Medical, Laser, and Surgical Management of Inadvertent Cyclodialysis Cleft With Hypotony

Cyclodialysis cleft is the result of separation of the meridional ciliary muscle fibers from the scleral spur, thereby providing a new drainage pathway of aqueous humor into the suprachoroidal space. Cyclodialysis has been used as a surgical option for aphakic glaucoma but more often occurs inadvertently during anterior segment surgery or because of blunt ocular trauma. The new drainage channel increases uveoscleral outflow and may result in chronic ocular hypotony. Choroidal effusion, cystoid macular edema, optic nerve edema, engorgement and stasis of retinal veins, retinal folds, shallow anterior chamber, and cataract are recognized complications of chronic ocular hypotony.

Medical management of inadvertent cyclodialysis cleft is a trial of topical 1% atropine sulfate for 6 to 8 weeks. Topical or systemic corticosteroid therapy is not indicated. If medical management is ineffective, noninvasive methods of cleft closure, such as argon laser photocoagulation to the cleft, should be attempted. Should conservative therapy fail, then surgical closure of the cleft is the final option.

We describe 7 patients who had an inadvertent cyclodialysis cleft that occurred following uneventful extracapsular cataract extraction with posterior chamber intraocular lens implantation in 5 patients, blunt trauma in 1 patient, and trabeculectomy in 1 patient (Table). Their ocular hypotony was successfully treated with topical 1% atropine in 4 patients, argon laser photocoagulation in 2 patients, and surgical closure in 1 pediatric patient.

Report of Cases. Case 1. A 52-year-old man underwent uneventful extracapsular cataract extraction with posterior chamber lens insertion in his right eye in April 1990. Postoperatively, he developed a 20% hypHEMA that cleared spontaneously within several days; however, he developed ocular hypotony of 4.0 to 6.0 mm Hg in the operated eye with reports of metamorphopsia. He was referred to us in consultation when the hypotony failed to resolve after 18 months and his visual acuity decreased from 20/20 immediately postoperatively to 20/50. The patient was seen at the Pennsylvania State University Milton S. Hershey Medical Center, Hershey, for the first time in November 1991. Medications included dexamethasone eye drops and aspirin.

On ophthalmic examination, his visual acuity was 20/100 OD and 20/20 OS. Central Amsler grid distortion was noted in the right eye and intraocular pressure (IOP) was 2.0 mm Hg OD and 9.0 mm Hg OS. Anterior chambers were deep, equal, and quiet in both eyes. The corneoscleral wound had healed nicely and no inadvertent filtration bleb was observed. The posterior capsule was opacified and there were posterior synechiae to the implant at the 5-o’clock position. The implant was well centered. Gonioscopy revealed a cyclodialysis cleft at the 11 through 1-o’clock positions with a peripheral anterior synechia bridging the cleft at the center. Fundoscopic examination of the right eye revealed a hyperemic and swollen optic nerve head. Retinal veins were tortuous and slightly distended. There were prominent chorioretinal folds as well as choroidal thickening extending into the macular area in that eye. Findings from the examination of the left eye were unremarkable.

The patient’s condition was diagnosed as inadvertent cyclodialysis cleft following cataract extraction. The treatment regimen of corticosteroid eye drops was discontinued and 1% atropine, 3 times daily, was ordered in an attempt to close the cleft. Over the next 6 weeks, the IOP increased to only 5 mm Hg and the cleft remained patent.

Argon laser treatment was then applied deep within the cleft to both the ciliary muscle and scleral side (100 applications, 100-µm spot size, 0.1-second duration, and 1.0- to 1.5-W power). After no subsequent rise in IOP was observed, a second argon laser treatment was applied 1 week after the initial treatment (160 applications, 100-µm spot size, and 1.0- to 1.5-W power). Treatment with topical 1% atropine was continued.

Four days later, the patient was seen in the emergency department reporting severe headache, nausea, and pain in the right eye. Visual acuity was 20/60 OD and 20/20 OS with moderate (2+) conjunctival hyperemia in the right eye and trace corneal epithelial edema. Intraocular pressure was 46.0 mm Hg OD. Combined treatment with 0.5% timolol hemihydrate, acetazolamide, 250 mg/d orally, and glycerin was initiated. Within 2 hours, the IOP was

<table>
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<th>Patient No.</th>
<th>Cause of Cleft</th>
<th>Preoperative Pressure, mm Hg</th>
<th>Postoperative Pressure, mm Hg</th>
<th>Duration of Cleft, mo</th>
<th>Type of Cleft Repair</th>
<th>Preoperative Visual Acuity</th>
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<td>2</td>
<td>24</td>
<td>21</td>
<td>Argon laser</td>
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<td>14</td>
<td>8</td>
<td>Medical</td>
<td>20/100</td>
<td>20/25</td>
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<td>12 mo</td>
<td>Argon laser</td>
<td>20/60</td>
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reduced to 24.0 mm Hg. Atropine therapy was discontinued; combined treatment with a topical corticosteroid and 0.5% timolol hemihydrate was prescribed. The patient returned to his referring physician for continued monitoring of his condition.

Case 2. A 72-year-old woman was referred to the retina service for evaluation of chorioretinal folds of the left eye. The patient had undergone uneventful extracapsular cataract extraction with posterior chamber intraocular lens implantation. Her uncorrected visual acuity was 20/30 OS 8 weeks after the operation. Intraocular pressure was not recorded. Three months postoperatively, her visual acuity dropped to 20/50 OS. Intraocular pressure was 16.0 mm Hg OD and 0.0 to 1.0 mm Hg OS. The anterior chamber was deep with mild flare (1+) and no cells. The implant was centered. Fundus examination showed no abnormality and intravenous fluorescein angiography demonstrated patchy choroidal filling. She was treated with 80 mg/d of oral prednisone for uveitis and possible macular edema. One month later, the patient's visual acuity was unchanged. The anterior chamber was quiet but the IOP remained 0.0 mm Hg OS. She was then referred to the glaucoma service for further evaluation and treatment 4 months after her initial surgery.

On our examination, her visual acuity was 20/40 OD and 20/200 OS. Her IOP was 19.0 mm Hg OD and 1.0 mm Hg OS. On gonioscopy, no peripheral anterior synchiae, angle recession, or filtration cleft was noted. No filtration bleb was visible on slitlamp examination. Fundus examination revealed optic disc edema extending into the peripapillary retina. No choroidal or retinal folds were visible. With the impression of possible anterior segment ischemia, the left eye was treated with dexamethasone sodium phosphate (Decadron) eye-drops every 2 hours.

Several weeks later, she was reexamined. Visual acuity was 20/100 OS and her IOP was 5.0 mm Hg OS. Careful gonioscopy at this time showed a small, less than 1 clock hour, cyclodialysis cleft between the 12- and 1-o’clock positions. The anterior chamber was deep and quiet. Therapy with 1% atropine eye-drops 3 times daily was begun. Three weeks after atropine therapy was initiated, her visual acuity was 20/70 OS and IOP was 17.0 mm Hg OS. At her final visit, 6 weeks later, visual acuity was 20/25 OS and IOP was 1.4 mm Hg OS. The cyclodialysis cleft was closed. Atropine therapy was tapered over a 3-week period.

Case 3. A 9-year-old boy was hit in his left eye with a BB 1 week before he was referred to our glaucoma service for evaluation and treatment of hypotony. Immediately following trauma, he developed a 10% hyphema for which he was hospitalized. The hyphema resolved uneventfully, but his IOP remained between 2.0 and 4.0 mm Hg OS. Computed tomography of the left orbit showed no abnormality for an intraocular or intraorbital foreign body. He was treated with prednisolone phosphate eye-drops, 1 drop 4 times daily, without effect.

When we examined the patient, he reported photophobia. His visual acuity was 20/15 OD and 20/200 OS. There was a myopic shift of −9.50 diopters in his left eye and with correction, visual acuity improved to 20/50 OS. Anisocoria was present, with an irregular, dilated left pupil. No relative afferent pupillary defect was present. The anterior chamber in the left eye was slightly shallow. There was no evidence of anterior chamber inflammation. Intraocular pressure was 12.0 mm Hg OD and 3.0 mm Hg OS. An iridodialysis was present from the 8:30- through 10-o’clock positions in the left eye. Gonioscopy demonstrated a wide open angle in the right eye and a narrow angle in the left eye with a cyclodialysis cleft from the 10- through 1-o’clock positions. The right fundus was normal; however, optic disc edema and minimal macular edema were noted in the left eye. No choroidal or retinal folds were present.

Topical corticosteroid therapy was discontinued and 1% atropine eye-drops, 3 times daily, were prescribed. When his condition was evaluated in 1 month, the clinical picture was unchanged. Atropine therapy was continued. When the patient was again seen 7 weeks later, his visual acuity was 20/40 OS and his IOP was 10.0 mm Hg OS. The macular edema was decreased. The patient returned to the referring ophthalmologist with instructions to taper the atropine therapy.

Case 4. A 72-year-old man underwent uneventful extracapsular cataract extraction with phacoemulsification and posterior chamber intraocular lens implantation in his left eye by his referring ophthalmologist on January 10, 1992. Immediately following the procedure, he was found to have ocular hypotony with an IOP of 0.0 to 2.0 mm Hg OS and gradual decline in his visual acuity. Combined treatment with a topical corticosteroid every 2 hours, cyclopentolate hydrochloride, and dexamethasone sodium phosphate ointment was started. He was ultimately referred to us 5 months postoperatively, when medical management was not alleviating his hypotony and decreased visual acuity.

When he was examined in consultation, his visual acuity was 20/30 OD and 20/200 pinholing to 20/60 OS. The eyes were without inflammation and the left anterior chamber was slightly shallow. The implant was in place. Gonioscopy revealed a small 1 clock hour cyclodialysis cleft superiorly. His IOP was 22.0 mm Hg OD and 0.0 to 2.0 mm Hg OS. Fundus examination showed choroidal thickening and mild macular edema of the left eye.

Treatment with 1% atropine eye-drops, 3 times daily, were prescribed. When his condition was evaluated in 1 month, the clinical picture was unchanged. Atropine therapy was continued. When the patient was again seen 7 weeks later, his visual acuity was 20/40 OS and his IOP was 10.0 mm Hg OS. The macular edema was decreased. The patient returned to the referring ophthalmologist with instructions to taper the atropine therapy.

Case 5. A 4½-month-old girl was referred to the pediatric ophthalmology service for bilateral congenital glaucoma in June 1993. She took no medications. Ocular history included an intermittent esotropia. During evaluation of her condition, she was found to be in good general health.
She fixed and followed well with the right eye and poorly with her left eye. Her pupils were dilated medically at the time. She was orthophoric with motor testing and had full motility. Epiphora was observed in both eyes. Horizontal corneal diameters were 12.0 mm OD and 3.0 mm OS. The left cornea appeared hazy on slitlamp examination; no Haab striae were noted. Intraocular pressure measured using a handheld electronic tonometer (Tono-Pen XL; Mentor Corp, Santa Barbara, Calif) was 19.0 mm Hg OD and 27.0 mm Hg OS. Funduscopic examination revealed a cup-disc ratio of 0.7 OD and 0.8 OS with normal vessels, macula, and periphery.

The patient underwent bilateral uncomplicated trabeculotomy 2 weeks apart. Postoperatively she was receiving a combination of polymyxin B sulfate–trimethoprim sulfate and 1% prednisolone phosphate eyedrops in both cases. Fundus examination 1 week postoperatively showed shallow choroidal detachments in the right eye. On subsequent follow-up visits, the right eye was found to be soft and hypotonic to palpation. On funduscopic examination 2½ months later, hypotony maculopathy and optic disc edema were present and the eye remained soft to palpation. Treatment with 1% atropine ointment, 3 times daily, was begun for a presumed cyclodialysis cleft. The patient was scheduled for examination under anesthesia.

Examination under anesthesia then revealed a shallow anterior chamber on the right side. The IOP was 0.0 mm Hg OD and 11 mm Hg OS. Funduscopic examination of the right eye showed prominent optic disc swelling. Gonioscopy of the right eye was performed and revealed a cyclodialysis cleft extending for about 2 clock hours in the superior nasal quadrant between the 10:30- and 12-o’clock positions. A 4 × 6-mm, partial-thickness, limbus-based scleral flap was created superiorly. A scleral incision was created 2.5 mm posterior to the limbus within the scleral flap. Incision was carefully continued deep to the sclera at the center of the scleral bed; fluid was found in the suprachoroidal space, most prominently in the superior nasal quadrant. Several 10-0 nylon sutures were used to close the cyclodialysis cleft in the superior nasal quadrant. The sutures were passed through the sclera, then through the ciliary body, then back to the sclera. On postoperative day 1, IOP taken with the handheld electronic tonometer was 11.0 mm Hg OS and optic disc edema was reduced. The patient’s condition was maintained on a combination therapy of 0.5% atropine ointment, 2 times daily, polymyxin B–trimethoprim, and 1% prednisolone acetate. Atropine therapy was slowly tapered over months. Visual acuity 4 years later was 10/16 OU and IOP was normal at 8.0 mm Hg OU.

Case 6. A 55-year-old woman underwent uneventful extracapsular cataract extraction with phacoemulsification and placement of a posterior chamber intraocular lens in the left eye on March 7, 1995. On the first postoperative day, she reported a mild headache. Visual acuity was 20/200 OS pinholing to 20/80 OS. The wound was secure and intact with a deep anterior chamber and a moderate (2+) cellular reaction. Intraocular pressure was 6 mm Hg. Combined therapy was started with polymyxin B–trimethoprim and 1% prednisolone phosphate eyedrops.

On evaluation 2 days later, her visual acuity was unchanged. There was a negative Seidel sign. There was no filtration bleb noted. The cornea was mildly edematous and guttata were observed. The IOP was 4.0 mm Hg. On gonioscopy, a small cyclodialysis cleft was noted superiorly. Funduscopic examination showed no evidence of retinal detachment or hypotonic maculopathy. One percent atropine, 2 times daily, was added to her treatment regimen.

Five days later, she was doing very well. Her visual acuity was 20/200 pinholing to 20/40 OS. The eye was quiet with a secure wound. Intraocular pressure was 14.0 mm Hg OS and the cyclodialysis cleft was closed on gonioscopy. Atropine therapy was tapered over 2 weeks. One month after surgery, her visual acuity was 20/40 OS. Intraocular pressure was 13.0 mm Hg bilaterally. The wound was well healed. The posterior chamber intraocular lens was well positioned. Funduscopic examination revealed no abnormality. The patient underwent YAG-laser capsulotomy at her 3-month postoperative visit. The patient’s visual acuity was 20/20 OS following that procedure.

Case 7. A 52-year-old man was referred to the retina service at our institution in January 2002 because of decreased vision in the left eye. This patient had a history of blunt trauma to the left eye in August 1999. He developed a traumatic cataract that was successfully removed by extracapsular cataract extraction in January 2000. There were no surgical or postoperative complications. Postoperatively, visual acuity was 20/25 OS, but gradually his visual acuity decreased. He also had persistent anterior chamber reaction. He was treated with 1 drop of 2% cyclopenotolate, every other day, and 1% prednisolone acetate eyedrops, 4 times daily, by his primary ophthalmologist. He was noted to have some posterior retinal changes and was referred for retinal evaluation.

On presentation, the patient reported painless, persistent, decreased vision. Visual acuity was 20/20 OD and 20/80 pinholing to 20/50 OS. He had a trace afferent pupillary defect in the left eye. Slitlamp examination of the eyelids, conjunctiva, and cornea were unremarkable. Anterior chambers were deep and quiet. Intraocular pressure was 16.0 mm Hg OD and 6.0 mm Hg OS. Funduscopic examination revealed chorioretinal folds through the macula and optic disc swelling of the left eye.

The patient was referred that day to the glaucoma service where gonioscopy revealed a superior cyclodialysis cleft encompassing 2 to 3 clock hours in the left eye. Because the patient had been receiving cyclopenotolate for almost 1 year, the decision was made to use an argon laser to close the cleft. He was discharged from clinic receiving 1% atropine, 4 times daily, to the left eye while awaiting argon laser treatment.

No changes were noted in 3 to 4 weeks, so he underwent argon laser treatment. The patient received 95 spots of 1500-mW power,
100-μm spot size, for 0.1-second duration to the cyclodialysis cleft. Four days afterward, the patient was examined and found to have an IOP of 8 to 9 mm Hg by applanation; the cyclodialysis cleft remained open. He underwent a second argon laser treatment of 1500 mW, 50-μm spot size, 0.1-second duration, and 45 spots to the cyclodialysis cleft.

Two days following his last argon laser treatment, the patient was seen in the emergency department reporting pain and blurred vision in the left eye. His visual acuity was hand motions OS. He exhibited moderately severe (3+) conjunctival hyperemia, microcystic corneal edema, and hazy view of the posterior segment in the left eye. Intraocular pressure was 53.0 mm Hg OS. No view of angle was seen with gonioscopy. Intraocular pressure was reduced by using a combination therapy of glycerin, acetazolamide, timolol maleate–dorzolamide hydrochloride, and brimonidine tartrate. The patient’s IOP was lowered to 31.0 mm Hg over a few hours and he experienced improved visual acuity of 20/300 OS and decreased corneal edema.

The next day, the IOP was 14.0 mm Hg. The patient continued the instillation of 1% atropine, 4 times daily, in the left eye. Four days later, the patient was noted to have decreased hypotony maculopathy. At his last visit, 2 months after argon laser treatment, visual acuity was 20/50 OS, IOP was 13.0 mm Hg, optic nerve edema was resolved, and chorioretinal folds had disappeared. He was sent back to the referring physician with instructions to taper the atropine therapy.

Comment. A cyclodialysis cleft, resulting from the disruption of the meridional ciliary muscle insertion into the scleral spur, may result in ciliochoroidal detachment and ocular hypotony.34 In 1900, Fuchs5 reported separation of the ciliary body and choroid following cataract extraction noted in pathologic analysis. His observations suggested that there was a direct communication between the anterior chamber and suprachoroidal space. Based on the observation, Heine7 described a new operation for aphakic glaucoma, which he termed, “cyclodialysis procedure.” In 1932, Elschniég reported on the pathologic findings in an eye 14 years after the cyclodialysis procedure was performed. He noted a direct communication between the anterior chamber and suprachoroidal space accompanied by a choroidal detachment.

Whether performed as a treatment for aphakic glaucoma, inadvertently produced following surgery on the anterior segment of the eye, or resulting from blunt trauma to the eye, persistent hypotony due to cyclodialysis is an uncommon but significant complication. An analysis of complications in a large series of cyclodialysis procedures revealed that hypotony was present in 24 (9%) of 291 cases.3 The magnitude of the hypotony is not proportional to the size of the cyclodialysis cleft.9 The mechanism of this hypotony is presumed to be internal filtration, decreased aqueous production, or both.10 Chandler and Maumenee11 believe that there is a generalized detachment of the ciliary body in post-cyclodialysis hypotony.

Hypotony may result in choroidal effusion, retinal thickening and folding, optic disc edema, macular edema, cataract, and shallow anterior chamber. Reduced visual acuity may be due to cataract; however, the major cause of visual loss is because of transudation of protein-rich fluid from the posterior blood vessels into the subretinal space in the posterior pole. Loss of vision caused by macular edema may become permanent if the condition is undetected and untreated.

It is speculative as to why and how patients develop cyclodialysis cleft. Unrecognized trauma to the angle and adjacent structures at the time of lens implantation may be the cause of inadvertent cyclodialysis. The most common cause of hypotony after cataract extraction is a leaking wound. Ocular hypotony itself is the most frequent cause of ciliochoroidal detachment following cataract extraction. Therefore, in all cases, prior to considering cyclodialysis cleft, a wound leak should be ruled out. Other causes for persistent hypotony such as retinal detachment, chronic inflammation, and anterior segment ischemia also should be excluded. Nevertheless, inadvertent cyclodialysis cleft resulting from extracapsular cataract extraction is a significant cause of persistent ocular hypotony following such procedures.

A careful gonioscopic examination of the anterior chamber angle is the key element in the diagnosis of iatrogenic or traumatic cyclodialysis cleft. Sometimes it is difficult to see the cleft itself, as it is often small and obscured by a narrowed anterior chamber angle. A shallow chamber generally is caused by diffuse choroidal effusion that leads to forward displacement of the lens iris diaphragm. It has been suggested that anterior chamber deepening by the injection of a viscoelastic agent into the anterior chamber may facilitate visualization of the cleft.12 Sometimes immersion B-scan ultrasonography may be helpful in the diagnosis of the small cyclodialysis cleft.13 Diagnosis of cyclodialysis cleft using conventional ultrasonography with a microconvex ultrasound probe of 9-MHz frequency has been described.14 More recently, high-resolution ultrasound biomicroscopy has been used to provide detailed information about the extent and location of a cyclodialysis cleft.15,16

Once the diagnosis of cyclodialysis is made, medical treatment consisting of topical 1% atropine, 2 to 3 times daily, should be attempted for up to 6 weeks. Miotics and corticosteroids should be avoided. A cyclodialysis cleft that results in ocular hypotony unaccompanied by significant ocular structural or functional abnormalities probably does not require treatment.

Since 1990, we have diagnosed cyclodialysis cleft in 7 patients: 1 eye (14.3%) following blunt ocular trauma, 3 eyes (71.4%) following extracapsular cataract extraction, and 1 eye (14.3%) after trabeculotomy. In 2 patients, ocular hypotony had been present 1 to 2 weeks prior to the diagnosis, in 3 patients within 3 to 5 months, and in 2 patients more than 1 year. Four (57.1%) of 7 eyes responded to medical therapy alone and the hypotony was reversed within 1 week after initiation of 1% atropine therapy. Two (28.6%) of 7 eyes had...
successful closure of the cleft with argon laser photocoagulation directed to the cyclodialysis cleft. One (14.3%) of 7 eyes (1 pediatric patient) required surgical closure of the cleft, as she was unable to cooperate at the slitlamp for attempts at argon laser closure of the cleft.

Argon laser therapy for cyclodialysis induced ocular hypotony was used for the first time by Joon-deph. The hypotony was reversed following 2 sessions of argon laser treatment. He suggested a power of 400 to 800 mW, 200-µm spot size, and 0.1- to 0.2-second duration. Multiple other investigators have described success with argon laser treatment. The mechanism by which laser treatment causes reversal of hypotony is unknown. Harbin assumes that swelling of the choroid following laser treatment closes the cleft and blocks aqueous flow into the suprachoroidal space, or, perhaps, the iritis that is caused by the treatment plays a role in altering the aqueous humor composition, thereby obstructing drainage.

Other methods of laser photocoagulation of cyclodialysis clefts have been successful. Alward et al used a modified endophotocoagulator probe as an external source to treat the eye of a 7-year-old boy with a traumatic hypotony cyclodialysis cleft after he failed to respond to medical treatment. In this technique, the endophotocoagulator is adapted for use with the Swan-Jacobs goniolens to more directly focus treatment to a cleft that is particularly difficult to visualize. Carolina et al accomplished diagnosis and treatment of cyclodialysis cleft by directly imaging and treating the cleft with an endoscopic laser, with the need for a goniolens. Brooks et al describe closure of a persistent cyclodialysis cleft in 3 patients using transcleral YAG laser photocoagulation with 6-J power, 20 applications, 2 to 3 mm behind the limbus in the area of the suspected cleft. Krohn reported using external transconjunctival cryotherapy at the presumed location of a cyclodialysis cleft to obtain successful cleft closure in a hypotonic eye following trabeculectomy ab externo.

If laser photocoagulation or noninvasive methods of cleft closure fail, a number of surgical interventions such as direct cyclohexy, ciliochoroidal diathermy, anterior scleral buckling, or vitreoretinal procedures may be performed. In direct cyclohexy, the detached ciliary body is sutured to the sclera near the opening of the cleft under direct visualization. A similar result can be obtained by suturing the iris base into the circumferential limbal stab incisions under a partial-thickness scleral flap. In 1971, Maumenee and Stark proposed the application of penetrating diathermy in the area surrounding the cleft following drainage of suprachoroidal fluid. They treated 6 patients with persistent hypotony due to cyclodialysis cleft—3 patients following intracapsular cataract extraction, 1 patient after surgical peripheral iridectomy, and 2 patients following inteventional cyclodiagnosis procedure. In some complicated cases, anterior scleral bucking with cryotherapy or diathermy has been successful.

Helbig and Foerster described management of hypotensive cyclodiagnosis with pars plana vitrectomy, gas tamponade, and cryotherapy. Most recently, Hoerauf et al described successful treatment of a traumatic cyclodialysis cleft of a severe and chronic nature with vitrectomy, cryotherapy, and gas endotamponade with the principle of direct mechanical apposition of the detached ciliary muscle to the scleral spur by the gas bubble and scar induction by cryotherapy. It is our belief that these kind of invasive procedures should be reserved for those cases that do not respond to medical treatment, laser treatment, or direct cyclohexy.

When the cyclodiagnosis cleft is closed, whether with 1% atropine therapy, argon laser treatment, or invasive surgery, there may be a period of acute ocular hypertension in the 2 weeks following the closure. This hypertension is caused by collapse of the aqueous drainage channels during the prolonged hypotony period and inability to reestablish drainage once the IOP is restored. The onset of this hypertensive episode tends to be delayed for several days following laser treatment; however, early pressure rise is common after surgical correction.

Medical treatment with β-blockers, carbonic anhydrase inhibitors, and hyperosmotic agents is indicated during the hypertensive stage. Miotics are contraindicated, as they could lead to opening of the cleft.

Remarkable visual recovery often occurs when the hypotony is reversed, even after several years’ duration. Delgado et al described a patient who had a 7-year history of hypotony prior to argon laser photocoagulation and gained improvement in visual acuity from 20/200 to 20/30 following cleft closure. Similarly, retinal choroidal changes and optic disc edema usually respond to normalization of IOP.

The clinician must suspect the presence of cyclodiagnosis cleft in any patient exhibiting persistent ocular hypotony following surgical or nonsurgical ocular trauma. Treatment options are variable and with proper recognition and therapy, most of these patients may have restoration of ocular function and vision.

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New Insights Into Progressive Visual Loss in Adult Retinopathy of Prematurity

An abnormal vitreous and aberrant vitreoretinal traction are significant factors in the development of cica-tricial changes and poor outcomes in neonates with retinopathy of prematurity. Late complications of retinopathy of prematurity (ie, retinal tears or rhegmatogenous retinal detachment) have also been described and these, too, can be attributed to an abnormal vitreoretinal relationship.  

Tasman and Brown observed that progressive visual loss may be noted in adult retinopathy of prematurity without a definitive clinical cause. Tangential vitreoretinal traction was speculated to result in cystoid macular edema or compromised photoreceptors with resulting pigmentary disturbances and visual deterioration. Herein we report our observations in a similar case of adult retinopathy of prematurity and with the aid of optical coherence tomography note that subclinical tractional retinal detachment may be the cause of visual loss in at least some of these cases.

Report of a Case. A 29-year-old woman had been delivered at gestational age 28 weeks with a birth weight of 1304 g. She had a history of retinopathy of prematurity. She was referred to our center for examination of a 7-month history of progressive visual loss in her left eye. The patient had previously undergone multiple procedures in the left eye for treatment of glaucoma and subsequent cataract extraction with intraocular lens placement at 16 years of age. Her baseline visual acuity was 20/60 OS before the onset of her recent symptoms. She had not previously undergone cryotherapy or laser treatment in either eye. Her right eye had been enucleated 2 years previously for end-stage glaucoma and complications following rhegmatogenous retinal detachment related to previous intraocular surgeries.

On her initial visit, her visual acuity measured 20/400 OS. Biomicroscopic examination results of the left eye revealed a quiet anterior segment with a posterior chamber intraocular lens and a central opening in the posterior capsule. Examination results of the right orbit revealed an ocular prosthesis. Funduscopy examination results of the left eye showed pulled retinal vessels at the optic nerve (ie, a dragged disc) with temporal dragging of the fovea. A small organized area of central vitreous attached to the optic disc was present. Retinal pigment epithelial change was noted in the macula, but no clinically appreciable cystoid macular edema, epiretinal membrane formation, macular hole, retinal detachment, or other macular pathologic features were noted. The peripheral retina was attached and had an avascular appearance consistent with a history of retinopathy of prematurity. The patient was asked to undergo fluorescein sodium angiography and optical coherence tomography testing.

At a follow-up examination 1 week later, the patient had a further decrease in visual acuity to 20/800 OS. Slitlamp and funduscopy examination results of the left eye remained unchanged. Fluorescein angiography results of the left eye revealed retinal pigment epithelial clumping without intraretinal edema or other abnormalities of the posterior segment other than dragging.