Argon Laser Peripheral Iridoplasty

Robert Ritch, MD; Jeffrey M. Liebmann, MD

Argon laser peripheral iridoplasty (ALPI) is a simple and an effective means of opening an appositionally closed angle in situations in which laser iridotomy either cannot be performed or does not physically eliminate appositional angle closure because mechanisms other than pupillary block are present. The procedure consists of placing contraction burns (long duration, low power, and large spot size) in the extreme iris periphery to contract the iris stroma between the site of the burn and the angle, physically pulling open the angle.[1-5] ALPI is extremely useful in reversing an attack of angle-closure glaucoma when medical treatment fails.[2,6]

Krasnov[7] first attempted to use laser energy placed near the iris root to separate iris and trabecular meshwork. The initial treatment procedure encompassed only 90° of the angle. The laser parameters used in these early attempts were more like penetrating burns than the slow contraction burns that later proved optimal, and were often unsuccessful because of insufficient retraction of the iris away from the meshwork. Kimbrough et al.[8] described a technique for direct treatment of 360° of the peripheral iris through a gonioscopy lens and termed the procedure gonioplasty. Their work served as the conceptual basis for the modern procedure.

From the Departments of Ophthalmology, The New York Eye and Ear Infirmary, New York, and the New York Medical College, Valhalla, NY.

Accepted for publication February 16, 1996.
Request reprints from Robert Ritch, MD, Glaucoma Service, The New York Eye and Ear Infirmary, 310 East 14th Street, New York, NY 10003.

Although ALPI is a simple procedure to perform, there are important aspects of technique that must be followed for a successful result. The diagnostic indications for ALPI require the ability to differentiate subtle gonioscopic findings, and the examiner must be proficient at indentation gonioscopy.[9,10] It is necessary to be familiar with the anatomic causes of angle-closure glaucoma and the means of diagnosing these clinically in order to understand the appropriate indications for the procedure.

THE ANATOMIC BASIS OF ANGLE-CLOSURE GLAUCOMA

Classification

Angle-closure glaucoma is an anatomic disorder. It comprises a final common pathway of several entities characterized by abnormal relationships of anterior segment structures. The initial step in this pathway is iris apposition to the trabecular meshwork, which can be caused by one abnormality or a combination of abnormalities in the relative or absolute sizes or positions of anterior segment structures or abnormal forces in the posterior segment that alter the anatomy of the anterior segment.[11] Classification of angle closure by the anatomic level of the cause of the block, defined by the structure producing the "forces" leading to the block, facilitates understanding of the various mechanisms, and appropriate treatment in any particular case becomes an exercise in deductive logic. We have defined four levels of block, from anterior to posterior.[11] Each level of block may have a component of each of the levels preceding it. The appropriate treat-

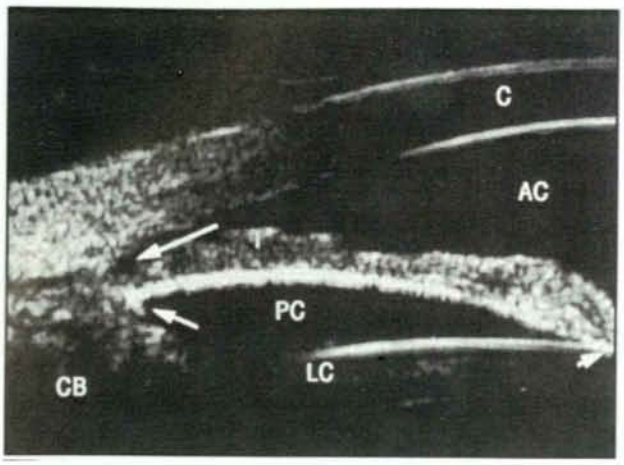


Figure 1. Ultrasound biomicrograph of an eye with relative pupillary block. The iridocorneal angle is almost closed as a result of iris bombe caused by increased pressure in the posterior chamber (PC). The ciliary sulcus (short arrow) is normal. The arrowhead indicates the site of iridolenticular apposition. Long arrow indicates angle recess. AC = anterior chamber; C = cornea; I = iris; LC = lens capsule; CB = ciliary body.

ment becomes more complex for each level of block, as each level may also require the treatments for lower levels of block.

Relative pupillary block is the underlying mechanism in approximately 90% of patients with angle closure. The remainder have either a mechanism or a combination of mechanisms other than or in addition to pupillary block. Only in the last decade have these other mechanisms become well recognized. Some can be made worse by routine treatment of angle closure, particularly when miotics are used for patients with intumescent or anteriorly subluxed lenses or malignant glaucoma.

Level 1—Block Originating at the Iris

In pupillary block, flow of aqueous from the posterior chamber to the anterior chamber is impeded between the anterior surface of the lens and the posterior surface of the iris. Aqueous pressure in the posterior chamber becomes higher than that in the anterior chamber, causing anterior iris bowing, narrowing of the angle, and acute or chronic angle-closure glaucoma (Fig. 1). The anterior segment structures and their anatomic relationships appear otherwise normal.

During indentation gonioscopy, pressure on the cornea forces aqueous humor into the angle, widening it to permit viewing over the iris convexity. The presence and extent of synechial closure and the depth of the angle can be determined. In pupillary block, there

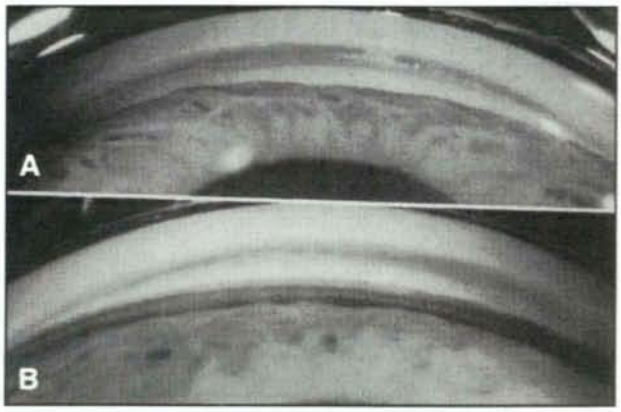


Figure 2. Indentation gonioscopy in an eye with pupillary block. (A) Before indentation. (B) After indentation.

is only aqueous humor in the posterior chamber to offer resistance to indentation gonioscopy, and the angle opens quite easily (Fig. 2). Laser iridotomy is the definitive treatment for pupillary block, allowing aqueous pressures in the anterior and posterior chambers to equalize and allowing the iris to assume a planar configuration.

Relative pupillary block is typically associated with hyperopia, although it may occur in eyes with any type of refractive error.[12,13] The anterior chamber depth and volume are smaller in hyperopes.[14] With age, the anterior chamber depth decreases[14-18] and lens thickness increases, resulting in forward movement of the anterior lens surface.[13,19-22] Compared with normal eyes, eyes with relative pupillary block have a smaller corneal diameter,[19,20] smaller radius of corneal curvature (steeper cornea),[13,19,20,23] shallower anterior chamber,[13,19,24] thicker lens,[13,19,20] smaller radius of anterior lens curvature (steeper lens surface),[13,25] more anterior lens position,[13,20] and shorter axial length.[13,19,20]

Level 2—Block Originating at the Ciliary Body

A large or anteriorly positioned ciliary body can maintain the iris root in proximity to the trabecular meshwork, creating a configuration known as plateau iris (Fig. 3).[26-29] On gonioscopy, the iris root angulates forward and then centrally. The iris root may be short and inserted anteriorly on the ciliary face, producing a shallow, narrow angle with a sharp drop-off of the peripheral iris. Before iridotomy, the anterior chamber is usually of medium depth and the iris surface mildly convex. After iridotomy, the iris surface is flat, although the angle remains narrow or appositionally closed.

When indentation gonioscopy is performed in

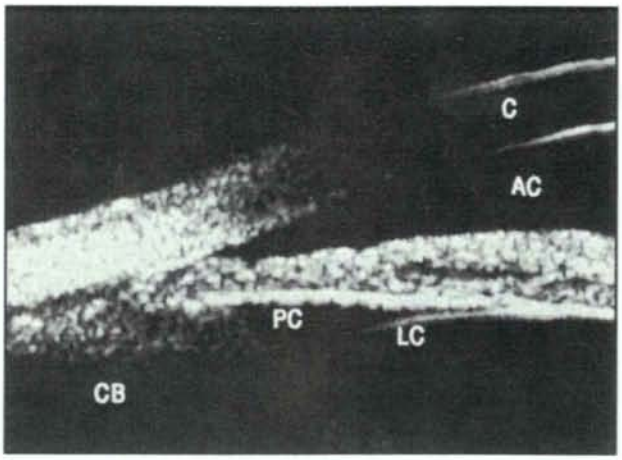


Figure 3. Ultrasound biomicrograph of an eye with plateau iris that has already undergone laser iridotomy. The iris surface is flat and the chamber appears normally deep. The iris root is thick and the entire periphery of the iris is supported by large and anteriorly positioned ciliary processes. (The abbreviations are explained in Figure 1's legend.)

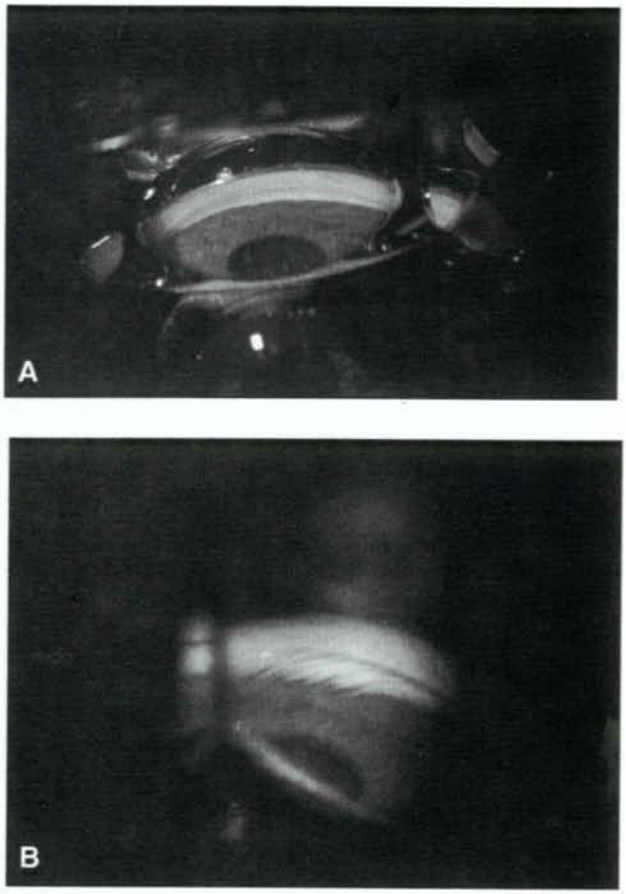


Figure 4. Typical gonioscopic appearance of plateau iris. (A) Before indentation, the angle is closed to mid-trabecular meshwork. The iris assumes a characteristically flat approach to the angle. (B) With indentation, the deepest displacement of the iris occurs at the lens equator.

such an eye, the ciliary processes prevent posterior movement of the peripheral iris. As a result, a sinuous configuration occurs (Σ sign), in which the iris follows the curvature of the lens, reaches its deepest point at the lens equator, and then rises again over the ciliary processes before dropping peripherally. Much more force is needed during gonioscopy to open the angle than in pupillary block because the ciliary processes must be displaced, and the angle does not open as widely (Fig. 4).

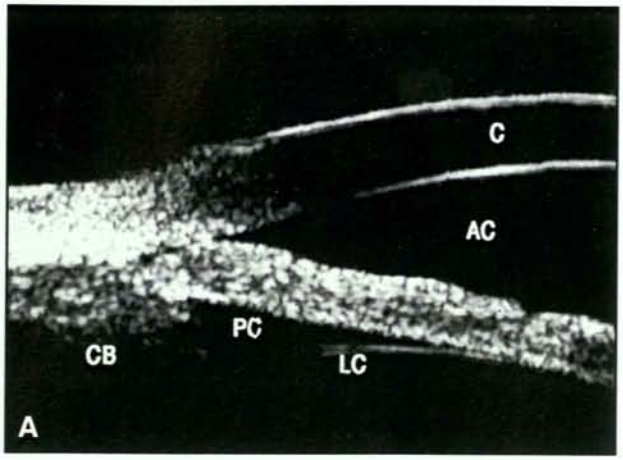
Plateau iris *syndrome* refers to the development of angle closure, either spontaneously or after pupillary dilation, in an eye with plateau iris configuration despite the presence of a patent iridectomy or iridotomy. Acute angle-closure glaucoma may develop in some patients.[30-33]

The extent or the "height" to which the plateau rises determines whether the angle will close completely or only partially (Fig. 5).[34] In the *complete* plateau iris syndrome, the angle closes to the upper meshwork or Schwalbe's line, blocking aqueous outflow and leading to a rise in intraocular pressure (IOP) (Fig. 9). This situation is far less common than the *incomplete* syndrome, in which the angle closes only partially, leaving the upper portion of the filtering meshwork open, so that IOP will not rise. However, peripheral anterior synechiae (PAS) can develop in these eyes up to years after a successful iridotomy initially produces what appears to be a widely opened angle. In addition, the angle can narrow further with age as a result of enlargement of the lens, so that an angle with plateau configuration that does not close after iridotomy may do so some years later. Periodic gonioscopy is required. Continued appositional angle closure in the presence of a patent iridotomy is an indication for ALPI.

Treatment must be targeted at the cause of angle closure (in this case, the ciliary body and iris root). If pupillary block either is not a component mechanism of the angle closure or has been eliminated by iridotomy, it is necessary to find a way to eliminate the physical blockage of the angle. This is accomplished by ALPI, which compresses the iris root and creates a space where none was before (Fig. 6).

Level 3—Block Originating at the Lens

Swelling of the lens may precipitate acute angle-closure glaucoma (phacomorphic glaucoma) because of the lens' pressing against the iris and ciliary body



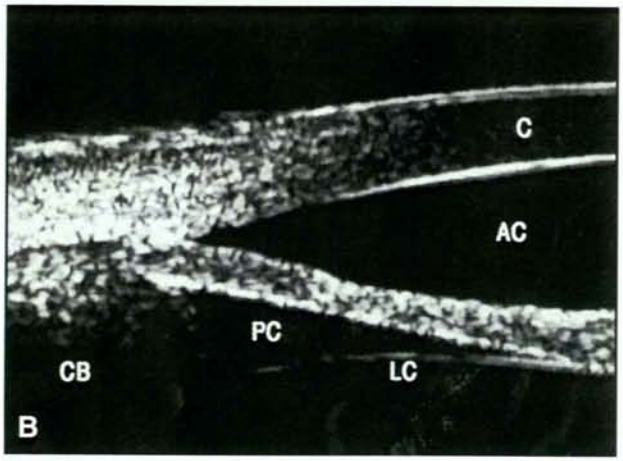
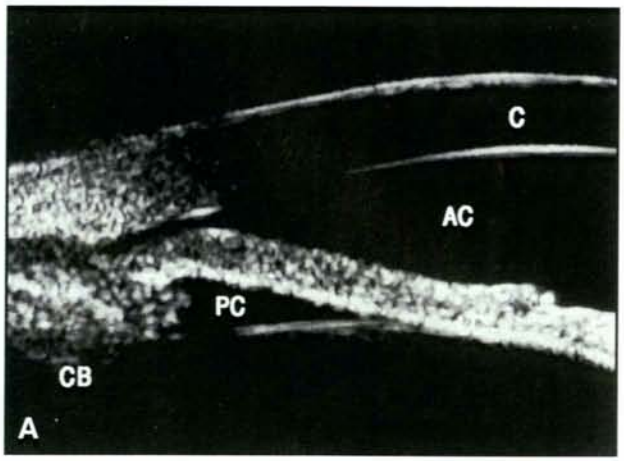


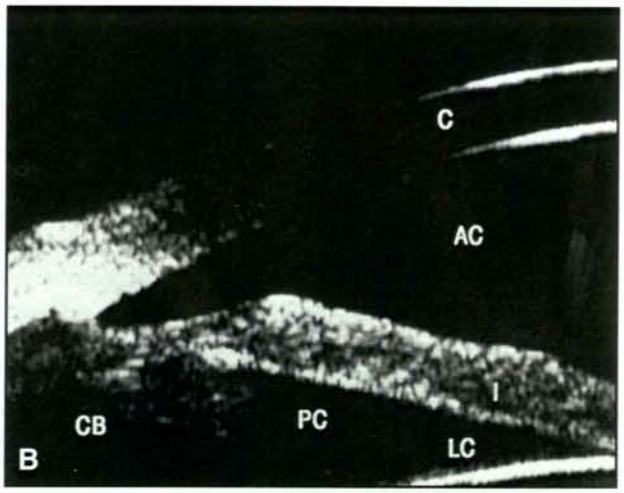
Figure 5. Ultrasound biomicroscopy of (A) an eye with a high plateau and (B) an eye with a mid-level plateau. (The abbreviations are explained in Figure 1's legend.)

and forcing them anteriorly (Fig. 7). Phacomorphic glaucoma is often unresponsive to medical therapy, and paradoxical reactions to pilocarpine,[35] which increases axial lens thickness and causes anterior lens movement, [36] further shallowing the anterior chamber,[37] are common.

ALPI is effective in breaking attacks of phacomorphic angle closure.[2] Very often, the eye is severely inflamed, as these patients have usually been referred after being treated unsuccessfully for a few days. Breaking the attack with ALPI allows 2 to 3 weeks for the inflammation and folds in Descemet's membrane to clear, permitting cataract extraction under conditions much closer to ideal. Any element of pupillary block is treated as soon as possible (usually within 2 to 3 days) after breaking the attack.

The situation is not so simple in cases of anterior lens subluxation caused by trauma or such hereditary disorders as Weill–Marchesani syndrome. In these





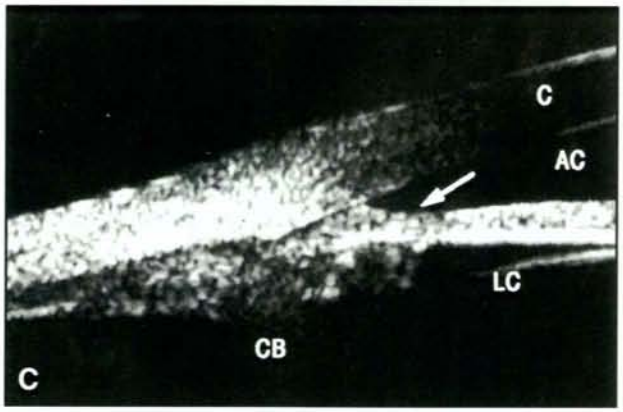


Figure 6. (A) Ultrasound biomicroscopic image of an eye with plateau iris. (B) A similar eye after argon laser peripheral iridoplasty. Note the compression of the iris root, creating a space between it and the trabecular meshwork. (C) A burn placed too centrally (arrow); there is no effect on the angle. (The abbreviations are explained in Figure 1's legend.)

cases, ALPI is less successful because the pressure of the normal-sized lens against the iris continues, with or without an iridotomy, as long as the underlying

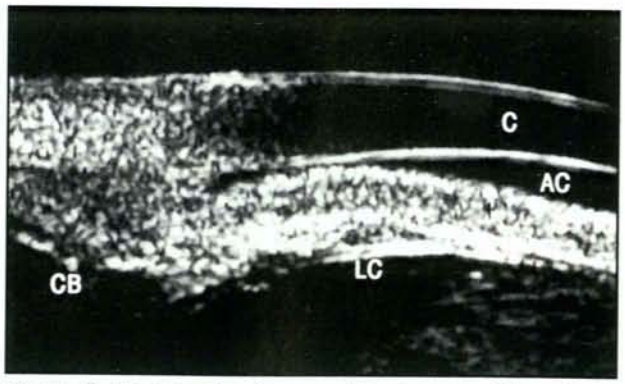


Figure 7. An eye with phacomorphic glaucoma. The anterior chamber is extremely shallow and the lens wedges the iris into the angle and against the cornea. (The abbreviations are explained in Figure 1's legend.)

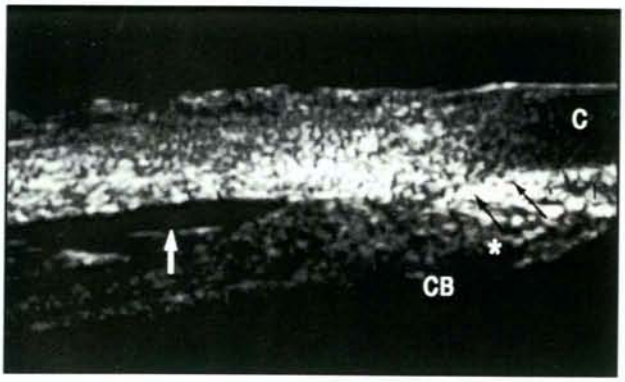


Figure 8. Ultrasound biomicrograph of an eye with malignant glaucoma. Anterior rotation of the ciliary processes (star) has forced the peripheral iris against the trabecular meshwork (black arrows). A shallow supraciliary effusion is present (white arrow). (The abbreviations are explained in Figure 1's legend.)

cause is present. Cycloplegics are useful if the zonules are intact, but these may not always be so.[38] A more complete discussion of this subject can be found elsewhere.[39,40] If not treated in time, forward lens movement can lead to malignant glaucoma, as discussed below.

Treatment must be oriented at the level of the lens. Lens removal is indicated for intumescent cataracts, but is prone to complications if performed during an acute attack of angle-closure glaucoma. ALPI is virtually always successful at breaking the attack, opening the angle enough for IOP to normalize, and providing time (usually about 2 weeks) for the cornea and anterior chamber to clear completely.

Level 4—Block Originating Posterior to the Lens

Also known as malignant (ciliary block) glaucoma, angle closure caused by forces posterior to the lens that push the lens–iris diaphragm forward presents the greatest diagnostic and treatment challenge of the angle-closure glaucomas. Analogous to pupillary block, in which the angle is occluded by iris because of a pressure differential between the posterior and anterior chambers, in ciliary block, a pressure differential is created between the vitreous and aqueous compartments by aqueous misdirection into the vitreous.

Malignant glaucoma, itself a final common pathway, can result from numerous causes and is a multifactorial disease in which the following components may play varying roles: (1) previous acute or chronic angle-closure glaucoma, (2) shallowness of the anterior chamber, (3) forward movement of the lens, (4) pupillary block by the lens or vitreous, (5) slackness of the zonules, (6) anterior rotation and/or swelling of the ciliary body, (7) thickening of the anterior hyaloid membrane, (8) expansion of the vitreous, and (9) posterior aqueous displacement into or behind the vitreous.[41-45]

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, which in turn causes direct angle closure by physically pushing the iris against the trabecular meshwork (Fig. 8).[46] Ultrasound biomicroscopy often reveals a shallow supraciliary detachment not evident on routine B-scan examination. This effusion appears to be the cause of the anterior rotation of the ciliary body and the forward movement of the lens–iris diaphragm. Some of the disorders that can lead to this picture include drug sensitivity (e.g., sulfonamides); angle closure after panretinal photocoagulation, central retinal vein occlusion, or scleral buckling procedures; uveal effusion from posterior segment inflammation; ciliary body swelling, inflammation, or cysts; and posterior segment tumors.

The effusions in many of these conditions, such as angle closure after panretinal photocoagulation (PRP) or scleral buckling, are self-limited, but treatment is indicated to prevent PAS formation and to lower IOP. A component of pupillary block is often present and the opposite angle often narrow, and if the cornea is clear, laser iridotomy can be performed. If appositional closure remains after iridotomy or if the cornea is not clear, ALPI again is almost always successful at opening the angle.

Approach to Acute Angle-Closure Glaucoma

Copious miotic treatment continues to be com-

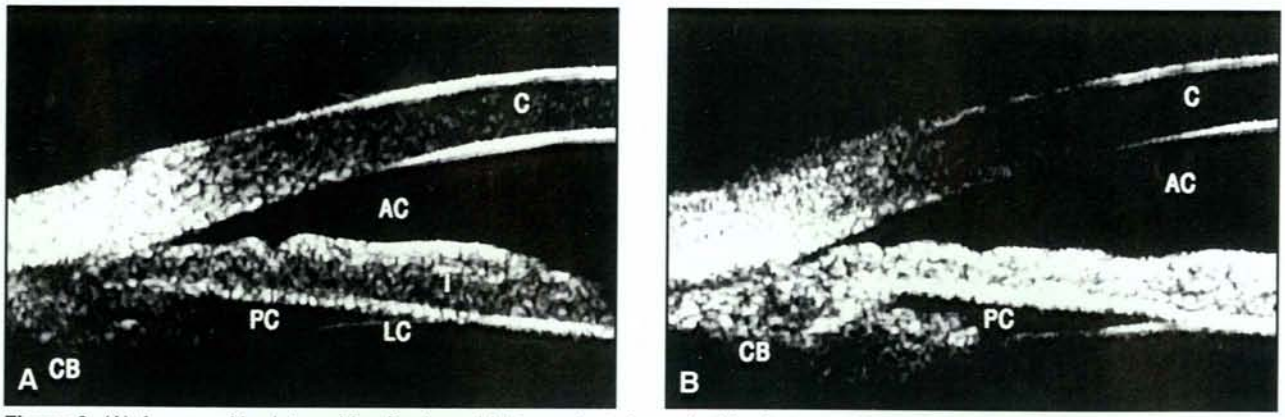


Figure 9. (A) An eye with plateau iris after laser iridotomy (not shown in this photograph) scanned with room lights on. The anterior chamber is moderately deep, the iris and iris root are comparatively thick, and the iris surface is planar. The ciliary processes are positioned anteriorly and the ciliary sulcus, although present, is minimally defined. The approach to the angle is relatively wide until the point at which the iris root angulates posteriorly, where the angle suddenly becomes extremely narrow. (B) The same eye scanned with room lights out. The pupil has dilated and the angle has closed. Argon laser peripheral iridoplasty can open the angle in such an eye by compressing and thinning the peripheral iris stroma. (The abbreviations are explained in Figure 1's legend.)

mon for acute angle-closure glaucoma. When IOP is more than 50 mm Hg, the pupil becomes unresponsive to miotics because of ischemia and paralysis of the iris sphincter. Pilocarpine may not only be ineffective, it may also paradoxically worsen the situation.[2,47-49] Miotics may increase the pupillary block by causing forward motion of the lens–iris diaphragm, and overtreatment with pilocarpine can severely exacerbate attacks of angle-closure glaucoma that are unresponsive to initial medical treatment. The attacks that respond paradoxically to pilocarpine are those with underlying level 3 or level 4 block. The following is our approach to a patient with acute angle closure.[50]

1. Examination of the affected eye and fellow eye with attention to central and peripheral anterior chamber depth and shape of the peripheral iris.

2. Administration of an oral hyperosmotic agent and, if desired, apraclonidine, a beta-blocker, and a carbonic anhydrase inhibitor.

3. Placement of the patient in the supine position to permit the lens to fall backward, if possible, with vitreous dehydration.

4. Reassessment of ocular findings after 1 hour. IOP is usually decreased, but the angle usually remains appositionally closed. One drop of 4% pilocarpine is given and the patient is reexamined 30 minutes later.

a. If the 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 the IOP is unchanged or elevated and the angle remains closed, level 3 or 4 block should be suspected, further pilocarpine should be withheld, and the attack should be broken by ALPI.[2,51,52]

We have performed ALPI in nearly 100 attacks of angle-closure glaucoma unresponsive to medical therapy, even after several days. All but one eye, which had total synechial closure, responded with normalization of IOP and opening of the angle. ALPI 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. Ciliary hyposecretion may help the situation. The alternative of waiting and prolonging medical therapy for several days seriously increases the possibility of irreversible damage to the iris, lens, drainage pathways, and optic nerve head.[53]

INDICATIONS FOR ALPI

Medically Unbreakable Attacks of Angle-Closure Glaucoma

An attack of angle-closure glaucoma that is unresponsive to medical therapy and in which corneal edema, a shallow anterior chamber, or marked inflammation precludes laser iridotomy, or that is unresponsive to successful iridotomy, may be broken with ALPI.[2,6,54,55]

During an attack, the iris is directly apposed to the trabecular meshwork. If PAS have not formed between

the iris and the trabecular meshwork, a contraction burn placed at the periphery of the iris can shrink the iris sufficiently to pull it away from the meshwork. Although ALPI has been reported to break PAS,[56] we have been unable to accomplish this.

Circumferential treatment of the iris opens the angle in those areas in which there are no PAS. All published series have reported virtually 100% success. In a prospective study of 10 eyes with medically unbreakable attacks lasting 2 to 5 days, the mean prelaser IOP was 54.9 mm Hg and 2 to 4 hours postlaser the IOP was 18.5 mm Hg.[55] Even when extensive PAS are present, the IOP is usually normalized within an hour or two, perhaps because of associated secretory hypotony. The effect lasts long enough for the cornea and anterior chamber to clear so that iridotomy can be performed. In cases in which an intumescent lens is responsible for the angle-closure attack, cataract extraction can be postponed until the intraocular inflammation has sufficiently resolved.

Plateau Iris Syndrome (Level 2)

In this condition, discussed above, the angle remains appositionally closed or occludable following laser iridotomy because of abnormally anteriorly positioned ciliary processes.[27,28]

Angle Closure Related to Size or Position of the Lens (Levels 3 and 4)

Angle closure caused by an enlarged lens or pressure posterior to the lens pushing it forward is not usually responsive to iridotomy, although a component of pupillary block may be present and should be eliminated by iridotomy. In these situations in which the angle remains appositionally closed after laser iridotomy, the apposition can often be partially or entirely eliminated by ALPI.[1,4,57,58] Once the angle has been opened and the IOP reduced, cycloplegics may be given cautiously to ascertain the mechanism of the angle closure.

Adjunct to Laser Trabeculoplasty

If a narrow but open angle results from plateau iris or anglé crowding, ALPI can retract the iris away from the trabecular meshwork.[3] In eyes in which most of the angle is visible but focal areas of narrowing are present because of iris irregularities or intraepithelial cysts, focal contraction burns are sufficient to widen these areas to permit trabeculoplasty burns to be placed at these sites.[59] Argon laser trabeculoplasty (ALT) can be performed immediately after ALPI when focal coagulation alone is necessary. If extensive treatment is necessary, it is probably better to perform the procedures on separate days.

Retinopathy of Prematurity

Angle closure may occur in very young children with retinopathy of prematurity as a result of forward shifting of the lens–iris diaphragm.[60-65] These children do not respond to iridotomy. In young adults with this condition, there appears to be a superimposed element of pupillary block, and iridotomy may be successful.[66,67]

Nanophthalmos

Nanophthalmos is characterized by hyperopia, small corneal diameter, thick sclera, and narrow angles.[68] Patients with nanophthalmos are anatomically predisposed to angle-closure glaucoma as a result of anterior chamber crowding. Angle-closure glaucoma usually appears between the ages of 20 and 50 years. Laser iridotomy is usually unsuccessful or only temporarily successful. Prophylactic iridotomy is not without risk. Bilateral nonrhegmatogenous retinal detachments have been described following laser iridotomy in these patients[69] and may be attributable to worsening of preexisting retinal or choroidal disease.[70] Flattening of the peripheral iris by argon laser was first reported in 1979 by Kimbrough et al.[8] Combined iridotomy and ALPI often brings the angle closure under control.[71] Uveal effusions have been reported after both laser iridotomy[69] and ALT.[72] The risks of surgical intervention include malignant glaucoma, expulsive suprachoroidal hemorrhage, and retinal detachment.[73] Posterior sclerotomy may or may not be successful at preventing uveal effusion.[71,74]

CONTRAINDICATIONS

Advanced Corneal Edema or Opacification

Moderate degrees of corneal edema are not a contraindication to ALPI when it is performed in order to break a medically unresponsive attack of angle-closure glaucoma. Extensive corneal opacification may present difficulties, because higher powers necessary to cause contraction of the iris may injure the cornea as well. Glycerin may help clear the cornea temporarily.

Flat Anterior Chamber

Corneal endothelial burns are generated by heat-

ing of the aqueous humor at the site of the laser burn and its reflux toward the corneal endothelium. If the iris is apposed to the cornea, any attempt at photocoagulation will result in damage to the corneal endothelium. If the anterior chamber is very shallow, laser applications should be timed enough apart so that heat generated can dissipate.

Synechial Angle Closure

ALPI will not relieve synechial angle closure and should not be used for this purpose, especially in eyes with uveitis, neovascular glaucoma, or iridocorneal endothelial syndrome.

TECHNIQUE

Pretreatment Measures

ALPI is performed on an outpatient basis using topical anesthesia and an Abraham lens. Apraclonidine is administered about 45 minutes prior to treatment. Shortly before treatment, 4% pilocarpine is applied topically to stretch the iris maximally from the iris root to the pupillary border. In some eyes, posterior synechiae or iris atrophy prevents constriction. In eyes predisposed to a paradoxical reaction to pilocarpine, the risk is minimized by the timing of the pilocarpine. However, miotics should not be continued following the procedure.

Laser Parameters

The laser is set to produce contraction burns (500-µm spot size, 0.5-second duration, and 200 to 400 mW of power), which pull the surrounding iris tissue forward toward the site of the burn and compact the stroma at the site of the burn. The short-term effect appears to be related to heat shrinkage of collagen, and the long-term effect is secondary to contraction of a fibroblastic membrane in the region of the laser application.[75]

The aiming beam should be directed to the most peripheral portion of the iris possible. Spot placement short of the iris root is ineffective. The patient should look in the same direction as the quadrant of iris being treated. It is useful to allow a thin crescent of the aiming beam to overlap the sclera at the limbus. The surgeon should begin with 200 mW for dark irides and 300 mW for light ones and should adjust the power as necessary to obtain visible stromal contraction. The foot pedal should be pressed for the entire duration of the burn, unless bubble formation and pigment release occur. Contraction is immediate and is usually accompanied by deepening of the peripheral anterior chamber at the site of the burn. If bubble formation occurs or if pigment is released into the anterior chamber, the power should be reduced. Occasionally, in light gray irides, a 200-µm spot size may be more effective in achieving significant stromal contraction.

Twenty to 24 spots are placed over 360°, leaving approximately 2 spot-diameters between each spot and avoiding large visible radial vessels if possible. Although rare, iris necrosis may occur if too many spots are placed too closely together. If this treatment is insufficient, more spots may be given at a later time. The presence of an arcus senilis should be ignored. An extremely shallow anterior chamber and corneal edema, which are relative contraindications to laser iridotomy, do not preclude ALPI.

Other laser settings reported for this type of burn, most commonly 200 µm, 0.1- or 0.2-second duration, and 200 mW of power, often provide insufficient contraction and result in bubble formation or pigment liberation into the anterior chamber. When used through the angled mirror of a gonioscopy lens, these settings are more likely to result in stromal destruction or inadvertent trabecular damage. A few angles have a very sharply defined plateau that, on indentation, forms almost a right angle and takes firm pressure to indent open. This often does not respond well to contraction burns placed with the Abraham lens, and instead requires burns placed through one of the angled mirrors of a Goldmann or Ritch lens directly into the peripheral angle. A 200-µm spot size should be used in this situation.

Postoperative Treatment

Immediately after the procedure, the patient is given a drop of topical steroid and apraclonidine. Gonioscopy should be performed to assess the effect of the procedure. Patients are treated with topical steroids four to six times daily for 3 to 5 days. IOP is monitored postoperatively as after any other anterior segment laser procedure and patients are treated as necessary if a postlaser rise occurs.

Approach to Angle Closure After Elimination of Pupillary Block

Once iridotomy has eliminated any component of pupillary block, the status of the iridocorneal angle should be reassessed by gonioscopy, both immediately after the procedure and after miotics have been dis-

continued. If ALPI was not used to break the attack, the angle may be open, partially open, or closed.

As many as one third of angles without PAS remain narrow after iridotomy, and approximately half of these are capable of closure with mydriasis.[76] Continued appositional angle closure in the presence of a patent iridotomy suggests plateau iris, a large lens, forward lens movement, or malignant glaucoma, and is an indication for ALPI.[4,5] If closure is essentially complete, we perform ALPI and then proceed as below, as cycloplegia at this point may be detrimental. If closure is partial, we discontinue miotics and reexamine the angle 2 days later. If it is still appositionally closed, ALPI should be performed. After this, the eye may be tested with a drop of cyclopentolate. If the anterior chamber deepens and the angle opens, the patient is maintained on cycloplegics if IOP is not adversely affected. If the angle closes because of iris crowding, no further cycloplegics are given.

If extensive PAS are present after ALPI, goniosynechialysis may be performed. In this procedure, PAS are surgically stripped from the angle wall to restore trabecular function; this is successful only if the PAS have been present for less than 1 year.[77] Promising results have been reported in both phakic and pseudophakic eyes.[78-80] It is effective both alone and in conjunction with other surgical procedures.[81] ALPI can be used postoperatively to further flatten the peripheral iris and prevent synechial reattachment.[82]

Complications

Mild postoperative iritis is common and responds to topical steroid treatment, seldom lasting more than a few days. The patient may experience transient ocular irritation.

Because ALPI is often performed on patients with extremely shallow peripheral anterior chambers, ill-defined, diffuse corneal endothelial burns may occur. These may be minimized by placing an initial contraction burn more centrally before placing the first peripheral burn. This first burn will deepen the anterior chamber peripheral to it, allowing the more peripheral burn to be placed with less corneal damage. Each peripheral burn deepens the circumferentially adjacent chamber. We have seen only one case of corneal decompensation following ALPI in a patient with preexisting Fuchs' dystrophy.

Hemorrhage does not occur. A transient rise in IOP can occur as with other anterior segment laser procedures. Lenticular opacification has not occurred with ALPI, and theoretically this problem would be highly unlikely.

The Need for Re-treatment

The duration of the effect of the ALPI depends on the level of block and its resolution. Eyes with plateau iris rarely, if ever, require re-treatment. However, angle closure may recur on the basis of lens enlargement with time. Pressure from the lens against the posterior iris may lead to gradual narrowing of the angle, possibly because of further anterior lens movement, or to stretching of the iris stroma. Re-treatment is most commonly needed in eyes in which the mechanism of the angle closure is the result of forward lens movement, particularly malignant glaucoma. Patients in whom angle closure results from intumescent lenses usually undergo cataract extraction. Patients should be observed gonioscopically at regular intervals and further treatment should be given if necessary.

REFERENCES

1. York K, Ritch R, Szmyd LJ. Argon laser peripheral iridoplasty: indications, techniques and results. *Invest Ophthalmol Vis Sci.* 1984;25(suppl):94.
2. Ritch R. Argon laser treatment for medically unresponsive attacks of angle-closure glaucoma. *Am J Ophthalmol.* 1982;94:197.
3. Ritch R. *Techniques of Argon Laser Iridectomy and Iridoplasty.* Palo Alto, CA: Coherent Medical Press; 1983.
4. Ritch R, Solomon IS. Glaucoma surgery. In: L'Esperance FA, ed. *Ophthalmic Lasers,* 3rd ed. St. Louis, MO: C. V. Mosby; 1989.
5. Ritch R. Argon laser peripheral iridoplasty: an overview. *J Glaucoma.* 1992;1:206–213.
6. 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.
7. Krasnov MM. Q-switched laser iridectomy and Q-switched laser goniopuncture. *Adv Ophthalmol.* 1977; 34:192–196.
8. Kimbrough RL, Trempe CS, Brockburst RJ, Simmons RJ. Angle-closure glaucoma in nanophthalmos. *Am J Ophthalmol.* 1979;88:572.
9. Forbes M. Gonioscopy with corneal indentation: a method for distinguishing between appositional closure and synechial closure. *Arch Ophthalmol.* 1966;76:488–497.
10. Gorin G. Re-evaluation of gonioscopic findings in angle-closure glaucoma: static versus manipulative

gonioscopy. *Am J Ophthalmol.* 1971;71:894.
11. 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.
12. 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;54:161–169.
13. Lowe RF. Primary angle-closure glaucoma: a review of ocular biometry. *Aust J Ophthalmol.* 1977;5:9–17.
14. Fontana SC, Brubaker RF. Volume and depth of the anterior chamber in the normal aging human eye. *Arch Ophthalmol.* 1980;98:1801–1808.
15. Heim M. Photographische Bestimmung der Tiefe und des Volumens der menschlichen Vorderkammer. *Ophthalmologica.* 1941;102:193–220.
16. Weekers R, Delmarcelle Y, Collignon J, Luyckx J. Mesure optique de la profondeur de la chambre antérieure: applications cliniques. *Doc Ophthalmol.* 1973; 34:413–434.
17. Kondo T, Miura M, Imamichi M. Anterior chamber volume in the normal human eye. *Acta Soc Ophthamol Jpn.* 1985;89:1099–1103.
18. Grosvenor T. Reduction in axial length with age: an emmetropizing mechanism for the adult eye? *Am J Optom Physiol Optics.* 1987;64:657–663.
19. Delmarcelle Y, Francois J, Goes F, Collignon–Brach J, Luyckx–Bacus J, Verbracken H. Biometrie oculaire cliniqie (oculometrie). *Bull Soc Belge Ophthalmol.* 1976;172:1–608.
20. Tomlinson A, Leighton DA. Ocular dimensions in the heredity of angle-closure glaucoma. *Br J Ophthalmol.* 1973;57:475–486.
21. Hoffer KJ. Axial dimension of the human cataractous lens. *Arch Ophthalmol.* 1993;111:914–918.
22. Lowe RF. Anterior lens displacement with age. *Br J Ophthalmol.* 1970;54:117–121.
23. Lowe RF, Clark BAJ. Posterior corneal curvature: correlations in normal eyes and in eyes involved with primary angle-closure glaucoma. *Br J Ophthalmol.* 1973;57:475.
24. Lee DA, Brubaker RF, Illstrup DM. Anterior chamber dimensions in patients with narrow angles and angle-closure glaucoma. *Arch Ophthalmol.* 1984;102:46–50.
25. Lowe RF, Clark BAJ. Radius of curvature of the anterior lens surface: correlations in normal eyes and eyes involved with primary angle-closure glaucoma. *Br J Ophthalmol.* 1973;57:471.
26. Tornquist R. Angle-closure glaucoma in an eye with a plateau type of iris. *Acta Ophthalmol.* 1958;36:413.
27. Pavlin CJ, Ritch R, Foster FS. Ultrasound biomicroscopy in plateau iris syndrome. *Am J Ophthalmol.* 1992;113:390–395.
28. Ritch R. Plateau iris is caused by abnormally positioned ciliary processes. *J Glaucoma.* 1992;1:23–26.
29. Wand M, Pavlin CJ, Foster FS. Plateau iris syndrome: ultrasound biomicroscopic and histological study. *Ophthalmic Surg.* 1993;24:129.
30. Godel V, Stein R, Feiler–Ofry V. Angle-closure glaucoma following peripheral iridectomy and mydriasis. *Am J Ophthalmol.* 1968;65:555–560.
31. Lowe RF. Primary angle-closure glaucoma: postoperative acute glaucoma after phenylephrine eye-drops. *Am J Ophthalmol.* 1968;65:552.
32. Lowe RF. Plateau iris. *Aust J Ophthalmol.* 1981;9:71.
33. Wand M, Grant WM, Simmons RJ, Hutchinson BT. Plateau iris syndrome. *Trans Am Acad Ophthalmol Otolarnygol.* 1977;83:122.
34. Lowe RF, Ritch R. Angle-closure glaucoma: clinical types. In: Ritch R, Shields MB, Krupin T, eds. *The Glaucomas,* vol. 2. St. Louis: C. V. Mosby; 1989:839–853.
35. Bleiman B, Schwartz AL. Paradoxical response to pilocarpine. *Arch Ophthalmol.* 1979;97:1305.
36. Abramson DH, Chang S, Coleman DJ, et al. Pilocarpine-induced lens changes: an ultrasonic biometric evaluation of dose response. *Arch Ophthalmol.* 1974;92:464.
37. 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.
38. Ritch R, Solomon LD. Argon laser peripheral iridoplasty for angle-closure glaucoma in siblings with Weill–Marchesani syndrome. *J Glaucoma.* 1992;1: 243–247.
39. Liebmann J, Ritch R. Glaucoma secondary to lens intumescence and dislocation. In: Ritch R, Shields MB, Krupin T, eds. *The Glaucomas.* St. Louis, MO: C. V. Mosby; 1989.
40. Ritch R, Lowe RF, Reyes A. Therapeutic overview of angle-closure glaucoma. In: Ritch R, Shields MB, Krupin T, eds. *The Glaucomas.* St. Louis, MO: C. V. Mosby; 1989:855–864.
41. Dueker D. Ciliary-block glaucoma: differential diagnosis and management. *J Glaucoma.* 1994;3:167–170.
42. Levene RZ. A new concept of malignant glaucoma. *Arch Ophthalmol.* 1972;87:497.

43. Shaffer RN, Hoskins HD Jr. Ciliary block (malignant) glaucoma. *Trans Am Acad Ophthalmol Otolarnygol.* 1978;85:215.
44. Simmons RJ. Malignant glaucoma. *Br J Ophthalmol.* 1972;56:273.
45. Weiss DI, Shaffer RN. Ciliary block (malignant) glaucoma. *Trans Am Acad Ophthalmol Otolarnygol.* 1972;76:450.
46. Phelps CD. Angle-closure glaucoma secondary to ciliary body swelling. *Arch Ophthalmol.* 1974;92:287.
47. Gorin G. Angle-closure glaucoma induced by miotics. *Am J Ophthalmol.* 1966;62:1063.
48. Mapstone R. Closed-angle glaucoma: theoretical considerations. *Br J Ophthalmol.* 1974;58:36–40.
49. Rieser JC, Schwartz B. Miotic induced malignant glaucoma. *Arch Ophthalmol.* 1972;87:706.
50. Kramer P, Ritch R. The treatment of angle-closure glaucoma revisited. *Ann Ophthalmol.* 1984;16:1101–1103. Editorial.
51. Ritch R, Solomon IS. Laser treatment of glaucoma. In: L'Esperance FA Jr, ed. *Ophthalmic Lasers,* 3rd ed., vol. 2. St. Louis, MO: C. V. Mosby; 1989:650–748.
52. Shin DH. Argon laser treatment for relief of medically unresponsive angle-closure glaucoma attacks. *Am J Ophthalmol.* 1982;94:821.
53. David R, Tessler Z, Yassur Y. Long-term outcome of primary acute angle-closure glaucoma. *Br J Ophthalmol.* 1985;69:261.
54. Matai A, Consul S. Argon laser iridoplasty. *Indian J Ophthalmol.* 1987;35:290–292.
55. 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.
56. Wand M. Argon laser gonioplasty for synechial angle closure. *Arch Ophthalmol.* 1992;110:353–367.
57. Burton TC, Folk JC. Laser iris retraction for angle-closure glaucoma after retinal detachment surgery. *Ophthalmology.* 1988;95:742–748.
58. Koster HR, Liebmann JM, Ritch R, Hudock S. Acute angle-closure glaucoma in a patient with acquired immunodeficiency syndrome successfully treated with argon laser peripheral iridoplasty. *Ophthalmic Surg.* 1990;21:501–502.
59. Metz D, Ackerman J, Kanarek I. Laser trabeculoplasty enhancement by argon laser iridotomy and/or iridoplasty. *Ophthalmic Surg.* 1984;15:535. Letter.
60. Cohen J, Alfano JE, Boshes LD, Palmgren C. Clinical evaluation of school age children with retrolental fibroplasia. *Am J Ophthalmol.* 1964;57:41–57.
61. Hittner HM, Rhodes LM, McPherson AR. Anterior segment abnormalities in cicatricial retinopathy of prematurity. *Ophthalmology.* 1979;86:803–816.
62. Pollard ZF. Secondary angle-closure glaucoma in cicatricial retrolental fibroplasia. *Am J Ophthalmol.* 1980;89:651.
63. McCormick AQ, Pratt–Johnson JA. Angle-closure glaucoma in infancy. *Can J Ophthalmol.* 1971;6:38–41.
64. Laws DE, Haslett R, Ashby D, O'Brien C, Clark D. Axial length biometry in infants with retinopathy of prematurity. *Eye.* 1994;8:427–430.
65. Kushner BJ. Ciliary block glaucoma in retinopathy of prematurity. *Arch Ophthalmol.* 1982;100:1078.
66. Ueda N, Ogino N. Angle-closure glaucoma with pupillary block mechanism in cicatricial retinopathy of prematurity. *Ophthalmologica.* 1988;196:15–18.
67. Smith J. Angle-closure glaucoma in adults with cicatricial retinopathy of prematurity. *Arch Ophthamol.* 1984;102:371.
68. O' Grady RB. Nanophthalmos. *Am J Ophthalmol.* 1971;71:1251.
69. Karjalainen K, Laatikainen L, Raitta C. Bilateral nonrhegmatogenous retinal detachment following neodymium-YAG laser iridotomies. *Arch Ophthalmol.* 1986;104:1134.
70. Singh OS, Belcher CD, Simmons RJ. Nanophthalmic eyes and neodymium-YAG laser iridectomies. *Arch Ophthalmol.* 1987;105:455.
71. Jin JC, Anderson DR. Laser and unsutured sclerotomy in nanophthalmos. *Am J Ophthalmol.* 1990;109:575–580.
72. Good WV, Stern WH. Recurrent nanophthalmic uveal effusion syndrome following laser trabeculoplasty. *Am J Ophthalmol.* 1988;106:234–235.
73. Hyams S. *Angle-closure Glaucoma.* Amsterdam: Kugler and Ghedini; 1990.
74. Calhoun FP. The management of glaucoma in nanophthalmos. *Trans Am Ophthalmol Soc.* 1975;73:97.
75. Sassani JW, Ritch R, McCormick S, et al. Histopathology of argon laser peripheral iridoplasty. *Ophthalmic Surg.* 1993;24:740–745.
76. Lowe RF. Primary angle-closure glaucoma investigations after surgery for pupillary block. *Am J Ophthalmol.* 1964;57:931.
77. Campbell DG, Vela A. Modern goniosynechialysis for the treatment of synechial angle-closure glaucoma. *Ophthalmology.* 1984;91:1052–1060.
78. Ando H, Kitagawa K, Ogino N. Results of goniosynechialysis for synechial angle-closure glaucoma after pupillary block. *Folia Ophthalmol Jpn.* 1990;41:883–886.

79. Nagata M, Nezu N. Goniosynechialysis as a new treatment for chronic angle-closure glaucoma. *Jpn J Clin Ophthalmol.* 1985;39:707–710.
80. Tanihara H, Nishiwaki K, Nagata M. Surgical results and complications of goniosynechialysis. *Graefes Arch Clin Exp Ophthalmol.* 1992;230:309–313.
81. Shingleton BJ, Chang MA, Bellows AR, Thomas JV. Surgical goniosynechialysis for angle-closure glaucoma. *Ophthalmology.* 1990;97:551–556.
82. Tanihara H, Nagata M. Argon-laser gonioplasty following goniosynechialysis. *Graefes Arch Clin Exp Ophthalmol.* 1991;229:505–507.