Clinical Practice Guide for the Diagnosis, Treatment and Management of Glaucoma

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Optometry Australia has developed this Clinical Practice Guide in consultation with an expert working group comprised of 9 experienced practitioners who work extensively in the area of glaucoma assessment and management.

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Optometry Australia Clinical Practice Guide – Open Angle Glaucoma

This Clinical Practice Guide provides evidence-based information about current best practice in the management of glaucoma. It is general information for optometrists, and is not a formal treatment or management protocol. It is a guide to aid clinicians in their diagnosis and management and does not replace advice on glaucoma management provided by regulatory agencies including the Optometry Board of Australia. It is the responsibility of all optometrists to be familiar and comply with all policies about the management of glaucoma, including policies of the OBA. Material in this document is based on the National Health and Medical Research Council (NHMRC) Guidelines for the Screening, Prognosis, Diagnosis, and Management of Glaucoma (2010). We recommend this document be read in conjunction with the NHMRC Guidelines.

Introduction

Glaucoma in all its forms results in progressive optic neuropathy which may ultimately lead to a loss of visual function. Optometrists, in their capacity as primary eye health care providers, play a pivotal role in the provision of eye care services to Australians who have been diagnosed with or are at risk of developing glaucoma. This role includes the detection, assessment, diagnosis and management of the condition. Optometrists play an essential role in preventing vision loss through early detection and intervention, and facilitating appropriate referral pathways.

In 2010, the World Health Organisation estimated that glaucoma was the cause of 8% of blindness, making it the second leading cause of blindness worldwide. [1] It is estimated that 4.5 million people[2] globally are bilaterally blind due to glaucoma and this number is due to rise to 11.2 million by 2020.[3] In 2005, the Australian Institute of Health and Welfare reported that 3% of all vision impairment and 16% of blindness caused in Australia in those aged 55 or over was due to glaucoma. [4]

It is important that optometrists are confident and competent in assessing patients with or at risk of developing glaucoma, so that they are able to provide evidence-based management and advice including appropriate communication, diagnosis and referral when indicated, and where the practitioner is therapeutically endorsed, management of the condition in accordance with the Optometry Board of Australia's guidelines.

Epidemiology of Open angle glaucoma

Glaucoma is the second leading cause of blindness worldwide and will have significant impact on the rising health costs as prevalence increases with an ageing population. It is estimated that there will be approximately 80 million people affected by this condition by 2020. 74% will have open angle glaucoma and women are expected to account for just over half of identified cases. [3] The Asian population represents 47% of those with glaucoma and 87% of those with angle closure glaucoma.[3] In Australia alone, the prevalence is expected to rise from 208,000 in 2008 to 379,000 in 2025[5] due to ageing of the population. Over the same period of time, the health system costs are estimated to increase from $AU355 million to $AU784 million. Total costs, including indirect costs (falls, aged care, depression, loss of productivity etc.) are estimated to reach up to $AU4.3 billion in Australia by 2025. [5] The prevalence of glaucoma increases exponentially with age with one in 10 Australians over the age of 80 being diagnosed with glaucoma. [6] The National Indigenous Eye Health Survey found an overall prevalence of glaucoma was 2.2% of Australian Indigenous Adults.[7]

Open Angle Glaucoma

Open angle glaucoma is a chronic progressive optic neuropathy leading to characteristic optic nerve defects and retinal nerve fibre layer losses and/or visual field defects in the presence of an open anterior chamber drainage angle. The intraocular pressure (IOP) may or may not be elevated.

Open angle glaucoma can be classified as primary when unrelated to other underlying conditions, or secondary when the glaucoma is a consequence of another disease process (e.g. pseudoexfoliation, uveitis etc.)

Secondary Open Angle Glaucoma

Secondary open angle glaucoma occurs as a consequence of another ocular disease process. The most common aetiologies are pseudoexfoliation and pigment dispersion. Some of the less common causes are inflammatory, angle recession (traumatic), neovascularisation (Rubeosis Iridis), lens related (phacolytic), tumour related, iridoschisis, Iridocorneal Endothelial (ICE) Syndrome and several of the phacomatoses. Treatment of the most common forms of secondary open angle glaucoma is typically the same as the treatment for primary open angle glaucoma (POAG).

Pseudoexfoliative Glaucoma (PXG)

Pseudoexfoliation (PXF) is a disorder where a fibrillar proteinacious substance is deposited both systemically and within ocular tissue. The majority of ocular deposits occur on the anterior lens capsule where pupillary movement leads to dispersion of the material and iris pigment into the trabecular meshwork.[8] The reduced aqueous outflow may result in a rise in intraocular pressure and secondary pseudoexfoliative glaucoma (PXG). The Blue Mountains eye study showed PXF was a risk factor for glaucoma independent of the IOP.[9] Approximately one-third of patients with PXF will progress to PXG. Typical patients are in the older age group (over 50 years of age). The condition is more common in people of Scandinavian descent.

Pigment Dispersion Glaucoma (PDG)

Pigment dispersion syndrome (PDS) occurs in patients with a deep anterior chamber and/or backwards bowing of the iris, where the pigmented posterior iris rubs against the lens zonules with subsequent dispersion of pigment onto the corneal endotheluim (Krukenberg spindle) and into the trabecular meshwork.[10,11] The resulting reduction in aqueous outflow may result in a rise in IOP and secondary pigment dispersion glaucoma (PDG). Approximately 20% of patients with PDS will progress to PDG. Typical patients are younger (age 30-50 years), male and myopic. [12,13]

Narrow Angle Glaucoma

Narrow angle glaucoma is a progressive optic neuropathy in the presence of a shallow anterior chamber and/or crowded anterior chamber angle. It has both chronic and acute forms. Management is primarily surgical via laser iridotomy and/or crystalline lens extraction, so will not be addressed in this document.

The Glaucoma Pathway

1. DETECTION

Optometrists perform the vast majority of routine eye examinations in Australia. As primary care eye practitioners, optometrists are ideally positioned to detect patients who have risk factors for glaucoma during a routine eye exam. It is the responsibility of all optometrists, whether therapeutically endorsed or not, to detect glaucoma suspects at routine eye examinations. The major areas of investigation include:

- Patient History
- Disc Assessment
- Biomicroscopy
- Intraocular Pressures

Patient History	Relevant Question	Considerations
Ocular history	• Blunt ocular trauma	Anterior segment trauma with angle recession
	• Hyphema	
Medical history	• High Risk Medications	e.g. long term Corticosteroids use
	• Diabetes	Almost twice the risk developing POAG
	• Peripheral Vasospasm	Raynaud's Phenomenon
	• Systemic Hypertension or Hypotension	Strong link between POAG and hypertention[18]
Risk Factors	• Age	Caucasians over the age of 50 and for African-descended people over the age of 40
	• Family History	Incidence in 1st degree relatives is 3-5x that of general population
	• Race	African-descended population most at risk Asian decent (for angle closure glaucoma- ACG)

	• Smoking	Greater than 1x the risk of developing glaucoma
	• Myopia	2-5x greater in patients with myopia Hyperopia (for ACG)

Table 1: Historical risk factors associated with an increased risk of developing open angle glaucoma that should be included in an initial optometric examination[11]

Disc Assessment

Enlarged cup-disc ratio	With consideration to disc size and insertion
Cup-Disc Ratio asymmetry	Asymmetric cup-disc ratio >0.2
Neuroretinal Rim	Thinning, focal narrowing or notching of the neuroretinal rim typically in the superior and inferior poles of the optic nerve head
Optic Disc Haemorrhage	Drance haemorrhage
Blood vessels at Optic Disc	Baring or bayoneting at the optic disc
Retinal Nerve Fiber Layer	Nerve fiber layer defect in the superior and inferior bundles
Peripapillary atrophy	Zone-beta PPA – represents loss of retinal pigment epithelium and choriocapillaris leaving intact choroid vasculature.

Table 2: Structural posterior fundus features associated with glaucoma that should be assessed in an initial optometric examination[11]

Biomicroscopy

Narrow angles	Van Herick or Shadow Test
Pseudoexfoliation	PXF on lens capsule and pupil margin
Pigment Dispersion	Iris transillumination defects and pigment deposits on the corneal endothelium
Other signs of Secondary Glaucoma	Iris rubeosis or atrophy, uveitis

Table 3: Structural anterior eye features with glaucoma that should be assessed in an initial optometric examination

Intraocular pressures

Approximately half of patients with open angle glaucoma have an intraocular pressure within the statistically normal range (less than 21 mmHg). This is known as normal tension glaucoma (NTG) [14].

Other patients present with elevated IOP levels (over 21 mmHg) and no evidence of optic nerve damage or loss of visual fields. This is known as ocular hypertension (OHT).[15] In general, the higher the IOP, the greater the conversion rate to glaucoma and the greater the severity and progression of the disease.

2. GLAUCOMA ASSESSMENT

People with glaucoma risk factors that have been identified during the routine eye exam are recommended to undergo further clinical investigations to determine if glaucoma is present. The glaucoma assessment can be made by all optometrists, whether therapeutically endorsed or not. If certain equipment is not available to the practitioner, a referral can be made to a colleague for specialised testing with interpretation and report.

It may be prudent to conduct the glaucoma assessment over several visits and at different times of the day to reduce patient fatigue and assist in determining any diurnal fluctuations in IOP.

Equipment

To comply with the Optometry Board of Australia's guidelines[16] for the care of patients with or at risk of developing glaucoma, as well as detection, diagnosis and management of the disease, optometrists must have the equipment to measure and/or assess the following areas or alternatively refer to another optometrist or an ophthalmologist for these assessments:

Area of Assessment	Equipment required
Intraocular Pressures (IOPs)	Tonometer – Applanation, iCare, Tonopen
Central Corneal Thickness	Pachymeter/Anterior OCT
Anterior Chamber Angle	Slit lamp/Gonioscope
Optic Nerve Head	Slit lamp and Fundus Lens
Retinal Nerve Fibre layer	Slit lamp and Fundus Lens
Threshold Visual Fields	Automated Threshold Perimetry (10-2, 24-2 and/or 30-2 tailored to the patient and degree of visual field loss)

Table 4: Major aspects of assessment required in a patient who is at risk or diagnosed with glaucoma.

Best practice standards for the diagnosis of glaucoma are further interpreted in the National Health and Medical Research Council (NHMRC) guidelines to include the following: [11]

- Comprehensive medical history
- Full eye examination:
    - Stereoscopic optic nerve head assessment
    - Gonioscopy
    - Retinal Nerve Fibre layer assessment
    - Intraocular pressures
    - Corneal thickness measurement
- Standard automated perimetry (white-on-white) including comparison with age-corrected normal on a point-wise, regional (e.g. hemifield) and global basis.

The Frequency Doubling Technology Matrix perimeter is also a valuable tool designed for fast and effective detection of visual field loss.[17] Some studies have shown that it offers a high sensitivity and specificity in the detection of early glaucomatous damage. Other equipment and techniques that may supplement a comprehensive glaucoma assessment include:

Optical Coherence Tomography (Posterior OCT)	• Ganglion cell layer • Neuroretinal Rim • Retinal Nerve Fibre layer
Optical Coherence Tomography (Anterior OCT)	• Anterior segment assessment including anterior chamber and corneal thickness
Scanning Lasers (HRT; GDx)	• Evaluation of Retinal Nerve Fibre layer and Neuro-retinal rim

Table 5: Other diagnostic equipment and tests available for glaucoma assessment

Supplemental tests

- Monocular colour vision assessment
- Contrast sensitivity function
- Selective perimetry:
    - Short Wavelength Automated Perimetry (SWAP)
    - Flicker Perimetry

3. DIAGNOSIS

Upon completion of the glaucoma assessment and with reference to the results of the glaucoma specific testing, a decision can be made regarding the clinical diagnosis of glaucoma.

A. Glaucoma

Three or more *structural* signs of optic nerve and/or nerve fibre layer damage indicate a very high probability of glaucoma. The structural signs may be via clinical examination of the disc and surrounding tissues, optic nerve photography and/or OCT imaging. The structural signs (table 2) are to be in concordance with each other .[18]

Visual field testing may show a reliable and repeatable threshold white-on-white visual field defect typical of glaucoma (e.g. nasal step, arcuate defect, paracentral scotoma, temporal wedge) or diffuse losses in one hemifield greater than the other (positive glaucoma hemifield test).

Clinical Pearl: Evidence supports that it is NOT necessary to have a visual field defect to make a diagnosis of glaucoma as structural losses can precede currently detectable functional losses in early glaucoma. However, in spite of technology (e.g. OCT) being available to assist with early and accurate detection of glaucoma, visual field assessment is as essential aspect of glaucoma assessment and management.[19]

B. Glaucoma suspect

A patient may be diagnosed as a glaucoma suspect if there are only 1 or 2 structural signs of disease, the visual field testing is unreliable or not repeatable, or the abnormal test results are not in concordance with each other.

People with glaucoma risk factors should be reviewed on a regular basis and the glaucoma assessment repeated to determine any *progression* in structural losses or visual function. The frequency of re-assessment should be based on the number of structural signs and the number of risk factors and would be between 6-24 months.[11]

4. TREATMENT

Treatment of glaucoma with topical anti-glaucoma medications is presently restricted to those optometrists holding a scheduled medicines endorsement by the OBA.

A. Severity Grading

Once a diagnosis of glaucoma is made, staging of the disease is essential to determine severity of vision loss, which helps in setting the appropriate target intraocular pressure, and selecting the best form of treatment (eye drops, laser or surgery).

Glaucoma is a disease continuum, with a number of grading scales. In general, 5 stages of glaucoma severity can describe most clinical presentations of the disease:

- **High Risk Ocular Hypertension (OHT)**[15]
    - Central corneal thickness less than 555 microns, IOP greater than 26 mmHg, age greater than 60 years, larger C/D ratio, higher Humphrey pattern standard deviation (PSD)
    - Risk of 15% or more by OHTS risk calculator http://ohts.wustl.edu/risk/calculator.html
- **Pre-perimetric Glaucoma**[18]
    - 3 or more structural signs of disease (optic nerve and/or GCC/RNFL abnormalities) *without* visual field defect by standard automated perimetry
- **Early Perimetric Glaucoma**[20]
    - 3 or more structural signs of disease (optic nerve and/or GCC/RNFL abnormalities) *with* repeatable visual field defect (Humphrey Mean Deviation (MD) 0 to -6 dB)
- **Moderate Perimetric Glaucoma**[20]
    - 3 or more structural signs of disease (optic nerve and/or GCC/RNFL abnormalities) *with* repeatable visual field defect (Humphrey Mean Deviation MD -6 to -12 dB) and not within 5 degrees of fixation by standard automated perimetry.
- **Severe Perimetric Glaucoma**[20]
    - 3 or more structural signs of disease (optic nerve and/or GCC/RNFL abnormalities) *with* severe repeatable visual field defect (Humphrey Mean Deviation MD worse than -12 dB) or any defect within 5 degrees of fixation by standard automated perimetry.

Clinical Stages of Primary Open Angle Glaucoma		
Stage		**Clinical Sign**
Mild (Early Perimetric Glaucoma)	**Optic Nerve**	• Mild concentric or partial localized narrowing of the neuroretinal rim. • Disc haemorrhages • Cup-disc asymmetry (≥0.2)
	Retinal Nerve Fibre Layer	o Less bright reflex o Fine striations o Only large retinal blood vessels clear
	Visual Field	• Nasal step, partial acute scotoma, paracentral defect, temporal wedge • Mean deviation (MD) less than -6dB • Damage limited to one hemifield with less than 25% of points involved.
Moderate	**Optic Nerve**	o Moderate concentric narrowing of the neuroretinal rim o Increased in the area of central disc pallor o Complete localised notch or loss of neuroretinal rim in one quadrant
	Retinal Nerve Fibre Layer	• Minimal brightness to reflex • All retinal blood vessels clear
	Visual Field	o Partial or full arcuate scotoma in at least one hemifield o Mean deviation between -6dB and -12dB
Severe	**Optic Nerve**	• Complete absence of neuroretinal rim in at least 3 quadrants • Bayoneting of blood vessels • Increased area of central disc pallor
	Retinal Nerve Fibre Layer	o Dark reflex o All retinal blood vessels clear
	Visual Field	• Advanced loss in both hemifields • 5-10 degrees central island of vision • Mean deviation worse than -12dB

Table 6: Classification of clinical stages and signs of glaucoma[21]

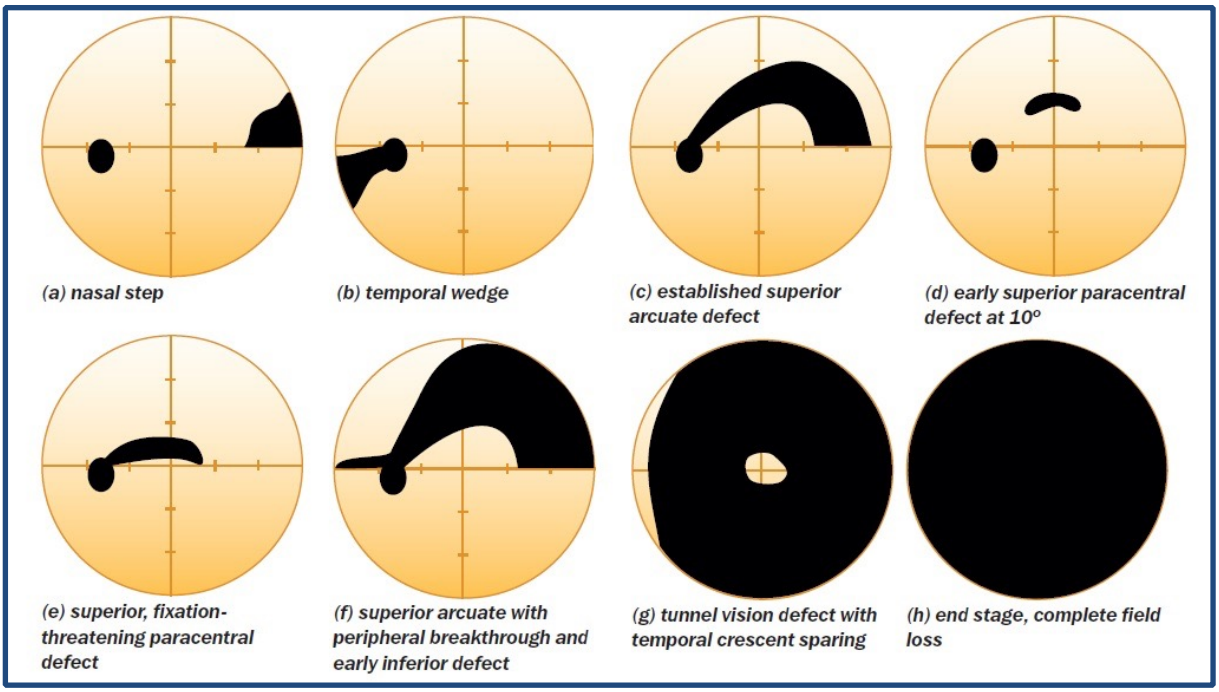

Figure 1: Visual Field defects* in different stages of Primary Open Angle Glaucoma [22]

**Visual field defect as depicted in Figure 1 above is only a graphical representation of visual field defects and it is important to note that many patients present with less classical signs.*

B. Decision to Treat

After consideration of the evidence of the effectiveness of treatment with the patient, an appropriate treatment plan is determined. Patients with Ocular Hypertension may be monitored closely to assess for any progression prior to initiating therapy.

The Ocular Hypertension Treatment Study (OHTS) [15] showed that 10% of patients with untreated elevated IOP progressed to glaucoma over a 5 year follow-up. The Early Manifest Glaucoma Trial (EMGT) [23] showed that 38% of patients with early perimetric glaucoma did not have progressive visual field loss over the course of the study when left untreated. The Collaborative Normal Tension Glaucoma Study (CNTGS) [24] showed that 50% of untreated study participants did not progress over 7 years of follow-up.

Patients with early disease and older patients aged >80 years may be monitored closely to assess the rate of progression prior to initiating therapy. Therapy may be delayed if the burden of treatment is high and the risk of significant symptomatic visual field loss is low in the patient's estimated lifespan. It may also be worth considering referring the patient for laser trabeculoplasty where appropriate for first line therapy for patients where medical therapy may be difficult.[19]

C. Setting Target IOP

Once determination of glaucoma severity is made, and a decision to initiate therapy is clinically appropriate, the target intraocular pressure needs to be set.

Target IOP is the estimated intraocular pressure required to slow or stop glaucomatous optic neuropathy. Target IOP is an estimate derived from the glaucoma clinical trials and depends on the severity of the disease, but needs to be individualised for each patient based on other ocular and systemic risk factors, family history, age, life expectancy etc.

No practitioner can know the true target IOP for any particular patient prior to initiating glaucoma therapy. The appropriateness of the target is only revealed once structural and function stability is assessed over a number of years. If the patient continues to progress even at their target pressure, the target IOP needs to be lowered further until stability of structure and function occurs.

Selection of Initial Target Intraocular Pressure (IOP)[11]

Severity	IOP Reduction
High Risk Ocular Hypertension and Pre-Perimetric Glaucoma	20% reduction in IOP
Early Perimetric Glaucoma	25% reduction in IOP
Moderate Perimetric Glaucoma	30% reduction in IOP
Advanced Glaucoma	Lower than 14mmHg

Table 7: Selection of Initial Target IOP

Progression: Evidence of repeatable progression at any severity stage mandates greater IOP reduction of another 10% over the existing target IOP.

D. Treatment options

Once target IOP has been determined, the optimal treatment to achieve target pressure needs to be selected. A number of treatment options are available including:

- Topical eye drop therapy
- Selective Laser Trabeculoplasty (SLT) (Alternative primary treatment)
- Ciliary body ablation
- Minimally invasive glaucoma surgery (MIGS)
- Surgery: Laser Trabeculectomy; Tube shunt
- Oral Acetazolamide

Historically glaucoma treatment begins with topical pharmacological eye drop therapy, followed by laser treatment in conjunction with or as an alternative to drops, and when these treatments fail to achieve stability of the disease, surgical intervention is required.

Topical pharmacotherapy is still the mainstay of treatment. Statistically 50% of patients can achieve target pressure with one topical agent, and 90% can achieve target pressure on maximal medical therapy (4 topical agents).[25]

Selective Laser Trabeculoplasty (SLT) is an alternative primary treatment to topical pharmacotherapy. Early use of SLT may be advantageous in pseudoexfoliative or pigmentary glaucoma, patients with allergy or contraindications to the use of certain classes of drugs, and where compliance and adherence to topical therapy is poor.

The Optometry Board of Australia's 'Guidelines for the use of scheduled medicines' outlines all board approved Scheduled 4 poisons that optometrists with a scheduled medicines endorsement are qualified to prescribe[16].

Drug selection

The ideal drug of choice should have the greatest clinical efficacy with the least ocular and systemic side effects and the lowest daily dosing schedule. In almost all cases a prostaglandin analogue meets these requirements. If the initial prostaglandin is partially effective but does not reach target IOP, consider substituting with a drug within the same class prior to changing to a different class of drug.

A monocular treatment trial should be considered for assessing response to target IOP. An alternative is to evaluate efficacy of response binocularly by comparing treated IOP to baseline pre-treatment IOP [26].

Medications available in Australia that are used in the management of glaucoma[11]

Preparations by Class	Mechanism of action	Efficacy	Daily Dosage	Wash-out period	Order of Treatment Choices	Side Effects[27]	Contraindications[27]
Prostaglandin analogues • **Latanoprost** 0.005% • **Travoprost** 0.004% • **Bimatoprost** 0.03% • **Tafluprost** 0.0015%	Increase Aqueous Outflow	25-30%	Once a day	4-6 weeks	First	Increase in iris pigmentation, darkening, lengthening of eyelashes, conjunctival hyperaemia	Intraocular inflammation (iritis, uveitis History of herpetic keratitis Aphakia or pseudophakia
Beta-Blockers *Non-Selective agents* • **Timolol** 0.25%, 0.5%, 0.1% *Selective agents* • **Betaxolol** 0.25%, 0.5%	Decrease Aqueous Production	20-25%	Once a day – Twice a day	2-5 weeks	First	**Common**: Blurred vision, decreased corneal sensitivity **Infrequent**: Bradycardia, Hypotension , Bronchospasm	Reversible Airway Disease e.g. asthma Bradyarrhythmia.
Alpha2-agonists • **Brimonidine** 0.2% • **Apraclonidine** 0.5%	Increase Aqueous Outflow and Decrease Aqueous Production	20-25%	Twice a day – Three times a day	1-3 weeks	Second	**Common:** Ocular irritation, dry mouth and nose, taste disturbance	Severe cardiovascular disease
Carbonic Anhydrase Inhibitors *Topical* • **Dorzolamide** 2% • **Brinzolamide** 1%	Decrease Aqueous Production	15-20%	Twice a day – Three times a day	1 week	Second	**Common:** Ocular irritation, bitter taste, FB sensation **Infrequent:** Vision Changes	Corneal grafts, Endothelial dystrophy. May cause corneal oedema. Allergy to sulfonamides

Preparations by Class	Mechanism of action	Efficacy	Daily Dosage	Wash-out period	Order of Treatment Choices	Side Effects[27]	Contraindications[27]
Carbonic Anhydrase Inhibitors *Systemic* • **Acetazolamide** 250mg	Decrease Aqueous Production	25-30%	Twice a day – Four times a day	3 days	Third	**Common:** CNS Depression and Lactic Acidosis	Adrenal or respiratory failure Sodium or potassium depletion
Cholinergics (Miotics) • **Pilocarpine** 1%, 2%	Increase Aqueous Outflow	20-25%	Three times a day – Four times a day	3 days	Third	**Common:** blurred vision, headache, myopia, miosis	Inflammatory disease. Uveitis, iritis, secondary glaucoma
Combination Therapies • **Combigan** (brimonidine 0.2%/timolol 0.5%) • **Cosopt** (dorzolamide 2%/timolol 0.5%) • **DuoTrav** (travoprost 0.004%/timolol 0.5%) • **Xalacom** (latanoprost 0.005%/timolol 0.5%) • **Ganfort** (bimatoprost 0.03%/timolol 0.5%) • **Azarga** (brinzolamide 1%/timolol 0.5%) • **Simbrinza** (brinzolamide 1%/brimonidine 0.2%)	As for individual components	25-30%	Twice a day Twice a day Once a day Once a day Once a day Twice a day Twice a day	As for Individual Components	Second	As for individual components	As for individual components

Table 8: Glaucoma Medication available in Australia

5. REVIEW

The goals of glaucoma therapy are:

- To lower a patient's baseline pressure to the target IOP to achieve stability in structure and function or slow down progression in structure and function
- To minimise treatment side effects and drug interactions for better quality of life
- To educate the patient on the chronic progressive nature of glaucoma so that compliance, adherence and persistence is maximised

Once treatment is initiated, regular review is mandatory to ensure optimal treatment outcomes. Routine review serves several purposes:

- To assess structure and function for any progressive change over time
    - i.e. to assess whether target pressure is achieved
- To adjust pharmacotherapy if target IOP is no longer met
    - i.e. there is evidence of glaucomatous progression
- To assess patient adherence and compliance with prescribed therapy
- To detect the development of side effects or intolerance to therapy
- To determine whether laser or surgery is now a more effective treatment option

A. Evaluation

Equipment and techniques required for review are the same as the initial assessment of glaucoma suspects, in particular comparison to stereo-disc photographs and visual field analysis. Optical Coherence Tomography (OCT) is also useful for progression analysis in addition to being a valuable diagnostic tool for routine clinical examination. Ultrasonic pachymetry does not typically need to be repeated unless there is a co-existing condition likely to change the corneal thickness.

B. Progression analysis

- The Ocular Hypertension Treatment Study (OHTS) showed that progression from ocular hypertension to glaucoma was most commonly detected by structural disc changes rather than visual field changes. [15]
- The Early Manifest Glaucoma Trial (EMGT) [23] showed that in patients with an existing glaucomatous visual field defect, progression was most commonly detected by visual field changes rather than by structural disc changes.
- Visual field progression (increased depth of scotoma, increased size of scotoma, new area of scotoma) needs to be confirmed on two or more occasions due to subjective variability and/or poor test reliability.

Inability to reach target IOP with maximum medical therapy and/or rapid progression of disease (>1.5 dB annual MD visual field loss) requires re-evaluation of the diagnosis in case of other pathology masquerading as glaucoma (e.g. retrobulbar orbital tumour) and referral for possible surgical intervention. Less than 10% of cases will fit this description.

C. Review schedule

Frequent review is required early in the treatment phase as patients are still adapting to topical eye drop use and compliance may still be poor. Prostaglandin eye drops may also take up to 6 weeks to reach full clinical efficacy.[28]

Ideally six threshold visual field tests should be performed in the first 2 years.[16] This will allow the perimeter's progression analysis software to determine true statistical progression from patient test-test variability. Current Medicare eligibility for the use of Computerised Perimetry Service items (10940 and 10941) is a maximum of two perimetry services in any twelve month period. Items can be claimed only in the presence of relevant ocular disease or suspected pathology of the visual pathways or brain.[29]

Once target IOP is met and structural and functional stability is achieved, the review schedule can be extended to every 6 months, which coincides with the maximum number of drug repeats that can be prescribed to patients.

6. REFERRAL OPTHALMOLOGY REFERRAL REQUIREMENTS

As with all ocular disease, referral to an ophthalmologist should be based on the clinical needs of the patient to ensure access to the most appropriate, evidence based, patient focused health-care in a timely fashion. OBA guidelines are subject to change; therefore optometrists are to ensure they practice in compliance with OBA guidelines that are currently in place.

The OBA Guidelines for the Use of Scheduled Medicines (2014) require referral letter to an ophthalmologist to be provided to the patient within the first four months of commencement of any glaucoma treatment.

Optometrists MUST also refer the patient to an ophthalmologist if:

- The initial management/treatment regime is failing to stabilise the condition and/or
- The patient is experiencing treatment side effects and/or
- If the patient needs assessment for potential surgical intervention or laser treatment.

References

1. Global data on visual impairments 2010 – World Health Organisation
2. World Health organization data from www.who.int/blindness/causes/priority/en/index6.html
3. Quigley HA, Broman AT. The number of people with glaucoma worldwide in 2010 and 2020. *Br J Ophthalmol* 2006; 90: 262-267
4. Vision problems among older Australians. Australian Institute of Health and Welfare 2005.
5. Mohamed Dirani PhD, Jonathan G Crowston MD PhD, Penny S Taylor PhD, Peter T Moore PhD, Sophie Rogers PhD, M Lynne Pezzullo PhD, Jill E Keeffe PhD and Hugh R Taylor MD. Economic impact of primary open-angle glaucoma in Australia. *Clinical and Experimental Ophthalmology* 2011; 39: 623–632.
6. Matthew D. Wensor, BOrth, Cathy A. McCarty, PhD, MPH, Yury L. Stanislaosky, PhD, MBBS, Patricia M. Livingston, PhD, Hugh R. Taylor, MD, FRACO. The Prevalence of Glaucoma in the Melbourne Visual Impairment Project. *Ophthalmology* 1998; 105: 733–9.
7. Taylor HR 2009. National Indigenous Eye Health Survey—Minum Barreng (Tracking Eyes). Melbourne: Indigenous Eye Health Unit, Melbourne School of Population Health in collaboration with the Centre for Eye Research Australia and the Vision CRC. Viewed 22 June 2016.
8. Rao A and Padhy D. Pattern of Pseudoexfoliation Deposits on the Lens and Their Clinical Correlation- Clinical Study and Review of Literature. PLoS One 2014; 9(12)
9. Mitchell P, Smith W, Attebo K, Healey PR. Prevalence of open-angle glaucoma in Australia. The Blue Mountains Eye Study. Ophthalmology. 1996 Oct; 103(10):1661-9.
10. Sowka J. Pigment dispersion syndrome and pigmentary glaucoma. *Optometry* 2004 75(2); 115-22
11. NHMRC Guidelines for the screening, prognosis, diagnosis, management and prevention of glaucoma 2010 https://www.nhmrc.gov.au/_files_nhmrc/publications/attachments/cp113_glaucoma_120404.pdf
12. Farrar SM, Shields MB,Miller KN, Stoup CM. Risk factors for the development and severity of glaucoma in the pigment dispersion syndrome. *Am J Ophthalmol* 1989; 108: 223–9.
13. Siddiqui Y, Ten Hulzen RD, Cameron JD, Hodge DO, Johnson DH.What is the risk of developing pigmentary glaucoma from pigment dispersion syndrome? Am J Ophthalmol 2003; 135: 794–9.

14. Lascaratos G, Garway-Heath DF, Burton R, Bunce C, Xing W, Crabb DP, Russell RA, Shah A; United Kingdom Glaucoma Treatment Study Group. The United Kingdom Glaucoma Treatment Study: a multicenter, randomized, double-masked, placebo-controlled trial: baseline characteristics. Ophthalmology 2013 Dec;120(12):2540-5

15. Kass MA, Heuer DK, Higginbotham EJ, Johnson CA, Keltner JL, Miller JP, Parrish RK 2nd, Wilson MR, Gordon MO. The Ocular Hypertension Treatment Study: a randomized trial determines that topical ocular hypotensive medication delays or prevents the onset of primary open-angle glaucoma. Arch Ophthalmol. 2002 Jun;120(6):701-13

16. Guidelines for Use of Scheduled Medicines. December 2014. Optometry Board of Australia. http://www.optometryboard.gov.au/Policies-Codes-Guidelines.aspx

17. Johnson CA, Wall M, Fingeret M, Lalle P. A Primer for Frequency Doubling Technology Perimetry. Skaneateles, New York: Welch Allyn, 1998.

18. Okoshi H, Kimura N, Hayashi H, Saito M, Endo N, Suzumura H and Usui M. Frequency of Normal-Tension Glaucoma found at health check-ups. Perimetry Update 1998/1999, pp. 443-451.

19. International Council of Ophthalmology. ICO Guidelines for Glaucoma Eye Care. 2015

20. Hodapp E, Parrish RK II, Anderson DR. Clinical decisions in glaucoma. St Louis: The CV Mosby Co; 1993. pp. 52–61.

21. American Optometric Association. Care of the Patient with Open Angle Glaucoma. 2011.

22. Broadway D. Visual field testing for glaucoma – a practical guide. Community Eye Health Journal. 2012: 25 (79-80): 66-70.

23. Heijl A, Leske MC, Bengtsson B, Hyman L, Bengtsson B, Hussein M; Early Manifest Glaucoma Trial Group. Reduction of intraocular pressure and glaucoma progression: results from the Early Manifest Glaucoma Trial. Arch Ophthalmol. 2002 Oct; 120(10):1268-79.

24. Anderson DR. Collaborative Normal Tension Glaucoma Study. Current Opinion in Ophthalmology. April 2003; 14(2): 86-90

25. Jost B Jonas; Wido M Budde; Andrea Stroux; Isabel M Oberacher-Velten; Anselm G Juenemann. IOP Profile in Glaucoma J Ophthalmic Vis Res 2010; 5 (2): 92-100

26. King A, Uppal S, Rotchford A, Lakshumanan A, Abedin A Henry E. Monocular trial of intraocular pressure-lowering medication: a prospective study. Ophthalmology. 2011 Nov; 118(11): 2190-5.

27. Australian Medicines Handbook, 2015.

28. Carl B. *Camras, Albert Aim, Peter Watson, Johan Stjernschantz,* MD, Latanoprost for Glaucoma Therapy, Ophthalmology *Volume 103, Number* 11, *November 1996*

29. Optometrical Services Schedule – Medicare Benefits Schedule Book (1 May 2016) *http://www.mbsonline.gov.au/internet/mbsonline/publishing.nsf/Content/1BC94358D4F276D3CA257CCF0000AA73/$File/201607-Optom.pdf*