How large must an iridotomy be?

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Abstract

Four cases of acute angle closure glaucoma in eyes with a small but patent Nd-YAG laser iridotomy are presented, and similar cases in the literature are reviewed. Theoretically a 15 μm diameter iridotomy should be large enough to prevent angle closure glaucoma due to pupil block. Mechanisms by which larger iridotomies fail to prevent angle closure glaucoma, and the role of provocation tests following iridotomy, are discussed. An iridotomy should be at least 150–200 μm in diameter if acute angle closure glaucoma is to be reliably prevented.

Various forms of iris surgery have been used to treat angle closure glaucoma since von Graefe's original description of the broad iridectomy in 1857.1 Surgical iridotomy was introduced by Curran in 1920.2 4 Peripheral iridectomy was described by Chandler in 1952.2 Argon laser iridotomy,3 and more recently Nd-YAG laser iridotomy,4 have already largely superseded surgical iridectomy. While laser surgery has many advantages over conventional surgery, it must not be forgotten that laser iridotomy represents a return to the use of smaller iris openings than those obtained by surgical peripheral iridectomy.5 Gifford noted in 1921 that very small surgical iridotomies were not as effective in controlling intraocular pressure as larger iridotomies.4

Reports are now emerging of acute angle closure glaucoma developing in the presence of a small but patent argon16 or Nd-YAG11,12 laser iridotomy. While cases of acute angle closure glaucoma following surgical peripheral iridectomy have generally been attributed to plateau iris syndrome,11,12 not all cases of acute angle closure glaucoma following a small laser iridotomy can be placed in this category.13,14 We have treated four patients who developed acute angle closure glaucoma despite the presence of a patent Nd-YAG laser iridotomy.

The mechanism of failure of very small iridotomies may be related to obstruction of aqueous flow at very small iridotomy size. Below a critical surface area significant iris bombé may occur at normal aqueous flow rates. When iris bombé is sufficient to cause iridocorneal contact throughout a critical extent of the angle, beginning superiorly, acute angle closure glaucoma results.15 The minimum 'safe' size of iridotomy has not been defined, but an approximate value may be calculated from a simple mathematical model.

Material and methods

We are undertaking a prospective randomised comparison of Nd-YAG laser iridotomy and surgical peripheral iridectomy in the treatment of acute angle closure glaucoma. The cases presented belong to the laser treated group in this study.

A Zeiss Visulas Nd-YAG laser and Zeiss anterior segment YAG contact lens were used in all treatments. Patients were given acetazolamide 500 mg orally one hour before treatment, and one pilocarpine eyedrop 2% 30 minutes before treatment.

A treatment site was chosen in the superior iris between 10 and 2 o'clock, approximately two-thirds of the distance from the pupil margin to the base of the iris. An iris crypt was used, when present. A single 5–10 mJ pulse was delivered to the treatment site. If this pulse did not penetrate the iris, further single pulses were delivered to the same site until penetration was achieved. A gush of aqueous fluid from the posterior chamber could be seen at the moment of penetration. Patency was assessed by direct observation of the posterior chamber or anterior lens capsule.

The size of iridotomies was measured by comparison with the 0.2 mm spot produced by a Haag-Streit slit-lamp focused on the iris plane.

Results: case reports

CASE 1

A 75-year-old hypermetropic man presented with right acute angle closure glaucoma of 48 hours' duration. The intraocular pressure was 78 mmHg, the anterior chamber was shallow, and the angle was closed throughout 360°. The left eye had a very narrow, open angle. Treatment with acetazolamide 500 mg intravenously and pilocarpine eyedrops 2% hourly resulted in a fall in intraocular pressure to 12 mmHg, with an open angle throughout 360°. Nd-YAG laser iridotomy was performed three days later. A treatment site 1 mm from the limbus at 10 o'clock was chosen. The iris was a light grey colour, with no crypts. Three 9-0 mJ pulses resulted in a small (200×100 μm) patent iridotomy. Minimal iris bleeding occurred at the time of treatment, but there were no other complications. One week after treatment the iridotomy was patent, though small. The intraocular pressure was 10 mmHg. No further eye medication was prescribed.

Two weeks after treatment the patient complained of blurred vision in the right eye. The intraocular pressure was 68 mmHg, and gonioscopy revealed 300° of angle closure superiority. Medical treatment with topical pilocarpine 2% and timolol 0·5% reduced the intraocular pressure to 13 mmHg, with an open angle. The iridotomy was small (50×100 μm) but patent. Trabeculectomy with peripheral iridectomy was performed, leading to an intraocular pressure of 10 mmHg, and 6/5 visual acuity.
CASE 2

A 65-year-old woman presented with left acute angle closure glaucoma of 24 hours’ duration. The intraocular pressure was 58 mmHg, the anterior chamber was shallow, and the angle was closed throughout 360°. The right anterior chamber was shallow, with 120° of angle closure superiorly, and the intraocular pressure was 15 mmHg.

After initial medical control left trabeculectomy was performed. Further examination of the right eye after treatment with pilocarpine eyedrops 2% revealed an open angle throughout 360°. Right Nd-YAG laser iridotomy was performed. The treatment site was 1 mm from the limbus, at 10 o’clock. The iris was brown, with no crypts. Four 9-7 mJ pulses were used to produce a patent iridotomy, and a further single 9-7 mJ pulse was used to enlarge the opening.

One week later the iridotomy was patent, though small. The intraocular pressure was 14 mmHg. At review three weeks later the right intraocular pressure was found to be 41 mmHg, and the pupil was dilated. Gonioscopy was not performed. The patient had been using cyclopentolate 1% drops to the left eye, and may have inadvertently contaminated the right eye. After treatment with pilocarpine eyedrops 2% the intraocular pressure returned to 16 mmHg. The iridotomy was patent, but small — 50×50 μm (Fig 1). Further laser treatment was given to enlarge the opening. Five 2-0 mJ Nd-YAG pulses were delivered to the sides of the existing iridotomy, enlarging the opening to 100×100 μm. After this treatment, eyedrops of Betamethasone 0-1% four times a day and tropicamide 1% once daily were prescribed. The patient returned four days later complaining of pain and reduced vision in the right eye. The intraocular pressure was 50 mmHg, and the pupil was mid-dilated and unreactive. The iridotomy was now situated very peripherally, with iris strands running forwards to the cornea, but appeared to be patent. Gonioscopy was not performed. The intraocular pressure returned to normal after treatment with acetazolamide 500 mg intravenously, and pilocarpine eyedrops 2% hourly. The iridotomy was patent, but small (100×100 μm). The angle was open on gonioscopy. Treatment with pilocarpine eyedrops 2% four times a day was prescribed.

A pilocarpine/phenylephrine provocation test as described by Mapstone was performed on the right eye one week later and was negative. Intermittent aching pain above the right eye continued, and further Nd-YAG laser enlargement of the iridotomy was performed six weeks later. Twenty-one 1-1 mJ pulses were delivered by means of a Wise 103-dioptre contact lens, resulting in some oozing of blood. A further 10 1-0 mJ pulses were delivered 30 minutes later. The resulting iridotomy measured 200×200 μm.

One week later all symptoms had resolved, and the iridotomy measured 100×200 μm. Pilocarpine/phenylephrine testing was again negative. However, provocation with Tropicamide eyedrops 1% produced a rise of 9 mmHg in IOP, with folds of iris blocking the trabecular meshwork over 270° on gonioscopy. At follow-up three weeks later the intraocular pressure was 17 mmHg, with no medication (Fig 2).

CASE 3

An 83-year-old woman presented with intermittent pain in the left eye. The intraocular pressure was 44 mmHg, the anterior chamber was deep, and Goldmann gonioscopy revealed 90° of angle closure super temporally. The plane of the iris was flat, with an anteriorly placed iris insertion. The right eye had a similar angle configuration, with normal intraocular pressure. Plateau iris configuration was diagnosed. Intraocular pressure in the left eye quickly returned to normal after treatment with acetazolamide 500 mg orally and pilocarpine eyedrops 2% hourly. Gonioscopy revealed 180° of angle closure superiorly. Nd-YAG laser iridotomy was performed three days later. The iris was light grey, with no crypts. A treatment site 1 mm from the limbus at 2 o’clock was selected. Four 10-9 mJ pulses produced a moderately large (200×400 μm) patent iridotomy. There was minimal iris bleeding at the time of treatment.
After this treatment, eyelids of Timolol 0.5% twice daily, cyclopentolate 1% twice daily, and betamethasone 0.1% four hourly were prescribed. Fourteen days later the patient returned with pain and reduced vision of 24 hours’ duration in the left eye. The intraocular pressure was 50 mmHg. After treatment with Pilocarpine eyelids 2% and timolol eyelids 0.5% the intraocular pressure fell to 7 mmHg. The iridotomy was patent and moderately large (200×400 μm). Identication gonioscopy revealed goniostechniae over 150° superiorly, with plateau iris configuration. A sector iridectomy was performed. On further follow-up the intraocular pressure in the left eye was found to be 28 mmHg on no treatment, falling to 21 mmHg on addition of timolol eyelids 0.5% twice daily. Gonioscopy revealed an open angle throughout 360°.

CASE 4
A 71-year-old hypermetropic woman presented with left acute angle closure glaucoma of eight hours’ duration. The intraocular pressure was 46 mmHg and the anterior chamber was shallow. The right eye had a normal intraocular pressure and a shallow anterior chamber. After medical treatment with acetazolamide 500 mg intravenously and pilocarpine eyelids 2% hourly the left intraocular pressure fell to 9 mmHg within two hours. Goldmann gonioscopy revealed 150° of angle closure superiorly. The right eye also had a very narrow angle, with 150° of angle closure superiorly.

Nd-YAG laser iridotomy was attempted in the left eye the next day. The iris was brown, with no crypts. Three 9.9 mJ pulses were delivered to two sites, with no penetration. Three days later four 10.2 mJ pulses were delivered, two to each of the previous treatment sites. The final pulse produced a very small, patent iridotomy at 2 o’clock, in the mid iris position. A single enlarging pulse of 9.2 mJ was delivered to this site on the following day. A very small (50 μm) iridotomy resulted. The patient was discharged home with betamethasone eyelids 0.1% twice daily and cyclopentolate eyelids 1% once daily to the left eye.

She returned six days later complaining of severe pain in the left eye. The intraocular pressure was 45 mmHg, and the anterior chamber was very shallow. The iridotomy was patent but very small. The intraocular pressure quickly fell to 8 mmHg after treatment with acetazolamide 500 mg intravenously and pilocarpine eyelids 2% hourly. A surgical peripheral iridectomy was performed the next day.

Postoperatively the patient was treated with betamethasone eyelids 0.1% four times daily and tropicamide eyelids 1% once daily. Two days later she complained of pain over the left eye. The pupil was dilated, and the peripheral iridectomy was patent. The intraocular pressure was 41 mmHg. Gonioscopy was not tolerated. The intraocular pressure fell to 10 mmHg after treatment with acetazolamide 250 mg orally twice daily. Timolol 2% four times daily, and timolol eyelids 0.5% twice daily. Long term treatment with pilocarpine eyelids 2% four hourly and timolol eyelids 0.5% twice daily has been necessary to maintain normal intraocular pressure in the left eye.

**MATHEMATICAL MODEL**
A very small iridotomy may be of inadequate physical size to by-pass pupil block adequately. Given a constant rate of aqueous flow, the pressure difference across an iridotomy increases as the size of the iridotomy decreases. Increased posterior chamber pressure relative to anterior chamber pressure will result in iris bombe. The degree of bombe is dependent on the rigidity of the iris and the pressure difference across the iris. At a critical level, dependent on peripheral anterior chamber depth, iris bombe may become sufficient to cause iridoconal contact over a large enough extent of the angle to precipitate acute angle closure glaucoma.10–12 19–20

The minimum ‘safe’ size of an iridotomy may be calculated from a simple mathematical model. Firstly, an assessment is made as to whether aqueous flow is laminar or turbulent through an iridotomy aperture of 10 μm. On the assumption that aqueous viscosity equals the viscosity of water ($\eta = 10^{-3}$ Newton seconds per square metre), aqueous density equals the density of water ($\rho = 1000$ kilograms per cubic metre), and aqueous flow, $Q$, is approximately 2 μl per minute (3.3×10^{-11} cubic metres per second), as measured by Bloom et al.,9 then Reynolds’ number for the system may be calculated.

If the iridotomy size (d) is 10 μm diameter, the aqueous velocity

$$v = \frac{flow}{\pi d^2} = 4 \times 10^{-5} \text{ ms}^{-1}$$

Reynolds’ number for the system

$$R = \frac{vd}{\eta} = 42$$

If $R$ is considerably less than 1000, flow is laminar. Aqueous flow through an iridotomy of diameter 10 μm or larger will be laminar.

Secondly, the iridotomy channel may be considered to be a pipe of length 50 μm on the assumption that the peripheral iris thickness is 50 μm. The pressure drop (pd) along a pipe length $l=50$ μm, diameter $d = 100$ μm, at flow rate $Q$ is expressed by the formula:

$$pd = 128\eta lQ^2/d^4$$

$$= 128 \times 10^{-3} \times 5 \times 10^{-3} \times 3.3 \times 10^{-11} / 142 \times 10^{-16}$$

$$= 0.672 \text{ Nm}^{-2}$$

$$= 0.00494 \text{ mmHg}, \text{ as 1 mmHg} = 136 \text{ Nm}^{-2}$$

An iridotomy of 100 μm diameter will have a pressure drop of 0.00494 mmHg across it on the assumption of total pupil block.

**Figure 3** Pressure difference across the iris as a function of iridotomy size.
However, as pd is proportional to 1/d, if the diameter of the iridotomy were 50 μm pd would be 0.079 mmHg, under the same conditions. If the iridotomy were 25 μm, the pd would be 1.27 mmHg. If the iridotomy were 12.5 μm, the pd would be 20.2 mmHg, and if the iridotomy were 10 μm the pd would be 49.4 mmHg. It is not difficult to conceive that a pressure difference of 10–20 mmHg across the iris could result in significant iris bombé, with iridocorneal contact and angle closure. Such a pressure difference could develop if the size of the iridotomy was between 10 and 15 μm in diameter (Fig 3).

Discussion
Curran observed in 1920 that in some patients with glaucoma the passage of aqueous through the pupil appeared to be impeded by the iris ‘hugging the lens over too great a surface extent’. Surgical iridotomy allowed free drainage of fluid from the posterior chamber to the anterior chamber, with posterior movement of the iris and a reduction in intraocular pressure. Barkan elaborated the concept of pupil block as the causative mechanism of iris bombé, with resulting angle closure. A peripheral iridectomy causes ‘collapse’ of iris bombé, resulting in widening of the ‘entrance’ to the angle. Peripheral anterior chamber depth, but not central anterior chamber depth, increases following peripheral iridectomy owing to resolution of iris bombe.

Failure of surgical peripheral iridectomy, or laser iridotomy, as evidenced by development of acute angle closure glaucoma, may occur by one of two mechanisms. A small number of cases of angle closure glaucoma are related to ‘plateau iris configuration’ an anteriorly placed iris root, flat iris plane, and a normal or near normal anterior chamber depth. Pupil dilatation results in peripheral iris bunching, with occlusion of the trabecular meshwork. As pupil block is not causative, surgical peripheral iridectomy or laser iridotomy are relatively ineffective in preventing further attacks of acute angle closure glaucoma. One of our patients (case 3) probably had plateau iris syndrome, and developed acute angle closure glaucoma following mydriasis despite the presence of a relatively large, patent iridotomy.

Case 4 developed acute angle closure glaucoma following mydriasis despite the presence of a small patent iridotomy and subsequently a larger surgical iridectomy. The mechanism of acute intraocular pressure rise may have been iris crowding in the angle. Plateau iris configuration was not present. Perhaps the term ‘angle crowding’ angle closure glaucoma would be a more appropriate term to use for eyes which develop acute angle closure glaucoma following mydriasis in the presence of a patent peripheral iridectomy. The cases of two other patients with moderately large peripheral iridectomies who developed acute angle closure glaucoma while using mydriatic drops have been reported.

Mydriatic or dark room provocation tests following peripheral iridectomy have been found to be positive in 3–19% of cases. Gonioscopy at the end of a positive test in eyes with patent peripheral iridectomies has revealed angle closure in some cases, due to angle crowding. However, in other eyes the angle has been open. Raised intraocular pressure in these eyes may be explained by the outflow facility lowering effect of anticholinergic drugs. Surgical peripheral iridectomy appears entirely to overcome pupil block. No cases of acute angle closure glaucoma due to iris bombe have been reported in the presence of a patent surgical iridectomy.

Mydriatic provocation tests following argon laser iridotomy and Nd-YAG laser iridotomy have given similar results to those performed following surgical peripheral iridectomy. However, pilocarpine/phenylephrine tests have been negative in all eyes tested following argon laser iridotomy or Nd-YAG laser iridotomy. The test produces only moderate mydriasis, and increased tone in both the sphincter and dilator muscles of the iris may prevent angle crowding. The test induces maximal pupil block, and failure to obtain any positive results suggests that pupil block was fully overcome in all cases tested.

Acute angle closure glaucoma in the presence of a small, patent argon laser iridotomy has previously been reported in two patients. Mandelkorn et al reported a case of acute angle closure glaucoma following a 50 μm diameter argon laser iridotomy. Further argon laser treatment was applied to the site, resulting in satisfactory long term pressure control. Brainard reported a similar case, in an eye with a 75 μm diameter argon laser iridotomy. Further argon laser treatment to enlarge the iridotomy to 125 μm resulted in satisfactory long term intraocular pressure control, suggesting inadequate iridotomy size was responsible for initial failure.

Acute angle closure glaucoma has also been reported in the presence of a small, but patent Nd-YAG laser iridotomy. Brazier reported two cases. One was treated by surgical peripheral iridectomy, with no further complications. The second was treated with miotics. Wishart and Hitchings reported two cases of acute angle closure glaucoma and two cases of subacute angle closure glaucoma in the presence of patent Nd-YAG laser iridotomies. In three cases the iridotomy was 100 μm diameter and in the fourth 150 μm. Gray et al reported two cases of acute angle closure glaucoma in the presence of a small, patent iridotomy. One developed acute angle closure glaucoma while using pilocarpine eye drops 4%, and was treated by trabeculectomy. The second was treated by a further iridotomy. Intraocular pressure subsequently remained normal in both cases. Two of our cases (cases 1 and 2) behaved in a very similar way to the two cases reported by Gray et al. Case 1 responded to trabeculectomy, and case 2 responded to laser enlargement of the iridotomy.

The occurrence of acute angle closure glaucoma in the presence of a small, but patent, iridotomy which responds to surgical peripheral iridectomy or laser enlargement, suggests the size of the initial iridotomy was inadequate.

Wishart studied iris bombe using slit-lamp photographic assessment of peripheral anterior chamber depth. In four eyes with very small iridectomies and progressive narrowing of the
angle, repeat laser iridotomy resulted in deepening of the peripheral anterior chamber. This study provides further evidence that very small iridotomies may not fully relieve iris bombé. We have calculated that the minimum functional iridotomy size should be approximately 15 μm, as previously suggested by Wheeler. However, our clinical experience, and that of others, shows that iridotomies in the range of 50-150 μm diameter may fail to prevent acute angle closure glaucoma.

Several factors may contribute to the reduction in size of an iridotomy of theoretically 'safe' size below that needed to prevent acute angle closure glaucoma. Most cases of acute angle closure glaucoma, following Nd:YAG laser iridotomy have occurred within one month of treatment. Naveh et al observed rapid diminution of Nd:YAG laser iridotomy size within an hour of treatment in six eyes. In each case a return to original treatment size occurred within two weeks. Localised iris oedema round the treatment site could be responsible.

Re-closure of Nd:YAG laser iridotomies due to iris pigment epithelium proliferation is unusual, and tends to occur 1-4 months after treatment. Re-closure resulted in acute angle closure glaucoma in two eyes reported on by Wishart and Hitchings and one eye reported on by Gray et al.

Case 2 developed acute angle closure glaucoma following mydriasis. Pupil dilatation might reduce the surface area of some iridotomies, and folds of iris tissue might functionally block an iridotomy when the pupil is dilated. Mydriatics might therefore produce acute angle closure glaucoma in the presence of a patent iridotomy by two mechanisms – pupil block due to reduction of iridotomy size, and angle crowding.

To answer the question, How large must an iridotomy be to prevent acute angle closure glaucoma? several factors must be considered. In forms of angle crowding angle closure glaucoma, such as the plateau iris syndrome, a large iridotomy will not prevent the development of acute angle closure glaucoma following mydriasis, as pupil block is not causative.

Theoretical modelling suggests an iridotomy of 10-15 μm diameter will prevent angle closure glaucoma due to pupil block. However, an iridotomy of greater size may fall below this critical level due to early post-treatment oedema, late pigment epithelium proliferation, or pupil dilatation. Wishart and Hitchings reported subacute angle closure glaucoma in an eye with a 150 μm diameter iridotomy. All other cases of acute angle closure glaucoma in the presence of a patent iridotomy have occurred in eyes with an iridotomy of less than 100 μm in diameter. The smallest potentially safe size of an iridotomy would therefore appear to be 100 μm. However, in order to incorporate a safety margin, we would recommend that iridotomies should be at least 150-200 μm in diameter.

Provocation tests have not been helpful in identifying eyes at risk, as eyes developing acute angle closure glaucoma following Nd:YAG laser iridotomy have done so before a provocation test had been performed. If provocation tests are to be employed, we suggest the use of tropicamide eyedrops 1% soon after the iridotomy has been performed, in order to detect cases at risk of developing acute angle closure glaucoma. Routine pupil dilatation to prevent posterior synechiae formation following laser iridotomy may be more safely achieved with phenylephrine eyedrops 10%, with measurement of intraocular pressure following dilatation. Lowe has shown that phenylephrine very rarely results in a rise in intraocular pressure following laser peripheral iridotomy, and the effect may be quickly reversed using thymoxamine eyedrops 0.5% if a pressure rise does occur.


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