Evaluation of Abduction Deficits in Pediatric Patients

A Case Report & Review of Differentials

■ Marie I. Bodack, O.D.

Abstract

Pediatric patients who present with abduction deficits may or may not be symptomatic. In asymptomatic patients, the deficit may be found only on routine extraocular muscle evaluation. Among the differentials for such a finding are spasm of the near reflex, sixth nerve palsy, Duane's retraction syndrome, and myasthenia gravis.

A 12-year-old Hispanic male presented for a routine eye examination. All external testing, with the exception of extraocular muscles, was within normal limits. On extraocular muscle testing, the patient exhibited a right abduction deficit in right gaze and reported pain. Refraction revealed low hyperopia. Ocular health was within normal limits. Because of the abduction deficit, the patient was referred to the neuro-ocular clinic for an evaluation where he was diagnosed with spasm of the near reflex. He was prescribed reading glasses and reported an improvement in his symptoms.

Abduction deficits, especially in pediatric patients, can prove to be challenging to the optometrist. In order to help arrive at a diagnosis, the case history should focus on neurological symptoms, developmental delays and birth history. Additional questions regarding recent colds, ear infections or contact with deer ticks can help narrow the differentials. Testing should focus on ruling out a neurological etiology. In cases where a neurological problem is suspected, the appropriate referrals must be made.

Key Words

abduction deficit, Duane's retraction syndrome, extraocular muscles, myasthenia gravis, sixth nerve palsy, spasm of the near reflex

INTRODUCTION

An abduction deficit can occur bilaterally or unilaterally. Among the etiologies for abduction deficits in pediatric patients are spasm of the near reflex, sixth nerve palsy, Duane's retraction syndrome and myasthenia gravis. Depending on the etiology, the patient may or may not be symptomatic. Consequently, the defect may be discovered only on careful routine extraocular muscle testing. When a practitioner observes an abduction defect, additional questions should be asked, and supplemental testing performed in order to rule out a neurological problem. In some cases, evaluation by a neurologist is required to confirm a diagnosis or to manage the patient.

CASE REPORT

History

A 12-year-old Hispanic male presented at a school-based eye clinic. He was referred by his school's social worker because of problems with his near vision that occurred after he broke his reading glasses. On questioning, he reported a loss of place when reading for a prolonged time. He also reported occasional itch to both eyes secondary to allergies to cats. He denied tearing, redness, burning, diplopia, headaches, nausea, dizziness, flashes and floaters.

The patient's medical history was positive for measles and chicken pox when younger. The family medical history was not significant. The patient was currently in sixth grade and had repeated second grade. His birth weight was 7.5 pounds and the delivery was uncomplicated. The patient's mother did not receive prenatal care during pregnancy and denied drug and alcohol use while pregnant.

Optometric evaluation

Uncorrected distance visual acuities were 20/30 (6/9) OD and OS. Uncorrected near visual acuities were 20/40 (6/12) OD and OS. Distance and near cover tests revealed orthophoria. The nearpoint of convergence was to the nose. Extraocular muscles were full initially, but on repeated testing, when the patient looked to the right, the left eye maintained fixation and the right eye went into an esotropia of approximately 45 prism diopters. Then, almost immediately the right eye released fully into abduction, while the left eye remained adducted, such that both eyes were correctly fixated. Both eyes were then able to make smooth versions to the left. The patient also reported pain during the time that the right eye released into abduction. Confrontations were full to finger counting OD and OS. Pupils were

equal, round, reactive, with no afferent pupillary defect. Randot stereopsis was 250 seconds. Color vision was normal, OD and OS, as measured with Ishihara plates.

Retinoscopy (non cycloplegic) revealed +0.25 DS OD, +0.50-0.75x130 OS with a variable reflex in each eye. Subjective refraction was +0.25 DS 20/20 (6/6) OD, plano 20/20 (6/6) OS. With a binocular balance addition of +0.25 DS OD, +0.50 DS OS the binocular acuity was 20/20 (6/6) OU. Near von Graefe testing indicated orthophoria. Near base-in ranges were 16/18/6. Near base-out ranges were 16/30/4. Negative relative accommodation (NRA) was +1.50. Positive relative accommodation (PRA) was –1.75.

Amplitude of accommodation, as measured with minus lenses was 5.50 diopters OD and OS. MEM retinoscopy over +0.50 DS OU revealed a +1.00 lag of accommodation in each eye.

The anterior segment examination was within normal limits. Finger tensions were soft and equal. As the parent did not consent to dilation, we could not perform a cycloplegic refraction or dilated fundus examination. Direct ophthalmoscopy revealed C/D ratios of 0.25 OD and OS with pink, distinct neural retinal rims. The vessels were of normal caliber and the maculae were flat with a foveal reflex present in both eyes.

We made the following diagnoses: accommodative insufficiency and anomalous eye movements. We also suspected malingering because of the discrepancy between the entering distance visual acuities and the refractive findings and resulting corrected distance acuities. We prescribed +0.75 DS OU for near work.

Because of the suspected malingering and anomalous eye movements, we contacted the patient's social worker to determine if there were any social or neurological problems. The social worker was not aware of any neurological conditions, but did report that the patient's father had a history of alcohol abuse and violence when drinking. The patient's father was receiving treatment, and the patient was receiving counseling. Because of the reported negative neurological history, the patient was referred to the neuro-ocular clinic at the State University of New York, State College of Optometry (SUNY) for an evaluation of his eye movements.

Neuro-ocular optometric evaluation

Evaluation at SUNY revealed an uncorrected visual acuity of 20/20 –3 (6/6) OD and OS, correctable to 20/20 (6/6) with +0.25 OD and +0.50 OS. Pupils were equal, round and reactive with no afferent pupillary defect. Extraocular muscles revealed a difficulty with and pain on abduction OD, during which time a pupillary constriction was noted. When retested in various manners, such as having the patient follow his reflection in a 20D lens, extraocular muscle excursions were full and no pain was reported. Cranial nerves II-VII were intact.

Anterior segment examination was within normal limits. Intraocular pressures, measured with Goldmann were 14 mm Hg OD and OS at 2:30pm. A dilated fundus examination revealed C/D ratios of 0.2 OU with pink, distinct, neural retinal rims. The maculae were flat OU with a foveal reflex in each eye. No holes, tears or retinal detachments were present in either eye.

The patient was diagnosed with spasm of the near reflex with a malingering component. The consulting optometrist felt that the patient was able to control his eye movements and voluntarily induce the spasm. The patient was told to use the glasses at near and to follow up with the school clinic and his social worker.

A follow-up examination in the school one month later revealed uncorrected distance VA of 20/20- (6/6) OD and OS. Corrected near acuities were 20/20 OD and OS. The patient reported that he was doing well with the glasses and used them for reading. Extraocular muscles were full initially when tested by the doctor. However, when an intern retested them, the abduction deficit reappeared and the patient reported pain. We advised the patient to continue to use the glasses for all academic work, including computer use, and to follow up in one year, or, earlier if problems developed. The patient also continues to meet with his social worker weekly. Vision therapy for the accommodative insufficiency was not a viable option because of logistical and other concerns.

DIFFERENTIALS

Among the differentials of abduction deficits are spasm of the near reflex, sixth nerve palsy, Duane's retraction syndrome, and myasthenia gravis.

Spasm of the Near Reflex

Spasm of the near reflex involves pupillary miosis, increased accommodation and increased convergence, all of which may vary in degree and duration.[1,2] During the spasm, either visual acuity or ocular motility can be affected.[3] Patients may be asymptomatic or complain of a variety of symptoms, including blurry vision or diplopia.[1-4] Cogan found that the most common symptom of these patients is blurry vision.[2] (Table 1)

Table 1. Symptoms of Patients with Spasm of the Near Reflex[2]

Blur 81%
Diplopia 62%
Headaches 56%
Eye Pain 38%
Dizziness 44%
Difficulty walking 38%

* Patients reported multiple symptoms

Onset of this condition can occur at any age, although one study found that the most common age of onset is between 15-20.[2] Women appear to be affected more than men; according to one study, 86% of those affected are female.[2]

In cases where the visual acuity is affected, the patient often presents with a dry myopic refraction that disappears with atropine. This "pseudomyopia" can be as high as 10 diopters.[2] Goldstein refers to a 1922 study of 30 patients with spasm of the near reflex, showing that the average amount of the spasm was 2.29 diopters.[1]

Unlike most cases of pseudomyopia, patients who have spasm of the near reflex will exhibit pupillary miosis.[5] The miosis may be intermittent or continuous, resulting in great variability of retinoscopy and refraction findings. Thus, if a spasm is suspected, the doctor should be attentive to pupil size and stability and perform retinoscopy.[5] Static retinoscopy has been reported to show an increase and variability of myopia, while dynamic retinoscopy shows a large lead of accommodation.[4]

In cases where vergence is affected, patients may present with a convergent strabismus or a unilateral or bilateral limitation of abduction.[1-4] They may progress from ortho or a low phoria to 50 degrees of esotropia during the spasm.[2]

Cogan reports that calling attention to the eyes, e.g., touching the lids, shining a bright light into the eyes, asking the pa-

tient to read the acuity chart or to look in a certain direction, can be the precipitating factor for the spasm.[2] He refers to two examples of the latter as a precipitating cause; one patient who exhibited an esotropia only when asked to look to the right, and the other who manifested an esotropia only when voluntarily looking upward.[2]

Most studies consider the etiology of this condition to be hysterical.[1-3,5] Patients who present with this condition have been described as "emotionally unstable" young adults.[3] They may suffer from anxiety, emotional distress or personality disorders.[4] However, there are reports of spasm of the near reflex being associated with neurological conditions including Arnold Chiari malformations, neurofibroma of the posterior fossa, pituitary adenoma and vestibulopathy.[5] It is important to note that in all of these cases, the neurological diagnosis was made prior to the discovery of the spasm, and it is unclear if there was a causality. Additionally, there are a number of cases in the literature of patients presenting with spasm of the near reflex after a head injury.[5,6]

There is little agreement on the appropriate treatment for these patients. Minus and plus lenses, nasal lens occlusion, cycloplegia with atropine or scopolamine, and patient reassurance have all been advocated.[1-4] The rationale for minus lenses is to partially correct the myopia induced by the spasm, whereas the rational for plus lenses is to help inhibit excess accommodation.[2,4] In some cases, treatment can involve patient adaptations, including head turn or tilt, or eye closure during the spasm.[2] There are reports of patients whose symptoms resolved without treatment.[1-3] Table 2 shows the treatments and percentage of relief in a study of 16 patients.

Table 2. Effective Treatment for Patients With Spasm of the Near Reflex[2]

Head Tilt 13%
Head Turn 13%
Atropine 44%
Minus Lenses 63%
No treatment/Spontaneous Resolution 25%

Sixth Nerve (Abducens) Palsy

A full discussion of sixth nerve palsies is beyond the scope of this paper, however, some important characteristics of this condition will be discussed. The importance of understanding sixth nerve palsies is heightened by the fact that an abducens nerve palsy is the most common cranial nerve palsy in pediatric patients.[7]

In cases of a sixth nerve palsy, the affected eye cannot abduct, whereas in a case of spasm of the near reflex, the eye can abduct normally. Therefore, it is important to repeat eye movement testing in patients with suspected abduction deficits to determine repeatability. Monocular duction testing is important because in cases of a sixth nerve palsy there will be restriction of abduction, while in cases of spasm of the near reflex ductions will be normal.[8]

A sixth nerve palsy can be either congenital or acquired. A sub classification of congenital palsies, congenital absence of abduction, is relatively rare and refers to a transient lateral rectus palsy secondary to damage to the abducens nerve during birth.[8-10] Its incidence increases as labor becomes increasing difficult, as illustrated by the Table 3.

Table 3. Incidence of Congenital Absence of Abduction With Method of Delivery[8]

C- Section	0%
Vaginal Delivery	0.1%
Forceps delivery	2.4%
Vacuum extraction	3.2%

Patients who present with congenital sixths are asymptomatic, whereas patients with acquired sixths are symptomatic for diplopia at distances greater than near, and may manifest a head turn away from the palsied muscle.[10] Acquired sixths are more serious, as their presence may indicate a neoplasm.[7-12]

There are discrepancies regarding the etiology of acquired abducens nerve palsies in children.[10-12] Variability can be explained by the setting in which the children were examined. For example, specialty hospitals presumably encounter more pathology than smaller community clinics. Holmes et al, who conducted a retrospective study of all patients from a certain geographic area diagnosed with sixth nerve palsies, found that the etiology of most sixth nerve palsies was undetermined.[11] On the other hand, Lee et al, who did a similar study on patients referred to a neuro-ophthalmologic practice in a children's hospital, found that most sixth nerve palsies were secondary to a neoplasm.[12] Table 4 illustrates these and other differences between the two studies:

In patients under the age of 3, the early stages of metastatic neuroblastoma can present as an isolated lateral rectus palsy.[10] Pontine gliomas, the most common brainstem glioma in early childhood or adolescence, can present with a unilateral or bilateral sixth nerve palsy at an average age of 6.5 years.[10] Often these tumors present with a seventh nerve palsy as well.[9] Additional symptoms may include headaches, nausea, vomiting and difficulties with gait.[9] Thirty five percent of patients with pontine gliomas will develop papilledema.[10] Therefore, dilated fundus examinations, with attention to the optic nerve and periphery are important in patients with a recent onset abduction deficit.

Other studies have shown a relationship between idiopathic intracranial hypertension (ICP) and a sixth nerve palsy in pre-pubertal pediatric patients.[13] According to one study, 60% of children under the age of 11 who were diagnosed with ICP also exhibited sixth nerve palsies.[13] Patient symptoms included headaches, diplopia, stiff neck, vomiting and malaise.

Table 4: Etiologies for CNVI Palsies in Pediatric Patients

Holmes et al (1999)[11]	Lee et al (1999)[12]
Undetermined 36%	Neoplasm 45%* Medulloblastoma 38% Brainstem glioma 29% Rhabdomyosarcoma 12%
Trauma 27%	Increased ICP 15%
Postviral 18%	Trauma 12%
S/P Tumor Resection 18%	Congenital 11% Duane's 75% Mobius 13%
Congenital 8%	Inflammation 7%
	Idiopathic 5%
	Miscellaneous 5% Aneurysm 25% Post circulation infarction 50% Cerebellar hemangioma 25%

*Includes patients whose palsy manifested pre and post surgery

On dilation, all patients showed optic nerve edema. Unlike adult patients who present with this condition, only 10% of pre-pubertal children with ICP are obese, and there does not appear to be a gender predilection.[13]

Other cases of acquired lateral rectus palsies have been reported following viruses, specifically varicella, and infections, such as meningitis and Lyme disease.[10-12] These palsies generally resolve within 2-4 months.[7-9] There is also a report of a bilateral sixth nerve palsy resulting from MDMA ("Ecstasy") abuse in a 17 year old.[14] Gradenigo's syndrome is the name given to a sixth nerve palsy with ipsilateral facial pain and earache.[7] This constellation of findings most commonly results from inflammation in the area of the petrous apex. However, tumors and aneurysms in this area can produce similar signs and must be ruled out.[7]

Up to 20% of sixth nerve palsies are bilateral.[7] Bilateral sixth nerve palsies may be confused with congenital esotropia and vice versa. If a child is suspected of having a bilateral sixth nerve palsy, some practitioners advocate examining for the "dolls eye response" by lifting the child and spinning him/her. If there are no ocular rotations, then a true paresis is present.[7]

It is also important to test for comitancy in these cases. Most childhood strabismus is comitant without a limitation of abduction.[9] Mekari-Sabbagh reports a case of a girl treated for bilateral esotropia for three years, until another practitioner noted non-comitancy and made the diagnosis of bilateral sixth nerve palsies. An MRI revealed a chordoma in the area of the pons. The child required two surgeries to remove the tumor, but still manifested an esotropia.[9]

If a patient presents with definite neurological signs, a neurological consult is in order. However, some practitioners monitor these patients every two to six weeks for improvement and refer them if the symptoms worsen or do not improve in six months.[7] However, Lee et al recommend neuroimaging immediately if papilledema or other neurological signs are present, and within one week in all other cases.[12]

In patients with chronic sixth nerve palsies, generally defined as lasting greater than six months, treatment options include observation, surgery or Botox injections.[15-16]

Duane's Retraction Syndrome

This condition has been classified as a type of congenital lateral rectus palsy.[10] In its most common form, it is characterized by a limitation of abduction, with a narrowing of the palpebral fissure and globe retraction on adduction.[7,8,17] The exact cause of this anomaly is not known. Studies have shown that Duane's arises from an agenesis (abnormal development) of the sixth nerve, which results in a miswiring of the third cranial nerve to the lateral rectus muscle.[7,8,17] Some have postulated that a teratogenic event in the second month of gestation is responsible for this condition because of its increase in Thalidomide births.[8]

In reality, three types of Duane's Exist:[8,17]

Type I 85% Limitation of Abduction, Globe retraction on attempted Adduction
Type II 14% Limitation of Adduction, Globe retraction on attempted Adduction
Type III 1% Limitation of Abduction and Adduction

Other findings in Duane's Type I can include a widening of the lids on attempted abduction, an up or downshoot of the eye on adduction, and a head turn towards the affected eye.[8,17] In Type I Duane's, the angle of the deviation does not frequently exceed 30 prism diopters.[17]

Duane's affects 0.1% of the general population, with 10% of patients having a positive family history.[8,17] In Type I, for unknown reasons, women are affected four times more frequently than men, and the left eye three times more frequently than the right.[8,17,18] Fifteen to 20% percent of cases are bilateral.[8,17,18] Vision in these cases is almost always normal.

Thirty to 50% of patients with Duane's have systemic associations, including Goldenhar's syndrome and Klippel-Fiel syndrome, the latter of which can occur with or without labyrinthine deafness.[8,17]

In general, patients with Duane's are asymptomatic.[18] If the patient presents with strabismus, there is often deep suppression and no diplopia.[18] In symptomatic patients complaints relate to poor binocular fusion and can include intermittent diplopia, fatigue and eyestrain with near tasks.[18] Vision therapy is an option for these patients. If there is a significant esotropia, surgery is an option.[18]

Myasthenia Gravis

As with other conditions characterized by intermittent signs and symptoms, myasthenia gravis should be included as a possibility when patients present with anomalous eye movements, possible nerve palsies, internuclear ophthalmoplegias or symptoms of intermittent diplopia or blur.[4,5]a

In children, myasthenia can be of three types: transient neonatal, congenital myasthenic syndromes and juvenile autoimmune myasthenia.[19] The neonatal form generally resolves quickly with no sequalae. Patients with congenital myasthenia can manifest symptoms, such as motor delays, failure to thrive and/or poor sucking ability, as early as infancy. If the signs are mild the child may not be diagnosed with myasthenia until 2 or older.[19] This form has a genetic component and is more common in girls than boys.[10]

The juvenile autoimmune (autoimmune) form of the disease manifests in older children, but onset is before age 17.[10] There can be overlap in the ages at which the congenital or juvenile forms appear.[19] Like the adult form, the juvenile form is grouped into ocular and systemic myasthenia.[19]

A study by Mullaney et al revealed that 21% of children with myasthenia exhibited the congenital form and 71% the juvenile form. The remaining 8% were diagnosed with transient neonatal myasthenia.[19]

In Mullaney's study, all of the patients with congenital myasthenia exhibited either ptosis, strabismus or ophthalmoplegia at some point during the course of the disease.[19] These signs were more prominent and stable than in patients with the juvenile form. Patients' symptoms and signs could fluctuate with medication dosage and compliance.

All patients with autoimmune mysathenia displayed a ptosis at some point during their disease, and most displayed a restriction in ocular motility when looking laterally.[19] Fifty six percent of the patients with juvenile myasthenia exhibited ocular signs only, most commonly a variable ptosis or strabismus.[19] Thirty six percent of these patients progressed from ocular to generalized myasthenia on the average of 7.8 months. Table 5 summarizes the ocular signs of the congenital and autoimmune myasthenias.

Table 5: Ocular Signs Associated with Myasthenia (in order of decreasing frequency)[19]	
Congenital Myasthenic Syndrome	**Autoimmune Myasthenia**
Ptosis	Ptosis
Strabismus	Incomitant Strabismus
Orbicularis Weakness	Horizontal Restriction of EOMs
Ophthalmoplegia	External Ophthalmoplegia
Slow Saccades	Comitant Strabismus

Although the definitive test for myasthenia is electron muscle biopsy of the intercostal muscles, revealing synaptic abnormalities, this test is not often performed if symptoms are mild.[19] Instead, the more common diagnostic tests are endrophonium chloride (Tensilon), or neuromuscular testing.[19] A positive family history of myasthenia may also help with the diagnosis of congenital myasthenia.

Because of different etiologic mechanisms, treatment is based upon the classification of myasthenia. The congenital form is not associated with autoimmune disease. Therefore, traditional immunosuppressive treatment is generally ineffective and may lead to a worsening of the disease.[19] In these cases, treatment is supportive. Children with the autoimmune form are treated with anticholinesterase medications, steroids or thymectomy, depending on disease severity.[19] In Mullaney's study, these patients entered remission, during which they were visually asymptomatic, after an average of two years of treatment.[19] According to one source, approximately, 30% of these patients undergo spontaneous remission by 15 years of age.[10]

Rosenberg describes two cases where adult patients presented with symptoms of intermittent diplopia and esotropia on lateral gaze.[20] Both patients showed improvement in their symptoms after injection of Tensilon and were initially diagnosed with ocular myasthenia. On subsequent testing, it was noted that both patients displayed miosis along with the esotropia. This miosis was initially missed and was important to support the revised diagnosis of spasm of the near reflex.

Conclusion

The evaluation of patients with abduction deficits or eye movement anomalies can prove to be difficult, and at times, challenging. Among the differentials are sixth nerve palsies, Duane's retraction syndrome, spasm of the near reflex, and myasthenia gravis. To aid in arriving at a diagnosis, the case history should focus on symptoms of diplopia and possible neurological signs such as nausea, vomiting, headaches, hemiparesis, and facial nerve palsies. In children, a developmental history should include questions about complications with birth and developmental milestones. Additional questions relating to recent colds or infections, as well as possible contact with deer ticks, can help narrow the differentials.

During the exam, the patient's behavior should also be observed, with particular attention to pupils, accommodation, vergence, and the presence or absence of neurological signs such as ataxia or nystagmus. In addition to the history and externals, a careful dilated fundus examination is important to rule out papilledema. In cases where a diagnosis is elusive or the findings are highly suggestive of a neurological problem, the appropriate neurological referrals must be made.

In the case reported here, the ocular health was normal, and there were no neurological signs or symptoms. The ability of the patient to demonstrate normal versions and ductions, and the presence of pupillary miosis, were important findings to support the diagnosis of spasm of the near reflex. Additionally, the fact that the patient was receiving counseling for his family situation, and might have been suffering from emotional distress or anxiety, is consistent with the profile of patients with this disorder. This case also supports Cogan's view[2] that attention to the eyes is a precipitating factor for the spasm; during this patient's follow up examination, he demonstrated the spasm only when an intern retested his extraocular muscles.

The Appendix (page 148) is presented as a guide to the parameters that must be considered in the differential diagnosis of abduction defects.

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Appendix
A Summary for the Differential Diagnosis of Abduction Defects

Disorder	Symptoms	Signs	History	Testing	Etiology	Management
Spasm of near reflex	Blur Diplopia Eye strain Headaches	Increase in myopia EOM abnormality Pupil constriction	Psychological problems possible	Repeat versions/ ductions Cycloplegic refraction Pupil size during retinoscopy	Psychogenic	Variable: plus or minus lenses Psychological reassurance
Sixth nerve palsy - Congenital	None	ET may be present	Forceps birth Family history	Dilation	Birth trauma Genetic	Monitor Surgery for ET cosmesis
Sixth nerve palsy – Acquired	Diplopia @ distance	Head Turn Headaches Vomiting Papilledema Possible contra hemiplegia Ipsilateral facial palsy	Recent trauma Neurological signs (nausea, vomiting, hemiplegia) Earache Colds Flu Contact with ticks	Dilation pupils depending on signs/symptoms: CBC, Lyme Titer, VDRL, FTA-ABS, MRI, Spinal Tap	Neoplasm Increased ICP Trauma Inflammation Idiopathic	STAT referral if papilledema present Neuro consult indicated if neuro signs May follow patient monthly May consider surgery or Botox if chronic
Duane's	Often asympto-matic Possible intermittent diplopia/ near symptoms	Globe retraction on abduction Narrow palpebral fissure on abduction	Family history	Binocular testing to check for suppression	Agenesis of CN VI	Monitor VT or surgery if there are symptoms
Myasthenia	Intermittent blur Diplopia	Anomalous EOMs Nerve palsy INO Ptosis	Developmental history, motor delays, failure to thrive	Repeat testing of EOMs, Tensilon, neuromuscular testing	Autoimmune	Referral to neurologist for tx Anticholinesterase medications Surgery

Corresponding author:
Marie I. Bodack, O.D.
Assistant Clinical Professor
SUNY State College of Optometry
33 West 42nd Street
New York, New York 10036
Date accepted for publication:
September 20, 2002