Comment. Pharmacologic diagnosis of Horner syndrome with the use of topical 5% or 10% cocaine has been the standard for years. 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 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 study 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.

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. Adverse 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. 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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third lasted 6 days. The corneal defect healed completely after the third application.

Case 2. A 17-year-old woman was referred to the eye casualty service with a 5-day history of left-sided Bell palsy associated with a painful eye. Examination revealed 4 mm of lagophthalmos associated with poor Bell phenomenon and a
corneal abrasion. Chloramphenicol ointment was prescribed, and the patient was advised to tape her left eyelid shut at night. Four days later, corneal exposure with a persistent corneal abrasion was still evident. Surgical tarsorrhaphy was refused; however, the patient consented to tarsorrhaphy with Indermil. The tarsorrhaphy lasted 7 days, during which the corneal abrasion healed. Function of the left facial muscles was restored during this period, and the exposure keratopathy resolved.

Case 3. A 40-year-old man was seen in the eye casualty with a 4-day history of facial nerve palsy secondary to herpes zoster infection. Corneal exposure with a central abrasion was evident. There was 5 mm of lagophthalmos. He was prescribed chloramphenicol ointment and advised to tape his eyelids shut at night. He subsequently developed a skin allergy to the tape and discontinued its use. A subsequent examination found a persistent central corneal abrasion. The patient refused surgical tarsorrhaphy but was agreeable to lateral tarsorrhaphy with Indermil. The first tarsorrhaphy lasted 6 days but his lower eyelid function remained poor, and worsening of the exposure keratitis occurred over the next 5 days. Glue tarsorrhaphy with Indermil was performed on 2 other occasions, each lasting 7 days. At the end of the third application, the corneal defect had healed and eyelid function recovered satisfactorily.

Comment. Exposure keratitis is a complication of facial nerve palsy. Without treatment, this may lead to corneal ulceration with severe visual loss from scarring and infection. In mild cases, exposure keratopathy can be managed conservatively with copious lubricants and eyelid taping, but in severely affected corneas, eyelid closure may be required to maintain corneal integrity. Surgical tarsorrhaphy and botulinum toxin–induced ptosis are 2 well-recognized methods of providing corneal protection. Both are effective but have their disadvantages.

Surgical tarsorrhaphy may be divided into suture tarsorrhaphy over bolsters (for short-term use) and reversible permanent eyelid adhesion tarsorrhaphy (for longer-term use). The former is commonly accepted as the gold standard for temporary eyelid closure. However, both forms of surgical tarsorrhaphy are time consuming, and there may be a risk of permanent scarring to the eyelids from surgery. In addition, patients often refuse surgical tarsorrhaphy for cosmetic reasons. On the other hand, botulinum toxin may not be available universally because of constraints of cost and expertise. Moreover, the induced ptosis is variable in its onset and duration, and there are risks associated with the injection.

An alternative approach is to perform glue-assisted tarsorrhaphy. Indermil is a tissue adhesive (N-butyl-2-cyanoacrylate monomer) that is widely used in surgery for the closure of skin wounds and internal wounds without the need for suturing. The use of Indermil has also been described in obstetric and gynecologic procedures, otolaryngologic procedures, hand surgery, and plastic and reconstructive surgery. The use of cyanoacrylate glue has been described previously in the management of corneal epithelial defects and other ocular problems. However, there has been little documentation to date on the successful use of licensed medical preparations of tissue glue for tarsorrhaphy.

Indermil-assisted tarsorrhaphy lasts for about a week and can easily be repeated when necessary. With regard to safety, a previous case series has suggested that there is no long-term morbidity from superglue contact with the eye. The technique is not a replacement for surgical tarsorrhaphy; however, it may be considered as an alternative in certain situations. First, the technique can be used to provide short-term corneal protection prior to recovery of facial nerve palsy. Second, it may serve as a temporary measure for exposure keratopathy while awaiting more definitive treatment. Third, it is of value in patients who refuse surgical intervention.

**Report of a Case.** A 38-year-old Mexican American man sought care because of decreased vision in both eyes for the past 9 months, although it had worsened in the past 6 weeks. He also complained of neck stiffness and headaches. He was a butcher, had lived in Mexico until age 22 years, and had visited there 2 years previously. His visual acuity was 20/70 OD and 20/100 OS. Fundus examination showed serous retinal detachments, and Vogt-Koyanagi-Harada syndrome was diagnosed.

He was treated with 100 mg of oral prednisone per day. His visual acuity improved slightly to 20/50 OD and 20/100 OS but then deteriorated to 20/200 OD and 20/60 OS. His serous detachments did not resolve. During the next 10 months, he was treated with 1 injection of 40 mg of sub-Tenon triamcinolone acetone in the right eye, 25 mg of oral methotrexate weekly, 60 mg of oral prednisone daily, and 1 injection of 4 mg of intraocular triamcinolone in the right eye. His visual acuity and serous retinal detachments did not improve.

On May 15, 2002, the patient’s visual acuity was 20/70 OD and 20/50 OS, and he had multiple serous retinal detachments with fibrin in each eye (Figure 1A). Fluorescein angiography showed multiple areas of leakage (Figure 1B).