

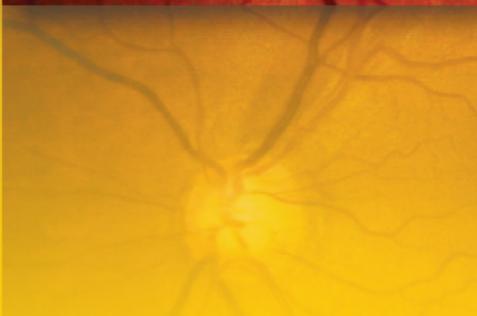
CLINICAL PRACTICE GUIDELINES

JUNE 2017

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MANAGEMENT OF GLAUCOMA

(Second Edition)



Ministry of Health
Malaysia



Academy of
Medicine Malaysia

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STATEMENT OF INTENT

These clinical practice guidelines (CPG) are meant to be guides for clinical practice, based on the best available evidence at the time of development. Adherence to these guidelines may not necessarily guarantee the best outcome in every case. Every healthcare provider is responsible for the management of his/her unique patient based on the clinical picture presented by the patient and the management options available locally.

UPDATING THE CPG

These guidelines were issued in 2017 and will be reviewed in a minimum period of four years (2021) or sooner if new evidence becomes available. When it is due for updating, the Chairman of the CPG or National Advisor of the related specialty will be informed about it. A discussion will be done on the need for a revision including the scope of the revised CPG. A multidisciplinary team will be formed and the latest systematic review methodology used by MaHTAS will be employed.

Every care is taken to ensure that this publication is correct in every detail at the time of publication. However, in the event of errors or omissions, corrections will be published in the web version of this document, which is the definitive version at all times. This version can be found on the websites mentioned above.

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LEVELS OF EVIDENCE	
Level	Study design
I	Evidence from at least one properly randomised controlled trial
II-1	Evidence obtained from well-designed controlled trials without randomisation
II-2	Evidence obtained from well-designed cohort or case-control analytic studies, preferably from more than one centre or group
II-3	Evidence from multiple time series with or without intervention. Dramatic results in uncontrolled experiments (such as the results of the introduction of penicillin treatment in the 1940s) could also be regarded as this type of evidence
III	Opinions of respected authorities based on clinical experience; descriptive studies and case reports; or reports of expert committees

SOURCE: US / CANADIAN PREVENTIVE SERVICES TASK FORCE 2001

In line with the current development in CPG methodology, the CPG Unit of MaHTAS is in the process of incorporating **Grading Recommendations, Assessment, Development and Evaluation (GRADE)** into its work process. The quality of each retrieved evidence and its effect size are carefully assessed/reviewed by the CPG Development Group. In formulating the recommendations, overall balances of the following aspects are considered in determining the strength of the recommendations:-

- Overall quality of evidence
- Balance of benefits versus harms
- Values and preferences
- Resource implications
- Equity, feasibility and acceptability

KEY RECOMMENDATIONS

The following recommendations are highlighted by the CPG Development Group as the key recommendations that answer the main questions addressed in the CPG and should be prioritised for implementation.

Recommendation 1

- Risk factors* should be identified in the management of glaucoma.

Recommendation 2

- Population-based screening for glaucoma is not advocated.
- Screening of glaucoma should be considered for patients with risk factors.

Recommendation 3

- Glaucoma diagnosis should be made based on combination of history (with presence of risk factors), ocular examination and investigation.

Recommendation 4

- Medical treatment in glaucoma should be individualised based on patient's characteristics and drug factors, and adjusted according to target intraocular pressure (IOP).
- Prostaglandin analogues should be used as first-line treatment in glaucoma.
- Patient education should be given to patients with glaucoma. This includes benefits and side effects of treatment, proper instillation technique of eye drop and compliance to treatment.

Recommendation 5

- Laser iridotomy should be performed in primary angle closure disease when indicated.
- Peripheral iridoplasty may be considered for initial treatment in acute angle closure.

Recommendation 6

- Intraoperative Mitomycin C during trabeculectomy should be used in glaucoma patients at risk of surgical failure.

Recommendation 7

- Glaucoma patients with blindness or low vision should be referred for vision rehabilitation which includes vocational, occupational and independent living.

Recommendation 8

- Ocular hypertension (OHT) patients should have comprehensive initial eye examination and assessment of risk factors for conversion to primary open angle glaucoma (POAG).
 - Central corneal thickness measurement should be performed.
- Treatment of OHT should be based on the risk of conversion to POAG.

Recommendation 9

- Comprehensive eye examination and risk assessment should be performed in primary open angle glaucoma suspect.
 - Diurnal intraocular pressure fluctuation should be considered in the assessment.

GUIDELINES DEVELOPMENT AND OBJECTIVES

GUIDELINES DEVELOPMENT

The members of the Development Group (DG) for these Clinical Practice Guidelines (CPG) were from the Ministry of Health (MoH) and Ministry of Higher Education. There was active involvement of a multidisciplinary Review Committee (RC) during the process of the CPG development.

The CPG update was done based on the CPG Management of Primary Open Angle Glaucoma, issued in 2008. In the update, the scope had been widened to include both primary open angle and angle closure glaucoma, and also selected conditions. A literature search was carried out using the following electronic databases: mainly Medline via Ovid and Cochrane Database of Systemic Reviews and others e.g. Embase, Pubmed and Guidelines International Network (refer to **Appendix 1** for **Example of Search Strategy**). The search was limited to literature published in the last ten years, on humans and in English. In addition, the reference lists of all retrieved literature and guidelines were searched to further identify relevant studies. Experts in the field were also contacted to identify further studies. All searches were conducted from 23 March 2015 to 29 July 2015. Literature searches were repeated for all clinical questions at the end of the CPG development process allowing any relevant papers published before 31 January 2017 to be included. Future CPG updates will consider evidence published after this cut-off date. The details of the search strategy can be obtained upon request from the CPG Secretariat.

References were also made to other CPGs on glaucoma such as Glaucoma: Diagnosis and Management of Chronic Open Angle Glaucoma and Ocular Hypertension (National Collaborating Centre for Acute Care, 2009), Primary Open-Angle Glaucoma PPP (American Academy of Ophthalmology, 2015), Terminology and Guidelines for Glaucoma 4th Edition (European Glaucoma Society, 2014) and Asia Pacific Glaucoma Guidelines Third Edition (Asian Pacific Glaucoma Society, 2016). The CPGs were evaluated using the Appraisal of Guidelines for Research and Evaluation (AGREE) II prior to them being used as references.

A total of 16 clinical questions were developed under different sections. Members of the DG were assigned individual questions within these sections (refer to **Appendix 2** for **Clinical Questions**). The DG members met 25 times throughout the development of these guidelines. All literature retrieved were appraised by at least two DG members using Critical Appraisal Skill Programme checklist, presented in evidence tables and further discussed in each DG meetings. All statements and

recommendations formulated after that were agreed upon by both the DG and RC. Where evidence was insufficient, the recommendations were made by consensus of the DG and RC. This CPG is based largely on the findings of systematic reviews, meta-analyses and clinical trials, with local practices taken into consideration.

The literature used in these guidelines were graded using the US/Canadian Preventive Services Task Force Level of Evidence (2001), while the grading of recommendation was done using the principles of GRADE (refer to the preceding page). The writing of the CPG follows strictly the requirement of AGREE II.

On completion, the draft of the CPG was reviewed by external reviewers. It was also posted on the MoH Malaysia official website for feedback from any interested parties. The draft was finally presented to the Technical Advisory Committee for CPG, and the HTA and CPG Council MoH Malaysia for review and approval. Details on the CPG development by MaHTAS can be obtained from Manual on Development and Implementation of Evidence-based Clinical Practice Guidelines published in 2015 (available at <http://www.moh.gov.my/index.php/pages/view/117>).

OBJECTIVES

To provide evidence-based recommendations in the management of glaucoma on the following aspects:

- screening
- diagnosis
- treatment

CLINICAL QUESTIONS

Refer to **Appendix 2**

TARGET POPULATION

i. Inclusion Criteria

- Adult patients (>18 years old) with:
 - primary open angle glaucoma
 - primary angle closure glaucoma
 - selected conditions such as ocular hypertension, glaucoma suspect, steroid-induced glaucoma, neovascular glaucoma and post-refractive surgery glaucoma

ii. Exclusion Criteria

- Secondary open angle glaucoma including pseudo exfoliation or pigment dispersion, primary congenital, infantile or childhood glaucoma

TARGET GROUP/USERS

This document is intended to guide those involved in the management of glaucoma in primary and secondary/tertiary care including:

- i. Doctors
- ii. Optometrists
- iii. Allied health professionals
- iv. Trainees and medical students
- v. Patients and their advocates
- vi. Professional societies

HEALTHCARE SETTINGS

Primary and secondary/tertiary care settings

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The draft guidelines were reviewed by a panel of experts from both public and private sectors. They were asked to comment primarily on the comprehensiveness and accuracy of the interpretation of evidence supporting the recommendations in the guidelines.

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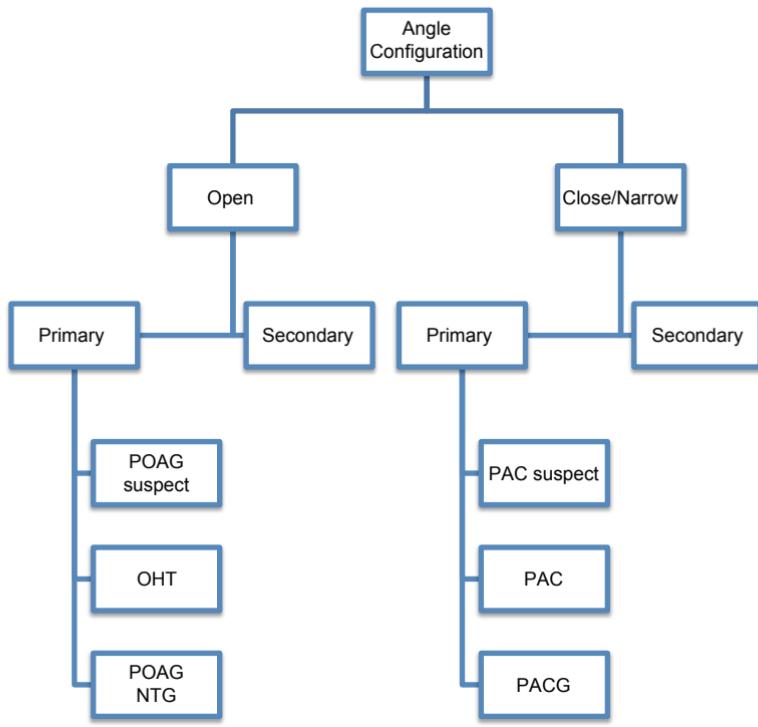
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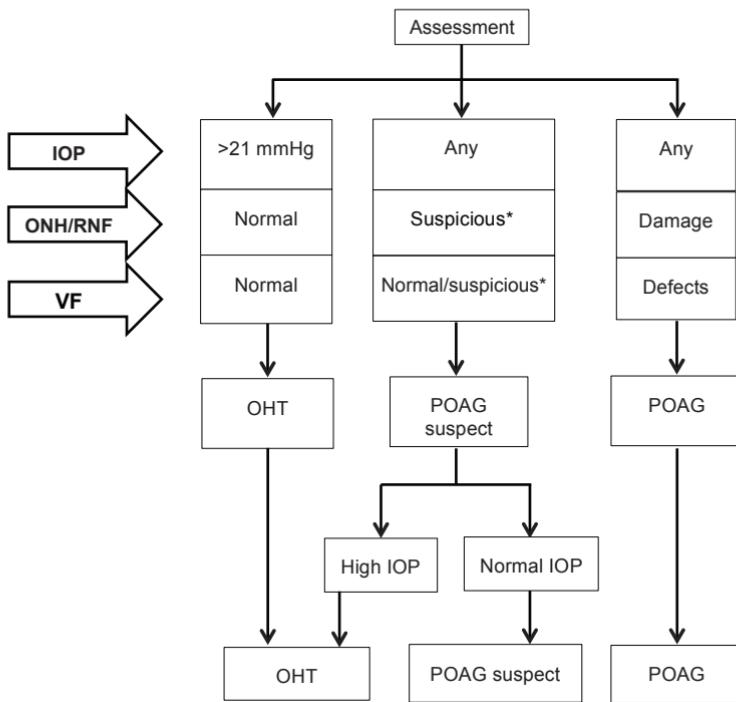
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ALGORITHM 1. CLASSIFICATION OF GLAUCOMA BASED ON ANGLE CONFIGURATION

- POAG = primary open angle glaucoma
- OHT = ocular hypertension
- NTG = normal tension glaucoma
- PAC = primary angle closure
- PACG = primary angle closure glaucoma

ALGORITHM 2. CLASSIFICATION OF PRIMARY OPEN ANGLE GLAUCOMA/OCULAR HYPERTENSION/ PRIMARY OPEN ANGLE GLAUCOMA SUSPECT

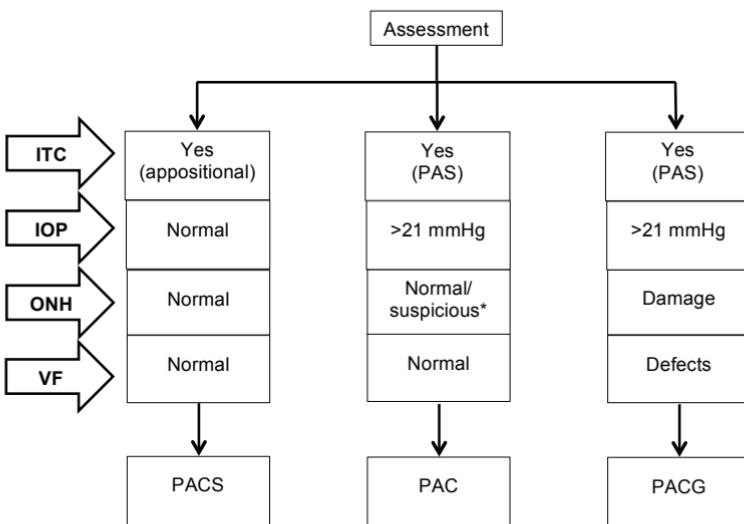


*ONH and/or peripapillary RNFL appearance and VF changes are suggestive of, but not definitive for glaucoma

IOP	= intraocular pressure
ONH	= optic nerve head
RNFL	= retinal nerve fiber layer
VF	= visual field
OHT	= ocular hypertension
POAG	= primary open angle glaucoma

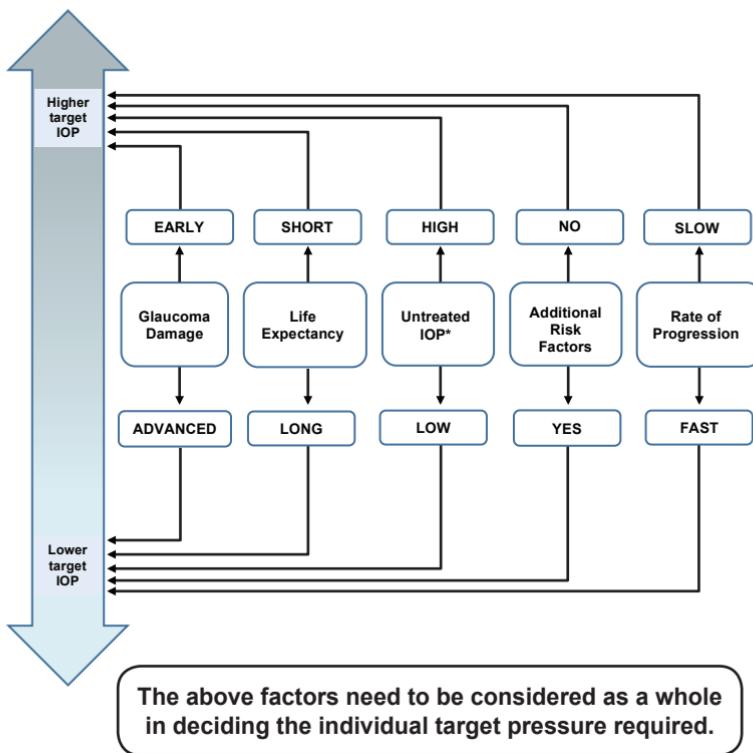
Modified: National Collaborating Centre for Acute Care. Glaucoma: Diagnosis and management of chronic open angle glaucoma and ocular hypertension. London: RCS; 2009

**ALGORITHM 3. CLASSIFICATION OF PRIMARY ANGLE CLOSURE
SUSPECT/PRIMARY ANGLE CLOSURE/
PRIMARY ANGLE CLOSURE GLAUCOMA**



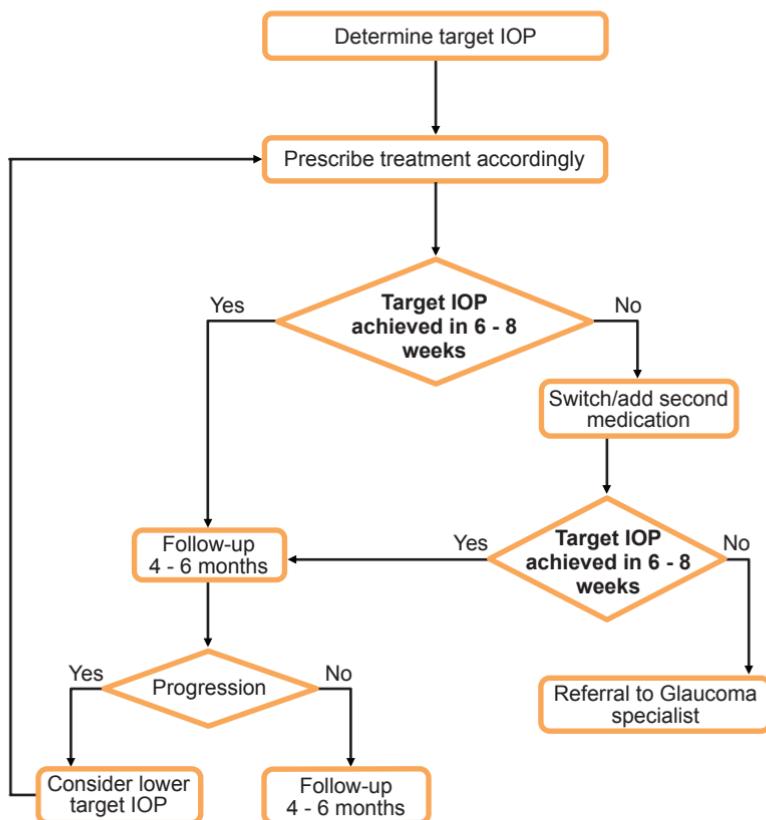
*ONH and/or peripapillary RNFL appearance is suggestive of, but not definitive for glaucoma

ITC	= iridotrabecular contact
IOP	= intraocular pressure
ONH	= optic nerve head
VF	= visual field
PAS	= peripheral anterior synechiae
PACS	= primary angle closure suspect
PAC	= primary angle closure
PACG	= primary angle closure glaucoma

ALGORITHM 4. SETTING THE TARGET IOP

*Consider central corneal

Source: European Glaucoma Society. Terminology and Guidelines for Glaucoma (4th Edition). Savona: Publicomm; 2014

ALGORITHM 5. ADJUSTMENT OF TARGET IOP

Modified: European Glaucoma Society. Terminology and Guidelines for Glaucoma (4th Edition). Savona: Publicomm; 2014

1. INTRODUCTION

Glaucoma is a chronic eye disease that damages the optic nerve, and can result in serious vision loss and irreversible blindness. It is the second leading cause of blindness worldwide.¹ Individuals with glaucoma are often asymptomatic and present at a late stage. Blindness from glaucoma is preventable if treatment is instituted early. The number of persons estimated to be blind as a result of primary glaucoma is 4.5 million, accounting for slightly more than 12% of all global blindness.² In Malaysia, the 2014 National Eye Survey II showed an estimated prevalence of blindness in those aged 50 and above was at 1.2% [presenting visual acuity (VA) <3/60 in the better eye] of which 6.6% was caused by glaucoma.³

As with any other chronic diseases, the management of glaucoma may be a burden on the economy. The direct and indirect economic impact of glaucoma on the population has been studied. The direct cost estimates for approximately two million citizens of United States of America and 300,000 citizens of Australia with glaucoma are USD2.99 billion⁴ and AUD144.2 million⁵ respectively. However, the true cost is probably higher as about half of the patients with glaucoma are unaware they have the disease.⁶⁻⁹

Resource use and direct cost of glaucoma management increase with worsening disease. The average direct cost of treatment ranges from USD623 per patient/year for glaucoma suspects or patients with early stage disease to USD2511 per patient/year for patients with end-stage disease. Medication costs composed the largest proportion of total direct cost for all stages of disease (24 - 61%).¹⁰ A similar trend is seen in Europe where resource utilisation and direct medical costs of glaucoma management increase with worsening disease severity. The direct cost of treatment is increased by an estimated €86 for each incremental step ranging from €455 per patient/year for stage 0 to €969 per patient/year for stage IV disease. Medication costs range from 42% to 56% of total direct cost for all stages of disease.¹¹

In East Asia, primary angle closure glaucoma (PACG) is an important cause of blindness. The cost of managing acute PACG in Singapore, which affects 12.2 per 100,000/year in those aged 30 and older is estimated annually to be USD261,742.¹²

The first edition of the Clinical Practice Guideline (CPG) published in 2008 was confined to POAG. Many new developments have emerged which led to variation in clinical practice. Based on this, the CPG has been updated and the scope widened to include PACG and special conditions. It aims to guide healthcare providers on evidence-based management of glaucoma in the Malaysian healthcare setting.

1.1 Epidemiology

Glaucoma is the leading cause of irreversible blindness. The global prevalence of POAG is 3.05% (95% CI 1.69 to 5.27) and PACG 0.50% (95% CI 0.11 to 1.36).^{13, level III} Overall prevalence of glaucoma in Asia is 3.54%. POAG is the predominant glaucoma subtype, followed by PACG and secondary glaucoma with prevalence of 2.34% (95% CI 0.96 to 4.55), 0.73% (95% CI 0.18 to 1.96%) and 0.47% (95% CI 0.09 to 1.48) respectively.^{14, level III}

Across ethnicity, people of African origin have the highest prevalence of glaucoma (6.11%, 95% CI 3.83 to 9.13) and POAG (5.4%, 95% CI 3.17 to 8.27). While Asians have the highest prevalence of PACG (1.2%, 95% CI 0.46 to 2.55).^{13, level III}

1.2 Definition and Classification

- Glaucoma is a chronic disease with progressive optic neuropathy characterised by morphological changes of the optic nerve head (ONH) and retinal nerve fibre layer (RFNL) with corresponding visual field (VF) defect.¹⁵
- In primary glaucoma, there are no identifiable causes.
- Based on the angle configuration, glaucoma is further divided into open angle and closed angle/angle closure.
 - In both types, there are risk factors i.e. ocular hypertension (OHT), primary open angle glaucoma (POAG) suspect, primary angle closure suspect (PACS) and primary angle closure (PAC). Refer to **Algorithm 1**.

- Primary open angle disease can be classified as:
 - OHT - presence of high IOP without glaucomatous optic neuropathy (GON) and VF changes.
 - POAG suspect - presence of normal IOP with suspicious ONH and/or VF changes.
 - POAG - presence of GON with corresponding VF defect.

Refer to **Algorithm 2** on **Classification of Primary Open Angle Glaucoma/Ocular Hypertension/Primary Open Angle Glaucoma Suspect.**

- Primary angle closure disease can be classified as:
 - primary angle closure suspect (PACS) - presence of appositional contact between peripheral iris and posterior trabecular meshwork (TM)
 - primary angle closure (PAC) - presence of occludable drainage angle with evidence of peripheral anterior synechiae (PAS), elevated intraocular pressure (IOP), iris whorling, glaukomflecken, lens opacities or excessive pigment deposition on TM without glaucomatous optic nerve damage
 - primary angle closure glaucoma (PACG) - PAC with evidence of glaucomatous optic nerve damage

Refer to **Algorithm 3** on **Classification of Primary Angle Closure Suspect/Primary Angle Closure/Primary Angle Closure Glaucoma.**

Modified: Foster PJ, Buhrmann R, Quigley HA, et al. The definition and classification of glaucoma in prevalence surveys. Br J Ophthalmol. 2002;86(2):238-42

PAC and PACG can present with or without acute angle closure (AAC). In Malaysian population, more than half do not present with AAC.^{16 - 17}, level III

2. RISK FACTORS

The identification of risk factors for POAG and PACS/PAC/PACG is important in their individual disease management.

2.1 Risk Factors for Primary Open Angle Glaucoma

2.1.1 Non-Ocular Factors

a. Advancing/older age

- Advancing age is a risk of POAG.^{18 - 20, level II-2}
- The overall prevalence of open angle glaucoma (OAG) by age was 0.3 (95% CI 0.1 to 0.5) in people aged 40 years and increased steeply to 3.3% (95% CI 2.5 to 4.0) in people aged 70 years.^{21, level I}

b. Ethnicity/race

- Individuals of West African, Afro-Caribbean or Hispanic/Latino ethnicity have a higher prevalence of POAG.²²

c. Positive family history among first-degree relatives (parents or siblings)

- Multiple genetic factors are likely to play a role.¹⁵
- Positive family history of glaucoma is associated with 3-fold increased risk of POAG.^{21, level I; 23, level III}
- Individuals with family history of glaucoma among siblings are four times more likely to develop glaucoma compared to two times for those whose parents have glaucoma. However, individuals whose children had a history of glaucoma are not at risk.²²

d. Obstructive Sleep Apnoea Syndrome

- Obstructive Sleep Apnoea (OSA) syndrome is associated with significantly increased risk of glaucoma.^{24, level III; 25, level II-2; 26, level III; 27, level II-2}

e. Diabetes mellitus

- Diabetes mellitus (DM) is associated with an increased risk of glaucoma (RR=1.37, 95% CI 1.20 to 1.57).^{28, level II-2}

2.1.2 Ocular factors

a. Intraocular pressure

- Elevated IOP significantly increases risk of POAG.^{19 - 20, level II-2; 23, level III}
- The diurnal variation of IOP is greater in glaucoma patients compared with normal individuals.²²
- Asymmetry of IOP by ≥ 3 mmHg between the two eyes is suspicious of glaucoma.²²

b. Central Corneal Thickness

- Thinner central corneal thickness (CCT) is associated significantly with risk of glaucoma.^{19 - 20, level II-2}

c. Myopia

- Myopia is significantly more prevalent in patients with POAG.^{23, level III}
- Myopic refractive error > -6 D may be a risk factor associated with glaucomatous optic neuropathy.^{29, level III}

d. Corneal hysteresis

- Corneal hysteresis is a risk factor for progression of glaucoma.^{30, level II-2; 31, level III}

2.1.3 Other Possible Risk Factors

- Higher systolic and diastolic blood pressure^{23, level III}
- Higher systolic perfusion pressure^{23, level III}
- Larger vertical CDR^{19 - 20, level II-2}
- Larger vertical CDR asymmetry^{19, level II-2}
- Higher pattern standard deviation (PSD) on Humphrey VF test^{19 - 20, level II-2}
- Male gender^{18, level II-2}

2.2 Risk Factors for Primary Angle Closure Suspect/Primary Angle Closure/Primary Angle Closure Glaucoma**2.2.1 Non-Ocular Factors****a. Advancing/older age**

- Increasing age is a risk factor for angle closure disease.^{32 - 34, level III}

b. Positive family history

- OR=1.65, 95% CI 1.16 to 2.34^{33, level III}

2.2.2 Ocular Factors**a. Elevated intraocular pressure**

- Increased IOP is a risk factor for PACG.^{32 - 33, level III}

b. Biometric measurement

These include:

- Decreasing axial length^{34, level III}
- Decreasing anterior chamber depth^{34, level III}
- Increased choroidal thickness^{35, level III}

2.2.3 Other possible risk factors

- DM (OR=3.18, 95% CI 1.34 to 7.58)^{32, level III}
- Female (OR=2.07, 95% CI 1.09 to 3.9)^{32, level III}
- Nuclear cataract (OR=1.23, 95% CI 1.01 to 1.48)^{34, level III}

Recommendation 1

- Risk factors* should be identified in the management of glaucoma.

*Refer to the preceding text.

3. SCREENING

Screening of glaucoma aims to identify individuals at risk or early stage of the disease to prevent or reduce blindness.

There is no strong evidence to advocate population-based screening for glaucoma.^{22; 36, level I; 37, level III}

However, screening should be considered for higher risk groups at the age of 40 years and be carried out by trained healthcare providers.^{21, level I; 22; 37, level III}

There is no single test or a group of tests that is accurate for glaucoma screening.^{38, level II-2} However, combination of optic disc (OD) assessment, RNFL assessment, tonometry and VF may be used.

Recommendation 2

- Population-based screening for glaucoma is not advocated.
- Screening of glaucoma should be considered for patients with risk factors especially:
 - age >40 years
 - family history of glaucoma
 - diabetes mellitus

Refer to **Chapter on Risk factors** for further details.

4. DIAGNOSIS

- Most glaucoma patients are asymptomatic. Their VA can remain normal even at advanced stages of the disease. As such, they are usually diagnosed during routine visit to eye care professionals for other eye problems.

The diagnosis of glaucoma is based on history (including presence of risk factors) and eye examination (ONH, RNFL, VF, IOP and gonioscopy). Certain investigations may help in the diagnosis of glaucoma. Confirmation of the diagnosis may require more than one visit.²²

4.1 History²²

4.1.1 Symptoms

- Asymptomatic
 - Reduced vision (acute and/or advanced glaucoma)
 - Ocular pain, redness, haloes)
 - Headache, nausea, vomiting
 - VF defect
- } mainly in acute angle closure

4.1.2 Ocular history

- History of ocular surgery, ocular trauma and use of topical steroids
- Refractive status; myopia (POAG), hyperopia >3 Diopter (angle closure)
- Refractive surgery

4.1.3 Systemic history

- Systemic history of DM, hypertension, migraine, Raynauld's phenomenon and use of systemic steroids
- OSA syndrome

4.1.4 Family history of glaucoma

4.1.5 Other risk factors (refer to Chapter 2)

4.2 Examination²²

4.2.1 Visual acuity

- Usually normal but may be reduced in acute or advanced glaucoma

4.2.2 Pupil

- Mid-dilated pupils (in AAC)
- Posterior synechiae
- Relative afferent pupillary defect in asymmetrical cases

4.2.3 Anterior chamber and lens

- Depth of anterior chamber (shallow in patient with PAC)
- PAS
- Iris whorling and/or atrophy
- Anterior subcapsular opacities (glaukomflecken)
- Cataract

4.2.4 Intraocular pressure measurement

- IOP should be measured preferably with Goldmann applanation tonometry (GAT) which is the gold standard.
- In non-ambulatory patients and those who have corneal disease, alternative tonometry such as tonopen, rebound tonometer etc. can be used.
- CCT needs to be measured with a pachymeter.
- Time of measurement should be recorded as IOP varies at different times of the day; peaks are often before noon.

- Acute angle closure (AAC) is diagnosed when IOP >21 mmHg with occludable angle in the presence of:
 1. any two of the following symptoms
 - ocular and periocular pain
 - nausea and/or vomiting
 - history of blurring of vision and haloes

AND

2. any three of the following signs
 - conjunctival injection
 - corneal epithelial oedema
 - mid-dilated unreactive pupil
 - shallow anterior chamber

4.2.5 Assessment of the angle

- Slit Lamp Examination of angles (Van Herick test, a slit lamp estimation of the angle depth - refer to **Appendix 3**)
- Gonioscopy
 - Evaluation of the angle is needed to determine the type of glaucoma.
 - The angle is considered open when gonoscopic findings (without indentation) show grades III or IV based on Shaffer's classification and it is considered narrow at grade II or less.

Refer to **Table 1** on **Gonioscopic chart and grading system**.

- Gonioscopy should always be performed in a dark room with a narrow slit beam, making sure that the beam does not enter the pupil which can cause the pupil to constrict.^{39, level III}
 - Indentation/dynamic gonioscopy is essential to:
 - differentiate appositional from synechial angle closure in patients with suspected angle closure
 - detect plateau iris configuration
- Refer to **Appendix 4** on **Indentation/Dynamic Gonioscopy**.

Table 1. Gonioscopic chart and grading system for gonioscopic findings (without indentation)

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

*TM=trabecular meshwork

Source: Asia-Pacific Glaucoma Society. Asia Pacific Glaucoma Guidelines (Third Edition). Amsterdam: Kugler Publication; 2016

4.2.6 Assessment of the optic disc and retinal nerve fibre layer

- Assessment should be done through a dilated pupil unless contraindicated. It can be done with:
 - slit lamp biomicroscopy using high power condensing lens (recommended method)
 - stereoscopic OD photography
 - red free illumination (either on slit lamp or photography)
 - direct ophthalmoscopy (limited to OD assessment)
- The OD should be examined for (refer to **Appendix 4**):
 - OD size
 - increase in the vertical CDR*
 - Inferior Superior Nasal Temporal (ISNT) rule (whereby the thickest neuro-retinal rim (NRR) is inferior and thinnest is temporal)
 - NRR notching or acquired pit of the OD*
 - asymmetry CDR between OD $>0.2^*$
 - OD haemorrhage
 - nasalisation and bayonetting of retinal vessels
 - peripapillary beta zone atrophy
- RNFL should be examined for thinning or loss (slit/wedge defects)**. Refer to **Appendix 5**.

*features that are indicated for **referral by primary care providers** to ophthalmology service

feature seen in fundus photo for **referral by primary care providers to ophthalmology service

4.3 Investigation

4.3.1 Automated visual field analysis*

- Standard Automated Perimetry (SAP)
 - Automated static threshold perimetry is currently the gold standard for VF assessment.
 - Commonly used threshold algorithms are Swedish Interactive Threshold Algorithm (SITA) Standard and SITA Fast in the Humphrey perimeter. Other available algorithms such as “Dynamic Strategy” in the Octopus perimeter may be used.
 - For those with very advanced disease, it may be necessary to consider:
 - Goldmann size V stimulus rather than size III
 - a perimetric strategy which focuses more closely on the remaining area of VF (Octopus M1 or M2 or the Humphrey 10-2)
- Non-conventional perimetry
 - There is insufficient evidence that Short Wave Automated Perimetry and Frequency Doubling Threshold Perimetry has any advantage over SAP.¹⁵

*In VF assessment, the tests must be reliable and reproducible.

VF defects suggestive of POAG are:

- classical defects - paracentral scotoma, nasal step and arcuate scotoma; temporal wedge is an uncommon feature
- early defects on SAP
 - Glaucoma hemifield test (GHT) graded as outside normal limits
 - a minimum of three clustered points (non-edge points) with significantly depressed sensitivity, of which one should have a significance of $p < 1\%$ on the pattern deviation plot
 - p -value of PSD $< 5\%$

4.3.2 Optic nerve head and retinal imaging

- In centres with Optical Coherence Tomography (OCT) machines, global RNFL is the best parameter to detect early glaucomatous structural damage.^{40, level III}
- Confocal scanning laser ophthalmoscopy (CSLO) may be used to detect structural change and the rate of rim area loss.^{41, level I}

4.3.3 Central Corneal Thickness

- Mean CCT measurement is $540 \pm 30 \mu\text{m}$ (mean \pm standard deviation in μm).¹⁵
- CCT affects IOP measurement. Thus, measurement of CCT is important as it aids in patient's management.²² However there is no validated conversion table.
- Thin CCT will result in falsely low IOP readings and vice versa.²²

4.3.4 Anterior Segment Optical Coherence Tomography

- Anterior Segment Optical Coherence Tomography (AS-OCT) is a rapid non-contact method of imaging the angle structures anterior to the ciliary body.

- AS-OCT tends to detect more angle closure compared with gonioscopy (gold standard).^{42, level III}

4.4 Staging of Glaucoma

The staging of glaucoma is based on SAP as shown in **Table 2.**

Table 2. Staging of Glaucoma

Stage: Humphrey MD score	Additional Criteria (at least one of the listed criteria must apply)
Stage 0: No or Minimal Defect	<ul style="list-style-type: none"> Does not meet any criteria for Stage 1
Stage 1: Early Defect < -6.00 dB	<ul style="list-style-type: none"> A cluster of ≥ 3 points on the pattern deviation plot in an expected location of the VF depressed below the 5% level, at least one of which is depressed below the 1% level Corrected PSD/PSD significant at $p < 0.05$ GHT outside normal limits
Stage 2: Moderate Defect ≥ -6.00 to -12.00 dB	<ul style="list-style-type: none"> $\geq 25\%$ but $< 50\%$ of points on the pattern deviation plot depressed below the 5% level, and $\geq 15\%$ but $< 25\%$ of points depressed below the 1% level At least one point within the central 5° with sensitivity of < 15 dB but no points in the central 5° with sensitivity of < 0 dB Only one hemifield containing a point with sensitivity < 15 dB within 5° of fixation
Stage 3: Advanced Defect ≥ -12.01 to -20.00 dB	<ul style="list-style-type: none"> $\geq 50\%$ but $< 75\%$ of points on pattern deviation plot depressed below the 5% level and $\geq 25\%$ but $< 50\%$ of points depressed below the 1% level Any point within the central 5° with sensitivity < 0 dB Both hemifields containing a point(s) with sensitivity < 15 dB within 5° of fixation
Stage 4: Severe Defect ≥ -20.00 dB	<ul style="list-style-type: none"> $\geq 75\%$ of points on pattern deviation plot depressed below the 5% level and $\geq 50\%$ but $< 50\%$ of points depressed below the 1% level At least 50% of points within the central 5° with sensitivity < 0 dB Both hemifields containing $> 50\%$ of points with sensitivity < 15 dB within 5° of fixation
Stage 5: End-Stage Disease	<ul style="list-style-type: none"> Unable to perform HVF in worst eye due to central scotoma or worst eye VA 6/60 or worse due to POAG. Fellow eye may be at any stage.

Source: Mills RP, Budenz DL, Lee PP et al. Categorizing the stage of glaucoma from pre-diagnosis to end-stage disease. Am J Ophthalmol. 2006; 141(1):24-30

Recommendation 3

- Glaucoma diagnosis should be made based on a combination of history (with presence of risk factors), ocular examination and investigation.
 - Essential ocular examination includes intraocular pressure measurement, optic disc assessment and gonioscopy.
 - Essential investigations include reliable visual field test.

5. TREATMENT

The aim of glaucoma treatment is to preserve maximal functional vision throughout a patient's lifetime without sacrificing his/her quality of life (QoL) and at a sustainable cost. QoL is affected by visual function, treatment regime and its adverse effects, financial burden of the treatment and the psychological effect of having a potentially blinding disease.

5.1 Principles of Treatment

Currently, lowering of IOP is the only proven efficient approach in preventing progression of glaucoma.²² The risk of progression is decreased by 10% with each mmHg of IOP reduction from baseline to the first follow-up visit (HR=0.90 per mmHg decrease, 95% CI 0.86 to 0.94).^{43, level I} IOP lowering can be achieved by either medication, laser treatment, surgery or any combination of these modalities.

Target IOP is an estimate of mean IOP at which further glaucomatous damage is likely to be prevented. It should be tailored to individual patients and may be adjusted during the course of the disease. It is set based on the following factors:²²

- pre-treatment IOP
- stage of optic nerve damage and VF defects
- rate of glaucoma progression
- age, life expectancy and visual requirements of the patient
- presence of glaucoma risk factors

Refer to **Algorithm 5 on Setting the Target IOP**.

In ocular hypertension (OHT), a treatment target IOP of ≤24 mmHg and IOP reduction of ≥20% from baseline is recommended.^{44, level I}

In established glaucoma:

- a 25% IOP reduction from baseline is protective against progression in early (mild) glaucoma^{43, level I}
- non-progression is seen in mild to moderate glaucoma with a mean IOP of 16.5 mmHg^{45, level I}
- an IOP of >14 mmHg is associated with greater worsening of VF defect in advanced stage^{46, level II-2}
- a 30% IOP reduction from baseline is protective against progression in NTG^{47, level I}
- non-progression is seen in mild to moderate PACG with IOP <12 mmHg in a local Malaysian population^{48, level II-2}

Based on the above findings and existing guidelines,^{15; 49} the following target IOP is suggested by the CPG DG (refer to **Table 3**).

Table 3. Suggested target IOP according to stage of glaucoma

Stage of glaucoma	Target IOP
OHT (if decision is made to treat)	≤24 mmHg with reduction of at least 20% from baseline IOP
Early (mild) disease	≤20 mmHg with reduction of at least 25% from baseline IOP
Moderate disease	≤17 mmHg with reduction of at least 30% from baseline IOP
Advanced disease	≤14 mmHg with reduction of at least 30% from baseline IOP

- Target IOP in glaucoma
 - should be individualised.
 - can be adjusted throughout the management of the condition*

*Refer to **Algorithm 5 on Setting the Target IOP**.

In AAC, the aims of treatment are to lower the IOP urgently and relieve the acute symptoms. These are achieved by multiple modalities. Refer to **Appendix 6**.

5.2 Medical Treatment

Medical treatment is usually the initial treatment of choice in glaucoma. It includes the use of topical and systemic anti-glaucoma medications that increase aqueous outflow, reduce aqueous production or both.

- Criteria for prescribing anti-glaucoma medications are:²²
 - patient's characteristics (stage of glaucoma, age, risk factors, co-morbidities, compliance, psychological and socio-economic status)
 - drug factors (efficacy, safety, dosing regimen, preservative/non-preservative, cost and availability of the drug)

There are six main pharmacologic classes of anti-glaucoma medications which can be used as monotherapy or in combination therapy. They are:

- prostaglandin analogues
- beta-blockers
- adrenergic agonists
- carbonic anhydrase inhibitors (CAIs)
- cholinergic agents
- osmotic agents

5.2.1 Prostaglandin analogues

Prostaglandin analogues are the first choice/first-line medications in glaucoma treatment. They have the highest IOP lowering effect and minimal systemic side effects among all topical anti-glaucoma medications. Their once daily dosing enhances compliance.²²

In a good meta-analysis of high quality RCTs, bimatoprost was significantly more efficacious in lowering IOP compared with latanoprost and travoprost at different time points in a day for POAG and OHT at 1 - 6 months follow-up.^{50, level I} The results on efficacy were supported by three meta-analysis conducted after that.^{51 - 53, level I}

A meta-analysis of 15 RCTs on normal tension glaucoma showed that prostaglandin analogues (latanoprost and bimatoprost) were the most significantly efficacious IOP-lowering medications compared with other medications on short-term follow-up (0.5 - 3 months).^{54, level I}

In chronic angle closure glaucoma (or PACG), prostaglandin analogues (latanoprost, travoprost and bimatoprost) are significantly efficacious in lowering IOP from baseline at 1 - 3 months and the IOP reduction is greater than timolol.^{55, level I}

In terms of side effects, the incidence of hyperaemia is significantly more in bimatoprost compared with latanoprost and travoprost.^{50, level I; 56, level I}

5.2.2 Beta-blockers

Beta-blockers are commonly used as anti-glaucoma medications. Currently, they are used as either first- or second-line medications in patients with no contraindications. There are two types of beta-blocker available which are selective and non-selective agents.

Beta-blockers are efficacious as monotherapy^{51, level I; 54, level I; 55 - 57, level I} and adjunctive therapy^{58, level I} in all types of glaucoma.

Topical beta-blockers can be the first-line medications when considering medical treatment of glaucoma in pregnancy. There is no significant difference in the risk of low birth weight infants between mothers prescribed with beta-blockers and those in the comparison cohort (OR=1.48, 95% CI 0.86 to 2.56).^{59, level III}

- Beta-blockers are contraindicated in individuals with:
 - respiratory problems such as asthma and chronic obstructive pulmonary disease
 - cardiac problems such heart block, cardiac failure and bradycardia
- Important systemic side effects of beta-blockers are bronchospasm, bradycardia, cardiac failure and syncope.

5.2.3 Adrenergic agonists

Brimonidine is efficacious as an adjunctive therapy^{58, level I} but in selected cases, it may be used as a monotherapy. Its common side effect is ocular allergy.

- Adrenergic agonists are contraindicated in individuals on monoamine oxidase inhibitor therapy.
- Side effects of adrenergic agonists include burning sensation and hyperaemia.

5.2.4 Carbonic anhydrase inhibitors

CAIs are available in topical and systemic forms. They are efficacious as an adjunctive therapy.^{58, level I} In selected cases, they may be used as monotherapy. Long-term use of systemic CAIs is not advisable due to their possible serious side effects.

- CAIs are contraindicated in individuals with:
 - sulphonamide allergy
 - renal calculi or failure
 - respiratory/metabolic acidosis and hypokalaemia
- Important systemic side effects of CAIs are:
 - Stevens-Johnson syndrome
 - angioedema
 - metabolic acidosis
 - electrolyte imbalance
 - blood dyscrasias

5.2.5 Cholinergic agents

Topical pilocarpine is mainly used as short-term, pre-laser treatment of patients with narrow angle. Its long-term use is not favoured due to its side effects (such as blurred vision, peri-orbital pain and stinging) and frequent dosing i.e. four times a day.²²

5.2.6 Osmotic agents

Osmotic agents are only available as systemic therapy. They are usually used in acute situations when rapid IOP reduction is desired. Commercially available agents are oral glycerol and intravenous mannitol.²²

- Osmotic agents are contraindicated in individuals with:
 - cardiac disease
 - renal disease
- Important systemic side effects of osmotic agents are:
 - dehydration
 - cardiac failure
 - hyponatraemia
 - acute renal failure
- Osmotic agents may cause hyperglycaemia in diabetic patients.

5.2.7 Fixed combination medications

Several fixed combination medications have been developed to maximise patient's compliance and QoL. Most fixed combination medications contain timolol as one of its components.

The six commonly used fixed combination medications significantly reduce IOP from baseline and the combination of prostaglandin analogue and timolol have the highest IOP reduction between 33.9% and 34.9%.^{60, level I}

Fixed combination medications are equally safe and efficacious at lowering IOP compared with their non-fixed components. Three main ocular side effects are hyperaemia, ocular irritation and keratitis which are more frequent in non-fixed combination.^{61, level I}

Fixed combination provides significantly improved long-term adherence compared with non-fixed combination in glaucoma treatment.^{62, level I}

Combination treatment, either as fixed or non-fixed, is not recommended as first-line treatment.¹⁵

General Principles in Medical Treatment in Glaucoma²²

- Determine an individualised target IOP and readjust treatment if necessary throughout the course of disease.
- Diagnostic parameters to be considered in deciding on anti-glaucoma medications are:
 - IOP levels and/or fluctuations
 - extent of OD damage
 - severity of VF defects
 - CCT
- Baseline parameters should be collected prior to initiating treatment and verified before modifying treatment.
- Choose monotherapy that:
 - provides greatest IOP lowering effects to achieve target IOP
 - has the best safety profiles e.g. least side effects, good tolerability, etc.
 - enhances compliance e.g. simple dosing regimen, minimal disruption to QoL, etc.
 - is available and affordable
- Treatment
 - Treatment is considered effective when the individual target IOP is achieved.
 - If the target IOP is not achieved:
 - switch within prostaglandin analogue or to a different class of medication
 - use adjunctive treatment either non-fixed or fixed combination
 - Generally, if more than two medications are required for IOP control; other forms of treatment should be considered.
- Patient education is important and it includes:
 - nature of the disease
 - benefits and side effects of treatment
 - proper instillation technique of eye drop*
 - importance of compliance (adherence, persistence and follow-up)

*Refer to Appendix 7 on Proper Instillation Technique of Eye Drop.
Refer to Appendix 8 on Main Features of Topical Anti-Glaucoma Medications.

Recommendation 4

- In glaucoma treatment, healthcare providers should aim to maintain maximal functional vision throughout a patient's lifetime without sacrificing his/her quality of life and at a sustainable cost.
- Medical treatment in glaucoma should be individualised based on patient's characteristics and drug factors, and adjusted according to target intraocular pressure (IOP).
- Prostaglandin analogues should be used as first-line treatment in glaucoma.
- If the target IOP is not achieved in glaucoma, consider switching anti-glaucoma medications or adding adjunctive treatment either non-fixed or fixed combination.
- Patient education should be given to patients with glaucoma. This includes benefits and side effects of treatment, proper instillation technique of eye drop and compliance to treatment.

5.2.8 Neuroprotection in glaucoma

There is insufficient evidence to support that neuroprotective medications are effective in preventing retinal ganglion cell death, and thus preserving vision in people with OAG.^{63, level I}

5.3 Laser Treatment

Laser treatment has become important in the management of glaucoma. It is indicated when medical therapy fails, as an adjunct or as a primary treatment where appropriate. There are several types of laser treatment used to treat glaucoma as shown below.

Types of Laser Treatment in Different Types of Glaucoma

- **Open Angle Glaucoma**
 - Laser trabeculoplasty for outflow enhancement
 - Trans-scleral cyclophotocoagulation (TSCP) for inflow reduction (usually for end-stage disease)
- **Angle Closure (\pm Glaucoma)**
 - Laser iridotomy for pupillary block relief
 - Laser peripheral iridoplasty for modification of iris contour
 - TSCP for inflow reduction (usually for end-stage disease)
- **Post-Filtering Surgery**
 - Laser suture lysis for outflow enhancement

Source: Asia-Pacific Glaucoma Society. Asia Pacific Glaucoma Guidelines (Third Edition). Amsterdam: Kugler Publications; 2016

5.3.1 Laser trabeculoplasty

Laser parameters used for Selective laser trabeculoplasty (SLT) and Argon laser trabeculoplasty (ALT) are shown in **Table 4**.

Laser trabeculoplasty is initially efficacious in 80 - 85% of treated eyes with a mean IOP reduction of 20 to 25% (6 - 9 mmHg).^{64, level III} It is also efficacious and safe in OHT.^{65, level I}

In a meta-analysis of six RCTs, there was no significant difference of IOP reduction between SLT and ALT up to two years in patients who were naive to laser (first laser trabeculoplasty). There was also no significant difference in adverse events between the two modes of treatment.^{66, level I}

These were supported by another meta-analysis in 2015, although one of the RCTs included patients treated with ALT. The meta-analysis also showed no difference in efficacy between SLT and medications. The overall safety profile of SLT was good, with most side effects being transient and amenable to medical therapy.^{67, level I}

In patients treated previously with ALT or SLT, retreatment with SLT is efficacious and safe in further lowering IOP.^{68, level II-2; 69, level I; 70, level II-1} SLT 360° is a more effective treatment compared with 90° or 180°.^{71, level I}

Higher baseline IOP is the main predictor for the success of laser therapy in both SLT^{72, level I} and ALT.^{46, level II-2; 73, level II-2; 74, level III}

Indications for laser trabeculoplasty in open angle glaucoma are:^{64, level III}

- failure or intolerance of medical therapy
- adjunct to medical therapy
- primary treatment if appropriate

Complications of laser trabeculoplasty are:^{64, level III}

- temporary blurred vision
- IOP spike with possible VF loss
- transient iritis
- PAS if placement of burns is too posterior or post-laser inflammation control is not effective (in ALT)
- endothelial burns if treatment is too anterior (in ALT)
- chronic increase in IOP
- corneo-refractive changes
- suprachoroidal effusion

5.3.2 Laser iridotomy

Laser iridotomy, a relatively non-invasive procedure is efficacious in relieving pupillary block. Complications of laser iridotomy are IOP spikes, temporary blurring of vision and corneal burn. These could be minimised by using proper technique.^{64, level III}

Indications for laser iridotomy in angle closure disease are:^{64, level III}

- PAC
- PACG
- PACS:
 - absolute indication - PAC in the fellow eye
 - relative indication - need for repeated dilated examination e.g. diabetic patients
 - poor access to regular ophthalmic care
 - confirmed family history of PACG

Refer to **Appendix 9 on Laser Iridotomy**.

5.3.3 Argon laser peripheral iridoplasty

Argon laser peripheral iridoplasty (ALPI) is a non-invasive procedure. It contracts the peripheral iris which results in widening of the anterior chamber angle and re-opens the appositionally closed segments. However, ALPI is associated with higher failure rates and lower IOP reduction compared with prostaglandin analogues in eyes with persistent appositional angle closure and raised IOP after laser iridotomy.^{75, level I}

Indications for ALPI in angle closure disease are as follows:^{64, level III}

- initial treatment in acute angle closure attack
- adjunctive measure when systemic medications fail to control IOP
- persistent occludable angle following laser iridotomy
- facilitate access to TM for laser trabeculoplasty
- an adjunct to goniosynechialysis

ALPI is contraindicated in area with PAS.

5.3.4 Cyclophotocoagulation

Cyclophotocoagulation (CPC) is a cyclodestructive procedure that reduces aqueous production by the ciliary epithelium. The procedure may be performed transscleral or endoscopically.

CPC is indicated in the following:^{64, level III}

- painful blind eyes or eyes with poor vision
- failed multiple filtering surgeries

It is also may be performed in sighted eyes when the benefits outweigh the risks for incisional surgery.^{76, level III}

Complications include:^{64, level III}

- pain
- persistent inflammation
- loss of visual acuity
- hypotony and phthisis
- scleral thinning or rupture
- pupillary distortion

- macular oedema
- retinal detachment
- aqueous misdirection syndrome
- sympathetic ophthalmia

Table 4 shows the summary of parameters and lenses for laser treatment.

Table 4. Parameters and lenses for laser treatment

Parameters	Laser procedures				
	SLT	ALT	Peripheral iridoplasty	Trans-scleral CPC	Laser iridotony*
Number of spots	30 - 50 (over 180°)	50 (over 180°)	10 - 40 (over 360°)	20 - 40 (over 180 - 360°)	
Spot size	400 µm	50 µm	500 µm	-	
Exposure time	3 nsec	0.1 sec	0.5 sec	0.5 - 2 sec	
Fluence (mJ/mm ²)	6	40,000	-	-	
Power	0.4 - 1.4 mJ	300 - 600 mW	150 - 240 mW		
Type of lenses	Latina SLT lens, Goldmann gonioscopy lens, Ritch trabeculoplasty lens, CGA© LASAG/Meridien CH, Magna View Gonio lens		Abraham (+66 D), Wise (+103 D), CGI©LASAG CH lens, central non-mirrored part of the Goldmann lens	Not needed	

*Refer to **Appendix 9**.

Source: Asia-Pacific Glaucoma Society. Asia Pacific Glaucoma Guidelines (Third Edition). Amsterdam: Kugler Publications; 2016

Recommendation 5

- Laser trabeculoplasty should be considered in primary open angle glaucoma when indicated*.
- Laser iridotomy should be performed in primary angle closure disease when indicated**.
- Peripheral iridoplasty may be considered for initial treatment in acute angle closure.
- Cyclophotocoagulation should be considered in refractory glaucoma.

*Refer to the preceding text.

**Refer to the preceding text.

5.4 Surgical Treatment

Surgery is indicated in glaucoma when the target IOP cannot be reached despite maximal medical therapy or when there is intolerance or non-compliance to medical therapy.²²

Available surgical options include trabeculectomy, implantation of glaucoma drainage devices (GDD) and non-penetrating glaucoma surgery.

5.4.1 Trabeculectomy

Trabeculectomy is the primary surgery of choice in POAG.²² However, the development of fibrosis may lead to failure of the surgical procedure. Thus, antimetabolites such as Mitomycin C (MMC) and 5-Fluorouracil are used to improve the success rate.²² Intraoperative MMC reduces the risk of surgical failure in primary trabeculectomy (RR=0.29, 95% CI 0.16 to 0.53).^{77, level I} However, the use of antimetabolites may increase complications such as bleb leaks, hypotony, late-onset blebitis and endophthalmitis.²²

5.4.2 Lens extraction

Clear lens extraction has beneficial effect in PAC and PACG with IOP >30 mmHg in terms of QoL ($p=0.005$) and IOP control ($p=0.04$) compared with standard laser PI. There is no significant serious adverse events reported.^{78, level I} This procedure should only be performed by an experienced surgeon.

In medically-controlled PACG with co-existing cataract, there are no significant differences in mean IOP between phacoemulsification alone and combined phacotrabeculectomy with adjunctive MMC at 6 - 24 months. However, phacoemulsification alone is associated with less post-operative complications ($p<0.001$).^{79, level I}

5.4.3 Combined trabeculectomy and lens extraction

In medically-uncontrolled PACG with coexisting cataract, combined phacotrabeculectomy with adjunctive MMC results in significantly lower mean IOP than phacoemulsification alone at three, 15 and 18 months of follow-up. However, combined surgery is associated with higher post-operative complications ($p<0.001$) and possible progression of optic neuropathy ($p=0.03$).^{80, level I}

In glaucoma patients who require cataract and filtering surgery, performing cataract surgery after trabeculectomy significantly increases the rates of bleb failure. However, the risk of failure is reduced if lens extraction is performed >6 months post-trabeculectomy.^{64, level III}

5.4.4 Glaucoma drainage devices

The use GDD such as Baerveldt, Ahmed and Molteno are usually reserved for patients with high risk of failure from augmented trabeculectomy. This includes eyes with previous failed filtering surgery, severe conjunctival or ocular surface diseases, active neovascular diseases, paediatric glaucomas or excessive conjunctival scarring.²²

Ahmed and Baerveldt implants are as efficacious as trabeculectomy in IOP reduction.^{81 - 82, level I} Although Molteno implant provides better and sustained IOP reduction,^{83, level II-2} there is no clinical trial to support this. Baerveldt implant has a higher qualified success rate than trabeculectomy at 5-year follow-up.^{82, level I}

Based on two multi-centre RCTs, Baerveldt implant had greater IOP reduction compared with Ahmed implant at 3 - 5 years of follow-up. However, serious complications occurred less frequently in Ahmed implant.^{84 - 85, level I} Ahmed and Baerveldt implants have similar rate of complications compared with trabeculectomy.^{81 - 82, level I} However, a lower rate of persistent hypotony has been reported with Baerveldt implant compared to trabeculectomy.^{82, level I}

5.4.5 Others (non-penetrating glaucoma surgery and minimally invasive glaucoma surgery)

Deep sclerostomy and viscocanalostomy are types of non-penetrating glaucoma surgery.²² There is limited evidence on viscocanalostomy or deep sclerostomy resulting in better IOP control compared with trabeculectomy.^{86, level I} Thus it is not suitable for patients who need low target IOP. Its advantages over trabeculectomy include lower risk of post-operative hypotony and bleb infection.²²

Minimally invasive glaucoma surgery is a group of procedures which include ab-interno and ab-externo techniques which aims for less tissue manipulation and side effects compared with standard filtering surgery. However, there is no good evidence to show its efficacy.¹⁵

Recommendation 6

- Intraoperative Mitomycin C during trabeculectomy should be used in glaucoma patients at risk of surgical failure.
- Early lens extraction may be considered as first-line treatment in primary angle closure and primary angle closure glaucoma.
- Glaucoma drainage devices may be offered in the treatment of refractory glaucoma.

6. FOLLOW-UP

Glaucoma patients need to be followed-up to monitor the effects of treatment, and to detect disease progression and any changes in patient's risk profile and systemic health that may affect glaucoma management plan.²²

Currently there is no retrievable evidence on this matter. The CPG DG uses existing guidelines^{15; 64, level III; 87; 88} and their expert opinion to address issues on follow-up schedule of glaucoma patients.

During each review, the following should be elicited:²²

- History
 - Ocular history
 - Medical and drug history
 - Local and systemic problems with ocular medications
 - General assessment of the impact of visual function on daily living
 - Frequency and time of last IOP lowering medications
 - Verification of compliance
- Ocular examination
 - VA in both eyes
 - IOP measurement in both eyes
 - Slip lamp for external eye examination
 - ONH evaluation
 - Gonioscopy, VF, ONH and RFNL (imaging/photography) are done according to the follow-up schedule or when indicated

The following are the recommended follow-up schedule for patients with glaucoma based on the target IOP and disease progression (refer to **Table 5**). However, it should be individualised according to the severity of disease and risk factors.

Table 5. Follow-up schedule for glaucoma and related conditions

Diagnosis	Follow-up schedule
OHT	Refer to Subchapter 10.1
POAG suspect	Refer to Subchapter 10.2
POAG and PACG	Target IOP achieved: No : 1 - 2 months Yes : 6 - 12 months
	Disease progression (structural and functional): Yes : 3 - 6 months No : 6 - 12 months

*Disease progression supersedes target IOP in determining interval of follow-up.

The following are the recommended investigations schedule for glaucoma patients and related conditions (refer to **Table 6**).

Table 6. Investigations schedule for glaucoma and related conditions

Diagnosis	VF	Gonioscopy	OD image	CCT
OHT	Refer to Subchapter 10.1			
POAG Suspect	Refer to Subchapter 10.2			
Open angle glaucoma	Stable: Yearly Progression: 4 - 8 months	Baseline and when indicated	Every 1 - 2 years	Baseline or when indicated
Angle closure glaucoma	Stable: Yearly Progression: 4 - 8 months	Baseline, post-laser iridotomy and 3-yearly or when indicated	Every 1 - 2 years	Baseline or when indicated

7. MONITORING OF PROGRESSION

Patients with glaucoma require life-long treatment and monitoring. Adjustment of treatment and target IOP depends on the evaluation of glaucoma progression (refer to **Algorithm 5 on Adjustment of Target IOP**). It is important to assess both optic nerve structure and function in detecting progression. The use of functional test may fail to detect progression in eyes with early glaucomatous damage. On the other hand, structural test may fail to detect progression in moderate to severe glaucomatous damage.^{89, level III}

7.1 Visual Field Assessment (Functional Test)

VF assessment using the white-on-white SAP is still considered to be the best method to monitor glaucomatous progression in patients with evident VF damage. It has direct relevance to patient's QoL. Although there is no standard method, there are several approaches used to analyse VF progression:^{90, level III}

- clinical judgement (based on the simple observation of a sequential series of VF tests)
- defect classification systems (usually used in research setting)
- trend analysis (regression analyses to measure rates of change)
- event analysis (comparison of a follow-up examination to the baseline VF)

Estimating the rate of progression is invaluable to guide therapeutic decisions and estimate the likelihood of visual impairment during the patient's lifetime.

- Changes in VF are determined by the:^{89, level III; 91, level III}
 - same SAP strategy and test pattern
 - threshold tests using full threshold and SITA standard testing strategies
 - sufficient number (minimum two) of reliable tests to establish a good baseline
 - sufficient number of examinations to detect change
 - Six VF examinations should be performed in the first two years to detect rapid progression (-2 dB/year or worse) and establish a good set of baseline data.¹⁵
 - VF progression should be correlated with clinical findings.

7.2 Optic Nerve and Retinal Nerve Fibre Layer Evaluation (Structural Test)

Imaging of the optic nerve and RNFL allows objective, standardised and reproducible ways to assess and continuously monitor glaucomatous damage. Three types of computer-based optic nerve imaging devices are available for glaucoma:⁹²

- CSLO
- OCT

To determine the progression of glaucoma, progression analysis programmes are being incorporated into the above devices using the patients' baseline images as references. Currently these programmes are still unable to predict future functional loss. Good quality images are important to facilitate progression analysis as poor quality image can lead to false positive or negative results.⁸⁹

Frequency of imaging should be similar to VF testing. Six imaging tests should be performed in the first two years to detect progression in high risk patients. A repeat imaging should be performed within three months after the baseline. Patients should be followed-up with the same test/method to monitor progression.¹⁵

- Imaging alone does not replace VF testing in monitoring progression.

8. REHABILITATION

Glaucoma could result in permanent visual impairment and blindness. Consultation with an optometrist trained in vision rehabilitation is advisable to enhance patient's residual vision and QoL.^{88; 93, level III} The rehabilitation significantly improves vision-related QoL in patients with glaucoma.^{94, level II-2}

Vision rehabilitation includes the use of optical and non-optical devices, and referral to other services (vocational, occupational and independent living) and psychosocial counselling.^{93, level III} Multidisciplinary approach improves scores of overall visual ability of visually-impaired patients including those with glaucoma at 30 days follow-up ($p<0.001$).^{95, level II-2}

- All patients with low vision and blindness should be registered with Social Welfare Department to be eligible for financial and social benefits.^{96, level III}

Recommendation 7

- Glaucoma patients with blindness or low vision should be referred for vision rehabilitation which includes vocational, occupational and independent living.

Refer to Appendix 10 on Categories of severity of visual impairment.

9. REFERRAL

The aim of referral is to ensure that the patients receive specialised and optimised care under glaucoma-trained healthcare providers. This chapter is written based on expert opinion of the CPG DG and evidence in other chapters in the guidelines.

- Indications for referral are as follows:
 - acute angle closure (immediate referral)
 - confirmation of diagnosis
 - progression of disease
 - issues related to medical treatment:
 - side effects
 - requirement of ≥ 2 medications
 - poor compliance or adherence
 - uncontrolled IOP despite maximum medical treatment requiring laser or surgical intervention

10. SPECIAL CONDITIONS

10.1 Ocular Hypertension

Clinical features of OHT are:

- untreated IOP >21 mmHg
- open anterior chamber angle
- normal OD and RNFL
- normal VF
- no clinical evidence of secondary causes of elevated IOP

Important diagnostic testing includes:

- measurement of IOP
- gonioscopy
- measurement of CCT
 - It is an essential part of ocular examination in the workup of OHT patients.
 - It aids in interpretation of IOP readings and stratification of patient risk for developing POAG.
 - In OHT patients, the average CCT is 570 µm. Eyes with CCT <555 µm are at greater risk of developing POAG compared to eyes with CCT of ≥588 µm.^{97, level II-2}
 - GAT may overestimate the actual IOP in eyes with thicker CCT and underestimate in eyes with thinner CCT.^{98 - 99, level III} IOP measurement should not be corrected as there is no generally accepted correction formula for it.
- OD and RNFL assessment and/or imaging
 - OD and RNFL should be assessed and secondary causes of any abnormalities should be excluded. Stereoscopic disc photographs are advocated as baseline documentation. Computerised digital imaging of the OD and RNFL can be used to provide quantitative information and to facilitate in monitoring of patients.^{41, level I}
- VF test for initial evaluation and as baseline for future monitoring of progression

Management of OHT consists of:

- monitoring for changes suggestive of glaucomatous damage in the OD, RNFL and VF
- evaluating and identifying patients at risk of POAG conversion in order to initiate treatment

The likelihood of patients with OHT developing POAG increases with the number and severity of risk factors. Some of the predictive factors for conversion to POAG in both Ocular Hypertension Treatment Study (OHTS) and European Glaucoma Prevention Study (EGPS) trials

are:¹⁹, level II-2; 97, level II-2

- older age
- higher IOP
- thinner CCT
- larger CDR
- higher Humphrey VF PSD

A risk calculator using the above predictive factors as parameters can determine the overall risk of developing POAG in five years.¹⁸, level II-2 It is available for free online from <http://ohts.wustl.edu/risk>. Based on OHTS data, low glaucoma 5-year risk was defined arbitrarily as <6%, moderate as 6 - 13% and high >13%.¹⁰⁰, level I

In OHTS, medical treatment of OHT was effective in delaying or preventing the onset of POAG. Lowering of IOP by 20% reduced risk for progression by 50% over five years duration. However, 90 - 95% of the patients did not progress to POAG and thus not all require treatment.
^{100 - 101}, level I Based from the OHTS data, treatment of patients with IOP ≥ 24 mmHg and $\geq 2\%$ annual risk of development of POAG is likely to be cost-effective.¹⁰², level I

The decision to treat OHT will need to be individualised based on:

- clinical findings
- risk assessment
- life expectancy
- patient's preferences
- treatment cost, risks and benefits

Monitoring schedule of OHT should be based on the risk of conversion to POAG.⁸⁷ In OHTS, a reduction of 20% from the mean baseline IOP was used as the target IOP.¹⁰¹, level I In EGPS, dorzolamide alone was not effective in preventing the onset of POAG at five years follow-up compared to placebo.¹⁰³, level I It has been suggested to begin by choosing a target pressure of 20% lower than the mean baseline IOP.⁹² The first choice of treatment is usually topical medication. Laser trabeculoplasty may be considered if there is drug intolerance, poor compliance or cost issues.

Refer Appendix 11 on OHT Pathway (OHT and POAG suspects with high IOP).

OHT patients need to be monitored long-term for the development of POAG regardless whether they are being treated or not. When there is evidence of changes suggestive of glaucomatous damage that is consistent with POAG (e.g. ONH appearance, RNFL or VF), then patients should be managed as POAG cases.

There is no evidence from studies on how OHT patients should be monitored and put on follow-up schedules. In OHTS, the first feature suggestive of POAG was either change in OD and/or VF.¹⁰⁴, level I Thus, both structural and functional assessments are essential when monitoring OHT patients. Computer-based imaging of the ONH and RNFL is a useful adjunct to clinical examination as it provides quantitative information and may aid in documenting progression.⁴¹, level I Therefore, clinical examination and imaging of the ONH and RNFL, with assessment of the VF is commonly needed to facilitate the evaluation of progressive glaucomatous damage.

Recommendation 8

- Ocular hypertension (OHT) patients should have comprehensive initial eye examination and assessment of risk factors for conversion to primary open angle glaucoma (POAG).
 - Central corneal thickness measurement should be performed.
- Treatment of OHT should be based on the risk of conversion to POAG*.

*Risk calculator can be used.

10.2 Primary Open Angle Glaucoma Suspect

POAG suspect is defined as an individual with clinical findings and/or risk factors that increase the likelihood of developing POAG.¹⁰⁵ The clinical findings include any of the following features:⁸⁷

- any IOP level
- suspicious glaucomatous appearance of OD (refer to **Subchapter 4.2.6**)
- normal or suspicious glaucomatous VF defects (refer to **Subchapter 4.3.1**)

Management of POAG suspect consists of:

- monitoring for changes suggestive of glaucomatous damage in the OD, RNFL and VF
- evaluating and identifying patients at risk of POAG conversion in order to initiate treatment

An individual with normal IOP, and changes in optic nerve and VF consistent with glaucomatous damage, should be diagnosed as having Normal Tension Glaucoma (NTG). It should be treated and monitored similar to POAG.⁸⁷

Conversion of glaucoma suspect to NTG is poorly understood. Non-IOP dependent factors, especially vascular dysregulation, are thought to play a relatively larger role.¹⁰⁶, level III

Risk factors of NTG are:

a. ocular

- disc haemorrhages (HR=2.72, 95% CI 1.39 to 5.32)¹⁰⁷, level II-2
- wide diurnal IOP fluctuation^{108, level III; 109, level II-2}
- presence of beta zone within peripapillary atrophy^{110, level III}

b. systemic

- OSA Syndrome^{26, level III}
- Raynaud's phenomenon⁸⁸
- migraine^{88; 107, level II-2}
- low blood pressure^{93, level III}
- nocturnal hypotension^{93, level III}
- low ocular perfusion pressure^{111, level II-2}
- low intracranial and cerebrospinal fluid (CSF) pressure^{112, level III}

Monitoring of POAG suspect may be done at intervals of:⁸⁷

- 1 to 2 years if there is low risk of conversion*
- 6 months to 1 year if there is high risk of conversion*

*Risk of conversion is clinically assessed based on age, IOP, CCT, and changes in ONH and VF.

If there is no change in the parameters after 3 - 5 years, the patient can be transferred from active glaucoma care to general ophthalmologist or community optometrist.⁸⁷

Refer to Appendix 12 on POAG Suspects with Normal IOP Pathway.

Recommendation 9

- Comprehensive eye examination and risk assessment should be performed in primary open angle glaucoma suspect. Diurnal intraocular pressure fluctuation should be considered in the assessment.

10.3 Steroid-Induced Glaucoma

Steroid use may give rise to secondary OHT which may infrequently lead to steroid-induced glaucoma. It can occur as a result of topical, systemic or intravitreal administration of steroids. However in clinical practice, most can be controlled by topical glaucoma medications but a small proportion requires surgery.^{113, level I; 114, level II-2} In a small study of 34 patients, glaucoma medications were discontinued in all patients by 18 months after cessation of steroid therapy.^{114, level II-2}

10.4 Neovascular Glaucoma

Neovascular glaucoma (NVG) is a severe form of secondary glaucoma in which the eye develops progressive neovascularisation of the iris and angle. It is induced by ocular microvascular disease with retinal

ischaemia. Initially fibrovascular membrane covers the angle, causing secondary open angle glaucoma. This can progress to angle closure glaucoma. The common conditions associated with NVG are proliferative diabetic retinopathy, central retinal vein occlusion and other conditions such as ocular ischaemic syndrome and tumours.

Management of NVG involves optimising treatment of the underlying disease and control of high IOP. The treatment for NVG includes:¹⁵

- medical treatment
 - topical and systemic IOP lowering medication (refer to **Subchapter on Medical Treatment**)
 - topical steroid
 - topical atropine
- laser/surgery
 - retinal ablation with laser or cryotherapy
 - filtering surgery with antimetabolites
 - GDD
 - cyclodestructive procedure
 - intravitreal or intracameral anti-vascular endothelial growth factor (anti-VEGF)

NVG is a difficult condition to manage and medical treatment usually fails to control the IOP. Surgical treatment provides better IOP control.

Trabeculectomy with MMC efficaciously reduces the elevated IOP in NVG. The extent of PAS and a history of vitrectomy are significant negative predictors of IOP reduction.^{115, level II-1} Both Ahmed valve implant and Molteno single plate implant significantly reduce IOP in patients with NVG up to six months.^{116, level III} There is no significant difference in the IOP lowering between TSCP and Ahmed implant in refractory NVG treatment. However, TSCP is less time consuming and easier to perform.^{117, level I} Rates of TSCP complications are higher in NVG and with treatment protocols using more than 80 Joule per session.^{64, level III}

Anti-VEGF may have a role in the treatment of NVG.

- There is no significant difference in regression of neovascularisation of iris and reduction of IOP between 1.25 mg and 2.5 mg intracameral bevacizumab followed by trabeculectomy at six months follow-up.^{118, level I}
- Intravitreal bevacizumab with PRP followed by Ahmed valve implantation is significantly efficacious in controlling IOP at 18 months^{119, level I} and causing complete regression of rubeosis iridis in NVG at 24 months.^{120, level I}
- However, intravitreal ranibizumab (0.5 mg) injection before surgery has no significant effect on IOP, BCVA, anti-glaucoma medications or post-operative complications in NVG treated with Ahmed glaucoma valve implantation up to 12 months.^{121, level II-1}

- There is insufficient evidence to recommend a preferred method of treatment for NVG. The treatment options need to be individualised.
- The key to prevent NVG is to optimise management of the underlying diseases.

10.5 Intraocular Pressure Monitoring in Post-Refractive Surgery Cases

Corneal laser refractive surgery is a popular method for correction of refractive errors. However, refractive surgery is relatively contraindicated in patients with glaucoma because of its potential problems. These include acute transient IOP elevation and damage to existing filtering blebs during corneal flap construction, steroid-induced IOP elevation and pressure-induced intra-lamellar stromal keratitis, and inaccuracy of post-operative IOP measurement which may influence the management of glaucoma.

Most IOP measuring devices are reliant on corneal biomechanics and thickness. Different corneal thickness and tissue properties have varying effects of IOP measurement by different IOP measuring devices. In post-refractive surgery, there is an underestimation of IOP by most of the devices because of thinner CCT. GAT is significantly influenced by CCT compared with Pascal dynamic contour tonometry and tonopen.^{98 - 99, level III; 122 - 123, level II-3}

Proper and accurate establishment, and documentation of baseline parameters (e.g. VF, IOP, CCT and OD image) prior to refractive surgery is essential in providing optimal monitoring for development and progression of glaucoma in future.

- In post-refractive surgery, there is an underestimation of IOP by most devices.
- Documentation of baseline parameters prior to refractive surgery is essential.

11. IMPLEMENTING THE GUIDELINES

The management of glaucoma should be guided by evidence-based approach in order to provide quality care to the patients. Several factors may affect the implementation of recommendations in the CPG.

11.1 Facilitating and Limiting Factors

Existing facilitators for application of the recommendations in the CPG include:

- wide dissemination of the CPG (soft- and hard-copies) to healthcare providers
- regular update on glaucoma management at conferences and scientific meeting locally
- public awareness during World Glaucoma Week

Existing barriers for application of the recommendations of the CPG are:

- limited knowledge and evolving understanding of glaucoma
- insufficient resources for integrated care at different level of service delivery
- variation in treatment practice and preferences
- no national glaucoma registry for further planning of services
- lack of awareness, poor access to eye care services and different cultural/religious beliefs among the patients

11.2 Potential Resource Implications

To implement the CPG, there must be strong commitment to:

- ensure widespread distribution of the CPG to healthcare providers via printed and electronic copies
- reinforce regular training with adequate funding of healthcare providers
- ensure trained multidisciplinary team is available at different levels of healthcare
- ensure widespread distribution of updated patient education materials

The following is proposed as **clinical audit indicator for quality management:**

$$\text{Percentage of newly-diagnosed glaucoma patients treated with prostaglandin analogue as first-line treatment} = \frac{\text{Number of newly-diagnosed glaucoma patients treated with prostaglandin analogue as first-line treatment in a period}}{\text{Number of newly-diagnosed glaucoma patients on medical treatment in the same period}} \times 100\%$$

Implementation strategies will be developed following the approval of the CPG by MOH which include launching of the CPG, Quick Reference and Training Module.

REFERENCES

- Prevention of Blindness and Visual Impairment (available at <http://www.who.int/blindness/causes/priority/en/index1.html>).
- Quigley HA, Broman AT. The number of people with glaucoma worldwide in 2010 and 2020. *Br J Ophthalmol.* 2006;90(3):262-267.
- Mustari Z, MA S, Hussein E, et al. The National Eye Survey 2014. Kuala Lumpur: Ophthalmology Service, Ministry of Health Malaysia;2015.
- Rein DB, Zhang P, Wirth KE, et al. The economic burden of major adult visual disorders in the United States. *Arch Ophthalmol.* 2006;124(12):1754-1760. Erratum in: *Arch Ophthalmol.* 2007 Sep;112(1759):1304.
- Taylor HR, Pezzullo ML, Keeffe JE. The economic impact and cost of visual impairment in Australia. *Br J Ophthalmol.* 2006;90(3):272-275.
- Varma R, Ying-Lai M, Francis BA, et al. Prevalence of open-angle glaucoma and ocular hypertension in Latinos: the Los Angeles Latino Eye Study. *Ophthalmology.* 2004;111(8):1439-1448.
- de Voogd S, Ikram MK, Wolfs RC, et al. Incidence of open-angle glaucoma in a general elderly population: the Rotterdam Study. *Ophthalmology.* 2005;112(9):1487-1493.
- Quigley HA, West SK, Rodriguez J, et al. The prevalence of glaucoma in a population-based study of Hispanic subjects: Proyecto VER. *Arch Ophthalmol.* 2001;119(12):1819-1826.
- Mitchell P, Smith W, Attebo K, et al. Prevalence of open-angle glaucoma in Australia. The Blue Mountains Eye Study. *Ophthalmology.* 1996;103(10):1661-1669.
- Lee PP, Walt JG, Doyle JJ, et al. A multicenter, retrospective pilot study of resource use and costs associated with severity of disease in glaucoma. *Arch Ophthalmol.* 2006;124(1):12-19.
- Traverso CE, Walt JG, Kelly SP, et al. Direct costs of glaucoma and severity of the disease: a multinational long term study of resource utilisation in Europe. *Br J Ophthalmol.* 2005;89(10):1245-1249.
- Wang JC, Chew PT. What is the direct cost of treatment of acute primary angle closure glaucoma? The Singapore model. *Clin Exp Ophthalmol.* 2004;32(6):578-583.
- Tham YC, Li X, Wong TY, et al. Global prevalence of glaucoma and projections of glaucoma burden through 2040: a systematic review and meta-analysis. *Ophthalmology.* 2014;121(11):2081-2090.
- Chan EW, Li X, Tham YC, et al. Glaucoma in Asia: regional prevalence variations and future projections. *Br J Ophthalmol.* 2016;100(1):78-85.
- European Glaucoma Society. Terminology and Guidelines for Glaucoma (4th Edition). Savona: Publiccomm; 2014.
- Liza-Sharmini AT, Sharina YN, Dolaboladi AJ, et al. Clinical presentation, severity and progression of primary angle closure in Malays. *Med J Malaysia* 2014;69(1):21-26.
- Liza-Sharmini AT, Ng GF, Nor-Sharina Y, et al. Clinical presentation, severity and progression of primary angle closure in Malay and Chinese patients. *Med J Malaysia.* 2014;69(6):245-251.
- Leske MC, Wu SY, Honkanen R, et al. Nine-year incidence of open-angle glaucoma in the Barbados Eye Studies. *Ophthalmology.* 2007;114(6):1058-1064.
- European Glaucoma Prevention Study (EGPS) Group., Miglior S, Pfeiffer N, et al. Predictive factors for open-angle glaucoma among patients with ocular hypertension in the European Glaucoma Prevention Study. *Ophthalmology.* 2007;114(1):3-9.
- Ocular Hypertension Treatment Study Group, European Glaucoma Prevention Study Group, Gordon MO, et al. Validated prediction model for the development of primary open-angle glaucoma in individuals with ocular hypertension. *Ophthalmology.* 2007;114(1):10-19.
- Burr JM, Mowatt G, Hernández R, et al. The clinical effectiveness and cost-effectiveness of screening for open angle glaucoma: a systematic review and economic evaluation. *Health Technol Assess.* 2007;11(41):iii-iv, ix-x, 1-190.
- Ministry of Health Malaysia. Management of Primary Open Angle Glaucoma. Putrajaya: MoH; 2008.
- Orzalesi N, Rossetti L, Omboni S, et al. Vascular risk factors in glaucoma: the results of a national survey. *Graefes Arch Clin Exp Ophthalmol.* 2007;245(6):795-802.

24. Sergi M, Salerno DE, Rizzi M, et al. Prevalence of normal tension glaucoma in obstructive sleep apnea syndrome patients. *J Glaucoma*. 2007;16(1):42-46.
25. Wu X, Liu H. Obstructive sleep apnea/hypopnea syndrome increases glaucoma risk: evidence from a meta-analysis. *Int J Clin Exp Med*. 2015;8(1):297-303.
26. Bilgin G. Normal-tension glaucoma and obstructive sleep apnea syndrome: a prospective study. *BMC Ophthalmol*. 2014;14:27.
27. Lin CC, Hu CC, JD H, et al. Obstructive sleep apnea and increased risk of glaucoma: a population-based matched-cohort study. *Ophthalmology*. 2013;120(8):1559-1564.
28. Zhao D, Cho J, Kim MH, et al. Diabetes, fasting glucose, and the risk of glaucoma: a meta-analysis. *Ophthalmology*. 2015;122(1):72-78.
29. Xu L, Wang Y, Wang S, et al. High myopia and glaucoma susceptibility the Beijing Eye Study. *Ophthalmology*. 2007;114(2):216-220.
30. Zhang C, Tatham AJ, Abe RY, et al. Corneal Hysteresis and Progressive Retinal Nerve Fiber Layer Loss in Glaucoma. *Am J Ophthalmol*. 2016;166:29-36.
31. Murphy ML, Pokrovskaya O, Galligan M, et al. Corneal hysteresis in patients with glaucoma-like optic discs, ocular hypertension and glaucoma. *BMC Ophthalmol*. 2017;17(1):1.
32. Senthil S, Garudadri C, Khanna RC, et al. Angle closure in the Andhra Pradesh Eye Disease Study. *Ophthalmology*. 2010;117(9):1729-1735.
33. Qu W, Li Y, Song W, et al. Prevalence and risk factors for angle-closure disease in a rural Northeast China population: a population-based survey in Bin County, Harbin. *Acta Ophthalmol*. 2011;89(6):e515-520.
34. Casson RJ, Marshall D, Newland HS, et al. Risk factors for early angle-closure disease in a Burmese population: the Meiktila Eye Study. *Eye (Lond)*. 2009;23(4):933-939.
35. Zhou M, Wang W, Huang W, et al. Is increased choroidal thickness association with primary angle closure? *Acta Ophthalmol* 2014;92(7):e514-520.
36. Hatt SR, Wormald R, Burr J. Screening for prevention of optic nerve damage due to chronic open angle glaucoma. *Cochrane Database Syst Rev*. 2006;(4):CD006129.
37. Hernández R, Rabindranath K, Fraser C, et al. Screening for open angle glaucoma: systematic review of cost-effectiveness studies. *J Glaucoma*. 2008;17(3):159-168.
38. Mowatt G, Burr JM, Cook JA, et al. Screening tests for detecting open-angle glaucoma: systematic review and meta-analysis. *Invest Ophthalmol Vis Sci*. 2008;49(12):5373-5385.
39. Liu CJ, Cheng CY, Hsu WM. Scanning laser polarimetry with variable corneal compensation in primary angle-closure glaucoma. *Ophthalmology*. 2008;115(8):1334-1339.
40. Leung CK, Medeiros FA, Zangwill LM, et al. American Chinese glaucoma imaging study: a comparison of the optic disc and retinal nerve fiber layer in detecting glaucomatous damage. *Invest Ophthalmol Vis Sci*. 2007;48(6):2644-2652.
41. Zangwill LM, Jain S, Dirkes K, et al. The rate of structural change: the confocal scanning laser ophthalmoscopy ancillary study to the ocular hypertension treatment study. *Am J Ophthalmol*. 2013;155(6):971-982.
42. Nolan WP, See JL, Chew PT, et al. Detection of primary angle closure using anterior segment optical coherence tomography in Asian eyes. *Ophthalmology*. 2007;114(1):33-39.
43. Leske MC, Heijl A, Hussein M, et al. Factors for glaucoma progression and the effect of treatment: the early manifest glaucoma trial. *Arch Ophthalmol*. 2003;121(1):48-56.
44. Kass MA, Heuer DK, Higginbotham EJ, et al. 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;120(6):701-713.
45. Bengtsson B, Leske MC, Hyman L, et al. Fluctuation of intraocular pressure and glaucoma progression in the early manifest glaucoma trial. *Ophthalmology*. 2007;114(2):205-209.
46. The Advanced Glaucoma Intervention Study (AGIS): 7. The relationship between control of intraocular pressure and visual field deterioration. The AGIS Investigators. *Am J Ophthalmol*. 2000;130(4):429-440.
47. Comparison of glaucomatous progression between untreated patients with normal-tension glaucoma and patients with therapeutically reduced intraocular pressures. Collaborative Normal-Tension Glaucoma Study Group. *Am J Ophthalmol*. 1998;126(4):487-497. Erratum in: *Am J Ophthalmol* 1999 Jan;112(1):1120.

48. Sharmini AT, Yin NY, Lee SS, et al. Mean target intraocular pressure and progression rates in chronic angle-closure glaucoma. *J Ocul Pharmacol Ther.* 2009;25(1):71-75.
49. Canadian Ophthalmological Society Glaucoma Clinical Practice Guideline Expert Committee, Canadian Ophthalmological Society. Canadian Ophthalmological Society evidence-based clinical practice guidelines for the management of glaucoma in the adult eye. *Can J Ophthalmol* 2009;44 Suppl 1:S7-93. Erratum in: *Can J Ophthalmol.* 2009 Aug;44(4):477.
50. Aptel F, Cucherat M, Denis P. Efficacy and tolerability of prostaglandin analogs: a meta-analysis of randomized controlled clinical trials. *J Glaucoma.* 2008;17(8):667-673.
51. Stewart WC, Konstas AG, Kraft B, et al. Meta-analysis of 24-h intraocular pressure fluctuation studies and the efficacy of glaucoma medicines. *J Ocul Pharmacol Ther.* 2010;26(2):175-180.
52. van der Valk R, Webers CA, Lumley T, et al. A network meta-analysis combined direct and indirect comparisons between glaucoma drugs to rank effectiveness in lowering intraocular pressure. *J Clin Epidemiol.* 2009;62(12):1279-1283.
53. Li T, Lindsley K, Rouse B, et al. Comparative Effectiveness of First-Line Medications for Primary Open-Angle Glaucoma: A Systematic Review and Network Meta-analysis. *Ophthalmology.* 2016;123(1):129-140.
54. Cheng JW, Cai JP, Wei RL. Meta-analysis of medical intervention for normal tension glaucoma. *Ophthalmology.* 2009;116(7):1243-1249.
55. Cheng JW, Cai JP, Li Y, et al. A meta-analysis of topical prostaglandin analogs in the treatment of chronic angle-closure glaucoma. *J Glaucoma.* 2009;18(9):652-657.
56. Honrubia F, García-Sánchez J, Polo V, et al. Conjunctival hyperaemia with the use of latanoprost versus other prostaglandin analogues in patients with ocular hypertension or glaucoma: a meta-analysis of randomised clinical trials. *Br J Ophthalmol.* 2009;93(3):316-321.
57. Vass C, Hirn C, Sycha T, et al. Medical interventions for primary open angle glaucoma and ocular hypertension. *Cochrane Database Syst Rev.* 2007;(4):CD003167.
58. Cheng JW, Li Y, Wei RL. Systematic review of intraocular pressure-lowering effects of adjunctive medications added to latanoprost. *Ophthalmic Res.* 2009;42(2):99-105.
59. Ho JD, Hu CC, HC L. Antiglaucoma medications during pregnancy and the risk of low birth weight: a population-based study. *Br J Ophthalmol.* 2009;93(10):1283-1286.
60. Cheng JW, Cheng SW, Gao LD, et al. Intraocular pressure-lowering effects of commonly used fixed-combination drugs with timolol: a systematic review and meta-analysis. *PLoS One.* 2012;7(9):e45079.
61. Cox JA, Mollan SP, Bankart J, et al. Efficacy of antiglaucoma fixed combination therapy versus unfixed components in reducing intraocular pressure: a systematic review. *Br J Ophthalmol.* 2008;92(6):729-734.
62. Barnebey HS, Robin AL. Adherence to Fixed-Combination Versus Unfixed Travoprost 0.004% / Timolol 0.5% for Glaucoma or Ocular Hypertension: a Randomized Trial. *Am J Ophthalmol.* 2016;pii: S0002-9394(16)30598-0.
63. Sena DF, Lindsley K. Neuroprotection for treatment of glaucoma in adults. *Cochrane Database Syst Rev.* 2013;(2):CD006539.
64. Asian Pacific Glaucoma Society. Asia Pacific Glaucoma Guidelines (Third Edition). Amsterdam: Kugler Publications; 2016.
65. Katz LJ, Steinmann WC, Kabir A, et al. Selective laser trabeculoplasty versus medical therapy as initial treatment of glaucoma: a prospective, randomized trial. *J Glaucoma.* 2012;21(7):460-468.
66. Wang W, He M, Zhou M, et al. Selective laser trabeculoplasty versus argon laser trabeculoplasty in patients with open-angle glaucoma: a systematic review and meta-analysis. *PLoS One.* 2013;8(12):e84270.
67. Wong MO, Lee JW, Choy BN, et al. Systematic review and meta-analysis on the efficacy of selective laser trabeculoplasty in open-angle glaucoma. *Surv Ophthalmol.* 2015;60(1):36-50.
68. Polat J, Grantham L, Mitchell K, et al. Repeatability of selective laser trabeculoplasty. *Br J Ophthalmol.* 2016;100(10):1437-1441.
69. Damji KF, Bovell AM, Hodge WG, et al. Selective laser trabeculoplasty versus argon laser trabeculoplasty: results from a 1-year randomised clinical trial. *Br J Ophthalmol.* 2006;90(12):1490-1494.

70. Latina MA, Sibayan SA, Shin DH, et al. Q-switched 532-nm Nd:YAG laser trabeculoplasty (selective laser trabeculoplasty): a multicenter, pilot, clinical study. *Ophthalmology*. 1998;105(11):2082-2088.
71. Nagar M, Ogunyomade A, O'Brart DP, et al. A randomised, prospective study comparing selective laser trabeculoplasty with latanoprost for the control of intraocular pressure in ocular hypertension and open angle glaucoma. *Br J Ophthalmol*. 2005;89(11):1413-1417.
72. Hodge WG, Damji KF, Rock W, et al. Baseline IOP predicts selective laser trabeculoplasty success at 1 year post-treatment: results from a randomised clinical trial. *Br J Ophthalmol*. 2005;89(9):1157-1160.
73. Heijl A, Peters D, Leske MC, et al. Effects of argon laser trabeculoplasty in the Early Manifest Glaucoma Trial. *Am J Ophthalmol*. 2011;152(5):842-848.
74. Tzimis V, Tze L, Ganesh J, et al. Laser trabeculoplasty: an investigation into factors that might influence outcomes. *Can J Ophthalmol*. 2011;46(4):305-309.
75. Narayanaswamy A, Baskaran M, Perera SA, et al. Argon Laser Peripheral Iridoplasty for Primary Angle-Closure Glaucoma: A Randomized Controlled Trial. *Ophthalmology*. 2016;123(3):514-521.
76. Zhekov I, Janjua R, Shahid H, et al. A retrospective analysis of long-term outcomes following a single episode of transscleral cyclodiode laser treatment in patients with glaucoma. *BMJ Open*. 2013;3(7 pii):e002793.
77. Wilkins M, Indar A, Wormald R. Intra-operative mitomycin C for glaucoma surgery. *Cochrane Database Syst Rev*. 2005;(4):CD002897.
78. Azuara-Blanco A, Burr J, Ramsay C, et al. Effectiveness of early lens extraction for the treatment of primary angle-closure glaucoma (EAGLE): a randomised controlled trial. *Lancet*. 2016;388(10052):1389-1397.
79. Tham CC, Kwong YY, Leung DY, et al. Phacoemulsification versus combined phacotrabeculectomy in medically controlled chronic angle closure glaucoma with cataract. *Ophthalmology*. 2008;115(12):2167-2173.e2162.
80. Tham CC, Kwong YY, Leung DY, et al. Phacoemulsification versus combined phacotrabeculectomy in medically uncontrolled chronic angle closure glaucoma with cataracts. *Ophthalmology*. 2009;116(4):725-731, 731.e721-723.
81. HaiBo T, Xin K, ShiHeng L, et al. Comparison of Ahmed glaucoma valve implantation and trabeculectomy for glaucoma: a systematic review and meta-analysis;. *PLoS One*. 2015;10(2):e0118142.
82. Gedde SJ, Schiffman JC, Feuer WJ, et al. Treatment outcomes in the Tube Versus Trabeculectomy (TVT) study after five years of follow-up. *Am J Ophthalmol*. 2012;153(5):789-803.e782.
83. Molteno AC, Bevin TH, Herbison P, et al. Long-term results of primary trabeculectomies and Molteno implants for primary open-angle glaucoma. *Arch Ophthalmol*. 2011;129(11):1444-1450.
84. Budenz DL, Barton K, Gedde SJ, et al. Five-year treatment outcomes in the Ahmed Baerveldt comparison study. *Ophthalmology* 2015;122(2):308-316.
85. Christakis PG, Tsai JC, Kalenak JW, et al. The Ahmed versus Baerveldt study: three-year treatment outcomes. *Ophthalmology*. 2013;120(11):2232-2240.
86. Eldaly MA, Bunce C, Elsheikha OZ, et al. Non-penetrating filtration surgery versus trabeculectomy for open-angle glaucoma. *Cochrane Database Syst Rev*. 2014;(2):CD007059.
87. National Collaborating Centre for Acute Care. *Glaucoma: Diagnosis and management of chronic open angle glaucoma and ocular hypertension*. London: RCS; 2009.
88. National Health and Medical Research Council. *NHMRC Guidelines for the screening, prognosis, diagnosis, management and prevention of glaucoma 2010*. Canberra: NHMRC; 2010.
89. Weinreb RN, Garway-Heath DF, Leung C, et al. Progression of glaucoma: the 8th consensus report of the World Glaucoma Association. Amsterdam: Kugler Publications; 2011.
90. Brusini P. Monitoring glaucoma progression. *Prog Brain Res*. 2008;173:59-73.
91. Chauhan BC, Garway-Heath DF, Goñi FJ, et al. Practical recommendations for measuring rates of visual field change in glaucoma. *Br J Ophthalmol*. 2008;92(4):569-573.
92. American Academy of Ophthalmology. *Primary Open-Angle Glaucoma PPP - 2015*. San Francisco: Elsevier Inc; 2015.

93. American Optometric Association. Care of the Patient with Open Angle Glaucoma. St. Louis: AOA; 2010.
94. Luo RJ, Liu SR, Tian Z, et al. Rehabilitation of vision disorder and improved quality of life in patients with primary open angle glaucoma. *Chin Med J (Engl)*. 2011;124(17):2687-2691.
95. Wang BZ, Pesudovs K, Keane MC, et al. Evaluating the effectiveness of multidisciplinary low-vision rehabilitation. *Optom Vis Sci*. 2012;89(9):1399-1408.
96. Perkhidmatan Optometri, Kementerian Kesihatan Malaysia. Prosedur Operasi Standard Penjagaan dan Rehabilitasi Visual Pesakit Penglihatan Terhad. Putrajaya: Majlis Optik Malaysia, KKM; 2009.
97. Gordon MO, Beiser JA, Brandt JD, et al. The Ocular Hypertension Treatment Study: baseline factors that predict the onset of primary open-angle glaucoma. *Arch Ophthalmol*. 2002;120(6):714-720.
98. Ku JY, Danesh-Meyer HV, Craig JP, et al. Comparison of intraocular pressure measured by Pascal dynamic contour tonometry and Goldmann applanation tonometry. *Eye (Lond)*. 2006;20(2):191-198.
99. Martinez-de-la-Casa JM, Garcia-Feijoo J, Vico E, et al. Effect of corneal thickness on dynamic contour, rebound, and goldmann tonometry. *Ophthalmology*. 2006;113(12):2156-2162.
100. Kass MA, Gordon MO, Gao F, et al. Delaying treatment of ocular hypertension: the ocular hypertension treatment study. *Arch Ophthalmol*. 2010;128(3):276-287.
101. Kass MA, Heuer DK, Higginbotham EJ, et al. 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;120(6):701-713.
102. Kymes SM, Kass MA, Anderson DR, et al. Management of ocular hypertension: a cost-effectiveness approach from the Ocular Hypertension Treatment Study. *Am J Ophthalmol*. 2006;141(6):997-1008.
103. Miglior S, Zeyen T, Pfeiffer N, et al. Results of the European Glaucoma Prevention Study. *Ophthalmology*. 2005;112(3):366-375.
104. Keltner JL, Johnson CA, Anderson DR, et al. The association between glaucomatous visual fields and optic nerve head features in the Ocular Hypertension Treatment Study. *Ophthalmology*. 2006;113(9):1603-1612.
105. American Academy of Ophthalmology. Primary Open-Angle Glaucoma Suspect PPP - 2015. San Francisco: Elsevier Inc.; 2016.
106. Sommer A, Tielsch JM. Primary open-angle glaucoma: a clinical-epidemiologic perspective. In: Shields MB, Van Buskirk M, editors. 100 years of progress in glaucoma. Philadelphia: Lippincott Raven; 1997.
107. Drance S, Anderson DR, Schulzer M, et al. Risk factors for progression of visual field abnormalities in normal-tension glaucoma. *Am J Ophthalmol*. 2001;131(6):699-708.
108. Choi J, Kook MS. Systemic and Ocular Hemodynamic Risk Factors in Glaucoma. *Biomed Res Int*. 2015;2015:141905.
109. Asrani S, Zeimer R, Wilensky J, et al. Large diurnal fluctuations in intraocular pressure are an independent risk factor in patients with glaucoma. *J Glaucoma*. 2000;9(2):134-142.
110. Park SC, Lee DH, Lee HJ, et al. Risk factors for normal-tension glaucoma among subgroups of patients. *Arch Ophthalmol*. 2009;127(10):1275-1283.
111. Leske MC, Heijl A, Hyman L, et al. Predictors of long-term progression in the early manifest glaucoma trial. *Ophthalmology*. 2007;114(11):1965-1972.
112. Ren R, Jonas JB, Tian G, et al. Cerebrospinal fluid pressure in glaucoma: a prospective study. *Ophthalmology*. 2010;117(2):259-266.
113. Kiddee W, Trope GE, Sheng L, et al. Intraocular pressure monitoring post intravitreal steroids: a systematic review. *Surv Ophthalmol*. 2013;58(4):291-310.
114. Sihota R, Konkal VL, Dada T, et al. Prospective, long-term evaluation of steroid-induced glaucoma. *Eye (Lond)*. 2008;22(1):26-30.
115. Kiuchi Y, Sugimoto R, Nakae K, et al. Trabeculectomy with mitomycin C for treatment of neovascular glaucoma in diabetic patients. *Ophthalmologica*. 2006;220(6):383-388.
116. Yalvac IS, Eksioglu U, Satana B, et al. Long-term results of Ahmed glaucoma valve and Molteno implant in neovascular glaucoma. *Eye (Lond)*. 2007;21(1):65-70.

117. Yildirim N, Yalvac IS, Sahin A, et al. A comparative study between diode laser cyclophotocoagulation and the Ahmed glaucoma valve implant in neovascular glaucoma: a long-term follow-up. *J Glaucoma*. 2009;18(3):192-196.
118. Gupta V, Jha R, Rao A, et al. The effect of different doses of intracameral bevacizumab on surgical outcomes of trabeculectomy for neovascular glaucoma. *Eur J Ophthalmol*. 2009;19(3):435-441.
119. Mahdy RA, Nada WM, Fawzy KM, et al. Efficacy of intravitreal bevacizumab with panretinal photocoagulation followed by Ahmed valve implantation in neovascular glaucoma. *J Glaucoma*. 2013;22(9):768-772.
120. Arcieri ES, Paula JS, Jorge R, et al. Efficacy and safety of intravitreal bevacizumab in eyes with neovascular glaucoma undergoing Ahmed glaucoma valve implantation: 2-year follow-up. *Acta Ophthalmol*. 2015;93(1):e1-6.
121. Tang M, Fu Y, Wang Y, et al. Efficacy of intravitreal ranibizumab combined with Ahmed glaucoma valve implantation for the treatment of neovascular glaucoma. *BMC Ophthalmol*. 2016;16:7.
122. Hamed-Azzam S, Briscoe D, Tomkins O, et al. Evaluation of intraocular pressure according to corneal thickness before and after excimer laser corneal ablation for myopia. *Int Ophthalmol*. 2013;33(4):349-354.
123. Aristeidou AP, Labiris G, Katsanos A, et al. Comparison between Pascal dynamic contour tonometer and Goldmann applanation tonometer after different types of refractive surgery. *Graefes Arch Clin Exp Ophthalmol*. 2011;249(5):767-773.

APPENDIX 1**EXAMPLE OF SEARCH STRATEGY**

The following Medical Subject Heading terms or free text terms were used either singly or in combination, search was limit to English, human and last 10 years:-

Clinical Question: What are the risk factors for primary glaucoma (open angle and angle closure glaucoma)?

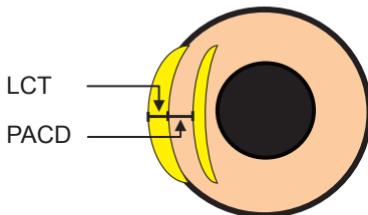
1. glaucoma/
2. glaucoma*.tw.
3. glaucoma, angle-closure/
4. glaucoma* adj1 (narrow angle or narrow-angle or closed angle or closed-angle or angle closure or angle-closure).tw.
5. glaucoma, open-angle/
6. glaucoma* adj1 (simpl* or open angle or open-angle).tw.
7. low tension glaucoma/
8. glaucoma* adj1 (low tension or normal tension).tw.
9. tension adj1 (glaucoma* low or glaucoma* normal).tw.
10. 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9
11. risk factors/
12. Risk adj1 factor*.tw.
13. 11 or 12
14. 10 and 13
15. limit 14 to ("all adult (19 plus years)" and english and humans and last 10 years)

APPENDIX 2**CLINICAL QUESTIONS**

1. What are the risk factors for primary glaucoma (open angle and angle closure glaucoma)?
2. Who to be screened for glaucoma?
What are the effective and safe screening tools/methods for glaucoma?
3. What are the criteria for glaucoma diagnosis?
4. What are the principles of treatment for glaucoma?
5. What are the effective and safe pharmacological treatments in glaucoma?
6. Is laser treatment effective and safe in primary open angle glaucoma?
7. What are the effective and safe surgical treatments in glaucoma?
8. What is the effective and safe follow-up schedule in glaucoma?
9. What is the effective method of monitoring patient's compliance and adherence in glaucoma?
10. What is the effective method of monitoring progression in glaucoma?
11. What are the effective and safe rehabilitation measures in glaucoma?
12. What are the safe and effective treatments in ocular hypertension?
13. How to diagnose and follow-up glaucoma suspect (optic disc abnormalities)?
14. What are the effective and safe treatments in patients with steroid-induced glaucoma?
15. What are the effective and safe treatments in neovascular glaucoma?
16. What are the effective and safe methods of intra-ocular pressure measuring in post-refractive surgery in glaucoma?

APPENDIX 3**VAN HERICK TEST**

- A narrow slit of light is projected onto the peripheral cornea at an angle of 60° as near as possible to the limbus.
- This results in a slit image on the surface of the cornea, the width of which is used as reference for the assessment of the conditions in the chamber angle [limbal corneal thickness (LCT)].
- The peripheral anterior chamber depth (PACD) can be described by the distance between the corneal slit image and the slit image on the iris



Van Herick Grade	Ratio of PACD to LCT	Schematic diagram
Grade 0	Closed angle closure	
Grade 1	<1:4 Angle closure likely Angle approx. 10°	
Grade 2	1:4 Angle closure possible Angle approx. 20°	
Grade 3	1:2 Angle closure unlikely	
Grade 4	1:1 (or > 1.1) Angle closure very unlikely	

Modified: Savage JA. Gonioscopy in the Management of Glaucoma. San Francisco: AAO; 2006

APPENDIX 4**INDENTATION/DYNAMIC GONIOSCOPY****Technique**

1. Explain the procedure to patient.
2. Instill topical anaesthesia.
3. Darken the room.
4. Set the slit lamp magnification to 10 - 25x, a fairly short (2 - 3 mm) slit and narrow light beam. The light beam should not pass through the pupil as this may cause pupil constriction.
5. Use a goniolens with a smaller posterior diameter than the corneal diameter (such as Sussman, or Zeiss 4-mirror goniolens). A coupling agent is not required.
6. Start with non-indentation gonioscopy.
7. In indentation gonioscopy, gentle pressure is placed on the cornea to force the aqueous humour into the anterior chamber angle. This causes posterior bowing of the iris which enables further assessment of the angle.
8. Too much pressure on the cornea may distort the anterior chamber angle and may give the observer the false impression of an open angle. This happens when the examiner notices the presence of Descemet membrane folds.
9. Examine each quadrant of the angle and record accordingly.

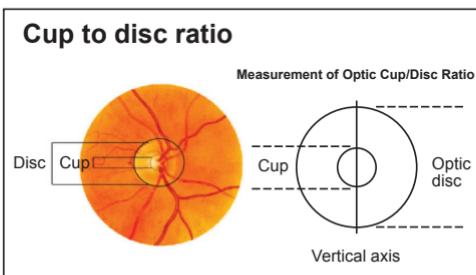
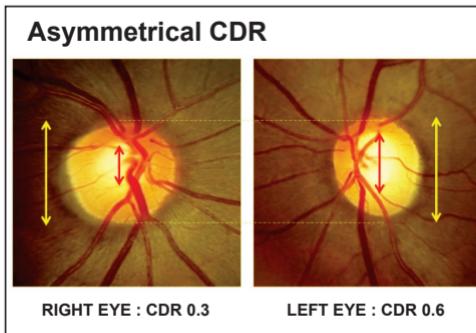
Refer to www.gonioscopy.org, for further details.

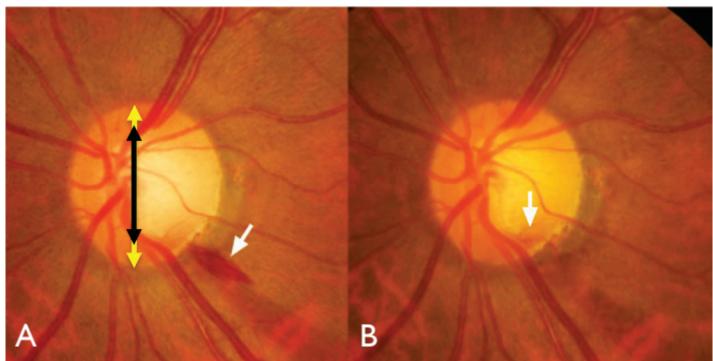
APPENDIX 5**ESTIMATION OF OPTIC DISC SIZE**

- OD size influences the estimation of CDR.
- A normal patient with a small disc will have a small cup, thus a smaller CDR. A normal patient with a large disc tend to have a large cup, thus a larger CDR.
- OD size can be estimated using a handheld convex lens and a slit lamp (using the adjustable beam height).
- A small beam is adjusted to the vertical diameter of the OD and its length is read on the scale of the slit lamp. It is most accurate in a dilated pupil.
- Based on the lens used, this value needs to be corrected using the magnification factor depending on lens power.

Lens used	Correction factor
+60D Volk	1.0
+78D Volk	1.13
Superfield Volk	1.50

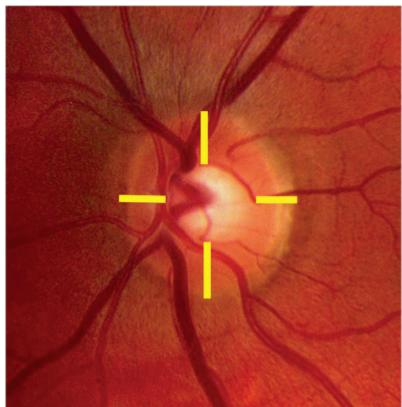
- A normal disc size is approximately 1.5 mm. A disc is considered small if it is ≤ 1.2 mm and large if it is ≥ 1.8





Right eye OD showing typical glaucomatous optic disc changes:

- ISNT rule not obeyed (inferior NRR thickness is the same as superior NRR)
- large vertical CDR 0.8 (ratio of black to yellow arrow)
- peripapillary OD haemorrhage and loss of RNFL (white arrow in picture A)
- focal neuro-retinal rim thinning/notching (white arrow picture B)

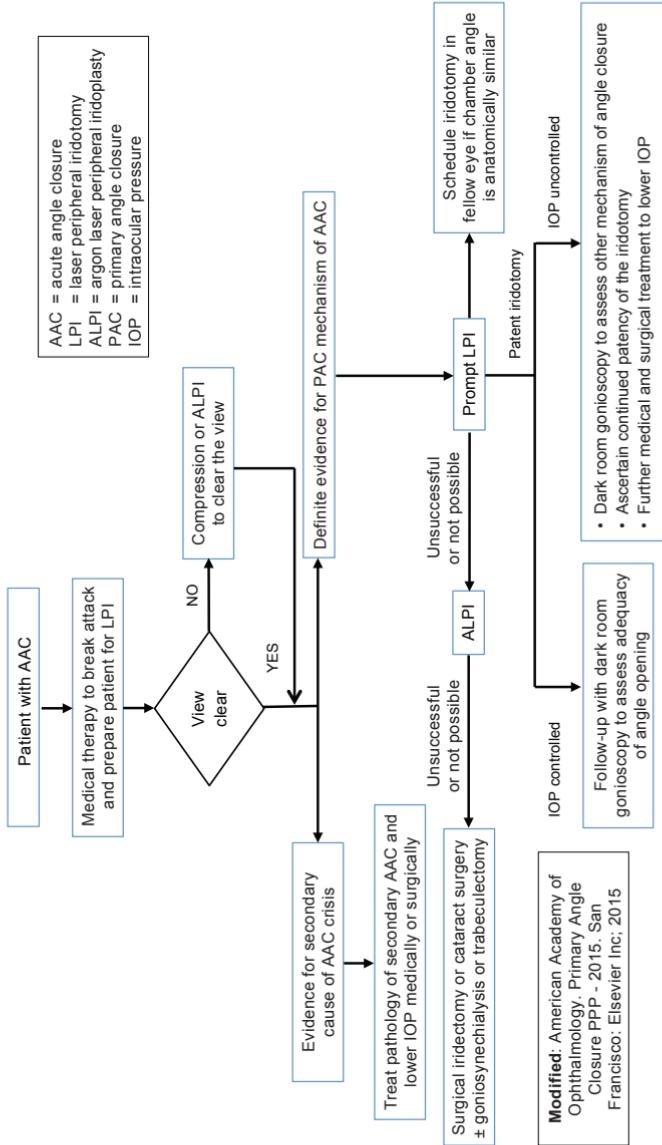


A normal OD photograph showing the thickness of neuro-retinal rim following the ISNT rule.

• The ISNT rule is useful in evaluating whether thinning is physiological or pathological.

• A healthy disc tends to have its thickest portion of NRR inferiorly, followed by superiorly and nasally, with the thinnest portion temporally. NRR thickness as shown in yellow bar follows ISNT rule.

Modified: American Academy of Ophthalmology. Gonioscopy in the Management of Glaucoma. San Francisco: AAO; 2006

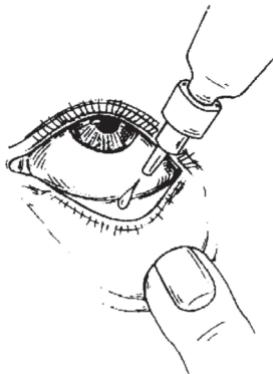
APPENDIX 6**MANAGEMENT ACUTE ANGLE CLOSURE**

APPENDIX 7**PROPER INSTILLATION TECHNIQUE OF EYE DROP****It is important to:**

- Wash hands before and after putting the eye drop.
- Apply the eye drop as directed by the doctor.

Instillation technique

1. Tilt the head back and look up.
2. Pull down the lower eyelid to form a pocket.
3. Hold the bottle vertically and bring it above the eye. Do not let the bottle tip touch the eye.
4. Squeeze one drop into the pocket of the lower eyelid.
5. If you're not sure a drop has entered the eye, instil a second drop.



6. Close the eye gently. Do not blink or squeeze the eyelid. Apply a gentle pressure on the lacrimal duct at a nasal corner of the eye with the index finger for two to three minutes.



7. If using more than one eye drops, wait at least five minutes before instilling the next eye drop.

APPENDIX 8

MAIN FEATURES OF TOPICAL ANTI-GLAUCOMA MEDICATIONS

Features	Prostaglandin analogues/prostamides	β -blockers	α_2 Adrenergic agonists	Topical CAIs	Cholinergic agents (direct-acting)
Mechanism of action	Increase in uveoscleral outflow	Reduction of aqueous production	<ul style="list-style-type: none"> Reduction of aqueous production Increase in uveoscleral outflow 	Reduction of aqueous production	Increase in trabecular outflow
IOP reduction efficacy	++++ 25 - 30% (better control of circadian IOP)	<ul style="list-style-type: none"> +++ 20 - 25% Selective beta-blockers has lower efficacy Efficacy maybe reduced by simultaneous treatment with systemic beta-blockers Tachyphylaxis may occur 	<ul style="list-style-type: none"> ++ to +++ 20 - 25% Long term use of apraclonidine may be limited due to allergic reactions and tachyphylaxis (lower incidence with brimonidine) 	+ to ++ 15 - 20%	+ to ++ 20 - 25%
Duration of effect	24 hours	12 hours	12 hours	8 - 12 hours	6 - 12 hours
Treatment option	First or second choice (switching within this class maybe of benefit as patient may respond differently)	First or second choice	Second choice (α_2 -selective agents maybe used as short term primary therapy following anterior segment procedures for preventing acute spike in IOP)	Second choice Third choice	Third choice

Features	Prostaglandin analogues/prostamides	β -blockers	α_2 Adrenergic agonists	Topical CAIs	Cholinergic agents (direct-acting)
Instillation frequency	<ul style="list-style-type: none"> Once daily (except unoprostone twice daily) Paradoxical effect (\uparrow IOP) may occur if more than once daily dosing More effective if administered in the evening 	1 - 2 times daily	2 - 3 times daily	2 - 3 times daily (Monotherapy - thrice daily dosing, adjunctive therapy - twice daily dosing)	3 - 4 times daily
Commercially available preparation	<ul style="list-style-type: none"> Latanoprost 0.005%* Travoprost 0.004%* Bimatoprost 0.03%, 0.01%* Tafluprost 0.0015%* Unoprostone 0.12%, 0.15% 	<p>Non-selective</p> <ul style="list-style-type: none"> Timolol 0.25%, 0.5%* Levocabenol 0.25%, 0.5%* Befunolol 0.5% Metipranolol 0.1%, 0.3% Carteolol 0.5%, 2% Pindolol 0.5% <p>β_1 selective</p> <ul style="list-style-type: none"> Betaxolol 0.25%, 0.5%* 	<p>α_2-selective</p> <ul style="list-style-type: none"> Brimonidine 0.15%* Apraclonidine 0.5%, 1% Clonidine 0.15%, 0.5% <p>Non-selective</p> <ul style="list-style-type: none"> Dipivefrin 0.1%, 0.5% Epinephrine 0.25%, 0.5% 	<p>Topical CAIs</p> <ul style="list-style-type: none"> Brinzolamide 1%* Dorzolamide 2%* 	<p>Direct-acting cholinergic agents</p> <ul style="list-style-type: none"> Pilocarpine 1%, 2%, 4%* Acetylcholine 2% Carbachol 0.75% - 3% Acethylcholine 1%
Drug combinations - additive effect					<p>Indirect-acting</p> <ul style="list-style-type: none"> Demecarium bromide 0.125% - 0.25% Ecothiopate iodide 0.03% - 0.25% Physostigmine
Prostaglandin analogues/prostamides			+	+	+
Beta-blockers	+		+		+
Adrenergic agonists	+	+	+	+	+
Topical CAIs	+	+	+	+	+
Cholinergic agents (direct-acting)	‡	+	+	+	

Features	Prostaglandin analogues/prostamides	β -blockers	α_2 Adrenergic agonists	Topical CAIs	Cholinergic agents (direct-acting)
Non-preservatives or with different preservative preparations	No	Yes* (both preparations available)	Yes* (preparation with different preservative available)	No	Yes* (non-preserved preparation available)
Fixed combination preparations		<ul style="list-style-type: none"> • Travoprost 0.004% & timolol 0.5% eye drops* • Bimatoprost 0.03% & timolol 0.5% eye drops* • Latanoprost 0.005% & timolol maleate 0.5% eye drops* • Tafluprost 0.0015% & timolol 0.5% eye drops* • Dorzolamide 2% & timolol 0.5% eye drops* • Brimonidine 0.2% & timolol 0.5% eye drops* • Brinzolamide 1% & timolol 0.5% eye drops* 	<ul style="list-style-type: none"> Brimonidine 0.2% & brinzolamide 1% eye drops 	Yes* Brimonidine 0.2% & brinzolamide 1% eye drops	
Beta-blockers	Yes*			No	Yes*
Adrenergic agonists	No	No*	No	Yes	Yes*
Topical CAIs	No	Yes*	Yes	No	No

*Drugs available in Malaysia

The information on various anti-glaucoma medications on this section only serves as a general guide and is not all inclusive.

Source:

- Ministry of Health Malaysia. Management of Primary Open Angle Glaucoma. Putrajaya: MoH; 2008
- European Glaucoma Society. Terminology and Guidelines for Glaucoma (4th Edition). Savona: Publicomm; 2014.
- Individual product information sheet

Safety Profiles of Topical Anti-glaucoma Medications

Safety Profiles	Prostaglandin analogues/prostanoides	β -blockers	α_2 Adrenergic agonists	Topical CAIs	Cholinergic agents (direct-acting)
Contraindications	<p>Relative contraindications:</p> <ul style="list-style-type: none"> • uveitis • Herpes Simplex Viral keratitis • cystoid macular oedema • Caution in: <ul style="list-style-type: none"> • complicated intraocular surgery (e.g. posterior capsule rupture) 	<ul style="list-style-type: none"> • Bronchial asthma, chronic obstructive pulmonary disease • Bradyarrhythmia, heart block, cardiac failure • Relative contraindication for $\beta 1$ selective 	<ul style="list-style-type: none"> • On monoamine oxidase inhibitor therapy • Children < 2 years old due to possibility of central nervous system suppression 	<ul style="list-style-type: none"> • Compromised corneal endothelium • Sulphonamide allergy • Severe renal impairment • Caution Hepatic impairment 	<ul style="list-style-type: none"> • Uveitic, neovascular and lens induced glaucoma • Aqueous misdirection syndrome
Pregnancy and nursing mothers	Human studies are lacking. Use only if potential benefit outweighs the potential risk to foetus/infant.	Human studies are lacking. Use only if potential benefit outweighs the potential risk to foetus/infant.	Human studies are lacking. Use only if potential benefit outweighs the potential risk to foetus/infant.	Human studies are lacking. Use only if potential benefit outweighs the potential risk to foetus/infant.	Human studies are lacking. Use only if potential benefit outweighs the potential risk to foetus/infant.
Common drug interactions	Chronic pilocarpine use may reduce the efficacy of these agents	Systemic beta-blockers Calcium antagonists Digitalis Catecholamine-depleting drugs	Central nervous system depressants (alcohol, barbiturates, opiates, sedatives, anaesthetics), tricyclic antidepressants	Caution in patients on steroid (potential for hypokalaemia)	Competitive interaction on outflow with prostaglandin
Topical allergies	±	±	++	±	±

Safety Profiles	Prostaglandin analogues/prostamides	β -blockers	α_2 Adrenergic agonists	Topical CAIs	Cholinergic agents (direct-acting)
Ocular adverse effects	+ to ++ Ocular discomfort (stinging, burning, foreign body sensation) Pruritis Photophobia Tearing Dry eye Blurred vision Asthenopia Allergy (conjunctivitis, eyelid erythema) Conjunctival hyperaemia (usually transient and non-infectious) Subconjunctival haemorrhage Hypertrichosis Blepharitis Eyelid skin darkening Corneal oedema Punctate epithelial keratopathy Reactivation of Herpes Simplex Virus keratitis Iris darkening Cataract Anterior uveitis Cystoid macula oedema	++ Ocular discomfort (stinging, burning, foreign body sensation) Pruritis Photophobia Tearing Conjunctival hyperaemia Decreased corneal sensitivity Punctate epithelial keratopathy Allergy (conjunctivitis, eyelid erythema)	++ Ocular discomfort (stinging, burning, foreign body sensation) Pruritis Allergy (conjunctivitis, eyelid erythema) Conjunctival hyperaemia Subconjunctival haemorrhage Lid retraction Pupil dilation (apraclonidine)	++ (Brinzolamide 1% causes less ocular discomfort) Ocular discomfort (stinging, burning, foreign body sensation) Pruritis Tearing Allergy (conjunctivitis, eyelid erythema) Blurred vision Transient myopia Punctate epithelial keratopathy	+++ Brow ache Lacration Miosis Dimness of vision Blurring of vision, myopic shift Ciliary spasm Aggravate pupillary block Retinal detachment

Safety Profiles	Prostaglandin analogues/prostamides	β -blockers	α_2 Adrenergic agonists	Topical CAIs	Cholinergic agents (direct-acting)
Systemic adverse effects	0	+ to +++ (Selective β -blockers has a wider safety margin - less systemic side effects especially cardiopulmonary side-effects)	+ to ++	0 to ++	0 to ++
Cardiovascular adverse effects	-	Bradycardia Hypotension Cardiac failure Nocturnal hypotension	-	-	Arrhythmia Flushing
Respiratory adverse effects	-	Bronchospasm	-	-	Bronchoconstriction
Neurology adverse effects	-	Syncope Drowsiness Anergy Fatigue Depression Aggravation of myasthenia gravis Memory impairment	-	-	Headache
Gastrointestinal tract (GIT) adverse effects	-	GIT discomfort Oral dryness	GIT discomfort Oral dryness	Throat irritation Altered taste	Salivation Abdominal cramps Diarrhoea Vomiting

Safety Profiles	Prostaglandin analogues/prostamides	β -blockers	α_2 Adrenergic agonists	Topical CAIs	Cholinergic agents (direct-acting)
Other adverse effects	-	Masked hypoglycaemia Hypercholesterolaemia Sexual dysfunction (loss of libido, impotence) Reduced effort tolerance Increased falls in the elderly	-	Urolithiasis	Urinary frequency

The information on various anti-glaucoma medications on this section only serves as a general guide and is not all inclusive.

Source:

- Ministry of Health Malaysia. Management of Primary Open Angle Glaucoma. Putrajaya: MoH; 2008
- European Glaucoma Society. Terminology and Guidelines for Glaucoma (4th Edition). Savona: Publicomm; 2014.
- South East Asia Glaucoma Interest Group. Asia Pacific Glaucoma Guidelines Second Edition. Hong Kong: Scientific Communication International; 2008
- Individual product leaflet

Application of Mitomycin C (MMC) or 5-Fluorouracil (5-FU) in Trabeculectomy

Antimetabolite	Timing of application	Dose and duration	Mode of application
MMC**	During surgery/intra-operatively	0.2 - 0.4 mg/ml applied for 1 - 5 minutes	Subconjunctival local application
5-FU**	<ul style="list-style-type: none"> comes in as a vial and in purple colour powder of 10 mg injection potency freshly reconstituted with distilled water or normal saline in concentration of 0.2 - 0.4 mg/ml 	<p>For primary surgery, a concentration of 0.4 mg/ml is applied for 1 minute, whereas for poor prognosis filtercases, the same concentration can be applied for 3 minutes.</p> <p>Post-operatively/prior to needling</p> <p>During surgery/ intra-operatively</p> <p>Post-operatively/prior to needling</p>	<p>Post-operative injection</p> <p>0.01 ml of MMC (0.4 mg/ml) and 0.02 ml of bupivacaine with epinephrine</p> <p>50 mg/ml for 1 - 3 minutes</p> <p>5 mg/0.1ml for up to 4 post-operative weeks</p>

** Antimetabolites usage should be individualised to each patient depending on the complexity of cases.

Care in the preparation and disposal of metabolites:

- It is recommended to prepare metabolite in a separate trolley using aseptic technique.
- The instruments used in MMC or 5-FU application should not be re-used during the surgery to avoid contamination of the surgical field.
 - The soaked sponges must be disposed in an incinerator or safely in concordance with bio-waste rules.
 - Disposal of leftover/unused antimetabolites should be taken with the same care as any other chemotherapies. It should be sent back to local pharmacy department for proper disposal.

Modified: South East Asia Glaucoma Interest Group. Asia Pacific Glaucoma Guidelines Second Edition. Hong Kong: Scientific Communication International; 2008

APPENDIX 9**LASER IRIDOTOMY****Pre-Laser Management**

- i. Get informed consent.
- ii. Instil pilocarpine 2%.
- iii. Consider brimonidine 0.15 - 0.2%, and/or β -blocker, and/or oral CAI, and/or steroid eye drops before the procedure to reduce post-treatment IOP spike/inflammation.
- iv. Instil topical anaesthesia.
- v. Use iridotomy lenses e.g. Abraham (+66 diopters) or equivalent.
- vi. Choose superior quadrants of the iris which is well covered by the upper eyelid, in a thin looking area or an iris crypt and placed as peripheral as possible.

Laser Setting for 'Sequential' Laser - Argon followed by Nd: Yag Laser

- Preparatory burns - Argon laser
 - Spot size: 200 - 500 μm
 - Exposure time: 0.2 - 0.5 sec
 - Power: 200 - 600 mW (depends on iris pigmentation; lower power for darker irides to avoid charring)
- Penetrating burns - Argon laser (chipping technique)
 - Spot size: 50 μm
 - Exposure time: 0.05 - 0.1 sec
 - Power: 600 - 1,000 mW (modify parameters depending on individual's response)
 - End point: presence of gush of aqueous and pigments
- Penetration laser burns - Nd:YAG laser
 - Power: 3 - 8 mJ
 - Number of shots: Usually 2 - 5 shots
 - Adequate iridotomy size: 200 - 500 μm

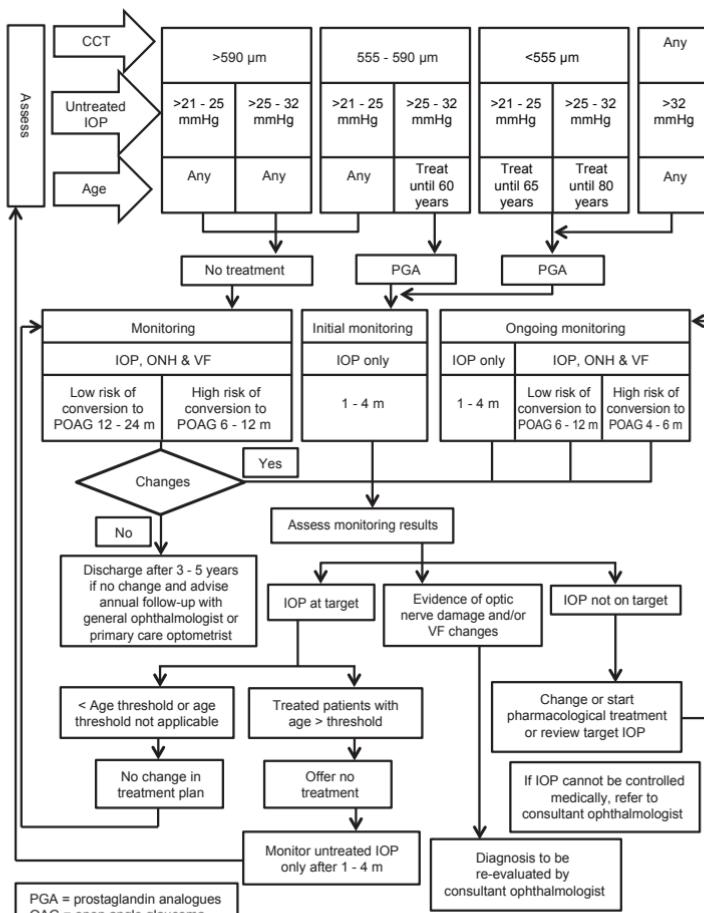
Modified: Asia-Pacific Glaucoma Society. Asia Pacific Glaucoma Guidelines (Third Edition). Amsterdam: Kugler Publication; 2016

APPENDIX 10**CATEGORIES OF SEVERITY OF VISUAL IMPAIRMENT**

Category of visual impairment	Snellen VA with best possible correction		Or central VF*
	Maximum less than	Minimum equal to or better than	
1 Moderate visual impairment	6/18	6/60	
2 Severe visual impairment	6/60	3/60	20° or less but more than 10°
3 Blindness	3/60	1/60 (counting finger at 1 meter)	10° or less but more than 5°
4 Blindness	1/60 (counting finger at 1 meter)	Light Perception	5° or less
5 Blindness	No Light Perception		

*VF restriction criteria is applicable even if VA is better than for that category of visual impairment.

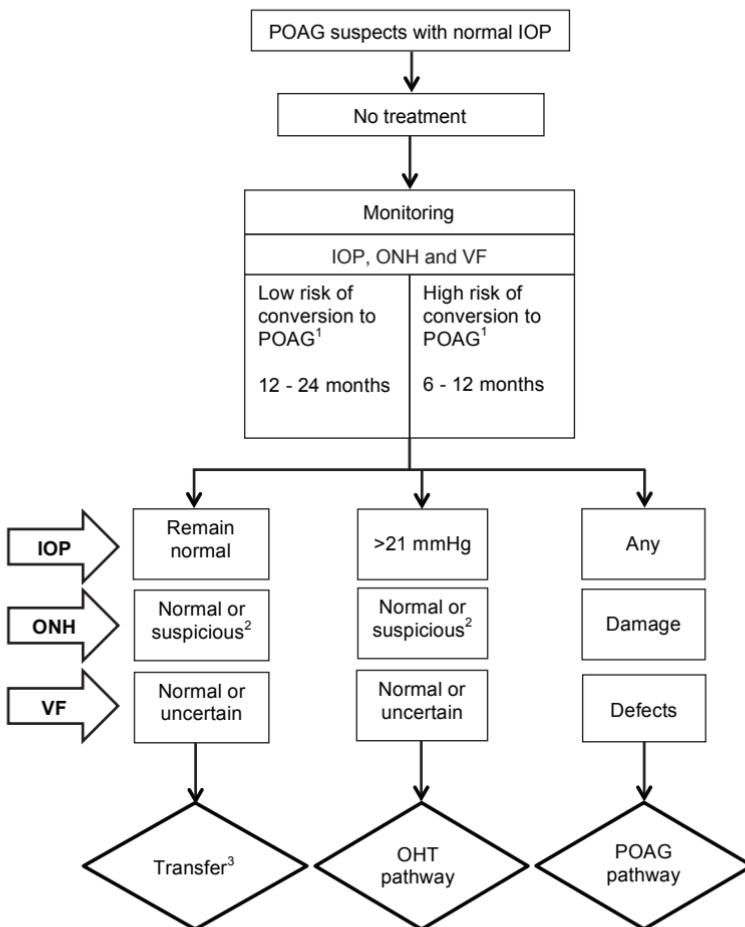
Modified: International Statistical Classification of Diseases and Related Health Problems 10th Revision (ICD-10)-WHO Version for 2016 (available at <http://apps.who.int/classifications/icd10/browse/2016/en#/H53-H54>)

APPENDIX 11**OCULAR HYPERTENSION PATHWAY (OHT AND POAG SUSPECTS WITH HIGH IOP)**

Modified: National Collaborating Centre for Acute Care. Glaucoma: Diagnosis and management of chronic open angle glaucoma and ocular hypertension. London: RCS; 2009

APPENDIX 12

POAG SUSPECTS WITH NORMAL IOP PATHWAY



¹To be clinically assessed based on age, IOP, CCT, changes in ONH and VF

²ONH and/or peripapillary RNFL appearance is suggestive of, but not definitive for glaucoma

³If there is no change in the parameters after 3 - 5 years, the patient can be transferred from active glaucoma care to general ophthalmologist or primary care optometrist for annual assessment.

Modified: National Collaborating Centre for Acute Care. Glaucoma: Diagnosis and management of chronic open angle glaucoma and ocular hypertension. London: RCS; 2009.

LIST OF ABBREVIATIONS

α	alpha
β	beta
€	Euro
5-FU	5-Fluorouracil
µg	microgram
µm	micrometre
AAC	acute angle closure
ALPI	Argon laser peripheral iridoplasty
ALT	Argon laser trabeculoplasty
AS-OCT	Anterior Segment-Optical Coherence Tomography
AUD	Australian Dollar
CAI(s)	carbonic anhydrase inhibitor(s)
CCT	central corneal thickness
CDR	cup: disc ratio
CI	confidence interval
CPC	cyclophotocoagulation
CPG(s)	clinical practice guidelines
CSLO	confocal scanning laser ophthalmoscopy
D	diopter
dB	decibel
DM	diabetes mellitus
EGPS	European Glaucoma Prevention Study
GAT	Goldmann applanation tonometry
GDD	glaucoma drainage devices
GIT	gastrointestinal tract
GHT	Glaucoma hemifield test
GON	glaucomatous optic neuropathy
HR	hazard ratio
IOP	Intraocular pressure
ISNT	Inferior Superior Nasal Temporal
LCT	limbal corneal thickness
mg	milligramme
mJ	millijoule
ml	millilitre
mm	millimetre
mm ²	millimetre square
MMC	Mitomycin C
mmHg	millimetre mercury
mW	milliwatt
MoH	Ministry of Health
NRR	neuro-retinal rim
nsec	nanosecond
NTG	normal tension glaucoma
NVG	neovascular glaucoma
OAG	open angle glaucoma
OCT	optical coherence tomography
OD	optic disc
OHT	ocular hypertension

OHT	ocular hypertension
OHTS	Ocular Hypertension Treatment Study
ONH	optic nerve head
OR	odds ratio
OSA	Obstructive Sleep Apnoea
PACG	primary angle closure glaucoma
PAC	primary angle closure
PACD	peripheral anterior chamber depth
PACG	primary angle closure glaucoma
PACS	primary angle closure suspect
PAS	peripheral anterior synechiae
PSD	Pattern Standard Deviation
POAG	primary open angle glaucoma
QoL	quality of life
RCT(s)	randomised controlled trial(s)
RNFL	retinal nerve fibre layer
RR	relative risk
SAP	Standard Automated Perimetry
sec	second
SITA	Swedish Interactive Threshold Algorithm
SLT	Selective laser trabeculoplasty
TM	trabecular meshwork
TSCP	trans-scleral cyclophotocoagulation
USD	United States Dollar
VA	visual acuity
VEGF	vascular endothelial growth factor
VF	visual field

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