The Rarity of Clinically Significant Rise in Intraocular Pressure after Laser Peripheral Iridotomy with Apraclonidine

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Objective: To determine the incidence of intraocular pressure (IOP) rise of varying degrees after laser peripheral iridotomy (LPI) in patients with and without glaucoma treated perioperatively with pilocarpine and apraclonidine.

Design: A retrospective chart review.

Participants: A total of 289 eyes in 179 patients with narrow occludable angles (NOA) (N = 148), open-angle glaucoma or ocular hypertension (OAG) (N = 115), or chronic-angle closure glaucoma (CACG) (N = 26) were reviewed.

Main Outcome Measures: The difference between preoperative and postoperative IOP, absolute postoperative IOP, and the need for acute IOP-lowering treatment was noted.

Results: Only 1.1% (95% confidence interval [CI], 0.03%–5.8%; 1 of 94) of patients and 0.7% (95% CI, 0.02%–3.7%; 1 of 148) of eyes with NOA experienced a rise of more than 10 mmHg 1 to 2 hours after LPI. The incidence of postoperative IOP greater than 25 mmHg and acute postoperative IOP-lowering management was 0% (95% CI, 0%–3.8%). Intraocular pressure in 1 of 115 eyes (0.9%, 95% CI, 0.02%–4.7%) with OAG rose more than 10 mmHg, requiring acute treatment. None of the 26 CACG eyes experienced a rise of more than 10 mmHg (95% CI, 0%–13.2%).

Conclusion: The IOP rise that requires further intervention after LPI with the perioperative use of pilocarpine and apraclonidine is very uncommon. In patients with NOA, routine postiridotomy IOP monitoring may not be required. *Ophthalmology 1998;105:2256–2259*

Almost from its inception as a means of producing an iridotomy in humans, laser peripheral iridotomy (LPI) has been known to result in an acute, transient rise in intraocular pressure (IOP) that may be greater than 10 mmHg in up to a third of eyes[1,2] and perhaps severe enough to induce irreversible ocular damage. Robin and Pollack[1] first demonstrated the efficacy of topical apraclonidine (Iopidine; Alcon, Ft. Worth, TX) in dramatically reducing this IOP response to LPI[3]; other investigators confirmed their findings.[4–6] For almost 10 years, the perioperative use of apraclonidine has been a standard adjunct to LPI. However, because no preoperative or operative factors have been consistently shown to predict which eyes will experience a potentially damaging rise in IOP, recent publications have continued to recommend the routine postoperative monitoring of IOP in all LPI patients.[7,8]

Our clinical impression suggested that clinically significant rises in IOP (potentially damaging rises requiring IOP-lowering intervention) are now exceedingly uncommon. To determine the frequency of such IOP rises, we looked at all LPI procedures performed at our institution over a 7-year period.

Materials and Methods

The charts of all patients undergoing LPI at the University of Wisconsin and one of its satellite clinics between February 1990 and December 1996 were reviewed. Data collected included primary diagnosis, patient age at the time of the procedure, which eye was treated, IOP measured before surgery and 1 to 2 hours after surgery by applanation tonometry, chronic ocular medications, the number of laser shots, and total energy used. Patients were almost all white. Specific notations of race and iris color were not available. All patients were treated 1 hour before surgery with pilocarpine 1% to 4% and 0.5 or 1% apraclonidine and immediately after surgery with apraclonidine. Most iridotomies were made with a neodymium:YAG (Nd:YAG) laser alone (N = 278); some were made with a combination of argon and Nd:YAG lasers (N = 11).

Eyes treated for acute angle closure were excluded from the analysis. Remaining eyes were divided into one of three diagnostic

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Table 1. Patient Demographics

	Diagnostic Group*		
	NOA	*OAG*	*CACG*
No. of patients	84	75	20
Patient age (yrs)	67.5	67.4	71.5
(SD)	(11.2)	(16.1)	(10.1)
Range	41.2–102.9	25.1–99.6	52.4–94.9
Preoperative IOP (mmHg)	16.0	22.8	24.1
(SD)	(2.9)	(6.5)	(12.1)
Range	8–21	10–45	3–54
No. of preoperative medications	0.01	0.75	0.95
(SD)	(0.11)	(0.89)	(0.94)
Range	0–1†	0–3	0–3
Total energy used (mJ)	49.1‡	38.6	41.0
(SD)	(84.4)‡	(70.0)	(36.2)
Range	3.1–1979	4.1–565	8–120

NOA = narrow occludable angle; OAG = open-angle glaucoma or suspect glaucoma; CACG = chronic angle-closure glaucoma; SD = standard deviation; IOP = intraocular pressure.

* One eye per patient, categorized by most severe diagnosis (CACG > OAG > NOA). The right eye was chosen if both eyes had same diagnosis.

† Includes patient treated with an oral agent for the fellow eye.

‡ Excludes single patient with extraordinary energy of 1979 mJ.

groups. The narrow occludable angle (NOA) group included those eyes that were judged by gonioscopy to have narrow occludable angles but no record of IOP greater than 21 mmHg. The open-angle glaucoma/ocular hypertension group (OAG) included those eyes that carried either a diagnosis of primary open-angle glaucoma or suspect glaucoma based on IOP greater than 21 mmHg on at least one occasion but also had anatomically narrow angles. The chronic angle closure group (CACG) included those eyes that carried a diagnosis of chronic-angle closure glaucoma defined by the presence of peripheral anterior synechiae.

Data for 179 patients (289 eyes) who underwent LPI were analyzed. In the group analysis (Tables 1 and 2), each patient was counted only once, even if both eyes with differing diagnoses were treated, to maintain the independence of observations. Patients were categorized according to the most severe diagnosis (CACG > OAG > NOA).

To detect the occurrence of rare events, the analysis of IOP rise included all eyes so that no event was excluded. In addition, because we were most concerned with capturing adverse events on a per-patient basis, we performed a worst-eye analysis as well (Tables 3–7). Patients whose two eyes fell into the same diagnostic group had only the eye with the greatest IOP rise after LPI chosen for further analysis. For patients whose eyes fell into different diagnostic groups, but were both treated, each eye was included in the analysis for its respective diagnostic group.

Exact 95% confidence intervals for the event rates were calculated based on the binomial distribution. Likelihood ratio tests from logistic regression analysis were used to examine the association between IOP rise and potential risk factors.

Results

Table 1 summarizes preoperative and operative factors for each diagnostic group. By definition, patients in the NOA group had preoperative IOPs of 21 mmHg or less; patients in the OAG and CACG groups had a wide range of preoperative IOPs. Laser energy used varied greatly in all diagnostic groups. A single patient in the NOA group underwent an extraordinarily prolonged procedure, which resulted in the mean energy used for this group drawing upward substantially. This patient did not have an IOP rise, and the mean energy listed for the NOA group in the table excludes this patient.

Using a single eye from each patient (right eye, if both treated) for analysis, Table 2 shows that for each diagnostic group, the mean difference between preoperative IOP and pressure was measured approximately 1 hour after LPI. The mean change for all groups was a decrease in IOP of several millimeters of mercury. The range for each group shows that maximum increases in IOP varied from a rise of 7 mmHg in a CACG patient to 8 mmHg in a NOA patient and to 12 mmHg in an OAG patient.

The analysis of all eyes, performed to capture rare events, shows the degree of IOP rises after LPI (Table 3). A single NOA patient's two eyes accounted for the only eyes in this group in which IOP rose more than 5 mmHg; one eye rose 12 mmHg to 24 mmHg and the other eye rose 8 mmHg. Review of this patient's records showed nothing remarkable about her ocular history or the LPI procedure itself. The only other eye to rise more than 10 mmHg after LPI was in the OAG group.

The occurrence and incidence of outcomes that might be regarded as clinically significant are listed in Tables 4 through 6. Again, to detect the occurrence of a rare outcome, all eyes were used for analysis. No eyes in the NOA group had a postoperative IOP of more than 25 mmHg. Eyes in the OAG and CACG groups had postoperative IOPs greater than 25 mmHg 6.1% and 15.4% of the time, respectively. These eyes were more likely to have had higher preoperative IOPs as well.

The IOP in two eyes, one each in the NOA and OAG groups, rose more than 10 mmHg. Only the eye in the OAG group required acute treatment. Thus, all 148 NOA eyes underwent LPI without an IOP spike requiring intervention. Although none of the eyes in the CACG group rose more than 10 mmHg or required intervention, the smaller number of eyes in this group precludes a meaningful conclusion.

Logistic regression (Table 7) showed no definite association between rise in IOP after LPI and primary diagnosis, preoperative IOP, preoperative medications, or the number of laser shots or total energy used.

Table 2. Change in Intraocular Pressure (IOP)

	Diagnostic Group*		
	NOA	*OAG*	*CACG*
No. of patients	84	75	20
Preoperative IOP (mmHg)	16.0	22.8	24.1
(SD)	(2.9)	(6.5)	(12.1)
Range	8–21	10–45	3–54
Postoperative IOP (mmHg)	12.8	17.4	18.8
(SD)	3.8	5.7	9.6
Range	4–23	9–40	3–40
Change in IOP (mmHg)	−3.2	−5.4	−5.3
(SD)	(3.5)	(6.2)	(8.9)
Range	(−10)–8	(−24)–12	(−26)–7

NOA = narrow occludable angle; OAG = open-angle glaucoma or suspect glaucoma; CACG = chronic angle-closure glaucoma; SD = standard deviation.

* One eye per patient, categorized by most severe diagnosis (CACG > OAG > NOA). The right eye was chosen if both eyes had same diagnosis.

Table 3. Rise in Intraocular Pressure (IOP) after Iridotomy

	Diagnostic Group*					
	NOA		OAG		CACG	
IOP rise (mmHg)	% (95% CI)	n (eyes)	% (95% CI)	n (eyes)	% (95% CI)	n (eyes)
1–5	12.8 (7.9–19.3)	19/148	9.6 (4.9–16.5)	11/115	11.5 (2.4–30.2)	3/26
6–10	0.7 (0–2.5)	1/148	1.7 (0.2–6.1)	2/115	7.7 (0.9–25.1)	2/26
≥11	0.7 (0–2.5)	1/148	0.9 (0.02–4.7)	1/115	0.0 (0–13.2)	0/26

NOA = narrow occludable angle; OAG = open-angle glaucoma or suspect glaucoma; CACG = chronic angle-closure glaucoma; CI = confidence interval (exact binomial).

* Each eye was categorized separately. Patients may appear twice.

Discussion

In 7 years and treatment of 289 eyes with LPI at our institution for narrow angles in a nonacute angle closure setting, only 2 eyes experienced a rise in IOP of more than 10 mmHg, and only 1 of these eyes required intervention. No eye without a history of ocular hypertension or glaucoma needed such intervention. To our knowledge, this is to date the largest reported series of patients undergoing LPI.

Several prospective, randomized control, double-masked studies using one eye per patient have shown the efficacy of apraclonidine in reducing IOP rise after LPI, albeit in relatively small series. Robin et al[3] found no IOP rises greater than 10 mmHg over baseline after LPI in 14 eyes with chronic narrow-angle glaucoma with disc or visual field changes. Brown et al,[4] in patients with unspecified diagnoses, found no IOP rises greater than 5 mmHg in 17 eyes. Kitazawa et al[5] and Sridharrao and Badrinath[6] found the rates of IOP rise greater than 10 mmHg to be 3.4% (1 of 29 eyes with CACG) and 3.2% (1 of 31 eyes with unspecified diagnoses), respectively. These studies are in agreement with our own findings that even in glaucomatous eyes, the incidence of IOP rise greater than 10 mmHg is low.

It is possible that this predominantly white population, most of whom had blue irides, has a lower rate of clinically significant IOP rises than would a more heterogeneous population. It generally is accepted that blue irides require less laser energy for penetration than dark ones. However, prior studies have indicated that neither race, iris color, nor any other identifiable factor is predictive of the IOP response after LPI.[1,3]

Table 4. Clinically Significant Intraocular Pressure (IOP) Rise after Iridotomy: Narrow Occludable Angle Group

	Eyes		Patients*	
	% (95% CI)	n	% (95% CI)	n
IOP > 25 mmHg	0.0 (0–2.5)	0/148	0.0 (0–3.8)	0/94
IOP rise > 10 mmHg	0.7 (0.02–3.7)	1/148	1.1 (0.03–5.8)	1/94
Need for treatment	0.0 (0–2.5)	0/148	0.0 (0–3.8)	0/94

CI = confidence interval.

* One eye per patient included. If both eyes have the same diagnosis, only the eye with the highest postoperative IOP is included. If eyes have differing diagnoses, the patient appears in both diagnostic groups.

Table 5. Clinically Significant Intraocular Pressure (IOP) Rise after Iridotomy: Open-angle Glaucoma Group

	Eyes		Patients*	
	% (95% CI)	n	% (95% CI)	n
IOP > 25 mmHg	6.1 (2.5–12.1)	7/115	7.9 (3.0–16.4)	6/76
IOP rise > 10 mmHg	0.9 (0.02–4.7)	1/115	1.3 (0.03–7.1)	1/76
Need for treatment	0.9 (0.02–4.7)	1/115	1.3 (0.03–7.1)	1/76

CI = confidence interval.

* One eye per patient included. If both eyes have the same diagnosis, only the eye with the highest postoperative IOP is included. If eyes have differing diagnoses, the patient appears in both diagnostic groups.

The decision to amend recommendations for routine postoperative IOP monitoring is difficult. It may be useful to place the risk of potentially clinically significant IOP rise after LPI in context. The risk of such IOP rise after cataract surgery is estimated to be 1.6%.[9] However, current standard of practice suggests that IOP monitoring several hours after LPI is appropriate, whereas after cataract surgery, it is not requisite, and patients are only seen the following day. One could speculate that our standard of care in this regard is informed more by our past than by a realistic assessment of current risk. For patients with narrow occludable angles and normal IOP at our institution, postoperative IOP monitoring contributed nothing to their care, as no additional intervention was made.

Table 6. Clinically Significant Intraocular Pressure (IOP) Rise after Iridotomy: Chronic Angle-closure Group

	Eyes		Patients*	
	% (95% CI)	n	% (95% CI)	n
IOP > 25 mmHg	15.4 (4.4–34.9)	4/26	20.0 (5.7–43.7)	4/20
IOP rise > 10 mmHg	0.0 (0–13.2)	0/26	0.0 (0–16.8)	0/20

CI = confidence interval.

* One eye per patient included. If both eyes have the same diagnosis, only the eye with the highest postoperative IOP is included. If eyes have differing diagnoses, patient appears in both diagnostic groups.

Table 7. Risk Factors for Intraocular Pressure (IOP) Rise after Iridotomy

Factor	OR	(95% CI)	P*
Diagnostic group			0.82
NOA	1.00		
OAG	1.03	(0.45, 2.50)	
CACG	1.50	(0.43 5.25)	
Preoperative IOP	0.95	(0.88, 1.02)	0.10
Preoperative medications	1.53	(0.95, 2.44)	0.09
No. of laser shots (per 10 shots)	1.00	(0.85, 1.20)	0.89
Total laser energy used (per 50 mJ)	1.01	(0.91, 1.13)	0.80

OR = odds ratio; CI = confidence interval; NOA = narrow occludable angle; OAG = open-angle glaucoma or suspect glaucoma; CACG = chronic angle-closure glaucoma.

* Logistic regression, likelihood ratio, one eye per patient, for the association of any IOP rise > 0 mmHg.

The limitations of this study include: (1) limited sample size; (2) homogeneous population; (3) lack of information regarding race and iris color; and (4) retrospective nature.

Nevertheless, within these limitations, the study suggests that IOP rises requiring further intervention after LPI are uncommon occurrences. In eyes with healthy optic nerves that could withstand relatively high IOPs for brief periods of time, routine IOP monitoring may be unnecessary. We invite other investigators to present data to assess more accurately the risk of IOP rise after LPI over a larger collective sample of patients.

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