathology.
- Clustered in neighboring test points (localized).
- Defect should correlate with the appearance of the optic disc and neighborhood.

* early/moderate cases

Other tests of optic nerve function

- Short wave length automated perimetry (blue on yellow perimetry)
- Frequency doubling perimetry

References:

1.2 Treatment Categories and Targets

**Why?**
Tailor treatment to severity of glaucoma, depending on disease stage and risk factors.

**What are the categories?**

**Group 1: Glaucoma with high risk of progressive visual loss**
Definite glaucomatous optic neuropathy (GON) with correlating visual field (VF) loss (moderate to advanced).
- Includes moderate to advanced normal pressure glaucoma (NPG).¹

**Group 2: Glaucoma with moderate risk of visual loss or glaucoma suspect with high risk of visual loss**
- Mild GON with correlating early VF loss.
- Mild to moderate NPG.²
- Ocular hypertension ≥30 mmHg with suspicious disc.³
- Primary angle closure with high intraocular pressure (IOP) or peripheral anterior synechiae (PAS).
- Angle neovascularization.

**Group 3: Glaucoma suspect at moderate risk of visual loss**
- Glaucoma-like disc appearance without detectable VF loss.
- Fellow of eye with established GON (exclude secondary unilateral glaucomas).⁴
- Ocular hypertension with suspicious disc.³

**Group 4: Glaucoma suspect with low risk of visual loss**

*More important:*
- ocular hypertension³,⁴
- older age³,⁴
- occludable angles (appositionally closed without PAS)
- pigment dispersion syndrome (PDS)
- pseudoexfoliation syndrome (PXFS)
- disc hemorrhage (DH)
- glaucoma suspect disc (GSD) – including disc asymmetry
- family history of glaucoma
- glaucoma gene(s).
Less important:
• steroid responder, steroid users
• myopia, peripapillary atrophy (PPA)
• diabetes mellitus
• uveitis
• systemic hypertension.

Multiple risk factors (RFs)
The presence of multiple RFs proportionally increases glaucoma risk and may elevate a patient to Group 3.

When?
Each visit.

How?
Modifiable mechanisms for RFs.

Objective
To maintain functional vision throughout the patient’s lifetime with minimal effect on quality of life.

Setting goals
Goal of intervention is risk factor reduction (RFR)
• IOP.
• Angle control.
• Treatment of predisposing disease/factors (diabetes mellitus, uveitis, steroids).

Rate of neural loss and life expectancy
• Treatment sets goals to preserve sight
• This should be a balance of the severity of the disease, the amount of neuronal function present and the life expectancy of the patient (the course of time 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 any glaucoma treatments
Stage of disease
Use the four treatment categories above.

Estimate rate of neural loss
Higher \(\rightarrow\) more aggressive RFR.

Severity of RFs
Higher or greater number \(\rightarrow\) more aggressive RFR.

Modifiers of goals
- Life expectancy.
- Ability to attend follow-up.
- Diseases that prevent accurate disc or field assessment.

Goals of angle control
- Deepen peripheral angle closure (AC).
- Iridotomy – reduce pupil block.
- Argon laser peripheral iridoplasty (ALPI) – flatten peripheral iris.
- Lens extraction (LE) – reduces pupil block, displaces iris posteriorly.
- Vitreous surgery.

IOP landmarks
Presenting (untreated) IOP.
IOP in fellow normal eye in unilateral secondary glaucoma.
Population mean and standard deviation IOP for normal eyes.

Target IOP
- Target IOP is based on the pressure reduction required to slow or halt disease progression
- When the target is achieved, the patient needs continued monitoring for structural and functional changes
- Target IOP needs to be individualized
- The benefits of further pressure reduction need to be weighed against the risks
Goals (target IOPs) in Group 1
Target pressure reduction of at least 30%, or close to episcleral venous pressure (7–12 mmHg if achievable safely).

Goals in Group 2
Target pressure reduction of at least 20%.

Goals in Group 3
Monitor closely for change. Treat if risk(s) increase(s) with target pressure reduction of at least 20%.

Group 4
Monitor, no treatment.

Goals of treating predisposing diseases
Prevent onset of GON by proper management of disease.

References:
Section 2
2.1 Initiation of Treatment

**Why should treatment be initiated?**

Glaucome is a progressive optic neuropathy: if left untreated, the patient may go blind.

**What should be treated?**

Assess patients as a whole with the aim of preservation of visual function. Intraocular pressure (IOP) is the only known causal risk factor\(^1\) and the only one that can be manipulated effectively.\(^2\)

Mechanisms that elevate IOP:
- primary unknown cause
- angle closure with or without glaucoma
- secondary glaucomas (see *Appendix 5: Secondary glaucomas – principles of management*).

**Multiple mechanisms for angle closure (refer to Appendix 6: Angle closure mechanisms)**

Angle closure is caused by different sites of blockage which can often occur at multiple levels simultaneously and/or sequentially:
- Site one: pupil block – iris bombé appearance
- Site two: anteriorly rotated ciliary processes that push the iris forward and/or thick peripheral iris – plateau iris configuration
- Site three: lens induced forward displacement of the iris – volcano configuration
- Site four: aqueous misdirection with accumulation of fluid in the vitreous pushing the entire lens–iris diaphragm forwards

**When to treat?**

In the presence or the likelihood of developing visual damage that will interfere with quality of life during the patient’s lifetime.

- Demonstrable functional and structural defect.
- Progressive structural defect.
- High risk of developing such damage.
- Anatomical disorder that leads to:
  - angle closure
  - angle closure glaucoma
  - occludable angle (absolute or relative)
    - angle closure in the fellow eye
    - confirmed family history of angle closure glaucoma
    - need for repeated dilated examinations
    - poor access to regular ophthalmic care
    - reported family history of angle closure glaucoma.
Treat the mechanism(s)

IOP reduction:
- medication(s)
- laser
- surgery.

Correct the abnormal anatomy:
- laser
- surgery.

(Once any angle closure component has been appropriately treated, the management is similar to open angle glaucoma).

Collaborate with colleague(s) to treat systemic problems.

References:


2.2 Medical Treatment

Why?

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

What?

Choice depends on mechanism of glaucoma as well as other factors (refer to Patient Assessment). For angle closure, medical treatment is only appropriate after peripheral iridotomy (PI).

Table 2.1: Efficacy, safety and dosing frequency of various drug classes

<table>
<thead>
<tr>
<th>Drug class</th>
<th>Daily Dosage</th>
<th>Efficacy</th>
<th>Side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Local</td>
</tr>
<tr>
<td>Adrenergic agonists</td>
<td>2x to 3x</td>
<td>++ to +++</td>
<td>++</td>
</tr>
<tr>
<td>β-blockers</td>
<td>1x to 2x</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Carbonic anhydrase inhibitors</td>
<td>Topical</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td></td>
<td>Systemic</td>
<td>+++</td>
<td>0</td>
</tr>
<tr>
<td>Cholinergics</td>
<td>3x to 4x</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>Hyperosmotic agents</td>
<td>Stat dose(s)</td>
<td>+++++</td>
<td>0</td>
</tr>
<tr>
<td>Prostaglandins and other lipid</td>
<td>1x</td>
<td>+++</td>
<td>+ to ++</td>
</tr>
<tr>
<td>receptor agonists*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Proprietary fixed combinations</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BB + CAI</td>
<td>2x</td>
<td>+++ to ++++</td>
<td>++</td>
</tr>
<tr>
<td>BB + PG</td>
<td>1x</td>
<td>++++ to ++++</td>
<td>+ to ++</td>
</tr>
<tr>
<td>BB + Pilo</td>
<td>2x</td>
<td>+++</td>
<td>+++</td>
</tr>
</tbody>
</table>

0 – rare
PG – Prostaglandins
BB – β-blockers
Pilo – Pilocarpine
CAI – Carbonic anhydrase inhibitor
*excluding unoprostone
### Table 2.2: Mechanism of action of different drug classes

<table>
<thead>
<tr>
<th>Mechanism of action</th>
<th>Drug class</th>
<th>Preparations</th>
</tr>
</thead>
</table>
| Reduction of aqueous inflow | Adrenergic agonists | • Brimonidine  
|                     | β-blockers                      | • Apraclonidine  
|                     | Carbonic anhydrase inhibitors   | • Timolol  
|                     |                                | • Levobunolol  
|                     |                                | • Carteolol  
|                     | Non-selective                  | • Betaxolol  
|                     | Systemic                      | • Acetazolamide  
|                     | Topical                        | • Methazolamide  
|                     |                                | • Dichlorphenamide  
|                     | Increase in aqueous outflow    | • Dorzolamide  
|                     | Cholinergics                   | • Brinzolamide  
|                     | • Increase trabecular outflow  | • Pilocarpine  
|                     | Prostaglandins and other lipid receptor agonists | • Carbachol  
|                     | • Increase uveoscleral outflow | • Latanoprost  
|                     |                                | • Travoprost  
|                     |                                | • Bimatoprost  
|                     |                                | • Unoprostone  

<table>
<thead>
<tr>
<th>Preparations by class</th>
<th>Contraindications*</th>
<th>Common drug interactions</th>
<th>Local side effects</th>
<th>Systemic side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Adrenergic agonists</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brimonidine</td>
<td>• Monoamine oxidase inhibitor therapy</td>
<td>CNS depressants (alcohol, barbiturates, opiates, sedatives, or anesthetics), tricyclic antidepressants</td>
<td>Burning, stinging, blurring, foreign-body sensation, itching, hyperemia, follicular conjunctivitis</td>
<td>Oral dryness, headache, fatigue, drowsiness</td>
</tr>
<tr>
<td>Apraclonidine</td>
<td>• Less than 2 years old</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| **β-blockers (non-selective)** | | | | |
| Timolol | • Absolutely contraindicated in bronchial asthma, chronic obstructive pulmonary disease, bradycardia, heart block | Systemic β-blockers, calcium channel blockers | Burning, stinging, photophobia, itching, tearing, decreased corneal sensitivity, hyperemia, punctate epithelial keratopathy | Bronchospasm, hypotension, bradycardia, heart block, mask hypoglycemia, adversely affects lipid profile (except carteolol), loss of libido, fatigue, aggravation of myasthenia gravis, depression, memory impairment, reduced exercise tolerance, increased falls in the elderly |
| Levobunolol          | • To be used cautiously in cardiac failure |                          |                    |                      |
| Carteolol            | | | | |
| Metipranolol         | | | | |

| **β-blockers (selective)** | | | | |
| Betaxolol             | Relatively contraindicated in bronchial asthma, chronic obstructive pulmonary disease, bradycardia, heart block, cardiac failure | As for non-selective β-blockers with wider safety margin | As for non-selective β-blockers with wider safety margin | |

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

*Known hypersensitivity to any component of the product, pregnancy*
Please refer to manufacturer’s summary of product characteristics before prescribing

*Known hypersensitivity to any component of the product, pregnancy

<table>
<thead>
<tr>
<th>Preparations by class</th>
<th>Contraindications*</th>
<th>Common drug interactions</th>
<th>Local side effects</th>
<th>Systemic side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Carbonic anhydrase inhibitors</strong>&lt;br&gt;(topical)</td>
<td>Relatively contraindicated with compromised corneal endothelium, sulfonamide allergy</td>
<td></td>
<td>Burning, stinging, itching, punctate epithelial keratopathy, blepharoconjunctivitis</td>
<td>Bitter taste</td>
</tr>
<tr>
<td>Dorzolamide&lt;br&gt;Brinzolamide</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Carbonic anhydrase inhibitors</strong>&lt;br&gt;(systemic)</td>
<td>Sulfonamide allergy, renal stones/failure, respiratory/metabolic acidosis, hypokalemia</td>
<td>Steroids, diuretics, digoxin</td>
<td>Transient myopia</td>
<td>Fatigue, lethargy, anorexia, gastrointestinal upset, weight loss, paresthesia, taste disturbance, Stevens-Johnson Syndrome, blood dyscrasias, renal stones, hypokalemia</td>
</tr>
<tr>
<td>Acetazolamide&lt;br&gt;Methazolamide&lt;br&gt;Dichlorphenamide</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Cholinergics</strong></td>
<td>Uveitic, neovascular and lens induced glaucomas&lt;br&gt;Post-drainage surgery&lt;br&gt;Aqueous misdirection syndrome</td>
<td></td>
<td>Pain, dimness of vision, blurring, myopic shift, retinal detachment, aggravate pupillary block</td>
<td>Headache, salivation, lacrimation, urinary frequency, diarrhea, abdominal cramps</td>
</tr>
<tr>
<td>Pilocarpine&lt;br&gt;Carbachol</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
## Preparations by class

<table>
<thead>
<tr>
<th>Preparations by class</th>
<th>Contraindications*</th>
<th>Common drug interactions</th>
<th>Local side effects</th>
<th>Systemic side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hyperosmotic agents</strong></td>
<td>Heart failure, pulmonary edema, renal failure</td>
<td>Chronic pilocarpine use may reduce efficacy of these agents</td>
<td>Blurred vision, burning, stinging, conjunctival hyperemia, foreign-body sensation, itching, increased iris/periorbital skin pigmentation, lash growth, punctate epithelial keratopathy, cystoid macular edema, reactivation of herpetic infection</td>
<td>Headaches, unpleasant taste, heart failure, pulmonary edema, death</td>
</tr>
<tr>
<td>Mannitol</td>
<td>Caution with hypertension</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| **Prostaglandins and other lipid receptor agonists** | Relatively contraindicated in the presence of active inflammatory ocular conditions, cystoid macular edema | Chronic pilocarpine use may reduce efficacy of these agents | Blurred vision, burning, stinging, conjunctival hyperemia, foreign-body sensation, itching, increased iris/periorbital skin pigmentation, lash growth, punctate epithelial keratopathy, cystoid macular edema, reactivation of herpetic infection | |
| Latanoprost | Caution following complicated intraocular surgery | | | |
| Travoprost | | | | |
| Bimatoprost | | | | |
| Unoprostone | | | | |

| **Proprietary fixed combinations** | As for individual components | | | |

Please refer to manufacturer's summary of product characteristics before prescribing

*Known hypersensitivity to any component of the product, pregnancy*
Choose the most appropriate medication

- Greatest chance of reaching target.
- 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 2–4 weeks.
- Assess side effects.
- If acceptable and effective, make treatment bilateral.

If response inadequate to achieve target pressure: switch before adding

- Switch to different class of medication (switching within lipid receptor agonist class may be useful, but compliance and regression to the mean need to be considered).
- Use the one-eyed therapeutic trial again.

Use more than one agent only if each has demonstrated efficacy but insufficient to reach target

- Apply this principle also to the fixed combinations.

Maximize the likelihood of compliance

- Establish therapeutic alliance with the patient and 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 of drop instillation

- Demonstrate the preferred method including punctal occlusion and eyelid closure for at least 3 minutes (‘double DOT’ – Digital Occlusion Technique, Don’t Open Technique).
- Ensure the patient can do it.
- If two or more drops being instilled, wait at least 5 minutes between drops.
- Provide educational material.

Suggested reading:

Figure 2.3: Medical Treatment Algorithm

Drug 1
Maximize compliance

- Reaches target? Side effects none / tolerable?
  - YES
    - Continue treatment and monitor
  - NO
    - Partial / side effects
      - Stop
      - Hold in reserve
    - Ineffective
      - Discard
      - Laser or surgery

Drug 2
Maximize compliance

- Reaches target? Side effects none / tolerable?
  - YES
    - Continue treatment and monitor
  - NO
    - Partial / side effects
      - Stop
      - Hold in reserve
    - Ineffective
      - Discard
      - Laser or surgery

Drug 3 or more (if appropriate)
Maximize compliance

- Reaches target? Side effects none / tolerable?
  - YES
    - Continue treatment and monitor
  - NO
    - Partial / side effects
      - Stop
      - Hold in reserve
    - Ineffective
      - Discard
      - Laser or surgery


2.3 Laser Treatment

Laser treatment for open angle glaucoma
- Outflow enhancement: laser trabeculoplasty.
- Inflow reduction: cyclophotocoagulation (usually for end stage).

Laser treatment for angle closure (± glaucoma)
- Pupillary block relief: laser iridotomy.
- Modification of iris contour: laser iridoplasty (gonioplasty).
- Inflow reduction: cyclophotocoagulation (usually for end stage).

Why?
- Relatively effective.
- Relatively non-invasive.

What?
Laser treatment to trabecular meshwork to increase outflow.

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

How?
Pre-laser management
- Explain the procedure.
- To reduce post-treatment intraocular pressure (IOP) spike or inflammation, consider 1% apraclonidine¹ or 0.2% brimonidine² and/or 2–4% pilocarpine and/or β-blocker and/or steroid drops before the procedure.
- Topical anesthesia.

Laser
- Argon green or blue–green (ALT).
- Diode (DLT).
- Frequency doubled Nd-YAG ("A"LT).
- Selective laser (SLT).

Lens*
- Goldmann gonioscopy lens.
- Ritch trabeculoplasty lens.
- CGA© LASAG CH.

*should be coated to minimize reflection and hazard to observers
Placement of laser spots
Between pigmented and non-pigmented trabecular meshwork (see Appendix 7a: Argon laser trabeculoplasty).

Parameters
- Power: 300–1200 mw depending on the reaction.
- Spot size: 50 µm (for Argon), 75 µm (for diode), 400 µm (for SLT).
- Duration: 0.1 sec (for Argon & diode), 3 ns for SLT.
- Number of burns: 30–50 spots evenly spaced over 180 degrees. Treat the remaining 180 degrees sequentially, or at the same time, as required.

Complications
- Temporary blurred vision.
- IOP spike with possible visual field loss.
- Transient iritis.
- Peripheral anterior synechiae if placement of burns is too posterior or post-laser inflammation control is not effective.
- Endothelial burns of treatment too anterior.
- Chronic increase in IOP.

Post-laser management
- Continue any current medical treatment.
- Especially if IOP spike prevention treatment is not available, re-check IOP at 1–6 hours after laser and again 24–48 hours.
- Topical steroid qid for 4–14 days (consider omitting with SLT).

Closer monitoring is suggested in certain cases
- Advanced glaucoma with severe field loss.
- One-eyed patient.
- 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.
**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?**
- Angle closure.
- Angle closure glaucoma.
- Occludable angle (absolute):
  - angle closure in the fellow eye
  - confirmed family history of angle closure glaucoma.
- Occludable angle (relative):
  - need for repeated dilated examinations
  - poor access to regular ophthalmic care.

**How?**
**Pre-laser management**
- Explain the procedure.
- Instill 2% or 4% pilocarpine.
- To reduce post-treatment IOP spike/inflammation, consider 1% apraclonidine¹ or 0.2% brimonidine² and/or β-blocker and/or oral carbonic anhydrase inhibitor and/or steroid drops before the procedure.
- Topical anesthesia.
- Topical glycerin, if the cornea is edematous.
- Superior 1/3 of iris (beneath upper lids) desirable.

**Laser:**
- Nd-YAG.
- Argon or krypton.

**Laser parameters for Nd-YAG laser**
- Energy: 2–5 mJ, use minimum energy, 1–3 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
Complications

- Temporary blurring of vision.
- Corneal epithelial and/or endothelial burns with Argon (latter aggravated by bubble formation and contact with endothelium).
- Intra-operative bleeding with Nd-YAG.
- IOP spikes.
- Post-operative inflammation.
- Posterior synechiae.
- Closure of iridotomy.
- Failure to penetrate.
- Localized lens opacities.
- Rarely: retinal damage, cystoid macular edema, malignant glaucoma, endothelial decompensation.

Laser parameters for Argon laser

Preparatory stretch burns:
- Spot size: 200–500 µm
- Exposure time: 0.2–0.5 sec
- Power: 200–600 mW

Penetration laser burns:
- Diameter: 50 µm
- Exposure time: 0.02 sec
- Power: 800–1000 mW

For pale blue or hazel iris:

First step, to obtain a gas bubble:
- Diameter: 50 µm
- Exposure time: 0.5 sec
- Power: 1500 mW

Second step, penetration through the gas bubble:
- Diameter: 50 µm
- Exposure time: 0.05 sec
- Power: 1000 mW

For thick dark brown iris: (chipping technique)
- Diameter: 50 µm
- Exposure time: 0.01–0.02 sec
- Power: 1500–2500 mW

Choose and modify parameters depending on individual response.
Laser Treatment

Post-laser management
- Particularly if IOP spike prevention treatment is not available, re-check IOP at 1–6 hours after laser and again at 24–48 hours.
- Topical steroid at least 4–6 times/day for 4–14 days depending on inflammation.
- Verify the patency of the peripheral iridotomy (PI).
- Repeat gonioscopy.
- Pupillary dilatation to break posterior synechiae when suspected.

Iridoplasty (gonioplasty)

Why?
- Reasonably effective.
- Relatively non-invasive.
- Adjunct to peripheral iridotomy.

What?
Laser treatment to contract the peripheral iris:
- to flatten the peripheral iris
- to widen the anterior chamber angle inlet.

When?
- Angle remains occludable following peripheral iridotomy (e.g., plateau iris).
- Help break an attack of acute angle closure.
- Facilitate access to trabecular meshwork for laser trabeculoplasty.
- Minimize the risk of corneal endothelial damage during iridotomy.

How?

Pre-laser management
- Explain the procedure.
- Instill 2% or 4% pilocarpine.
- To reduce post-treatment IOP spike/inflammation, consider 1% apraclonidine® or 0.2% brimonidine® and/or β-blocker and/or oral carbonic anhydrase inhibitor and/or steroid drops before the procedure.
- Topical anesthesia.
- Topical glycerin, if the cornea is edematous.

Procedure
- Argon green or blue–green.
- Diode laser.

Lens
- CGI© LASAG CH.
- Goldmann 3-mirror lens.
- Placement of laser spot: aim at the most peripheral location, avoid corneal arcus.
**Laser parameters**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Power</td>
<td>200 to 400 mW according to the reaction</td>
</tr>
<tr>
<td>Spot size</td>
<td>200–500 µm</td>
</tr>
<tr>
<td>Duration</td>
<td>0.2–0.5 sec</td>
</tr>
<tr>
<td>Number of burns</td>
<td>30 to 50 applications over 360 degrees leaving at least 1–2 spot diameters between spots</td>
</tr>
</tbody>
</table>

**Endpoint**

- Iris contraction with peripheral anterior chamber deepening and more visible angle in line with the laser applications.

**Complications**

- Mild iritis.
- Corneal endothelial burns.
- IOP spikes.
- Peripheral anterior and/or posterior synechiae.

**Post-operative treatment**

- If IOP spike prevention treatment is not available, check IOP within 1–6 hours and then 24–48 hours depending on the status of the patient.
- Topical corticosteroids 4–6 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 angle closure that might necessitate further intervention.
- Pupillary dilatation to break posterior synechiae when suspected.

---

**Cyclophoto-coagulation**

**Why?**

Preferable to cyclocryoablation or cyclodiathermy.

**What?**

Reduces aqueous production by 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?**

**Pre-laser management**

- Explain procedure.
- Topical and sub-Tenon’s or retro/peri-bulbar anesthesia.
- General anesthesia when indicated.
Techniques

• Transpupillary.
• Transscleral.
• Endolaser.
• Conservative, incremental applications avoiding 3 and 9 o’clock positions.

Non-contact transscleral Nd-YAG laser
Lasag Microruptor 2 using thermal mode.

<table>
<thead>
<tr>
<th>Laser parameters</th>
</tr>
</thead>
<tbody>
<tr>
<td>Power: 8–10 J and with maximum defocusing, 1–1.5 mm posterior to the limbus</td>
</tr>
<tr>
<td>Number of burns: 32 over 360 degrees</td>
</tr>
</tbody>
</table>

Contact transscleral Nd-YAG laser
Continuous wave Nd-YAG laser with transscleral contact probe.

<table>
<thead>
<tr>
<th>Laser parameters</th>
</tr>
</thead>
<tbody>
<tr>
<td>Power: 4–7 J</td>
</tr>
<tr>
<td>Duration: 0.5–0.7 sec</td>
</tr>
<tr>
<td>Number of burns: 30–40 over 360 degrees</td>
</tr>
<tr>
<td>Location: 1.0–2.0 mm from limbus</td>
</tr>
</tbody>
</table>

Contact transscleral diode laser
Diode laser with transscleral contact probe (see Appendix 7b: Contact transscleral diode laser).

<table>
<thead>
<tr>
<th>Laser parameters</th>
</tr>
</thead>
<tbody>
<tr>
<td>Power: 1.0–2.5 W</td>
</tr>
<tr>
<td>Duration: 0.5–2.0 sec</td>
</tr>
<tr>
<td>Number of burns: 20–40 over 180–360 degrees</td>
</tr>
<tr>
<td>Location: 1.0–2.0 mm from limbus</td>
</tr>
</tbody>
</table>

Endolaser

• Diode endoscopic laser.
• Argon or krypton laser.

<table>
<thead>
<tr>
<th>Laser parameters</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depends on laser system used, consult the instruction manual and clinical updates</td>
</tr>
</tbody>
</table>
Complications

- Pain.
- Persistent inflammation.
- Loss of visual acuity.\textsuperscript{4,5}
- Hypotony.\textsuperscript{6}
- Scleral thinning.\textsuperscript{7,8}
- Macular edema.
- Retinal detachment.\textsuperscript{9}
- Aqueous misdirection syndrome.\textsuperscript{10}
- Phthisis.\textsuperscript{11}
- Sympathetic ophthalmia.\textsuperscript{12}
- Failure to control IOP – multiple procedures may be needed.

Post-operative management

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

References:


2.4 Surgery

Open angle glaucoma
• Outflow enhancement: penetrating and non-penetrating filtering surgery.
• Glaucoma drainage device.

Chronic angle closure glaucoma
• Pupillary block relief: iridectomy.
• Outflow enhancement: trabeculectomy.
• Widening of anterior chamber angle inlet: lens extraction.
• Angle surgery: goniosynechialysis.
• Glaucoma drainage device.

Acute angle closure (± glaucoma)
• Pupillary block relief: iridectomy.
• Outflow enhancement: trabeculectomy.
• Angle surgery: goniosynechialysis.
• Widening of anterior chamber angle inlet: lens extraction.

Childhood glaucoma
• Angle surgery: goniotomy and trabeculotomy.
• Outflow enhancement: trabeculectomy with or without trabeculotomy.
• Glaucoma drainage device.

Why?
• Reasonably effective.¹²
• Reasonably safe.
• Widely available.

What?
• Penetrating filtering surgery:
  – trabeculectomy
  – trabeculectomy with anti-fibrotics.
• Non-penetrating surgery (with or without implant):
  – deep lamellar sclerectomy
  – viscocanalostomy.
• Glaucoma drainage implant.
• Surgical iridectomy: largely replaced by laser iridotomy (refer to Laser Treatment).
• Lens extraction for lens-induced angle closure glaucomas.
• Goniosynechialysis.
• Vitrectomy for aqueous misdirection.


Asia Pacific Glaucoma Guidelines

Childhood surgery

- Goniotomy (primary treatment – to be performed at specialist centers).
- Trabeculotomy (primary treatment – to be performed at specialist centers).

When?

- Failed medical and/or laser treatment.¹
- Anticipated failure of medical/laser treatments (e.g., very high IOP).²,³
- Patient preference.
- Other forms of therapy are inappropriate: compliance, side effects and/or socioeconomic problems.

How?

Pre-operative assessment
Identify risk factors for failure and treat where applicable.⁴
- Asian, African, Hispanic ethnicity.
- Previous surgery.
- Young age.
- Aphakia.
- Pseudophakia.
- Active ocular inflammation.
- Prolonged use of topical glaucoma medications.⁵
- Tendency to form keloids.
- Neovascular glaucoma.

Surgical technique

- Select appropriate technique.

Enhancement of surgery

- Use of anti-fibrotic agents:
  - intra-operative⁴
  - post-operative⁴,⁶,⁷
- Use of anti-inflammatory agent:
  - systemic corticosteroids⁸,⁹
  - other agents (e.g., NSAIDs)
- Use of laser suture lysis or releasable sutures¹⁰

Post-operative management

- Examine first post-operative day.
- Topical steroids for 6–12 weeks.
- Topical antibiotics for 14 days or more.
- Cycloplegics for 2–6 weeks.
- Analgesics.
- Intensive individualized post-operative care.
Surgery

Use of anti-fibrotics in surgery

**Why?**
Scarring is the major cause for failure following filtration surgery. Anti-fibrotics have been shown to inhibit scarring and to increase the success rate.

**What?**
Commonly used: 5-Fluorouracil (FU), Mitomycin-C (MMC)
Others: \(\beta\) radiation.

**When?**
- For high risk of failure following standard filtering surgery. This includes repeat surgery, neovascular glaucoma, glaucoma in uveitis, glaucoma in aphakia, younger age, black race.
- In primary surgery, especially where a lower target pressure is required.\(^{11, 12}\)
- To increase the success rates with artificial drainage devices.
- With needling of a failed filter.

In these instances, the enhanced success rates with anti-fibrotics may make the complications associated with their use more acceptable.

**How?**

**A. Application during surgery:**

**Dose**
- Sponge soaked in MMC (varying doses of 0.2 to 0.4 mg/ml applied for 1–5 minutes), 0.4 mgs/mL for 1 minute for primary surgery and the same concentration for 3 minutes for poor prognosis filters.
- Sponge soaked in 5-FU (50 mgs per mL) for 1–3 minutes.
- For subconjunctival use prior to needling of a bleb: a mixture of 0.01 ml of MMC (0.4 mg/mL) and 0.02 mL of bupivicaine with epinephrine.\(^{13}\)

**Mode of application**
- Sponge placed under the conjunctiva. A little extra dissection allows multiple sponges and a large surface area treatment.\(^{14}\)
- If used prior to needling, 0.01 mL of MMC (0.4 mg/mL) and 0.02 mL of bupivicaine with epinephrine can be injected subconjunctivally superior to the bleb. A needle is used to perforate the area of subconjunctival fibrosis and re-establish flow.

**Removal of residual drug when applied during surgery**
- Copious irrigation of the treated area with balanced salt solution, normal saline or ringer lactate solution.

**B. Post-operative application:**

5-FU is also used as post-operative injections of 5 mg/0.1 mL for up to 4 post-operative weeks. The injection may be given alongside or behind the bleb, or sometimes 90 to 180 degrees away using preferably a 30 gauge needle. The number of injections is titrated according to the appearance of the bleb. Care is taken to avoid spillage on the cornea. The conjunctiva
over the area of injection may be tamponaded with a cotton bud for about 1 minute after the injection.

*Note: The use of anti-fibrotic agents can be associated with sight threatening complications and they must be used with caution. Use of an algorithm developed by those experienced in the use of such agents is desirable.*

---

### Glaucoma drainage implants

**Why?**  
An implant allows aqueous to flow from the anterior chamber into a maintained episcleral space from where it can be absorbed into surrounding blood vessels.

**What?**  
Molteno, Ahmed, Baerveldt.

**When?**  
Where there is a very high risk of failure of trabeculectomy even with anti-fibrotics – these eyes invariably have severe, refractory glaucoma.

- Previously failed trabeculectomies with anti-fibrotics.
- 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 (e.g., irido-corneal endothelial syndrome, neovascular glaucoma).

**How?**  
This surgery, and the management of the patient post-operatively is complicated. An ophthalmologist with appropriate training and experience should perform it.

Depending on the surgeon’s preference, one of the drainage implants is positioned on the scleral surface, usually in the superotemporal or superonasal quadrant (or both for a two-plate implant) and connected to the anterior chamber by an attached tube.

To avoid immediate post-operative hypotony, the tube must be occluded either by a valve, or by a suture (venting slits may be needed to avoid high IOP until the suture is removed or dissolves). A hypertensive phase is common and requires medical control as well as anti-fibrotic therapy.

**Complications from these implants**

- Failure to control IOP.
- Hypotony.
- Corneal decompensation.
- Peripheral anterior synechiae.
- Pupillary distortion.
- Cataract.
- Endophthalmitis and erosion of the tube and/or plate(s).
Cataract and glaucoma are both common conditions and which often co-exist. A recent review has assessed current surgical management.\(^{20}\)

**Table 2.4: Evidence for surgical management of cataract and glaucoma\(^{20}\)**

<table>
<thead>
<tr>
<th>Review comments</th>
<th>Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>MMC (but not 5-FU) has a small benefit (2–4 mmHg) for ECCE*-trabeculectomy</td>
<td>B</td>
</tr>
<tr>
<td>Two-site surgery provides slightly lower IOP (1–3 mmHg) than one-site surgery</td>
<td>C</td>
</tr>
<tr>
<td>IOP is lowered more (1–3 mmHg) by phacoemulsification than by ECCE in combined procedures</td>
<td>C</td>
</tr>
<tr>
<td>Two-staged versus combined procedures</td>
<td>I</td>
</tr>
<tr>
<td>Alternative glaucoma procedures versus trabeculectomy in combined procedures</td>
<td>I</td>
</tr>
</tbody>
</table>

Evidence grades:
- A – strong
- B – moderate
- C – weak
- I – insufficient

For the surgical management of coexisting cataract and glaucoma, there is some evidence of efficacy for using MMC.

*Extracapsular cataract extraction.
References:


Section 3
3.1 Follow-up

**Why?**

The aim of follow-up is:
- to detect progression
- to detect effects of treatment
- to detect any change in health that may affect the glaucoma management plan.

**What?**

The follow-up process starts with the management plan made at the initiation of therapy. At the follow-up visits the doctor should:
- briefly discuss the patient’s subjective well being and visual function
- reassess risk factors: especially intraocular pressure (IOP) and gonioscopic change(s)
- reassess structure and function of the optic nerve
- estimate rate of (any) progression
- identify adverse effect(s) of treatment
- assess compliance
- identify change(s) in current medical and ophthalmological problems
- discuss quality of life issue(s)
- reinforce appropriate patient information:
  - revise management
  - plan follow-up.

**When?**

The more severe the damage, the worse the risk factors, the sooner should be the follow-up.

**Patient’s subjective wellbeing and visual function**

- Patients will often wish to tell the doctor how they feel their condition has (or has not) changed.
- This discussion helps build a good doctor–patient relationship.

Subjective changes in vision with glaucoma are rare, but in advanced disease, changes in the following qualities of vision may indicate a deterioration of glaucomatous optic neuropathy (GON):
- night vision
- dark adaptation
- glare
- stereopsis
- acuity (high and low contrast)
- missing pieces of vision.
Intraocular pressure (IOP)

- IOP is the only currently 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 all fluctuations.
- Repeat unexpected readings at the same visit and soon after.

Causes of change in IOP at follow-up

Increased IOP:
- progression of disease
- gradual loss of efficacy of a drug (tachyphylaxis)
- poor compliance

Reduced IOP:
- therapeutic effect

Reduced or increased IOP:
- variation during the day and between days
- change in systemic medications

Gonioscopic changes

- Maintain baseline examination conditions.
- Perform gonioscopy regularly in patients with angle closure and periodically in patients with open angles.
- Look for increased appositional and/or synechial closure.
- Pupil size changes have dynamic effects on the angle.
- Look for change in angle width, synechiae, and pigmentation.
Optic disc

Progression of GON usually occurs over a long period, which can make the detection of change difficult.

The occurrence of the following indicate GON progression (refer to Appendix 8: Glaucomatous optic neuropathy):

- disc hemorrhage
- focal rim notching
- change in vessel position
- wedge-type nerve fiber layer defects
- generalized rim thinning
- increased cup–disc ratio.

Where baseline optic disc photographs and serial photography are available, detection of these changes is substantially enhanced. If photographs are 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.

Visual field

Change is frequent in perimetry. Usually only a small proportion is owing to GON progression.

Causes of change

- Learning – field performance usually improves over the first few attempts.
- Reliability changes, poor concentration may cause general depression, look for false negatives.
- False negative errors may indicate progression. False positive errors reflect poor reliability and may mask progression. Fixation losses may reflect poor reliability and mask progression, or may reflect technical errors.
- Progression of disease.
- Cataract – causes generalized depression and masks relative scotomas.
- Pupil size changes – miosis causes generalized field depression; minimum 3 mm recommended.
- Retinal disease (e.g., vein occlusion, macular degeneration, significant diabetic retinopathy).
- Retinal laser.
- Miscellaneous artifacts.
- Change in measurement strategy (Fastpac vs. Full Threshold vs. SITA).
Detecting progression

Progression is characterized by:
- widening or deepening of an existing scotoma
- development of a new glaucomatous scotoma
- occasionally generalized field depression (although this is usually caused by media opacity or miosis).

(Refer to Appendix 9: Field progression print-outs).

Changes in visual field should be confirmed by at least one or more repeat tests.

There is a close correlation between glaucomatous changes in structure of the optic disc and consequent visual field loss. However, there may be considerable variation in morphology of a “normal” disc, and in patients’ ability to perform visual field tests adequately.

Changes should be regarded skeptically until the deviation exceeds the standard deviation of serial measurements.

Adverse effects of treatment should be actively sought using general and specific questioning. These include:
- General effects: self-rated health, feelings about/attitude towards treatment.
- Systemic effects: respiratory, cardiovascular, digestive, neurological.
- Local effects: stinging/burning, blurring, itching, redness.

- The patient's quality of life (QOL) should be estimated and the impact of the glaucoma management on QOL assessed.
- This forms part of the assessment of burden of disease and burden of treatment.
**SEAGIG DECISION SQUARE FOR GON**

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

**Table 3.1: SEAGIG Decision Square for GON**

<table>
<thead>
<tr>
<th>Risk</th>
<th>Disease status</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Stable</td>
</tr>
<tr>
<td>Increased</td>
<td>+</td>
</tr>
<tr>
<td>Uncertain</td>
<td>Reassess risk</td>
</tr>
<tr>
<td>Stable</td>
<td>–</td>
</tr>
</tbody>
</table>

**Risk factors***

The following factors confer a higher risk of loss of vision from glaucoma:

- high or rising IOP
- any appositional angle closure
- any peripheral anterior synechiae (PAS), or an increase in PAS, if seen before
- longer life expectancy.

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

**Disease progression**

- Stable disease indicates no change in optic disc or visual field status
- Uncertain disease status indicates a change in visual field that is not consistent with the status of the disc.
- Progressing disease indicates changes consistent with glaucoma in the optic disc and visual field.

Add + if the rate of progression appears rapid.
THE GLAUCOMA LIFE STORY (GLS)

The determinants of the GLS are:
- state of damage
- life expectancy
- rate of progression.

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 is the key factor in 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 aging 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 color in the graph represents the risk of blindness from glaucoma. The green area represents low risk and red represents high risk.
Follow-up timing

The target pressure is the IOP at which it is believed the patient can retain vision for the rest of his/her life. However this needs to be checked from time to time.

Follow-up timing is determined by the treatment regime if this has changed. If the patient is stable the timing is determined mainly by the extent of damage: for glaucoma suspect, 6–24 months; for mild damage, 6–12 months; moderate damage, 4–6 months; severe damage, 1–4 months.

References:


Case Detection

The Epidemiology section outlines our current understanding of the magnitude of glaucoma blindness in Asia. This large burden raises the question of screening the population to detect and initiate prompt treatment for glaucoma.

The World Health Organization recommends that certain defined criteria be fulfilled before any population-based screening is undertaken. 1

• The disease must be an important public health problem.
• There must be a recognizable latent or early stage, during which persons 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 patients 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 of the disease.
• The cost of case finding must be economically balanced in relation to possible expenditure on medical care as a whole.

Other questions that need to be asked before embarking on any screening program are listed below.2

1. Does early diagnosis lead to improved clinical outcomes in terms of visual function and quality of life?

2. Can the health system cope with the additional clinical time and resources required to confirm the diagnosis and provide long term care for those who screen positive for a chronic disease such as glaucoma?

3. Will the patients in whom early diagnosis is achieved comply with subsequent recommendations and treatment regimens?

4. Are the cost, accuracy and acceptability of the screening tests adequate for our purpose?

Glaucoma fits some of the criteria required for screening but others 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.
Detecting early glaucoma is ideal, but requires a sensitive test that leads to many false positives. For screening, a test should have a reasonably high sensitivity with as high specificity as possible. Prevent Blindness America suggests 95–98% specificity and 85% sensitivity for moderate-to-severe glaucoma.4

<table>
<thead>
<tr>
<th>Population-based screening</th>
<th>Case detection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients are sought, there is an implied pledge that the process is going to make them better but this may not be true.</td>
<td>Relies on detection of glaucoma in patients who present themselves for other complaints. Patients seek us out; we treat them to the best of our ability but without the guarantee of a cure.</td>
</tr>
<tr>
<td>Obliged to establish a diagnosis and treatment using sophisticated techniques, which may not be widely available for general screening. Without the requisite equipment, trained personnel and infrastructure, screening is not justified.</td>
<td>Based on detection of glaucoma in ‘at risk’ patients where the prevalence of glaucoma is higher. Thus, most of the tests described below – tonometry, ophthalmoscopy and perimetry – have a reasonably high positive predictive value.</td>
</tr>
<tr>
<td>The patients who turn out to be false positives carry the burden of being labelled. The consequences may be severe.³</td>
<td>Patients who actually have the disease but have tested negative are given a clean bill of health, which can be dangerous.</td>
</tr>
<tr>
<td>Patients who actually have the disease but have tested negative are given a clean bill of health, which can be dangerous.</td>
<td>Many countries in the region may not have the requisite infrastructure to follow-up and categorize test positives or even treat them appropriately.</td>
</tr>
<tr>
<td>The general physician can play an important role in the diagnosis of open angle glaucoma. Ophthalmoscopy and frequency doubling perimetry are feasible in a physician’s office.</td>
<td>Screening cannot be a one-time affair, and even developed countries may ill afford to screen the population at large for glaucoma and handle the burden of further testing, treating and follow-up.</td>
</tr>
<tr>
<td>Most elderly patients, diabetics and myopes (all at risk for glaucoma), often visit the offices of ophthalmologists and optometrists for other eye care needs. Follow-up is also easier.</td>
<td>Most elderly patients, diabetics and myopes (all at risk for glaucoma), often visit the offices of ophthalmologists and optometrists for other eye care needs. Follow-up is also easier.</td>
</tr>
</tbody>
</table>
Primary open angle glaucoma (POAG)

According to population-based studies in Western countries, the prevalence of POAG is five times that of PACG. However, half the glaucoma blindness in the world is caused by angle closure.

As approximately 75% of subjects with PACG in Asia have optic nerve damage, screening strategies that detect functional damage in POAG may also be suitable for PACG. Such tests will not detect eyes without functional damage, nor eyes at risk for angle closure.

- Tonometry will only detect angle closures that have a raised IOP.
- Optic disc examination and perimetry will only detect angle closure that has damaged the disc or visual field.

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

---

<table>
<thead>
<tr>
<th>Test</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tonometry</td>
<td>At cut off of &gt;21mm:</td>
<td></td>
<td>Poor sensitivity and specificity</td>
</tr>
<tr>
<td></td>
<td>47.1%&lt;sup&gt;5&lt;/sup&gt;</td>
<td>92.4%&lt;sup&gt;5&lt;/sup&gt;</td>
<td>Half of the patients with POAG have IOPs &lt;22 mmHg at a single screening</td>
</tr>
<tr>
<td>Automated perimetry</td>
<td>97%&lt;sup&gt;6&lt;/sup&gt;</td>
<td>84%&lt;sup&gt;6&lt;/sup&gt;</td>
<td>Poor sensitivity and specificity, Test can be made more specific or sensitive, Time-consuming and laborious</td>
</tr>
<tr>
<td>Frequency doubling perimetry</td>
<td>90–94%&lt;sup&gt;7–9*&lt;/sup&gt;</td>
<td>91–96%&lt;sup&gt;7–9*&lt;/sup&gt;</td>
<td>Rapid, Relatively inexpensive</td>
</tr>
<tr>
<td>Disc and nerve fiber layer examination</td>
<td>Cup–disc ratio of 0.55 cut off:</td>
<td></td>
<td>Best performed using slit lamp biomicroscopy with a 60, 78 or 90D lens, Direct ophthalmoscope is a reasonable alternative, Inter-observer agreement of disc examination by clinical methods or fundus photographs is poor</td>
</tr>
<tr>
<td></td>
<td>59%&lt;sup&gt;10&lt;/sup&gt;</td>
<td>73%&lt;sup&gt;10&lt;/sup&gt;</td>
<td><strong>Clinic-based study with selected normals which may overestimate specificity</strong></td>
</tr>
</tbody>
</table>

* **Clinic-based study with selected normals which may overestimate specificity**
Methods to identify eyes at risk of angle closure include anterior chamber depth as well as anterior chamber depth/axial length ratio. The sensitivity and specificity of these techniques do not make them appropriate for screening.

Other easier techniques include the flash light test and van Herick test. In the flash light test a light is shone from the temporal side onto the cornea, parallel with but anterior to the iris. A shadow on the nasal limbus identifies an eye with a shallow anterior chamber, at risk for closure. The sensitivity of the flash light test is 80–86% and specificity is 69–70%.\textsuperscript{15,16}

The van Herick test uses a slit beam to compare the peripheral anterior chamber depth with the thickness of the cornea. The sensitivity and specificity of the test is 61.9% and 89.3%, respectively.\textsuperscript{15} Expressing the test in decimals yields similar results.\textsuperscript{16}

The flashlight and van Herick tests are also inappropriate for screening on their own.

If the van Herick test is positive AND the IOP is raised, the specificity improves to 99.3%. This is high enough actually to treat the patient as having angle closure.

**Population-based screening**

This is not recommended as a strategy. Population-based screening is especially inappropriate for developing countries without an adequate infrastructure. Adequate infrastructure here implies the availability of expertise (trained ophthalmologists), time and instrumentation required to confirm the diagnosis among test positives in an appropriate manner. It also means the availability of expertise (trained surgeons) and instrumentation to treat appropriately those in whom the diagnosis is confirmed. The operative word is “appropriate” and implies modern preferred practice. The requirements for diagnosis and management are covered in the relevant sections.

Each country will need to make a decision on population-based screening based on an assessment of the ground reality. Thus, more developed countries may opt to target high risk groups for screening.

**Case detection**

Any person over the age of 35 who seeks ophthalmic attention for any reason should have a comprehensive ophthalmic examination. As far as the diagnosis of glaucoma is concerned, this would include the following.
<table>
<thead>
<tr>
<th>Test</th>
<th>Ideal</th>
<th>Acceptable</th>
<th>Less than ideal</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tonometry</td>
<td>Applanation tonometry</td>
<td>Tonopen</td>
<td>Pneumotonometer or Schiotz tonometer</td>
<td></td>
</tr>
<tr>
<td>Dilated evaluation of the optic disc</td>
<td>Dilated stereoscopic evaluation by slit lamp biomicroscopy, fundus photography</td>
<td>Direct ophthalmoscope</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| Slit lamp biomicroscopy and van Herick test                        | NA                                                                    | NA                              | NA                               | • The flash light and/or van Herick tests are not appropriate tools to diagnose angle closure  
• A positive flash light or van Herick test requires confirmation by gonioscopy  
• If the flash light test is negative (less than 1/3 of the iris on the nasal side of the pupil covered by shadow) AND the van Herick test is negative (an anterior chamber depth of more than 1/4 thickness of the peripheral cornea), an occludable angle is highly unlikely.\textsuperscript{13} In this situation a gonioscopy for the purpose of ruling out an occludable angle may not be necessary |
| Gonioscopy                                                          | Indentation gonioscopy using a Sussman, Zeiss or Posner lens           | Goldmann single or two mirror with ‘manipulation’ |                                   | • Mandatory for every glaucoma suspect, irrespective of whether the suspicion is based on a raised IOP, optic disc, or visual field findings  
• Ideal to have both types of gonioscopes available to carry out a dynamic examination |
| Visual field examination (if the IOP is >21 mmHg and/or the disc is suspicious) | A full threshold test (includes SITA) using a calibrated white-on-white automated perimeter or Goldmann perimetry | Frequency doubling perimeter (FDP), Hensons visual field screener, or a Bjerrum screen |                                   | • A trained technician must perform the Goldmann perimetry, FDP, Hensons and Bjerrum screen |
Currently, the optimal method for detection of individuals with glaucoma is periodic routine comprehensive eye examinations. Feasibility of this depends on the current state of the healthcare system in the individual country. In lieu of the ideal, case detection should be relied on.

**Definitions**

<table>
<thead>
<tr>
<th>Definition</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Screening</strong></td>
<td>Population-based detection of glaucoma</td>
</tr>
<tr>
<td><strong>Case detection (opportunistic screening)</strong></td>
<td>Active detection of glaucoma when persons visit clinics and hospitals for other purposes</td>
</tr>
<tr>
<td><strong>Prevalence</strong></td>
<td>The proportion of patients with the target disorder (glaucoma) in the population tested</td>
</tr>
<tr>
<td><strong>Sensitivity</strong></td>
<td>The ability of a test to correctly identify those who have glaucoma (true positives)</td>
</tr>
<tr>
<td><strong>Specificity</strong></td>
<td>The ability of a test to correctly identify those who do not have glaucoma (true negatives or normal)</td>
</tr>
<tr>
<td><strong>Positive predictive value</strong></td>
<td>The proportion of patients with positive test results who actually have glaucoma</td>
</tr>
<tr>
<td><strong>Negative predictive value</strong></td>
<td>The proportion of patients with negative test results who do not have glaucoma</td>
</tr>
</tbody>
</table>

The predictive value of a test is dependent on the prevalence of glaucoma in the population being tested. As shown in Figure 1, assuming all other factors remain constant, the positive predictive value (PPV) will increase with increasing prevalence.

*Figure 1: Positive predictive value (PPV)*

![Graph showing positive predictive value (PPV) vs. prevalence (%)](image-url)
With a low prevalence of glaucoma, most of those who test positive will in fact be false positives.

In order to increase the effectiveness of all 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 the elderly, persons with family history of glaucoma, diabetics, hypermetropes (angle closure) and myopes (open angle).

References:
Appendix 1: How to test calibration of a Goldmann tonometer

1. Set the tonometer in position on its slit lamp stand, with its Perspex biprism head in place and the tension on the circular dial on its right side (from the examiner’s side of the slit lamp) set at 5 mmHg. The head should lean slightly forwards (away from the examiner).

2. Slowly twirl the circular dial counterclockwise until the head rocks back towards you. The tension should read 0–2 mmHg below zero (Figure A).

3. Slowly twirl the dial clockwise until the head rocks forwards again. The tension should read 0–2 mmHg (Figure B).

4. Remove the calibration rod from its box. Firmly screw into position the holding bracket that slides along the rod so that the closest mark in front of the center one (i.e., on the other side of the center from you) is aligned as exactly as you can (Figure C).

5. Slip the rod and its holder into the receptacle on the right side of the tonometer. The head will rock 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 20–23 mmHg.

7. Slowly twirl the circular dial counterclockwise until the head rocks backwards. The tension on the dial should read 17–20 mmHg.

8. Remove the rod and holding bracket from the tonometer and reposition the bracket so that it is aligned exactly with the most forward mark on the rod – furthest away from you (Figure D).

9. Replace the rod in its bracket in the tonometer receptacle. The tonometer head should rock backwards, towards you.

10. Slowly twirl the dial clockwise until the head rocks forwards. The tension should read 60–64 mm Hg.

11. Slowly twirl the dial counter-clockwise until the head rocks backwards – the tension should read 56–60 mm Hg.

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

(Figures A, B and D – Courtesy of Haag-Streit AG and Mandarin Opto-Medic Co Pte Ltd)
Appendix 2: Tonometry mires

(Courtesy of Ivan Goldberg, Australia)
Excess corneal applanation (IOP lower than tonometer reading)

(Courtesy of Ivan Goldberg, Australia)
Insufficient corneal applanation (IOP higher than tonometer reading)

(Courtesy of Ivan Goldberg, Australia)
Correct endpoint corneal applanation (IOP equals tonometer reading)
Appendix 3a: Gonioscopy

Gonioscopy

• Biomicroscopic examination of the anterior chamber angle
• Essential for glaucoma diagnosis, treatment and prognosis

Methods

Gonioscopic contact lens permits the angle to be seen.

1. Direct gonioscopy
   Place the Koepe goniolens on an anesthetized cornea with the patient supine. Fill the space between the lens and the cornea with a contact fluid (e.g., saline or methylcellulose). View the angle with a handheld biomicroscope and an illuminator.

2. Indirect gonioscopy
   At the slit lamp, place a mirrored lens (e.g., Goldmann-type or four-mirror indentation) on the cornea.

Indentation (pressure/dynamic) gonioscopy

With a four-mirror indirect contact lens press on the cornea to displace fluid into the angle to expose anatomic landmarks and to determine the presence of peripheral anterior synechiae.
Appendix 3b: Goniogram/gonioscopic chart

Grading system for gonioscopic findings (without indentation):

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

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 0</td>
<td>Iridocorneal contact</td>
</tr>
<tr>
<td>Grade I</td>
<td>Peripheral anterior chamber depth between iris and corneal endothelium is less than 1/4 corneal thickness (occludable)</td>
</tr>
<tr>
<td>Grade II</td>
<td>Greater than 1/4 but less than 1/2 of corneal thickness</td>
</tr>
<tr>
<td>Grade III</td>
<td>Greater than or equal to 1/2 of corneal thickness (non-occludable)</td>
</tr>
</tbody>
</table>

B.

1. Shaffer

<table>
<thead>
<tr>
<th>Grade</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>closed</td>
<td>10º</td>
<td>20º</td>
<td>30º</td>
<td>40º</td>
</tr>
</tbody>
</table>

2. Modified Shaffer

| Schwalbe’s line is not visible | Schwalbe’s line is visible | Anterior TM is visible | Scleral spur is visible | Ciliary band is visible |

C. Spaeth

1. Iris insertion
   - Anterior to Schwalbe’s line or TM
   - Behind Schwalbe’s line
   - Centered at scleral spur
   - Deep to scleral spur
   - Extremely deep/on ciliary band

2. Angular width
   - Slit
   - 10º
   - 20º
   - 30º
   - 40º

3. Peripheral iris configuration
   - queerly concave
   - regular
   - steep

4. TM pigment
   - 0 (none) to 4 (maximal)

Reference:
Appendix 3c: Corneal wedge diagram

A gonioscopic view of the drainage angle at high magnification (x16 or x25). 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).
Appendix 4: How to optimize patient performance in subjective perimetry

1. Choose the most appropriate investigation
   • Test pattern – 24–2: early/moderate damage and glaucoma suspects; 10–2: advanced damage or paracentral scotomas.
   • Test strategy – e.g., SITA (Humphrey field analyzer): most patients and suspects.

2. Patient set-up at the perimeter
   • Use near lens power based on current refraction.
   • Support the patient’s feet comfortably so that the thighs are horizontal.
   • Support the patient’s back.
   • Adjust chin rest height so the forehead touches its holding band easily.
   • Cover other eye fully – some patients prefer it open, some, closed.
   • Support the arms so shoulders and neck do not tire.

3. Instructions to the patient before starting the test
   • “We are getting you to do this test to give us information. We want to see how full and perfect your vision is, or if it isn’t, we want to know where the damage is, and what sort of damage it is.”
   • “The test is not difficult, but to get the best information for your care, it needs to be done in a particular way.”
   • “The key to success is to look straight ahead all the time. (Point where you want them to look.) Let the light come to you – don’t go looking for it.”
   • “You won’t see the light a good deal of the time, so don’t worry if time seems to be passing without a light appearing. The machine makes the light very dim so that it can tell when you can just see it.”
   • “Press the button when you think you see the light. All the lights you see, count – they can be fuzzy, dim, bright, it doesn’t matter.”
   • “Blink whenever you need to, but do so when you press the button. That will stop your eyes drying out and hurting, and you won’t miss any lights.”
   • “Hold the button down when you want to rest. That will pause the machine. Release the button when you want to continue. Remember you can rest as often as you like. You’re the one controlling the machine.”
   • “Let’s have a practice run now so you can get a feel for the whole thing.” (This is essential for perimetric novices, but may be important for many others as well. Run the demonstration program.)

4. Patient support during the test
   • Do not abandon the patient during the test – have your technician return regularly and frequently to supervise.
   • Reassure and encourage the patient during the test.
   • Restart the test if the performance is proving unreliable. Try to identify and to rectify the cause of the problem. Do not disparage or “blame” the patient.
   • Consider rescheduling the test if the patient cannot cope.
   • Be patient, more patient, and then even more patient.
Appendix 5: Secondary glaucomas – principles of management

<table>
<thead>
<tr>
<th>Strategy</th>
<th>An example for the approach to management – uveitic glaucoma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diagnose the underlying cause(s)</td>
<td>Diagnose uveitis and its cause(s)</td>
</tr>
<tr>
<td>Treat the underlying cause(s)</td>
<td>Anti-inflammatories</td>
</tr>
<tr>
<td>Identify the mechanism(s)</td>
<td>Posterior synechiae with pupil block</td>
</tr>
<tr>
<td>Treat the mechanism(s) – they may change over the course of the disease</td>
<td>Laser peripheral iridotomy</td>
</tr>
<tr>
<td>Medical therapy first-line agents are aqueous inflow inhibitors</td>
<td>β-blockers; α2-agonists; Carbonic anhydrase inhibitors</td>
</tr>
</tbody>
</table>
Appendix 6: Angle closure mechanisms

Site one: pupil block – iris bombé appearance

(Courtesy of Paul Chew, Singapore)

Site two: anteriorly rotated ciliary processes that push the iris forward and/or thick peripheral iris – plateau iris configuration

(Courtesy of Paul Chew, Singapore)

Site three: lens induced forward displacement of the iris – volcano configuration

(Courtesy of Paul Chew, Singapore)

Site four: aqueous misdirection with accumulation of fluid in the vitreous pushing the entire lens-iris diaphragm forwards

(Courtesy of Ningli Wang, China)
Appendix 7a: Argon laser trabeculoplasty (ALT)

About 100 equally spaced laser spots (diameter 50 microns) each for 0.1 seconds are applied over 360 degrees of trabecular meshwork, often in two sessions of 180 degrees, separated by 1–2 weeks. Ideally the spots should be applied over Schlemm’s canal, avoiding the iris root: at the junction of the anterior 1/3 and posterior 2/3 of the meshwork. The energy level should be set to induce a reaction from a slight transient blanching of the treated area, to small bubble formation.

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Appendix 7b: Contact transscleral 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 shown, the fibre-optic laser tip is 1.5mm behind the anterior edge of the footplate and protrudes 0.7mm. The laser tip should be placed over the ciliary body. Indentation improves energy delivery and blanches conjunctival blood vessels. The figure shows a relatively posterior ciliary body treatment, which may improve pressure reduction.
Appendix 8: Glaucomatous optic neuropathy

Moderate glaucomatous optic neuropathy
- Localized loss of both inferior and superior neuroretinal rim
- A classic inferior notch (small arrows)
- Nerve fiber layer defect in both superior and inferior arcuate area (large arrows)

Advanced glaucomatous optic neuropathy
- Neuroretinal rim thinning
- The cup extends to the disc rim
- Circumlinear blood vessel baring
- Bayoneting of the blood vessels
- Peripapillary atrophy

Nerve fiber layer hemorrhage
- Splinter, superficial flame-shaped, hemorrhage at disc margin (large arrow)
- Localized loss of neuroretinal rim at corresponding area
- Laminar dots are visible
- A pitlike notch at superotemporal rim is formed (small arrows)
Appendix 9: Field progression print-outs

New scotoma

10-06-2002 S/TA-Standard

GHT: Outside

normal limits

5.4 mm

20/20

Fovea: 36 dB
FL: 2/15
FN: 0 %
FP: 0 %

MD: -4.09 dB P < 1 %
PSD: 5.64 dB P < 0.5 %

18-10-2003 S/TA-Standard

GHT: Outside

normal limits

6.4 mm

20/20

Fovea: 36 dB
FL: 0/16
FN: 0 %
FP: 2 %

MD: -7.73 dB P < 0.5 %
PSD: 8.10 dB P < 0.5 %

(Courtesy of Prin RojanaPongpun, Thailand)

Deepening and enlarging scotoma

04-03-2003 S/TA-Standard

GHT: Outside

normal limits

5.6 mm

20/20

Fovea: 28 dB
FL: 0/15
FN: 0 %
FP: 8 %

MD: -7.23 dB P < 0.5 %
PSD: 5.82 dB P < 0.5 %

16-10-2003 S/TA-Standard

GHT: Outside

normal limits

6.4 mm

20/20

Fovea: 35 dB
FL: 0/15
FN: 0 %
FP: 3 %

MD: -8.06 dB P < 0.5 %
PSD: 7.94 dB P < 0.5 %

(Courtesy of Prin RojanaPongpun, Thailand)
Suggested Areas for Further Research

Prevalence and incidence of primary open angle glaucoma and primary angle closure glaucoma in Asia Pacific

Natural history of glaucoma in Asia Pacific

Natural history of angle closure

Risk factors – ranking risk factors among Asians according to importance

Normal values of central corneal thickness and optic disc parameters in Asian countries

Applicability of ‘ISNT Rule’ in Asian eyes

Target pressure reduction – evidence on extent of reduction required

Clinical classification of angle closure glaucoma based upon visual outcomes

Clarification of mechanisms responsible for angle closure in Asian people

Structural and functional change pattern, rate and extent in angle closure glaucoma versus open angle glaucoma

Randomized, controlled trials of all aspects of management of angle closure – particularly roles of laser iridotomy, laser iridoplasty, lens extraction and filtering surgery

Treatment outcomes in angle closure glaucoma versus open angle glaucoma – medical, laser and surgery

Efficacy of screening and prophylaxis of angle closure

Cost-efficient glaucoma screening program
### Definition of Terms

<table>
<thead>
<tr>
<th>Term</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angle neovascularization</td>
<td>New vessel formation within or on the surface of angle structures with or without formation of a fibrovascular membrane.</td>
</tr>
<tr>
<td>Anterior ischemic optic neuropathy (AION)</td>
<td>Optic nerve head ischemia resulting from disturbance in the short posterior ciliary artery circulation.</td>
</tr>
<tr>
<td>Aqueous misdirection syndrome</td>
<td>Misdirection of aqueous into the vitreous resulting from an anatomical abnormality at the level of lens, zonule/anterior vitreous and ciliary processes.</td>
</tr>
<tr>
<td>Cup–disc ratio (CDR)</td>
<td>The fractional decimal value obtained dividing the cup diameter with the disc diameter. The closer the value is to 1, the worse the damage.</td>
</tr>
<tr>
<td>Glaucomatous optic neuropathy (GON)</td>
<td>Characteristic pattern of damage to the optic nerve head caused by glaucoma.</td>
</tr>
<tr>
<td>Glaucoma suspect disc (GSD)</td>
<td>Optic nerve head appearance suggestive of glaucomatous damage.</td>
</tr>
<tr>
<td>Neovascular glaucoma (NVG)</td>
<td>Glaucoma resulting from a fibrovascular membrane across the angle in response to ischemia.</td>
</tr>
<tr>
<td>Normal pressure glaucoma (NPG)</td>
<td>Characteristic glaucomatous optic neuropathy in the presence of statistically normal intraocular pressure.</td>
</tr>
<tr>
<td>Occludable angle</td>
<td>Clinical term for an angle that is gonioscopically open but narrow enough to be considered at risk of closure.</td>
</tr>
<tr>
<td>Ocular hypertension (OH)</td>
<td>Intraocular pressure more than two standard deviations above the population mean with open angles and no evidence of glaucomatous optic neuropathy or visual field loss (with normal central corneal thickness).</td>
</tr>
<tr>
<td>Peripapillary atrophy (PPA)</td>
<td>Zone of retinal choroidal atrophy abutting the optic nerve head.</td>
</tr>
<tr>
<td>Peripheral anterior synechiae (PAS)</td>
<td>Permanent adhesions between the peripheral iris and other angle structures.</td>
</tr>
<tr>
<td>Definition of Terms</td>
<td></td>
</tr>
<tr>
<td>---------------------</td>
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</tr>
<tr>
<td><strong>Pigment dispersion syndrome (PDS)</strong></td>
<td>Abnormal scattering of iris pigment into the anterior segment of the eye.</td>
</tr>
<tr>
<td><strong>Plateau iris configuration</strong></td>
<td>An ocludable angle in the absence of pupil block.</td>
</tr>
<tr>
<td><strong>Plateau iris syndrome</strong></td>
<td>Angle closure in the presence of patent iridectomy.</td>
</tr>
<tr>
<td><strong>Posner-Schlossman Syndrome</strong></td>
<td>Episodic anterior uveitis and presumed trabeculitis with secondary elevation of IOP.</td>
</tr>
<tr>
<td><strong>Primary angle closure suspect (PACS)</strong></td>
<td>An eye in which appositional contact between the peripheral iris and posterior trabecular meshwork is present or considered possible. Epidemiologically, this has been defined as an angle in which 180–270 degrees of the posterior trabecular meshwork cannot be seen gonioscopically.</td>
</tr>
<tr>
<td><strong>Primary angle closure (PAC)</strong></td>
<td>PACS with either statistically raised IOP and/or peripheral anterior synechiae.</td>
</tr>
<tr>
<td><strong>Primary angle closure glaucoma (PACG)</strong></td>
<td>PAC with glaucomatous optic neuropathy.</td>
</tr>
<tr>
<td><strong>Primary open angle glaucoma (POAG)</strong></td>
<td>Chronic progressive optic neuropathy with characteristic changes in the optic nerve head and/or visual field in the absence of secondary causes.</td>
</tr>
<tr>
<td><strong>Primary open angle glaucoma suspect</strong></td>
<td>Significant risk factors for glaucoma (e.g., ocular hypertension, family history) and/or glaucoma suspect disc in the absence of frank glaucomatous optic neuropathy or visual field loss.</td>
</tr>
<tr>
<td><strong>Pseudoexfoliation syndrome (PXFS)</strong></td>
<td>Deposition of an abnormal fibrillo-granular protein predominantly in the anterior segment of the eye.</td>
</tr>
<tr>
<td><strong>Secondary angle closure glaucoma</strong></td>
<td>Glaucomatous optic neuropathy with angle closure and an identifiable (non-primary) cause.</td>
</tr>
<tr>
<td><strong>Secondary open angle glaucoma</strong></td>
<td>Raised intraocular pressure in the presence of identifiable secondary cause(s). Without treatment it is presumed this will cause glaucomatous optic neuropathy.</td>
</tr>
</tbody>
</table>
Index

A
Adrenergic agonists, 33, 34, 35
  factors influencing treatment, 12
Angle closure
  incidence, 5
  mechanisms, 31, Appendix 6
  signs of, 18
  surgery, 51
Anterior chamber
  depth, risk factor for glaucoma, 6
  examination, 17
Anti-fibrotics
  use in surgery, 53

B
β-blockers, 33, 34, 35
  factors influencing treatment, 13

C
Cataract
  cause of blindness, 5
  and glaucoma surgery, 55
Carbonic anhydrase inhibitors
  factors influencing treatment, 12
Cholinergics, 33, 34, 36
Cup–disc ratio, 20
Cyclophotocoagulation, 46

D
Disc haemorrhage, Appendix 8
  treatment category, 25
Double DOT, 39

E
Episcleral venous pressure, 28

F
Flash light test
  tool for case detection, 70, 71

G
Gonioscopic changes
  assessment at follow-up, 60
Gonioscopy, 17
  corneal wedge diagram, Appendix 3c
  gonioogram, Appendix 3b
  methods, Appendix 3a
  tool for case detection, 71
H
Hyperosmotic agents, 33, 37

I
Intraocular pressure
  causes of change at follow-up, 60
  factors influencing, 15
  targets, 27
Iridoplasty
  laser, 45
  modifiers of treatment goals, 27
Iridotomy
  laser, 43
  modifiers of treatment goals, 27
Iris examination, 17
ISNT rule, 21

L
Lens
  examination, 17
  extraction, 27

N
Neuroretinal rim, 21

O
Occulable angles
  treatment category, 25
Ocular hypertension
  treatment category, 25
One-eyed therapeutic trial, 38
Optic disc
  dilated evaluation of, 71
  examination, 19
  methods of assessment, 21
  photography and imaging, 20
  reassessment at follow-up, 61
  recording, 29
  size, 20
Optic nerve head examination, 19
Index

P

Perimetry
- automated, 22
- frequency doubling, 23, 71
- how to optimize patient performance, Appendix 4
- minimum acceptable resources for examination, 14
- tool for case detection, 71

Peripapillary atrophy, 18
- treatment category, 25

Pigment dispersion syndrome
- treatment category, 25

Posner-Schlossman syndrome
- past ophthalmic history of, 12

Primary open angle glaucoma
- epidemiology, 5, 69
- surgery, 51

Primary angle closure glaucoma
- epidemiology, 7, 65
- surgery, 50

Prostaglandins and other lipid receptor agonists, 33, 34, 37

Pseudoexfoliation syndrome
- treatment category, 25

R

Retinal nerve fiber layer examination, 19

Risk factors
- reassessment at follow-up, 60
- reduction, 26

S

Scotoma, 62, Appendix 9

SEAGIG decision square, 63

Secondary glaucoma
- cause of uniocular blindness, 5
- principles of management, Appendix 5

Surgery, 51
- penetrating filtering, 51
- non-penetrating, 51
- use of anti-fibrotics, 53

Slit lamp
- examination, 15, 16
- minimum acceptable resources for examination, 14
- tool for case detection, 71

Synechiae
- peripheral anterior, 18
- risk factor for progression, 63
- treatment category, 25

T

Tonometry
- tool for case detection, 71
- Goldmann-style applanation tonometry, 15
- how to test calibration, Appendix 1
- measurement errors, 16, Appendix 2
- minimum acceptable resources for examination, 14
- Tonopen, 15
- Perkin's tonometer, 15

Trabeculoplasty
- laser, 41, Appendix 7a

V

van Herick technique, 17
- tool for case detection, 70, 71

Visual field
- causes of change in, 61
- characteristics of glaucomatous defects, 23
- examination, 23
- treatment categories, 25