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# The Effectiveness and Safety of Micropulse Cyclophotocoagulation in the Treatment of Ocular Hypertension and Glaucoma

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***Purpose:*** To evaluate the effectiveness and safety of primary and adjunctive micropulse cyclophotocoagulation (MPCPC) in the treatment of ocular hypertension (OHT) and glaucoma.

***Design:*** Retrospective cohort study.

***Participants:*** Ocular hypertension and all severities of glaucoma (including treatment-naïve and well-sighted eyes) and all types of glaucoma (including normal-tension glaucoma [NTG]).

***Methods:*** Consecutive eyes with glaucoma or OHT that underwent MPCPC (Iridex Cyclo G6 Glaucoma Laser System, Mountain View, CA) between 2016 and 2018 were identified.

***Main Outcome Measures:*** Intraocular pressure (IOP), visual acuity, glaucoma medications, and ocular adverse events.

***Results:*** Three hundred ninety-nine MPCPC surgeries, on 342 eyes of 214 patients, were analyzed. Laser power ranged from 900 to 2800 mW. Main diagnoses in descending prevalence were primary open-angle glaucoma (55.9%), chronic angle-closure glaucoma (10.8%), neovascular glaucoma (9.0%), NTG (6.5%), and OHT (5.5%). Mean baseline IOP was 19.8±7.4 mmHg and IOP reduction was 22.7%, 20.2%, 20.7%, and 23.7% at postoperative months (POMs) 1, 3, 6, and 12 ($P < 0.0001$ for all time points). The end point of 20% or more mean IOP reduction from baseline was achieved by 67.8% of the study cohort at POM 12. Additional mean IOP reduction of 16.4% ($P < 0.0001$) was achieved with each re-treatment. Analysis based on IOP stratification demonstrated 30.5% mean IOP reduction at POM 12 when baseline IOP was more than 21 mmHg and 20.1% when it was 21 mmHg or less (71% of overall cohort; $P < 0.0001$). Analysis based on laser power stratification demonstrated mean IOP reduction of 31.5% at POM 12 with laser power of 2500 mW or more and 17.8% with laser power of less than 2500 mW ($P < 0.02$). Overall, the mean number of topical glaucoma medications was unchanged from baseline to POM 12. Greater baseline IOP and number of baseline topical glaucoma medications were significant predictors of effectiveness in the regression analysis. No patients demonstrated persistent inflammation or hypotony, phthisis bulbi, or sympathetic ophthalmia.

***Conclusions:*** In patients with OHT or glaucoma, MPCPC demonstrated effectiveness and safety in IOP reduction sustained to 1 year. Baseline IOP of 21 mmHg or less subgroup demonstrated a more limited response. A dose-response relationship is suggested with respect to laser power and repeat treatments. *Ophthalmology Glaucoma 2020;3:181-189 © 2020 by the American Academy of Ophthalmology*

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Glaucoma, one of the leading causes of irreversible blindness worldwide,[1] is treated with topical ocular medication, oral medication, laser therapy, and incisional surgery. This optic neuropathy is distinguished from ocular hypertension (OHT), which is characterized by high intraocular pressure (IOP) of more than 21 mmHg without damage to the optic nerve and therefore vision loss. The mainstay of treatment for all types of glaucoma is IOP reduction. Cycloablative procedures, a form of glaucoma treatment, are far from novel. In 1831, Hancock portrayed his ideas on cyclotomy, and the earliest forms of cycloablation were through methods including cyclectomy, cryotherapy, and diathermy.[2–6] Thereafter, cyclophotocoagulation was introduced[7] using such lasers as the ruby, neodymium:yttrium–aluminum–garnet, argon, krypton, and diode.[8–12] Diode laser is now the mainstay for cycloablation. The advancement of lasers used for cyclodestruction has led to decreased rates of vision loss, intraocular inflammation, severe pain, hypotony, phthisis, and sympathetic ophthalmia.[13]

Traditional transscleral cyclophotocoagulation (TSCPC) is a cyclodestructive procedure that uses an 810-nm wavelength laser. Continuous laser energy is delivered and absorbed by melanin in the pigmented ciliary body epithelium, resulting in coagulative necrosis, reduction of aqueous humor secretion, and subsequent decrease in IOP. Micropulse cyclophotocoagulation (MPCPC; Iridex Cyclo G6 Glaucoma Laser System) is an alternative to traditional diode TSCPC. Although MPCPC uses the same wavelength as conventional TSCPC, it differs in that MPCPC fragments


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the continuous laser beam into short repetitive bursts of energy via on-and-off duty cycles. This new laser delivery platform allows tissues to cool in between cycles, and despite it being categorized as cycloablative, data support MPCPC as not being truly cyclodestructive in nature.[14] Evidence also supports MPCPC as an effective and safer alternative to traditional TSCPC in IOP reduction.[15] However, with MPCPC being novel compared with more established glaucoma procedures, the literature is limited with regard to the number of studies with a large cohort size, significant follow-up duration, and fixed treatment protocol.

To our knowledge, this study encompasses the largest consecutive MPCPC cohort published to date. The objective of this retrospective study was to investigate the effectiveness and safety of MPCPC in the treatment of OHT and all severities of glaucoma (including treatment-naïve and well-sighted eyes) and all types of glaucoma (including normal-tension glaucoma [NTG]).

## Methods

### Study Design

This was a retrospective cohort study approved by the William Osler Health Systems Research Ethics Board. Patient consent was obtained, and all research adhered to tenets of the Declaration of Helsinki. Consecutive eyes with glaucoma or OHT that received MPCPC treatment between May 1, 2016, and May 19, 2018, were included. The study had no exclusion criteria.

### Baseline Measures

Baseline parameters included age, gender, ethnicity, glaucoma type, disease severity, previous laser treatments or surgeries, number of glaucoma medications (topical and oral), IOP (via Goldmann applanation tonometry), and Snellen corrected distance visual acuity (CDVA). The Canadian Ophthalmological Society clinical practice guidelines[16] were used for staging of disease.

### Outcome Measures

Postoperative data were recorded at postoperative months (POMs) 1, 3, 6, and 12. The primary outcome measure was effectiveness as measured by IOP reduction. The effectiveness end point was 20% or more mean IOP reduction from baseline, achieved with or without medication. Subgroup response to MPCPC (i.e., NTG, pretreatment IOP of 21 mmHg or less, laser power, and repeat treatments) was evaluated.

Secondary outcome measures included number of glaucoma medications, vision loss, and ocular adverse effects. Vision loss was defined as a decrease of 2 lines or more of Snellen CDVA not attributable to a shift in refractive error as determined by pinhole visual acuity (VA) or subjective autorefraction. Recorded adverse events included IOP spike (defined as IOP of more than 25% from baseline), persistent hypotony (defined as IOP of 5 mmHg or less on 2 or more consecutive visits persisting for 3 months or more), serous choroidal detachment, persistent inflammation (defined by inflammation persisting for 3 months or more), macular edema, symptomatic mydriasis, cataract, vision loss, painful eye, and phthisis bulbi. Ocular surface disease was defined by Dry Eye Workshop (DEWS) II criteria.[17]

### Micropulse Cyclophotocoagulation Procedure

The treatment was performed by 2 surgeons (D.Y. or E.T.) using a fixed treatment protocol and standardized technique. All patients received 1 g acetaminophen, if no contraindications existed, before arrival at the surgical center. On arrival, lidocaine 2% gel, topical tetracaine, and topical brimonidine were instilled in the preoperative holding area. At least 30 minutes before the laser procedure, a 0.5-mlsubconjunctival injection of 2% lidocaine with epinephrine was administered in each hemisphere. The injection was aimed at being posterior from the limbus with effort to avoid blood vessels to minimize occurrence of subconjunctival hemorrhage. All patients received concurrent intravenous neurolept anesthesia using a combination of midazolam and fentanyl, with dosage titrated to the patient's need. The Cyclo G6 Laser in its micropulse treatment mode with the MicroPulse P3 handpiece was used for all treatments. Micropulse cyclophotocoagulation laser settings were maintained at 80 seconds per hemisphere for a total of 160 seconds in each eye with a duty cycle of 31.3% (0.5-ms duration, 1.1-ms interval). Power, the only variable, ranged from 900 to 2800 mW and was titrated at the discretion of the surgeon based on visual acuity, glaucoma severity, baseline IOP, target IOP, and previous MPCPC response. In general, patients with higher baseline IOP were treated with higher laser power. Laser power was decreased if audible "pops" were heard or if the patient could not tolerate the current power. The laser probe's fiber optic probe was oriented with the curved side toward the limbus and the flat side toward the eyelid. The handpiece was positioned 1 to 2 mm posterior to the limbus and was held perpendicular to the globe. The mobilization of the probe was via a continuous sliding arc motion. Dwell time was 10 seconds per pass in each hemisphere, with a total of 8 passes per hemisphere (80 seconds total treatment time per hemisphere). The 3- and 9-o'clock positions, although not marked, were avoided, as well as any site with scleral thinning, glaucoma drainage device, or filtering bleb. Moderate to firm steady pressure was applied on the probe for well-controlled contact with the globe. A coupling liquid interface (lidocaine 2% gel in conjunction with a viscous artificial tear) was used.

### Postoperative Management

Patients were prescribed difluprednate 0.05% 4 times daily for the first week after laser treatment. At the discretion of the surgeon and with consideration of target IOP, medical therapy was reduced via a stepwise approach starting with oral glaucoma medication. Retreatment was considered if IOP reduction was over target. Retreatment was not considered if a lack of effectiveness became apparent or if significant adverse events occurred. Repeat MPCPC occurred at least 1 month from initial treatment.

### Statistical Analysis

Data analyses were carried out using SAS software version 9.4 (SAS Institute, Cary, NC) and R software version 3.6.1 (R Foundation for Statistical Computing, Vienna, Austria). A $P$ value of less than 0.05 was set for significance. Intraocular pressure spikes were excluded for analyses pertaining to effectiveness. Preoperative and postoperative data were compared using bivariate and multivariate regression analysis, with adjustments made for the following confounding variables: baseline IOP, baseline logarithm of the minimum angle of resolution (logMAR) VA, baseline oral glaucoma medications, baseline number of topical glaucoma medications, history of glaucoma surgery, history of cataract surgery, age, gender, ethnicity, diagnoses, severity, surgeon, eye, laser power, and postoperative period. Descriptive statistics were reported as mean ± standard deviation for continuous variables and

as percentages for categorical variables. A linear mixed model was applied to control for outcome dependence in regression analysis. The analysis accounted for the interrelationships between both eyes of the same patient.

## Results

### Study Population and Baseline Characteristics

A total of 399 MPCPC surgeries on 342 eyes of 214 patients were analyzed. Table 1 illustrates the demographic statistics of the cohort. Mean patient age was 67±13 years (range, 26–97 years), and 46% of patients (n = 98) were women. Most patients were white (53.3% [n = 114]), followed by Asian (35.0% [n = 75]). The diagnoses in descending prevalence were primary open-angle glaucoma (55.9% [n = 223]), chronic angle-closure glaucoma (10.8% [n = 43]), neovascular glaucoma (9.0% [n = 36]), NTG (6.5% [n = 26]), and OHT (5.5% [n = 22]). Most of the eyes had undergone prior glaucoma medical treatment (77.7% [n = 310]) and glaucoma surgery (86.2% [n = 344]), with selective laser trabeculoplasty being most common (74.4% [n = 297]). Among the eyes, 22.3% (n = 89) had not undergone any form of glaucoma treatment (i.e., no previous glaucoma medications, laser therapy, or surgery).

Table 1. Baseline Characteristics of Cohort

| Characteristic | Data |
|---|---|
| No. of MPCPC procedures | 399 |
| No. of eyes | 342 |
| No. of patients | 214 |
| No. of eyes requiring re-treatment | 57 |
| Mean age (SD), yrs | 67 (13) |
| Median age (range), yrs | 69 (26–97) |
| Female gender, no. (%) | 98 (46) |
| Ethnicity, no. of patients (%) | |
| White | 114 (53) |
| Asian | 75 (35) |
| Hispanic | 17 (8) |
| Black | 8 (4) |
| Systemic comorbidity, no. patients (%) | |
| Diabetes mellitus | 69 (32) |
| Hypertension | 102 (48) |
| Diagnosis, no. of eyes (%) | |
| Primary open-angle glaucoma | 223 (56) |
| Chronic angle-closure glaucoma | 44 (11) |
| Neovascular | 36 (9) |
| Normal-tension glaucoma | 26 (7) |
| Other secondary glaucoma* | 25 (6) |
| Ocular hypertension | 22 (5) |
| Pseudoexfoliation | 17 (4) |
| Mixed mechanism | 7 (2) |
| Severity of disease, no. of eyes (%) | |
| Ocular hypertension | 22 (5) |
| Early | 121 (30) |
| Moderate | 82 (21) |
| Advanced | 177 (44) |
| Prior treatment, no. of eyes (%) | |
| Glaucoma medications | 310 (78) |
| Any glaucoma procedure† | 344 (86) |
| Selective laser trabeculoplasty | 297 (74) |
| Laser peripheral iridotomy | 63 (16) |
| Drainage device | 33 (8) |
| Trabeculectomy | 8 (2) |
| Cataract extraction | 191 (48) |
| Naïve to any form of therapy | 89 (22) |

MPCPC = micropulse cyclophotocoagulation; SD = standard deviation.
*For example, pigment dispersion, traumatic, and uveitic or inflammatory glaucoma.
†Some eyes received more than 1 glaucoma procedure previously.

A latitude of ±1 month was observed for all postoperative time points. The follow-up cohort at POMs 1, 3, 6, and 12 comprised 94.0% (n = 375), 60.2% (n = 240), 37.0% (n = 148), and 33.6% (n = 134) of the original 399 surgeries, respectively. Overall pretreatment mean IOP was 19.8±7.4 mmHg, with 28.6% (n = 114) showing baseline IOP of more than 21 mmHg and 71.4% (n = 285) showing baseline IOP of 21 mmHg or less. With regard to laser treatment, 62.4% (n = 249) received treatment with laser power of less than 2500 mW and 37.6% (n = 150) received treatment with laser power of 2500 mmHg or more.

### Primary Outcome Measure: Effectiveness

Overall IOP reduction was 22.7%, 20.2%, 20.7%, and 23.7% from baseline IOP measurement, corresponding to mean IOP of 15.3±6.0 mmHg, 15.8±6.6 mmHg, 15.7±5.7 mmHg, and 15.1±6.3 mmHg at POMs 1, 3, 6, and 12, respectively ($P < 0.0001$ for all; Fig 1). The end point of 20% or more mean IOP reduction from baseline was achieved in 53.7% of the total cohort at POM 1 (n = 183), 49.3% of the total cohort at POM 3 (n = 106), 52.7% of the total cohort at POM 6 (n = 68), and 67.8% of the total cohort at POM 12 (n = 82). One or more repeat MPCPC treatments was administered to 14.3% of the cohort (n = 57). Micropulse cyclophotocoagulation re-treatment provided additional mean IOP reduction of 16.4% (from 18.9±6.1 mmHg to 15.8±7.1 mmHg) with each repeat treatment ($P < 0.0001$; n = 57).

Subanalysis of the NTG cohort demonstrated mean IOP reduction from baseline of 7.4% at POM 1 ($P = 0.16$). Mean IOP at baseline and POM 1, respectively, were 14.8±4.1 mmHg (n = 26) and 13.7±4.9 mmHg (n = 26). The follow-up cohort at POM 12 was too limited for analysis (n = 4).

Subanalysis of the chronic angle-closure glaucoma cohort demonstrated mean IOP reduction from baseline of 14.0% at POM 1 ($P = 0.03$) and 27.5% at POM 12 ($P = 0.01$). Mean IOP at baseline, POM 1, and POM 12 was 17.1±7.6 mmHg (n = 44), 14.7±6.0 mmHg (n = 38), and 12.4±1.7 mmHg (n = 14), respectively.

Subanalysis based on IOP stratification demonstrated mean IOP reduction at POM 1 of 32.0% when baseline IOP was more than 21 mmHg and 17.1% when baseline IOP was 21 mmHg or less ($P < 0.0001$). The corresponding values at POM 12 were 30.5% and 20.1% ($P < 0.0001$). Mean IOP at baseline, POM 1, and POM 12, respectively, was 28.2±8.0 mmHg (n = 114), 19.2±7.9 mmHg (n = 103), and 19.6±8.3 mmHg (n = 38) in the cohort with IOP of more than 21 mmHg and 16.4±3.3 mmHg (n = 285), 13.6±3.9 mmHg (n = 238), and 13.1±3.6 mmHg (n = 83) in the cohort with IOP of 21 mmHg or less.

Subanalysis based on laser power stratification demonstrated mean IOP reduction at POM 1 of 28.7% with laser power of 2500 mW or more and 18.3% with laser power of less than 2500 mW ($P < 0.0001$). The corresponding values at POM 12 were 31.5% and 17.8%, respectively ($P = 0.02$). Mean IOP at baseline, POM 1,

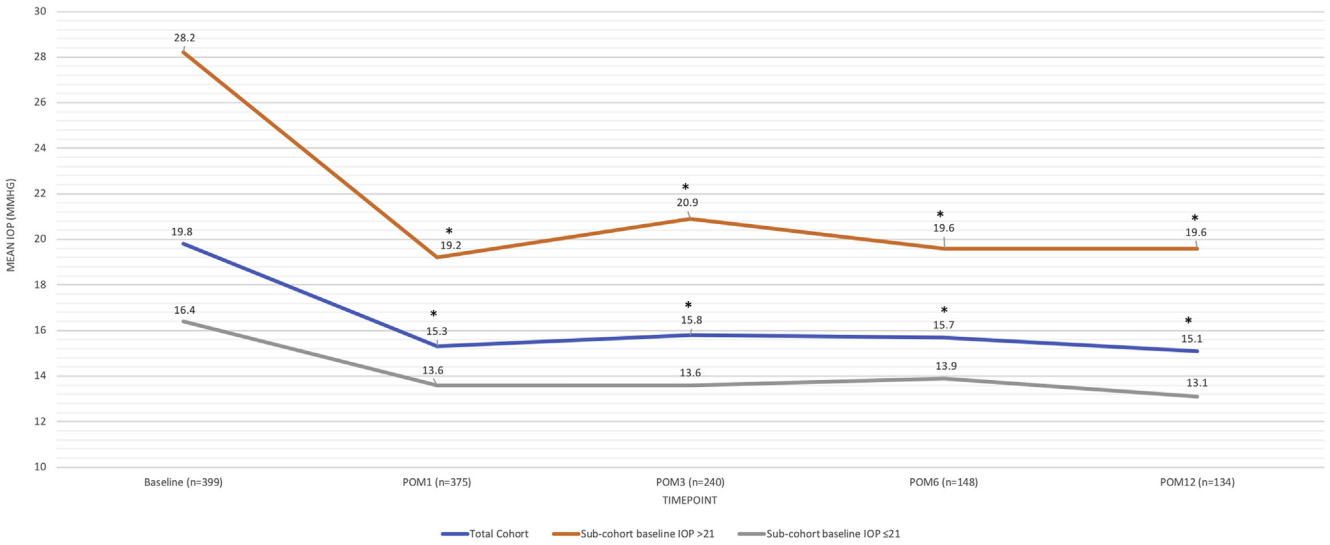


**Figure 1.** Graph showing 1-year mean intraocular pressure (IOP) progression curve of 3 cohorts (total and 2 cohorts based on IOP stratification). The x-axis represents each evaluation time point, and the y-axis represents mean IOP in mmHg. The asterisk denotes *P* value of less than 0.05. POM = postoperative month.

and POM 12 was 21.6±9.4 mmHg (n = 150), 15.4±7.5 mmHg (n = 128), and 14.8±7.7 mmHg (n = 41), respectively, in the cohort receiving laser power of 2500 mW or more and 18.6±5.6 mmHg (n = 249), 15.2±4.8 mmHg (n = 213), and 15.3±5.4 mmHg (n = 80) in the cohort receiving laser power of less than 2500 mW.

Multivariate analysis for effectiveness demonstrated that higher baseline IOP, greater number of baseline topical glaucoma medications, and surgeon were significant predictors of IOP reduction ($P < 0.0001$, $P < 0.03$, and $P < 0.003$, respectively). Each 1-mmHg increase in baseline IOP was associated with a −0.37-mmHg reduction in IOP (95% confidence interval [CI], −0.32 to −0.41 mmHg); IOP stratification was not used in the multivariate analysis. The cohort of one surgeon (D.Y.) achieved greater IOP reduction (−1.36 mmHg; 95% CI, −2.24 to −0.48 mmHg). Regression estimates for number of baseline topical glaucoma medications were as follows: −0.89 mmHg for 1 medication (95% CI, −1.71 to −0.08 mmHg), −1.05 mmHg for 2 medications (95% CI, −2.04 to −0.05 mmHg), −1.28 mmHg for 3 medications (95% CI, −3.12 to 0.55 mmHg), and −4.80 mmHg for 4 medications (95% CI, −8.82 to −0.78 mmHg). Other predictors did not reach statistical significance in the multivariate analysis, in particular, disease severity, ethnicity, and diagnoses ($P = 0.67$, $P = 0.86$, and $P = 0.92$, respectively). The dose-response relationship between laser power and effectiveness lessened after the regression model-controlled baseline IOP ($P = 0.57$). The regression model demonstrated no difference in IOP reduction between surgeons ($P = 0.27$) unless baseline IOP was introduced as a covariate ($P = 0.01$). Baseline IOP was the only predictor between surgeons that reached statistical significance ($P = 0.03$).

Table 2. Mean Change in Number of Topical Glaucoma Medications after Micropulse Cyclophotocoagulation

| Time Point | No. | Mean (Standard Deviation) |
|---|---|---|
| Baseline | 399 | 1.6 (1.1) |
| Postoperative month 1 | 375 | 1.4 (1.1) |
| Postoperative month 3 | 240 | 1.5 (1.1) |
| Postoperative month 6 | 148 | 1.6 (1.1) |
| Postoperative month 12 | 134 | 1.6 (1.1) |

## Secondary Outcome Measures

**Glaucoma Medications.** Before surgery, 77.7% of patients (n = 310) were receiving topical glaucoma medications, and 70.9% (n = 266), 78.2% (n = 186), 79.7% (n = 118), and 79.1% (n = 106) of patients were receiving topical glaucoma medications at POMs 1, 3, 6, and 12, respectively. Overall, the mean number of topical glaucoma medications was 1.6±1.1 at baseline and 1.4±1.1, 1.5±1.1, 1.6±1.1, and 1.6±1.1 at POMs 1, 3, 6, and 12, respectively ($P = 0.01$, $P = 0.27$, $P = 0.79$, and $P = 0.91$, respectively; Table 2). Of the 25 patients initially receiving oral glaucoma medication, 15 (60.0%) and 18 (72.0%) ceased after surgery at POM 1 and POM 12, respectively.

**Visual Acuity.** Change in lines of Snellen CDVA compared with baseline at each postoperative time point is demonstrated in Table 3. Percentages were calculated based on number of patients at that time point. At POM 1, 50.1% (n = 188) experienced no change, 35.2% (n = 132) experienced a decrease of 1 line or more, and 14.7% (n = 55) experienced an increase of 1 line or more. Nine eyes of 9 patients showed CDVA of light perception to no light perception at various time points after treatment. Eight of the 9 eyes showed preoperative CDVA of 20/400 or worse (20/400, n = 1; counting fingers, n = 5; hand movements, n = 2). The cause of visual loss was as follows: 1 eye demonstrated CDVA intervisit fluctuation, with light perception vision being measured before baseline assessment; 4 eyes demonstrated glaucoma progression; 1 eye demonstrated proliferative diabetic retinopathy; 1 eye demonstrated proliferative diabetic retinopathy and glaucoma progression; and 2 eyes demonstrated penetrating keratoplasty graft failure.

Table 3. Change in Corrected Distance Visual Acuity (Lines of Snellen Visual Acuity) Compared with Baseline at Each Postoperative Time Point

| Vision Range | % (No.) | | | |
|---|---|---|---|---|
| | *Postoperative Month 1* | *Postoperative Month 3* | *Postoperative Month 6* | *Postoperative Month 12* |
| Lost to LP or NLP | <1 (3) | 5 (5) | 5 (9) | 3 (4) |
| −11 | <1 (1) | 0 (0) | 0 (0) | 0 (0) |
| −10 | 0 (0) | 0 (0) | <1 (1) | 2 (2) |
| −9 | <1 (2) | 0 (0) | 0 (0) | 0 (0) |
| −8 | 1 (4) | 0 (0) | <1 (1) | 0 (0) |
| −7 | <1 (2) | 0 (0) | <1 (1) | 4 (5) |
| −6 | <1 (3) | 1 (3) | 2 (3) | <1 (1) |
| −5 | 1 (5) | 3 (6) | 2 (3) | 1 (2) |
| −4 | 2 (8) | 3 (7) | 4 (6) | 4 (5) |
| −3 | 4 (14) | 8 (19) | 6 (11) | 4 (5) |
| −2 | 10 (38) | 10 (24) | 11 (20) | 12 (16) |
| −1 | 14 (52) | 18 (44) | 18 (29) | 14 (19) |
| 0 | 50 (188) | 37 (88) | 31 (46) | 37 (49) |
| 1 | 8 (30) | 11 (26) | 11 (16) | 13 (18) |
| 2 | 5 (17) | 3 (8) | 3 (5) | 2 (3) |
| 3 | <1 (3) | 1 (2) | 1 (2) | 2 (2) |
| 4 | <1 (3) | 1 (3) | 1 (2) | 3 (4) |
| 5 | <1 (2) | 1 (2) | 1 (2) | 2 (2) |
| 6 | 0 (0) | 2 (4) | 0 (0) | 0 (0) |
| 7 | 0 (0) | 1 (3) | 0 (0) | 0 (0) |
| Total | 372 | 239 | 148 | 133 |

LP = light perception; NLP = no light perception.

Table 4 demonstrates vision changes based on a 3-cohort stratification of Snellen VA: VA of 20/40 or better, VA of 20/50 to 20/80, and VA of 20/100 or worse. When comparing between stratification groups from baseline to POM 12, patient percentages were relatively similar. More specifically, at POM 12, a 6% increase was observed in the number of patients in the cohort with VA of 20/40 or better (58% vs. 52%), a 5% decrease was observed in the number of patients in the cohort with VA of 20/50 to 20/80 (11% vs. 16%), and a 1% decrease was observed in the number of patients in the cohort with VA of 20/100 or worse (31% vs. 32%). At POM 1, 16.4% of patients (n = 61) demonstrated vision loss (defined by 2 lines or more of Snellen CDVA), with visual recovery to baseline occurring in 53.6% (n = 30) by POM 12. The cause of visual loss at POM 1 (Table 5) most commonly was attributable to, in descending order of prevalence, ocular surface disease (OSD; 21.3% [n = 13]), cataract (8.2% [n = 5]), and retinal or macular pathology (6.6% [n = 4]). No cases of neurotrophic keratitis were observed.

Table 4. Stratification of Snellen Corrected Distance Visual Acuity at Each Time Point

| Time Point | No. | Snellen Corrected Distance Visual Acuity Range (%) | | |
|---|---|---|---|---|
| | | *20/40 or Better* | *20/50–20/80* | *20/100 or Worse* |
| Baseline | 399 | 52 | 16 | 32 |
| Postoperative month 1 | 375 | 52 | 16 | 32 |
| Postoperative month 3 | 240 | 52 | 19 | 29 |
| Postoperative month 6 | 148 | 48 | 20 | 32 |
| Postoperative month 12 | 134 | 58 | 11 | 31 |

Table 5. Cause of Vision Loss (≥2 Lines*) at Postoperative Month 1

| Cause | No. (%) |
|---|---|
| Ocular surface disease | 13 (21.3) |
| Cataract | 5 (8.2) |
| Retinal or macular pathology† | 4 (6.6) |
| Anterior uveitis | 3 (4.9) |
| Symptomatic mydriasis | 3 (4.9) |
| Hypotony or serous choroidal detachment | 2 (3.2) |
| Corneal abrasion | 1 (1.6) |
| Posterior capsule opacification | 1 (1.6) |
| Intraocular pressure spike | 1 (1.6) |
| Unspecified | 28 (46.0) |
| Total | 61 (100) |

*Not attributable to a shift in refractive error as determined by pinhole visual acuity or subjective autorefraction.
†For example, proliferative diabetic retinopathy, epiretinal membrane, ischemic macula, and macular pucker.

Stratifying by laser energy used, vision loss at POM 1 was 27.0% (n = 38) in the cohort with laser power of 2500 mW or more and 16.7% (n = 39) in the cohort with laser power of less than 2500 mW ($P = 0.07$). Of the 57 repeat MPCPC treatments, vision loss was 12.3% (n = 7). All 7 eyes demonstrated vision loss from the initial treatment such that subsequent re-treatment did not result in further deterioration of vision. Second treatment accounted for 82.5% (n = 47) of repeat MPCPC procedures.

Multivariate analysis for vision loss demonstrated increased logMAR VA compared with baseline at all postoperative time points ($P < 0.0001$). Lower baseline logMAR VA and greater number of baseline topical glaucoma medications were significant predictors of increased logMAR VA ($P < 0.0001$ and $P < 0.01$, respectively).

**Adverse Events.** Ocular adverse events at POM 1 are demonstrated in Table 6. The most common adverse event was vision loss (16.3% [n = 61]), followed by IOP spike (9.1% [n = 14]), cataract (2.7% [n = 7]), iritis (1.6% [n = 6]), symptomatic mydriasis (1.6% [n = 6]), hypotony (1.2% [n = 4]), and vitreous hemorrhage (0.3% [n = 1]). No cases of persistent inflammation, persistent hypotony, macular edema, painful eye, phthisis bulbi, or sympathetic ophthalmia were observed. Eight patients required

Table 6. Ocular Adverse Events after Micropulse Cyclophotocoagulation at Postoperative Month 1

| Adverse Event | No. of Eyes (%)* |
|---|---|
| Visual acuity loss | 61 (16.3) |
| Intraocular pressure spike | 34 (9.1) |
| Cataract | 7 (1.9) |
| Iritis | 6 (1.6) |
| Symptomatic mydriasis | 6 (1.6) |
| Hypotony | 4 (0.3) |
| Vitreous hemorrhage | 1 (0.3) |

*Three hundred seventy-five eyes at postoperative month 1.

additional glaucoma surgical intervention during the study period: gel microstent (XEN; Allergan Inc, Dublin, Ireland; n = 6), trabecular micro-bypass stent (iStent inject; Glaukos Corporation, San Clemente, CA; n = 1), and supraciliary microstent (CyPass; Novartis, Basel, Swizerland; n = 1).

## Discussion

Kuchar et al[18] defined surgical success as IOP of 21 mmHg or less or a reduction of at least 20%. Twenty percent as a value of significance for reduction of IOP has been used in previous glaucoma studies, such as the MPCPC studies of Williams et al[19] and Zaarour et al[20] and the Tube Versus Trabeculectomy Study.[21] Our study demonstrated mean IOP reduction of 20% or more from baseline maintained to 1 year in 68% of patients, whereas Sarrafpour et al[22] demonstrated this effectiveness end point in 76% of patients. Zaarour et al demonstrated an attrition of effectiveness over time in that although 87% (n = 65) of their cohort achieved 20% or more IOP reduction at POM 1, only 57% (n = 34) achieved the same end point at POM 12. On the contrary, effectiveness was sustained in our study: 54% and 68% achieved 20% or more IOP reduction at POM 1 (n = 181) and POM 12 (n = 82) respectively. Our study demonstrated mean IOP reduction of 23% at POM 1, whereas other studies by Sarrafpour et al[22] and Emanuel et al[23] demonstrated mean IOP reduction of closer to 40% at POM 1. Our study demonstrated mean IOP reduction of 24% at 1 year, whereas Sarrafpour et al[22] demonstrated mean IOP reduction of 46% at the corresponding time point. The more modest, although statistically significant, IOP reduction in our study may not be entirely comparable with that of other published cohorts because of the inherent differences in our patient population. Our cohort showed demonstrably lower mean baseline IOP: 71% showed IOP of 21 mmHg or less before treatment. Other published cohorts demonstrated higher pretreatment IOPs: those of Tan et al,[24] Kuchar et al,[18] Aquino et al,[15] Emanuel et al,[23] and Sarrafpour et al[22] showed baseline IOPs of 39.3 mmHg, 37.9 mmHg, 36.5 mmHg, 27.7 mmHg, and 25.5 mmHg, respectively. Patients with lower baseline IOP have demonstrated reduced treatment effect to therapy elsewhere in the literature,[25] an effect we found consistent in our subcohort with baseline IOP of 21 mmHg or less who showed IOP reduction at POM 1 of only 17% (mean IOP from 16.4 mmHg to 13.6 mmHg). Our multivariate regression findings further support this because baseline IOP was found to be one of the strongest predictors of effectiveness. In our study, because the subgroup with baseline IOP of 21 mmHg or less comprised nearly three quarters of the cohort, our overall mean IOP reduction was skewed. Conclusions drawn from IOP stratification, namely 32.0% mean IOP reduction when pretreatment IOP was more than 21 mmHg, is likely more meaningful and on par with effectiveness results of other published data. The literature on re-treatment is limited.[22] The ability to reduce IOP further by 16% in our cohort with each repeat MPCPC treatment was an important finding, because consideration could be made for more routine re-treatment to augment effectiveness if target IOP is not achieved after initial MPCPC.

Our cohort was broad in that it was inclusive of the following: OHT eyes, eyes with all types of glaucoma (including NTG) and all levels of severity (including well-sighted eyes), surgically naive eyes, and treatment-naïve eyes. This contrasts with the cohorts of Emanuel et al[23] and Aquino et al,[15] which comprised refractory glaucoma only, and that of Kuchar et al,[18] which comprised advanced glaucoma only. Although the NTG cohort was small, a limited IOP response to MPCPC in this subgroup and in the subgroup with baseline IOP of 21 mmHg or less was demonstrated. These data may help to guide patient selection for MPCPC treatment. Laser power in our study ranged from 900 to 2800 mW. The use of subconjunctival anesthesia may have allowed for higher treatment power to be tolerated by patients. Laser power of 2500 mW or more augmented effectiveness by 10% (29% IOP reduction with power of 2500 mW or more vs. 18% with power of less than 2500 mW) at POM 1 without increased risk of visual loss. Thirty-eight percent of our cohort (n = 150) received laser power of 2500 mW or more, suggesting a skew in our data set toward the cohort that received power of less than 2500 mW (62.4% [n = 249]). In our study, a positive correlation between laser power and effectiveness was demonstrated with laser power stratification. Similarly, Sarrafpour et al[22] found a dose-response relationship between laser power and effectiveness with 57% IOP reduction at 2500 mW, the highest power setting used in their cohort, and 30% reduction at 2000 mW. Laser power in other studies was typically lower (2000 mW or less).[15,18,23] In our study, the dose-response relationship between laser power and effectiveness diminished when baseline IOP was controlled for in the regression analysis. Given that baseline IOP was a strong predictor for IOP reduction and a confounder for the association between laser power and IOP reduction, titration of laser power to higher baseline IOP may be a study limitation. Randomization of laser power may have allowed for a better determination of the dose-response relationship between laser power and effectiveness. Although a specific dose-response titration curve for each power setting would have been interesting, our study was not designed to ascertain this.

It is recognized that lower laser power may be offset by longer treatment duration to maintain equivocal total laser energy delivered. The duration of 160 seconds per eye in our study varied from that of previous studies, and currently a lack of consensus prevails in the literature regarding treatment duration. One hundred sixty seconds was selected in accordance with the MPCPC training in Canada that surgeons received from the manufacturer, Iridex Corporation. Sarrafpour et al[22] and Tan et al[24] used a duration of 100 seconds per eye. Kuchar et al[18] used 100 to 240 seconds per eye. Emanuel et al[23] used a higher setting than in our cohort, with a range between 180 and 360 seconds. Fifty percent of their cohort received 360 seconds of treatment, which is double to triple that of the aforementioned studies. In our study, treatment duration was kept as a control with power varied.

More than half of our study's cohort (52%) did not demonstrate visual changes at POM 1. Interestingly, 14% showed an improvement of 1 line or more of Snellen CDVA

at the same time point. Of the 34% of patients who showed a decrease of 1 line or more of CDVA at POM 1, the VA of more than half was correctable with refraction or pinhole VA. We postulate that refractive error may be the result of tear film instability, ciliary body rotation, zonular tightening, or lenticular changes. Only 16% (n = 61) at POM 1 experienced vision loss, as defined by a loss of 2 lines or more of Snellen VA, with OSD being the most prevalent cause. All patients were treated for OSD before MPCPC intervention. Although the mechanism of OSD aggravation after MPCPC has not been well elucidated in the literature, we suggest 4 potential mechanisms: limbal stem cell destruction in the limbal area, goblet cell destruction in the postlimbal area, release of inflammatory mediators, and potential noncompliance with postoperative topical steroid medications. Because no cases of neurotrophic keratitis were present in our cohort, limbal stem cell deficiency seems less plausible. Nevertheless, it is reassuring that 54% of patients (n = 30) who experienced visual loss recovered to baseline CDVA by POM 12. Furthermore, in our 3-cohort stratification of Snellen CDVA (VA of 20/40 or better, VA of 20/50 to 20/80, and VA of 20/100 or worse), percentages of patients in each stratification group at baseline compared with POM 12 were similar. In contrast, Sarrafpour et al[22] demonstrated greater visual loss at POM 1 compared with our cohort, with 38% of patients having lost 2 lines or more of Snellen VA. Furthermore 19% did not recover to baseline vision by POM 3 or 6.

Traditionally, cyclodestructive procedures were reserved for poorly sighted eyes with refractory glaucoma resulting from significant complications.[3,26] Persistent hypotony, painful eye, phthisis bulbi, and sympathetic ophthalmia are some of the most feared complications in glaucoma surgery. None of these major complications were reported in our study, but other complications, such as macular edema, easily could be missed or not noted in a retrospective chart review. The Tube Versus Trabeculectomy study[21] highlighted persistent hypotony as a marker of failure in the affected eye, with 13% of patients (n = 3) in the tube group and 31% of patients (n = 13) in the trabeculectomy group experiencing persistent hypotony. With traditional TSCPC, Ramli et al[27] demonstrated that 39% of their cohort (n = 16) experienced persistent hypotony extending to 12 months. Aujla et al[28] identified the presence of phthisis bulbi in 6% of patients (n = 4) after traditional TSCPC. Aquino et al[15] demonstrated persistent hypotony in 22% of patients (n = 5) and phthisis bulbi in 4% of patients (n = 1) in the traditional TSCPC cohort as opposed to no cases of either adverse event in the MPCPC cohort. Both Kuchar et al[18] and Emanuel et al[23] reported no incidence of phthisis bulbi or symptomatic ophthalmia in their MPCPC cohorts. Sarrafpour et al[22] reported no incidence of macular edema or phthisis in their cohort after MPCPC. In our study, the lack of persistent inflammation, persistent hypotony, painful eye, phthisis, and sympathetic ophthalmia was consistent with published data to date and supports the favorable safety profile of MPCPC.

Although some evidence supports MPCPC as being less painful than traditional TSCPC,[27] any type of cyclophotocoagulation may cause significant pain. The ocular surface is innervated by long ciliary nerves that branch from the nasociliary nerve, which is connected to the ophthalmic branch (V1) of cranial nerve V (trigeminal nerve) that feeds directly into the brainstem. We used subconjunctival anesthesia in addition to concurrent intravenous neurolept anesthesia for patient comfort. However, swelling of the conjunctiva resulting from anesthetic volume and the presence of subconjunctival heme may have blunted absorption and penetration of the laser energy, potentially leading to decreased treatment effect. To increase the likelihood of achieving targeted tissue effect, moderate to firm globe pressure was applied and higher titration of laser power was used.

In our treatment protocol, we included brimonidine 0.15% before surgery to reduce the amount of subconjunctival hemorrhage.[29–31] In the current design of the MicroPulse P3 probe, the laser filament protrudes from the end of the probe over a small surface area. This can be traumatic because the probe is pressed against the globe and swept across the treatment area repeatedly. Consideration may be given to re-designing the MicroPulse P3 probe with a larger surface area of contact and a less conspicuous protrusion of the laser filament to reduce pressure and trauma on the globe. A coupling agent (lidocaine 2% gel in conjunction with a viscous artificial tear) was used to increase transmission of energy at the recommendation of the laser manufacturer. Iridex Corporation estimated that without a coupling agent, transmission of laser energy would decrease by 50% because of scattering of light energy. Alternative local anesthesia such as sub-Tenon's, peribulbar, or retrobulbar may lead to less swelling of the conjunctiva and decreased risk of subconjunctival hemorrhage; however, it is associated with other significant risks such as vision loss and systemic side effects. Although we did not transilluminate to localize the position of the ciliary body at the time of laser treatment,[24] transillumination may allow for a more targeted and individualized approach to treatment structures. Recommendations from Iridex Corporation suggest that increasing the dwell time from 10-second passes per hemisphere to 20-second passes may correlate with greater effectiveness. This, along with safety implications, have yet to be substantiated in the literature. Mydriasis often is caused by damage to the short ciliary nerves. Mydriasis was recorded as a complication only when patients were symptomatic, usually as a result of photosensitivity, glare, or loss of accommodation. Given that only symptomatic mydriasis was recorded in our study, it is likely that we underestimated mydriasis secondary to MPCPC. Although we made every effort to avoid the 3- and 9-o'clock positions, variable excyclotorsion of the globe with the patient in the supine position has been well established. Marking the 3- and 9-o'clock position at the limbus with the patient upright before treatment may mitigate the risk of damaging ciliary nerves.

Sarrafpour et al[22] reported that 43% of patients achieved a reduction of at least 1 topical medication at POM 12, and 73.3% (n = 11) ceased oral glaucoma medications at the same time point. The latter result is consistent with the findings in our study. Sixty percent of our cohort ceased

oral medications after surgery at POM 1 and sustained this to POM 12, which further supports the effectiveness of MPCPC in controlling IOP. However, our study differed in that the reduction in the mean number of topical glaucoma medications between baseline and POM 1 ($P = 0.01$) was not sustained to POM 12 ($P = 0.91$). A plausible explanation has been reported in the literature that may account for the discrepancy. Higher mean preoperative IOP of 25.5 mmHg in the cohort of Sarrafpour et al[22] was associated with greater mean number of baseline topical glaucoma medications ($3.1\pm1.1$). In contrast, our cohort reported a mean of $1.6\pm1.1$ baseline topical glaucoma medications, which suggests that MPCPC may be more successful in reducing glaucoma medications in patients with relatively higher baseline IOP. This was supported by our regression analysis in that higher baseline IOP and greater number of baseline topical medications were significant predictors for effectiveness. Extrapolating, in patients with fewer topical glaucoma medications and lower pretreatment IOP, MPCPC may be better suited as adjunctive therapy, rather than as replacement therapy.

Because MPCPC is a relatively novel procedure in the glaucoma treatment paradigm, the evidence regarding effectiveness and safety is limited. It is difficult to compare studies given that treatment parameters may not be defined thoroughly and standardization of treatment parameters currently is lacking. As such, we expounded on our technique fully. Parameters include, but are not limited to, local versus systemic anesthesia, liquid interface coupling agent, probe orientation, position from limbus, angulation of probe, pressure on globe, dwell time, quadrants versus hemisphere, power, duration of treatment, postoperative medication, and repeat treatments. As mentioned previously, duration of treatment was constant in all patients with only 1 variable, titrated laser power, which is similar to the treatment protocol of Sarrafpour et al.[22]

Finally, although MPCPC is becoming more widely adopted, the mechanism of action of MPCPC is yet to be elucidated fully. Although categorized as cycloablative, data support MPCPC as not being truly cyclodestructive in nature.[24] No blanching of the ciliary epithelium of the ciliary body occurs, as demonstrated by Tan et al,[24] which is in marked contrast to TSCPC. Anatomically, MPCPC with the orientation of the probe perpendicular to the probe 1 to 2 mm behind the limbus targets pars plana, rather than pars plicata, the part of the ciliary body responsible for aqueous production,[15] making decreased aqueous production a less likely mechanism of action. Preliminary research demonstrates contraction of the trabecular meshwork with MPCPC laser energy being applied, which suggests IOP lowering at least potentially in part via increased aqueous outflow.[32] Inflammation of the ciliary body may reduce aqueous formation and possibly enhance uveoscleral outflow.[33,34] However, if the mechanism of action of MPCPC was attributable to inflammation, theoretically the IOP-lowering effect should be transient and dissipate as soon as inflammation is resolved. Activation of a cellular biochemical cascade may decrease IOP, although publications in this area are pending.

In conclusion, this study adds to the growing body of evidence in support of the effectiveness and safety of MPCPC. It is important to acknowledge that our study was limited in its retrospective nature and in the number of patients lost to follow-up over a 1-year period. Nevertheless, this series represents a large MPCPC cohort and encompasses the following: eyes with OHT and all types and severities of glaucoma (including NTG, well-sighted eyes, and treatment-naïve eyes), repeat treatments, and detailed specifications of laser treatment parameters. In our study, MPCPC demonstrated effectiveness in lowering IOP maintained to 1 year with a favorable safety profile. Subgroups with NTG and baseline IOP of 21 mmHg or less demonstrated a more limited response, which may guide patient selection. Furthermore, a dose-response relationship with respect to laser power and repeat treatments is suggested. Laser power of 2500 mW or more proved more effective, and each repeat treatment provided significant additional IOP reduction. Baseline IOP and number of baseline topical glaucoma medications were the strongest predictors for effectiveness in the regression analysis. Further rigorous studies on MPCPC, which are prospective and include treatment parameters and expanded cohorts (not limited to refractory glaucoma and poorly sighted eyes) with longer follow-up, are warranted to provide clarity on the role of this emerging therapy. We also encourage further dialogue on treatment protocol for more objective comparison of outcomes between studies and guidance for technique standardization going forward.

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## References

1. Flaxman SR, Bourne RRA, Resnikoff S, et al. Global causes of blindness and distance vision impairment 1990−2020: a systematic review and meta-analysis. *Lancet Glob Health*. 2017;5(12):1221−1234.
2. Bietti G. Surgical intervention on the ciliary body; new trends for the relief of glaucoma. *J Am Med Assoc*. 1950;142(12):889−897.
3. De Roetth Jr A. Cryosurgery for the treatment of glaucoma. *Trans Am Ophthalmol Soc*. 1965;63:189−204.
4. Walton DS, Grant WM. Penetrating cyclodiathermy for filtration. *Arch Ophthalmol*. 1970;83(1):47−48.
5. Bellows AR, Grant WM. Cyclocryotherapy in advanced inadequately controlled glaucoma. *Am J Ophthalmol*. 1973;75(4):679−684.
6. Coleman DJ, Lizzi FL, Driller J, et al. Therapeutic ultrasound in the treatment of glaucoma. II. Clinical applications. Ophthalmology. 1985;92(3):347−353.
7. Weekers R, Lavergne G, Watillon M, et al. Effects of photocoagulation of ciliary body upon ocular tension. *Am J Ophthalmol*. 1961;52(2):156−163.
8. Wise JB, Witter SL. Argon laser therapy for open-angle glaucoma: a pilot study. *Arch Ophthalmol*. 1979;97:319−322.
9. Beckman H, Kinoshita A, Rota AN, Sugar HS. Transscleral ruby laser irradiation of the ciliary body in the treatment of intractable glaucoma. *Trans Am Acad Ophthalmol Otolaryngol*. 1972;76(2):423−436.
10. Gray RH, Nairne JH, Ayliffe WH. Efficacy of Nd-YAG laser iridotomies in acute angle closure glaucoma. *Br J Ophthalmol*. 1989;73(3):182−185.

11. Immonen IJ, Puska P, Raitta C. Transscleral contact krypton laser cyclophotocoagulation for treatment of glaucoma. *Ophthalmology*. 1994;101(5):876–882.
12. Hennis HL, Stewart WC. Semiconductor diode laser transscleral cyclophotocoagulation in patients with glaucoma. *Am J Ophthalmol*. 1992;113(1):81–85.
13. Ndulue JK, Rahmatnejad K, Sanvicente C, et al. Evolution of cyclophotocoagulation. *J Ophthalmic Vis Res*. 2018;13:55–61.
14. Dastiridou AI, Katsanos A, Denis P, et al. Cyclodestructive procedures in glaucoma: a review of current and emerging options. *Adv Ther*. 2018;35(12):2103–2127.
15. Aquino MC, Barton K, Tan AM, et al. Micropulse versus continuous wave transscleral diode cyclophotocoagulation in refractory glaucoma: a randomized exploratory study. *Clin Exp Ophthalmol*. 2015;43(1):40–46.
16. Rafuse PE, Buys YM, Damji KF, et al. Canadian Ophthalmological Society evidence-based clinical practice guidelines for the management of glaucoma in the adult eye. *Can J Ophthalmol*. 2009;44(1):S7–S54.
17. Craig JP, Nichols KK, Nichols JJ, et al. TFOS DEWS II definition and classification report. *Ocul Surf*. 2017;15(3):276–283.
18. Kuchar S, Moster MR, Reamer CB, et al. Treatment outcomes of micropulse transscleral cyclophotocoagulation in advanced glaucoma. *Lasers Med Sci*. 2016;31(2):393–396.
19. Williams AL, Moster MR, Rahmatnejad K, et al. Clinical efficacy and safety profile of micropulse transscleral cyclophotocoagulation in refractory glaucoma. *J Glaucoma*. 2018;27(5):445–449.
20. Zaarour K, Abdelmassih Y, Arej N, et al. Outcomes of micropulse transscleral cyclophotocoagulation in uncontrolled glaucoma patients. *J Glaucoma*. 2019;28(3):270–275.
21. Gedde SJ, Schiffman JC, Feuer WJ, et al. Treatment outcomes in the tube versus trabeculectomy (TVT) study after five years of follow-up. *Am J Ophthalmol*. 2012;153(5):789–803.
22. Sarrafpour S, Saleh D, Ayoub S, et al. Micropulse transscleral cyclophotocoagulation: a look at long-term effectiveness and outcomes. *Ophthalmol Glaucoma*. 2019;2(3):167–171.
23. Emanuel ME, Grover DS, Fellman RL, et al. Micropulse cyclophotocoagulation: initial results in refractory glaucoma. *J Glaucoma*. 2017;26(8):726–729.
24. Tan AM, Chockalingam M, Aquino MC, et al. Micropulse transscleral diode laser cyclophotocoagulation in the treatment of refractory glaucoma. *Clin Exp Ophthalmol*. 2010;38(3):266–272.
25. Lindén C, Heijl A, Jóhannesson G, et al. Initial intraocular pressure reduction by mono-versus multi-therapy in patients with open-angle glaucoma: results from the glaucoma intensive treatment study. *Acta Ophthalmol*. 2018;96(6):567–572.
26. Covell LL, Batungbacal RT. Cyclodiathermy in glaucoma. *Am J Ophthalmol*. 1955;40(1):77–82.
27. Ramli N, Htoon HM, Ho CL, et al. Risk factors for hypotony after transscleral diode cyclophotocoagulation. *J Glaucoma*. 2012;21(3):169–173.
28. Aujla JS, Lee GA, Vincent SJ, Thomas R. Incidence of hypotony and sympathetic ophthalmia following trans-scleral cyclophotocoagulation for glaucoma and a report of risk factors. *Clin Exp Ophthalmol*. 2013;41(8):761–772.
29. Rodríguez-Galietero A, Martínez JV, Del Buey A, et al. Use of brimonidine before LASIK with femtosecond laser-created flaps for the correction of myopia: a contralateral eye study. *J Refract Surg*. 2010;26(1):28–32.
30. Pasquali TA, Aufderheide A, Brinton JP, et al. Dilute brimonidine to improve patient comfort and subconjunctival hemorrhage after LASIK. *J Refract Surg*. 2013;29(7):469–475.
31. Norden RA. Effect of prophylactic brimonidine on bleeding complications and flap adherence after laser in situ keratomileusis. *J Refract Surg*. 2002;18(4):468–471.
32. Khan F, Krishna R. Transscleral micropulse CPC in open-angle glaucoma. Glaucoma Today. *January/February*. 2019;54–55. http://glaucomatoday.com/2019/02/transscleral-micropulse-cpc-in-open-angle-glaucoma/; Accessed 29.05.19.
33. Liu GJ, Mizukawa A, Okisaka S. Mechanism of intraocular pressure decrease after contact transscleral continuous-wave Nd:YAG laser cyclophotocoagulation. *Ophthalmic Res*. 1994;26(2):65–79.
34. Schubert HD, Agarwala A. Quantitative CW Nd:YAG pars plana transscleral photocoagulation in post-mortem eyes. *Ophthalmic Surg*. 1990;21(12):835–839.


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Abbreviations and Acronyms:
**CDVA** = corrected distance visual acuity; **CI** = confidence interval; **IOP** = intraocular pressure; **logMAR** = logarithm of the minimum angle of resolution; **MPCPC** = micropulse cyclophotocoagulation; **NTG** = normal-tension glaucoma; **OHT** = ocular hypertension; **OSD** = ocular surface disease; **POM** = postoperative month; **TSCPC** = transscleral cyclophotocoagulation; **VA** = visual acuity.


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