

Angle Closure Glaucoma

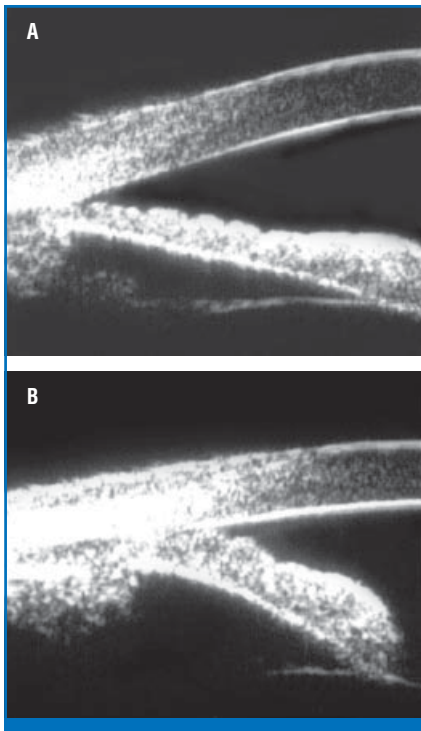
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Gonioscopy

Accurate assessment of narrow or closed angles requires precise 4-mirror dynamic gonioscopy.¹ Use of the Goldmann lens is much less accurate. Pressure on the cornea forces aqueous humour into the angle, widening it. The presence and extent of synechial closure and the depth of the angle can be determined. The angle should be assessed with respect to iris convexity, width, depth, and the dimensions of peripheral anterior synechiae (PAS) or the presence of other pathology.

When assessing a narrow angle for

Figure 1. Ultrasound biomicrograph of an eye under light (A) and dark (B) conditions. The angle, which appears open in the light, is actually occludable in the dark.



occludability, gonioscopy in a completely darkened room using the smallest square of slit-beam light to avoid stimulating the pupillary light reflex is of the utmost importance (figure 1). The quadrant of angle to be assessed is examined with the 4-mirror lens without pressure on the cornea and with the patient looking sufficiently far in the direction of the mirror so that the examiner can see deeply into the angle. The angle is observed while the pupil dilates in the dark. The narrowest quadrant is usually the superior angle (inferior mirror). PAS may be deep in the angle and difficult to see when the angle is very narrow and the iris very convex. Appositional closure and PAS most commonly form initially in the superior angle.² When angle closure is limited to the superior angle, maximum pigmentation of the trabecular meshwork may occur there.³ This pigment is characteristically blotchy and scattered over the trabecular meshwork.

The Anatomic Basis of Angle Closure Glaucoma

Angle closure glaucoma (ACG) is an anatomic disorder comprising a final common pathway of iris apposition to the trabecular meshwork resulting from various abnormal relationships of anterior segment structures. These in turn result from 1 or more abnormalities in the relative or absolute sizes or positions of anterior segment structures or posterior segment forces that alter anterior segment anatomy.⁴ ACG can be caused

- by 1 or several of the following factors:
- abnormalities in the *relative* sizes or positions of anterior segment structures
 - abnormalities in the *absolute* sizes or positions of anterior segment structures
 - abnormal *forces* in the posterior segment which alter the anatomy of the anterior segment.

The forces causing iris apposition to the trabecular meshwork may be viewed as originating at 4 anatomical levels: the posterior chamber (pupillary block), the ciliary body (plateau iris), the lens (phacomorphic glaucoma), and posterior to the lens (malignant glaucoma).⁴ The more posterior the level, the more complex is diagnosis and treatment, since each level may have a component of the mechanism peculiar to each of the levels preceding it.

Pupillary Block (Aqueous Pressure)

Inhibition of aqueous flow from the posterior to the anterior chamber creates a relative pressure gradient between the 2 chambers and pushes the iris anteriorly, causing narrowing or closure of the angle (figure 2). Relative pupillary block typically occurs in hyperopic eyes, which have a shorter than average axial length, more shallow anterior chamber, thicker lens, more anterior lens position, smaller corneal diameter, and smaller radius of corneal curvature.⁵⁻⁸ In absolute pupillary block, posterior synechiae between the iris and lens are responsible. When pupillary block develops, the iris assumes a bombé configuration, creating an angle that is narrow throughout its approach. Dynamic (indentation) gonioscopy forces the entire iris posteriorly, opening the angle. If synechiae are absent, the angle opens widely.

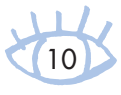
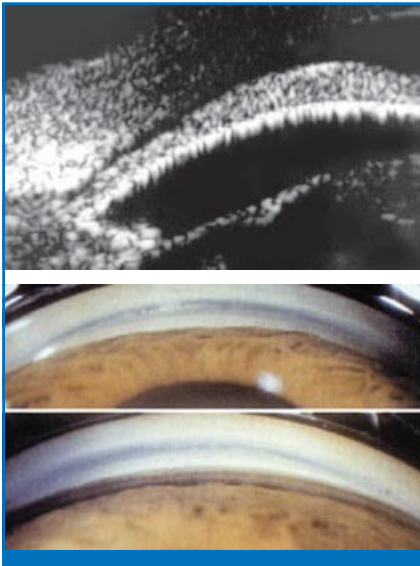


Figure 2. Pupillary block.

Pupillary block does not affect central anterior chamber depth. Anterior lens movement may occur in some eyes in the prone position, of possible importance in provocative testing and in the aetiology of some attacks of ACG. Miotic-induced ciliary muscle constriction relaxes the zonules, producing anterior lens movement and increased lens thickness and curvature, all of which augment pupillary block. Exfoliation syndrome, presumably because of zonular involvement, appears to predispose to anterior lens movement and increased pupillary block.⁹

With chronic appositional closure, acute angle closure may develop or PAS may form and lead to chronic angle closure glaucoma (CACG). Laser iridotomy eliminates the pressure differential between the anterior and posterior chambers and relieves the iris convexity. The iris configuration becomes planar and the angle widens. The region of iridolenticular contact actually increases, as aqueous flows through the iridotomy rather than the pupillary space.¹⁰

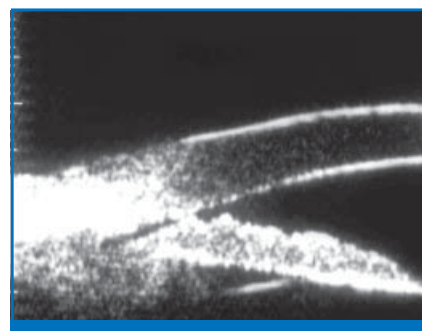
The physiological factors that convert relative pupillary block to absolute pupillary block remain poorly understood,

as are those that determine whether an eye will develop acute or chronic angle closure. Absolute pupillary block is most commonly triggered when the pupil is mid-dilated (approximately 3.5 to 6 mm in diameter). In this position, the combination of pupillary block and relaxation of the peripheral iris, allowing its forward displacement into the anterior chamber, are maximised. Most attacks occur during the evening, beginning mildly and rapidly increasing in severity. Approximately one-third of patients describe episodes of intermittent or subacute angle closure having occurred before the acute attack. The most common precipitating events include illness, emotional stress, trauma, intense concentration, and pharmacological pupillary dilation.

Plateau Iris (Ciliary Body Pressure)



In plateau iris, the ciliary processes are large or anteriorly situated, supporting the iris root against the trabecular meshwork (figure 3).¹¹⁻¹³ Iris cysts may also cause a situation equivalent to plateau iris.¹⁴ The anterior chamber is usually of medium depth and the iris surface slightly convex. On gonioscopy, the iris root angulates forward and then centrally. With dynamic gonioscopy, the ciliary processes prevent posterior movement of the peripheral iris, resulting in a configuration in which the slit beam follows

Figure 3. Plateau iris.

the curvature of the iris to its deepest point at the periphery of the lens where the ciliary processes begin, then rises again over the ciliary processes before dropping peripherally (double hump sign). Greater force is needed to open the angle than in pupillary block because the ciliary processes must be displaced, and the angle does not open as widely. In a morphometric study of the ciliary sulcus, Orgül *et al.* proposed that the displacement of the pars plicata from the peripheral iris to the iris root during embryogenesis may be incomplete in eyes of shorter axial length.¹⁵

Until recently, plateau iris syndrome was considered rare. We have differentiated 2 subtypes.¹⁶ In the complete syndrome, which is rare, intraocular pressure (IOP) rises when the angle closes with pupillary dilation. In the incomplete syndrome, it does not. The differentiating factor is the height of the plateau with respect to the angle structures. If the angle closes to the upper meshwork or Schwalbe's line, IOP rises, since aqueous outflow is completely blocked, whereas if the angle closes partially, leaving the upper portion of the filtering meshwork open, aqueous humour can still exit the eye. This is far more common and its detection is important, as these patients can develop PAS years after a successful iridotomy produces what appears as a well-opened angle.

Patients with plateau iris tend to be female, younger (in the 30 to 50 years age group) and less hyperopic than those with relative pupillary block, and often have a family history of ACG. Except in the rare younger patients (aged 20 to 30 years old), some element of pupillary block is also present. If plateau iris was not diagnosed before iridotomy and IOP is elevated post-laser treatment, careful gonioscopy should be performed. If the angle is open, secondary damage to

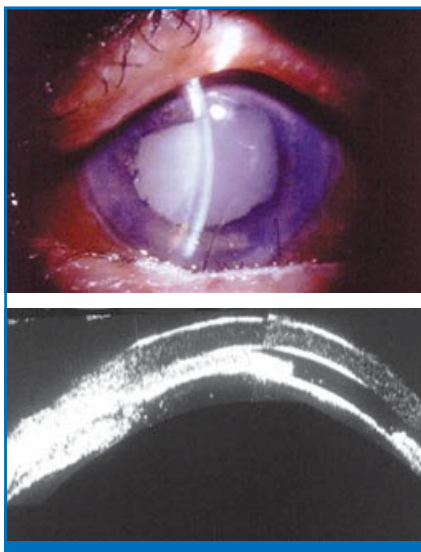


the trabecular meshwork or pigment liberation with dilation are the most likely causes. If the angle is closed, the differential diagnosis, besides plateau iris, should include malignant glaucoma, in which the anterior chamber is extremely shallow; PAS, which can be ruled out by dynamic gonioscopy; or incomplete iridectomy.

Phacomorphic Glaucoma (Lens Pressure)

Swelling of the lens may shallow the anterior chamber and precipitate acute ACG due to the lens pressing against the iris and ciliary body and forcing them anteriorly (figure 4). Pilocarpine produces a paradoxical reaction, which increases axial lens thickness and causes anterior lens movement, further shallowing the anterior chamber.¹⁷ Pilocarpine, even in elderly patients, increases axial lens thickness and causes anterior lens movement, further shallowing the anterior chamber.¹⁸ Argon laser peripheral iridoplasty (ALPI) is effective in treating attacks of phacomorphic angle closure.¹⁹

Figure 4. Phacomorphic glaucoma.



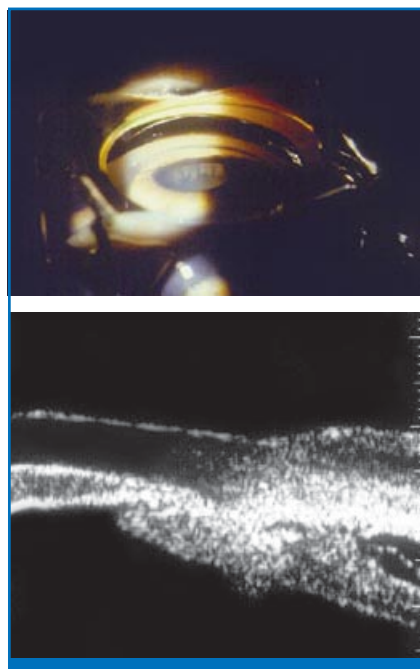
Malignant Glaucoma (Vitreous Pressure)

Angle closure caused by forces posterior to the lens which push the lens-iris diaphragm forward presents a great diagnostic and treatment challenge. Malignant (ciliary block) glaucoma is a multifactorial disease in which the following components may play varying roles:

- previous acute or chronic angle closure glaucoma
- shallowness of the anterior chamber
- forward movement of the lens
- pupillary block by the lens or vitreous
- slackness of the zonules
- anterior rotation and/or swelling of the ciliary body
- thickening of the anterior hyaloid membrane
- expansion of the vitreous
- posterior aqueous displacement into or behind the vitreous.

In ciliary block, a pressure differential is created between the vitreous and aqueous compartments by aqueous misdirection into the vitreous.

Figure 5. Malignant glaucoma.



Swelling or anterior rotation of the ciliary body with forward rotation of the lens-iris diaphragm and relaxation of the zonular apparatus causes anterior lens displacement (figure 5). Ultrasound biomicroscopy (UBM) usually reveals a shallow supraciliary detachment not evident on routine B-scan. This effusion appears to be the cause of the anterior rotation of the ciliary body and the forward movement of the lens-iris diaphragm. This, combined with aqueous misdirection into the vitreous, increases vitreous pressure, pushing the lens-iris diaphragm forward and causing angle closure by physically pushing the iris against the trabecular meshwork in a manner similar to that in phacomorphic glaucoma.

In predisposed eyes, miotic therapy can have a pronounced effect on lens position and trigger malignant glaucoma. Unequal anterior chamber depths, a progressive increase in myopia, or progressive shallowing of the anterior chamber are clues to the correct diagnosis. Malignant glaucoma may occur following cataract surgery with posterior chamber intraocular lens implantation. The differential diagnosis includes pupillary block, choroidal haemorrhage, and ciliochoroidal effusion with anterior rotation of the ciliary body and secondary angle closure. Shallowing of the central anterior chamber occurs in pseudophakic malignant glaucoma, but not in pupillary block. Rupture of the anterior hyaloid face is usually curative and allows aqueous to move into the anterior segment. Several patients with presumed aqueous misdirection in whom an annular ciliary body detachment had caused anterior movement of the ciliary body have been examined. Whether a posterior diversion of aqueous flow is present in these disorders is unknown.

Chronic Angle Closure Glaucoma



Chronic angle closure glaucoma refers to an eye in which portions of the anterior chamber angle are permanently closed by PAS. Variable and sometimes conflicting terminology has been used to describe somewhat differently appearing forms. The problem arose from the fact that the terminology developed prior to the advent of indentation gonioscopy and laser iridotomy, so that the mechanisms of ACG were poorly understood. The term CACG is used to denote eyes in which chronic appositional closure without PAS has led to elevated IOP or in which appositional closure with the formation of PAS has occurred in the presence of normal IOP. A patient who is noted after iridotomy for acute angle closure glaucoma (AACG) to have PAS is also considered to have had CACG prior to developing the AACG. Prolonged apposition or repeated subacute attacks lead to gradual PAS formation. These usually begin in the superior angle, which is narrower than the inferior angle,^{20,21} as pinpoint synechiae reaching to the mid-trabecular meshwork and then gradually expanding in width. In early cases, in which appositional closure is present and IOP is normal, but in which PAS has not yet formed, the term chronic appositional closure is preferred.

Eyes with progressive PAS formation may eventually develop AACG when pupillary block closes off the remaining open portions of the angle. Many patients, however, develop gradual angle closure, elevated IOP and glaucomatous damage in the absence of symptoms. The presentation is similar to that of chronic open angle glaucoma, with progression of glaucomatous cupping and visual field loss. PAS may also form during an acute

attack, remaining after iridotomy has opened the unaffected portions of the angle. These PAS are usually high and broad. When first observed at this stage, it is impossible to determine whether the PAS formed before or during the attack, or at both times.

In eyes with darker irides, particularly in Asians, a second mechanism of progressive angle closure is more common. The closure is circumferential and begins in the deepest portion of the angle. Closure occurs more evenly in all quadrants, so that the angle progressively becomes more shallow. The appearance over time is of a progressively more anterior iris insertion. The PAS gradually creep up the ciliary face to the scleral spur and then to the trabecular meshwork. Lowe termed this creeping angle closure.²² Creeping angle closure occurs in eyes with slightly deeper anterior chambers than are found in AACG. Gradual shortening of the angle in the presence of iris bombé brings the peripheral iris close to the external angle wall more anteriorly, narrowing the gap between the iris and the trabecular meshwork. Eventually, AACG may supervene or the PAS may permanently occlude the trabecular meshwork and lead to elevated IOP and glaucomatous damage.

The IOP in eyes with CACG may be normal or elevated. As PAS formation progresses in the absence of intermittent attacks, IOP rises gradually as less functional meshwork becomes available. In eyes with intermittent attacks, IOP rises more rapidly relative to the extent of PAS formation caused by recurrent damage to the trabecular meshwork by the transient angle closure.

Combined Mechanism Glaucoma



Combined mechanism glaucoma refers to situations in which both open angle

and angle closure components are present. Most commonly, angle closure glaucoma is treated successfully with iridotomy, eliminating all appositional closure, and IOP still remains elevated with or without the presence of PAS of any extent. Conversely, open angle glaucoma may later develop angle closure, either because of the natural development of pupillary block or because of exacerbation by miotic therapy. Exfoliation syndrome commonly predisposes to combined mechanism glaucoma.⁹ Here, open angle glaucoma can develop independently years after iridotomy for angle closure, with progressive blockage of the trabecular meshwork. In all of these cases, the residual open angle component is treated as open angle glaucoma.

Mixed Mechanism Glaucoma



This term is often used interchangeably with combined mechanism glaucoma, but should not be, as it creates additional confusion. It is better to reserve it to describe an eye with angle closure due to more than one contributory mechanism. When pupillary block is eliminated by iridotomy and the angle opens to a greater degree than before the iridotomy, and appositional closure remains on the basis of plateau iris, phacomorphic glaucoma, or malignant glaucoma, a mixed mechanism may be said to be present.

Miotic-induced Angle Closure Glaucoma



Prolonged miotic treatment in eyes with open angle glaucoma and narrow angles may lead to pupillary block and angle closure glaucoma. CACG has been seen to develop after several years of miotic therapy in eyes that initially had wide open



angles. In some eyes, zonular relaxation occurs more readily than in others, so that anterior lens movement and an increase in axial lens thickness may facilitate pupillary block and angle closure. In other eyes, there is little change in the lens, but progressively increasing pressure in the posterior chamber gradually pushes the peripheral iris against the trabecular meshwork. It is our impression that eyes with exfoliation syndrome are particularly prone to develop miotic-induced angle closure. In these eyes, the iris is thicker and stiffer than normal due to deposition of exfoliation material within the stroma. In addition, zonular weakness allows the lens to move forward, leading to pupillary block.

Less commonly, miotic therapy can

have a pronounced effect on lens position and trigger malignant glaucoma.²³⁻²⁶ Unequal anterior chamber depths, a progressive increase in myopia, or progressive shallowing of the anterior chamber are clues to the correct diagnosis.

Treatment of Angle Closure Glaucoma



The use of miotics to constrict the pupil and draw the peripheral iris away from the trabecular meshwork was formerly the main approach to treatment of AACG. The more severe and prolonged the attack, the more frequently miotics were applied. A typical recommended regimen was 4% pilocarpine every 5 minutes for 4 doses, every 15 minutes for 4 doses, then every hour for 4 doses

or until the attack stopped. However, when IOP is over 60 mm Hg, the pupil is unresponsive to miotics because of ischaemia and paralysis of the iris sphincter. Pilocarpine may not only be ineffective, but it may paradoxically worsen the situation even in pupillary block. Although the miotic effect of pilocarpine is blocked when IOP is extremely high, ciliary muscle contraction and anterior movement of the lens-iris diaphragm are not. High doses of pilocarpine may produce cholinergic toxicity, which may not be noticed because of the nausea and vomiting associated with the acute angle closure attack. Strong miotics, such as echothiophate, should not be used because they can increase both the pupillary block and vascular congestion.



The New York Eye and Ear Infirmary Approach to Acute Angle Closure Glaucoma

The New York Eye and Ear Infirmary approach to a patient with AACG is as follows.²⁷

1. Careful history of symptoms relating to intermittent angle closure attacks, attacks in the other eye, use of prescription or non-prescription drugs which may precipitate attacks, and type of activity preceding the attack.
2. Examination of the affected eye and other eye with attention to central and peripheral anterior chamber depth as well as the shape of the peripheral iris.
3. Administration of oral isosorbide and one or more topical aqueous suppressants. Intravenous acetazolamide can be given according to the surgeon's preference.
4. The patient lies supine to permit the

lens to fall posteriorly with vitreous dehydration.

5. The eye is reassessed after 1 hour. IOP is usually decreased, but the angle usually remains appositionally closed. One drop of 2% or 4% pilocarpine is given and the patient re-examined 30 minutes later.
 - a. If IOP is reduced and the angle is open, the patient may be treated medically with topical low-dose pilocarpine, aqueous suppressants and steroids, until the eye quiets and laser iridotomy may be performed.
 - b. If IOP is unchanged or elevated and the angle remains closed, lens-related angle closure should be suspected, further pilocarpine is withheld, and the attack bro-

ken by argon laser peripheral iridoplasty.^{19,28,29}

Peripheral iridoplasty does not eliminate pupillary block and is not a substitute for laser iridotomy, which must be performed as soon as the eye is quiet. However, even in eyes with extensive synechial closure, IOP is lowered sufficiently for a few days for the inflammation to resolve. Peripheral iridoplasty is much safer than attempting surgical iridectomy on an inflamed eye with elevated IOP. The risks of intra-operative surgery are avoided and, even if malignant glaucoma is present, the angle remains open long enough for inflammation to clear. Peripheral iridoplasty is highly effective in ameliorating attacks of angle closure glaucoma in Asian eyes.³⁰⁻³⁵

Treatment of Chronic Angle Closure Glaucoma

It is important to recognise early stages of appositional angle closure in the absence of PAS and to recognise deep, circumferential angle closure.³⁶ Laser iridotomy is indicated for all stages of CACG.^{37,38} Iridotomy will open those areas of the angle not involved by PAS and prevent further synechial closure. Miotic treatment may enhance the development of CACG in the absence of an iridotomy. When miotic-induced angle closure occurs, the approach to treatment should be determined by assessing the medications necessary to control the glaucoma. If a patient is taking dipivefrin, its discontinuation may be enough to open the angle and allow the patient to continue taking miotics, presuming the IOP remains under control. If the patient has been treated with miotics alone, substitution of aqueous suppressants may suffice. If the patient requires miotics for IOP control, then laser iridotomy is warranted.

If the angle remains appositionally closed or spontaneously occludable after laser iridotomy, ALPI is indicated to prevent progressive damage to or further appositional and/or synechial closure of the angle.^{29, 39-41} If, after ALPI, some of the angle still remains appositionally closed, low-dose pilocarpine, such as 2% at bedtime, often suffices to maintain the patency of the angle.

The need for continued medical treatment after iridotomy is determined by the level of IOP and the extent of glaucomatous damage. Treatment is similar to that of open angle glaucoma. Repeated gonioscopy is necessary. The need for further surgery cannot be predicted from the level of initial IOP or the gonioscopic changes. Argon laser

trabeculoplasty has been reported to be both successful⁴² and unsuccessful⁴³ after iridotomy in combined mechanism glaucoma. We have found it to be reasonably successful overall. If the pressure remains uncontrolled and glaucomatous damage develops, filtration surgery is indicated. There is an increased chance of developing malignant glaucoma following filtration surgery in patients who have had angle closure glaucoma.⁴⁴

Goniosynechialysis is a surgical procedure designed for the purpose of physically stripping PAS from the angle wall and restoring trabecular function.⁴⁵⁻⁵⁰ A paracentesis track is made into the anterior chamber and the chamber allowed to shallow slightly. Massage is performed at the limbus to force aqueous from the posterior chamber into the anterior chamber. A viscoelastic agent is injected and the angle visualised with direct gonioscopy. An irrigating cyclo-dialysis spatula is used to separate a small segment of PAS at a time, with an anterior to posterior movement.

Goniosynechialysis is successful only if the synechiae have been present for less than 1 year. Although it has not become popular in the United States, it has in Asia, where promising results have been reported in both phakic and pseudophakic eyes. It is effective both alone and in conjunction with other surgical procedures. ALPI can be used postoperatively to further flatten the peripheral iris and prevent synechial reattachment.⁴⁶ Complications include bleeding, iridodialysis, and marked inflammation.

Acknowledgement

Supported in part by The New York Glaucoma Research Institute, New York, NY, USA.

References

1. Palmberg P. Gonioscopy. In: Ritch R, Shields MB, Krupin T, eds. *The Glaucomas*. St Louis: CV Mosby Co, 1989:345-359.
2. Bhargava SK, Leighton DA, Phillips CI. Distribution of goniosynechiae and iridotrabecular contact. *Arch Ophthalmol* 1973;**89**:369-372.
3. Desjardins D, Parrish RK. Inversion of anterior chamber pigment as a possible prognostic sign in narrow angles. *Am J Ophthalmol* 1985;**100**:480-481.
4. Ritch R, Liebmann J, Tello C. A construct for understanding angle-closure glaucoma: the role of ultrasound biomicroscopy. *Ophthalmol Clin North Am* 1995;**8**:281-293.
5. Delmarcelle Y, François J, Goes F, et al. Biometrie oculaire clinique (oculometrie). *Bull Soc Ophthalmol Belge* 1976;**fascicule 1**:172.
6. Lee DA, Brubaker RF, Illstrup DM. Anterior chamber dimensions in patients with narrow angles and angle-closure glaucoma. *Arch Ophthalmol* 1984;**102**:46-50.
7. Lowe RF. Primary angle-closure glaucoma: a review of ocular biometry. *Aust J Ophthalmol* 1977;**5**:9-17.
8. Tomlinson A, Leighton DA. Ocular dimensions in the heredity of angle-closure glaucoma. *Br J Ophthalmol* 1973;**57**:475-486.
9. Ritch R. Exfoliation syndrome: clinical findings and occurrence in patients with occludable angles. *Trans Am Ophthalmol Soc* 1994;**92**:845-944.
10. Caronia RM, Liebmann JM, Stegman Z, et al. Iris-lens contact increases following laser iridotomy for pupillary block angle-closure. *Am J Ophthalmol* 1996;**122**:53-57.
11. Pavlin CJ, Ritch R, Foster FS. Ultrasound biomicroscopy in plateau iris syndrome. *Am J Ophthalmol* 1992;**113**:390-395.
12. Ritch R. Plateau iris is caused by abnormally positioned ciliary processes. *J Glaucoma* 1992;**1**:23-26.
13. Wand M, Pavlin CJ, Foster FS. Plateau iris syndrome: ultrasound biomicroscopic and histological study. *Ophthalmic Surg* 1993;**24**:129.
14. Augsburger JJ, Affel LL, Benarosh DA. Ultrasound biomicroscopy of cystic lesions of the iris and ciliary body. *Trans Am Ophthalmol Soc* 1996;**94**:259-274.
15. Orgül SI, Daicker B, Büchi ER. The diameter of the ciliary sulcus: a morphometric study. *Graefes Arch Clin Exp Ophthalmol* 1993;**231**:487-490.
16. Lowe RF, Ritch R. Angle-closure glaucoma: Clinical types. In: Ritch R,



- Shields MB, Krupin T, eds. The Glaucomas. St. Louis: CV Mosby Co, 1989:839-853.
17. Ritch R. The pilocarpine paradox. *J Glaucoma* 1996;**5**:225-227.
 18. Abramson DH, Franzen LA, Coleman DJ. Pilocarpine in the presbyope: Demonstration of an effect on the anterior chamber and lens thickness. *Arch Ophthalmol* 1973;**89**:100-102.
 19. Ritch R. Argon laser treatment for medically unresponsive attacks of angle-closure glaucoma. *Am J Ophthalmol* 1982;**94**:197.
 20. Bhargava SK, Leighton DA, Phillips CI. Early angle closure glaucoma: distribution of iridotrabecular contact and response to pilocarpine. *Arch Ophthalmol* 1973;**89**:369.
 21. Mapstone R. Partial angle closure. *Br J Ophthalmol* 1977;**61**:525.
 22. Lowe RF. Primary creeping angle-closure glaucoma. *Br J Ophthalmol* 1964;**48**:544.
 23. Gorin G. Angle closure glaucoma induced by miotics. *Am J Ophthalmol* 1966;**62**:1063-1066.
 24. Levene RZ. A new concept of malignant glaucoma. *Arch Ophthalmol* 1972;**87**:497-506.
 25. Merritt JC. Malignant glaucoma induced by miotics postoperatively in open-angle glaucoma. *Arch Ophthalmol* 1977;**95**:1988-1990.
 26. Rieser JC, Schwartz B. Miotic induced malignant glaucoma. *Arch Ophthalmol* 1972;**87**:706-708.
 27. Kramer P, Ritch R. The treatment of angle-closure glaucoma revisited [editorial]. *Ann Ophthalmol* 1984;**16**:1101-1103.
 28. Ritch R, Solomon IS. Laser treatment of glaucoma. In: L'Esperance FAJ, ed. Ophthalmic lasers. 3rd ed. St Louis: CV Mosby Co, 1989:650-748.
 29. Ritch R, Liebmann JM. Argon laser peripheral iridoplasty: a review. *Ophthalmic Surg Lasers* 1996;**27**:289-300.
 30. Lam DSC, Lai JSM, Tham CCY. Immediate argon laser peripheral iridoplasty (ALPI) as treatment for acute attack of primary angle-closure glaucoma (PACG): a preliminary study. *Ophthalmology* 1998;**105**:2231-2236.
 31. Chew P, Chee C, Lim A. Laser treatment of severe acute angle-closure glaucoma in dark Asian irides: the role of iridoplasty. *Lasers Light Ophthalmol* 1991;**4**:41-42.
 32. Chew PTK, Yeo LMW. Argon laser iridoplasty in chronic angle-closure glaucoma. *Int Ophthalmol* 1995;**19**:67-70.
 33. Fu YA, Liaw ZC. Argon laser goniotomy with trabeculoplasty in the treatment of chronic angle-closure glaucoma. *Trans Soc Ophthalmol Sin* 1986;**25**:481-485.
 34. Lim AS, Tan A, Chew P, et al. Laser iridoplasty in the treatment of severe acute angle closure glaucoma. *Int Ophthalmol* 1993;**17**:33-36.
 35. Agarwal HC, Kumar R, Kalra VK, Sood NN. Argon laser iridoplasty: a primary mode of therapy in primary angle-closure glaucoma. *Indian J Ophthalmol* 1991;**39**:87-90.
 36. Lowe RF. Primary angle-closure glaucoma investigations after surgery for pupillary block. *Am J Ophthalmol* 1964;**57**:931.
 37. Gieser D, Wilensky J. Laser iridectomy in the management of chronic angle-closure glaucoma. *Am J Ophthalmol* 1984;**98**:446.
 38. Ritch R. The treatment of chronic angle-closure glaucoma [editorial]. *Ann Ophthalmol* 1981;**13**:21-23.
 39. Ritch R. Techniques of argon laser iridectomy and iridoplasty. Palo Alto: Coherent Medical Press, 1983.
 40. Ritch R, Liebmann J, Solomon IS. Laser iridectomy and iridoplasty. In: Ritch R, Shields MB, Krupin T, eds. The Glaucomas. St. Louis: CV Mosby Co, 1989:581-603.
 41. Ritch R, Solomon LD. Argon laser peripheral iridoplasty for angle-closure glaucoma in siblings with Weill-Marchesani syndrome. *J Glaucoma* 1992;**1**:243-247.
 42. Shirakashi M, Iwata K, Nakayama T. Argon laser trabeculoplasty for chronic angle-closure glaucoma uncontrolled by iridotomy. *Acta Ophthalmol* 1989;**67**:265-270.
 43. Wishart PK, Nagasubramanian S, Hitchings RA. Argon laser trabeculoplasty in narrow angle glaucoma. *Eye* 1987;**1**:567.
 44. Eltz H, Gloor B. Trabeculectomy in cases of angle-closure glaucoma — successes and failures. *Klin Mbl Augenheilkd* 1980;**177**:556.
 45. Tanihara H, Nishiwaki K, Nagata M. Surgical results and complications of goniosynechialysis. *Graefes Arch Clin Exp Ophthalmol* 1992;**230**:309-313.
 46. Tanihara H, Nagata M. Argon-laser goniotomy following goniosynechialysis. *Graefes Arch Clin Exp Ophthalmol* 1991;**229**:505-507.
 47. Sharpe ED, Thomas JV, Simmons RJ. Goniosynechialysis. In: Thomas JV, Belcher CDI, Simmons RJ, eds. Glaucoma Surgery. St. Louis: CV Mosby, 1992.
 48. Nagata M, Nezu N. Goniosynechialysis as a new treatment for chronic angle-closure glaucoma. *Jpn J Clin Ophthalmol* 1985;**39**:707-710.
 49. Campbell DG, Vela A. Modern goniosynechialysis for the treatment of synechial angle-closure glaucoma. *Ophthalmology* 1984;**91**:1052-1060.
 50. Ando H, Kitagawa K, Ogino N. Results of goniosynechialysis for synechial angle-closure glaucoma after pupillary block. *Folia Ophthalmol Jpn* 1990;**41**:883-886.

