# Treatment of Chronic Ocular Hypotony

## Kronik Oküler Hipotenide Cerrahi Tedavi Yaklaşımları

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**ABSTRACT**

Diagnosis, etiopathogenesis and especially surgical treatment options of chronic ocular hypotony are discussed in this review. Main mechanisms of chronic hypotony are increased aqueous outflow and reduced aqueous production. While increased filtration can be effectively treated by surgery in most cases, there is no effective and longstanding therapy for ciliary body dysfunction. Dissection and removal of cyclic membranes and relief of traction on ciliary body provide a moderate increase in intraocular pressure. Occlusion of aqueous outflow via implanting a capsular tension ring to the iridocorneal angle offers effective increase in intraocular pressure in patients having some healthy ciliary epithelium. However, in eyes with no or minimal aqueous production, blockage of the iridocorneal angle would not work. Ciliary body transplantation or ventriculo-vitreal (cerebrovitreal) shunt applications may be future treatment options for protecting these eyes from development of phytisis bulbi.

**Key Words:** Hypotony, surgical therapy, etiopathogenesis of Hypotony.

**ÖZ**

Bu derlemede kronik oküler hipotoninin tanı, etyopatogenezi ve cerrahi başta olmak üzere tedavi seçenekleri tartışılmıştır. Hipotoninin temel nedeni; artmış aköz hümör dışa akımı ve azalmış aköz hümör üretimidir. Aşırı filtrasyon çoğu vakada cerrahi olarak iyi tedavi edilebilebirken, siliyer disfonksiyonunun etkin ve kalıcı bir tadavisi yoktur. Siklitik membranların temizliği ve traksiyonların rahatlatılması göz içi basıncında orta düzeyde bir artış sağlamaktadır. Bir miktar aköz salınımının olduğu durumlarda dışa akım yolunun kapsül germe halkası ile blokajı hipotoniyi tedavi etmede etkin bulunmuştur. Fakat, aköz hümör salınımının olmadığı gözlerde bu blokaj başarısız olacaktır. Siliyer doku transplantasyonu veya ventrikülo-vitreal (serebrovitreal) şant uygulamaları bu gözlerde fitizis bulbi gelişimini önlemede umut vadeden tedavi yöntemleri olabilir.

**Anahtar Sözcükler:** Hipotoni, cerrahi tedavi, hipotoni etyopatogenezi.

## INTRODUCTION

Chronic ocular hypotony(COH) is the main cause of eye loss in situations where anatomical integrity is preserved after trauma or intraocular surgery. COHis a devastating process for both ophthalmologists and patients, resulting in blurred visioni painful eye and phthisis bulbi.Ocular Hypotony is defined as the intraocular pressure(IOP) below 6 mmHg, but severe visual loss occurs below 4 mmHg.[1-4] Hypotony develops when aqueous humor production falls to 10% of normal.[5] The prolongation of the hypotony resultsin a decreased production of aqueous humor by impairing the blood aqueous barrier, which causes a vicious cycle of hypotony.[3] Clinically, structural and functional defects such as cataract, corneal edema, maculopathy, papillary edema and visual loss develop and hypotony can result in phytisis bulbi in untreated eyes.[4]

Suppression of inflammation by corticosteroids is the basis of medical treatment.Intravitreal viscoelastic agents, perfluorocarbon, gas and silicone oil injections are also well known surgical treatment options.[2-5] In selected cases, re-

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moving epiciliary and anterior proliferative vitreoretinopathy (PVR) membranes may be effective. The level of atrophy of the ciliary epithelium directly affects the outcome of all treatment options.The development of new methods to ensure full recovery is required.In this review, etiopathogenesis and surgical treatment of COH approaches will be discussed.

## CAUSES OF HYPOTONY

### Increased Filtration

Traumatic lacerations and surgical wound leakage cause hypotony externally. Cyclodialysis, ciliocoroidal detachment, retinal detachment, and extensive retinectomies cause hypotony internally with internal leakage from the suprachoroidal space.[2,4]

### Decreased Production

The hypotony that develops as a consequence of ciliary body failure is the most difficult type to cope with. Inflammation is a primary component of the pathogenesis of COH. Inflammation reduces aqueous production via prostaglandins and at the same time causes an increase in uveoscleral outflow.[6] Anterior PVR and cyclitic membranes cause chronic traction, which damages the choroidal blood flow of the ciliary body and reduces aqueous production.[5] Having more than 2 clock-face of ciliary dialysis is sufficient for hypotony.[7] Ciliochoroidal detachment creates a hypotonic cycle by increasing uveoscleral outflow and reducing aqueous production.

Hypotony may develop following vitreoretinal surgery;excessive laser and cryo applications under the repressive effect of silicone oil,[8]ciliary atrophy, proliferative membranes, deep scleral indentation, extensive retinotomy and retinectomy,[9-11] lensectomy, toxic effect of silicone oil, ciliochoroidal and retinal detachment.[4]

### Medical Treatment

Corticosteroids constitute the basis of medical treatment because of its effect on inflammation control.The increase in IOP is thought to be due to suppression of inflammation and increased outflow resistance.[12,13] It has been shown that oral, periocular, topical and intravitreal steroids are effective in the treatment of COH.[14,15] It is believed that the short-term control provided by steroid therapy has broken the vicious circle of hypotony. A current treatment alternative is topical 2% ibopamine administration, a nonselective dopaminergic agent. It has been reported that this treatment provides sustained IOP elevation in cases of resistant hypotonia that has undergone vitreoretinal surgery several times due to retinal detachment, but it is inadequate in functional success and difficult to use due to local side effects of the drug.[16,17]

### Surgical Treatment

Surgical method should focus on the underlying cause of COH.If the underlying cause is treated properly, significant structural and functional improvement has been observed, even in eyes with hypotony for a long time.[18]

Intraocular injections

Clinical studies show that sodium hyaluronate provides adequate vitreous support.[19] Injection of viscoelastic material into the anterior chamber or vitreous cavity provides effective but transient treatment for COH.[20-22] It is thought to be more effective on postoperative early hypotony and to break the vicious cycle of hypotony by eliminating the possible shallow detachment in the ciliary body.[20,23] If ciliary insufficiency is present, the chances of success are low.Complications such as prolonged inflammation and endophthalmitis due to repeated injections limit their use.The use of viscoelastic material at high concentration reduces the injection frequency.[20, 21, 24]

Repeated fluid-gasexchange in the hypotony after PVR surgery also prevents phytisis bulbi temporarily, but silicone oil may be needed for a longer duration of filling effect in some cases.[26] Silicone oil injection is a mandatory therapeutic option in the treatment of COH[2 ,4, 27-29] when the ciliary epithelium and it's extensions are ischemic and atrophic. Although permanent vitreous support provided by silicone oil may be sufficient to maintain IOP, the feeding of school tissues due to aqueous humor deficiency and oxygenation is impaired. In this situation, damage to the ocular tissues may progress and phytisis bulbi may develop.

In cases with the ciliary body disfunction that is due to epiciliary membranes; If these epiciliary , cyclitic membranes are cleaned and ciliochoroidal detachment is absent, ocular tonus can usually be restored with long-term silicone oil tamponade. Additional silicone oil injections may be required due to increased ocular volume induced by silicone oil to maintain IOP and achieve better visual functions.[30]

Removing anterior PVR membranes and epiciliary, cyclitic membranes

Ciliochoroidal detachment and ciliary body dysfunction are the leading causes of hypotony after vitreoretinal surgery. Anterior PVR, also referred to as proliferative vitreociliopathy, causes tractional ciliochoroidal detachment. This reduces aqueous production by disrupting the choroidal blood flow of ciliary body.[7] Cyclitic membranes cause hypotonia by a similar mechanism.Vitreoretinal surgery with transpupillary approach, in which cyclitic membranes have been removed and ciliary body traction have been relieved, provides an effective and sustainable IOP increase in selected cases.[27,31] Ciliary body can be assessed preoperatively by

ultrasonic biomicroscopy or by direct visualization during surgery.Regardless of etiology, removal of membranes and relieving ciliary tractions were found to be sufficient for hypotony control in cases where ciliary extensions were normal.[27,31,32] However, it is emphasized that this surgery is a rescue treatment and the functional success is achieved in a limited number of patients.[11,31] In the presence of severe ciliary atrophy, this surgical approach is ineffective.In this case, permanent silicone oil tamponade is inevitable to obtain enough IOP to prevent the development of phytisis bulbi.[27] Endoscopic vitrectomy is thought to be superior to the conventional method because it reveals tractions that can be overlooked due to scleral indentation.Especially young age and low numbers of previous vitreoretinal surgeries have been associated with positive outcome.

Reparation of ciliary body dialysis

Cyclodialysis, which can be seen as a complication of blunt trauma or intraocular surgery, is defined as the separation of the ciliary muscle from the scleral spur.Due to the direct aqueous passage between the anterior chamber and the suprachoroidal space, uveoscleral outflow is increased.Gonioscopy in anteriorly placed dialysis and ultrasound biomicroscopy in posterior dialysis are more useful to identify the dialysis.Anterior chamber narrowing is rare despite excessive filtration, if anterior chamber is shallow, the diagnosis can be made with gonioscopy after the anterior chamber is formed with viscoelastic material. The cyclodialysis cleft may occasionally close up as it rarely causes a sudden increase in IOP.Cycloplegia made with topical atropine can help the ciliary body to shift to the sclera by reducing the tonus of the ciliary muscle.[33] Arguments such as argon laser photocoagulation, diode and YAG laser cyclophotocoagulation have been found effective in the case of medical treatment failures.[33-35]

Methods used in the surgical treatment of closure-resistant cyclodialysis cleft are; direct cyclopexy, cryotherapy, anterior scleral buckling, pars plana vitrectomy with gas endotamponade or with endoscopic suturation, capsular tension ring suturation at sulcus and 3 piece intraocular lens application.[36-40]

Placement of capsular tension ring to the iridocorneal angle

The primary pathway responsible for the aqueous humoral outflow is the trabecular meshwork. Blocking the iridocorneal angle will increase IOP by reducing aqueous outflow from the trabecular meshwork.Argon laser-induced sclerosis in the trabecular meshwork has treated chronic hypotony by increasing aqueous outflow resistance.[41]

Gürelik et al. placed a capsular tension ring (CTR) to the iridocorneal angle to blockthe aqueous ourflowflow in COH patients andachieved a sustained increase in IOP and a significant improvement in visual function (Gürelik G, Dişli G: A New Surgical Technique to treat hypotony. AAO absract, 2014).The surgical technique is simple.A CTR (11-13 mm or 12-14 mm) is placed to the iridocorneal angleviaa small corneal incision (Figs. 1a, 1b, 1e, 1d). It is emphasized that

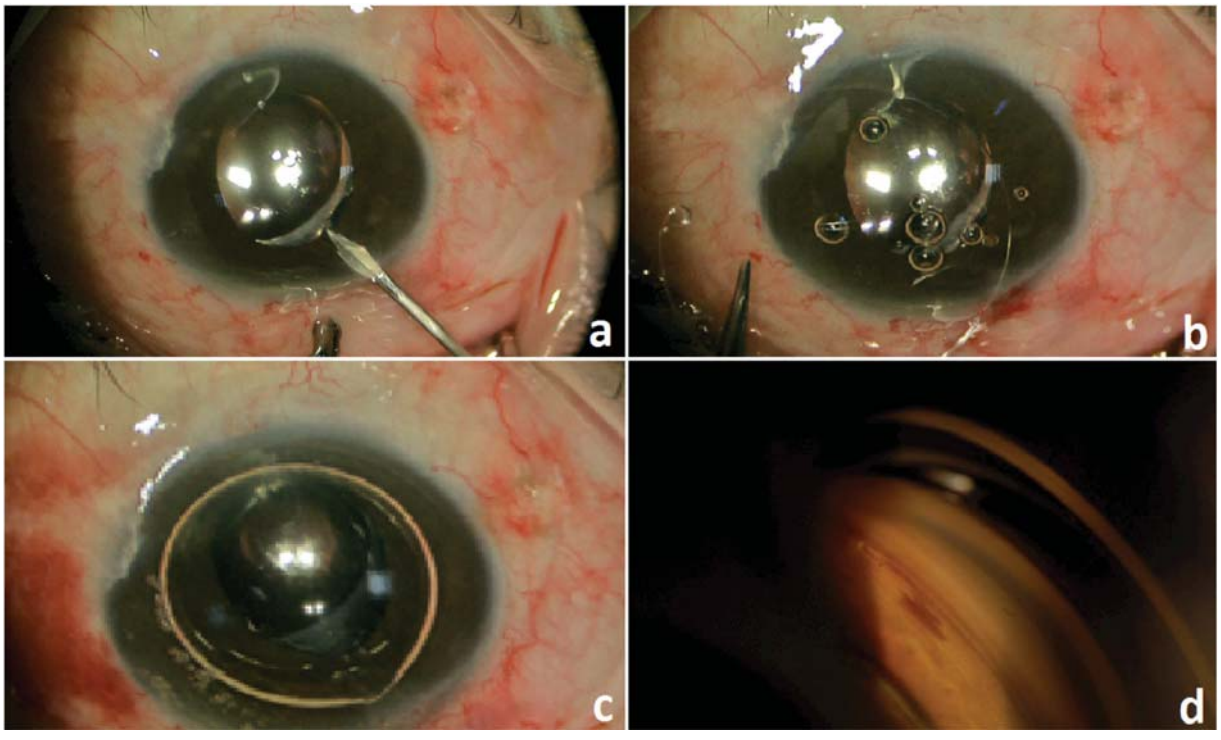


**Figure 1.** *Insertion of capsular tension ring in the iridocorneal angle*. **1a:** *limbal incision,* **1b:** *insertion of the capsule tension ring,* **1c:** *intracameral air injection*, **1d:** *gonioscopy image of the capsular tension ring.*

the success rate of this technique depends on the amount of remaining intact secreting ciliary epithelium.

**New and Promising Surgical Therapeutic Options in The Future**

Ciliary Body Transplantation

As described in detail above, complete loss of function of ciliary epitheliumappears after traumatic, surgical or inflammation-induced ciliary tissue damage. This dysfunction is irreversibe and thedevelopment of the phytisis bulbi due to ocular hypotonia is inevitable if not treated efficiently. Another option in this regard is to provide a functional healthy tissue. Since it was difficult and traumatic to reach the posterior of the iris, ciliary tissue allografts are placed in the anterior surface of the iris or iridocorneal angle in experimental models (Fig. 2a, 2b).The anterior chamber is immunologically protected and nutritional support makes it a suitable stage for tissue transplantation.It has been shown that the ciliary tissue graft placed in the intact anterior chamber of the immunosuppressed host is well perfused andbe able to produce aqueous humor and it's epithelial cell morphology is protected (Figs. 3a, 3b).[42-44] Although the results of ciliary tissue transplantation can not be predicted in the eyes with hypotonia , it seems to be a promising method to treat COH. However, the need for immunosuppression is a serious limiting factor and there is no clinical studies yet.

Cerebro-Vitreal shunt

There are no medical and surgical treatments to stimulate aqueous release from the damaged ciliary body in studies carried out so far.

As a second treatment option for these none aqueous secreting eyes a new surgical model was planned. In order to fill the eye with an aqueous humor-like fluid, cerebrospinal fluid was transferred into the vitreous cavity via shunt tube in an experimental study.

For this purpose, Gurelik et al. have defined a new experimental hypotony model that is effective and can be created in a short time. Severe hypotony was provided in rabbit eyes undergoing 360 degree argon laser endoscopic cyclocoagulation after lensectomy and vitrectomy (Figures 4a, 4b, 4c). After that procedure, some hypotonic eyes were treated by filling the eyes with balanced salt solution from the outside using the vitreal shunt system (Figures 5a-d).This study has shown that vitreal shunting is feasible (Gurelik G et al, unpublished information). An additional experimental study showed cerebro-vitreal shunting prevented eyes from phythisis bulbi in experimentally induced severe hypotony eyes. (Gurelik G et al, unpublished information)

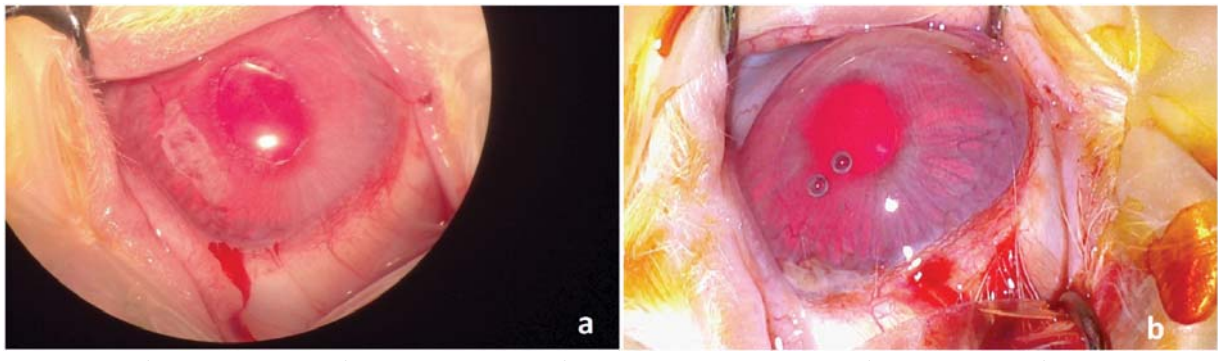


**Figure 2.** *Ciliary tissue transplantation* **2a:** *transplantation on iris,* **2b:** *transplantation in angle region*

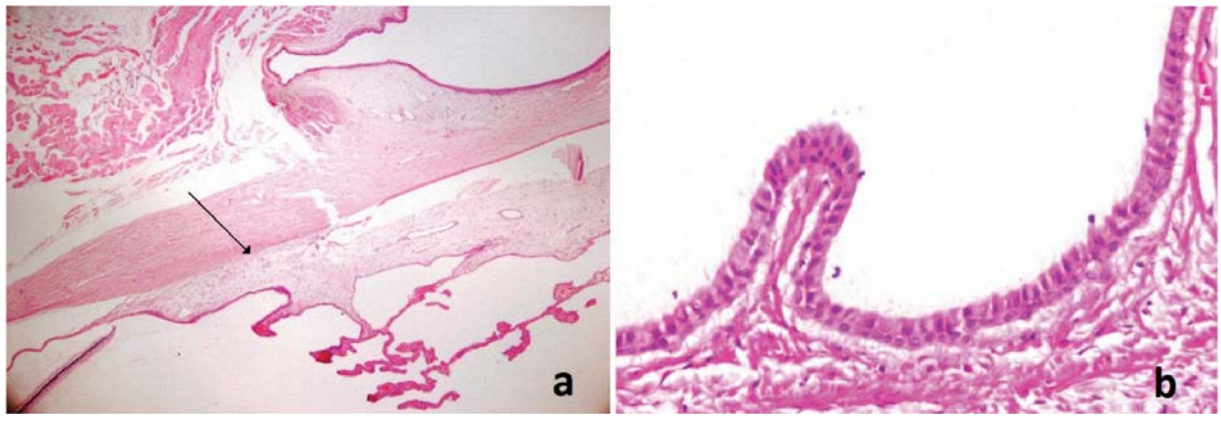


**Figure 3.** *Hematoxylin-eosin-stained histopathology preparations (X400)* **3a:** *vascular connection between graft tissue and iris* **3b:** *double-row normal epithelial cells in ciliary body.*

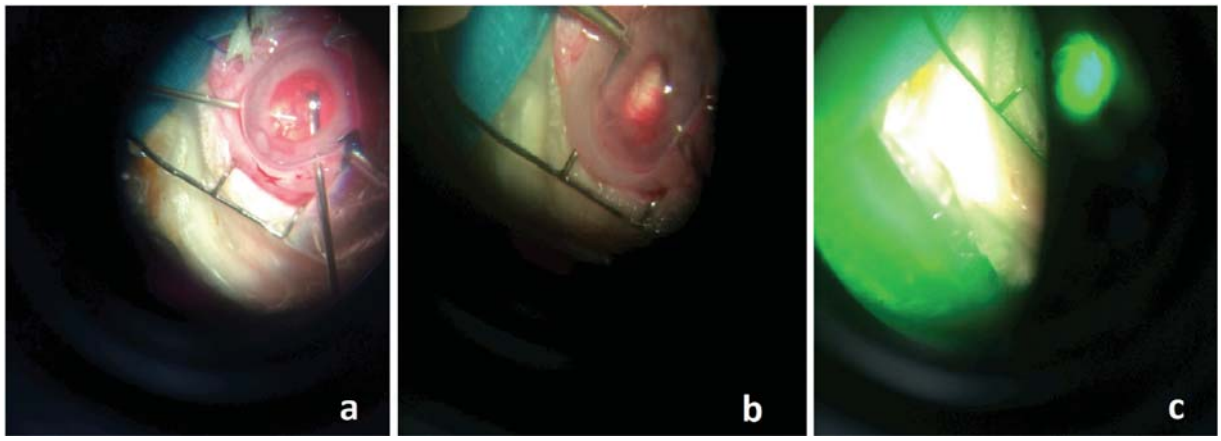


**Figure 4.** *New experimental hypotony model* **4a:** *Lensectomy and vitrectomy surgery* **4b:** *Scleral buckling* **4c:** *Argon laser endocyclophotocoagulation.*

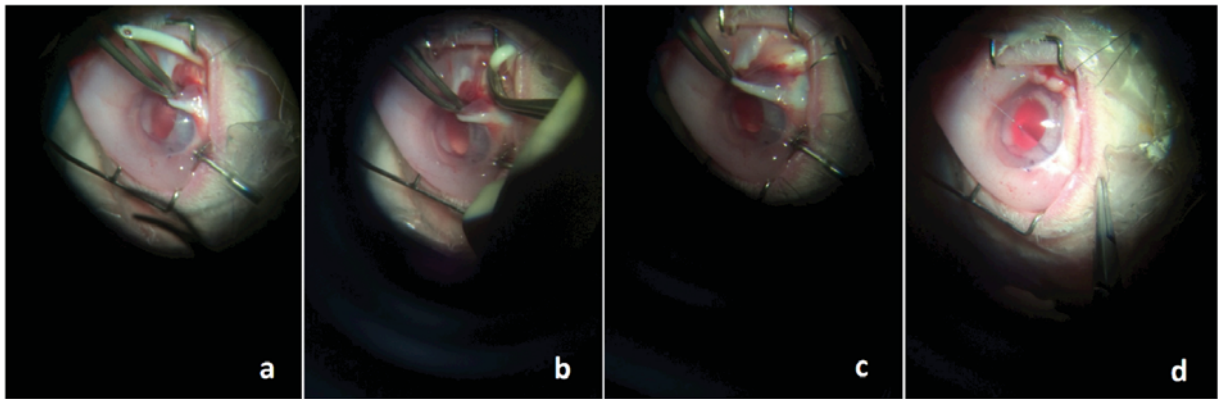


**Figure 5a-d.** *Vitreal shunt placement into the eye.*

The aqueous humor and cerebrospinal fluid have very close physical and chemical properties. In addition, IOP is similar to normal brain spinal fluid pressure (4-13 mmHg). This preliminary studieshave created a prototype for the subsequent cerebro-vitreal shunt procedure to treat COH.

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