Narrow Angle Glaucoma Laser Procedures

Ryan Kern, O.D., F.A.A.O.
Financial Disclosures:

• None
### Table 7.2 Classification of the Glaucomas Based on Initial Events

<table>
<thead>
<tr>
<th>A.</th>
<th>B.</th>
<th>C.</th>
<th>D.</th>
<th>E.</th>
<th>F.</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Open-angle glaucomas without other known ocular or systemic disorders</td>
<td>1. Chronic open-angle glaucoma</td>
<td>2. Normal-tension glaucoma</td>
<td>B. Angle-closure glaucomas without other known ocular or systemic disorders</td>
<td>1. Pupillary block glaucomas</td>
<td>2. Combined mechanism glaucoma</td>
</tr>
<tr>
<td></td>
<td>c. Fuchs endothelial corneal dystrophy</td>
<td>2. Glaucomas associated with disorders of the retina, choroid, and vitreous</td>
<td>d. Neovascular glaucoma</td>
<td>e. Glaucomas associated with retinal detachment and vitreoretinal abnormalities</td>
<td></td>
</tr>
<tr>
<td>E. Glaucomas associated with intraocular tumors</td>
<td>1. Malignant melanoma</td>
<td>2. Retinoblastoma</td>
<td>F. Glaucomas associated with elevated episcleral venous pressure</td>
<td></td>
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<tr>
<td>F. Glaucomas associated with increased episcleral venous pressure</td>
<td>1. Glaucomas associated with inflammation</td>
<td>a. Glaucomas associated with uveitis</td>
<td>b. Glaucomas associated with keratitis, episcleritis, and scleritis</td>
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<tr>
<td></td>
<td>5. Glaucomas associated with hemorrhage</td>
<td>10. Glaucomas after intraocular surgery</td>
<td>a. Ciliary block (malignant) glaucoma</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. Glaucomas after intraocular surgery</td>
<td></td>
<td></td>
<td></td>
<td>2. Glaucomas associated with uveitis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>a. Glaucomas associated with uveitis</td>
<td>b. Glaucomas associated with keratitis, episcleritis, and scleritis</td>
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<tr>
<td></td>
<td>2. Steroid-induced glaucoma</td>
<td>3. Steroid-induced glaucoma</td>
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<td></td>
<td>5. Glaucomas associated with corneal surgery</td>
<td>10. Glaucomas after intraocular surgery</td>
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<tr>
<td></td>
<td>a. Ciliary block (malignant) glaucoma</td>
<td>2. Glaucomas associated with uveitis</td>
<td></td>
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<tr>
<td></td>
<td>b. Glaucomas in pseudophakia and aphakia</td>
<td>3. Steroid-induced glaucoma</td>
<td></td>
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</tr>
<tr>
<td></td>
<td>c. Epithelial, fibrous, and endothelial proliferation</td>
<td>4. Glaucomas associated with corneal surgery</td>
<td></td>
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<tr>
<td></td>
<td>d. Glaucomas associated with vitreous surgery</td>
<td>e. Glaucomas associated with vitreoretinal surgery</td>
<td></td>
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</tr>
</tbody>
</table>

### Table 7.3 Classification of the Glaucomas Based on Mechanisms of Outflow Obstruction

#### OPEN-ANGLE GLAUCOMA MECHANISMS

<table>
<thead>
<tr>
<th>A.</th>
<th>B.</th>
<th>C.</th>
<th>D.</th>
<th>E.</th>
<th>F.</th>
</tr>
</thead>
<tbody>
<tr>
<td>A.</td>
<td>Metatubular (membrane overgrowth)</td>
<td>1. Fibrovascular membrane (neovascular glaucoma)</td>
<td>2. Endothelial layer, often with Descemet-like membrane</td>
<td></td>
<td></td>
</tr>
<tr>
<td>B.</td>
<td>a. Iridocorneal endothelial syndrome</td>
<td>b. Posterior polymorphous dystrophy</td>
<td>C. Penetrating and nonpenetrating trauma</td>
<td></td>
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</tr>
<tr>
<td>C.</td>
<td>Epithelial downgrowth</td>
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<tr>
<td>D.</td>
<td>Fibrous ingrowth</td>
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<tr>
<td>E.</td>
<td>Inflammatory membrane</td>
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<tr>
<td>F.</td>
<td>a. Fuchs heterochromic iridocyclitis</td>
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<tr>
<td>G.</td>
<td>b. Luxuriant intestinal keratitits</td>
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</tbody>
</table>

#### ANGLE-CLOSURE GLAUCOMA MECHANISMS

<table>
<thead>
<tr>
<th>A.</th>
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<th>D.</th>
<th>E.</th>
<th>F.</th>
</tr>
</thead>
<tbody>
<tr>
<td>A.</td>
<td>Anterior (“pulling”) mechanism</td>
<td>1. Contracture of membranes</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>B.</td>
<td>a. Neovascular glaucoma</td>
<td>b. Iridocorneal endothelial syndrome</td>
<td></td>
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<tr>
<td>C.</td>
<td>c. Posterior polymorphous dystrophy</td>
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<tr>
<td>D.</td>
<td>d. Penetrating and nonpenetrating trauma</td>
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<tr>
<td>E.</td>
<td>e. Epithelial downgrowth</td>
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<td>F.</td>
<td>f. Stromal contraction</td>
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</tbody>
</table>

### Developmental Anomalies of the Anterior Chamber Angle

<table>
<thead>
<tr>
<th>A.</th>
<th>B.</th>
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<th>E.</th>
<th>F.</th>
</tr>
</thead>
<tbody>
<tr>
<td>A.</td>
<td>High insertion of anterior uvea</td>
<td></td>
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</tr>
<tr>
<td>B.</td>
<td>1. Congenital (“infantile”) glaucoma</td>
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<tr>
<td>C.</td>
<td>2. Juvenile glaucoma</td>
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<tr>
<td>D.</td>
<td>3. Glaucomas associated with other developmental anomalies</td>
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<tr>
<td>E.</td>
<td>B. Incomplete development of trabecular meshwork</td>
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<tr>
<td>F.</td>
<td>1. Axenfeld-Rieger syndrome</td>
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</tr>
<tr>
<td>G.</td>
<td>2. Peters anomaly</td>
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<tr>
<td>H.</td>
<td>3. Glaucomas associated with other developmental anomalies</td>
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</tr>
<tr>
<td>I.</td>
<td>C. Iridocorneal adhesions</td>
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</tr>
<tr>
<td>J.</td>
<td>1. Broad strands (Axenfeld-Rieger syndrome)</td>
<td></td>
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</tr>
<tr>
<td>K.</td>
<td>2. Fine strands that contract to close angle (aniridia)</td>
<td></td>
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</tr>
</tbody>
</table>
Laser Peripheral Iridotomy

• Acute Primary Angle Closure
• Subacute, Chronic, and Combined Mechanisms of Angle Closure?
• Primary Angle Closure Suspect
• Some Forms of Secondary Angle Closure?
• Pigmentary Dispersion?
• Aqueous Misdirection?
• Silicone Oil Pupillary Block?
Glaucoma Classification

- “Primary” angle closure glaucoma classically refers to closure of the anterior chamber angle by apposition of the iris to the trabecular meshwork, or more anterior, inciting a spike in IOP which results in glaucomatous optic nerve damage.
- This “primary” closure is classically related to “normal” anatomy resulting in pupillary block.
- Normal anatomy, meaning there is no disease process or dysgenesis of the eye.
- Non-pathologic refractive error is considered “normal anatomy”, example is a typical hyperopic patient with a smaller axial length and therefore a short, more congested eye/anterior chamber.
Glaucoma Classification

• “Secondary” angle closure classically refers to angle closure glaucoma of a disease or dysgenesis process

• Authors argue that all glaucoma is secondary to an etiology, and therefore it may be more appropriate to refer to angle closure glaucoma in relation to initial event or exact mechanism of obstruction
PHYSIOLOGICAL PUPILLARY BLOCK

1. Iris has large arc of contact with anterior surface of lens

2. Resistance to aqueous flow from posterior to anterior chamber (*relative pupil block*)

3. Pupil dilates, peripheral iris becomes more flaccid and pushed anteriorly

4. Iris lies against trabecular meshwork → impede aqueous humor drainage → ↑ IOP
REVERSE PUPILLARY BLOCK IN PIGMENTARY GLAUCOMA
Pupillary Block

• Most common form of angle closure glaucoma
• Initiating event is the result of increased resistance to flow of aqueous humor between the pupillary portion of the iris and the anterior lens surface
• Associated with mid-dilation of the pupil, where it is proposed to have greatest potential for iris/lens contact during normal function
• Functional block causes increased fluid pressure in the posterior chamber resulting in forward shift of the iris
• Anterior movement of peripheral iris causes a bowing and contact of the iris anterior to or with the trabecular meshwork
• This bow and contact is termed iris bombe
Pupillary Block

• Related to:
  • A thicker/more anatomically placed lens
  • A smaller diameter, shorter posterior curvature cornea
  • A shorter axial length

• Possible that a mid-dilated pupil, hypothesized to be anywhere from 3.5-6mm in diameter, may potentiate highest risk of pupillary block induced angle closure in a patient with “normal” anatomy
The 4 Forms of Angle Closure – Basis of Symptoms and Clinical Findings

• Acute angle closure glaucoma

• Sub-acute angle closure glaucoma

• Chronic angle closure glaucoma

• Combined mechanism glaucoma
Symptoms and Signs of Angle Closures

- Pain/deep ache typically following trigeminal distribution (eye, facial, jaw pain)
- Mid-dilated pupil
- Blurred vision from corneal edema secondary to elevated IOP causing endothelial decompensation, stromal stretch, and epithelial microcystic edema
- Visual halos from presumed epithelial microcystic edema
- Red eye from ciliary flush and conjunctival congestion
- May also have nausea, vomiting
Occludable?

- 180 degrees or 2 quadrants of iridotrabecular contact constitutes a suspect for angle closure determined by gonioscopy
- Indentation gonioscopy with Sussman or Posner 4 mirror lenses
- Symptoms? Other signs?
- Risk factors?
- OCT/UBM?
• **Plateau iris configuration**
  
  • Based on gonioscopic findings (pre-surgically) with a narrow/closed angle with a flat iris that does not have iris bombe, there still may be a relative pupillary block component, which is relieved after iridotomy

• **Plateau iris syndrome**

  • Based (post-surgically) on the persistent presence of a closed/narrowed angle that did not allow deepening of the anterior chamber and opening of the angle

• Anterior displacement of the ciliary body and anterior insertion of iris root
Plateau Iris Syndrome

4. This patient presented with plateau iris syndrome. Notice how the peripheral iris is mechanically positioned against the trabecular meshwork.
Iridoplasty

- Peripheral burns applied to the iris causing contraction effectively pulling the iris out of the angle
- Applied when LPI does not change presence of occluded or potentially occluded angle in the setting of plateau iris syndrome
- Conduct an exam with basic entrance testing, may consider anterior segment OCT/UBM and address macular/retinal status
- Informed consent, pre/post operative brimonidine, pre-operative pilocarpine, post operative prednisolone acetate 1gtt QID 1 week
- 200-300mW
- 500um spot size
- Circumferential pattern with minimal spacing between burns
- Does not burn complete holes in the iris, but exposes darkly pigmented epithelial layers

Rewind Back to Occludable??? Do We LPI???

- 180 degrees or 2 quadrants of iridotrabecular contact constitutes a suspect for angle closure determined by gonioscopy
- Indentation gonioscopy with Sussman or Posner 4 mirror lenses
- Symptoms? Other signs?
- Risk factors?
- OCT/UBM?
- LPI on an asymptomatic patient???
Organizing Angle Closure

<table>
<thead>
<tr>
<th>Disease Stage</th>
<th>Associated Signs</th>
<th>Recommended Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>PACS (primary angle closure suspect)</td>
<td>Appositional contact but no PAS; normal IOP and optic nerve. Trabecular meshwork at risk.</td>
<td>LPI? Or observation?</td>
</tr>
<tr>
<td>PAC (primary angle closure)</td>
<td>High IOP and/or PAS (i.e., trabecular meshwork dysfunction) but no optic nerve damage.</td>
<td>Phaco if IOP is above 30 mmHg (as per the EAGLE study)</td>
</tr>
<tr>
<td>PACG (primary angle-closure glaucoma)</td>
<td>High IOP and/or PAS with optic nerve damage.</td>
<td>Phaco, with or without trabeculectomy or GDD</td>
</tr>
</tbody>
</table>

https://www.reviewofophthalmology.com/arts/the-asymptomatic-pac-suspect-lpi-or-no-lpi
Effectiveness of early lens extraction for the treatment of primary angle-closure glaucoma (EAGLE): a randomised controlled trial

Augusto Azuara-Blanco, Jennifer Burr, Craig Ramsay, David Cooper, Paul J Foster, David S Friedman, Graham Scotland, Mehdi Javanbakht, Claire Cochrane, John Norrie, for the EAGLE study group

Summary

Background Primary angle-closure glaucoma is a leading cause of irreversible blindness worldwide. In early-stage disease, intraocular pressure is raised without visual loss. Because the crystalline lens has a major mechanistic role, lens extraction might be a useful initial treatment.

Methods From Jan 8, 2009, to Dec 28, 2011, we enrolled patients from 30 hospital eye services in five countries. Randomisation was done by a web-based application. Patients were assigned to undergo clear-lens extraction or receive standard care with laser peripheral iridotomy and topical medical treatment. Eligible patients were aged 50 years or older, did not have cataracts, and had newly diagnosed primary angle closure with intraocular pressure 30 mm Hg or greater or primary angle-closure glaucoma. The co-primary endpoints were patient-reported health status, intraocular pressure, and incremental cost-effectiveness ratio per quality-adjusted life-year gained 36 months after treatment. Analysis was by intention to treat. This study is registered, number ISRCTN44464607.

Findings Of 419 participants enrolled, 155 had primary angle closure and 263 primary angle-closure glaucoma. 208 were assigned to clear-lens extraction and 211 to standard care, of whom 351 (84%) had complete data on health status and 366 (87%) on intraocular pressure. The mean health status score (0·87 [SD 0·12]), assessed with the European Quality of Life-5 Dimensions questionnaire, was 0·052 higher (95% CI 0·015–0·088, p=0·005) and mean intraocular pressure (16·6 [SD 3·5] mm Hg) 1·18 mm Hg lower (95% CI −1·99 to −0·38, p=0·004) after clear-lens extraction than after standard care. The incremental cost-effectiveness ratio was $14284 for initial lens extraction versus standard care. Irreversible loss of vision occurred in one participant who underwent clear-lens extraction and three who received standard care. No patients had serious adverse events.

Interpretation Clear-lens extraction showed greater efficacy and was more cost-effective than laser peripheral iridotomy, and should be considered as an option for first-line treatment.
Longitudinal Changes of Angle Configuration in Primary Angle-Closure Suspects:
The Zhongshan Angle-Closure Prevention Trial

Yuzhen Jiang, MSc, MD\textsuperscript{1,2}, Dolly S. Chang, MD, PhD\textsuperscript{2,3}, Haogang Zhu, MSc, PhD\textsuperscript{1}, Anthony P. Khawaja, MPhil, FRCPoPhth\textsuperscript{4}, Tin Aung, PhD, FRCS(Ed)\textsuperscript{5}, Shengsong Huang, MSc, MD\textsuperscript{2}, Qiyan Chen, BA, MA\textsuperscript{2}, Beatriz Munoz, MSc\textsuperscript{5}, Carlota M. Grossi, BSc, PhD\textsuperscript{1}, Mingguang He, MD, PhD\textsuperscript{1,2}, David S. Friedman, MD, PhD\textsuperscript{1,2,6}, and Paul J. Foster, PhD, FRCS(Ed)\textsuperscript{1,2,7}

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* These authors contributed equally to this work.

Abstract

Objective—To determine longitudinal changes in angle configuration in the eyes of primary angle-closure suspects (PACS) treated by laser peripheral iridotomy (LPI) and in untreated fellow eyes.

Design—Longitudinal cohort study.

Participants—Primary angle-closure suspects aged 50 to 70 years were enrolled in a randomized, controlled clinical trial.

Methods—Each participant was treated by LPI in 1 randomly selected eye, with the fellow eye serving as a control. Angle width was assessed in a masked fashion using gonioscopy and anterior segment optical coherence tomography (AS-OCT) before and at 2 weeks, 6 months, and 18 months after LPI.

Main Outcome Measures—Angle width in degrees was calculated from Shaffer grades assessed under static gonioscopy. Angle configuration was also evaluated using angle opening distance (AOD250, AOD500, AOD750), trabecular-iris space area (TISA500, TISA750), and angle recess area (ARA) measured in AS-OCT images.

Results—No significant difference was found in baseline measures of angle configuration between treated and untreated eyes. At 2 weeks after LPI, the drainage angle on gonioscopy widened from a mean of 13.5° at baseline to a mean of 25.7° in treated eyes, which was also confirmed by significant increases in all AS-OCT angle width measures ($P<0.001$ for all variables). Between 2 weeks and 18 months after LPI, a significant decrease in angle width was observed over time in treated eyes ($P<0.001$ for all variables), although the change over the first 5.5 months was not statistically significant for angle width measured under gonioscopy ($P = 0.18$), AOD250 ($P = 0.167$) and ARA ($P = 0.83$). In untreated eyes, angle width consistently decreased across all follow-up visits after LPI, with a more rapid longitudinal decrease compared with treated eyes ($P$ values for all variables $\leq 0.003$). The annual rate of change in angle width was equivalent to 1.2°/year (95% confidence interval [CI], 0.8–1.6) in treated eyes and 1.6°/year (95% CI, 1.3–2.0) in untreated eyes ($P<0.001$).

Conclusions—Angle width of treated eyes increased markedly after LPI, remained stable for 6 months, and then decreased significantly by 18 months after LPI. Untreated eyes experienced a more consistent and rapid decrease in angle width over the same time period.
Risk of Closure in Asymptomatic Patients in General?

<table>
<thead>
<tr>
<th>Region</th>
<th>Enrollment criteria</th>
<th>Age</th>
<th>Mean follow-up period</th>
<th>Number of cases</th>
<th>Number that developed APAC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Guangzhou, China⁶</td>
<td>(1) anterior chamber depth ≤2 mm; (2) peripheral anterior chamber depth ≤1/4 CT; or (3) iris light band ratio ≤1/4</td>
<td>≥40 years</td>
<td>4.8 years (one to six years)</td>
<td>485</td>
<td>6 (1.2 percent)</td>
</tr>
<tr>
<td>Chicago, USA⁷</td>
<td>(1) anterior chamber depth &lt;2 mm; (2) anterior chamber angle that the initial examining ophthalmologist believed was narrow enough to be capable of closure</td>
<td>Mean age: 62.1 years (range: 36.9 to 84.3 years)</td>
<td>2.7 years (one to six years)</td>
<td>129</td>
<td>8 (6.2 percent)</td>
</tr>
<tr>
<td>Vellore, India⁸</td>
<td>(1) Nonvisibility of the filtering trabecular meshwork for 180 degrees or more; (2) IOP less than 22 mmHg; and (3) no peripheral anterior synechiae in the angle.</td>
<td>Mean age: 54.8 years (range: 36 to 65 years)</td>
<td>Five years, or the time to having met the endpoints</td>
<td>48</td>
<td>0</td>
</tr>
</tbody>
</table>

*Based on Zhang, Liu, Wang et al, 2017*¹⁷

https://www.reviewofophthalmology.com/article/the-asymptomatic-pac-suspect-lpi-or-no-lpi

- What about with dilation?
  - 3 separate studies report closures in narrow angle patients with instance between 0-1.3%
We’ve Come Full Circle. Occludable??? Do We LPI???

• 180 degrees or 2 quadrants of iridotrabecular contact constitutes a suspect for angle closure determined by gonioscopy
• Indentation gonioscopy with Sussman or Posner 4 mirror lenses
• Symptoms? Other signs?
• Risk factors?
• OCT/UBM?

• LPI on an asymptomatic patient???

• It is up to the clinician to perform the procedure using all considerations as guidelines
Contraindications

• Unclear media (corneal edema, uveitis, etc.)
• Chamber is too shallow
• Active uveitis
• Uncontrolled glaucoma without pupillary block component
• Uncooperative patients
Pre-Operative Exam

- Visual acuity
- Basic entrance exam testing
- SLE
  - Consider eyelid position and iris crypts
- IOP (Goldmann)
- Gonioscopy
- Undilated fundus view to fullest extent possible, note macular integrity and consider macular OCT
- Anterior segment imaging (OCT, UBM)
Pre-Operative Exam

• Informed consent*
  • Educate the patient on what the procedure is, why we are doing it, what outcome to expect, risks and benefits of doing/not doing the procedure, alternative procedures, and complications
Pre-Operative Exam

- Only doing procedure on 1 eye per visit
- Instill 1 gtt of 1% or 2% pilocarpine in surgical eye
- Instill 1 gtt of brimonidine in surgical eye
- Instill 1 gtt of proparacaine in each eye
- Energy settings?
Energy Settings

- **Argon/Green/532nm Laser**
  - Typically not used
  - Can still use or use to pre-treat
  - May provide utility regarding darker iris pigmentation
  - Coagulative
  - Spot size, 50um
  - Duration, 0.1s
  - Power, 300-1200mW

- **Nd:YAG 1064nm Laser**
  - More commonly used
  - May have less utility regarding darkly pigmented eyes, although still generally used more often than green laser
  - Photodisruptive
  - Offset, zero microns, can consider slight defocus
  - Spot size, fixed ~8um
  - Duration, 3-7ns
  - Energy, various accepted “norms”, may say 1-6mJ or 2-6mJ, we usually recommend 3.0-6.0mJ
  - Pulse, 1-3
Pre-Operative Exam

- Educate on what to expect during the procedure
- Fill button lens with viscous fluid/gel
- Place on patient’s surgical eye
- Locate appropriate position for procedure
  - Ideally an iris crypt at the 11:00, 1:00, 3:00, or 9:00 position
  - Alight HeNe beams over point of interest and fire laser
- Proceed deeper into the same location if not through iris and repeat
- Proceed deeper into same area until through iris with adequate sized PI
  - About 150-200um size is required for therapeutic effect, we recommend about 0.5-1.0mm
Procedure

• Alight HeNe beams over point of interest and fire laser
• Proceed deeper into the same location if not through iris and repeat
• Proceed deeper into same area until through iris with adequate sized PI
• Size matters
  • About 150-200um size is required for therapeutic effect, we recommend about 0.5-1.0mm
Does Position Matter?

Dysphotopsia after temporal versus superior laser peripheral iridotomy: a prospective randomized paired eye trial

Vanessa Vera 1, Abdulla Naqi 1, Graham W Belovay 1, Devesh K Varma 2, Iqbal Ilke K Ahmed 3

Affiliations + expand
PMID: 24531024  DOI: 10.1016/j.sjoph.2014.02.010

Abstract

Purpose: To determine if the location of neodymium-yttrium-aluminum-garnet laser peripheral iridotomy (LPI) is related to the occurrence of postoperative visual dysphotopsia.

Design: Randomized, prospective, single-masked, paired-eye comparative clinical trial.

Methods: Setting: Private subspecialty clinic in Mississauga, Canada. Study population: Patients with primary angle closure or primary angle-closure suspects were recruited and randomized to receive LPI temporally in one eye and superiorly in the other. Patients were masked to the location of treatment in each eye. Intervention: Temporal or superior LPI. Main outcome measures: Occurrence of new-onset linear dysphotopsia. Other visual disturbances also were assessed using a questionnaire before and 1 month after intervention. Secondary outcome measures included eyelid position, laser parameters, and any intraoperative complications.

Results: A total of 208 patients were recruited to the study, of which 169 (84%) completed it. New-onset linear dysphotopsia was reported in 18 (10.7%) eyes with superior LPI versus 4 (2.4%) eyes with temporal LPI (P = .002). Eleven eyes (6.5%) with superior LPI reported linear dysphotopsia despite complete eyelid coverage of the iridotomy. No significant differences were found with other visual disturbances between them. There was more pain experienced by the temporal LPI (2.8 ± 2.2 vs 2.1 ± 2.0; P = .001), despite no difference in laser energy or number of shots. Intraoperative rates of hemorrhage were similar (8.9% vs 10.1%; P = .71).

Conclusions: Temporal placement of LPI is safe and was found to be less likely to result in linear dysphotopsia as compared with superior placement. Temporal iris therefore may be considered a preferred location for LPI.

Comparison of New Visual Disturbances after Superior versus Nasal/Temporal Laser Peripheral Iridotomy: A Prospective Randomized Trial

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PMID: 29096997  DOI: 10.1016/j.sjoph.2017.09.015

Abstract

Purpose: To determine whether laser peripheral iridotomy (LPI) location affects postoperative dysphotopsia symptoms.

Design: Multicenter, randomized, prospective, single-masked trial.

Participants: Fifty hundred-fifty-nine South Indian patients 30 years of age or older diagnosed as primary angle-closure suspects (PACS) or with primary angle closure (PAC) or primary angle-closure glaucoma (PACG) in both eyes.

Methods: Patients were randomized to either bilateral superior or bilateral nasal/temporal LPI. Occurrence of new visual disturbances was evaluated before and 2 weeks after LPI using a questionnaire based on the 7-item dysphotopsia symptoms described by Spaeth et al.

Main outcome measures: New-onset dysphotopsia symptoms.

Results: Superior LPI (n = 285) and nasal/temporal LPI (n = 274) patients were matched for age (P = 0.6), gender (P = 0.7), and distribution of PACS versus PAC or PACG (P = 0.7). Similar initial laser energy settings were used in both groups (P = 0.3), although superior LPIs required more shots (P = 0.006) and greater total energy (P < 0.001) than nasal/temporal LPIs. No significant differences in postoperative anterior chamber reaction (P = 0.7) or LPI area (P = 0.9) were noted between the 2 groups. No group differences were noted regarding the proportion of patients demonstrating 1 or more dysphotopsia symptoms before LPI (15.8% for superior vs 13.9% for nasal/temporal; P = 0.1) or any individual dysphotopsia symptom (P > 0.2 for all). After LPI, 8.9% of all patients reported 1 or more new symptoms; the most common consisting of linear dysphotopsias, glare, and blurring in 2.7%–4.3% and 4.2% of patients, respectively. Patients undergoing superior LPI were not more likely to describe the new onset of 1 or more dysphotopsia symptoms as compared with patients undergoing nasal/temporal LPI (8.4% vs 9.5%; P = 0.7), nor did the frequency of any new individual symptoms differ by group (P ≥ 0.3 for all). In multivariate logistic regression analysis, neither LPI location nor LPI area nor total laser energy predicted higher odds of new postoperative dysphotopsias (P > 0.1 for all).

Conclusions: Laser peripheral iridotomy likely is safe with respect to visual dysphotopsias regardless of location, LPI size, and amount of laser energy used.
Post-Operative Exam

• Encourage patient procedure went well
• Instill 1 gtt of brimonidine in post surgical eye
• Recheck IOP and VA 30 minutes after procedure
• Rx prednisolone acetate QID in post surgical eye for 1 week
• Re-educate on signs and symptoms of complications
• Follow up in 1 week for evaluation of post surgical eye, and procedure fellow eye if appropriate
Complications

- IOP spike and inflammation most common
- Hyphema
- Dysphotopsia
- Synechiae, correctopia, floaters, monocular diplopia, retinal detachment


Lavanya R;Baskaran M;Kumar RS;Wong HT;Chew PT;Foster PJ;Friedman DS;Aung T; "Risk of Acute Angle Closure and Changes in Intraocular Pressure after Pupillary Dilation in Asian Subjects with Narrow Angles." *Ophthalmology*, U.S. National Library of Medicine, 2012, pubmed.ncbi.nlm.nih.gov/22218999/.


