A GUIDE TO GLAUCOMA FOR PRIMARY HEALTH CARE PROVIDERS

A companion document to NHMRC Guidelines for the screening, prognosis, diagnosis, management and prevention of glaucoma 2010
Table of Contents

Introduction 1

The context of glaucoma 3

Identifying those at risk of glaucoma 5

Questions to ask patients with suspected glaucoma 6
Risk factors explained:
  Age 7
  Family history and genetics 8
  Ethnic origin 8
  Myopia 9
  Long-term steroid users 9
  Frequency of visits to eye care providers 9
  Smoking 9
  Diabetes 9
  Migraine and peripheral vasospasm 10
  Eye injury 10
  Systemic blood pressure 10

Glaucoma prognosis 13

Normal tension glaucoma 14
Ocular hypertension 14
Early primary open angle glaucoma 15
Advanced primary open angle glaucoma 15
Angle closure glaucoma 16

Diagnosis of glaucoma 17

Professional roles in diagnosis 18
  1. Degree of diagnostic suspicion 18
  2. Degree of urgency and severity 18
  3. Referral/cooperative management 19
What should be examined to identify angle closer? 20
What should be examined to identify open angle glaucoma? 21
Managing patients with glaucoma 23
  Open angle glaucoma 23
  Angle closure glaucoma 23

Monitoring: long-term care 27
  Indicators to change regimen 27
    After surgery for primary open angle glaucoma 29
    After surgery for angle closure 29
  Professional roles within the team 29
  Questions to ask your patients with glaucoma at review 29

Medication 31
  Side effects 32
  Topical medications 35
    Initiating treatment 35
    Application of topical medications 35
  Changing medication regimes 36
  Medication in acute angle closure crisis 36
  Managing glaucoma successfully within specific comorbid conditions 36
  Medication-induced glaucoma 36
    Open angle glaucoma 36
    Angle closure and angle closure glaucoma 37
  Specific patient groups 37

Laser therapy and surgery 39
  Laser therapy 39
    Laser iridotomy 39
    Laser iridoplasty 39
    Laser trabeculoplasty 39
    Combination laser surgery 39
    Cyclodestructive procedures 39
  Common surgical interventions 40
    Trabeculectomy 40
Introduction

A companion document to NHMRC Guidelines for the screening, prognosis, diagnosis, management and prevention of glaucoma 2010 (the Guide) has been developed to provide guidance for primary health care providers who encounter patients with the symptoms of glaucoma or with patients who have already been diagnosed with glaucoma. The Guide is a companion document to the NHMRC Guidelines for the Screening, Prognosis, Diagnosis, Management and Prevention of Glaucoma 2010 (the Glaucoma Guidelines).

This Guide aims to inform primary healthcare providers on key strategies and their responsibilities in:

- Identifying those at risk of developing glaucoma
- Risk factors that can be elicited from the patient’s history
- Questions to ask patients with suspected glaucoma
- Examining eye function
- Questions to ask patients with glaucoma
- Managing glaucoma successfully within specific comorbid conditions
- Having an understanding of angle closure glaucoma
- Seeking additional resources for professionals and consumers are available part of the glaucoma network

A summary of recommendations are provided at the beginning of each chapter in the Guide to assist the primary health care provider in the screening, prognosis, diagnosis, management and prevention of glaucoma. Definitions and anatomy related to glaucoma has been provided in the Appendices.

For more detailed information on the recommendations please consult the Glaucoma Guidelines that are available on NHMRC website at www.nhmrc.gov.au/publications/index.htm.
The context of glaucoma

In Australia

Glaucoma is a leading cause of blindness and vision loss. There is robust data to suggest that in most first world countries, 50% of glaucoma cases are undetected. Glaucoma Australia is a national charity that promotes eye health. This group estimates that over 300,000 Australians have glaucoma, although many people may not know it.

There is limited information regarding the prevalence and incidence of glaucoma within the Indigenous population of Australia. The Australian Bureau of Statistics ‘National Aboriginal and Torres Strait Islander Health Survey 2004-05’ provided data on long-term eyesight problems, however, glaucoma was not specifically reported in the data. It is currently assumed that Indigenous Australians are no more at-risk of glaucoma than Caucasians.

Glaucoma in the global population

Glaucoma is a global health problem expected to affect 60.5 million people by 2010. Glaucoma affects individuals through the full age span. It is estimated that up to two-thirds of people with glaucoma are undetected. Once established, glaucoma is progressive and usually relentless, and the damage to the eye is irreversible. Significant reductions in annual healthcare costs have been proposed if individuals with glaucoma are diagnosed and treated early. In general, in white populations around the world, the prevalence and incidence of glaucoma increases significantly with age in adults. People aged 70+ years have approximately three-times greater risk of developing glaucoma than people aged 40 years.

Major risk factors for developing glaucoma include elevated intraocular pressure (IOP), increased cup:disc ratio, disc rim haemorrhage, reduced central corneal thickness, age older than 40 years, strong family history and specific ethnicity.

Whilst people of African-American and Inuit descent are more at-risk of developing different forms of glaucoma, on current data, Australian Indigenous populations appear to be no more at-risk of developing any form of glaucoma than Caucasian-descent Australians.
Population screening for glaucoma is currently not considered to be cost-effective. There is no consensus in the literature on the recommended frequency of screening even for at-risk subgroups. An optimal test, or group of tests, for glaucoma screening has not been identified. However there is emerging evidence that several tests are potentially feasible for detecting glaucoma in a screening program, including assessment of optic disc, visual field and IOP.1,11,12
Identifying those at-risk of glaucoma

Recommendation 5

Identify and assess glaucoma patients and suspects (those at high risk of the disease).

Good Practice Points

- Identification is essential in order to make therapeutic decisions, whom to treat, and how aggressively to treat each person.
- All involved in their health care need to adopt a standard approach to risk factor assessment for each individual.

Recommendation 6

Detect glaucoma earlier

Good Practice Points

- Perform regular eye health checks for Caucasians over the age of 50, and for African-descended people over the age of 40.
- Perform regular eye health checks for all first-degree relatives of glaucoma patients, commencing 5-10 years earlier than the age of onset of glaucoma in their affected relative. Remind all glaucoma patients to alert first-degree relatives of the benefits of early and regular eye checks.
- Survey for glaucoma particularly in patients greater than 50 years of age, with any myopia, with abnormal blood pressure, with a history of migraine, with diabetes, with peripheral vasospasm, with eye injury and/or with ongoing steroid use.
- Monitor for glaucoma particularly in patients greater than 70 years of age, with IOP >21 mmHg, large and/or asymmetric cup-to-disc ratio (compared with disc size), disc haemorrhage, and thin central corneal thickness.
There is a strong body of research, developed over many years, that has established the risk factors for glaucoma development and progression. Many of these can be elicited from a patient history taken during the course of a consultation by primary health care providers, and by an eye examination with an ophthalmoscope (no pupil dilation). Full eye examinations are not usually performed by General Practitioners.

Where appropriate, it is a primary health care provider’s responsibility to educate patients about glaucoma and to alert them to the risk factors associated with it.

Questions to ask patients with suspected glaucoma

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>What are your symptoms?</td>
<td></td>
</tr>
<tr>
<td>Do you have any first-degree relatives with eye disorders (e.g. parents or siblings)?</td>
<td></td>
</tr>
<tr>
<td>What is your age?</td>
<td></td>
</tr>
<tr>
<td>Do you have existing eye conditions? (e.g. myopia and hypermetropia, eye trauma)</td>
<td></td>
</tr>
<tr>
<td>Do you have other medical problems? (e.g. diabetes)</td>
<td></td>
</tr>
<tr>
<td>Are you of African or Asian descent?</td>
<td></td>
</tr>
<tr>
<td>Are you taking any prescription or over-the-counter medicines, if so what?</td>
<td></td>
</tr>
<tr>
<td>Are you pregnant or breastfeeding?</td>
<td></td>
</tr>
<tr>
<td>When did you last have an eye examination?</td>
<td></td>
</tr>
<tr>
<td>Have you heard of glaucoma?</td>
<td></td>
</tr>
<tr>
<td>What do you know about glaucoma?</td>
<td></td>
</tr>
<tr>
<td>Would you like details on how to contact organisations offering glaucoma education and support services?</td>
<td></td>
</tr>
</tbody>
</table>
Risk factors explained

Age

Advancing age is a major risk factor for the development of glaucoma.

The prevalence of glaucoma is four to 10 times higher in the older age groups than in individuals in their forties. Damage to the optic nerve from glaucoma is uncommon before the age of 50 years in Caucasians, however it occurs at least a decade earlier in people of African descent.

Age-specific glaucoma prevalence data is available from the two Australian studies, based mainly on white Australians (Figure 2). Updates are required to this data given the increasingly multicultural nature of the Australian population (see Recommendation 6).

Figure 1: Age-specific prevalence of open angle glaucoma (extracted from Weih et al4 p1969)

![Age-specific prevalence of open angle glaucoma](image)

**POINT OF NOTE**

Caucasians over 50 years of age are at moderate risk, and those over 80 years of age are at high risk of developing glaucoma. A rational approach to screening for glaucoma is therefore required for Caucasians over the age of 50 years.

For Caucasians without other significant risk factors for glaucoma, a glaucoma assessment could be included in the health assessment for people aged 45-49 years (inclusive) who are at risk of developing chronic disease and the health assessment for people aged 75 and older using one of four time-based Medicare Item Numbers 701 to 707 undertaken by general medical practitioners.
Family history and genetics

A family history of glaucoma puts an individual at greater risk of developing the disease. In close relatives of individuals with primary open angle glaucoma (POAG), the prevalence is three to six times that of the general population.

<table>
<thead>
<tr>
<th>POINT OF NOTE</th>
</tr>
</thead>
<tbody>
<tr>
<td>A primary health care provider should advise all patients with glaucoma to inform all close relatives to undergo ocular examination as early as possible. This should occur at the age that is recommended for their ethnic group, or five to ten years earlier than the age of onset of glaucoma in their relative.</td>
</tr>
</tbody>
</table>

Ethnic origin

People of African descent have been reported to have an age-adjusted prevalence for POAG, 4.3 times greater than Caucasians. Prevalence of primary angle closure glaucoma (PACG) is highest among those of Asian or Inuit descent, with rates in these populations reported to be three to 10 times higher than in other ethnic groups.

<table>
<thead>
<tr>
<th>POINT OF NOTE</th>
</tr>
</thead>
<tbody>
<tr>
<td>The Working Committee note a new report ‘National Indigenous Eye Health Survey: Minum Barreng Full Report’ prepared by Anna-Lena Arnold, Ross A. Dunn and members of the National Indigenous Eye Health Survey Team (NIEHS) which was published 02 October 2009.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>POINT OF NOTE</th>
</tr>
</thead>
<tbody>
<tr>
<td>For individuals from high-risk ethnic backgrounds, appropriate surveillance activities include, but are not limited to, patient education regarding glaucoma, individual risk, consideration of concurrent medications, and advice to attend regular standard ocular examinations.</td>
</tr>
</tbody>
</table>
Myopia

Glaucoma and myopia have a strong familial basis and thus may share a common genetic link. The prevalence of open angle glaucoma (OAG) among people with myopia ranges from 1.4% to 4.3%. A dose–response relationship between OAG and myopia has been postulated (the higher the myopia, the more likely an individual would be to develop OAG).

Long-term steroid users

Corticosteroids are the main cause of drug-induced glaucoma (secondary glaucoma related to an external causation). Steroids administered by any route are associated with increases in IOP. Steroidal-like substances can also be found in traditional and natural medicines.

<table>
<thead>
<tr>
<th>POINT OF NOTE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Surveillance activities include, but are not limited to, patient education about risk, consideration of concurrent medications, and encouraging attendance at basic ocular checks.</td>
</tr>
</tbody>
</table>

Frequency of visits to eye care providers

The longer the time since the last visit to an eye care provider, the higher the risk of undiagnosed glaucoma.

Smoking

Evidence for the association of smoking with POAG is controversial. There is some evidence to suggest that current smoking is associated with POAG, but not past smoking.

Diabetes

The association between diabetes mellitus and POAG is controversial. The most recent data from Burr et al reported almost twice the risk of OAG onset among individuals with diabetes, compared with those without diabetes. The current understanding is that people with diabetes are at increased risk of developing POAG, and should be targeted for blindness-prevention programs.
Migraine and peripheral vasospasm

Migraine headache and peripheral vasospasm have been identified as risk factors for progressive glaucomatous optic nerve damage. Peripheral vasospasm has also been proposed as one possible mechanism for, or a factor contributing to, optic nerve damage in glaucoma.

Eye injury

Eye trauma is widely accepted as a risk factor for glaucoma. Traumatic glaucoma can occur immediately after a blunt trauma or penetrating eye injury, or years later. It is usually considered as secondary OAG. Eye trauma with angle recession is a risk factor for OAG.

Systemic blood pressure

Diastolic blood pressure largely determines perfusion pressure to the eye and is related to IOP. Low systemic blood pressure, including the nocturnal dip, may pose a risk for normal tension glaucoma (NTG), and POAG. There is limited evidence concerning the relationship between high systemic blood pressure and glaucoma. It is likely to be a complex relationship as patient age and the duration of systemic hypertension both impact upon the hypertensive state.

POINT OF NOTE

Recent publications, which were outside the scope of the literature review undertaken for these guidelines, indicate that reduced ocular perfusion pressure is strongly associated with glaucoma progression.
Reminder to health care providers

Health care providers should use a *patient's history* to elicit information about risk factors that are significantly associated with developing most types of glaucoma:

- elevated or fluctuating intraocular pressure
- strong family history of glaucoma
- advanced age
- African or Asian ethnicity
- current diabetes
- myopia
- rural location.

Health care providers should use a *patient examination* to elicit information about other risk factors that are significantly associated with developing most types of glaucoma:

- elevated or fluctuating intraocular pressure
- significant alterations in cup:disc ratio and cup:disc ratio asymmetry
- nerve fibre layer defects
- optic disc haemorrhage.

An assessment of these risk factors should aid in therapeutic decision-making regarding who to treat, when to treat, how to treat, and how aggressively to treat.

If appropriate, health care providers may also consider *other risk factors* which have more limited evidence of their association with developing most types of glaucoma:

- central corneal thickness
- current smoking
- current migraine and peripheral vasospasm
- long-term steroid use
- previous eye injury.
Glaucoma prognosis

 Recommendation 4

Assess risk of conversion from ocular hypertension to glaucoma.

**Good Practice Points**

- Patients at low risk of conversion should be considered for monitoring.
- Patients at high risk of conversion should be considered for treatment.
- Educate patients on the risks and consequences of conversion to glaucoma.

 Recommendation 2

Reduce intraocular pressure.

 Recommendation 3

Monitor visual field and determine rate of any field loss.

In the literature, the natural history of glaucoma is poorly defined and heterogeneous. An individual’s risk of progressive and sight-threatening glaucoma cannot be predicted with precision, however there is improving evidence to specifically identify candidates for treatment. If treatment decisions wait until there are overt signs of disease, this generally results in irreversible optic damage and likely disease progression.

However, all glaucoma treatments carry some side effects (e.g. development of cataracts), and therefore the trade-off between risk and benefit should be carefully considered in each patient’s case.12
Communication with patients

While lowering intraocular pressure slows or halts glaucoma progression, all interventions carry risk. Potential benefit and possible harm (the therapeutic index) need to be balanced carefully, with patient involvement where possible, in decision making.

Normal tension glaucoma

Individuals with glaucoma who have IOP in the ‘normal’ range are labelled as having NTG. There is sound evidence that IOP-lowering treatment is effective in preserving visual field in people with NTG.36

Ocular hypertension

10% of patients with ocular hypertension will progress to POAG within five years. However, the mean probability of progressing to POAG becomes greater with increasing IOP levels.1 Conversion time to POAG from ocular hypertension is significantly shorter for individuals not undergoing treatment.36

Risk factors for progression from ocular hypertension to glaucoma include elevated IOP, increased cup:disc ratio, older age, and thinner corneas.37 The relative proportion of the population converting from ocular hypertension to POAG can be reduced by 50% with appropriate IOP-lowering treatment.38,39

Communication with patients

It is essential that patients understand the risks for, and consequences of, progression to glaucoma and the value of treatment.

Rates of conversion to glaucoma are initially low, however any progression and visual loss is irreversible. Timely treatment can reduce the chance of progression and/or conversion by 50%.
Early primary open angle glaucoma

The literature provides sound evidence that without treatment, individuals with ocular hypertension and early POAG will convert more rapidly to advanced stages of the disease, with the inherent risks of visual field loss.

Allowing for considerable variability reported in the literature, an estimate of the average time for patients with POAG to progress to blindness without treatment is 23 years, and with treatment, 35 years. Topical IOP-lowering treatment is effective for most individuals, as it reduces the rate of progression of glaucomatous damage.

Communication with patients

With treatment 20 years ago, the average time to unilateral blindness for patients with primary open angle glaucoma was approximately 17 years. Untreated patients progress at approximately twice the speed of treated patients. In the last 20 years the rates of glaucoma blindness have dropped due to earlier diagnosis and more effective intraocular pressure-lowering treatment which significantly improves prognosis in the majority of cases. It is therefore important to comply with treatment and discuss any concerns with treatment with your health care provider.

Advanced primary open angle glaucoma

Patients with more severe glaucoma at diagnosis (i.e. those diagnosed later) are more likely to go blind. There is scant evidence on the impact of risk factors on the progression and outcomes of patients with severe and advanced glaucoma. The Advanced Glaucoma Intervention Study suggests that older age, lower formal education, male gender and diabetes are significant risk factors for the progression of advanced glaucoma to blindness. Reduction in maximal IOP and IOP fluctuation has some benefits for some patients, even in the advanced stage of glaucoma. However not every individual will gain the same benefits from treatment. A larger reduction in IOP is required to prevent progression in patients with more advanced glaucoma, when loss of vision is threatened. Patients with IOP below 14mmHg are reported to have the least progression.
Communication with patients

Higher rates of progression and visual loss may occur in patients who have been diagnosed late, or who already suffer from more advanced forms of glaucoma. However, evidence continues to support the benefits of active intraocular pressure reduction, even when patients have advanced stage glaucoma.

Angle closure glaucoma

Primary angle closure suspect (PACS) is an anatomical predisposition to closure with signs of narrowing of the angle (appositional contact between iris and trabecular meshwork) but without permanent occlusion or signs of adhesion (synechiae) between the iris and trabecular meshwork.

The treatment of primary angle closure-related glaucoma is two-fold; one is to manage the compromised angle and the other is to manage the glaucomatous nerve damage which is no different to the management of primary open angle glaucoma. Moreover, the prognosis regarding visual field and optic disc damage is thought to be identical, depending upon the pressure and patient susceptibility, so the previous section on primary open angle glaucoma prognosis is thought to pertain to angle closure.
Diagnosis of glaucoma

Recommendation 8

Assess with a comprehensive medical history, a full eye examination and investigate appropriately.

**Good Practice Points**

- A comprehensive medical history: identify all relevant risk factors, relevant co-morbidities and concurrent topical and systemic medications, and assess the impact of visual dysfunction, social environment and support networks that may affect adherence to a treatment program. Co-morbidities include hypertension, diabetes, thyroid disease, depression, asthma, liver and renal disease.

- A full eye examination: anterior segment evaluation including gonioscopy, optic nerve and retinal nerve fibre layer exam stereoscopic optic disc and retinal nerve fibre assessment with a permanent record, IOP and corneal thickness measurements.

- Appropriate investigations: standard automated perimetry (white-on-white) including comparison with age-corrected normals on a point-wise, regional (e.g. hemifield) and global basis, optic disc photography and imaging of the optic nerve and optic nerve fibre layer.

- Careful and informed interpretation of results from all imaging and functional tests in order to detect disease or to detect progression. With the multi-faceted nature of glaucoma and the large variability in normal values of all tests, consider results from all tests and assessments.

A diagnosis of glaucoma should be made on the basis of multiple sources of information, including the presenting history, an assessment of relevant risk factors, and an ocular examination reflecting structure and function of the eye. Initial consultation should elicit a complete medical, surgical, personal and occupational history, and ascertain relevant risk factors. This consultation should be followed by a comprehensive clinical examination including slit lamp examination, tonometry, fundus and optic nerve head examination, gonioscopy, and corneal thickness measurement. This examination may be in conjunction with special investigations to document the extent of structural damage to the optic nerve head and the retinal nerve fibre layer, using optic nerve and retinal
nerve fibre layer analysis or disc photography, computer-assisted visual field analysis. Children with suspected glaucoma should be referred to a specialist health care provider in the field.

A systematic approach should be used to elicit diagnostic information. In certain cases, glaucoma can present as a medical emergency. Confirmatory diagnosis of glaucoma may require more than one consultation with a health care provider, and the involvement of an ophthalmologist. A diagnosis is generally made on the basis of characteristic degenerative changes in the optic disc, and matching defects in visual fields.

Professional roles in diagnosis

The NHMRC guidelines encourage the establishment and nurturing of networks between primary health care providers, and between primary health care providers and ophthalmologists, to ensure that the best quality comprehensive care is provided to patients suspected of having, or diagnosed with, glaucoma.

Given the limited evidence base and ongoing changes in professional skills and responsibilities in Australia, the Working Committee notes that there are three essential issues that direct the most appropriate management pathway for a patient.

1. **Degree of diagnostic suspicion:** In the primary health care setting, if the degree of diagnostic suspicion of glaucoma is low, unnecessary referral of a patient to an ophthalmologist may lead to system overload. Low-risk patients may well be monitored by the most appropriate primary health care provider within the patient’s location, using established ocular health care networks for advice. If the degree of diagnostic suspicion of glaucoma is high however, the network should still be used for advice, and the appropriate decision may be a direct referral to a health care provider able to initiate treatment.

2. **Degree of urgency and severity:** If suspicion is very high with marked signs of nerve damage, and/or the IOP is very high then patients need urgent referral, with or without IOP-lowering treatment in the meantime, depending upon the waiting period for referral. Acute angle closure presents as a medical emergency and requires immediate referral to a specialist.
3. Referral/cooperative management: The Working Committee recommends that the professional roles, responsibilities and referral pathways are best determined in individual cases based on location, resources, skill-base of local health care providers and patient choice. Classically, referral occurs to an ophthalmologist when significant suspicion of glaucoma is raised. In some parts of Australia optometrists and or general practitioners can initiate treatment.

Irrespective of their location and professional networks, health care providers involved in diagnosing glaucoma should have the skills and equipment to measure IOP (by Goldmann Applanation Tonometry or a well calibrated non-contact tonometer, anterior chamber assessment and gonioscopy), visual field and optic disc.

Health care providers involved only with screening for, and/or diagnosis of, glaucoma, should receive appropriate training and continuing support from health care providers who manage glaucoma. Students in each health care discipline should be alerted to the importance of cooperation between disciplines in the screening, diagnosis and management of glaucoma.

<table>
<thead>
<tr>
<th>POINT OF NOTE</th>
</tr>
</thead>
<tbody>
<tr>
<td>In rural/remote settings, fundus photography is valuable if the results are to be relayed to a diagnosing health care provider.</td>
</tr>
</tbody>
</table>
What should be examined to identify angle closure?

- Assess anterior chamber and angle with gonioscopy and biomicroscopy
  
  *Key signs of closure*
  - peripheral anterior synechaie
  - trabecular meshwork pigment patches
  - iris insertion above a scleral spur
  - angle structures (trabecular meshwork) not being visible

- Assess and record eye structure with the best available instrument
  - refractive status
  - pupil size and reactivity
  - external appearance eye

- Assess and record eye function with best available instrument

- Assess IOP using best available instrument and taking patient preference into consideration
What should be examined to identify open angle glaucoma?

- Assess anterior chamber and angle with gonioscopy and biomicroscopy
  
  **Key signs**
  - abnormal trabecular meshwork
  - abnormal ciliary base (angle or cyclodialysis cleft)
  - blood reflux in Schlemm’s canal

- Assess and record eye structure with the best available instrument
  
  - ocular examination including
    - refractive status
    - pupil size and reactivity
    - external appearance eye
    - optic nerve head
    - visual field
  
  **Key signs**
  - typically superotemporal or inferotemporal optic disc neuroretinal rim loss with excavation
  - disc haemorrhage
  - increased cup:disc ratio and cup:disc ratio asymmetry
  - nerve fibre layer atrophy
  - peripapillary atrophy

- Assess and record eye function with best available instrument
  
  **Key signs**
  - defects that are
    - asymmetrical and cross midline
    - located in mid periphery
    - clustered in neighbouring points
    - correlate to defects on optic disc

- Assess IOP using best available instrument and taking patient preference into consideration
  
  **Key levels**
  - less than 21mmHg – consider NTG
  - over 26mmHg consider ocular hypertension
  - consider diurnal variation
Managing patients with glaucoma

Open angle glaucoma

Figure 2 presents the current best evidence for the most appropriate medical care of the ‘average’ patient with OAG. There are many factors which impact on the decision-making of the different health care providers who are responsible for the management of individual patients. This guideline does not consider the wider aspects of a patient journey with glaucoma, for instance social, emotional and economic elements. This guideline notes however that it is important that appropriate support is provided by health care providers to the patient with glaucoma and their carer(s). This may be provided by ensuring that they establish links to important consumer groups such as Glaucoma Australia, and by providing clear written and verbal communication. In advanced stages, low vision rehabilitation is a valuable adjunct to the glaucoma management plan.

Angle closure glaucoma

Figure 3 represents the current best evidence for the most appropriate care of the ‘average’ patient with intermittent or chronic angle closure. Angle closure may present as an acute crisis, requiring emergency management. These guidelines provide clear information regarding the signs and symptoms of an angle closure crisis.
Figure 2: Open angle glaucoma pathway

Patient with suspected open angle closure

- History
- Function
- Structure
- IOP

Risk assess

Discuss risk benefit. Reach consensus to undergo intervention. Provide literature to underpin understanding.

Diagnosis made and/or high-risk status

- Set target IOP
- Trial first choice medication
  - Successful MONITOR
  - Medically unresponsive
  - Adherence or administrative issues

NO diagnosis and low-risk status

- MONITOR

Diagnosis made and/or high-risk status

- Set target IOP
- Trial first choice medication
  - Successful MONITOR
  - Medically unresponsive
  - Adherence or administrative issues
  - Trial second and/or third choice
    - Successful MONITOR
    - Laser therapy
      - Consider length of time before failure
        - Long term
        - Short term
        - Rapid failure
          - Successful MONITOR
          - Filtering surgery with antimetabolite
            - Successful MONITOR
            - Rapid failure
              - Aqueous shunts
              - Cyclodestructive surgery
                - Ongoing care and MONITORING
Figure 3: Intermittent or chronic angle closure patient pathway

Patient with suspected angle closure

Discuss risk benefit. Reach consensus to undergo intervention. Provide literature to underpin understanding

Angle closure diagnosed

Peripheral Iridotomy (P.I.)

YAG Laser P.I.

Unilateral for chronic closure

Bilateral for acute or whenever access to medical services restricted

Unilateral Surgical P.I.

For acute closure where YAG P.I. not possible

Ongoing monitoring for ALL patients

Consider patency of angle

Open

MONITOR

Closing rapidly and/or significantly

Consider cause

Synechial closure without posterior force

Plateau iris or mild ciliary block NO cataract

Plateau iris or mild ciliary block WITH cataract

Ciliary block (severe)

Drainage surgery trabeculectomy

Iridoplasty

Lens extraction and IOL

MONITOR

Consider patency of angle

Open

MONITOR

Closing

Narrow post iridoplasty

Vitrectomy and anterior hyloidectomy

Ongoing monitoring for ALL patients
Monitoring: long-term care

The aims of monitoring patients diagnosed with glaucoma are to detect progression, evaluate the effects of treatment, re-assess risk factors for progression and note changes in health that may influence glaucoma management plans. Monitoring requires the performance of visual field, optic disc, gonioscopy and intraocular pressure measurement by suitably trained and equipped practitioners. Primary health care providers play an important role by prompting attendance in order to monitor glaucoma progression. Appropriate monitoring plans will ensure that patients who are at risk of glaucoma, and patients with established glaucoma, do not worsen through inadequate, or inappropriate medical care.

While it is not always possible to stop disease progression, it can usually be slowed significantly with appropriate treatment. Similar to diagnosis, monitoring is not based on a single test; rather it is based on a combination of test methodologies and technological tools. Lowering IOP is the strategy with the greatest evidence of effectiveness to achieve these goals. Therefore IOP measurement is vital in follow-up, with changes in visual field and optic nerve being the criteria for the alteration of target IOP. Once glaucoma has been diagnosed and patients placed on a treatment regimen, monitoring the patient’s capacity for adherence to the regimen and engaging the patient with treatment maintenance (including attendance at future appointments) is essential to best practice. The patient’s risk profile, disease state and capacity to self-manage dictate the frequency of review.

Indications to change regimen

The indications for adjusting a glaucoma management plan are:

• target IOP is not achieved
• the patient has progressive optic nerve or visual field damage despite achieving the target IOP. The validity of the diagnosis and target pressure should be reassessed. Additional evaluation may identify conditions that are contributing to the progression of damage, and serve as a justification to escalate treatment. These evaluations include obtaining diurnal IOP measurements, repeating the central corneal thickness measurement to verify a thin cornea or a change in corneal thickness after refractive surgery, or seeking evidence of unrecognised low blood pressure. A neurologic evaluation also may be considered.
• the patient is intolerant of the prescribed medication regimen
• the patient does not adhere to the prescribed medication regimen
• contraindications to individual medicines develop, and/or
• stable optic nerve status and low IOP occurs for a prolonged period in a patient on pressure-lowering medications. Under these circumstances, a carefully monitored attempt to reduce the medication regimen may be appropriate.\textsuperscript{13}

Downward adjustment of target pressure should be made in the event of progressive optic disc or visual field change. Upward adjustment of target pressure should be considered if the patient has been stable, and/or if the patient either requires less medication because of side effects, or personal choice. Whenever medication regimen changes are implemented, a follow-up visit is indicated within two to eight weeks to assess the response, as well as side effects from washout of the old medication, and onset of maximum effect of the new medication.\textsuperscript{44}

### POINT OF NOTE

Clinical judgement on a case-by-case basis is essential.

For newly diagnosed patients with glaucoma, and those who have undergone significant changes in treatment, assess the visual field two to three times per year, in the first two years, and then one to two times per year thereafter depending upon other risks, signs and symptoms.

Image the optic nerve every one to two years in glaucoma suspects and annually in glaucoma patients. A significant exception is for patients with substantial glaucomatous optic disc damage, with little remaining nerve tissue, and vertical cup:disc ratios (0.9 – 1.0). In these cases optic nerve imaging has little chance of detecting change in the remaining few fibres; there may not be a need to image at all.

Many field abnormalities on initial testing may not reproduce on subsequent tests. There are a number of techniques which can be used to assess the visual field.
After surgery for primary open angle glaucoma

After laser therapy or surgical treatment, a proportion of patients will be able to reduce or cease their medication. This may raise issues for monitoring. Health care providers should be sure that patients understand the chronic nature of their disease and the continued need for monitoring. A member of the health care team should take responsibility for monitoring these patients despite their independence from medication management.

After surgery for angle closure

After iridotomy, patients may be classified as residual open angle, or a mix of open angle and peripheral anterior synechiae. Patients in whom glaucomatous damage has occurred should be monitored as recommended for POAG. Patients who do not have glaucomatous optic neuropathy should be monitored in a manner similar to a POAG suspect.

Professional roles within the team

Using established networks, primary health care providers are encouraged to ensure the patients with glaucoma whom they are monitoring see an ophthalmologist at proscribed intervals.

Questions to ask your patient with glaucoma at review

- How are you? How are your eyes and vision?
- Are you managing to take your medication as discussed? If no, what are the problems and difficulties you face?
- Is there anything about your problems or your treatment plan that you would like explained?
- Are you experiencing any side effects from the medication?
- Do you have other medical problems? If yes, have they been exacerbated recently?
- Are you taking any prescription or over-the-counter medicines, if so what?
- Do you have plans to conceive/are you already pregnant? If yes, do you plan to breastfeed?
- When did you last attend an eye examination or have your condition monitored?
- Do you require further glaucoma information, at a patient level or would you benefit from contacting a glaucoma patient association?
Medication

Medication is the first management choice\(^1\) for most patients with glaucoma. Medication reduces IOP by enhancing aqueous outflow and/or reducing aqueous production. There are five main families of glaucoma medications, each with recognised actions, side effects and contraindications: beta-blockers, prostaglandin analogues, alpha2-agonists, carbonic anhydrase inhibitors and cholinergic agonists (see Table 1). Hyperosmotic medications such as mannitol are given to lower IOP in emergency situations.

The time taken to achieve maximal reduction in IOP is dependent on both the individual and the type of medication used. Initial reduction in IOP typically occurs within minutes to hours after administration, while maximal reduction in IOP can take weeks to months. Response to newly-initiated medications should be assessed after two to four weeks.

When prescribing glaucoma medication, many factors should be considered including IOP-lowering potency, additive effects, interaction with concomitant medications and disease states, side effects and ease of administration. Persistence with and adherence to medication regimens is vital in the management of chronic disease. Glaucoma medication must be suited to an individual’s capacity to effectively self-administer.

Many pharmacies have the capacity to provide a medicines profile, listing the prescription, OTC and complementary medicines being taken by a particular patient. The profiles are used to support patients in managing their medicines and can also be used as an effective communication tool when seeing other health professionals.

Primary health care providers should note that medications and how to use them are constantly being refined by research, and the development of new products. It is recommended that a pharmacist is involved in multidisciplinary networks of health care providers to provide specialist knowledge of medicines and their actions.

Ophthalmologists often authorise changes in a patient’s medication and should do in communication with a General Practitioner.

---

\(^1\) NB First choice refers to medications that a treating health care provider prefers to use as the initial intervention. First line refers to a medication that has been approved by an official controlling body for initial intervention (European Guideline Society 2003). This guideline refers to first choice as it provides guidance to health care provider.
Side effects

Health care providers should not underestimate the potentially significant side effects associated with medications for glaucoma. Side effects can be life-threatening. Particular caution should be exercised when prescribing medications for infants and the elderly, who may be more susceptible to side effects. Some side effects occur immediately, but most occur over time. This is why optimum management of patients with glaucoma should include regular monitoring and review of medication regimens. Details of side effects related to glaucoma medications are reported in Table 1.
### Table 1: Medications available in Australia that are used in the management of glaucoma

<table>
<thead>
<tr>
<th>Preparations by class</th>
<th>Mechanism of action</th>
<th>Efficacy</th>
<th>Daily dosage</th>
<th>Topical ophthalmic side effects</th>
<th>Systemic side effects</th>
<th>Order of treatment choices</th>
</tr>
</thead>
</table>
| **Prostaglandin Analogues** | Increase aqueous outflow | 25-30% | 1x | • Blurred vision  
• Burning  
• Stinging  
• Conjunctival hyperaemia  
• Foreign-body sensation  
• Itching  
• Reversible macular oedema  
• Increased pigmentation of the iris/peri-orbital skin  
• Longer-darker, and thicker lashes  
• Reactivation of herpetic infection  
• Iritis/uveitis | • Unlikely, but possible  
• Consult product information | **FIRST** |
| Latanoprost 0.005%  
Travoprost 0.004%  
Bimatoprost 0.03% | | | | | |
| **Beta-blockers** | Decrease aqueous production | 20-25% | 1x to 2x | • Burning  
• Stinging  
• Photophobia  
• Itching  
• Tearing  
• Decreased corneal sensitivity  
• Hyperaemia  
• Punctate keratitis  
• Diplopia | • Bronchospasm  
• Hypotension  
• Bradycardia  
• Heart block  
• Mask hypoglycaemia  
• Adversely affects lipid profile  
• Impotence  
• Fatigue  
• Depression  
• Reduced exercise tolerance  
• Syncope  
• Confusion  
• Alopecia | **FIRST** |
| Non-selective agents  
Timolol 0.25%, 0.5%, 0.1%  
Levobunolol 0.25% | | | | | |
| Selective agents  
Betaxolol 0.25%, 0.5% | | | | | |
| **Alpha-agonists** | Increase aqueous outflow and decrease aqueous production | 20-25% | 2x to 3x | • Ocular allergic reaction  
• Burning  
• Stinging  
• Blurring  
• Foreign-body sensation  
• Itching  
• Hyperaemia  
• Lid retraction  
• Conjunctival blanching  
• Photophobia  
• Mydriasis (Apraclonidine) | • Central nervous system depression  
• Oral dryness  
• Headache  
• Fatigue  
• Drowsiness | **SECOND** |
| Brimonidine 0.2%  
Apraclonidine 0.5% | | | | | |
<table>
<thead>
<tr>
<th>Preparations by class</th>
<th>Mechanism of action</th>
<th>Efficacy</th>
<th>Daily dosage</th>
<th>Topical ophthalmic side effects</th>
<th>Systemic side effects</th>
<th>Order of treatment choices</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Proprietary fixed combinations</strong></td>
<td>As for individual components</td>
<td>25-30%</td>
<td>2x</td>
<td>• As for individual components</td>
<td>• As for individual components</td>
<td><strong>SECOND</strong></td>
</tr>
<tr>
<td>Combigan (brimonidine 0.2%/timolol 0.5%)</td>
<td></td>
<td></td>
<td>2x</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cosopt (dorzolamide 2%/timolol 0.5%)</td>
<td></td>
<td></td>
<td>2x</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duo Trav (travoprost 0.004%/timolol 0.5%)</td>
<td></td>
<td></td>
<td>1x</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Xalacom (latanoprost 0.005%/timolol 0.5%)</td>
<td></td>
<td></td>
<td>1x</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Carbonic anhydrase inhibitors</strong></td>
<td>Decrease aqueous production</td>
<td>15-20%</td>
<td>2x to 3x</td>
<td>• Burning</td>
<td>• Bitter taste</td>
<td><strong>SECOND</strong></td>
</tr>
<tr>
<td>Topical</td>
<td>Dorzolamide 2%</td>
<td></td>
<td></td>
<td>• Stinging</td>
<td>• Headache</td>
<td></td>
</tr>
<tr>
<td>Brinzolamide 1%</td>
<td></td>
<td></td>
<td></td>
<td>• Itching</td>
<td>• Nausea</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Punctate epithelial keratopathy</td>
<td>• Fatigue</td>
<td></td>
</tr>
<tr>
<td><strong>Systemic</strong></td>
<td></td>
<td></td>
<td>25-30%</td>
<td>2x to 4x</td>
<td></td>
<td><strong>THIRD</strong></td>
</tr>
<tr>
<td>Acetazolamide 250mg</td>
<td></td>
<td></td>
<td></td>
<td>• Transient myopia</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• (Up to 50% of patients do not tolerate acetazolamide)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Fatigue/lethargy</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Anorexia/weight loss</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Gastro intestinal upset</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Paraesthesia</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Depression</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Loss of libido</td>
<td></td>
</tr>
<tr>
<td><strong>Cholinergics (Miotics)</strong></td>
<td>Increase aqueous outflow</td>
<td>20-25%</td>
<td>3x to 4x</td>
<td>• Eye pain</td>
<td>• Headache</td>
<td><strong>THIRD</strong></td>
</tr>
<tr>
<td>Pilcarpine 1%, 2%</td>
<td></td>
<td></td>
<td></td>
<td>• Decrease in night vision</td>
<td>• Salivation</td>
<td></td>
</tr>
<tr>
<td>Carbachol 1.5%, 3%</td>
<td></td>
<td></td>
<td></td>
<td>• Blurred vision</td>
<td>• Urinary frequency</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Miosis</td>
<td>• Diamphoea</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Topical medications

Initiating treatment

There is general consensus that for most patients with glaucoma, initial topical medication management should commence in one eye only, using the other eye as a control to check for therapeutic response. The response to lowering the IOP should be checked within two to six weeks, as should adherence to the medication regimen and instillation method. Two to four weeks is generally considered to be a suitable time frame for the medication to reach full effect before extending treatment to the fellow eye.

Application of topical medications

Patient adherence to their medication regimen, and their capacity to self-instill eye drops safely and effectively is of paramount importance when determining the most appropriate medication regimen for the individual. Patients should therefore be instructed carefully on how best to administer the medication to ensure accurate, effective and appropriate instillation. The preferred method for eye drop self-instillation includes holding the head horizontal with punctual occlusion and eyelid closure for three minutes (DOUBLE DOT: Digital Occlusion of Tear Duct and Don’t Open Technique) as systemic absorption can be reduced (by up to 70%) with this technique. If two or more drops are being instilled to the same eye, there should be an interval of at least five minutes between drops.

Glaucoma Australia has produced a DVD on ‘How to instill eye drops’ with funding from the Department of Health and Ageing. Health care providers are encouraged to become familiar with this resource, and to recommend it to their patients.

<table>
<thead>
<tr>
<th>POINT OF NOTE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Effective education on the instillation of eye drops includes:</td>
</tr>
<tr>
<td>• demonstrating the technique to the patient and carers</td>
</tr>
<tr>
<td>• observing patient and carers instilling the drops correctly</td>
</tr>
<tr>
<td>• repeating education, demonstration and observation until the health care provider is satisfied that patient and carers are fully capable of instilling the drops correctly.</td>
</tr>
</tbody>
</table>
Changing medication regimens

Change in well-tolerated medication regimens, and the use of additional medications are only supported in situations where target IOP has not been reached, despite the patient’s adherence to the regimen.

Medication in acute angle closure crisis

When acute angle closure presents as a crisis, medication management is usually initiated to lower IOP quickly, to reduce pain and to clear corneal oedema in preparation for laser therapy. Medications that suppress aqueous humor formation may be ineffective because they will have decreased capacity to reduce aqueous formation if the ciliary body is ischemic. Refer to the Therapeutic Guidelines: Emergency Medicine 2008 for clear guidance on the latest medications to be used in an emergency situation.

Managing glaucoma successfully within specific comorbid conditions

Glaucoma often occurs comorbidly to pre-existing health conditions. Primary health care providers should consider the potential interaction of glaucoma medications with medications for other conditions, as well as the effect of glaucoma medications on the pathophysiology of the pre-existing health conditions. Problematic pre-existing conditions include diabetes, depression, hyperthyroidism, asthma, renal and hepatic disease, chronic obstructive pulmonary disease, and cardiovascular disease. Further information is available in the NHMRC Glaucoma Guidelines (p122-128).

Medication-induced glaucoma

Open angle glaucoma

Corticosteroids are the main culprits in medication-induced glaucoma. Medication-induced glaucoma should be considered as secondary glaucoma related to its external causation. Corticosteroids raise the IOP when administered in any form.
**POINT OF NOTE**

Any patient taking steroids on a long-term basis is advised to undergo regular ocular checks to monitor intraocular pressure.

---

**Angle closure and angle closure glaucoma**

Adrenergic, anticholinergics (many antidepressants) and sulpha-based medications can precipitate an acute angle closure crisis. Further information is available in the NHMRC Glaucoma Guidelines (p127-128).

**POINT OF NOTE**

A large number of over-the-counter and prescription medications have been linked with acute angle closure crisis and/or raised intraocular pressure. Counseling could be offered for individuals who are identified as being at risk of angle closure glaucoma, regarding medication use.

---

**Specific patient groups**

When prescribing or monitoring glaucoma medications, health care providers should consider the special needs of pregnant women, breastfeeding mothers, children and other vulnerable groups of patients at risk of, or with, glaucoma. Further information regarding these specific considerations are available in the NHMRC Glaucoma Guidelines. (p129-135).
Laser therapy and surgery

Laser therapy

Laser therapy is currently considered for patients who fail to maintain IOP within the specified target range with medications. Emerging evidence suggests that laser therapy may become a first choice strategy for IOP reduction for some patients.

Laser iridotomy

Laser iridotomy is used to treat angle closure. This technique creates a hole in the iris in order to break the pupil block, which is the most common cause of angle closure. It is most frequently undertaken by Nd:YAG laser iridotomy, however when this form is not available, an argon laser may be utilised.

Laser iridoplasty

Laser iridoplasty is used in angle closure following iridotomy when the angle remains appositionally closed or occludable. Contraction burns are applied to the peripheral iris to pull it away from the trabecular meshwork.

Laser trabeculoplasty

Laser trabeculoplasty is used in OAG. Applications to the trabecular meshwork alter the drainage tissue, generally increasing aqueous outflow.

Combination laser surgery

Iridotomy is often combined with iridoplasty, where laser is applied to shrink the peripheral iris away from the trabecular meshwork to improve the aqueous flow.

Cyclodestructive procedures

Transcleral cyclophotocoagulation is a form of laser therapy which treats glaucoma by damaging the ciliary body. The laser is aimed through the sclera at the ciliary body, which secretes aqueous humor. This form of laser
treatment lowers IOP by decreasing aqueous humor production. Currently, cyclodestructive procedures are commonly performed using a transscleral laser delivery system, however they can also be performed endoscopically.\textsuperscript{11}

Common surgical interventions

Trabeculectomy

\textit{Incisional filtering microsurgery} involves surgically creating a drainage channel between the anterior chamber and subconjunctival space (see Figure 4). This is not a true fistula because the subconjunctival space is only loose connective tissue with a large capacity for fibrosis. The surgical dissection and subsequent aqueous flow are believed to stimulate this fibrosis which reduces the outflow of aqueous over time. Standard trabeculectomy five-year survival is reported to be 80\%.\textsuperscript{35}

Trabeculectomy may be undertaken as a primary procedure, or when laser therapy does not successfully lower IOP, or if the IOP begins to rise.

Since excessive scarring in the operative area will lead to failure of filtering surgery, anti-fibrotic medications such as 5-fluorouracil and Mitomycin C are frequently applied locally to retard healing.

Figure 4: Purpose of incisional filtering microsurgery
\textit{(Source: Members of the Working Committee)}
Resources

Consumer-orientated organisations

Glaucoma Australia
http://www.glaucoma.org.au/

Glaucoma Australia's (formerly The Glaucoma Foundation of Australia Inc.) mission is to minimise visual disability from glaucoma. Their sole purpose is:

- increasing community awareness and understanding of glaucoma and the need for regular eye checks
- supporting glaucoma patients and their families, especially with information
- funding glaucoma research.

Vision Australia
http://www.visionaustralia.org/

Vision Australia is a living partnership between people who are blind, sighted or have low vision. They are united by their passion that in the future people who are blind or have low vision will have access to and fully participate in every part of life they choose.

Royal Society for the Blind
http://www.rsb.org.au/

The Royal Society for the Blind (RSB) is the primary provider of services for South Australians who have severe vision impairment. These services are delivered by a professional, committed and highly qualified team supported by volunteers, drawn from all age groups and walks of life. Blindness or vision impairment can have a severe impact on a person’s lifestyle. The RSB is here to assist people to overcome their vision impairment and participate independently in the community.
Guide Dogs Australia
http://www.guidedogsaustralia.com/

Guide Dogs Australia is a brand that represents all of Australia’s state based Guide Dog organisations. Together, as the nation’s leading providers of orientation and mobility services, including Guide Dogs, they assist people who are blind or have a vision impairment gain the freedom and independence to move safely and confidently around the community and to fulfil their potential.

Association for the Blind of WA
http://www.abwa.asn.au/

Their mission is to maximise the quality of life of people who are blind or vision impaired by building confidence, promoting wellness, and creating connection.

Health Insite
http://www.healthinsite.gov.au/topics/glaucoma

Through this site you will find a wide range of up-to-date and quality assessed information on important health topics such as glaucoma, diabetes, cancer, mental health and asthma.

Profession-specific organisations

Australian Ophthalmic Nurses Association
www.aonavic.com.au
www.aonansw.org.au
www.aona.org.au

The Australian Ophthalmic Nurses Association is the professional association for Ophthalmic Nursing in Australia, with branches located NSW, QLD and VIC. The association aims to provide and communicate current information from a variety of clinical aspects, including nursing, medical and allied health professionals.

Royal Australian College of General Practitioners
http://www.racgp.org.au/

The Royal Australian College of General Practitioners is the professional organisation that focuses on supporting general practitioners in improving
health and wellbeing for all Australians and improving the safety and quality of general practice.

The Royal Australian College of General Practitioners has branches across Australia.

**Orthoptic Association of Australia**  

The Orthoptic Association of Australia Inc (OAA) is the national peak body for Orthoptists in Australia. The role of the OAA is to:
- promote and develop the profession of orthoptics
- represent and support its members
- contribute to excellence in eye health care in the community.

**Optometrists Association Australia**  

Optometrists Association Australia is the professional association for Australian optometrists. The website includes a ‘find an optometrist’ function, enabling consumers to find an optometrist by location.

**The Pharmacy Guild of Australia**  

The Pharmacy Guild of Australia is the professional body representing community pharmacies in Australia. The website includes a ‘Find a Pharmacy’ function, enabling consumers and health professionals to find a pharmacy by location.

**The Royal Australian and New Zealand College of Ophthalmologists (RANZCO)**  
[http://www.ranzco.edu/](http://www.ranzco.edu/)

The College’s mission is the improvement of the already high standard of eye care in Australia and New Zealand. In pursuit of this mission, the College provides a variety of services centered on its core roles as a higher educational institution and learned society.
Pregnancy-specific information

New South Wales
Mother Safe
Phone (02) 9382 6539, Toll free (NSW) 1800 647 848

Queensland
Queensland Drug Information Centre
Phone (07) 3636 7098
Information for health professionals only.

South Australia
Women’s and Children’s Hospital
Phone (08) 8161 7222

Victoria
Royal Women’s Hospital
Phone (03) 9344 2277

Western Australia
Women’s and Children’s Health Services
Phone (08) 9340 2723
References


Appendix I
Definitions and anatomy of the eye related to glaucoma

There is no definitive definition of glaucoma, largely due to the number of separate classifications of the disease. There is broad agreement that any form of glaucoma can be described as a progressive optic neuropathy with characteristic visual field loss, optic disc and nerve fibre degeneration. The NHMRC Glaucoma Working Committee recommended the use of the definition by Burr, Azuara-Blanco & Avenell: ‘Glaucoma describes a group of eye diseases in which there is progressive damage to the optic nerve characterised by specific structural abnormalities of optic nerve head and associated patterns of visual field loss’ (p2). There are four main ways to classify glaucoma:

1. Mechanism of aqueous flow impairment leading to open angle glaucoma or angle closure glaucoma.
2. Aetiology and presence of associated factors with the impairment (primary or secondary glaucoma).
3. Age of the population (congenital, infantile, juvenile or adult). Glaucoma of any age group may be classified within the above broad headings.
4. Temporal nature of symptoms i.e. acute/chronic or intermittent angle closure. This is most often used in angle closure glaucoma.

The pathophysiology of IOP elevation in glaucoma is primarily related to alteration in aqueous flow, although research is beginning to understand the role of optic nerve vulnerability and particular mechanisms of injury, including connective tissue and neural alteration, vascular dysfunction, genetic factors and responses to local excitotoxic conditions.

Open angle glaucoma

OA G is a progressive optic neuropathy. In the absence of other identifiable causes, it is classified as POAG. There is a characteristic acquired atrophy of the optic nerve, and loss of retinal ganglion cells and their axons. POAG is a disease in which elevated IOP is combined with a progressive optic neuropathy, resulting in characteristic excavation of the optic nerve head and corresponding visual field defects. This is associated with an open anterior chamber angle, by gonioscopic appearance. POAG is arbitrarily distinguished from NTG using an IOP cut-off of 21mmHg.
**Secondary OAG** can be caused by a variety of substances that mechanically block the outflow of aqueous through the anterior chamber angle, resulting in an elevation of IOP. These substances include pigment, exfoliation material, and red blood cells. Secondary OAG can also result from alterations in the structure and function of the trabecular meshwork, due to insults such as trauma, inflammation, and ischemia. In secondary OAG, elevated IOP causes progressive typical glaucomatous optic neuropathy and visual field loss. In several forms of secondary glaucoma, pathomechanisms are combined, leading to both secondary OAG and angle closure glaucoma.

**Normal tension glaucoma**

NTG is a subtype of POAG where IOP remains within the statistically determined usual range, but progressive optic neuropathy and visual defects typical of glaucoma occur. The criteria that are widely used to define NTG include:

- a mean untreated IOP consistently equal to or less than 21mmHg or median IOP equal to or less than 20mmHg on diurnal testing, with no single measurement greater than 24mmHg
- open drainage angles on gonioscopy
- typical optic disc damage with glaucomatous cupping and loss of neuroretinal rim
- absence of any secondary cause for a glaucomatous optic neuropathy (trauma, steroids, uveitis)
- visual field defect compatible with glaucomatous cupping (disc to field correlation).
Angle closure glaucoma

Angle closure glaucoma can present in either primary or secondary forms, in acute or chronic situations. Patients may have both, and present with acute attacks superimposed on a chronic condition. In primary angle closure, the eye is at risk of developing glaucomatous optic disc damage, particularly when associated with elevated IOP. When optic disc damage occurs, the eye is deemed to have progressed from primary angle closure to PACG. If acute angle closure is suspected, some components of the examination (optic disc imaging, visual field testing) may need to be postponed as patients may present as medical emergencies. Providing appropriate and timely treatment becomes the priority.

**Primary angle closure** is defined as appositional or synechial closure of the anterior-chamber angle, the commonest mechanism of which is usually pupillary block. In pupillary block, the front lens surface is anterior to the plane of the iris insertion into the ciliary body base, which causes resistance to aqueous humor flow. The resistance increases as the lens position is further forward, whether in primary angle closure or secondary angle closure. The resultant pressure gradient between the posterior and anterior chambers causes a forward bowing of the peripheral iris so that the iris covers all or part of the filtering portion of the trabecular meshwork (appositional angle closure), which can lead to elevation of IOP. Prolonged or repeated contact of the peripheral iris with the trabecular meshwork may lead to adhesions known as peripheral anterior synechiae and residual functional damage to the trabecular meshwork. The angle closure may or may not be associated with elevated IOP or glaucomatous optic neuropathy, and may occur in either an acute or chronic form.

**Acute angle closure** has at least two of the following symptoms (ocular or periocular pain, nausea and/or vomiting, history of intermittent blurring of vision with halos) and at least three of the following signs (raised IOP 21mmHg, conjunctival injection, corneal epithelial oedema, mid-dilated unreactive pupil, or shallow anterior chamber in the presence of an occludable angle).

**Secondary angle closure** is associated with a closed anterior chamber as identified on gonioscopy. The European Glaucoma Society suggests that the pathophysiology of secondary angle closure includes phakomorphic eyes and cases of inflammation.
Figure 5: The anatomy of the eye
(Source: Members of the NHMRC Working Committee)

Figure 6: An illustration of open angle and angle closure glaucoma with trabecular meshwork (Source: www.angleclosureglaucoma.cn)
Childhood glaucoma

Childhood glaucoma is a potentially blinding optic neuropathy invariably associated with raised IOP, characteristic optic disc changes and associated with specific visual field defects. Typically in infants, the signs of glaucoma are corneal haze, tearing of the eye with striking photophobia, and enlargement of the eyeball (buphthalmos). Childhood glaucoma is defined by age and mechanism of onset.

Pigmentary glaucoma

Pigmentary glaucoma presents similarly to POAG, with additional key signs including:

- pigment on the anterior surface of the iris often as concentric rings within the iris furrows
- spoke-like transillumination defects in the midperiphery of the iris
- pigment in the anterior and posterior chambers, and possibly Krukenberg’s spindles on the corneal endothelium
- a dense, homogeneously pigmented trabecular meshwork, especially posteriorly
- an open, deep anterior chamber angle with possible posterior bowing (concavity) of the iris
- rise of the IOP to rather high levels, with dramatic fluctuation
- pigment release resulting from pupillary dilation or strenuous exercise which requires assessment of the IOP after dilation.

Pseudoexfoliation glaucoma

Pseudoexfoliation glaucoma presents similarly to POAG, with additional key signs of:

- distribution of pseudoexfoliative material on the pupillary margin of the iris and, on the surface of the lens, as a central translucent disc with curled edges surrounded by an annular clear zone
- a peripheral granular zone on the anterior surface of the lens, best viewed through a dilated pupil
transillumination defects in the iris near the pupil, and patchy pigmentation of the trabecular meshwork located in the superior angle and anterior to Schwalbe’s line. Pigment granules may form a whorled pattern over the sphincter muscle on the surface of the iris

- depigmentation of pupillary ruff
- poor pupillary response to topical mydriatic medications
- accelerated cataract formation
- trabecular pigmentation which may precede the appearance of pseudoexfoliative material on the surface of the lens, even though this material is present in the conjunctiva.

Neovascular glaucoma

Neovascular glaucoma is a secondary glaucoma variously referred to as hemorrhagic glaucoma, thrombotic glaucoma, congestive glaucoma, rubeotic glaucoma, and diabetic hemorrhagic glaucoma. Causes of neovascular glaucoma include numerous secondary ocular and systemic diseases that share one common element, this being retinal ischemia/hypoxia and subsequent release of an angiogenesis factor. This angiogenesis factor causes new blood vessel growth from pre-existing vascular structures. Depending on the progression of neovascular glaucoma, it can cause glaucoma either through secondary open angle or secondary closed angle mechanisms. This is accomplished through the growth of a fibrovascular membrane over the trabecular meshwork in the anterior chamber angle, resulting in obstruction of the meshwork and/or associated peripheral anterior synechiae.
Appendix 2

Companion document details

Purpose of the companion document

This companion document presents a summary for primary health care providers of the current best evidence presented in the NHMRC guideline for Screening, Prognosis, Diagnosis, Management and Prevention of Glaucoma. The purpose of the guideline is to inform practice for Australian health care providers, particularly utilising a multidisciplinary team approach.

Guideline development period

The systematic review of the literature underpinning these guidelines was completed in September 2008 (available on www.nhmrc.gov.au). The guideline was completed in June 2009, and public consultation occurred during October-November 2009. This guideline was commissioned and funded by NHMRC.

Centre for Allied Health Evidence
University of South Australia
(Technical Team)

Professor Karen Grimmer-Somers
Ms Janine Dizon
Ms Judith Lowe
Ms Anthea Worley
Ms Lucyllynn Lizarondo

NHMRC Expert Working Committee

Professor William Morgan (Ophthalmologist)
Lions Eye Institute (CHAIR)
Associate Professor Ivan Goldberg (Ophthalmologist)
Eye Associates Glaucoma Services Sydney Eye Hospital
Professor Jonathon Crowston (Ophthalmologist)
Centre for Eye Research Australia
Associate Professor David Mackey (Epidemiologist/Ophthalmologist)
Royal Victorian Eye and Ear Hospital
Professor Algis Vingrys (Optometrist)
Department of Optometry and Visual Sciences University of Melbourne
Dr Philip Anderton (Optometrist rural)
Associate Professor Amanda McBride (General Practitioner with interest in Glaucoma)
Head of General Practice School of Medicine
The University of Notre Dame Australia, Sydney and GP in Woollahra
Dr Genevieve Napper (Optometrist, low-vision service provider)
Victorian College of Optometry
Mr Grant Martin (Director, Professional Services)
Pharmaceutical Society of Australia
Ms Jill Grasso (Ophthalmic Nurse)
Representing the Ophthalmic Nurses Association
Ms Beverly Lindsell (Glaucoma Australia Representative)
Glaucoma Australia
Ms Tania Straga (Orthoptist)
Representing the Orthoptic Association of Australia
Ms Helen Robbins
Representing the Optometrists Association Australia (Observer)

Internal reference group

Mr Luke Grzeskowiak, Pharmacist
University of South Australia

NHMRC project staff

Ms Vesna Cvjeticanin
Ms Carla Rodeghiero
Mr Fethon Ileris
Ms Tess Winslade
Ms Kay Currie
Ms Marion Hewitt
Appendix 3

Abbreviations

- IOP: Intraocular pressure
- mmHg: Millimetres of mercury
- NHMRC: National Health and Medical Research Council (Australia)
- nm: Nanometre
- NTG: Normal tension glaucoma
- OAG: Open angle glaucoma
- PACG: Primary angle closure glaucoma
- POAG: Primary open angle glaucoma
### Glossary of terms

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Afferent pupillary defect</td>
<td>A defect of the pupillary reflex characterised by less constriction of both pupils when the affected eye is stimulated by light relative to that occurring when the unaffected eye is stimulated, as with the swinging flashlight test. The defect is also known as the Marcus Gunn pupil.</td>
</tr>
<tr>
<td>African</td>
<td>The literature variably refers to the increased risk of glaucoma occurring in people of African descent. This refers to people who trace their ancestry to Africa, whether this be African-Americans, African-Carribbeans, East Africans, Sub-Saharan Africans or West Africans.</td>
</tr>
<tr>
<td>Anterior chamber</td>
<td>The space in the eye, filled with aqueous humor that is bordered anteriorly by the cornea and a small portion of the sclera and posteriorly by a small portion of the ciliary body, the iris, and that portion of the lens which presents through the pupil.</td>
</tr>
<tr>
<td>Argon laser trabeculoplasty</td>
<td>Light stimulation of the trabecular meshwork of the angle of the anterior chamber by an argon laser beam to facilitate aqueous humor outflow.</td>
</tr>
<tr>
<td>Aqueous humor</td>
<td>The clear, watery fluid that fills the anterior and posterior chambers of the eye.</td>
</tr>
<tr>
<td>Biomicroscopy</td>
<td>Examination of ocular tissue using a bright focal source of light with a slit of variable width and height and a binocular microscope with variable magnification.</td>
</tr>
<tr>
<td>Confocal scanning laser</td>
<td>The recording of two-dimensional sectional images for the evaluation of ocular tissue, using a confocal laser imaging system displayed digitally in real time.</td>
</tr>
<tr>
<td>ophthalmoscopy</td>
<td></td>
</tr>
<tr>
<td>Confocal scanning laser</td>
<td>The recording of a series of images along the axial axis of the eye enabling the three-dimensional reconstruction of the topography of the surface of the specific tissue under examination using a confocal laser imaging system.</td>
</tr>
<tr>
<td>tomography</td>
<td></td>
</tr>
<tr>
<td>Term</td>
<td>Definition</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Cup:disc ratio</td>
<td>The ratio of the diameter of the area of excavation of the surface of the optic disc to that of the diameter of the optic disc in any given meridian, often either the horizontal or vertical meridian. Cup:disc ratio is a value obtained by dividing the cup diameter by the disc diameter. The closer this value is to 1, the greater the level of tissue loss and therefore damage to the disc.</td>
</tr>
<tr>
<td>Excitotoxicity</td>
<td>The stimulation of neurons to death by excessive levels of excitatory neurotransmitters.</td>
</tr>
<tr>
<td>Filtration surgery</td>
<td>Surgical procedures (e.g. thermal sclerostomy, posterior or anterior lip sclerectomy, trephination, trabeculectomy) used to create an alternative pathway for the outflow of aqueous humor to lower intraocular pressure.</td>
</tr>
<tr>
<td>Fundus photography</td>
<td>The use of a camera with optics and an illumination system that permits photographing the fundus of the eye.</td>
</tr>
<tr>
<td>Genetic mutation</td>
<td>The alteration of DNA sequencing by changes in the genome.</td>
</tr>
<tr>
<td>Glaucoma</td>
<td>Glaucoma describes a group of eye diseases in which there is progressive damage to the optic nerve characterised by specific structural abnormalities of optic nerve head and associated patterns of visual field loss.</td>
</tr>
<tr>
<td>Glaucoma suspect</td>
<td>A person suspected of having glaucoma has some but not all of the criteria required for a glaucoma diagnosis. They may have one or more of the following: suspicious optic disc, optic disc margin haemorrhage, occludable drainage angle, peripheral anterior synechiae or elevated intraocular pressure.</td>
</tr>
<tr>
<td>Gonioscopy</td>
<td>A diagnostic procedure to examine the angle of the anterior chamber in which a specialised corneal contact lens and a biomicroscope are used.</td>
</tr>
<tr>
<td>Health care provider</td>
<td>Any member of the glaucoma team who provides input into the patient’s glaucoma journey. Health care providers involved with glaucoma in Australia may include, but are not limited to, ophthalmologists, general medical practitioners, optometrists, ophthalmic nurses and orthoptists.</td>
</tr>
<tr>
<td>Hypermetropia</td>
<td>A defect of vision in which a person is able to focus on objects in the distance, but not on close objects.</td>
</tr>
<tr>
<td>Term</td>
<td>Definition</td>
</tr>
<tr>
<td>----------------------------------</td>
<td>-------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Intraocular pressure</td>
<td>The pressure within the eye due to the balance between the formation and drainage of the aqueous humor.</td>
</tr>
<tr>
<td>Multifactorial inheritance</td>
<td>The determination of phenotype by multiple genetic and environmental factors, each making a small contribution.</td>
</tr>
<tr>
<td>Myopia</td>
<td>A vision condition in which close objects are seen clearly, but objects farther away appear blurred.</td>
</tr>
<tr>
<td>Myocilin</td>
<td>A protein believed to be associated with primary open angle glaucoma found both extraocular and in the trabecular meshwork, optic nerve, retina, cornea, iris, ciliary body, and sclera.</td>
</tr>
<tr>
<td>Nerve fibre layer</td>
<td>The layer of the retina that comprises unmyelinated axons of retinal ganglion cells.</td>
</tr>
<tr>
<td>Neuroprotection</td>
<td>The use of pharmacological, genetic alteration, and other means to attenuate a destructive cellular environment thereby protecting neurons from secondary degeneration caused by a variety of primary insults (ischemia/hypoxia, stroke, trauma, degeneration).</td>
</tr>
<tr>
<td>Neuroretinal rim</td>
<td>The tissue between the optic cup and disc margins.</td>
</tr>
<tr>
<td>Nocturnal dip</td>
<td>The decrease in systemic blood pressure during sleep.</td>
</tr>
<tr>
<td>Optic nerve</td>
<td>The cranial nerve (N II) that carries visual impulses from the retina to the brain.</td>
</tr>
<tr>
<td>Perimetry</td>
<td>Determination of the extent of the visual field for various types and intensities of stimuli for the purpose of diagnosing and localising disturbances in the visual pathway.</td>
</tr>
<tr>
<td>Peripapillary area</td>
<td>Tissue surrounding the optic nerve head.</td>
</tr>
<tr>
<td>Polygenic</td>
<td>The traits or diseases caused by the impact of many genes, each with a small additive effect on phenotype.</td>
</tr>
<tr>
<td>Posterior chamber</td>
<td>The space in the eye delimited by the posterior surface of the iris, the ciliary processes, and the valleys between them, the zonule of Zinn, and the anterior surface of the crystalline lens. It includes the canal of Hanover, the canal of Petit, and the retrolental space of Berger.</td>
</tr>
<tr>
<td>Term</td>
<td>Description</td>
</tr>
<tr>
<td>-------------------------------------------</td>
<td>-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Pulsatile ocular blood flow</td>
<td>The indirect assessment of choroidal blood flow by estimating the influx of blood into the eye during cardiac systole from an evaluation of the continuous IOP pulse wave.</td>
</tr>
<tr>
<td>Puncta</td>
<td>Puncta are tiny openings along the eyelid margin through which tears drain.</td>
</tr>
<tr>
<td>Refraction</td>
<td>Clinically, the determination of the refractive errors of an eye, or eyes (e.g. myopia, hyperopia, astigmatism, anisometropia).</td>
</tr>
<tr>
<td>Reverse pupillary block</td>
<td>Blockage of the movement of aqueous from the anterior to the posterior chamber leading to a concave anatomical configuration of the peripheral iris.</td>
</tr>
<tr>
<td>Selective laser trabeculoplasty</td>
<td>Use of a q-switched Nd:YAG laser to target trabecular meshwork endothelial cells without provoking coagulative necrosis, to improve aqueous outflow.</td>
</tr>
<tr>
<td>Short-wavelength automated perimetry</td>
<td>A form of automated perimetry that isolates the blue cone mechanism of the visual system by utilising a two-colour incremental thresholding technique consisting of a large blue target on a bright yellow background.</td>
</tr>
<tr>
<td>Tonometry</td>
<td>A procedure for measurement of the pressure within the eye. Clinically, tonometry measures the intraocular tension.</td>
</tr>
<tr>
<td>Trabecular meshwork</td>
<td>The meshwork of connective tissue that is located between the canal of Schlemm and the anterior chamber, and which is involved in drainage of aqueous humor from the eye.</td>
</tr>
<tr>
<td>Trabeculectomy</td>
<td>Surgical creation of a fistula to allow aqueous outflow from the anterior chamber to the subconjunctival tissue space, bypassing the trabecular meshwork/Canal of Schlemm outflow pathway.</td>
</tr>
<tr>
<td>Visual acuity</td>
<td>The clearness of vision that depends on the sharpness of the retinal image and the integrity of the retinal and visual pathway. It is expressed as the angle subtended at the anterior focal point of the eye by the detail of the letter or symbol recognised.</td>
</tr>
<tr>
<td>Visual field</td>
<td>The area or extent of space visible to an eye in a given position.</td>
</tr>
</tbody>
</table>