PREFERRED PRACTICE PATTERN®

Primary Angle Closure

AMERICAN ACADEMY OF OPHTHALMOLOGY
The Eye M.D. Association

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As a service to its members and the public, the American Academy of Ophthalmology has developed a series of guidelines called Preferred Practice Patterns that **identify characteristics and components of quality eye care.** (See Appendix 1.)

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All PPPs are reviewed by their parent panel annually or earlier if developments warrant and updated accordingly. To ensure that all PPPs are current, each is valid for 5 years from the "approved by" date unless superseded by a revision. Preferred Practice Pattern guidelines are developed by the Academy's H. Dunbar Hoskins Jr., M.D. Center for Quality Eye Care without any external financial support. Authors and reviewers of PPPs are volunteers and do not receive any financial compensation for their contributions to the documents. The PPPs are externally reviewed by experts and stakeholders before publication.

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INTRODUCTION

The Preferred Practice Pattern® (PPP) guidelines have been written on the basis of three principles.

- Each PPP should be clinically relevant and specific enough to provide useful information to practitioners.
- Each recommendation that is made should be given an explicit rating that shows its importance to the care process.
- Each recommendation should be given an explicit rating that shows the strength of evidence that supports the recommendation and reflects the best evidence available.

In the process of revising this document, a detailed literature search of PubMed and the Cochrane Library was conducted on December 3, 2008 and April 24, 2009 on the subject of primary angle closure (PAC) for the years from January 1, 2004 to the date of the search. Details of the literature search are available at www.aao.org/ppp. The results were reviewed by the Glaucoma Panel and used to prepare the recommendations, which they rated in two ways. The panel first rated each recommendation according to its importance to the care process. This "importance to the care process" rating represents care that the panel thought would improve the quality of the patient's care in a meaningful way. The ratings of importance are divided into three levels.

- Level A, defined as most important
- Level B, defined as moderately important
- Level C, defined as relevant but not critical

The panel also rated each recommendation on the strength of evidence in the available literature to support the recommendation made. The “ratings of strength of evidence” also are divided into three levels.

- Level I includes evidence obtained from at least one properly conducted, well-designed, randomized, controlled trial. It could include meta-analyses of randomized controlled trials.
- Level II includes evidence obtained from the following:
  - Well-designed controlled trials without randomization
  - Well-designed cohort or case-control analytic studies, preferably from more than one center
  - Multiple-time series with or without the intervention
- Level III includes evidence obtained from one of the following:
  - Descriptive studies
  - Case reports
  - Reports of expert committees/organizations (e.g., PPP panel consensus with peer review)

Evidence is that which supports the value of the recommendation as it relates to the quality of care. The committee believes that it is important to make available the strength of the evidence underlying the recommendation. In this way, readers can appreciate the degree of importance the committee attached to each recommendation, and they can understand what type of evidence supports the recommendation.

The ratings of importance and the ratings of strength of evidence are given in bracketed superscripts after each recommendation. For instance, “[A:II]” indicates a recommendation with high importance to clinical care [A], supported by sufficiently rigorous published evidence, though not by a randomized controlled trial [II].

The sections entitled “Orientation” and “Background” do not include recommendations; rather they are designed to educate and provide summary background information and rationale for the recommendations that are presented in the Care Process section. A summary of the major recommendations for care is included in Appendix 2. Appendix 3 has an algorithm for the management of patients with acute angle-closure crisis (AACC). Appendix 4 contains the International Statistical Classification of Diseases and Related Health Problems (ICD) codes for the disease entities that the PPP covers.

ORIENTATION

DISEASE DEFINITION

Primary angle closure is appositional or synechial closure of the anterior chamber angle caused by multiple mechanisms.[1-3] Pupil block is considered to be a key element in the pathogenesis of most instances of PAC. The pressure in the posterior chamber is higher than the anterior chamber, causing an anterior bowing of the iris, which may crowd the angle in predisposed eyes. Certain factors can increase this pressure disparity between the two chambers (e.g., pupil dilation and thickening of the crystalline lens with age), which can result in iris apposition to the anterior chamber angle structures. In a minority of cases, this can happen acutely, resulting in acute angle closure.

Other important mechanisms play a role in angle closure, including the relative position and thickness of the ciliary body, the location of the iris insertion into the ciliary body, and the volume of the iris. Furthermore, it is clear that the crystalline lens size, shape, or position may play an important role in determining which eyes develop angle closure. Prolonged or repeated contact of the peripheral iris with the trabecular meshwork may lead to functional damage of the trabecular meshwork and the development of peripheral anterior synechiae (PAS). Angle closure may or may not be associated with elevated intraocular pressure (IOP) or glaucomatous optic neuropathy, and may occur in either an acute or chronic form. Secondary forms of angle closure can also occur (e.g., iridocorneal endothelial syndrome, inflammation, or neovascularization). This PPP focuses on PAC.

CLASSIFICATION AND CLINICAL CHARACTERISTICS OF ANGLE CLOSURE

Primary angle closure is generally bilateral, although 90% of acute attacks are unilateral.[4,5] Patients with angle closure and those at risk may be categorized as follows:

Primary Angle-Closure Suspect

Iridotrabecular contact (ITC), as observed on noncompressive gonioscopy, is defined as the iris seeming to touch the anterior chamber angle at the posterior pigmented trabecular meshwork or more anterior structures. It is the hallmark of PAC. The extent of ITC required to diagnose an eye as having angle closure has been the subject of debate. Consensus holds that a person with 180 degrees or more of ITC in primary gaze on gonioscopy is at risk of angle-closure glaucoma or an acute attack of angle closure. A person with this amount or more of ITC, no PAS, and normal IOP is considered a primary angle-closure suspect (PACS).[6] Only one study (carried out in south India) has reported on the natural history of these eyes. About one in four in this population developed elevation in IOP or PAS over 5 years.[7,8] Further longtitudinal studies in a variety of ethnic groups are needed.

Primary Angle Closure and Primary Angle-Closure Glaucoma

Any eye that has at least 180 degrees of ITC and an elevated IOP or PAS with no secondary cause for the PAS is classified as having PAC. The presence of high IOP and/or PAS is evidence that the ITC noted on gonioscopy may be causing permanent changes to the eye. When glaucomatous optic neuropathy is present (as defined in the Primary Open-Angle Glaucoma PPP[9]), the eye has progressed from PAC to primary angle-closure glaucoma (PACG).

Plateau iris configuration is used to refer to eyes that continue to have ITC after iridotomy, with a gonioscopic appearance of the peripheral iris closely apposed to the angle despite a deep central anterior chamber. Nearly one-third of PAC eyes treated with iridotomy have an angle that retains significant ITC.[10-12] Yet, on pupil dilation, the plateau iris configuration eyes do not have a significant increase in IOP, and no longitudinal study has shown that their risk of long-term development of PACG is higher than those eyes whose angles widen more after iridotomy. Rarely, plateau iris configuration eyes do have recurrent high IOP spikes after iridotomy; these eyes are called plateau iris syndrome and require more extensive treatments.

Acute Angle-Closure Crisis

If much of the chamber angle is obstructed suddenly, the IOP can rise rapidly to high levels. This may cause pressure-induced corneal edema (experienced as blurred vision and occasionally as multicolored haloes around lights), a mid-dilated pupil, vascular congestion, eye pain, and/or headache; this is termed acute angle-closure crisis or AACC. High IOP may be accompanied by nausea and vomiting. Acute angle-closure crisis may be self-limited and resolve spontaneously or may occur repeatedly. Untreated, this entity may cause permanent vision loss or blindness. The fellow eye is also at high risk of AACC.

PATIENT POPULATION

The patient population includes individuals of all ages, most of whom are adults over 50 who have risk factors that include abnormally high transpupillary resistance to aqueous humor movement, most often in association with pupil block.

ACTIVITY

Diagnosis and management of a patient with angle closure on gonioscopy.

PURPOSE

The purpose of treatment is to preserve visual function and maintain quality of life by preventing PACG or AACC from developing.

GOALS

- Identify those patients who are at risk of developing PACG or AACC or in whom it is present
- Manage an acute attack of angle closure
- Reverse or prevent angle closure by using laser iridotomy and/or iridoplasty or, if necessary, incisional iridectomy to alleviate pupil block
- Observe patients for chronic IOP elevation, progression of synechial angle closure, identification of unsuspected plateau iris by repeat gonioscopy after iridotomy, or optic nerve damage, and manage as indicated
- Evaluate the fellow eye for evidence of angle closure or an anatomic narrow angle in AACC and appropriately perform an iridotomy
- Educate the patient and family members about the characteristics of the disease and involve them in the management of it

BACKGROUND

EPIDEMIOLOGY

There are considerable differences in the prevalence of angle closure among ethnic groups. The highest rates are reported in Inuit,[13-15] Chinese,[16-20] and other Asian[21-29] populations; lower rates are reported in populations of African and African-derived origin[30-32] and European and European-derived origin[33-41] (see Table 1). Primary angle-closure glaucoma may account for nearly as many cases of glaucoma as open-angle glaucoma in some Asian populations.[21,42,43] Worldwide, 0.7% of people over 40 are estimated to have angle-closure glaucoma.[43] It is estimated that 21 million people worldwide will have angle-closure glaucoma in 2020.[43] In China, PACG is estimated to cause unilateral blindness (visual acuity <3/60 or visual field ≤10°) in 1.5 million individuals and bilateral blindness in another 1.5 million.[42]

TABLE 1 **PREVALENCE OF ANGLE CLOSURE**

Population	Location	Reported Prevalence (%)
Inuit	Alaskan Inuit[1,2]	2.65–3.8 (age >40)
	East Greenlandic Inuit[3]	2.5 (age ≥40)
Chinese	Taiwan[4]	3.0 (age ≥40)
	Guangzhou[5]	1.5 (age ≥50)
	Mongolia[6]	1.4 (age ≥40)
	Beijing[7]	1.2 (age ≥40)
	Singapore[8]	1.1 (age 40–79)
Other Asian	Burma[9]	2.5 (age ≥40)
	South Africa* [10]	2.30 (age ≥40)
	Southern India[11]	1.08 (age ≥40)
	Thailand[12]	0.9 (age ≥50)
	Southern India[13]	0.87 (age ≥40)
	Southern India[14]	0.5 (age ≥40)
	Bangladesh[15]	0.4 (age ≥40)
	Japan[16]	0.34 (age ≥40)
	Japan[17]	0.6 (age ≥40)
Hispanic	Arizona, U.S.[18]	0.10 (age >40)
African and African-derived	Northern Cape, S.A.* [10]	2.30 (age ≥40)
	Baltimore, U.S.[19]	0.6 (age ≥40)
	Tanzania[20]	0.58 (age ≥40)
	Temba, South Africa[21]	0.50 (age ≥40)
European and European-derived	Baltimore, U.S. (unpublished data)	0.60 (age ≥40)
	Italy[22]	0.60 (age >40)
	Blue Mountains, Australia[23]	0.27 (age ≥49)
	Bedford, U.K.[24]	0.17 (age >40)
	Ireland[25]	0.09 (age ≥50)
	Wales, U.K.[26]	0.09 (age ≥40–75)
	Melbourne, Australia[27]	0.06 (age ≥40)
	Beaver Dam, U.S.[28]	0.04 (age ≥43)
	Rotterdam[29]	0 (age ≥55)
	Sweden[30]	0 (age 55-69)

* Population in study is of mixed race ancestry mainly from Southeast Asia, but there was some African and White admixture.

NOTE: The studies reporting prevalence used different definitions of disease; therefore, these rates are not directly comparable.

1. Van Rens GH, Arkell SM, Charlton W, Doesburg W. Primary angle-closure glaucoma among Alaskan Eskimos. Doc Ophthalmol 1988;70:265-76.
2. Arkell SM, Lightman DA, Sommer A, et al. The prevalence of glaucoma among Eskimos of northwest Alaska. Arch Ophthalmol 1987;105:482-5.
3. Bourne RR, Sorensen KE, Klauber A, et al. Glaucoma in East Greenlandic Inuit--a population survey in Ittoqqortoormiit (Scoresbysund). Acta Ophthalmol Scand 2001;79:462-7.
4. Congdon NG, Quigley HA, Hung PT, et al. Screening techniques for angle-closure glaucoma in rural Taiwan. Acta Ophthalmol Scand 1996;74:113-9.
5. He M, Foster PJ, Ge J, et al. Prevalence and clinical characteristics of glaucoma in adult Chinese: a population-based study in Liwan District, Guangzhou. Invest Ophthalmol Vis Sci 2006;47:2782-8.

6. Foster PJ, Baasanhu J, Alsbirk PH, et al. Glaucoma in Mongolia. A population-based survey in Hovsgol province, northern Mongolia. Arch Ophthalmol 1996;114:1235-41.
7. Xu L, Zhang L, Xia CR, et al. The prevalence and its effective factors of primary angle-closure glaucoma in defined populations of rural and urban in Beijing [in Chinese]. Zhonghua Yan Ke Za Zhi 2005;41:8-14.
8. Foster PJ, Oen FT, Machin D, et al. The prevalence of glaucoma in Chinese residents of Singapore: a cross-sectional population survey of the Tanjong Pagar district. Arch Ophthalmol 2000;118:1105-11.
9. Casson RJ, Newland HS, Muecke J. Prevalence of glaucoma in rural Myanmar: the Meiktila Eye Study. Br J Ophthalmol 2007;91:710-4.
10. Salmon JF, Mermoud A, Ivey A, et al. The prevalence of primary angle closure glaucoma and open angle glaucoma in Mamre, western Cape, South Africa. Arch Ophthalmol 1993;111:1263-9.
11. Dandona L, Dandona R, Mandal P, et al. Angle-closure glaucoma in an urban population in southern India. The Andhra Pradesh eye disease study. Ophthalmology 2000;107:1710-6.
12. Bourne RR, Sukudom P, Foster PJ, et al. Prevalence of glaucoma in Thailand: a population based survey in Rom Klao District, Bangkok. Br J Ophthalmol 2003;87:1069-74.
13. Vijaya L, George R, Arvind H, et al. Prevalence of angle-closure disease in a rural southern Indian population. Arch Ophthalmol 2006;124:403-9.
14. Ramakrishnan R, Nirmalan PK, Krishnadas R, et al. Glaucoma in a rural population of southern India: the Aravind comprehensive eye survey. Ophthalmology 2003;110:1484-90.
15. Rahman MM, Rahman N, Foster PJ, et al. The prevalence of glaucoma in Bangladesh: a population based survey in Dhaka division. Br J Ophthalmol 2004;88:1493-7.
16. Shiose Y, Kitazawa Y, Tsukahara S, et al. Epidemiology of glaucoma in Japan--a nationwide glaucoma survey. Jpn J Ophthalmol 1991;35:133-55.
17. Yamamoto T, Iwase A, Araie M, et al. The Tajimi Study report 2: prevalence of primary-angle closure and secondary glaucoma in a Japanese population. Ophthalmology 2005;112:1661-9.
18. 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):1819-26.
19. Tielsch JM, Katz J, Singh K, et al. A population-based evaluation of glaucoma screening: the Baltimore Eye Survey. Am J Epidemiol 1991;134:1102-10.
20. Buhrmann RR, Quigley HA, Barron Y, et al. Prevalence of glaucoma in a rural East African population. Invest Ophthalmol Vis Sci 2000;41:40-8.
21. Rotchford, AP, Kirwan JF, Muller MA, et al. Temba glaucoma study: a population-based cross-sectional survey in urban South Africa. Ophthalmology 2003;110: 376-82.
22. Bonomi L, Marchini G, Marraffa M, et al. Prevalence of glaucoma and intraocular pressure distribution in a defined population. The Egna-Neumarkt Study. Ophthalmology 1998;105:209-15.
23. Mitchell P, Smith W, Attebo K, Healey PR. Prevalence of open-angle glaucoma in Australia. The Blue Mountains Eye Study. Ophthalmology 1996;103:1661-9.
24. Bankes JL, Perkins ES, Tsolakis S, Wright JE. Bedford glaucoma survey. Br Med J 1968;1:791-6.
25. Coffey M, Reidy A, Wormald R, et al. Prevalence of glaucoma in the west of Ireland. Br J Ophthalmol 1993;77:17-21.
26. Hollows FC, Graham PA. Intra-ocular pressure, glaucoma, and glaucoma suspects in a defined population. Br J Ophthalmol 1966;50:570-86.
27. Wensor MD, McCarty CA, Stanislavsky YL, et al. The prevalence of glaucoma in the Melbourne Visual Impairment Project. Ophthalmology 1998;105: 733-9.
28. Klein BE, Klein R, Sponsel WE, et al. Prevalence of glaucoma. The Beaver Dam Eye Study. Ophthalmology 1992;99:1499-504.
29. Dielemans I, Vingerling JR, Wolfs RC, et al. The prevalence of primary open-angle glaucoma in a population-based study in The Netherlands. The Rotterdam Study. Ophthalmology 1994;101:1851-5.
30. Bengtsson B. The prevalence of glaucoma. Br J Ophthalmol 1981;65:46-9.

RISK FACTORS

The following demographic and ocular factors have been implicated as risk factors for the presence of PAC.

Demographic Risk Factors

- Family history of angle closure[44,45]
- Older age[4,18,41]
- Female sex[4,46]
- Asian or Inuit descent [15,18,47-49]

Ocular Risk Factors

- Hyperopia[50-52]
- Shallow peripheral anterior chamber depth[5,50,52-58]
- Shallow central anterior chamber depth[52,59-63]
- Steep corneal curvature[64]
- Thick crystalline lens[52,65]
- Short axial length[52,65,66]

NATURAL HISTORY

If patients with unilateral AACC and high IOP do not receive treatment, glaucomatous optic neuropathy can occur rapidly.[67] Untreated fellow phakic eyes are at increased risk for developing acute angle closure.[68,69] Following resolution of an AACC, there is evidence that retinal nerve fiber layer thickness significantly decreases within 16 weeks after the attack, probably consisting of both resolution of axonal swelling and frank axonal atrophy.[67] In one study, 4 to 10 years following an AACC attack, 18% of eyes were blind (10% from glaucoma), 48% of eyes developed glaucomatous optic neuropathy, and 58% of eyes had vision worse than 20/40.[70] Thus, visual morbidity from AACC is significant. The natural history in untreated patients with AACC and PACG is to develop progressive vision loss that may result in bilateral blindness.

CARE PROCESS

PATIENT OUTCOME CRITERIA

- Preservation of visual function
- Maintenance of quality of life

DIAGNOSIS

Patients may or may not have symptoms of angle closure. Primary angle-closure suspect is a diagnosis based on the presence of ITC on gonioscopy. Primary angle closure is based on a combination of the presence of ITC on gonioscopy and either elevated IOP and/or PAS.[6]

Patients may be asymptomatic presenting for routine eye care, or they may present with sudden onset of symptoms and signs typical of AACC (such as pain, redness, congestion, decreased vision, corneal edema, very high IOP). The initial history and ophthalmic examination includes the components of the comprehensive adult medical eye evaluation,[71] with special attention to those factors that specifically bear upon primary and secondary causes of angle closure (see below).

History

The patient should be asked about symptoms that suggest intermittent angle-closure attacks (e.g., blurred vision, halos around lights, eye pain, headache, eye redness).[A:III] Review of the patient's family history may identify a relative with acute angle-closure glaucoma.[44,45,72] Specific questioning includes asking about the use of topical or systemic medication (e.g., sulfonamides,[73] topiramate[74] [e.g., Topamax; Ortho-McNeil Neurologics, Titusville, NJ], phenothiazines,[75] and anticholinergics) that may induce angle narrowing.

Ophthalmic Examination

Components of the physical evaluation that are particularly relevant for the diagnosis and management of angle closure and AACC follow.

Refractive status[A:III]

Hyperopic eyes, especially in older patients, have narrower anterior chamber angles[50] and are at increased risk of PAC.[51] Assessment of actual refractive status by retinoscopy or manifest refraction in the AACC eye may be postponed until a subsequent visit. However, approximating the refractive status is appropriate by measuring the eyeglass power to determine the possibility of hyperopia or refracting the fellow eye.

Pupil [A:III]

- Size and shape (may be asymmetric or oval in involved eye during or following an acute attack)
- Reactivity (may be poorly reactive or mid-dilated during an acute attack and tonic after an attack)
- Relative afferent pupillary defect (may be present in chronic angle closure or asymmetric optic nerve damage)

Slit-lamp biomicroscopy[A:III]

- Conjunctival hyperemia (in acute cases)
- Central and peripheral anterior chamber depth narrowing
- Anterior chamber inflammation suggestive of a recent or current attack
- Corneal swelling with or without microcystic edema (in acute cases)
- Iris abnormalities, including diffuse or focal atrophy, posterior synechiae, abnormal pupillary function, irregular pupil shape, and a mid-dilated pupil (suggestive of a recent or current attack)
- Lens changes including cataract and glaukomflecken (patchy, localized, anterior subcapsular lens opacities)
- Corneal endothelial cell loss[76-79]

Determination of IOP[A:III]

Intraocular pressure is measured in each eye, preferably using a contact applanation method (typically Goldmann tonometry) before gonioscopy. Measuring central corneal thickness should be postponed until resolution of an acute attack.[80] [A:III]

Gonioscopy[A:III]

Gonioscopy of both eyes should be performed on all patients in whom angle closure is suspected to evaluate the angle anatomy, appositional closure, and presence of PAS.[81] Compression (indentation) gonioscopy with a four-mirror or similar lens is particularly helpful to determine if visible appositional closure is actually permanent, synechial closure and, if so, for the extent of such PAS. Gonioscopy should be performed in a dark room with a bright, short (approximately 1 mm in length) beam that does not pass through the pupil to avoid inducing pupillary constriction, which can widen the angle.[82]

Gonioscopic visualization of the angle may be impaired secondary to corneal edema in the setting of acute angle-closure glaucoma. Topical glycerin may be used to clear the cornea to obtain a better view. (See www.gonioscopy.org and Selected Reference Texts section for discussion of the techniques of gonioscopy.)

Anterior segment imaging

Anterior segment imaging can be considered. There is good evidence demonstrating general agreement between findings on gonioscopy and anterior segment imaging, including ultrasound biomicroscopy (UBM) and anterior segment optical coherence tomography.[83-88] These technologies may prove useful in evaluating for secondary causes of angle closure (see Differential Diagnosis section) and to elucidate plateau iris (see Figures 1 to 7).

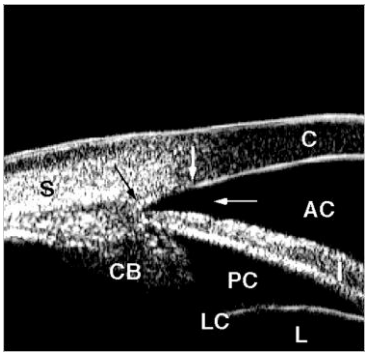

FIGURE 1. Normal angle anatomy by ultrasound biomicroscopy

AC = anterior chamber, C = cornea, CB = ciliary body with ciliary processes at tip, I = iris, L = lens, LC = lens capsule, PC = posterior chamber, black arrow = scleral spur, thick white arrow = Schwalbe's line, thin white arrow = angle recess or approach

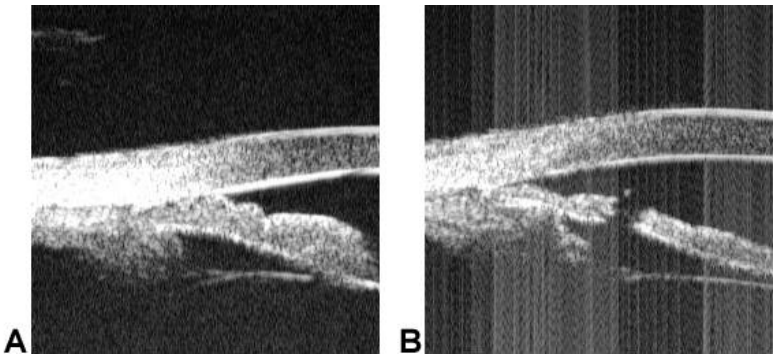

FIGURE 2. Angle closure before (A) and after (B) laser iridotomy with opening of the angle recess seen in image (B).

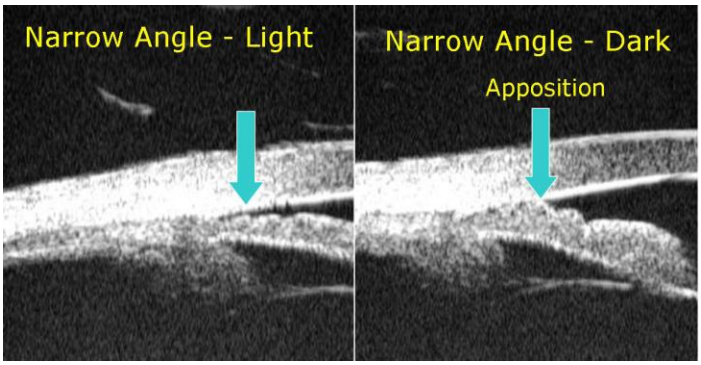

FIGURE 3. Comparative ultrasound biomicroscopy images of a narrow, occludable angle seen to be barely open in the light (left) and appositionally closed in the dark (right), denoted by arrow.

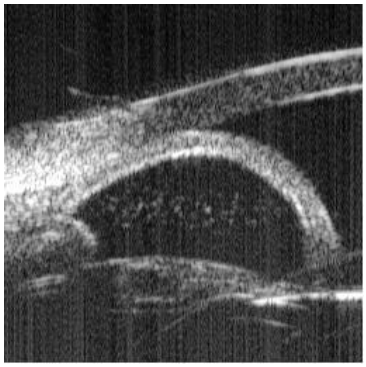

FIGURE 4. Iris bombé and acute angle closure as seen on ultrasound biomicroscopy.

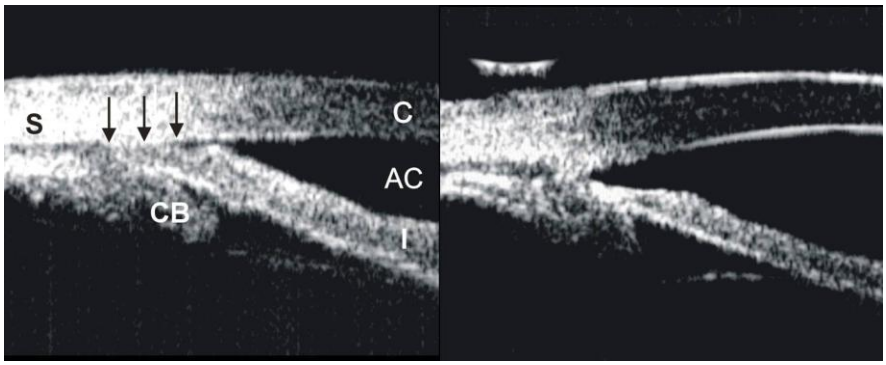

FIGURE 5. Plateau iris syndrome before (left) and after (right) laser peripheral iridoplasty, with opening of the angle recess and iris-trabecular meshwork apposition (black arrows) after iridoplasty, which results from the thermal laser energy absorbed by the iris tissue leading to atrophy and thinning of the iris periphery, seen in the photo on the right. This thinning leads to elimination of angle closure and intraocular pressure elevation with dilation seen after iridoplasty.
AC = anterior chamber, C = cornea, CB = ciliary body with ciliary processes, S = sclera.

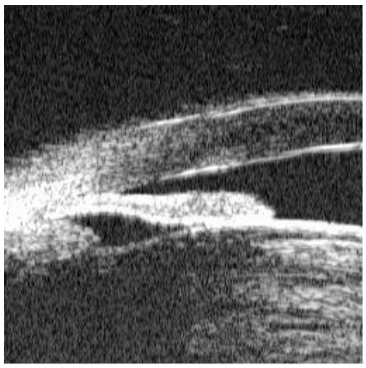

FIGURE 6. Angle closure due to a large, intumescent lens seen on ultrasound biomicroscopy.

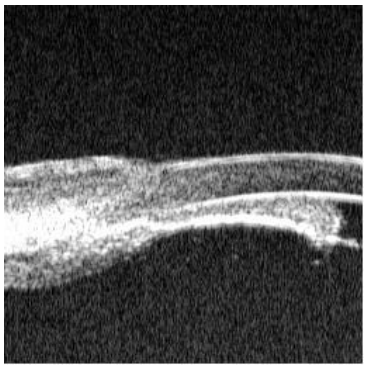

FIGURE 7. Angle closure with complete flattening of the iris against the corneal endothelium and loss of the anterior chamber due to aqueous misdirection/ malignant glaucoma seen on ultrasound biomicroscopy.

All images courtesy of Pat-Michael Palmiero, MD. Adapted with permission from the American Academy of Ophthalmology Compass Glaucoma Panel. Compass: Clinically Relevant Review for Practicing Ophthalmologists. Glaucoma review outline. Diagnostic tests: ultrasound biomicroscopy. Available to Academy members only at: http://one.aao.org/CE/EducationalContent/CompassExam.aspx.

Provocative testing

Careful gonioscopic examination and synthesis of the clinical findings have replaced the use of provocative tests to make therapeutic decisions for patients at risk for PAC.

Other components of the initial evaluation

Although a dilated examination may not be advisable in patients with ITC, an attempt should be made to evaluate the fundus and optic nerve head using the direct ophthalmoscope or indirect ophthalmoscopy at the slit-lamp biomicroscope with a 90-diopter lens.[A:III] For patients with PAC or PACS who are not having an acute attack, pupil dilation is not recommended until an iridotomy has been performed, since dilation can precipitate acute attacks.[46] [A:III] Evaluation and documentation of the optic nerve head, retinal nerve fiber layer, and visual fields may be postponed until an acute attack is adequately treated. (See Primary Open-Angle Glaucoma PPP for a detailed description of the evaluation.[9])

Differential diagnosis

Because PACS and PAC tend to be bilateral, the observation of a wide open angle in the fellow eye suggests a diagnosis other than PAC. Other entities that cause secondary anterior chamber angle closure and that may be unilateral or bilateral include the following:

- Neovascular glaucoma
- Inflammation resulting in both PAS and posterior synechiae, which can result in a secluded pupil with iris bombé
- Iridocorneal endothelial syndrome
- Uveal effusion associated with systemic medications (e.g., topiramate, sulfonamides, phenothiazines, anticholinergics), retinal vascular occlusion, inflammation, tight scleral buckle, or panretinal photocoagulation
- Suprachoroidal effusions
- Malignant glaucoma
- Lens-induced angle closure (e.g., from a phacomorphic or subluxed lens)
- Developmental disorders (e.g., nanophthalmos, retinopathy of prematurity, persistent hyperplastic primary vitreous)
- Iris or ciliary body mass lesions or cysts
- Other secondary causes of pupil block (e.g., aphakia without an iridectomy, phakic intraocular lens [IOL], anterior chamber IOL, silicone oil)
- History of blunt or penetrating trauma
- Axenfeld-Rieger syndrome
- Epithelial downgrowth

MANAGEMENT

Goals

The goals of managing a patient with PAC are as follows:

- Reverse or prevent angle-closure process
- Control IOP
- Prevent damage to the optic nerve

Primary Angle-Closure Suspect

No completed clinical trials have documented the benefit of iridotomy for PACS, yet the relative safety of this procedure has allowed its wider use in the hope of preventing AACC and PACG. Present cross-sectional data indicates that the majority of those with PACS will not develop either PAC or PACG. In patients with ITC, and normal IOP without PAS, iridotomy may be considered to reduce the risk of developing angle closure.[A:III] Alternatively, patients with ITC may be followed for development of IOP elevation, evidence of progressive narrowing, or synechial angle

closure,[A:III] since iridotomy can be associated with bothersome postoperative glare/diplopia. Hastening of cataract and posterior synechiae are occasional consequences.

Other factors that may influence the decision to perform prophylactic laser iridotomy in PACS include the following:

- Medication is required that may provoke pupillary block
- Symptoms are present that suggest prior acute angle closure
- The patient's health status or occupation/avocation makes it difficult to access immediate ophthalmic care (e.g., the patient resides in a nursing facility, travels frequently to developing parts of the world, works on a merchant vessel), or in poorly compliant patients

Patients with PACS who have not had an iridotomy should be warned that they are at risk for AACC and that certain medicines (e.g., over-the-counter decongestants, motion-sickness medication, anticholinergic agents) could cause pupil dilation and induce AACC.[46] [A:III] They should also be informed about the symptoms of AACC and instructed to notify their ophthalmologist immediately if symptoms occur.[89] [A:III]

Plateau Iris Configuration and Syndrome

The success of prophylactic peripheral laser iridoplasty after laser iridotomy to prevent PACG and AACC in eyes with plateau iris has been reported in a case series.[90] However, a recent Cochrane review found no randomized controlled trials to provide evidence to support the use of this procedure in these eyes.[91] Given the lack of convincing evidence in the literature for prophylactic use of iridoplasty in these patients, and since iridoplasty can be painful for the patient and may cause inflammation, the decision of whether to observe or treat these eyes is left to the judgment of the treating ophthalmologist. Eyes with recurrent high IOP after iridotomy when the pupil is dilated (plateau iris syndrome) should undergo further therapy, including iridoplasty, chronic miotic therapy, or other surgical procedures.[A:III]

Primary Angle Closure and Primary Angle-Closure Glaucoma

Patients with PAC may have elevated IOP as a result of a chronic compromise of aqueous outflow due to appositional or synechial angle closure, or damage to the trabecular meshwork from previous intermittent AACC. Iridotomy is indicated for eyes with PAC or PACG.[6,92] [A:III]

Complications of laser iridotomy include increased IOP; laser burn to the cornea, lens, or retina; late onset corneal edema; development of posterior synechiae; hyphema; iritis; and the development of a ghost image in the patient’s vision.

Surgery and Postoperative Care

The ophthalmologist who performs the laser iridotomy or incisional iridectomy has the following responsibilities:[93,94] [A:III]

- Obtain informed consent from the patient or the patient’s surrogate decision maker after discussing the risks, benefits, and expected outcomes of surgery[A:III]
- Ensure that preoperative evaluation confirms the need for surgery[A:III]
- Perform at least one IOP check within 30 minutes to 2 hours of surgery[95-97] [A:III]
- Prescribe topical corticosteroids in the postoperative period[A:III]
- Ensure that the patient receives adequate postoperative care[A:III]

Preoperative miotics facilitate laser iridotomy or iridectomy. Medications should be used perioperatively to avert sudden IOP elevation, particularly for patients who have severe disease.[95] [A:III]

Follow-up evaluations after surgery should include the following elements:[A:III]

- Evaluation of the patency of iridotomy
- Measurement of IOP
- Gonioscopy with compression/indentation to assess the extent of PAS, if it was not performed immediately after iridotomy

- Pupil dilation to decrease the risk of posterior synechiae formation
- Fundus examination as clinically indicated

Following iridotomy for PAC, persistent or progressive elevations of IOP and complications may occur for several reasons:

- Trabecular damage or formation of synechiae may have occurred during iridocorneal apposition
- If the iridotomy becomes occluded, pupil block may recur. Reoperation is indicated.
- Factors other than pupil block may lead to angle closure and may have gone unrecognized until after the iridotomy. These include plateau iris syndrome and secondary causes of pupil block (see Differential Diagnosis section).
- Angle closure may have been superimposed on pre-existing open-angle glaucoma or on another cause of IOP elevation, such as exfoliation syndrome[98-101]

Additional treatment of PAC or PACG after the component of pupil block has been alleviated by iridotomy is directed at lowering IOP to prevent or retard pressure-induced optic nerve damage and is similar to the treatment of primary open-angle glaucoma.[9] Reopening the angle by laser iridoplasty or surgical lysis of synechiae (goniosynechialysis) may improve aqueous outflow, especially when it is performed within 6 to 12 months of an acute attack.[102-105]

Additional management for elevated IOP that threatens the optic nerve is the same as for primary open-angle glaucoma (see Primary Open-Angle Glaucoma PPP[9]) and includes chronic topical ocular hypotensive agents, laser trabeculoplasty if, in the judgment of the treating ophthalmologist, sufficient open trabecular meshwork exists after laser iridotomy to expect a reasonable IOP reduction, and incisional surgery (trabeculectomy or tube shunt). In addition, a growing body of evidence indicates that cataract extraction alone may lead to substantial IOP lowering in some PACG patients and can be considered as an option for treatment.[106-111]

Acute Angle-Closure Crisis

Acute attack management

The initial treatment of AACC is aimed at lowering IOP to relieve the acute symptoms and potentially harmful high IOP. Several methods have been studied, including medical therapy alone, laser peripheral iridotomy (if the iris can be visualized and the procedure can be performed), peripheral laser iridoplasty, and paracentesis.[112-114] However, most patients are treated acutely with medications, and the iridotomy is performed as soon as feasible.

Iridotomy (or iridectomy) allows aqueous to bypass the pupil block and eliminates the pressure gradient between the posterior and anterior chambers.

In AACC, medical therapy is usually initiated first to lower the IOP to reduce pain and clear corneal edema.[A:III] Iridotomy should then be performed as soon as possible.[A:III] Medical therapy includes some or all of the following, based on the patient's overall physical and medical status:[115]

- Topical beta-adrenergic antagonists[116]
- Topical alpha$_2$-adrenergic agonists[117]
- Topical or systemic carbonic anhydrase inhibitors[116]
- Topical miotics[116,118]
- Systemic hyperosmotic agents

Agents that suppress aqueous humor formation (beta-adrenergic antagonists, carbonic anhydrase inhibitors) may be ineffective, because they may have decreased ability to reduce aqueous formation if the ciliary body is ischemic. As the configuration of the iris becomes less bowed with a small pupil, treatment with miotics may open the angle; however, miotic therapy is frequently ineffective when the IOP is markedly elevated because of pressure-induced ischemia of the pupillary sphincter. Often, systemic hyperosmotic agents need to be used to achieve a rapid decrease in IOP in the setting of AACC. Corneal indentation performed with a four-mirror gonioscopic lens, cotton-tipped applicator, or tip of a muscle hook may help break pupil block.[119]

Laser iridotomy is the preferred surgical treatment, because it has a favorable risk-benefit ratio.[10,120,121] [A:II] Iridotomy relieves pupil block and can prevent or retard the formation of PAS.

Timely treatment may prevent damage to the optic nerve, trabecular meshwork, iris, lens, and cornea. If an iridotomy cannot be performed due to corneal edema, the cornea may sometimes be cleared with topical hyperosmotic agents or anterior chamber paracentesis. Once the attack is broken, it is usually possible to perform a definitive laser iridotomy immediately or soon afterwards.[122-129]

When laser iridotomy is not possible or if the AACC cannot be medically broken, laser peripheral iridoplasty (even with a cloudy cornea),[128] paracentesis,[114] and incisional iridectomy remain effective alternatives.[A:III] When incisional iridectomy is required and extensive synechial closure is recognized or suspected, simultaneous primary filtering surgery may be considered. Concurrent glaucomatous cupping of the optic disc often indicates a prolonged chronic course before the onset of acute symptoms, and filtering surgery can be considered, especially if the presence of considerable PAS is confirmed on gonioscopy. Filtering surgery in eyes with unbroken acute angle closure are at high risk of developing a flat anterior chamber and aqueous misdirection postoperatively.[130]

Lens extraction for angle closure

Numerous studies document that lens extraction significantly widens the anterior chamber angle in eyes with narrow, occludable angles and in angle-closure glaucoma.[106-110] In prospective and retrospective studies, cataract surgery also has been shown to lower postoperative medication requirements and decrease complications compared with surgical iridectomy or trabeculectomy in angle-closure glaucoma[131-140] and AACC.[141] Although there is evidence that lens extraction with or without goniosynechialysis[102,142-144] can substantially lower IOP, it has been documented that cataract extraction alone does not result in as low an IOP as trabeculectomy and cataract surgery.[145] The risks and benefits of different procedures in eyes with angle closure should be considered when determining the most appropriate surgery.

A randomized trial of cataract surgery soon after AACC is broken compared with routine follow-up after iridotomy alone found that individuals with high IOP (>55 mmHg) were much less likely to require IOP-lowering therapy if they had early cataract extraction.[141] In this study, patients had phacoemulsification within 5.7 ± 3.3 (mean ± standard deviation) days and iridotomy within 4.3 ± 2.7 (mean ± standard deviation) days of the AACC. Early cataract surgery can be considered after AACC in patients with a high risk of developing uncontrollable IOP. However, cataract surgery in AACC eyes may have greater risk of operative complications due to the small dimensions of the chamber and the tendency for choroidal expansion.

Fellow-eye management

The fellow eye of a patient with AACC should be evaluated, because it is at high risk for a similar event. The fellow eye should receive a prophylactic laser iridotomy promptly if the chamber angle is anatomically narrow,[A:II] since approximately half of fellow eyes of acute angle-closure patients can develop acute attacks within 5 years.[68,69,89,92,146-148] These attacks can occur within days of presentation (in fact, 10% of cases present with bilateral AACC) and, therefore, consideration of immediate laser peripheral iridotomy in the fellow eye is warranted. Laser iridotomy in the fellow eye should be performed at the initial visit if the eye in AACC cannot have a successful iridotomy in the acute setting because of poor visualization of the iris due to corneal edema. Chronic miotic therapy is not an appropriate alternative, either for prophylaxis of the fellow eye or for treatment of established angle closure, and it is not a substitute for iridotomy. About 40% of fellow eyes treated with miotics can develop an acute attack within 5 years, and many eyes with angle closure can develop progressive formation of synechial angle closure with miotic use.[69,148] Prophylactic laser peripheral iridotomies are effective in preventing acute angle closure in the fellow eye.[147]

FOLLOW-UP EVALUATION

The recommendations for follow-up evaluations after iridotomy for angle closure apply to ongoing management and not to visits for other purposes. Following iridotomy, patients may have an open anterior chamber angle or an anterior chamber angle with a combination of open sectors with areas occluded by PAS. Following iridotomy, patients with a residual open angle or a combination of open angle and some PAS with or without glaucomatous optic neuropathy should be followed at

least annually, with special attention to repeat gonioscopy to determine interval changes such as increased extent of PAS or development of secondary angle closure from cataract progression and increased lens thickness.[A:III] Subsequent follow-up intervals depend on the clinical findings and judgment of the treating ophthalmologist.

PROVIDER AND SETTING

The performance of certain diagnostic procedures (e.g., tonometry, perimetry, pachymetry, optic disc imaging, and photography) may be delegated to appropriately trained and supervised personnel. However, the interpretation of results and medical and surgical management of disease require the medical training, clinical judgment, and experience of the ophthalmologist. Most diagnostic and therapeutic procedures can be undertaken safely on an outpatient basis. Hospitalization may be indicated for intensive treatment of an AACC so that patients can be monitored closely after surgical procedures associated with a high risk of serious short-term postoperative complications. It may also be indicated for patients in whom surgical complications have occurred or for patients who have special medical or social needs.

COUNSELING/REFERRAL

If the diagnosis or management of PAC, PACS, AACC, or PACG is in question or is refractory to treatment, consultation with or referral to an ophthalmologist with special training or experience in managing these conditions may be desirable. Patients with significant visual impairment or blindness may benefit from appropriate vision rehabilitation and social services.[149] More information on vision rehabilitation, including materials for patients, is available at www.aao.org/smartsight.

APPENDIX 1. QUALITY OF OPHTHALMIC CARE CORE CRITERIA

Providing quality care
is the physician's foremost ethical obligation, and is
the basis of public trust in physicians.
AMA Board of Trustees, 1986

Quality ophthalmic care is provided in a manner and with the skill that is consistent with the best interests of the patient. The discussion that follows characterizes the core elements of such care.

The ophthalmologist is first and foremost a physician. As such, the ophthalmologist demonstrates compassion and concern for the individual, and utilizes the science and art of medicine to help alleviate patient fear and suffering. The ophthalmologist strives to develop and maintain clinical skills at the highest feasible level, consistent with the needs of patients, through training and continuing education. The ophthalmologist evaluates those skills and medical knowledge in relation to the needs of the patient and responds accordingly. The ophthalmologist also ensures that needy patients receive necessary care directly or through referral to appropriate persons and facilities that will provide such care, and he or she supports activities that promote health and prevent disease and disability.

The ophthalmologist recognizes that disease places patients in a disadvantaged, dependent state. The ophthalmologist respects the dignity and integrity of his or her patients, and does not exploit their vulnerability.

Quality ophthalmic care has the following optimal attributes, among others.

- The essence of quality care is a meaningful partnership relationship between patient and physician. The ophthalmologist strives to communicate effectively with his or her patients, listening carefully to their needs and concerns. In turn, the ophthalmologist educates his or her patients about the nature and prognosis of their condition and about proper and appropriate therapeutic modalities. This is to ensure their meaningful participation (appropriate to their unique physical, intellectual and emotional state) in decisions affecting their management and care, to improve their motivation and compliance with the agreed plan of treatment, and to help alleviate their fears and concerns.
- The ophthalmologist uses his or her best judgment in choosing and timing appropriate diagnostic and therapeutic modalities as well as the frequency of evaluation and follow-up, with due regard to the urgency and nature of the patient's condition and unique needs and desires.
- The ophthalmologist carries out only those procedures for which he or she is adequately trained, experienced and competent, or, when necessary, is assisted by someone who is, depending on the urgency of the problem and availability and accessibility of alternative providers.
- Patients are assured access to, and continuity of, needed and appropriate ophthalmic care, which can be described as follows.
  - The ophthalmologist treats patients with due regard to timeliness, appropriateness, and his or her own ability to provide such care.
  - The operating ophthalmologist makes adequate provision for appropriate pre- and postoperative patient care.
  - When the ophthalmologist is unavailable for his or her patient, he or she provides appropriate alternate ophthalmic care, with adequate mechanisms for informing patients of the existence of such care and procedures for obtaining it.
  - The ophthalmologist refers patients to other ophthalmologists and eye care providers based on the timeliness and appropriateness of such referral, the patient's needs, the competence and qualifications of the person to whom the referral is made, and access and availability.
  - The ophthalmologist seeks appropriate consultation with due regard to the nature of the ocular or other medical or surgical problem. Consultants are suggested for their skill, competence, and accessibility.

They receive as complete and accurate an accounting of the problem as necessary to provide efficient and effective advice or intervention, and in turn respond in an adequate and timely manner.

- The ophthalmologist maintains complete and accurate medical records.
- On appropriate request, the ophthalmologist provides a full and accurate rendering of the patient's records in his or her possession.
- The ophthalmologist reviews the results of consultations and laboratory tests in a timely and effective manner and takes appropriate actions.
- The ophthalmologist and those who assist in providing care identify themselves and their profession.
- For patients whose conditions fail to respond to treatment and for whom further treatment is unavailable, the ophthalmologist provides proper professional support, counseling, rehabilitative and social services, and referral as appropriate and accessible.

- Prior to therapeutic or invasive diagnostic procedures, the ophthalmologist becomes appropriately conversant with the patient's condition by collecting pertinent historical information and performing relevant preoperative examinations. Additionally, he or she enables the patient to reach a fully informed decision by providing an accurate and truthful explanation of the diagnosis; the nature, purpose, risks, benefits, and probability of success of the proposed treatment and of alternative treatment; and the risks and benefits of no treatment.
- The ophthalmologist adopts new technology (e.g., drugs, devices, surgical techniques) in judicious fashion, appropriate to the cost and potential benefit relative to existing alternatives and to its demonstrated safety and efficacy.
- The ophthalmologist enhances the quality of care he or she provides by periodically reviewing and assessing his or her personal performance in relation to established standards, and by revising or altering his or her practices and techniques appropriately.
- The ophthalmologist improves ophthalmic care by communicating to colleagues, through appropriate professional channels, knowledge gained through clinical research and practice. This includes alerting colleagues of instances of unusual or unexpected rates of complications and problems related to new drugs, devices or procedures.
- The ophthalmologist provides care in suitably staffed and equipped facilities adequate to deal with potential ocular and systemic complications requiring immediate attention.
- The ophthalmologist also provides ophthalmic care in a manner that is cost effective without unacceptably compromising accepted standards of quality.

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APPENDIX 2. MAJOR RECOMMENDATIONS FOR CARE

DIAGNOSIS

History

The patient should be asked about symptoms that suggest intermittent angle-closure attacks (e.g., blurred vision, halos around lights, eye pain, headache, eye redness).[A:III] Review of the patient's family history may identify a relative with acute angle-closure glaucoma.[44,45,72] Specific questioning includes asking about the use of topical or systemic medication (e.g., sulfonamides,[73] topiramate[74] [e.g., Topamax; Ortho-McNeil Neurologics, Titusville, NJ], phenothiazines,[75] and anticholinergics) that may induce angle narrowing.

Ophthalmic Examination

Refractive status[A:III]

Hyperopic eyes, especially in older patients, have narrower anterior chamber angles[50] and are at increased risk of primary angle closure (PAC).[51] Assessment of actual refractive status by retinoscopy or manifest refraction in the acute angle closure crisis (AACC) eye may be postponed until a subsequent visit. However, approximating the refractive status is appropriate by measuring the eyeglass power to determine the possibility of hyperopia or refracting the fellow eye.

Pupil [A:III]

- Size and shape (may be asymmetric or oval in involved eye during or following an acute attack)
- Reactivity (may be poorly reactive or mid-dilated during an acute attack and tonic after an attack)
- Relative afferent pupillary defect (may be present in chronic angle closure or asymmetric optic nerve damage)

Slit-lamp biomicroscopy[A:III]

- Conjunctival hyperemia (in acute cases)
- Central and peripheral anterior chamber depth narrowing
- Anterior chamber inflammation suggestive of a recent or current attack
- Corneal swelling with or without microcystic edema (in acute cases)
- Iris abnormalities, including diffuse or focal atrophy, posterior synechiae, abnormal pupillary function, irregular pupil shape, and a mid-dilated pupil (suggestive of a recent or current attack)
- Lens changes including cataract and glaukomflecken (patchy, localized, anterior subcapsular lens opacities)
- Corneal endothelial cell loss[76-79]

Determination of Intraocular Pressure[A:III]

Intraocular pressure (IOP) is measured in each eye, preferably using a contact applanation method (typically Goldmann tonometry) before gonioscopy. Measuring central corneal thickness should be postponed until resolution of an acute attack.[80] [A:III]

Gonioscopy[A:III]

Gonioscopy of both eyes should be performed on all patients in whom angle closure is suspected to evaluate the angle anatomy, appositional closure, and presence of peripheral anterior synechiae (PAS).[81] Compression (indentation) gonioscopy with a four-mirror or similar lens is particularly helpful to determine if visible appositional closure is actually permanent, synechial closure and, if so, for the extent of such PAS.

Other components of the initial evaluation

Although a dilated examination may not be advisable in patients with iridotrabecular contact (ITC), an attempt should be made to evaluate the fundus and optic nerve head using the direct ophthalmoscope or indirect ophthalmoscopy at the slit-lamp biomicroscope with a 90-diopter lens.[A:III] For patients with PAC or PAC suspect who are not having an acute attack, pupil dilation is not recommended until an iridotomy has been performed, since dilation can precipitate acute attacks.[46 [A:III]]

Primary Angle-Closure Suspect

In patients with ITC, and normal IOP without PAS, iridotomy may be considered to reduce the risk of developing angle closure.[A:III] Alternatively, patients with ITC may be followed for development of IOP elevation, evidence of progressive narrowing, or synechial angle closure,[A:III] since iridotomy can be associated with bothersome postoperative glare/diplopia.

Patients with PAC suspect who have not had an iridotomy should be warned that they are at risk for AACC and that certain medicines (e.g., over-the-counter decongestants, motion-sickness medication, anticholinergic agents) could cause pupil dilation and induce AACC.[46 [A:III]] They should also be informed about the symptoms of AACC and instructed to notify their ophthalmologist immediately if symptoms occur.[89 [A:III]]

Primary Angle Closure and Primary Angle-Closure Glaucoma

Iridotomy is indicated for eyes with PAC or primary angle-closure glaucoma (PACG).[6,92 [A:III]]

Acute Angle-Closure Crisis

Acute attack management

In AACC, medical therapy is usually initiated first to lower the IOP to reduce pain and clear corneal edema.[A:III] Iridotomy should then be performed as soon as possible.[A:III] Laser iridotomy is the preferred surgical treatment, because it has a favorable risk-benefit ratio.[10,120,121 [A:II]]

When laser iridotomy is not possible or if the AACC cannot be medically broken, laser peripheral iridoplasty (even with a cloudy cornea),[128] paracentesis,[114] and incisional iridectomy remain effective alternatives.[A:III]

Fellow-eye management

The fellow eye of a patient with AACC should be evaluated, because it is at high risk for a similar event. The fellow eye should receive a prophylactic laser iridotomy promptly if the chamber angle is anatomically narrow,[A:II] since approximately half of fellow eyes of acute angle-closure patients can develop acute attacks within 5 years.[68,69,89,92,146-148]

Surgery and Postoperative Care

The ophthalmologist who performs the laser iridotomy or incisional iridectomy has the following responsibilities: [93,94 [A:III]]

- Obtain informed consent from the patient or the patient's surrogate decision maker after discussing the risks, benefits, and expected outcomes of surgery[A:III]
- Ensure that preoperative evaluation confirms the need for surgery[A:III]
- Perform at least one IOP check within 30 minutes to 2 hours of surgery[95-97 [A:III]]
- Prescribe topical corticosteroids in the postoperative period[A:III]
- Ensure that the patient receives adequate postoperative care[A:III]

Preoperative miotics facilitate laser iridotomy or iridectomy. Medications should be used perioperatively to avert sudden IOP elevation, particularly for patients who have severe disease.[95 [A:III]]

Follow-up evaluations after surgery should include the following elements:[A:III]

- Evaluation of the patency of iridotomy
- Measurement of IOP
- Gonioscopy with compression/indentation to assess the extent of PAS, if it was not performed immediately after iridotomy
- Pupil dilation to decrease the risk of posterior synechiae formation
- Fundus examination as clinically indicated

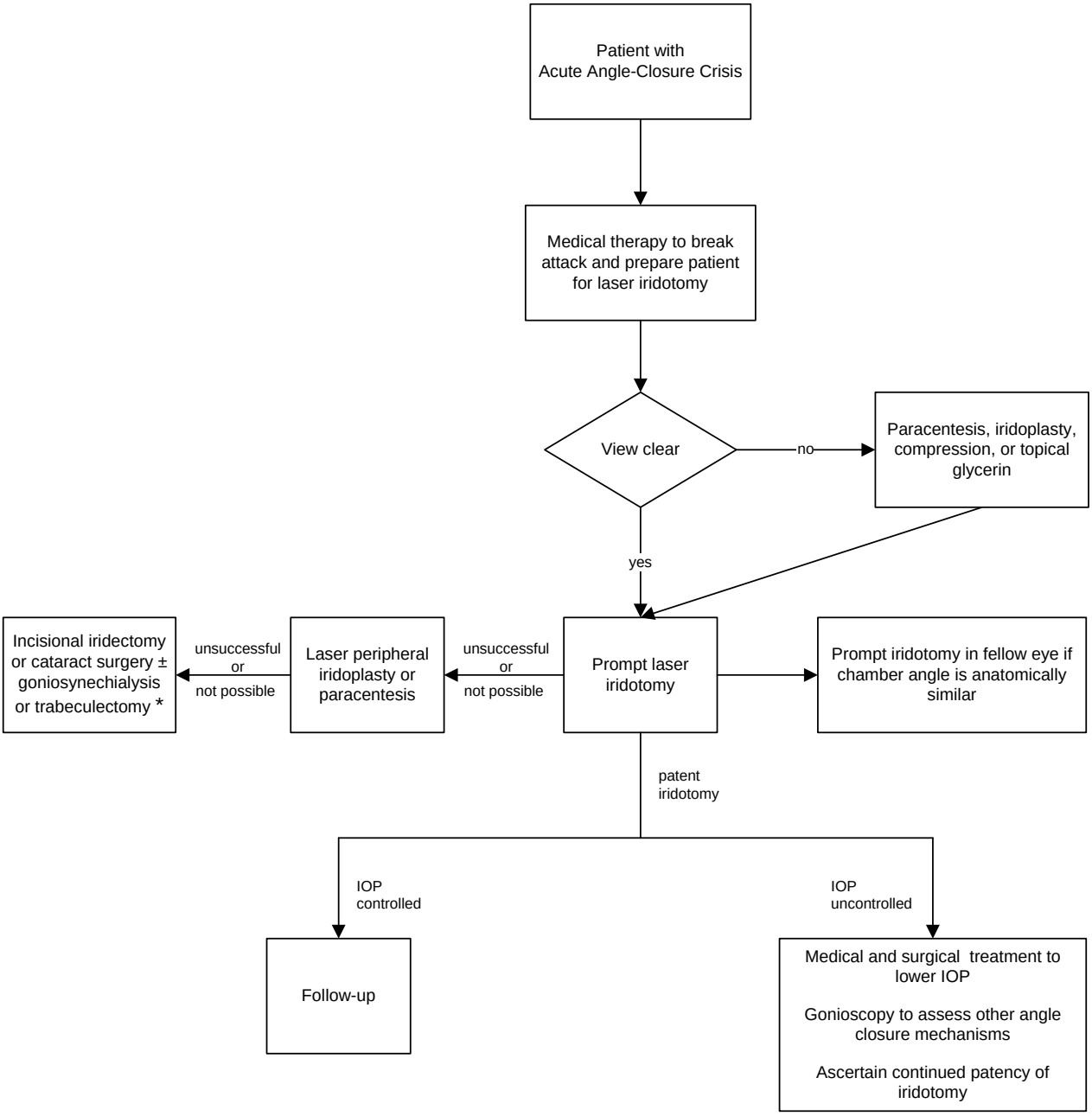
FOLLOW-UP EVALUATION

Following iridotomy, patients with a residual open angle or a combination of open angle and some PAS with or without glaucomatous optic neuropathy should be followed at least annually, with special attention to repeat gonioscopy to determine interval changes such as increased extent of PAS or development of secondary angle closure from cataract progression and increased lens thickness.[A:III] Subsequent follow-up intervals depend on the clinical findings and judgment of the treating ophthalmologist.

COUNSELING/REFERRAL

If the diagnosis or management of PAC, PAC suspect, AACC, or PACG is in question or is refractory to treatment, consultation with or referral to an ophthalmologist with special training or experience in managing these conditions may be desirable. Patients with significant visual impairment or blindness may benefit from appropriate vision rehabilitation and social services.[149] More information on vision rehabilitation, including materials for patients, is available at www.aao.org/smartsight.

APPENDIX 3. ALGORITHM FOR THE MANAGEMENT OF PATIENTS WITH ACUTE ANGLE-CLOSURE CRISIS



* Indicated for extensive synechial closure or optic nerve damage.

APPENDIX 4. INTERNATIONAL STATISTICAL CLASSIFICATION OF DISEASES AND RELATED HEALTH PROBLEMS (ICD) CODES

The Primary Angle Closure Preferred Practice Pattern covers the entity of primary angle-closure glaucoma (ICD-9 #365.20) and related entities with the following ICD-9 classifications:

- Acute angle-closure glaucoma (365.22)
- Intermittent angle closure (365.21)
- Chronic angle-closure glaucoma (365.23)
- Residual stage of angle-closure glaucoma (365.24)
- Anatomical narrow angle (365.02)

SUGGESTED REFERENCE TEXTS

- Allingham RR, Damji KF, Freedman S, Moroi SE, Shafranov G, Shields MB, eds. Shields' Textbook of Glaucoma. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2005.
- Alward WLM. www.gonioscopy.org. Accessed September 16, 2010.
- Alward WLM, Longmuir RA. Color Atlas of Gonioscopy. 2nd ed. San Francisco, CA: American Academy of Ophthalmology; 2008.
- Anderson DR, Patella VM. Automated Static Perimetry. 2nd ed. St. Louis, MO: CV Mosby Co.; 1999.
- Epstein DL, Allingham RR, Shuman JS, eds. Chandler and Grant's Glaucoma. 4th ed. Baltimore, MD: Lippincott Williams and Wilkins; 1997.
- Ritch R, Shields MB, Krupin T, eds. The Glaucomas. 2nd ed. St. Louis, MO: CV Mosby Co.; 1996.
- Stamper RL, Lieberman MF, Drake MV. Becker-Shaffer's Diagnosis and Therapy of the Glaucomas. 8th ed. Philadelphia, PA: Mosby Elsevier; 2009.
- Tasman W, Jaeger EA, eds. Duane's Ophthalmology. 15th edition. Philadelphia, PA: Lippincott Williams & Wilkins; 2009.
- Weinreb RN, Friedman DS, eds. Angle Closure and Angle-Closure Glaucoma. World Glaucoma Association Consensus Series - 3. The Netherlands: Kugler Publications; 2006.

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REFERENCES

1. Anderson DR, Jin JC, Wright MM. The physiologic characteristics of relative pupillary block. Am J Ophthalmol 1991;111:344-50.
2. Tiedeman JS. A physical analysis of the factors that determine the contour of the iris. Am J Ophthalmol 1991;111:338-43.
3. Jin JC, Anderson DR. The effect of iridotomy on iris contour. Am J Ophthalmol 1990;110:260-3.
4. Seah SK, Foster PJ, Chew PT, et al. Incidence of acute primary angle-closure glaucoma in Singapore. An island-wide survey. Arch Ophthalmol 1997;115:1436-40.
5. Friedman DS, Gazzard G, Foster P, et al. Ultrasonographic biomicroscopy, Scheimpflug photography, and novel provocative tests in contralateral eyes of Chinese patients initially seen with acute angle closure. Arch Ophthalmol 2003;121:633-42.
6. Weinreb RN, Friedman DS, eds. Angle Closure and Angle Closure Glaucoma. World Glaucoma Association Consensus Series-3. The Netherlands: Kugler Publications, 2006.
7. Thomas R, Parikh R, Muliyil J, Kumar RS. Five-year risk of progression of primary angle closure to primary angle closure glaucoma: a population-based study. Acta Ophthalmol Scand 2003;81:480-5.
8. Thomas R, George R, Parikh R, et al. Five year risk of progression of primary angle closure suspects to primary angle closure: a population based study. Br J Ophthalmol 2003;87:450-4.

9. American Academy of Ophthalmology Glaucoma Panel. Preferred Practice Pattern® Guidelines. Primary Open-Angle Glaucoma. San Francisco, CA: American Academy of Ophthalmology; 2010. Available at: www.aao.org/ppp.
10. Quigley HA. Long-term follow-up of laser iridotomy. Ophthalmology 1981;88:218-24.
11. He M, Friedman DS, Ge J, et al. Laser peripheral iridotomy in primary angle-closure suspects: biometric and gonioscopic outcomes: the Liwan Eye Study. Ophthalmology 2007;114:494-500.
12. He M, Friedman DS, Ge J, et al. Laser peripheral iridotomy in eyes with narrow drainage angles: ultrasound biomicroscopy outcomes. The Liwan Eye Study. Ophthalmology 2007;114:1513-9.
13. Van Rens GH, Arkell SM, Charlton W, Doesburg W. Primary angle-closure glaucoma among Alaskan Eskimos. Doc Ophthalmol 1988;70:265-76.
14. Arkell SM, Lightman DA, Sommer A, et al. The prevalence of glaucoma among Eskimos of northwest Alaska. Arch Ophthalmol 1987;105:482-5.
15. Bourne RR, Sorensen KE, Klauber A, et al. Glaucoma in East Greenlandic Inuit--a population survey in Ittoqqortoormiit (Scoresbysund). Acta Ophthalmol Scand 2001;79:462-7.
16. Congdon NG, Quigley HA, Hung PT, et al. Screening techniques for angle-closure glaucoma in rural Taiwan. Acta Ophthalmol Scand 1996;74:113-9.
17. He M, Foster PJ, Ge J, et al. Prevalence and clinical characteristics of glaucoma in adult Chinese: a population-based study in Liwan District, Guangzhou. Invest Ophthalmol Vis Sci 2006;47:2782-8.
18. Foster PJ, Baasanhu J, Alsbirk PH, et al. Glaucoma in Mongolia. A population-based survey in Hovsgol province, northern Mongolia. Arch Ophthalmol 1996;114:1235-41.
19. Xu L, Zhang L, Xia CR, et al. The prevalence and its effective factors of primary angle-closure glaucoma in defined populations of rural and urban in Beijing [in Chinese]. Zhonghua Yan Ke Za Zhi 2005;41:8-14.
20. Foster PJ, Oen FT, Machin D, et al. The prevalence of glaucoma in Chinese residents of Singapore: a cross-sectional population survey of the Tanjong Pagar district. Arch Ophthalmol 2000;118:1105-11.
21. Casson RJ, Newland HS, Muecke J, et al. Prevalence of glaucoma in rural Myanmar: the Meiktila Eye Study. Br J Ophthalmol 2007;91:710-4.
22. Salmon JF, Mermoud A, Ivey A, et al. The prevalence of primary angle closure glaucoma and open angle glaucoma in Mamre, western Cape, South Africa. Arch Ophthalmol 1993;111:1263-9.
23. Dandona L, Dandona R, Mandal P, et al. Angle-closure glaucoma in an urban population in southern India. The Andhra Pradesh eye disease study. Ophthalmology 2000;107:1710-6.
24. Bourne RR, Sukudom P, Foster PJ, et al. Prevalence of glaucoma in Thailand: a population based survey in Rom Klao District, Bangkok. Br J Ophthalmol 2003;87:1069-74.
25. Vijaya L, George R, Arvind H, et al. Prevalence of angle-closure disease in a rural southern Indian population. Arch Ophthalmol 2006;124:403-9.
26. Ramakrishnan R, Nirmalan PK, Krishnadas R, et al. Glaucoma in a rural population of southern India: the Aravind comprehensive eye survey. Ophthalmology 2003;110:1484-90.
27. Rahman MM, Rahman N, Foster PJ, et al. The prevalence of glaucoma in Bangladesh: a population based survey in Dhaka division. Br J Ophthalmol 2004;88:1493-7.
28. Shiose Y, Kitazawa Y, Tsukahara S, et al. Epidemiology of glaucoma in Japan--a nationwide glaucoma survey. Jpn J Ophthalmol 1991;35:133-55.
29. Yamamoto T, Iwase A, Araie M, et al. The Tajimi Study report 2: prevalence of primary angle closure and secondary glaucoma in a Japanese population. Ophthalmology 2005;112:1661-9.
30. Tielsch JM, Katz J, Singh K, et al. A population-based evaluation of glaucoma screening: the Baltimore Eye Survey. Am J Epidemiol 1991;134:1102-10.
31. Buhrmann RR, Quigley HA, Barron Y, et al. Prevalence of glaucoma in a rural East African population. Invest Ophthalmol Vis Sci 2000;41:40-8.
32. Rotchford AP, Kirwan JF, Muller MA, et al. Temba glaucoma study: a population-based cross-sectional survey in urban South Africa. Ophthalmology 2003;110:376-82.
33. Bonomi L, Marchini G, Marraffa M, et al. Prevalence of glaucoma and intraocular pressure distribution in a defined population. The Egna-Neumarkt Study. Ophthalmology 1998;105:209-15.

34. Mitchell P, Smith W, Attebo K, Healey PR. Prevalence of open-angle glaucoma in Australia. The Blue Mountains Eye Study. Ophthalmology 1996;103:1661-9.
35. Bankes JL, Perkins ES, Tsolakis S, Wright JE. Bedford glaucoma survey. Br Med J 1968;1:791-6.
36. Coffey M, Reidy A, Wormald R, et al. Prevalence of glaucoma in the west of Ireland. Br J Ophthalmol 1993;77:17-21.
37. Hollows FC, Graham PA. Intra-ocular pressure, glaucoma, and glaucoma suspects in a defined population. Br J Ophthalmol 1966;50:570-86.
38. Wensor MD, McCarty CA, Stanislavsky YL, et al. The prevalence of glaucoma in the Melbourne Visual Impairment Project. Ophthalmology 1998;105:733-9.
39. Klein BE, Klein R, Sponsel WE, et al. Prevalence of glaucoma. The Beaver Dam Eye Study. Ophthalmology 1992;99:1499-504.
40. Dielemans I, Vingerling JR, Wolfs RC, et al. The prevalence of primary open-angle glaucoma in a population-based study in The Netherlands. The Rotterdam Study. Ophthalmology 1994;101:1851-5.
41. Bengtsson B. The prevalence of glaucoma. Br J Ophthalmol 1981;65:46-9.
42. Foster PJ, Johnson GJ. Glaucoma in China: how big is the problem? Br J Ophthalmol 2001;85:1277-82.
43. Quigley HA, Broman AT. The number of people with glaucoma worldwide in 2010 and 2020. Br J Ophthalmol 2006;90:262-7.
44. Leighton DA. Survey of the first-degree relatives of glaucoma patients. Trans Ophthalmol Soc U K 1976;96:28-32.
45. Perkins ES. Family studies in glaucoma. Br J Ophthalmol 1974;58:529-35.
46. Wolfs RC, Grobbee DE, Hofman A, de Jong PT. Risk of acute angle-closure glaucoma after diagnostic mydriasis in nonselected subjects: the Rotterdam Study. Invest Ophthalmol Vis Sci 1997;38:2683-7.
47. Congdon N, Wang F, Tielsch JM. Issues in the epidemiology and population-based screening of primary angle-closure glaucoma. Surv Ophthalmol 1992;36:411-23.
48. Nguyen N, Mora JS, Gaffney MM, et al. A high prevalence of occludable angles in a Vietnamese population. Ophthalmology 1996;103:1426-31.
49. Lai JS, Liu DT, Tham CC, et al. Epidemiology of acute primary angle-closure glaucoma in the Hong Kong Chinese population: prospective study. Hong Kong Med J 2001;7:118-23.
50. Van Herick W, Shaffer RN, Schwartz A. Estimation of width of angle of anterior chamber. Incidence and significance of the narrow angle. Am J Ophthalmol 1969;68:626-9.
51. 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 Ophthalmol 1970;54:161-9.
52. Lavanya R, Foster PJ, Sakata LM, et al. Screening for narrow angles in the Singapore population: evaluation of new noncontact screening methods. Ophthalmology 2008;115:1720-7.
53. Wojciechowski R, Congdon N, Anninger W, Teo Broman A. Age, gender, biometry, refractive error, and the anterior chamber angle among Alaskan Eskimos. Ophthalmology 2003;110:365-75.
54. Lee DA, Brubaker RF, Ilstrup DM. Anterior chamber dimensions in patients with narrow angles and angle-closure glaucoma. Arch Ophthalmol 1984;102:46-50.
55. Wishart PK, Batterbury M. Ocular hypertension: correlation of anterior chamber angle width and risk of progression to glaucoma. Eye (Lond) 1992;6 (Pt 3):248-56.
56. Lowe RF. Primary angle-closure glaucoma. Inheritance and environment. Br J Ophthalmol 1972;56:13-20.
57. Alsbirk PH. Anatomical risk factors in primary angle-closure glaucoma. A ten year follow up survey based on limbal and axial anterior chamber depths in a high risk population. Int Ophthalmol 1992;16:265-72.
58. Salmon JF, Swanevelder SA, Donald M. The dimensions of eyes with chronic angle-closure glaucoma. J Glaucoma 1994;3:237-43.

59. Alsbirk PH. Anterior chamber depth and primary angle-closure glaucoma. I. An epidemiologic study in Greenland Eskimos. Acta Ophthalmol (Copenh) 1975;53:89-104.
60. Kurita N, Mayama C, Tomidokoro A, et al. Potential of the Pentacam in screening for primary angle closure and primary angle closure suspect. J Glaucoma 2009;18:506-12.
61. He M, Huang W, Zheng Y, et al. Anterior chamber depth in elderly Chinese: the Liwan eye study. Ophthalmology 2008;115:1286-90.
62. Xu L, Cao WF, Wang YX, et al. Anterior chamber depth and chamber angle and their associations with ocular and general parameters: the Beijing Eye Study. Am J Ophthalmol 2008;145:929-36.
63. Aung T, Nolan WP, Machin D, et al. Anterior chamber depth and the risk of primary angle closure in 2 East Asian populations. Arch Ophthalmol 2005;123:527-32.
64. Sihota R, Lakshmaiah NC, Agarwal HC, et al. Ocular parameters in the subgroups of angle closure glaucoma. Clin Experiment Ophthalmol 2000;28:253-8.
65. George R, Paul PG, Baskaran M, et al. Ocular biometry in occludable angles and angle closure glaucoma: a population based survey. Br J Ophthalmol 2003;87:399-402.
66. Lavanya R, Wong TY, Friedman DS, et al. Determinants of angle closure in older Singaporeans. Arch Ophthalmol 2008;126:686-91.
67. Aung T, Husain R, Gazzard G, et al. Changes in retinal nerve fiber layer thickness after acute primary angle closure. Ophthalmology 2004;111:1475-9.
68. Bain WE. The fellow eye in acute closed-angle glaucoma. Br J Ophthalmol 1957;41:193-9.
69. Lowe RF. Acute angle-closure glaucoma. The second eye: an analysis of 200 cases. Br J Ophthalmol 1962;46:641-50.
70. Aung T, Friedman DS, Chew PT, et al. Long-term outcomes in Asians after acute primary angle closure. Ophthalmology 2004;111:1464-9.
71. American Academy of Ophthalmology Preferred Practice Patterns Committee. Preferred Practice Pattern® Guidelines. Comprehensive Adult Medical Eye Evaluation. San Francisco, CA: American Academy of Ophthalmology; 2010. Available at: www.aao.org/ppp.
72. Salmon JF. Predisposing factors for chronic angle-closure glaucoma. Prog Retin Eye Res 1999;18:121-32.
73. Panday VA, Rhee DJ. Review of sulfonamide-induced acute myopia and acute bilateral angle-closure glaucoma. Compr Ophthalmol Update 2007;8:271-6.
74. Fraunfelder FW, Fraunfelder FT, Keates EU. Topiramate-associated acute, bilateral, secondary angle-closure glaucoma. Ophthalmology 2004;111:109-11.
75. Li J, Tripathi RC, Tripathi BJ. Drug-induced ocular disorders. Drug Saf 2008;31:127-41.
76. Setala K. Corneal endothelial cell density after an attack of acute glaucoma. Acta Ophthalmol (Copenh) 1979;57:1004-13.
77. Olsen T. The endothelial cell damage in acute glaucoma. On the corneal thickness response to intraocular pressure. Acta Ophthalmol (Copenh) 1980;58:257-66.
78. Bigar F, Witmer R. Corneal endothelial changes in primary acute angle-closure glaucoma. Ophthalmology 1982;89:596-9.
79. Markowitz SN, Morin JD. The endothelium in primary angle-closure glaucoma. Am J Ophthalmol 1984;98:103-4.
80. Aghaian E, Choe JE, Lin S, Stamper RL. Central corneal thickness of Caucasians, Chinese, Hispanics, Filipinos, African Americans, and Japanese in a glaucoma clinic. Ophthalmology 2004;111:2211-9.
81. Bhargava SK, Leighton DA, Phillips CI. Early angle-closure glaucoma. Distribution of iridotrabecular contact and response to pilocarpine. Arch Ophthalmol 1973;89:369-72.
82. Gazzard G, Foster PJ, Friedman DS, et al. Light to dark physiological variation in iridotrabecular angle width. Br J Ophthalmol 2004. Available at: http://bjo.bmj.com/cgi/content/full/88/11/DC1/1. Accessed September 16, 2010.
83. Ishikawa H, Liebmann JM, Ritch R. Quantitative assessment of the anterior segment using ultrasound biomicroscopy. Curr Opin Ophthalmol 2000;11:133-9.

84. Kaushik S, Jain R, Pandav SS, Gupta A. Evaluation of the anterior chamber angle in Asian Indian eyes by ultrasound biomicroscopy and gonioscopy. Indian J Ophthalmol 2006;54:159-63.
85. Barkana Y, Dorairaj SK, Gerber Y, et al. Agreement between gonioscopy and ultrasound biomicroscopy in detecting iridotrabecular apposition. Arch Ophthalmol 2007;125:1331-5.
86. Nolan WP, See JL, Chew PT, et al. Detection of primary angle closure using anterior segment optical coherence tomography in Asian eyes. Ophthalmology 2007;114:33-9.
87. Friedman DS, He M. Anterior chamber angle assessment techniques. Surv Ophthalmol 2008;53:250-73.
88. Wong HT, Lim MC, Sakata LM, et al. High-definition optical coherence tomography imaging of the iridocorneal angle of the eye. Arch Ophthalmol 2009;127:256-60.
89. Wilensky JT, Kaufman PL, Frohlichstein D, et al. Follow-up of angle-closure glaucoma suspects. Am J Ophthalmol 1993;115:338-46.
90. Ritch R, Tham CC, Lam DS. Long-term success of argon laser peripheral iridoplasty in the management of plateau iris syndrome. Ophthalmology 2004;111:104-8.
91. Ng WS, Ang GS, Azuara-Blanco A. Laser peripheral iridoplasty for angle-closure. Cochrane Database of Systematic Reviews 2008, Issue 3. Art. No.: CD006746. DOI: 10.1002/14651858.CD006746.pub2.
92. Saw SM, Gazzard G, Friedman DS. Interventions for angle-closure glaucoma: an evidence-based update. Ophthalmology 2003;110:1869-78; quiz 78-9, 930.
93. American Academy of Ophthalmology. Policy Statement. Pretreatment Assessment: Responsibilities of the Ophthalmologist. San Francisco, CA: American Academy of Ophthalmology; 2006. Available at: http://one.aao.org/CE/PracticeGuidelines/ClinicalStatements.aspx.
94. American Academy of Ophthalmology. Policy Statement. An Ophthalmologist's Duties Concerning Postoperative Care. San Francisco, CA: American Academy of Ophthalmology; 2006. Available at: http://one.aao.org/CE/PracticeGuidelines/ClinicalStatements.aspx.
95. Robin AL, Pollack IP, deFaller JM. Effects of topical ALO 2145 (p-aminoclonidine hydrochloride) on the acute intraocular pressure rise after argon laser iridotomy. Arch Ophthalmol 1987;105:1208-11.
96. Rosenblatt MA, Luntz MH. Intraocular pressure rise after argon laser trabeculoplasty. Br J Ophthalmol 1987;71:772-5.
97. Barnes SD, Campagna JA, Dirks MS, Doe EA. Control of intraocular pressure elevations after argon laser trabeculoplasty: comparison of brimonidine 0.2% to apraclonidine 1.0%. Ophthalmology 1999;106:2033-7.
98. Ritch R. Exfoliation syndrome and occludable angles. Trans Am Ophthalmol Soc 1994;92:845-944.
99. Gross FJ, Tingey D, Epstein DL. Increased prevalence of occludable angles and angle-closure glaucoma in patients with pseudoexfoliation. Am J Ophthalmol 1994;117:333-6.
100. Brusini P, Tosoni C, Miani P. Increased prevalence of occludable angles and angle-closure glaucoma in patients with pseudoexfoliation. Am J Ophthalmol 1994;118:540.
101. Franks WA, Miller MH, Hitchings RA, Jeffrey MN. Secondary angle closure in association with pseudoexfoliation of the lens capsule. Acta Ophthalmol (Copenh) 1990;68:350-2.
102. Campbell DG, Vela A. Modern goniosynechialysis for the treatment of synechial angle-closure glaucoma. Ophthalmology 1984;91:1052-60.
103. Shingleton BJ, Chang MA, Bellows AR, Thomas JV. Surgical goniosynechialysis for angle-closure glaucoma. Ophthalmology 1990;97:551-6.
104. Tanihara H, Nishiwaki K, Nagata M. Surgical results and complications of goniosynechialysis. Graefes Arch Clin Exp Ophthalmol 1992;230:309-13.
105. Wand M. Argon laser gonioplasty for synechial angle closure. Arch Ophthalmol 1992;110:363-7.
106. Steuhl KP, Marahrens P, Frohn C, Frohn A. Intraocular pressure and anterior chamber depth before and after extracapsular cataract extraction with posterior chamber lens implantation. Ophthalmic Surg 1992;23:233-7.
107. Yang CH, Hung PT. Intraocular lens position and anterior chamber angle changes after cataract extraction in eyes with primary angle-closure glaucoma. J Cataract Refract Surg 1997;23:1109-13.

108. Hayashi K, Hayashi H, Nakao F, Hayashi F. Changes in anterior chamber angle width and depth after intraocular lens implantation in eyes with glaucoma. Ophthalmology 2000;107:698-703.
109. Tham CC, Lai JS, Lam DS. Changes in AC angle width and depth after IOL implantation in eyes with glaucoma. Ophthalmology 2001;108:428-9.
110. Greve EL. Primary angle closure glaucoma: extracapsular cataract extraction or filtering procedure? Int Ophthalmol 1988;12:157-62.
111. Keenan TD, Salmon JF, Yeates D, Goldacre M. Trends in rates of primary angle closure glaucoma and cataract surgery in England from 1968 to 2004. J Glaucoma 2009;18:201-5.
112. Lai JS, Tham CC, Chua JK, et al. To compare argon laser peripheral iridoplasty (ALPI) against systemic medications in treatment of acute primary angle-closure: mid-term results. Eye (Lond) 2006;20:309-14.
113. Lam DS, Tham CC, Lai JS, Leung DY. Current approaches to the management of acute primary angle closure. Curr Opin Ophthalmol 2007;18:146-51.
114. Lam DS, Chua JK, Tham CC, Lai JS. Efficacy and safety of immediate anterior chamber paracentesis in the treatment of acute primary angle-closure glaucoma: a pilot study. Ophthalmology 2002;109:64-70.
115. Ritch R, Lowe RF. Angle-closure glaucoma: therapeutic overview. In: Ritch R, Shields MB, Krupin T, eds. The Glaucomas. St. Louis: CV Mosby Co, 1996:1521-31.
116. Choong YF, Irfan S, Menage MJ. Acute angle closure glaucoma: an evaluation of a protocol for acute treatment. Eye (Lond) 1999;13 (Pt 5):613-6.
117. Aung T, Oen F, Wong H-T, et al. Randomised controlled trial comparing the effect of brimonidine and timolol on visual field loss after acute primary angle closure. Br J Ophthalmol 2004;88:88-94.
118. Koyabashi H, Koyabashi K, Kiryu J, Kondo T. Pilocarpine induces an increase in the anterior chamber angular width in eyes with narrow angles. Br J Ophthalmol 1999;83:553-8.
119. Anderson DR. Corneal indentation to relieve acute angle-closure glaucoma. Am J Ophthalmol 1979;88:1091-3.
120. American Academy of Ophthalmology Committee on Ophthalmic Procedures Assessment. Laser peripheral iridotomy for pupillary-block glaucoma. American Academy of Ophthalmology. Ophthalmology 1994;101:1749-58.
121. Robin AL, Pollack IP. Argon laser peripheral iridotomies in the treatment of primary angle closure glaucoma. Long-term follow-up. Arch Ophthalmol 1982;100:919-23.
122. Ritch R. Argon laser peripheral iridoplasty: an overview. J Glaucoma 1992;1:206-13.
123. Shin DH. Argon laser iris photocoagulation to relieve acute angle-closure glaucoma. Am J Ophthalmol 1982;93:348-50.
124. Ritch R. Argon laser treatment for medically unresponsive attacks of angle-closure glaucoma. Am J Ophthalmol 1982;94:197-204.
125. Lai JS, Tham CC, Lam DS. Limited argon laser peripheral iridoplasty as immediate treatment for an acute attack of primary angle closure glaucoma: a preliminary study. Eye (Lond) 1999;13 (Pt 1):26-30.
126. Lam DS, Lai JS, Tham CC. Immediate argon laser peripheral iridoplasty as treatment for acute attack of primary angle-closure glaucoma: a preliminary study. Ophthalmology 1998;105:2231-6.
127. Tham CC, Lai JS, Lam DS. Immediate argon laser peripheral iridoplasty for acute attack of PACG (addendum to previous report). Ophthalmology 1999;106:1042-3.
128. Lam DS, Lai JS, Tham CC, et al. Argon laser peripheral iridoplasty versus conventional systemic medical therapy in treatment of acute primary angle-closure glaucoma : a prospective, randomized, controlled trial. Ophthalmology 2002;109:1591-6.
129. Lai JS, Tham CC, Lam DS. The efficacy and safety of combined phacoemulsification, intraocular lens implantation, and limited goniosynechialysis, followed by diode laser peripheral iridoplasty, in the treatment of cataract and chronic angle-closure glaucoma. J Glaucoma 2001;10:309-15.
130. Aung T, Tow SL, Yap EY, et al. Trabeculectomy for acute primary angle closure. Ophthalmology 2000;107:1298-302.

131. Wishart PK, Atkinson PL. Extracapsular cataract extraction and posterior chamber lens implantation in patients with primary chronic angle-closure glaucoma: effect on intraocular pressure control. Eye (Lond) 1989;3 (Pt 6):706-12.
132. Gunning FP, Greve EL. Uncontrolled primary angle closure glaucoma: results of early intercapsular cataract extraction and posterior chamber lens implantation. Int Ophthalmol 1991;15:237-47.
133. Acton J, Salmon JF, Scholtz R. Extracapsular cataract extraction with posterior chamber lens implantation in primary angle-closure glaucoma. J Cataract Refract Surg 1997;23:930-4.
134. Gunning FP, Greve EL. Lens extraction for uncontrolled angle-closure glaucoma: long-term follow-up. J Cataract Refract Surg 1998;24:1347-56.
135. Roberts TV, Francis IC, Lertusumitkul S, et al. Primary phacoemulsification for uncontrolled angle-closure glaucoma. J Cataract Refract Surg 2000;26:1012-6.
136. Hayashi K, Hayashi H, Nakao F, Hayashi F. Effect of cataract surgery on intraocular pressure control in glaucoma patients. J Cataract Refract Surg 2001;27:1779-86.
137. Jacobi PC, Dietlein TS, Luke C, et al. Primary phacoemulsification and intraocular lens implantation for acute angle-closure glaucoma. Ophthalmology 2002;109:1597-603.
138. Khokar K, Pangtey M. Phacoemulsification in filtered chronic angle closure glaucoma eyes. Clin Experiment Ophthalmol 2002;30:256-60.
139. Ming Zhi Z, Lim AS, Yin Wong T. A pilot study of lens extraction in the management of acute primary angle-closure glaucoma. Am J Ophthalmol 2003;135:534-6.
140. Kubota T, Toguri I, Onizuka N, Matsuura T. Phacoemulsification and intraocular lens implantation for angle closure glaucoma after the relief of pupillary block. Ophthalmologica 2003;217:325-8.
141. Lam DS, Leung DY, Tham CC, 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.
142. Harasymowycz PJ, Papamatheakis DG, Ahmed I, et al. Phacoemulsification and goniosynechialysis in the management of unresponsive primary angle closure. J Glaucoma 2005;14:186-9.
143. Matsumura M, Ido W, Shirakam Y, Zoizumi K. Treatment of primary closed angle glaucoma with cataract by lysis of peripheral anterior synechiae and intraocular lens implantation. Jpn J Clin Ophthalmol 1991;45:1567-9.
144. Teekhasaenee C, Ritch R. Combined phacoemulsification and goniosynechialysis for uncontrolled chronic angle-closure glaucoma after acute angle-closure glaucoma. Ophthalmology 1999;106:669-74; discussion 74-5.
145. Tham CC, Kwong YY, Leung DY, et al. Phacoemulsification versus combined phacotrabeculectomy in medically uncontrolled chronic angle closure glaucoma with cataracts. Ophthalmology 2009;116:725-31.
146. Edwards RS. Behaviour of the fellow eye in acute angle-closure glaucoma. Br J Ophthalmol 1982;66:576-9.
147. Ang LP, Aung T, Chew PT. Acute primary angle closure in an Asian population: long-term outcome of the fellow eye after prophylactic laser peripheral iridotomy. Ophthalmology 2000;107:2092-6.
148. Snow JT. Value of prophylactic peripheral iridectomy on the second eye in angle-closure glaucoma. Trans Ophthalmol Soc U K 1977;97:189-91.
149. American Academy of Ophthalmology Vision Rehabilitation Committee. Preferred Practice Pattern® Guidelines. Vision Rehabilitation for Adults. San Francisco, CA: American Academy of Ophthalmology; 2007. Available at: www.aao.org/ppp.

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