Primary Angle-Closure Glaucoma: A Comprehensive Meta-Narrative Review

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Absract

Primary angle-closure glaucoma, while less common than primary open-angle glaucoma, carries a 4- to 5-fold greater risk of severe visual morbidity. The identification of individuals at high risk of the disease enables proactive rather than reactive intervention, which helps mitigate the possibility of potentially serious consequences. Recognition is facilitated by careful case history, clinical examination, and ancillary imaging, while management is an evolving paradigm, informed by a number of relatively recent investigations, that may involve medications, laser procedures, surgery, or a combination thereof. This series of 4 papers, drawing upon relevant peer-reviewed literature, will endeavour to provide a comprehensive yet focused synthesis and synopsis of the contemporary diagnosis and management of primary angle-closure glaucoma.

Keywords

primary angle-closure glaucoma; gonioscopy; anterior segment optical coherence tomography; pupillary block; plateau iris; angle-closure continuum; laser peripheral iridotomy; cataract extraction

Introduction

Although primary angle-closure glaucoma represents 1 of every 4 of cases of glaucoma, it is responsible for 50% of glaucoma-related blindness worldwide.[1-3] In light of the fact that the number of people with the disease is projected to increase to 34 million by the year 2040, it represents a sizable public health challenge, particularly in Asia, where it may cause as much as 90% of glaucoma-related blindness.[4,5]

A number of epidemiologic and anatomic risk factors for angle closure have been identified, making careful case history and clinical examination of paramount importance.[6,7] Given that angle-closure glaucoma is a disease of ocular anatomy that is felt to be largely preventable with early identification and treatment, under-detection and the permanent vision loss that can follow is of significant concern.[8-10] Unfortunately, particularly in North America, angle-closure disease is very underdiagnosed, at least in part because gonioscopy (which remains the gold standard for assessing the anterior chamber angle) is performed infrequently and often poorly.[11-13]

Gonioscopy detects irido-trabecular contact, helps differentiate appositional from synechial closure and pupillary block from plateau iris, and informs disease staging.[14-17] A growing number of ancillary imaging modalities (including Scheimpflug imaging, ultrasound biomicroscopy, and anterior segment photography and optical coherence tomography) have become valuable complements too but, being largely 2-dimensional and static, are not replacements for gonioscopy.[9,18-20] That being said, these procedures have helped identify a number of novel biometric parameters quantifying the relationship between anterior segment structures, identifying risk factors for angle closure, and informing

Suggested citation

MacDonald D. Primary Angle-Closure Glaucoma: A Comprehensive Meta-Narrative Review. *Canadian Journal of Optometry*. 2025;87(3):45-57.
DOI : 10.15353/cjo.v87i3.6526

management decision making.[7,21-24] Both clinical and ancillary assessment help determine the relative influence of mechanisms including pupillary block, plateau iris, peripheral iris anatomy, and lens morphology, and have demonstrated that in many cases, multiple mechanisms are contributory.[9,10,25-27]

Following examination, the patient can be situated within a disease continuum that spans primary angle-closure suspect, primary angle-closure, and primary angle-closure glaucoma (more commonly chronic than acute, the latter considered a true ocular emergency) based upon the presence of iridotrabecular contact, intraocular pressure elevation and/or peripheral anterior synechiae (PAS) formation, and ultimately, glaucomatous optic neuropathy.[2,28-31] Although advancement through these stages remains poorly- characterized, recent data suggests that most patients progress slowly, and many not at all.[2,32]

Treatment of angle-closure disease involves medications, laser procedures (most commonly laser peripheral iridotomy), and incisional surgery, with controversy persisting around their positioning in the continuum.[33-35] The effectiveness of cataract surgery in primary angle-closure glaucoma has long been recognized,[36,37] and more recently the role of both cataract and clear lens extraction at other stages of disease severity has been the subject of extensive investigation.[38-40]

Discussion

Glaucoma, an optic neuropathy leading to the death of retinal ganglion cells (RGCs), is the second leading cause of blindness worldwide, affecting approximately 80 million individuals in 2020, a number projected to increase by nearly 50% over the next 20 years.[5,41,42] The disease can be broadly categorized by mechanism as open angle or angle closure,[43] and further subdivided as primary or secondary.[28,44] Primary angle-closure glaucoma (PACG) is disease that develops in an anatomically predisposed eye in the absence of other ocular or systemic abnormalities. It represents 1 of every 4 of cases of glaucoma, but is responsible for 50% of glaucoma-related blindness, suggesting that the risk of severe vision loss is 4- to 5-fold higher in ACG than open-angle disease.[1,2,17,45] Indeed, review of the American Association of Ophthalmology Intelligent Research in Sight (IRIS) Registry found that blindness in at least 1 eye impacted 1 in 9 patients in the United States with newly diagnosed PACG, including 1 in 3 patients under the age of 40 years.[46] Moreover, by 2040 the number of people with PACG is projected to increase to 34 million, with over 5 million (approximately 1 in 7) being bilaterally blind.[5]

Despite its visually devastating impact, ACG is a very understudied disease. In early 2023, only 85 investigations of angle closure were registered at ClinicalTrials.gov, versus over 860 related to open-angle glaucoma, OAG.[47]

Particularly in North America, the disease is also very underdiagnosed, with primary OAG being identified 32 times more often than primary ACG.[26] In some ways this may be a self-fulfilling prophecy as eye care providers tend to assume that the prevalence of PACG in the United States and Canada is very low.[46] Another large contributor to this disparity is an error of omission. Gonioscopy, a test that will be reviewed in detail in this report, is critical in differentiating OAG from ACG and primary from secondary glaucoma, but is performed less than half the time in both the initial workup of patients being investigated for glaucoma, and in the 5 years preceding glaucoma surgery.[11,48] In fact, as many as 1 in 8 patients referred for cataract surgery were found to have undocumented narrow angles,[13] and one in ten referred for primary OAG were subsequently diagnosed with primary ACG.[49] This suggests that gonioscopy is performed infrequently and when it is performed, it is often performed poorly. Under-detection and the attendant risk of irreversible vision loss is of significant concern, given that PACG is a disease of ocular anatomy, and considered preventable with early identification and prophylactic treatment.[8-10,25]

Epidemiology and Risk Factors

Prevalence data for PACG is sparse and subject to differences in disease definition and stage, and study methodology. Estimates are approximately 0.6% globally, ranging from 0.25% in North America to 2.65% among Inuit.[5,10,18,35,50-52]

In general, the same can be said about disease incidence, with rates varying from 4.7/100,000 per year in Finland to 15.5/100,000 per year in Singapore, increasing as one moves from west to east.[18,53]

Moreover, given that the majority of ACG in Asia is chronic and asymptomatic, published figures likely significantly underestimate true disease incidence.[54]

A number of risk factors for PACG have been identified, none of which should be considered in isolation but rather as building blocks of an individual's risk profile.[6] Much like OAG, perhaps the strongest is advancing age, with the prevalence of ACG being nearly 50 times higher for Europeans ≥70 years of age than for those aged 40 to 49 (0.95% versus 0.02%), and the risk of progression tripling with each passing decade.[7,50] This is felt to be primarily secondary to age-related thickening of the crystalline lens (increasing ~0.75 mm between the ages of 30 and 80) reducing anterior chamber depth and increasing the risk of pupillary block.[7,8,52,55] Conversely, ACG in patients of Asian descent and those under the age of 40 tend to be related to iris anatomy.[56-59] Disease mechanisms will be reviewed in detail later in this series.

In patients of any age, risk factors for ACG include a thicker (particularly if >5.5 mm) and/or more anteriorly positioned lens, short axial length (particularly if <21 mm), smaller and steeper cornea, narrow peripheral angle, and shallow central anterior chamber depth (particularly if <2.5 mm).[3,8,18,28,53,60-63] Indeed, the mean ACD of individuals with PACG is between 0.5 mm (in southeast Asians) and 1.0 mm (in Europeans) less than that of controls, while mean axial length is ~0.75 mm shorter.[8,55] In Chinese individuals with open angles (Shaffer grade IV, one of several classification systems that will be reviewed in this paper) the ACD averages 2.73 mm, versus 1.94 mm in those with closed angles (Shaffer grade 0).[64]

Although hyperopia is a strong risk factor for PACD,[3] age-related nuclear sclerosis may simultaneously thicken the lens and induce a myopic shift, meaning that older patients may appear less hyperopic but still be at high risk of angle closure.[36,55]

ACG is 3 to 5 times more common in women, who tend to have smaller eyes, shallower ACD, and longer lifespans than men.[3,18,41,52,55,65]

The disease is strongly related to ethnicity. Worldwide, over 75% of individuals with PACG are from Asia, where approximately 90% of glaucoma-related blindness is due to angle closure.[5,52,66-68] Indeed, nearly half the cases of PACG are found in China alone. As many as 80% of Chinese glaucoma diagnoses are PACG, and in 2001, it was estimated that nearly 30 million Chinese individuals had occludable angles.[4] Moreover, given the rapid increase and aging of the already large and elderly Chinese population, the number of people with PACG is expected to increase by nearly 75%, to approximately 14.5 million, by the year 2050.[65,68]

Intriguingly, it has been hypothesized that the prevalence of a shallow ACD, a strong risk factor for PACG, is an evolutionary compensatory measure to prevent corneal freezing among individuals residing in cold climates, including northern China.[69,70]

Patients with pseudoexfoliation (PEX) must be monitored carefully for more than OAG. In as many as 1 in 4 individuals with PEX, the zonule laxity that accompanies the condition (and increases with advancing age) may lead to anterior shift of the crystalline lens, decreased ACD, increased lens vault (one of many parameters that will be reviewed in detail in this series), and angle crowding.[35,36,71]

Pharmacologic mydriasis (causing pupillary block) and/or uveal effusion (causing anterior displacement of the lens/iris diaphragm) arising from the use of topical and systemic medications can precipitate iatrogenic angle closure in predisposed individuals.[72,73] Eye care providers will be familiar with topical mydriatic agents, while systemic drugs include but are not limited to antimigraine agents (particularly sumatriptan, with an odds ratio [OR] for angle closure of 12.6), sulfonamide antibiotics, diuretics, anti-inflammatory agents, antiseizure medications (particularly topiramate, OR 5.1), serotonergic drugs (particularly duloxetine, OR 4.0), benzodiazepines (OR up to 3.1), tricyclic antidepressants and monoamine oxidase inhibitors (OR up to 2.6, alpha- and beta-adrenergic agonists (including over-the-counter decongestants, OR up to 2.2), and antihistamines and anticholinergics (OR of up to 1.9).[73-75] Being pragmatic, balancing the benefits of these medications with the still relatively small risk of angle closure, individuals with pre-existing risk factors for ACG using drugs with increased OR should be monitored carefully, counselled on the signs and symptoms of APAC, and in rare cases may benefit from prophylactic treatment, which will be reviewed later in this series.[74,76]

Assessment of the Anterior Chamber Angle: Van Herick Assessment

As previously noted, the diagnosis of glaucoma (including the differentiation of ACG from OAG, and primary from secondary mechanisms) cannot

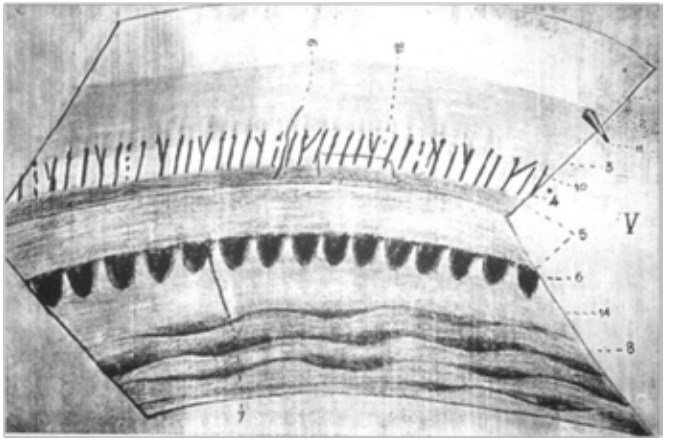
Figure 1a (on left). An example of a van Herick grade 3 angle, considered not occludable

Figure 1b (on right). An example of a van Herick grade 1 angle, considered occludable

be made without assessing the status of the anterior chamber angle. The gold standard for doing so remains gonioscopy, although other methods do exist.[6,12]

For a half-century, the van Herick assessment (VHA) has been utilized to compare peripheral ACD (also termed limbal ACD, LACD) to peripheral corneal thickness in the screening of non-glaucomatous patients, using the biomicroscope and a bright white light angled 60° from the visual axis.[77] An angle is considered "open" when the peripheral ACD is >1/4 (>25%) of the peripheral corneal thickness (Figure 1; Castaneda-Diez, 2011).[45] Sensitivity and specificity of a VHA ≤25% vary considerably between studies (the former from 54 to 99%, the latter from 53 to 95%), and even when performed by glaucoma specialists, VHA fails to detect as many as 40% of angles judged to be occludable by gonioscopy.[78-80] Both intra- and inter-observer variability can be significant. The VHA gives no information about iris contour or angle structures,[20] nor can it detect PAS or any secondary mechanisms.[79,80] Moreover, while the VHA is often performed temporally, in approximately two-thirds of individuals, the narrowest anterior chamber angle is elsewhere, which would result in an underestimation of angle closure risk.[81] Additionally, the successful application of the VHA depends upon reasonable corneal clarity at the limbus, something that is by no means guaranteed, perhaps particularly so in more at-risk Asian populations.[78] While the VHA may be helpful as a reasonably sensitive (albeit not specific) screening test in a busy general clinic, clinical assessment of anterior chamber angle configuration is best accomplished with gonioscopy."[79,80,82]

Figure 2. A remarkably detailed drawing of the angle produced by Alexios Trantas in the late 1800s



Assessment of the Anterior Chamber Angle: Gonioscopy

Gonioscopy (derived from γωνία and σκόπηση, the Greek words for "angle" and "observe") has a long and fascinating history, beginning in the late 1800s with Alexios Trantas first observing the angle, a serendipitous discovery as he was attempting to visualize the extreme peripheral retina. The evolution continued through the early 1900s with Maximilian

Figure 3. A beautifully detailed illustration of the angle by Emil Bethke that appeared in the first comprehensive text dealing with gonioscopy, written by Manuel Uribe y Troncoso and published in the late 1940s

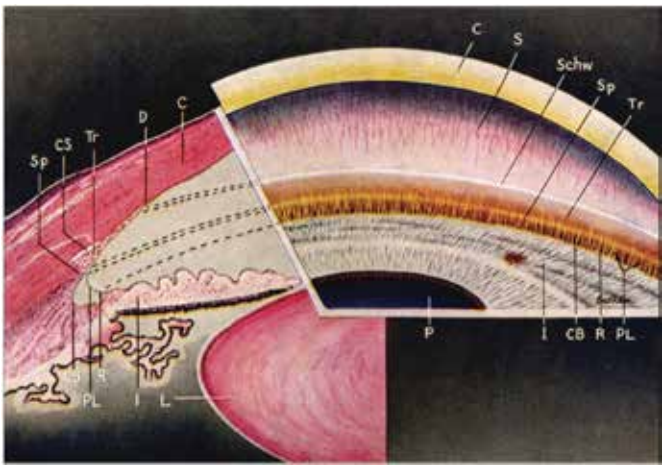
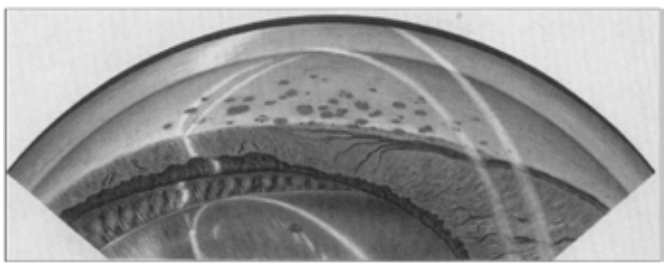


Figure 4. Using a 4-prism corneal lens that he personally developed in the mid-1950s



Lee Allen (who originally trained as an ocularist) produced extremely detailed drawings, including this carbon dust, pencil, and ink sketch depicting peripheral anterior synechiae.

Salzmann and Carl Zeiss (high plus lenses for direct gonioscopy), the 1920s and '30s with Leonhard Koeppe and Otto Barkan (angle-based surgery), and the 1940s with Hans Goldmann (indirect gonioscopy using a slit lamp biomicroscope and larger 3-mirror contact lens).[43,84-88] The smaller 4-mirror Zeiss, Posner, Sussman, and Volk corneal lenses that followed required no coupling medium, allowed compression/indentation gonioscopy, and remain in common use today.[89]

Specific to ACG, gonioscopy helps detect iridotrabecular contact (ITC, the hallmark of the disease), and compression/indentation helps differentiate appositional from synechial closure, and pupillary block from plateau iris.[17]

Direct gonioscopy using a high plus contact lens (often the prototypical +50D Koeppe lens) is rarely used in clinical practice but plays a critical role in pediatric examinations and angle-based surgery. It provides a panoramic, upright, and non-inverted view of the entire angle (and with the simultaneous use of 2 lenses, both angles).[90-92]

Indirect gonioscopy using a mirrored lens at the biomicroscope is more easily integrated into daily practice. It provides an inverted view of the angle opposite the mirror being used. The lens may be large in diameter (12 to 15 mm) with a steep radius of curvature (7.4 mm) and require a viscous coupling medium (the prototypical Goldmann scleral lens) or be smaller (9 mm), flatter (7.85 mm), and use the patient's tears as the coupling medium (Zeiss, Posner, Sussman, or Volk corneal lenses).[20] Only the latter allow for compression/indentation gonioscopy, but for that reason require gentle pressure to avoid inadvertently deepening the angle and temporarily reducing IOP.[88,93]

Performing Indirect Gonioscopy

The stepwise performance of indirect gonioscopy can be summarized as follows:

1. Instill anesthetic in both eyes and explain the procedure to the patient
2. Place the lens on the cornea
    a. the patient may initially look up then straight ahead to control the lids and facilitate lens placement
    b. care must be taken to keep contact pressure extremely light to avoid artificially deepening the angle
3. Use low ambient lighting and a short narrow beam of the dimmest possible illumination
    a. care must be taken to avoid inadvertent pupil constriction, which can also artificially deepen the angle
4. Be systematic: begin with the superior mirror (examining the inferior angle) and proceed clockwise
    a. when an individual is in an upright posture, the inferior angle tends to be

Figure 5a (top left). The most posterior angle structure visible is the peripheral iris, the contour of which can be described as concave, flat, or convex, while the angle of approach can vary from shallow to steep

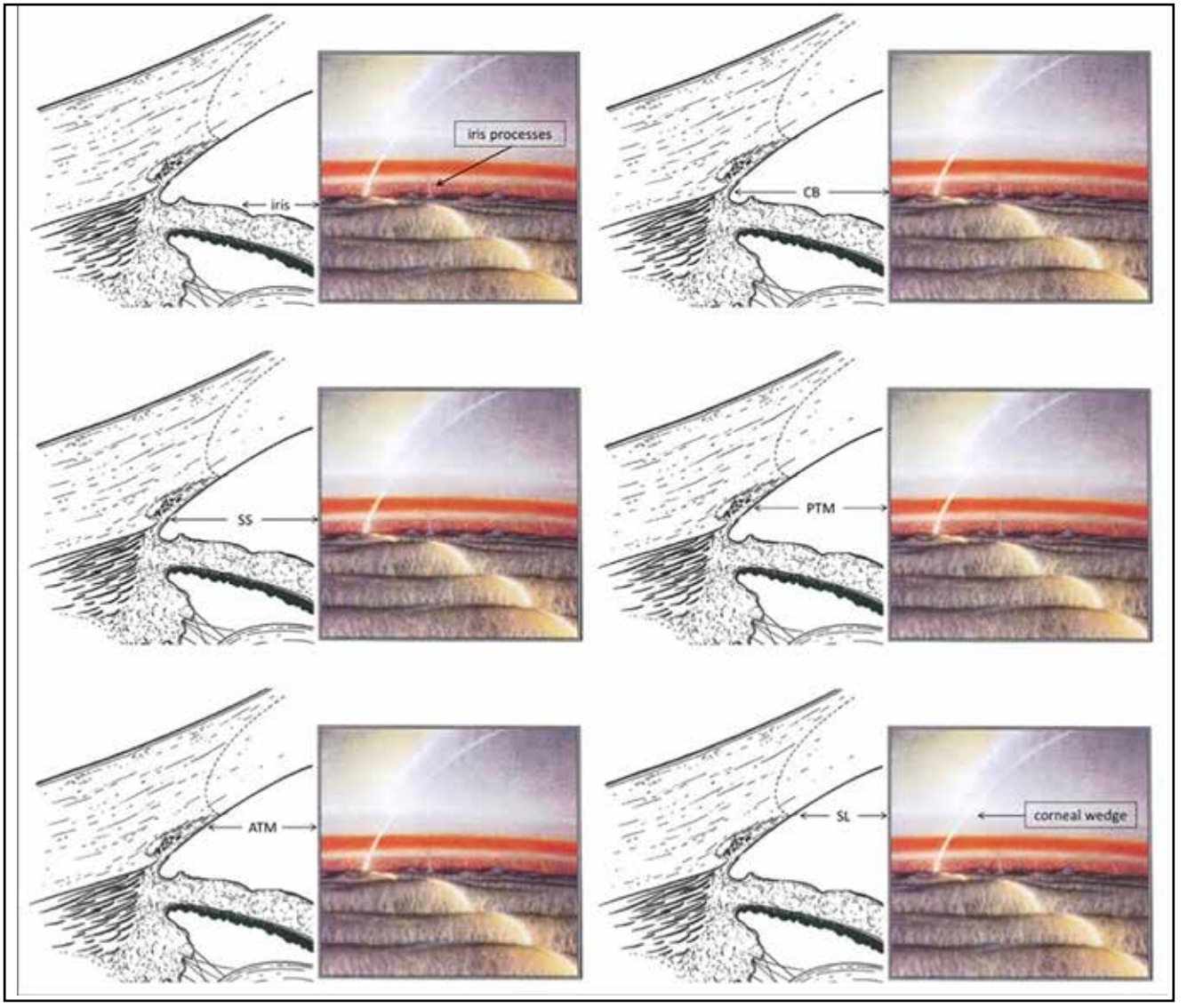
Figure 5b (top right). The ciliary body band (CB or CBB) is noted as a variably coloured structure anterior to the root of the iris

Figure 5c (middle left). The scleral spur (SS) represents the insertion of the ciliary muscle into the sclera, and can be localized as a whitish-grey band between the CBB and posterior (usually pigmented) trabecular meshwork

Figure 5d (middle right). The posterior (usually pigmented) trabecular meshwork (PTM) is the functional two-thirds of the TM found just anterior to the SS, overlying Schlemm's canal (visibility of PTM suggests that the angle is open in that particular area)

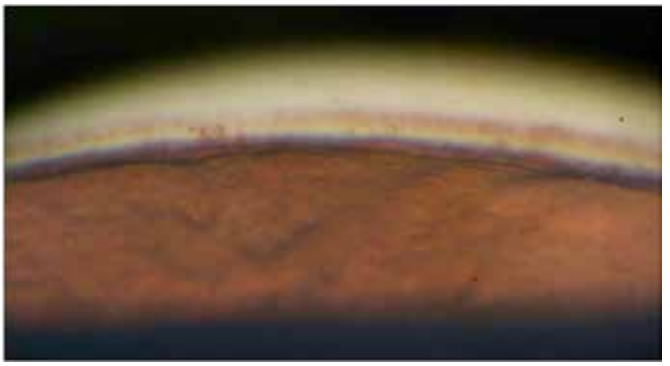
Figure 5e (bottom left). The anterior non- or lightly pigmented trabecular meshwork (ATM) is the non-functional one-third of the TM, having more of a ground-glass appearance

Figure 5f (bottom right). Schwalbe line (SL) is a fine whitish linear opacity that marks the peripheral termination of Descemet membrane, lying between ATM and corneal endothelium (if no angle structures posterior to SL are visible, it suggests that the angle is very narrow or closed in that particular area)



Particularly in cases of pigment dispersion or exfoliation, SL may be variably pigmented: while this may aid in its identification, it can also cause confusion by mimicking PTM. Using a slightly off-axis narrow slit beam, SL can be localized as the intersection of the light reflexes from the anterior and posterior corneal surfaces.

Figure 6. A gonioscopic photograph of an open angle, depicting (most posteriorly) the brown iris surface ending at the pigmented ciliary body, benign iris processes extending across the brighter scleral spur to the posterior pigmented trabecular meshwork, and (most anteriorly) the brighter Schwalbe line and corneal endothelium



deepest and most pigmented, making angle structures easier to identify[18]

5. Use mid to high magnification to identify angle details
6. The lens may be tilted slightly toward the angle being visualized (that is, away from the mirror being used) to "look over" a steep iris and see deeper into an open angle[92]
    a. the lens may also be tilted toward any bubbles to eliminate them
        i. the occasional bubble can be taken as a positive sign that the pressure being used is not excessive
7. Compress/indent to differentiate appositional from synechial closure and help in the identification of plateau iris
    a. in appositional closure, the iris will move posteriorly with gentle pressure, assuming a slightly convex profile; in areas of synechial closure, the peripheral iris will remain in contact with the posterior cornea
    b. in plateau iris, while the central iris bows posteriorly with gentle pressure, the peripheral iris does not; rather, it appears to bulge forward because of an anteriorly displaced ciliary body, creating the "double hump" sign, to be discussed in more detail later in this series
    c. in the case of an anteriorly displaced crystalline lens, the iris moves only slightly posteriorly while retaining a convex profile

Interpreting Gonioscopy

The angle structures visible during gonioscopy, from posterior to anterior (that is, from open to closed) are as follows,[20,91] and presented as Figures 5a through 5f.

- peripheral iris: note the contour (concave, flat, or convex) and angle of approach of the iris
- ciliary body band (CBB): a pink, brown/tan, or grey band anterior to the iris root
    - the CBB is typically wider in a myopic than in a hyperopic eye
- scleral spur (SS): the insertion of the ciliary muscle to the sclera forms a whitish-grey band between the CBB and posterior trabecular meshwork
    - the SS may be obscured by benign iris processes or pathologic PAS (to be reviewed in more detail later in this report)
- posterior (pigmented) trabecular meshwork (PTM): the functional two-thirds of the TM lying anterior to the SS and overlying Schlemm's canal
    - PTM visibility suggests that the angle is open in that area[82]
- anterior trabecular meshwork (ATM): the non- or lightly pigmented non-functional anterior one-third of the TM has more of a ground-glass appearance
- Schwalbe's line (SL): a fine opaque whitish line that marks the peripheral termination of Descemet's membrane, lying between the ATM and the corneal endothelium
    - visibility of no structures posterior to SL suggests that the angle is extremely narrow or closed in that area
    - SL may be very prominent (posterior embryotoxon) and/or variably pigmented (Sampaolesi's line, suggesting the presence of pigment dispersion or pseudoexfoliation, the latter of which can increase the risk of angle closure)
        - care must be taken to avoid misidentifying a pigmented SL as pigmented TM,

Figure 7a (on left). An angle viewed without compression/indentation shows few definitively visible angle structures, with perhaps a small section of posterior pigmented trabecular meshwork at the extreme left side of the light beam

Figure 7b (on right). With compression/indentation the angle widens, making the posterior pigmented trabecular meshwork and scleral spur visible in the left half of the light beam, while a posterior synechiae persists on the right side of the light beam, precluding visibility of any angle structures in that region

potentially leading to a closed angle being misclassified as open

- SL may also be localized by using a slightly off-axis slit lamp beam and noting where the anterior and posterior reflections of the corneal wedge meet (Figure 5f). This can be particularly helpful in a very lightly pigmented and/or very narrow angle where more posterior structures may not be visible[91]
  - if uncertainly persists, gentle compression/indentation may be applied to an angle thought to be open to see whether any more posterior structures become visible. If so, the angle was narrower than initially thought

- cornea: the posterior surface of the cornea can be visualized anterior to SL
  - if the cornea is the only visible structure, the angle is closed in that area

Obviously, gonioscopy is skill intensive, somewhat time consuming, and notwithstanding attempts at standardization, remains largely subjective.[17,20,92] Notwithstanding these limitations, gonioscopy is *in vivo*, requires little in the way of specialized instrumentation, provides dynamic real-time 3-dimensional information on much more than simply angle width, and despite some of the amazing technology to be reviewed later in this series, remains the gold standard for angle assessment.[12,82]

Of course, differentiating abnormal from normal requires a familiarity with the latter. Gonioscopy should be performed regularly (which also helps overcome what can be a steep learning curve) and not reserved solely for difficult cases in which abnormality is anticipated.[20]

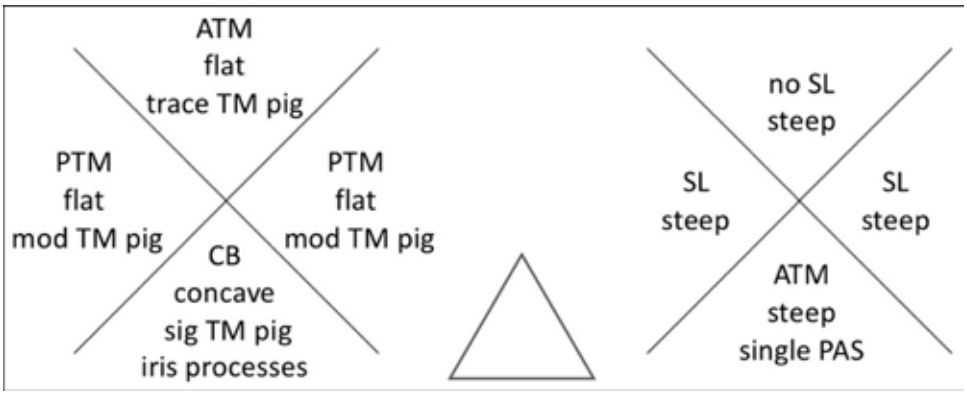
A gonioscopic photograph of an open anterior chamber angle is presented as Figure 6 (Castaneda-Diez, 2011),[45] and one of an appositionally-closed angle, pre- and post-compression/indentation, is depicted in Figure 7 (Castaneda-Diez, 2011).[10,45,89]

Gonioscopic Grading Systems

Over the past 80 years, a number of standardized grading systems that attempt to correlate gonioscopic appearance with the risk of angle closure have been proposed, including those of Shaffer, Spaeth, and Sheie.[14-16]

The Shaffer system,[15] likely the most commonly used standardized classification in clinical practice,[20] is based on the angle between the anterior surface of the iris and posterior surface of the cornea. Closure is felt to be very unlikely at grade IV (≥35°, CBB seen) or III (≥20°, SS seen), possible at grade II (≤20°, ATM but not PTM seen), very likely/

Figure 8. Hybrid qualitative system



Rather than risk the confusion that an angle grading system can cause, clinicians may simply note the most posterior angle structure visible, and include a description of iris approach and any abnormalities (including amount of pigmentation and presence of synechiae), for each quadrant of both eyes (as a hypothetical example, in the temporal quadrant of this patient's right eye, the iris approach was flat and the most posterior structure visible was a moderately pigmented PTM, suggesting an open angle; however, in the superior quadrant of the left eye, the iris approach was steep and SL was not visible, suggesting a closed angle)

imminent at grade I (≤10°, SL seen), and present at grade 0 (0°, no angle structures seen without compression/indentation).[45,92] This system, however, gives no information on iris contour or insertion, nor angle pigmentation.

The Spaeth system[16] is essentially a more complex extension of the Shaffer system. In addition to angle of iris approach, it describes level of iris insertion (from A to E as the insertion moves from extremely anterior to posterior) and contour (as steep, regular, or queer, the latter referring to a relatively concave peripheral contour). Iris contour may also be noted as b (bowed forward), f (flat), c (concave), or p (plateau), and a description of angle pigmentation (from 0 [no visible pigmentation] to 4+ [dense black pigment]) and iris processes (U, along angle recess; V, to TM; W, to SL) may be added. The Spaeth system also allows for incorporation of the results of dynamic and/or compression/indentation gonioscopy, making it the most comprehensive (and complicated) grading scheme.[84,92]

The Sheie system[14] grades the angle based on visible structures, from grade 0 and I (CBB completely and partially seen respectively; closure very unlikely) through grade II (SS seen; closure unlikely) and grade III (PTM not seen; closure likely), to grade IV (no visible structures; angle closed). Sheie's grading of PTM pigmentation (from grade 0 [no pigment] to grade IV [dense pigment]) is used to this day.

As if angle grading systems weren't complex enough, it is important to recognize that the Shaffer and Scheie systems are opposite: that is, a Shaffer grade I angle is very narrow, while a Scheie grade I angle is wide open.[14,15]

Moreover, studies using anterior segment optical coherence tomography (AS-OCT) have shown that ordinal grades of angle status (e.g., 0 through IV) do not represent equal intervals of progression through the angle-closure continuum, and highlighted "the need for a better method of tracking the natural history of angle-closure disease using gonioscopy in conjunction with quantitative data."[94]

To help minimize confusion, perhaps the most useful grading system in a clinical setting is a qualitative hybrid: in each quadrant, the most posterior visible angle structure is noted, with a description of iris approach (e.g., concave, flat, or steep) and any angle abnormalities (e.g. PAS, pigment, recession, or neovascularization). An example of how this particular system may appear in a clinical record (in essence, simply recording what you see, rather than assigning number or letter grades) is presented as Figure 8.

Pending

Part two of this four-part series will deal with ancillary imaging modalities, including anterior segment optical coherence tomography and quantitative anterior segment parameters. Part three will review the primary angle-closure disease spectrum, including the diagnosis and treatment of acute primary angle closure, and pathophysiologic mechanisms. The final section will delve into treatment, highlighting laser, medical, and surgical interventions applicable to different stages of the PACD continuum.

Conclusion

Primary angle-closure glaucoma remains an important yet, unfortunately and unnecessarily, an underdiagnosed cause of glaucoma-related visual impairment. Through recognition of risk factors, careful clinical assessment, and the judicious use of evolving ancillary imaging modalities, primary care optometrists can identify this largely preventable disease of ocular anatomy early in the angle-closure spectrum. This sets the stage for effective medical, procedural, and surgical intervention, means to the end of preventing vision loss and preserving vision-related quality of life.

Disclosures

Funding: This study did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Competing interests: All authors have completed the ICMJE uniform disclosure form and declare no conflict of interest.

AI Statement: The authors confirm no generative AI or AI-assisted technology was used to generate content.

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References

1. Quigley HA. The number of people with glaucoma worldwide. *Br J Ophthalmol.* 1996;80:389-93.
2. He M, et al. Laser peripheral iridotomy for the prevention of angle closure: a single-centre, randomised controlled trial. *Lancet.* 2019;393:1609-18.
3. Shan S, et al. Global incidence and risk factors for glaucoma: a systematic review and meta-analysis of prospective studies. *J Glob Health.* 2014;14:04252.
4. Foster PJ, Johnson GJ. Glaucoma in China: how big is the problem? *Br J Ophthalmol.* 2001;85:1277-85.
5. Tham YC, et al. Global prevalence of glaucoma and projections of glaucoma burden through 2040. A systematic review and meta-analysis. *Ophthalmology.* 2014;121:2081-90.
6. Thomas R, Walland MJ. Management algorithms for primary angle closure disease. *Clin Exp Ophthalmol.* 2013;41:282-92.
7. Xu BY, et al. Ocular biometric risk factors for progression of primary angle closure disease. The Zhongshan Angle Closure Prevention Trial. *Ophthalmology.* 2022;129:267-75.
8. Tarongoy P, et al. Angle-closure glaucoma: the role of the lens in the pathogenesis, prevention, and treatment. *Surv Ophthalmol.* 2009;54:211-25.
9. Nongpiur ME, et al. Angle closure glaucoma: a mechanistic review. *Curr Opin Ophthalmol.* 2011a;22:96-101.
10. Sun X, et al. Primary angle closure glaucoma: what we know and what we don't know. *Prog Ret Eye Res.* 2017;58:26-45.
11. Coleman AL, et al. Use of gonioscopy in Medicare beneficiaries before glaucoma surgery. *J Glaucoma.* 2006;15:486-93.
12. Smith SD, et al. Evaluation of the anterior chamber angle in glaucoma. A report by the American Academy of Ophthalmology. *Ophthalmology.* 2013;120:1985-97.
13. Varma DK, et al. Proportion of undetected narrow angles or angle closure in cataract surgery referrals. *Can J Ophthalmol.* 2017a;52:366-72.
14. Scheie HG. Width and pigmentation of the angle of the anterior chamber: a system of grading by gonioscopy. *AMA Arch Ophthalmol.* 1957;58:510-2.
15. Shaffer RN. Primary glaucomas, gonioscopy, ophthalmoscopy and perimetry. *Trans Am Acad Ophthalmol Otolaryngol.* 1960;62:112-27.
16. Spaeth GL. The normal development of the human anterior chamber angle: a new system of descriptive grading. *Trans Ophthalmol Sci UK.* 1971;91:709-39.
17. Radhakrishnan S, Chen L. Diagnosis and monitoring of primary angle closure. *Curr Ophthalmol Rep.* 2015;3:51-7.

18. He M, et al. Angle-closure glaucoma in East Asian and European people: different diseases? *Eye.* 2006a;20:3-12.
19. Nongpiur ME, et al. Lens vault, thickness and position in Chinese subjects with angle closure. *Ophthalmology.* 2011b;118:474-9.
20. Riva I, et al. Anterior chamber angle assessment techniques: a review. *J Clin Med.* 2020;9:3814.
21. Pavlin CJ, et al. Ultrasound biomicroscopy in plateau iris syndrome. *Am J Ophthalmol.* 1992;113:390-5.
22. Foo LL, et al. Determinants of angle width in Chinese Singaporeans. *Ophthalmology.* 2012;119:278-82.
23. Jones LW, et al. (2018). Diagnostic instruments. In N. Efron (Ed.), *Contact Lens Practice* (3rd ed., pp. 327-45). Elsevier.
24. Porporato N, et al. Role of anterior segment optical coherence tomography in angle-closure disease: a review. *Clin Exp Ophthalmol.* 2018;46:147-57.
25. Ritch R. Plateau iris is caused by abnormally positioned ciliary processes. *J Glaucoma.* 1992;1:23-6.
26. American Academy of Ophthalmology Glaucoma Panel. *Preferred Practice Pattern Guidelines. Primary Angle Closure*. San Francisco: AAO; 2015.
27. Wang N, et al. Primary angle closure glaucoma in Chinese and Western populations. *Chin Med J.* 2002;115:1706-15.
28. Foster PJ, et al. The definition and classification of glaucoma in prevalence surveys. *Br J Ophthalmol.* 2002;86:238-42.
29. Thomas R, et al. Five year risk of progression of primary angle closure suspects to primary angle closure: a population based study. *Br J Ophthalmol.* 2003a;87:450-4.
30. Thomas R, et al. Five year risk of progression of primary angle closure to primary angle closure glaucoma: a population based study. *Acta Ophthalmol Scand.* 2003b;81:480-5.
31. Day AC, Gazzard G. Missed opportunities in preventing acute angle closure – needlessly blind? *JAMA Ophthalmol.* 2022;140:604-5.
32. Baskaran M, et al. The Singapore Asymptomatic Narrow Angles Laser Iridotomy Study. Five-year results of a randomized controlled trial. *Ophthalmology.* 2022;129:147-58.
33. Robin AL, Pollack IP. Argon laser peripheral iridotomies in the treatment of primary angle closure glaucoma. *Arch Ophthalmol.* 1982;100:919-23.
34. Tham CC, et al. Phacoemulsification versus combined phacotrabeculectomy in medically uncontrolled chronic angle closure glaucoma with cataracts. *Ophthalmology.* 2009;116:725-31.
35. Wright C, et al. Primary angle-closure glaucoma: an update. *Acta Ophthalmol.* 2016;94:217-25.
36. Jacobi PC, et al. Primary phacoemulsification and intraocular lens implantation for acute angle-closure glaucoma. *Ophthalmology.* 2002;109:1597-1603.
37. Lam DSC, et al. Randomized trial of early phacoemulsification versus peripheral iridotomy to prevent intraocular pressure rise after acute primary angle closure. *Ophthalmology.* 2008;115:1134-40.
38. Azuara-Blanco A, et al. Effectiveness of early lens extraction for the treatment of primary angle-closure glaucoma (EAGLE): a randomized controlled trial. *Lancet.* 2016;388:1389-97.
39. Chan PP, Tham CC. Commentary on effectiveness of early lens extraction for the treatment of primary angle-closure glaucoma (EAGLE). *Ann Eye Sci.* 2017;2:21.
40. Song MK, et al. Glaucomatous progression after lens extraction in primary angle closure disease spectrum. *J Glaucoma.* 2020;29:711-7.
41. Quigley HA, Broman AT. The number of people with glaucoma worldwide in 2010 and 2020. *Br J Ophthalmol.* 2006;90:262-7.
42. Weinreb RN, et al. The pathophysiology and treatment of glaucoma: a review. *JAMA.* 2014;311:1901-11.
43. Barkan O. Glaucoma classification: causes and surgical control. *Am J Ophthalmol.* 1938a;21:1099-1113.
44. Jonas JB, et al. Glaucoma. *Lancet* 2017;390:2183-93.
45. Castaneda-Diez R, et al. (2011). Current Diagnosis and Management of Angle-Closure Glaucoma. In P. Gunvant (Ed.), *Glaucoma - Current Clinical and Research Aspects* (pp. 145-168). IntechOpen. doi:10.5772/662
46. Shah SN, et al. Prevalence and risk factors of blindness among primary angle closure patients in the United States: an IRIS® Registry analysis. *Ophthalmology.* 2024;259:131-40.
47. Azuara-Blanco A. Use of biometric data after laser peripheral iridotomy – individualized monitoring strategy for angle closure prevention. *JAMA Ophthalmol.* 2023;141:524.
48. Fremont AM, et al. Patterns of care for open-angle glaucoma in managed care. *Arch Ophthalmol.* 2003;121:777-83.

49. Varma DK, et al. Undetected angle closure in patients with a diagnosis of open-angle glaucoma. *Can J Ophthalmol.* 2017b;52:373-8.
50. Day AC, et al. The prevalence of primary angle closure glaucoma in European derived population: a systematic review. *Br J Ophthalmol.* 2012;96:1162-7.
51. Zhang H, et al. Clinical characteristics, rates of blindness, and geographic features of primary angle-closure disease in hospitals of the Chinese Glaucoma Study Consortium. *Can J Ophthalmol.* 2021;56:299-306.
52. Zhang N, et al. Prevalence of primary angle closure glaucoma in the last 20 years: a meta-analysis and systematic review. *Front Med.* 2021;7:624179.
53. Vijaya L, et al. Six-year incidence of angle-closure disease in a South Indian population: the Chennai Eye Diseases Incidence Study. *Am J Ophthalmol.* 2013;156:1308-15.
54. Wong TY, et al. Rates of hospital admissions from primary angle closure glaucoma among Chinese, Malays, and Indians in Singapore. *Br J Ophthalmol.* 2000;85:900-2.
55. Salmon JF. Predisposing factors for chronic angle-closure glaucoma. *Prog Ret Eye Res.* 1998;18:121-32.
56. Congdon NG, et al. Biometry and primary angle-closure glaucoma among Chinese, white and black populations. *Ophthalmology.* 1997;104:1489-95.
57. Ritch R, et al. Angle closure in younger patients. *Ophthalmology.* 2003;110:1880-9.
58. Stieger R, et al. Prevalence of plateau iris syndrome in young patients with recurrent angle closure. *Clin Exp Ophthalmol.* 2007;35:409-13.
59. Xu Y, et al. The ocular biometry characteristics of young patients with primary angle-closure glaucoma. *BMC Ophthalmol.* 2022;22:150.
60. Lowe RF. Aetiology of the anatomical basis for primary angle-closure glaucoma. Biometrical comparisons between normal eyes and eyes with primary angle-closure glaucoma. *Br J Ophthlamol.* 1970;54:161-9.
61. Lee DA, et al. Anterior chamber dimensions in patients with narrow angles and angle-closure glaucoma. *Arch Ophthalmol.* 1984;102:46-50.
62. Nolan WP, et al. Screening for primary angle closure in Mongolia: a randomised controlled trial to determine whether screening and prophylactic treatment will reduce the incidence of primary angle closure glaucoma in an east Asian population. *Br J Ophthalmol.* 2003;87:271-4.
63. Zhang Y, et al. Development of angle closure and associated risk factors: the Handan Eye Study. *Acta Ophthalmologica.* 2022;100:e253-e261.
64. Ng WT, Morgan W. Mechanisms and treatment of primary angle closure: a review. *Clin Exp Ophthalmol.* 2012;40:e218-e228.
65. Cheng JW, et al. The prevalence of primary glaucoma in mainland China: a systematic review and meta-analysis. *J Glaucoma.* 2013;22:301-6.
66. Congdon N, et al. Issues in the epidemiology and population-based screening of primary angle-closure glaucoma. *Surv Ophthalmol.* 1992;36:411-23.
67. Arkell SM, et al. The prevalence of glaucoma among Eskimos of northwest Alaska. *Arch Ophthalmol.* 1987;105:482-5.
68. Song P, et al. National and subnational prevalence and burden of glaucoma in China: a systematic analysis. *J Glob Health.* 2017;7:020705.
69. Casson RJ, et al. Gonioscopy findings and presence of occludable angles in a Burmese population: the Meiktila Eye Study. *Br J Ophthalmol.* 2007;91:856-9.
70. Qu 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:e515-e520.
71. Mohammadi M, et al. Evaluation of anterior segment parameters in pseudoexfoliation disease using anterior segment optical coherence tomography. *Am J Ophthalmol.* 2022;234:199-204.
72. Lachkar Y, et al. Drug-induced acute angle closure glaucoma. *Curr Opin Ophthalmol.* 2007;18:129-33.
73. Yang MC, Lin KY. Drug-induced acute angle-closure glaucoma: a review. *J Curr Glaucoma Pract.* 2019;13:104-9.
74. Wu A, et al. A review of systemic medications that may modulate the risk of glaucoma. *Eye.* 2020;34:12-28.
75. Na KI, Park SP. Association of drugs with angle closure. *JAMA Ophthalmol.* 2022;140:1055-63.
76. Foster PJ, et al. Association, risk, and causation – examining the role of systemic medications in the onset of acute angle-closure episodes. *JAMA Ophthalmol.* 2022;140:1064-5.
77. van Herick W, et al. Estimation of width of angle of anterior chamber: incidence and significance of the narrow angle. *Am J Ophthalmol.* 1969;68:626-9.

78. Congdon NG, et al. Screening techniques for angle-closure glaucoma in rural Taiwan. *Acta Ophthalmol Scand.* 1996;74:113-9.

79. Johnson TV, et al. Low sensitivity of the Van Herick method for detecting gonioscopic angle closure independent of observer expertise. *Am J Ophthalmol.* 2018;195:63-71.

80. Thompson AC, et al. Risk factors associated with missed diagnoses of narrow angles by the van Herick technique. *Ophthalmol Glaucoma.* 2018;1:108-14.

81. Gispets J, et al. Sources of variability of the van Herick technique for anterior angle estimation. *Clin Exp Optom.* 2014;97:147-51.

82. Friedman DS, He M. Anterior chamber angle assessment techniques. *Surv Ophthalmol.* 2008;53:250-73.

83. Dellaporta A. Historical notes on gonioscopy. *Surv Ophthalmol.* 1975;20:137-49.

84. Alward WLM. A history of gonioscopy. *Optom Vis Sci.* 2011;88:29-35.

85. Lowe RF. Curran, Barkan, and Chandler: a history of pupillary obstruction and narrow angle glaucoma. *J Glaucoma.* 1995;4:419-26.

86. Gloor BR. Hans Goldmann (1899-1991). *Eur J Ophthalmol*. 2010;20(1):1-11. doi:10.1177/112067211002000101.

87. Wong D, Fishman M. Lee Allen, The Man, The Legend. *J Ophthalmic Photogr.* 1990;12:51-67.

88. Hughes MO, et al. Lee Allen, Ocularist. *J Ophthalmic Prosthetics.* 2009;34:13-25.

89. Forbes M. Gonioscopy with corneal indentation: a method for distinguishing between appositional closure and synechial closure. *Arch Ophthalmol.* 1966;76:488-92.

90. Barkan O. Technic of goniotomy. *Arch Ophthalmol.* 1938b:19:217-23.

91. Fellman RL. (1998). Gonioscopy. In N.T. Choplin & D.C. Lundy (Eds.), A*tlas of Glaucoma* (1st ed., pp. 39-55.). Martin Dunitz Ltd.

92. Singh P, et al. Gonioscopy: a review. *Open J Ophthalmol.* 2013;3:118-21.

93. Schirmer KE. Gonioscopy and artefacts. *Br J Ophthalmol.* 1967;51:50-3.

94. Phu J, et al. Assessment of angle closure spectrum disease as a continuum of change using gonioscopy and anterior segment optical coherence tomography. *Ophthalmic Physiol Opt.* 2020;40:617-31.