



Figure 3. At postoperative month 8, the flap is well positioned with only mild interface haze.

every hour, and oral prednisone at a dosage of 50 mg/d for 3 days. There was no recurrence of epithelial ingrowth or diffuse lamellar keratitis. Culture results remained negative. All sutures were removed on postoperative day 11. The prednisone was tapered, and the ofloxacin treatment was discontinued. At postoperative month 8, there was mild interface haze (**Figure 3**). In the affected eye, the uncorrected visual acuity was 20/25, and the best spectacle-corrected visual acuity was 20/20 with a refractive error of -0.50 D + 0.75 D \times 72.

Comment. Previous methods of correcting a completely avulsed corneal flap following LASIK have been limited to epithelial supportive measures including application of a bandage contact lens, topical steroids to minimize haze, prophylactic topical antibiotics, lubrication, and vigilant observation to detect secondary infection. Although the lamellar flap is thought to be refractive neutral, secondary haze or irregular astigmatism may cause complications in these eyes.^{5,6} However, if the amputated flap is found, consideration can be given to replacement of the flap. The free cap should be carefully inspected to evaluate whether it is still viable. If the integrity of the free cap has been excessively compromised (ie, traumatically shredded) or it has become necrotic, it

should not be replaced. If it remains viable, any epithelialization of the stromal bed should be carefully removed, and the free corneal flap should be cultured, irrigated, and replaced with or without sutures. Suturing the flap may prevent epithelial ingrowth and assist in stretching the previously folded flap to prevent striae formation. Caution should be exercised to prevent misalignment of the flap or replacing the flap with the epithelial side down. Misalignment of the flap may not only result in irregular astigmatism but also predispose the patient to the development of epithelial ingrowth. Careful inspection of the flap under high magnification will demonstrate a smooth, shiny surface (Bowman membrane) and a slightly dull, rough surface (stroma). A bandage contact lens is placed over the eye. Prophylactic broad-spectrum antibiotics and topical steroids may also be indicated. During the postoperative period, the culture results should be monitored and the patient should receive daily follow-up to provide the earliest diagnosis of a secondary infection.

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The Use of Apraclonidine in the Diagnosis of Horner Syndrome in Pediatric Patients

Horner syndrome refers to a condition where oculosympathetic pathway damage or dysfunction can cause ptosis, miosis of the pupil, and anhydrosis.¹ Congenital Horner syndrome is most commonly idiopathic or due to a traumatic birth. Acquired Horner syndrome in chil-

dren is often postsurgical. Other causes include neuroblastoma, trauma, and brainstem processes.²

The diagnosis of this condition may be confirmed by instilling topical cocaine in each eye. The Horner pupil dilates poorly in comparison with the healthy pupil.³

Apraclonidine hydrochloride is an α -adrenergic receptor agonist that is approved for the treatment of elevated intraocular pressure following argon laser trabeculoplasty. A prior study⁴ of 6 adult patients with Horner syndrome showed that instillation of 1% apraclonidine into both eyes produced mydriasis in the affected eye only. In fact, all patients experienced a reversal of their baseline anisocoria. In every case, the miotic pupil on the Horner side dilated to become larger than the healthy side, whereas the size of the pupil on the unaffected side remained unchanged. Four patients had postganglionic Horner syndrome (third-order neuron) and 2 had preganglionic Horner syndrome (first-order and second-order neurons).

We discuss 4 pediatric patients with a diagnosis of Horner syndrome who underwent pharmacologic testing with apraclonidine. For comparison, we discuss 2 children pharmacologically tested with apraclonidine who had anisocoria but did not have Horner syndrome. To our knowledge, there is no prior report of the use of apraclonidine as a diagnostic test in a pediatric patient.

Report of Cases. *Case 1.* A 6-month-old boy was first seen in our office because of a 2-month history of anisocoria. He was the product of an uncomplicated pregnancy and was born full-term by vaginal delivery. Forceps were not used during the delivery. There was some fetal bradycardia related to contractions toward the end of the labor. He was found to have a nuchal cord and mild neck bruising. There was no evidence of shoulder dystocia, and his health had been good. His development was age-appropriate, and he reached for objects preferentially with his right hand. His parents reported that they did not notice any anisocoria before the infant was 4 months of age.

On examination, his vision was central, steady, and maintained in both eyes. In normal room lighting, the pupils measured 3 mm OD and 2 mm OS. In the dark, the size of the pupils changed to 5 mm OD and 3 mm OS. No inverse ptosis (elevation of the lower lid) was present, but there was approximately 1 mm of left upper lid ptosis. The conjunctiva, cornea, iris, and lens appeared healthy in both eyes. Motility was full, and the patient was orthotropic at nearby light reflex testing. Neurologic evaluation showed full range of motion of his neck and back. The patient kept his left hand fist with flexion contractures of the third, fourth, and fifth fingers.

We instilled 1% apraclonidine in each eye prior to any other pharmacologic agent that day. The patient was evaluated 1 hour later, and in normal room lighting, the right pupil remained 3 mm. The left pupil increased from 2 mm to 4 mm, resulting in a reversal of the anisocoria. The patient's mother reported that he was sleepier than usual that afternoon, but by evening his activity level had returned to baseline.

Computed tomography findings of the head, cervical spine, and neck were completely normal. Computed tomography results of the abdomen and pelvis were normal with no masses. Because of the left upper extremity weakness, a magnetic resonance image of the cervical and thoracic spine was ordered. These results were also normal. A diagnosis of Klumpke paralysis was made by the neurologist. The anisocoria secondary to a left-sided Horner syndrome was confirmed on 3 separate office visits.

Case 2. A 4-month-old boy was born by complicated vaginal delivery with a resultant brachial plexus injury on the left side. He displayed left upper extremity weakness with almost no motor strength in the left hand. His left pupil was noted to be smaller than his right pupil by his mother.

On examination, his vision was central, steady, and maintained in both eyes. There was 1 mm of left upper lid ptosis and 1 mm of left lower lid inverse ptosis. In bright lighting, the pupils measured 4 mm OD and 3 mm OS. In a darkened room, the

pupil size changed to 6 mm OD and 4 mm OS. One hour after the instillation of 1% apraclonidine in each eye, the right pupil measured 4 mm and the left pupil measured 5 mm, resulting in a reversal of anisocoria. The infant displayed no adverse effects from the topical medication.

Case 3. A 14-year-old boy was first seen in our office because of a 2-year history of anisocoria. His medical history was only remarkable for a positive purified protein derivative, which was treated with a 6-month course of antibiotics. A family photo album was reviewed and pictures of the boy prior to age 12 years failed to show anisocoria or ptosis, whereas photographs of the boy at age 12 years and older did show mild ptosis and anisocoria. On examination, he displayed a miotic right pupil with a greater amount of anisocoria in the dark. Mild right upper lid ptosis was present, as well as right lower lid inverse ptosis. No iris heterochromia was present.

One hour after instillation of 1% apraclonidine in each eye, the right pupil size increased from 2 mm to 4.5 mm, and the left pupil remained unchanged at 4 mm (**Figure 1**). Results from magnetic resonance imaging of his neck and chest radiography were normal, with no masses noted. The family declined any additional workup because of the chronicity of the condition.

Case 4. A 2-month-old boy was first seen in our office for a nasolacrimal duct obstruction. His mother also stated that asymmetry of the pupil size had been noticed for at least 1 month. On examination, there was left upper lid ptosis of 2 mm and left lower lid inverse ptosis. The right pupil was 4 mm and increased to 6 mm in dim light. The left pupil was 3 mm and did not change in dim light. The rest of the results of the ocular examination were normal except for eyelid crusting. The patient returned another day for testing with 1% apraclonidine. One hour after instillation, the right pupil changed from 4.5 mm to 4 mm, whereas the left pupil increased from 2.5 mm to 7 mm.

The patient then underwent urine testing. The results showed creatine (Cr) levels of 0.06 g/dL (normal range, 0.02-0.32 g/dL), ho-



Figure 1. Case 3. Left, Miotic right pupil. Right, Reversal of anisocoria 1 hour after instilling 1% apraclonidine in both eyes.



Figure 2. Case 6. Left, Miotic right pupil. Right, Right pupil is still miotic compared with the left pupil 1 hour after instillation of 1% apraclonidine in both eyes.

movanillic acid levels of 65.6 mg/g of Cr (normal range, 9.1-36.0 mg/g of Cr), and vanillylmandelic acid levels of 112.7 mg/g of Cr (normal range, 5.5-26.0 mg/g of Cr).

Because of the abnormal urine test results, computed tomography of his neck, chest, abdomen, and pelvis was performed, which revealed a 2-cm mass extending superiorly to the middle left thyroid lobe. The mass was resected and found to be a stage 1 neuroblastoma.

Case 5. A 5-month-old healthy full-term girl was first seen in our office with at least a 2-month history of anisocoria. On examination, there

was no ptosis or inverse ptosis. The pupils measured 5 mm OD and 3 mm OS. Both pupils became 1 mm larger in dim lighting. The rest of the ophthalmic examination was unremarkable. Two days later, 1% apraclonidine was instilled in each eye. One hour later, there was no change in pupil size.

Case 6. A 7-year-old boy returned for a routine follow-up examination of anisocoria. At the time of his initial examination in 1999, he underwent magnetic resonance imaging of his head and neck and radiography of the chest. He also had creatine and vanillylman-

delic acid urine tests. All test results were normal.

His right pupil measured 2 mm in room lighting and dilated to 3 mm in dim illumination. His left pupil measured 4 mm in room lighting and dilated to 6 mm in dim illumination. He had no ptosis, inverse ptosis, or iris heterochromia. The rest of the results from his ocular examination were unremarkable.

The patient received 1 drop of 1% apraclonidine in both eyes and returned to our office 1 hour later. There was no change in pupil size (**Figure 2**).

Comment. Pharmacologic diagnosis of Horner syndrome with the use of topical 5% or 10% cocaine has been the standard for years.⁵ Cocaine is a controlled substance that must be prepared by individual pharmacies for local use and has become more difficult to obtain in recent years. We have found the parents of children with anisocoria and possible Horner syndrome are often hesitant to allow the use of cocaine as a diagnostic tool when suggested.

In healthy eyes, 1% apraclonidine produces little or no dilation of the pupils. However, a previous study⁴ showed that 1 hour after 1% apraclonidine was instilled in both eyes of 6 adult patients with Horner syndrome, a reversal of anisocoria occurred. This occurred whether the Horner syndrome was preganglionic or postganglionic. The mydriatic response observed in eyes affected with Horner syndrome is due to the denervation supersensitivity of the α -1 receptors on the iris dilator muscle.⁴

Topical apraclonidine is readily available and has been used in the past to treat glaucoma with minimal adverse effects. To our knowledge, we report the first use of this agent in the pharmacologic diagnosis of pediatric Horner syndrome. We discussed 4 patients (cases 1-4) with Horner syndrome, all of whom experienced a reversal of anisocoria after receiving 1% apraclonidine. To contrast this, we discussed an additional 2 patients with anisocoria not related to Horner syndrome (cases 5 and 6), who experienced no change in pupil size. This reversal of anisocoria is easier to observe clinically than the asymmetric mydriasis cocaine produces.

Based on the findings in our cases and the prior article with 6 adult patients with Horner syndrome, apraclonidine may play a role in the diagnosis of Horner syndrome. In fact, the diagnosis of neuroblastoma in case 4 may have been delayed if we had not tested the patient with 1% apraclonidine.

If it could be shown that 0.5% apraclonidine is as effective in causing a reversal of anisocoria in Horner syndrome as 1% apraclonidine, it would be an even more attractive agent to use for this purpose. Ad-

verse effects would be minimized, and 0.5% apraclonidine is more readily available. Although the previously published article using apraclonidine to diagnose adults with Horner syndrome had cases of preganglionic and postganglionic lesions, the number of patients was small. Sensitivity and specificity of this diagnostic test need to be investigated. Also, with all pharmacologic tests based on denervation hypersensitivity, a false-negative result may occur in the acute setting (where denervation hypersensitivity has not yet developed).

Further studies are also necessary to evaluate the safety profile of the instillation of this agent in infants. Another α -adrenergic receptor agonist used to lower intraocular pressure, 0.2% brimonidine tartrate, has caused apnea, bradycardia, hypotension, somnolence, and lethargy in children.⁶ One of the infants in our study who received 1 drop of 1% apraclonidine in both eyes was somewhat sleepier than usual that afternoon, but this resolved without adverse sequelae. The other patients did not experience any adverse effects.

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The Use of N-Butyl Cyanoacrylate (Indermil) in Lateral Tarsorrhaphy

Exposure keratitis occurs in facial nerve palsy and may lead to visual loss resulting from corneal damage unless it is treated appropriately. Tarsorrhaphy may be required in more severe cases. This report describes a simple way of performing temporary tarsorrhaphy in the outpatient setting.

Report of Cases. Three consecutive patients with exposure keratopathy were treated with N-butyl-2-cyanoacrylate (Indermil; Henkel Loctite Corporation, Dublin, Ireland) tarsorrhaphy. Indermil-assisted tarsorrhaphy is simple and is easily performed in the outpatient setting. The eyelid is cleaned with isotonic sodium chloride solution and thoroughly dried with a cotton bud. The patient is instructed to close his or her eyes, and Indermil is applied directly to the eyelid margin (**Figure 1**). The glue should be applied as a thin film by mounting a Southampton (**Figure 2**) or lacrimal cannula at the end of the tube. Light pressure is then applied to the eyelid margins with cotton buds for 30 seconds to enhance adhesion. The patient is advised to avoid wetting the eyelids for the next few hours.

Case 1. A 45-year-old man was seen in the eye casualty service, complaining of decreased vision in the right eye. Four weeks previously, he had undergone surgery to remove an acoustic neuroma. An examination revealed a large central corneal abrasion in the right eye, with absent corneal sensation and lower motor neuron facial nerve palsy (**Figure 3**).

Conservative treatment with topical lubricants and antibiotics failed. The patient was offered surgical tarsorrhaphy, but the offer was declined. Instead, Indermil was used to close the lateral eyelids (**Figure 4**). The tarsorrhaphy was satisfactory and lasted 4 days, during which the epithelial defect decreased in size. Thereafter, tarsorrhaphy was repeated twice without complications. The second application lasted 7 days, and the