Published in final edited form as:

J Glaucoma. 2012 February ; 21(2): 83–88. doi:10.1097/IJG.0b013e31820bd1ce.

Intermediate term safety and efficacy of transscleral cyclophotocoagulation after tube shunt failure

Peter J. Ness, MD¹, Mahmoud A. Khaimi, MD², Robert M. Feldman, MD¹, Rania Tabet, MD¹, Steven R. Sarkisian Jr, MD², Gregory L. Skuta, MD², Alice Z. Chuang, PhD¹, and Kimberly A. Mankiewicz, PhD¹

¹ Robert Cizik Eye Clinic, The University of Texas Medical School at Houston, Richard S. Ruiz, MD Department of Ophthalmology and Visual Science, Houston, TX USA

² Dean McGee Eye Institute, University of Oklahoma College of Medicine, Department of Ophthalmology, Oklahoma City, OK, USA.

Abstract

Purpose—To determine the efficacy and safety of diode transscleral cyclophotocoagulation (TSCPC) after tube shunt failure.

Patients and Methods—The patient population consisted of 32 eyes of 31 patients with uncontrolled glaucoma. Each eye had a previously implanted aqueous tube shunt and was currently on maximally tolerated medication. Each eye also underwent TSCPC treatment using the Iridex (Mountain View, CA) diode laser with a maximum of 360 degrees of treatment. All 31 charts were reviewed for data pertaining to demographics, treatment, ocular history, and follow-up clinical examinations. Safety was evaluated by complication data. Efficacy was evaluated in terms of TSCPC treatment parameters (number of laser applications, laser power, application duration, and degrees of ciliary body treated), intraocular pressure (IOP), number of hypotensive medications, and any further treatment required.

Results—With a mean (SD) follow-up of 17.1 (16.3) (median = 11.7) months from the last treatment, the mean IOP decreased from 28.6 (10.2) mmHg to 16.8 (7.5) mmHg (35% reduction) at 3 months ($n = 30$, $p < 0.0001$) and to 14.7 (7.9) mmHg (43% reduction) at 1 year ($n = 13$, $p < 0.0001$). Complications included hypotony ($n = 4$), hyphema ($n = 2$), failed corneal transplant ($n = 1$), and loss of light perception ($n = 5$).

Conclusions—TSCPC has a significant ocular hypotensive effect on glaucoma refractory to both tube shunt and medical therapy. The safety of this intervention remains unclear in this high risk patient population and warrants further study.

Keywords

transscleral; cyclophotocoagulation; tube shunt; diode

Correspondence to: Robert M. Feldman, MD, Robert Cizik Eye Clinic, 6400 Fannin, 18th Floor, Houston, TX, 77030, USA. Phone: (713)-559-5200; FAX: (713)-500-0682; rmfeldman@swbell.net .

This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

Introduction

The 2 most common primary glaucoma surgical procedures are trabeculectomy and aqueous tube shunt implantation.¹ While trabeculectomy has a longer history of effective intraocular pressure (IOP) control in humans, Baerveldt glaucoma implants (tube shunts) have recently been reported to be superior as second line surgery.^{2, 3} Other popular types of tube shunts include the Molteno, Ahmed, and Krupin designs, all of which have been proven clinically safe and successful.⁴⁻⁶ Although tube shunts are generally effective, they fail in some patients, either by not attaining sufficient IOP control or causing complications in the patient that necessitate their removal.

Semiconductor diode transscleral cyclophotocoagulation (TSCPC) has been used for over 15 years to treat patients with glaucoma refractory to medical and surgical therapies, usually as the final therapeutic or palliative attempt to control IOP.⁷ Histopathological studies have proven that TSCPC effectively induces coagulative necrosis of the pars plicata with mild extension to the pars plana in the area of treatment.^{8, 9} TSCPC decreases production of aqueous, thus lowering IOP.

Cyclodestructive procedures were first introduced in the 1930's as cyclodiathermy,¹⁰ then evolved into cyclocryotherapy,^{11, 12} and lastly into today's laser cyclophotocoagulation using the argon,¹³ ruby,¹⁴ and neodymium:yttrium-aluminum-garnet (Nd:YAG) lasers.¹⁵ Numerous publications have reported significant ocular hypotensive effects in various types of glaucoma using diode TSCPC; furthermore, fewer complications have been encountered compared to previous modalities.¹³⁻¹⁷ By and large, TSCPC has been studied as a treatment option for glaucoma refractory to medical and/or surgical means, but a small and growing number of investigations are giving promising results for TSCPC as a primary surgical method.¹⁸⁻²⁰

Although a growing number of surgeons are using TSCPC in patients with failed tube shunts, little evidence supporting using TSCPC in patients with glaucoma refractory to tube shunts and medical therapy has been published. The aim of our study is to evaluate the efficacy and safety of TSCPC after tube shunt failure in eyes that still possess a functioning tube shunt and are on maximally tolerated medical therapy. To the best of our knowledge, our study describes the largest sample size published to date and is the first study to analyze and discuss prognostic factors for efficacy in such a situation.

Patients and Methods

The patients included in this study were treated at the Robert Cizik Eye Clinic, formerly the Hermann Eye Center, in Houston, TX or at the Dean McGee Eye Institute in Oklahoma City, OK. We first obtained approval for this study from the University of Texas Health Science Center at Houston and University of Oklahoma Institutional Review Boards, then retrospectively reviewed 48 charts of patients treated with diode TSCPC between January 1, 2003 and December 1, 2007. This research adhered to the tenets of the Declaration of Helsinki and was HIPAA-compliant. Patients had to be at least 18 years of age with an eye or eyes that had previously undergone tube shunt implantation and currently had an IOP above target while on maximum medical therapy. Eyes were excluded if they had received cyclodestructive therapy prior to tube shunt implantation or the initiation of the investigation period, so as to avoid confounding factors introduced by previously therapy. Other reasons for exclusion were removal of the tube shunt before TSCPC or availability of less than 1 week of post-TSCPC follow-up data. Of the charts reviewed, 32 eyes from 31 patients were included in our study.

We reviewed each chart for demographic data, type and date of tube shunt implant, any tube shunt revisions or removal, TSCPC procedural details, and follow-up period. The pertinent procedural details gathered were the number of applications (or “shots”), degrees of ciliary body treated, energy setting, and duration of each application. Pre-TSCPC data collected included IOP, the number of IOP-lowering medications, and best corrected visual acuity (BCVA). A single pre-TSCPC IOP value was obtained by averaging the last 3 measurements within the 3 months prior to the TSCPC procedure. Follow-up data collected consisted of follow-up duration, IOP, number of IOP-lowering medications, BCVA, any further surgeries, and any complications at each visit.

The outcome of the study was both efficacy and safety of TSCPC for treatment of glaucoma refractory to tube shunt and medical therapy. We evaluated efficacy in terms of post-TSCPC IOP, number of IOP-lowering medications, and BCVA. The number and type of complications determined safety of the procedure.

Numerous patients required multiple sessions of TSCPC, with varying numbers of applications and degrees of treatment. We defined the “initial treatment” as all TSCPC treatment given within the first 6 months and not to exceed 360 degrees. We intended to undertreat many patients at their first TSCPC session to avoid hypotony, anticipating that further TSCPC sessions may be required before the desired hypotensive effect was achieved. We classified any treatments received outside of these criteria as “further surgeries.”

We performed data analyses using SAS 9.1 (SAS Institute Inc., Cary, NC). We used the frequency and percentage to summarize discrete variables: site, race, gender, type of glaucoma, type of shunt, and complications. We used the mean, standard deviation (SD), and range to summarize continuous variables: age, duration from shunt to TSCPC, IOP, number of IOP-lowering medications, BCVA (logMar), and follow-up duration. We utilized paired *t*-tests to compare efficacy variables pre-TSCPC and post-TSCPC at each follow-up. We used a mixed-effect regression model with backward elimination to identify the risk and prognostic factors that may affect the outcome of IOP, the number of IOP-lowering medications, and BCVA. The prognostic factors studied were site, age, gender, race, type of glaucoma, type of shunt, pre-TSCPC IOP, number of applications per eye, and duration from shunt to TSCPC. A *P*-value < 0.05 was considered statistically significant.

Results

We studied a total of 32 eyes from 31 patients. Patient demographics data are listed in Table 1. Prior to TSCPC, all eyes had been surgically implanted with at least 1 tube shunt. In the 32 eyes, 35 tube shunts had been placed, as 3 of the eyes had a second shunt placement. The various types of tube shunts were Baerveldt anterior chamber (*n* = 17, 53.1%), Baerveldt pars plana (*n* = 1, 3.1%), Ahmed (*n* = 8, 25.0%), Ahmed two-plate (*n* = 2, 6.3%), double-plate Molteno (*n* = 2, 6.3%), Eagle Vision (*n* = 1, 3.1%), and unknown (*n* = 1, 3.1%). The mean time between shunt placement surgery and TSCPC was 2.7 (2.2) years (range 0.3-8.8). Table 2 lists the TSCPC treatment parameters. The “initial treatment” consisted of 1 TSCPC session for 25 eyes, 2 sessions for 6 eyes and 3 sessions for only 1 eye.

The mean (SD) IOP was significantly reduced from 28.6 (10.2) mmHg to 16.8 (7.5) mmHg at 3-month follow-up (*n* = 30, *p* < 0.0001) and to 14.7 (7.9) mmHg at 1-year follow-up (*n* = 13, *p* < 0.0001) (Figure 1). Two prognostic factors were found to affect the reduction of IOP at 3-month follow-up: 1) mean pre-TSCPC IOP (0.93 [0.13] mmHg greater reduction in IOP for each additional 1 mmHg of pre-TSCPC IOP, *p* < 0.0001) and 2) total number of TSCPC applications (0.50 [0.15] mmHg greater reduction in IOP for every additional application, *p* = 0.0033).

The mean (SD) number of IOP-lowering medications was significantly reduced from 2.7 (1.3) to 1.7 (1.5) at 3-month follow-up ($p < 0.0001$) and to 1.8 (1.2) at 1-year follow-up (Figure 2). Two prognostic factors were identified to be associated with the reduction of number of IOP-lowering medications after TSCPC: 1) total number of TSCPC applications (0.08 [0.02] fewer medications for every additional shot, $p=0.0055$) and 2) number of pre-TSCPC medications (0.37 [0.17] fewer medications for every additional pre-TSCPC medication, $p = 0.034$).

At 3-month follow-up, 12 out of 30 (40%) eyes had lost at least 2 lines of BCVA, 10 (33.3%) eyes had no change, and 4 (13.3%) eyes had a better BCVA from pre-TSCPC examination (Figure 3). For statistical purposes, we considered qualitative changes in vision at the poor end of the spectrum (i.e. changes between count fingers and hand motion or hand motion and light perception) to move in increments of 3 lines of BCVA. The mean (SD) visual acuity declined 0.19 (0.42) logMar from pre-TSCPC ($p=0.0220$) at 3-month follow-up. Upon final follow-up visit, 18 (56.25%) eyes had a decrease of at least 2 lines of BCVA, 10 (31.25%) eyes had no change, and 2 (6.25%) eyes had an improved BCVA than before the procedure. No prognostic factors were found to affect BCVA.

Complications are listed in Table 3. Hypotony was defined in our study as IOP ≤ 5 mmHg over 2 consecutive follow-up visits. One eye experienced multiple complications, including an early hyphema (resolved over the first 2 months), hypotony, and later failure of penetrating keratoplasty (PKP). Another eye started with an extremely high pretreatment IOP of 58 mmHg, then developed a temporary hyphema, and eventually decreased in vision from count fingers to no light perception. A total of 5 eyes lost light perception during follow-up, 2 of which carried the diagnosis of neovascular glaucoma, 2 more had secondary glaucoma and 1 had primary open angle glaucoma.

Discussion

The results of our study provide evidence that TSCPC can be effective after tube shunt failure. When a glaucomatous eye has been refractory to both previous tube shunt implantation(s) and maximum medical therapy, the next step in treatment is neither clear nor easy. Our study provides the largest sample size to date, of which we are aware, of patients in such a situation.

Table 4 compares our study with other significant TSCPC reports. Semchyshn et al has published the only other study known to us to focus on eyes that have received tube shunts. 13 Our reduction in IOP is more modest than in their results but comparable to general TSCPC results in other studies. 13, 16, 18, 21-26 One significant difference in study design between our study and Semchyshn's is inclusion of 9 pediatric eyes (out of 21 total) by Semchyshn. 13 This difference in study design, among other sources of variability between any 2 retrospective studies, such as the treatment protocol, may account for the difference.

It is encouraging that TSCPC seems relatively efficacious even for patients who have been refractory to other treatments. The mean IOP reduction was sustained through 1 year in our study, as in most others. 13, 16, 25, 26 Additionally, the 2 statistically significant prognostic factors for efficacy make sense logically. We found the greater the number of TSCPC applications, the greater the subsequent IOP reduction. IOP reduction ought to be proportional to the number of ciliary processes destroyed; therefore, the more TSCPC applications, the greater the effect. We also found a direct relationship between higher pre-TSCPC IOP and greater reduction in IOP after TSCPC. A higher IOP before treatment corresponds with higher aqueous overproduction. Since a given area of ciliary body epithelium produces more aqueous in these eyes, a certain number of TSCPC applications

should destroy that much more aqueous production potential and, consequently, lower the IOP more. We investigated other prognostic factors (see Patients and Methods section), but none were found to be statistically significant due to the small sample size of each type of tube shunt and high variability in treatment parameters. Specifically, we found no correlation between shunt type or patient race and efficacy. Perhaps a larger sample size would reveal further prognostic factors.

The most common complication in our study was the loss of 2 or more lines of visual acuity from baseline, which is similar to other publications, although our rate was somewhat higher than others reported.^{13, 16, 18, 21-26} Loss of visual acuity following TSCPC has become a complication of great interest in recent years, and mixed results have emerged.^{15, 26} Egbert et al¹⁸ prospectively randomized 92 eyes with primary open angle glaucoma to 1 of 2 TSCPC treatment protocols as a primary surgery and then compared results to the paired eye of each patient, where the other eye of the pair was treated only with medical therapy. That author reported 23% of eyes in both the TSCPC group and the paired medical treatment group experienced a decrease in visual acuity, concluding that TSCPC does not hasten the deterioration of vision any more than standard medical therapy. In a recent study, Rotchford et al reported approximately 30% of eyes with difficult to control glaucoma and BCVA $\geq$ 20/60 to lose 2 or more lines of BCVA after diode TSCPC.²⁷

Most studies show positive results pertaining to visual acuity after TSCPC,^{13, 16, 18, 21-26} but it is unclear why there is such a high degree of variability. Our patients seem to have lost more vision, on the whole, compared to these other reports. Our study population included a very high risk group of glaucoma patients, with diagnoses such as neovascular glaucoma, and all failed tube shunt therapy, which likely skews their outcomes to be worse than those with more mild cases. Again, the study by Semchyshn et al¹³ most closely resembles ours, and it showed similar visual acuity losses. It is possible that the presence of a tube shunt somehow worsens the vision losses. Both tube shunt-focused studies (Semchyshn¹³ and ours) report the highest rates of loss of light perception, 15.6% and 19%, respectively, while others have significantly lower rates.^{16, 18, 21-26}

Other complications commonly reported in the literature are hypotony, hyphema, and phthisis.^{13, 16, 21-24} Prolonged hypotony (IOP $\leq$ 5 mmHg) is quite variable from study to study, ranging from 0-14%. Murphy et al²³ reported a 9.5% (n = 25) occurrence of hypotony in their series of 263 eyes with refractory glaucoma and, furthermore, stated that there was a correlation between high pre-TSCPC IOP or high mean energy delivered by TSCPC and hypotony. The rate of hyphema has been reported previously as below 2%,^{16, 23, 24} which is lower than the 6.3% found in our study. One of the 2 eyes that experienced temporary hyphema had neovascular glaucoma, which may account for the higher rate of hyphema we saw in our study. The charts did not document pain since many patients received prophylactic treatment.

The limitations of our study are found in its retrospective nature and relatively short duration of follow-up for many of the patients. Because of the small number of potential subjects, a proper prospective comparison study was impractical. Therefore, variation in treatment protocols and follow-up duration made statistical analyses more difficult and interpretations vague. Also, follow-up at 1 year was only 40%. Longer follow-up may reveal a clearer clinical picture, including long-term efficacy and late onset complications. The strengths of the study are its dual-site structure and large sample size of patients that had tube shunt failure.

Conclusion

Diode TSCPC is effective to control IOP in the setting of tube shunt failure. The number of IOP-lowering medications used may be significantly reduced, possibly improving the quality of life and simplifying the treatment for patients. Although complications were not uncommon, most were transient. It is unclear whether loss of vision in this high risk patient population was due to the disease state or the TSCPC treatment. Enduring complications lack solid correlation to any prognostic factors.

Acknowledgments

Financial Support: Supported in part by National Eye Institute Vision Core Grant P30EY10608, Research to Prevent Blindness, New York, NY, and the Hermann Eye Fund, Houston, TX

References

1. Joshi AB, Parrish RK 2nd, Feuer WF. 2002 survey of the American Glaucoma Society: practice preferences for glaucoma surgery and antifibrotic use. *J Glaucoma*. 2005; 14:172–174. [PubMed: 15741822]
2. Gedde SJ, Herndon LW, Brandt JD, et al. Surgical complications in the Tube Versus Trabeculectomy Study during the first year of follow-up. *Am J Ophthalmol*. 2007; 143:23–31. [PubMed: 17054896]
3. Gedde SJ, Schiffman JC, Feuer WJ, et al. Treatment outcomes in the tube versus trabeculectomy study after one year of follow-up. *Am J Ophthalmol*. 2007; 143:9–22. [PubMed: 17083910]
4. Goulet RJ 3rd, Phan AD, Cantor LB, et al. Efficacy of the Ahmed S2 glaucoma valve compared with the Baerveldt 250-mm² glaucoma implant. *Ophthalmology*. 2008; 115:1141–1147. [PubMed: 18164068]
5. Malik R, Ellingham RB, Suleman H, et al. Refractory glaucoma--tube or diode? *Clin Experiment Ophthalmol*. 2006; 34:771–777. [PubMed: 17073900]
6. Schwartz KS, Lee RK, Gedde SJ. Glaucoma drainage implants: a critical comparison of types. *Curr Opin Ophthalmol*. 2006; 17:181–189. [PubMed: 16552254]
7. Hennis HL, Stewart WC. Semiconductor diode laser transscleral cyclophotocoagulation in patients with glaucoma. *Am J Ophthalmol*. 1992; 113:81–85. [PubMed: 1728152]
8. Feldman RM, el-Harazi SM, LoRusso FJ, et al. Histopathologic findings following contact transscleral semiconductor diode laser cyclophotocoagulation in a human eye. *J Glaucoma*. 1997; 6:139–140. [PubMed: 9098823]
9. McKelvie PA, Walland MJ. Pathology of cyclodiode laser: a series of nine enucleated eyes. *Br J Ophthalmol*. 2002; 86:381–386. [PubMed: 11914203]
10. Dunphy EB, Albaugh CH. Cyclodiathermy: An Operation for the Treatment of Glaucoma. *Trans Am Ophthalmol Soc*. 1941; 39:193–213. [PubMed: 16693249]
11. Bietti G. Surgical intervention on the ciliary body; new trends for the relief of glaucoma. *J Am Med Assoc*. 1950; 142:889–897. [PubMed: 15405993]
12. Polack FM, De Roeth A Jr. Effect of Freezing on the Ciliary Body (Cyclocryotherapy). *Invest Ophthalmol*. 1964; 3:164–170. [PubMed: 14166273]
13. Semchyshyn TM, Tsai JC, Joos KM. Supplemental transscleral diode laser cyclophotocoagulation after aqueous shunt placement in refractory glaucoma. *Ophthalmology*. 2002; 109:1078–1084. [PubMed: 12045047]
14. Nouredin BN, Zein W, Haddad C, et al. Diode laser transcleral cyclophotocoagulation for refractory glaucoma: a 1 year follow-up of patients treated using an aggressive protocol. *Eye (Lond)*. 2006; 20:329–335. [PubMed: 15877101]
15. Ansari E, Gandhewar J. Long-term efficacy and visual acuity following transscleral diode laser photocoagulation in cases of refractory and non-refractory glaucoma. *Eye (Lond)*. 2007; 21:936–940. [PubMed: 16628239]

16. Frezzotti P, Mittica V, Martone G, et al. Longterm follow-up of diode laser transscleral cyclophotocoagulation in the treatment of refractory glaucoma. *Acta Ophthalmol.* 2010; 88:150–155. [PubMed: 19432863]
17. Schlote T, Grub M, Kynigopoulos M. Long-term results after transscleral diode laser cyclophotocoagulation in refractory posttraumatic glaucoma and glaucoma in aphakia. *Graefes Arch Clin Exp Ophthalmol.* 2008; 246:405–410. [PubMed: 17965873]
18. Egbert PR, Fiadoyor S, Budenz DL, et al. Diode laser transscleral cyclophotocoagulation as a primary surgical treatment for primary open-angle glaucoma. *Arch Ophthalmol.* 2001; 119:345–350. [PubMed: 11231767]
19. Grueb M, Rohrbach JM, Bartz-Schmidt KU, et al. Transscleral diode laser cyclophotocoagulation as primary and secondary surgical treatment in primary open-angle and pseudoexfoliative glaucoma. Long-term clinical outcomes. *Graefes Arch Clin Exp Ophthalmol.* 2006; 244:1293–1299. [PubMed: 16550406]
20. Kramp K, Vick HP, Guthoff R. Transscleral diode laser contact cyclophotocoagulation in the treatment of different glaucomas, also as primary surgery. *Graefes Arch Clin Exp Ophthalmol.* 2002; 240:698–703. [PubMed: 12271364]
21. Bloom PA, Tsai JC, Sharma K, et al. “Cyclodiode“. Trans-scleral diode laser cyclophotocoagulation in the treatment of advanced refractory glaucoma. *Ophthalmology.* 1997; 104:1508–1519. discussion 1519-1520. [PubMed: 9307649]
22. Hauber FA, Scherer WJ. Influence of total energy delivery on success rate after contact diode laser transscleral cyclophotocoagulation: a retrospective case review and meta-analysis. *J Glaucoma.* 2002; 11:329–333. [PubMed: 12169970]
23. Murphy CC, Burnett CA, Spry PG, et al. A two centre study of the dose-response relation for transscleral diode laser cyclophotocoagulation in refractory glaucoma. *Br J Ophthalmol.* 2003; 87:1252–1257. [PubMed: 14507761]
24. Pokroy R, Greenwald Y, Pollack A, et al. Visual loss after transscleral diode laser cyclophotocoagulation for primary open-angle and neovascular glaucoma. *Ophthalmic Surg Lasers Imaging.* 2008; 39:22–29. [PubMed: 18254347]
25. Pucci V, Tappainer F, Borin S, et al. Long-term follow-up after transscleral diode laser photocoagulation in refractory glaucoma. *Ophthalmologica.* 2003; 217:279–283. [PubMed: 12792134]
26. Wilensky JT, Kammer J. Long-term visual outcome of transscleral laser cyclotherapy in eyes with ambulatory vision. *Ophthalmology.* 2004; 111:1389–1392. [PubMed: 15234142]
27. Rotchford AP, Jayasawal R, Madhusudhan S, et al. Transscleral diode laser cycloablation in patients with good vision. *Br J Ophthalmol.* 2010; 94:1180–1183. [PubMed: 20576775]

Figure 1.
Mean Intraocular Pressure (IOP) over the follow-up duration

Figure 2.
Mean Number of Glaucoma Medications over the follow-up duration

Figure 3. Scatterplot of best corrected visual acuity (BCVA) at 3 months compared to preoperative examination. Snellen visual acuity has been converted to logMar scale. Diagonal line represents no change in visual acuity. 2.0 = count fingers, 2.3 = hand motion, 2.6 = light perception, 2.9 = no light perception

Table 1

Demographics

No. of patients/eyes treated at Robert Cizik Eye Clinic	19/20
No. of patients/eyes treated at Dean McGee Eye Institute	12/12
Left/Right	15/17
Race (No.)	
Black	10
Hispanic	6
White	12
Asian	1
Unknown	2
Age (y) (mean (SD) [median, range])	66.3 (16.3) [68.9, 29-92]
Female/Male	15/16
Follow-up (mo) (mean (SD) [median, range])	17.1 (16.3) [11.7, 2-68]
Clinical Diagnosis (no. eyes (percent))	
Primary open angle glaucoma	17 (54.8%)
Secondary glaucoma	8 (25.8%)
Neovascular glaucoma	4 (12.9%)
Congenital glaucoma	1 (3.2%)
Unknown	1 (3.2%)

No. = number; y = years; mo = months; SD = standard deviation

Table 2

TSCPC Treatment Parameters

Parameter	Range	Mean (SD)
No. of applications (“shots”) per eye	2 – 37	14.9 (8.6)
Total degrees per eye	90 - 360	224.5 (84.1)
Median energy (W)	1.20 - 3.00	1.96 (0.43)
Duration (s)	2 - 4	3.4 (0.9)
TSCPC sessions in initial treatment per eye	1 - 3	1.25 (0.51)

TSCPC = transscleral cyclophotocoagulation; SD = standard deviation; No. = number; W = watts

Table 3

Complications

Hypotony (IOP $\leq$ 5mmHg over 2 consecutive f/u visits)	4 (12.5%)
Loss of light perception	5 (15.6%)
Loss of 2 or more BCVA lines from pre-TSCPC over 2 consecutive f/u visits	18 (56.3%)
Hyphema	2 (6.3%)
Failed PKP	1 (3.1%)

IOP = intraocular pressure; f/u = follow-up; BCVA = best corrected visual acuity; TSCPC = transscleral cyclophotocoagulation; PKP = penetrating keratoplasty

Table 4

Comparison of TSCPC Studies

Study	Design	Diagnosis	Mean Follow-up	IOP Reduction (mmHg)	Visual Acuity (VA) Results	Complications
Semchishyn et al. (2002) ^{7*}	Retrospective case series, 21 eyes	Refractory glaucoma after TS, max med tx	26.9 mo	35.7 (14.7) (pre-TSCPC) to 13.6 (7.1) (final)	52% lost ≥ 1 line; 48% no change or improved	Hypotony: 14% Phthisis: 5% Band K: 5% Loss of LP: 19%
Frezza et al. (2009) ¹⁰	Prospective, cohort study, 124 eyes	Refractory glaucoma	17 mo (36 eyes followed to ≥ 3 y)	29.9 (8.9) (pre-TSCPC) to 20.8 (8.0) (final)	22.5% lost (12.9% lost ≥ 2 lines); 73.5% no change; 4.0% improved	AC reaction (3+ cells): 2.4% Hyphema: 1.6% Loss of LP: 2%
Egbert et al. (2001) ¹³	Prospective, randomized to 2 TSCPC protocols, paired analysis with patient's other eye, 92 eyes	Primary surgery for POAG	13.2 (6.0) mo	48% of eyes ≤ 22 mmHg at final visit; $\geq 20\%$ reduction in 54% of eyes starting > 22 mmHg and 15% of eyes starting ≤ 22 mmHg	23% of eyes with TSCPC lost some VA; 23% of eyes with only med tx lost some VA; 70% with TSCPC had no change; 6% VA improved	Atonic pupil: 28%
Pucci et al. (2003) ²⁰	Retrospective, case series, 120 eyes	Refractory glaucoma	26 (8) mo	30.4 (3.1) (pre-TSCPC) to 20.3 (1.8) (final)	24.2% of eyes lost VA; 55% were stable ± 1 line; 20.8% gained	N/R
Murphy et al. (2003) ²¹	Retrospective case series, 2 sites, 263 eyes	Refractory glaucoma	17 mo	40.7 (13.7) (pre-TSCPC) to 17.7 (10.9) (final)	19.8% lost ≥ 2 lines; 74.6% of eyes had no change; 5.3% improved	Hypotony: 9.5% Phthisis: 5.3% Uveitis: 1.9% Hyphema: 0.4%
Pokroy et al. (2008) ²²	Retrospective case series, 25 eyes	POAG or NVG	≥ 6 mo	POAG: 29.2% NVG: 36.6%	POAG: 36% lost ≥ 2 lines; NVG: 50% lost ≥ 2 lines	Hypotony: 4% Decompensation of PKP: 4%
Wilensky et al. (2004) ²³	Retrospective case series, 21 eyes (14 diode TSCPC, 7 Nd:YAG)	Ambulatory vision ($\geq 20/80$)	40.7 mo	N/R	81% of eyes lost ≤ 1 line; 5% improved by 3 lines; 14% lost ≥ 3 lines	N/R
Bloom et al. (1997) ²⁴	Retrospective cases series, 210 eyes	Refractory glaucoma	10 mo	34.1 (1.4) (pre-TSCPC) to 20.1 (1.3) (final)	28% lost VA; 62% unchanged; 10% gained	Hypotony: 0.95% Decompensation of PKP: 9.52% Phthisis: 0.48% Mac pucker: 0.48% Hyphema: 0.48% Loss of LP: 3%

Study	Design	Diagnosis	Mean Follow-up	IOP Reduction (mmHg)	Visual Acuity (VA) Results	Complications
Hauber et al. (2002)25	Retrospective case series, 47 eyes	Refractory glaucoma	18 of 47 eyes to 1 y	29.4 (10.6) (pre-TSCPC) to 15.7 (12.0) (1 mo), 16.2 (1.3) (1 y)	17% lost 1 line; 13% lost ≥2 lines; 68% no change; 2% gained 3 lines	Hyphema: 4%
Present study*	Retrospective case series, 32 eyes	Refractory glaucoma after TS, max med tx	17.06 (16.3) mo	28.6 (7.5) (pre-TSCPC) to 16.8 (7.5) (3 mo), 14.7 (7.9) (1 y)	56.25% lost ≥2 lines; 31.25% had no change; 6.25% gained VA	Hypotony: 12.5% Loss of LP: 15.6% Hyphema: 6.3% Failed PKP: 3.1%

TSCPC = transscleral cyclophotocoagulation; IOP = intraocular pressure; TS = tube shunt; tx = therapy; band K = band keratopathy; LP = light perception; AC = anterior chamber; N/R = not reported; PKP = penetrating keratoplasty; mac = macular; mo = month; y = year

* All eyes included in these two studies had tube shunts implanted, while no other studies had any known eyes with tube shunts