Proboscis Lateralis

Proboscis lateralis describes a rudimentary nasal structure or appendage that is located off-center from the vertical midline of the face. Proboscis lateralis is a rare craniofacial malformation frequently associated with abnormalities of the eyes and/or ocular adnexa. We report a case with ipsilateral colobomatous microphthalmia and choanal atresia.

Report of a Case. A 3.5-kg black male infant was born at 40 weeks' gestation by uncomplicated spontaneous vaginal delivery with Apgar scores of 8 and 9 at 1 and 5 minutes. At birth, the patient was noted to have a 2.5 × 1.1-cm trunk-like appendage (Figure 1) arising from his left medial canthus with a clear mucoid discharge draining from an orifice at its distal end. Also noted were left microphthalmos, left choanal atresia, and a mildly hypoplastic left nasal ala. Prenatal history was negative for consanguinity, exposure to alcohol, ionizing radiation, prescription medications, or recreational drugs. The patient's mother denied any family history of blindness, craniofacial abnormalities, mental retardation, or other congenital defects. Chromosomal analysis was reported as 46,XY. Findings from examination of the left eye were remarkable for microphthalmia with a horizontal corneal diameter of 5.0 mm. The anterior chamber was well formed and the lens was clear. Funduscopic examination results were remarkable for a posterior chorioretinal coloboma with a partial retinal detachment. Findings from examination of the right eye were normal.

Computed tomographic images (Figure 2 and Figure 3) demonstrated normal cerebral parenchyma, ventricular architecture, and mid-line anatomy. Hypoplasia of the left nasal passage with left-sided choanal atresia was present. A defect in the medial wall of the left orbit was noted, and a tubular soft tissue structure extended from the medial aspect of the preseptal soft tissue and appeared continuous with the nasal cavity and ethmoid sinus. The left globe was small and dysplastic with a colobomatous cyst posteriorly.

Surgical excision of the proboscis was performed at age 4 months. Since the nasal alae were relatively well developed, reconstruction was unnecessary, and the soft tissue appendage was simply amputated from its origin at the superior aspect of the left medial canthus. The fistulous tract to the ethmoid sinus was excised, and the choanal atresia was then repaired.

Pathologic findings revealed an oblong, skin-covered, tubular mass.
proboscis lateralis measuring 2.5 cm in length \times 1.1 \text{ cm} in greatest diameter. At the distal end of the appendage was a 3-mm orifice that was patent to probing. Microscopic examination of sections from the mass (Figure 5 and Figure 6) revealed a hamartomatous malformation covered by hair-bearing skin. The stromal soft tissues were noted to be composed of fibroadipose tissue with abundant bundles of skeletal muscle. A central canal was lined by squamous mucosa distally, changing to respiratory-type mucosa more proximally. Small plates of hyaline cartilage as well as several normal-appearing peripheral nerves were noted in the soft tissues adjacent to the central canal.

Comment. Proboscis lateralis is a rare congenital anomaly in which a tubular, nose-like structure is seen to arise from the medial canthus, which is very rare, may generate a lateral proboscis in the absence of any other facial anomalies. Usually, however, the nasal placode develops abnormally, which may result in legion midfacial anomalies, but continues to act on mesenchymal tissue, allowing it to fuse into the tubular lateral proboscis.

Although the proboscis generally arises from the area of the medial canthus, exceptionally rare cases have been described in which the anomalous structure arises from the lateral canthus,4 nasal root, chin,10 or is present bilaterally.11 This trunk-like appendage is generally 2 to 3 cm in length and 1 cm in diameter and has a central tract lined with respiratory epithelium. The tract drains at a dimpled opening at the distal end of the proboscis and may be continuous with the paranasal sinuses proximally. Generally, there is heminasal hypoplasia or aplasia on the side of the proboscis although, in rare cases, the nose is normal. Anomalies often affect the nasal cavity as well as the nares, and complete closure of the nasal cavity at the piriform aperture may be seen in cases in which heminasal aplasia is present.2 Cleft lip and/or palate may also be present. To the best of our knowledge, this is the first reported case of proboscis lateralis associated with choanal atresia.

Of interest to the ophthalmologist is the frequent association of abnormalities of the eye and ocular adnexa with proboscis lateralis. Although Wang et al9 reported that lids complete with both eyelashes and a conjunctival lining. Selenkoff, however, is often credited with first describing proboscis lateralis in an 1884 autopsy.2
ocular defects are rare in patients with proboscis lateralis, a subsequent review of the literature by Boo-Chai noted that 24 of 34 patients with proboscis lateralis had associated anomalies of the ipsilateral eye and/or ocular adnexa. These abnormalities included anophthalmia, microphthalmia, microcornea, lenticular opacities, cyclopaean eye, and colobomas of the choroid, retina, iris, and eyelids. The presence or absence of ocular abnormalities was used by Boo-Chai to help categorize patients with proboscis lateralis into 4 groups: group I has a lateral proboscis with a normal nose (but may have ocular findings); group II, lateral proboscis with an ipsilateral deformity of the nose; group III, ipsilateral deformity of the nose and the eye and/or ocular adnexa; and group IV, cleft lip and/or palate in addition to the nasal and ocular abnormalities.

It is noteworthy that most patients with proboscis lateralis do not have serious central nervous system abnormalities, in stark contrast to a mid-line proboscis, which is often indicative of holoprosencephaly. Nonetheless, proboscis lateralis may coexist with central nervous system anomalies, and early neuroimaging is indicated to rule out intracranial abnormalities.

Because there is some variability in facial anomalies and the degree of nasal hypoplasia seen with proboscis lateralis, management must be individualized. When marked hypoplasia or aplasia of the nasal ala is present, reconstruction is indicated. The structure and texture of the proboscis make it an ideal substrate for nasal reconstruction, and for this reason, the proboscis should not be excised if future nasal reconstruction is anticipated. Depending on the size and location of the proboscis and the degree of nasal hypoplasia, a variety of techniques may be used to reconstruct an aesthetically acceptable nare, including use of the proboscis as a pedicle flap.

In our patient, ipsilateral nasal hypoplasia was minimal, and simple amputation of the proboscis and excision of the fistula connecting it to the ethmoid sinus was appropriate. However, endoscopic repair of the choanal atresia was complicated by the narrow nasal opening and required conversion to a transpalatal approach. At one time, it was suggested that the affected eye be enucleated to prevent formation of a fistula to the meninges and subsequent meningitis. There is no evidence, however, that such communication occurs, and enucleation is not recommended unless tumor is suspected.

Because of the variety of maxillofacial and ocular disease seen with proboscis lateralis, optimal care of the patient warrants a multidisciplinary approach that may involve an otolaryngologist or ophthalmologist, maxillofacial surgeon, plastic surgeon, and ophthalmologist.

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Late Occurrence of Diffuse Lamellar Keratitis After Laser In Situ Keratomileusis

Diffuse lamellar keratitis (DLK) is a noninfectious inflammatory complication associated with laser in situ keratomileusis (LASIK). Post-LASIK sterile interface keratitis has also been described as “sands of the Sahara syndrome” and “central focal interface opacity after LASIK.” The corneal infiltrates may be focal or multifocal but remain confined to the lamellar interface without extension, anterior chamber reaction, or associated epithelial defect. Common to all previously reported cases is an onset within 1 month after LASIK treatment, enhancement, or flap manipulation. We report 2 cases of DLK appearing after the immediate postoperative period (2-7 months after LASIK), 1 of which had bilateral involvement.

Report of Cases. Case 1. A 48-year-old woman with euthyroidism and a treatment history of hypothyroidism underwent bilateral sequential LASIK using a 180-µm Hansatome microkeratome (Bausch and Lomb, Rochester, NY) and a Summit Apex Plus excimer laser (Summit Technology, Waltham, Mass). Preoperative refraction of −4.75 +1.50 × 092 OD and −4.50 +1.75 × 085 OS yielded a best-corrected visual acuity of 20/20 OU. Fifty percent epithelial flap defects in both eyes and mild DLK in the left eye were present on postoperative day 1. The defects healed with bandage soft contact lens wear and the DLK resolved after 1 week of intensive flurometholone treatment. One month postoperatively, uncorrected visual acuity was 20/40 OD and 20/30 OS. Refraction of +0.25 +1.50 × 102 OD and −0.75 +0.75 × 092 OS yielded a best-corrected visual acuity of 20/25 OU. The flaps were well healed with the exception of mild interpalpebral punctate epithelial staining in both eyes. She was offered bilateral lower punctal plugs, which she declined.

Two months postoperatively she complained of a sudden onset of redness and a foreign body sensation in her left eye. Examination was remarkable for an uncorrected visual acuity of 20/40 OS. Slitlamp biomicroscopy showed minimal diffuse conjunctival hyperemia and a focal 2 × 2-mm nonsupplicative infiltrate in the lamellar interface inferocentral to the pupil. Mild edema of the flap without epithelial defects was noted. The anterior chamber was quiet. A regimen of hourly topical ofloxacin was started. Examination 24 hours later revealed a visual acuity of counting fingers. Slitlamp biomicroscopy showed severe diffuse conjunctival hyperemia, diffuse stromal edema with a large central epithelial defect, and a diffuse lamellar infiltrate without anterior or posterior extension (Figure 1). The lamellar flap was lifted and debrided of soft infiltrate. Scrapings were sent for aerobic, anaerobic, atypical mycobacterial, and fungus staining and culturing. The interface was irrigated with fortified vancomycin and tobramycin eye drops, the flap was repositioned, and the bandage soft contact lens was applied. Fortified vancomycin and tobramycin eye drops were administered hourly. One day later, discrete granular infiltrates reappeared in the interface. Stains and cultures revealed no organisms. Topical prednisolone acetate was given every 2 hours to treat DLK and then hourly when cul-

![Figure 1](image-url)
tures remained without growth after 48 hours. On complete reepithelialization, the bandage soft contact lens was removed and the dose of fortified antibiotics was decreased to 4 times per day. Prednisolone was taken hourly. On day 21, uncorrected visual acuity was 20/60 OS. Refraction of plano +1.00 × 180 yielded a best-corrected visual acuity of 20/30 OS. Slitlamp biomicroscopy showed mild interface scarring centrally, 2+ granular infiltrates, and 1+ stromal flap edema (Figure 2).

The left eye remained stable during the prednisolone dose taper. However, 3 months postoperatively, her right eye showed a small epithelial erosion without keratitis. On follow-up day 3, slitlamp biomicroscopy of the right eye revealed a healing erosion with few focal interface opacities and mild diffuse granular infiltrates (Figure 3). The keratitis resolved after a 5-day regimen of prednisolone acetate taken hourly and ofloxacin taken 4 times per day.

Case 2. A 51-year-old woman with myopic amblyopia currently taking thyroxine for hypothyroidism underwent bilateral LASIK for treatment of high myopia. She came for a second opinion regarding management of superior limbal keratoconjunctivitis in the right eye 7 months postoperatively. Refraction of −1.75 yielded a visual acuity of 20/40 OD. Slitlamp biomicroscopy was remarkable for mild superior conjunctival hyperemia, redundancy, and rose Bengal staining. She underwent superior conjunctival resection and was given tobramycin/dexamethasone ointment to apply 4 times per day. Three days after resection, she complained of worsening pain and decreased vision. On examination, her best-corrected visual acuity was 20/80 OD. The superior conjunctival defect was healing well without suppuration. Diffuse granular infiltrates were visible in the flap interface without epithelial defects or anterior chamber reaction. Her regimen was switched to ofloxacin taken 4 times per day and prednisolone acetate taken hourly. Follow-up examination 48 hours later showed best-corrected visual acuity to be 20/40 OD with completely resolved interface keratitis.

Comment. The causes of DLK are not clearly defined. A rare postoperative complication of LASIK, with an estimated incidence of 3 in 400,2 DLK is thought to be a secondary inflammatory response to a variety of potential agents within the space of the flap interface. Four clinical stages have been described, ranging from non–visually significant focal infiltrates to diffuse infiltrates with stromal necrosis.5 Known to occur within a week after either primary LASIK or enhancement procedures, DLK has also been reported after epithelial flap debridement to reduce visually significant basement membrane irregularities 1 month after LASIK enhancement.6

Unusual in our cases is the late onset of DLK. In the most severe case, the lack of initial epithelial defect, confinement of infiltrate to the interface, rapid progression despite hourly ofloxacin treatment, rapid improvement with intensive topical steroids, and negative microbiology culture results strongly support a diagnosis of DLK rather than...
infectious keratitis. One case series review of infectious ulcerative keratitis following LASIK reported that all cases had epithelial defects and none had infiltrate confined only to the interface.5 Our case 1 had documented recurrent epithelial erosions and typical DLK in the acute postoperative period. Case 2 had probable microdisruption of the superior corneal epithelium after manipulation of the superior conjunctiva. We propose a mechanism whereby an inflammatory reaction to corneal surface disruption occurs in the potential space of the flap interface resulting in a clinical picture of lamellar keratitis.

We report these 2 cases to bring attention to the possibility of DLK occurring beyond the immediate postoperative period, especially when the potential for corneal surface disruption exists. As more patients undergo LASIK, more cases of late-onset DLK will be seen. Care must be taken to exclude infectious keratitis. However, the prognosis for recovery is good following intensive topical steroid treatment.

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Retinal Degeneration Associated With Congenital Transcobalamin II Deficiency

Transcobalamin II (TCII) is a cobalamin (Cbl)-binding plasma protein that promotes the cellular uptake of Cbl (vitamin B12) by many tissues. Transcobalamin II deficiency is a rare autosomal recessive disorder.1,2

Report of a Case. A girl, born to healthy nonconsanguineous parents, was seen at age 3 months with pallor, lethargy, failure to thrive, and hypotonia. At age 7 months, she was seen by a physician because of pallor, purpura, hypotonia, myoclonia, epileptiform episodes of blinking, and chronic upper respiratory infections. Her blood cell count revealed severe pancytopenia. Serum Cbl levels were in the low to normal range. Methylmalonyl aciduria and homocystinuria were detected. The total unsaturated Cbl binding capacity of serum, measured as previously described,2 was 48 pmol/L (reference range, 440-880 pmol/L), without binding of [14C]Cbl to TCII. Immunoactive TCII serum levels were 95 pmol/L (reference, >370 pmol/L). Culture findings from the patient’s fibroblasts incubated with 35S-methionine expressed immunoactive radio-labeled TCII with the same molecular weight as native TCII, but no Cbl-binding TCII was secreted into the culture medium. The patient was treated with intramuscular hydroxocobalamin, 1000 µg every 10 days for 1 year, and subsequently with oral hydroxocobalamin (1000 µg per day) and oral folic acid until age 16 years.

At age 13 years, she was seen for headaches, lipothyama, epileptiform myoclonic episodes, cerebellar dysfunction, impairment of the pyramidal track, and was found to be moderately retarded. There were no ocular abnormalities. However, at age 16 years, she complained of a decrease in visual acuity, found to be 20/30 OU. Findings from fundus examination revealed a dark oval in the macula, surrounded by a ring of hypopigmentation. These findings were bilateral and symmetrical. The peripheral fundus examination showed a diffuse area of salt and pepper retinopathy associated with rare bone spicule formation (Figure 1). Fluorescein angiography showed an ovoid zone of hypofluorescence in the macula surrounded by a diffuse hyperfluorescence (Figure 1). Indocyanine green angiography findings are shown in Figure 2. Visual field analysis demonstrated bilateral annular perifoveal scotomata. Electroretinogram analysis showed decreased amplitude of both A and B waves. The patient was then started on intramuscular hydroxocobalamin (5000 µg 3 times per week). The episodes of myoclonia and the cor- donal posterior pyramidal signs decreased, as did the methylmalonyl aciduria and homocystinuria. At age 19 years, best-corrected visual acuity, visual field analysis, electroretinogram recording, and fluorescein and indocyanine green angiographies remained unchanged.

Comment. The retinal findings in this patient support the diagnosis of "unusual" pigmentary retinopathy, with salt-and-pepper fundi. A peculiar bilateral brown macular ovoid zone was observed, which was hypofluorescent on fluorescein and indocyanine green angiographic studies. This retinal degeneration may be similar to the ophthalmologic complications reported in some patients with the inherited Cbl deficiency. Indeed, 5 patients with inherited Cbl deficiency and a similar retinopathy to that described in our patient have been reported, but most of them died in infancy.1,3,4

To our knowledge, this is the first report of retinal dystrophy associated with congenital TCII deficiency. It is notable that while the patient was receiving high doses of intramuscular hydroxocobalamin
supplementation, no progression of retinal degeneration was observed.

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Interferon-Associated Retinopathy and Cystoid Macular Edema

Many cases of interferon-associated retinopathy have been reported since the first case in 1990.1 This disease, characterized by retinal hemorrhage and cotton-wool spots, has a good prognosis.2,3 We describe the first patient with interferon-associated retinopathy with decreased vision due to bilateral cystoid macular edema (CME).

Report of a Case. On February 4, 1999, a 24-year-old man with chronic, active hepatitis C began treatment...
with interferon beta at a daily dose of $4 \times 10^6$ U for 9 weeks. His corrected visual acuity was 20/20 OU and no abnormal fundus findings were noted in either eye before treatment. About 1 month later, the platelet count decreased from $350 \times 10^9$/L to $160 \times 10^9$/L and the triglyceride level increased markedly from 1.26 to 4.73 mmol/L (112-419 mg/dL). The patient also developed proteinuria and hypoalbuminemia (serum albumin level decreased from 0.039 to 0.023 g/L). Three days before the end of treatment, many cotton-wool spots and retinal hemorrhages were found in both fundi. Although the corrected visual acuity remained at 20/20 OU, the interferon therapy was terminated. However, the bilateral retinopathy worsened 11 days after the termination of interferon treatment (Figure 1). The visual acuity decreased to 20/40 OU because of CME.

Fluorescein angiography (Figure 2) showed capillary nonperfusion at the cotton-wool spots, capillary microaneurysms, and diffuse capillary dilatation. In the late phase, diffuse leakage from the dilated capillaries resulted in the pooling of fluorescein, with a petaloid appearance in the center of the macula. No treatment except routine follow-up was given.

Thirty-three days after the termination of interferon therapy, the CME disappeared and the visual acuity improved to 20/20 OU. The retinopathy resolved and the visual acuity remained stable.

Comment. Interferon-associated retinopathy is typically characterized by retinal hemorrhages and cotton-wool spots. However, various atypical interferon-associated ocular complications have been reported, including oculomotor nerve paralysis; optic disc edema; subconjunctival, preretinal, and vitreous hemorrhage; and retinal vein occlusion. Although severe visual losses due to atypical ocular complications have been reported, there have been no cases reported of visual decrease due to CME secondary to interferon-associated retinopathy.

Although the pathogenesis of interferon-associated retinopathy is not known, the deposition of immune complexes in vessels, immunological dysfunction, and increased adhesion of activated leukocytes to vascular walls have been suggested. Diabetes mellitus and

![Figure 1](image1.png)

**Figure 1.** Fundus photograph 11 days after the termination of interferon treatment showing a large number of cotton-wool spots and retinal hemorrhages. Dilatation and tortuosity of the retinal veins and mild tortuosity of the retinal arteries are noted. A, Right eye. B, Left eye.

![Figure 2](image2.png)

**Figure 2.** Fluorescein fundus angiography 11 days after the termination of interferon treatment. A, Arteriovenous-phase angiogram (22 seconds, right eye) showing capillary nonperfusion at the site of cotton-wool spots, capillary microaneurysms, and diffuse capillary dilatation. Angiographic findings from the left eye were similar (angiogram not shown). B, Late-phase fluorescein angiogram (326 seconds, right eye) showing marked diffuse leakage of fluorescein. Pooling of fluorescein with a petaloid appearance is noted in the center of the macula. Marked diffuse leakage of fluorescein and pooling of fluorescein in the cystoid spaces were observed in the left eye (angiogram not shown) as noted in the right eye.
hypertension, decreased platelet counts, and increased triglyceride levels during interferon treatment are risk factors of interferon-associated retinopathy. Our patient did not have any underlying systemic disease that might involve a risk of interferon-associated retinopathy. However, the platelet count decreased and triglyceride level increased after interferon therapy, resulting in a blood condition that might have caused a susceptibility to retinopathy. Our patient had hypoalbuminemia, which might have been responsible for the CME. Hypoalbuminemia decreases plasma oncotic pressure, and therefore decreases the oncotic pressure difference between the plasma and interstitial fluid. As a result, the influx of water from the interstitial space to the capillary may decrease, as explained by the Starling law. This decreased water reabsorption may have led to the marked cystoid macular edema in this situation, where interferon has already damaged retinal vessels and consequently increased vascular permeability.

Our findings suggest that interferon-associated retinopathy progresses and the visual acuity decreases even after the termination of interferon therapy. They also suggest that hypoalbuminemia due to proteinuria is a risk factor of CME. However, interferon-associated retinopathy has a good visual prognosis without any treatment even when accompanied by CME.

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Prophylaxis of Vasovagal Reaction With Atrohist Plus

Vasovagal reactions (VVRs) can present a treatment dilemma for medical professionals, and fear of causing such reactions can prevent ophthalmologists from performing necessary examinations. Atenolol, midodrine hydrochloride, paroxetine, fludrocortisone acetate, as well as salt and fluid intake have demonstrated efficacy in treating the disorder. Other vasoconstrictors and selective serotonin reuptake inhibitors are being studied, but to our knowledge, there are no medications to prevent isolated incidents of VVR with known triggers. We describe 2 patients who experienced VVRs on applanation tonometry or instillation of dilating drops. Subsequent reactions were prevented with oral Atrohist Plus (a combination of phenylephrine hydrochloride, phenylephrine hydrochloride, chlorpheniramine maleate, phenylpropanolamine hydrochloride, atropine sulfate, and scopolamine hydrobromide; Vintage Pharmaceuticals Inc, Charlotte, NC) administered 1 hour prior to examination.

Report of Cases. Case 1. A 41-year-old healthy man with an ocular history of myopia and a family history of glaucoma was seen for complaints of decreased visual acuity for several months. After instillation of a fluorescein sodium–benoxinate hydrochloride solution, the applanation tonometry demonstrated the intraocular pressure (IOP) of 21 mm Hg OU, and immediately after applanation, the patient fainted. Owing to lightheadedness, he refused additional drops for dilation. The undilated fundus examination revealed a cup-disc ratio of 0.50 U. The patient was followed as a glaucoma suspect, and after each IOP measurement, he felt faint, sweaty, or light-headed. On one occasion, he experienced a severe VVR, became lightheaded, tremulous, diaphoretic, and fainted. Blood pressure and pulse were measured immediately following this presumed VVR and were found to be normal, 134/84 mm Hg and 74 bpm, respectively. No formal testing for VVR was performed.

Repeated IOP examinations were indicated, but additional tonometry measurements incurred a high risk of another syncopal episode. Since there are no oral medications identified in the literature to prevent isolated incidents of VVR, intravenous atropine was suggested by a cardiologist. As an alternative to the intravenous medication, Atrohist Plus was selected, and 1 tablet was administered to the patient 1 hour prior to applanation measurement. Applanation tonometry was performed, and the patient did not experience any symptoms of VVR. One tablet of oral Atrohist Plus was administered 1 hour prior to his next 3 office visits, and no symptoms of VVR were reported.

Case 2. A 21-year-old healthy man with no medical problems was known to experience a VVR and faint after the instillation of dilating drops. The patient had an ocular history of myopia and contact lens wear and was seen for a comprehensive examination and laser in situ keratomileusis consultation. One tablet of Atrohist Plus was administered approximately 1 hour prior to the eye examination. A complete eye examination was performed, including applanation tonometry followed by instillation of dilating drops (1% tropicamide and 2.5% phenylephrine). No symptoms of VVR were reported.

Comment. Predictable VVRs and fainting during tonometry or instillation of eye medications are uncommon occurrences but may force the ophthalmologist to choose between a comprehensive eye examination and an adverse event. The use of the oral medication, Atrohist Plus, can be a useful tool in the prevention of VVR when the ophthalmologist is aware of the precipitating factor. The primary indication for use is for relief from irritation of sinus, nasal, and upper respiratory tissues.
owing to its vasoconstrictive properties and subsequent drying of the mucosa. The drug is contraindi-
cated in patients receiving mono-
amine oxidase inhibitors, patients
with asthma, patients younger than
12 years, women who may be preg-
nant, or patients with known hy-
persensitivity to antihistamines or
sympathomimetics. In addition,
acute angle-closure glaucoma may
be precipitated by the dilating ef-
fects of this oral medication; there-
fore, narrow angles should be ruled
out before its administration.3

Atrohist Plus presumably pre-
vents VVRs through the anticholin-
ergic effects of atropine, hyoscya-
mine, and scopolamine and the
adrenergic effects of phenyleph-
rine and phenylpropanolamine. The
anticholinergics act by blocking ace-
tylcholine at the muscarinic recep-
tors in smooth muscle, cardiac
muscle, and sinoatrial and atrioven-
tricular nodes as well as in exo-
crine glands, resulting in increased
heart rate and blood pressure and
decreased heart block. Phenylephrine
acts primarily on $\alpha_1$ adrenergic re-
ceptors in arterial smooth muscle to
cause vasoconstriction, while phen-
ylpropanolamine acts on both $\alpha_1$ and $2$ and $\beta_1$ receptors to cause va-
soconstriction as well as increased
heart rate, contractility, and car-
diac output. The combination of
medications most likely acts by pre-
venting the bradycardia and hypo-
tension associated with VVR.4

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Correction

Spelling Error. In the Photo Essay titled “Iris Creep Pro-
ducing Corectopia in Response to Molteno Implants,”
published in the February issue of the ARCHIVES (2001;
119:304), “corectopia” should have been spelled with only
1 “r.” Additionally, in that same article, “ectropium uvae”
should have been written, “ectropion uveae.”