A Benign Syndrome of Transient Loss of Accommodation in Young Patients

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Objective: To describe a benign condition of transient, isolated accommodation paralysis in young patients as a specific entity.

Design: Case series of children and young adults with transient loss of accommodation who were referred to the neuro-ophthalmology clinic at the Meir Medical Center from 1997 to 2006. Five young patients who complained of an inability to read had full neuro-ophthalmological examinations. Those who were found to have isolated accommodation paralysis without any other related ocular or systemic findings were prescribed reading glasses and followed up.

Results: All 5 patients had isolated loss of accommodation. No one had other ocular, neurological, or systemic abnormalities that could be associated with accommodation paralysis; they all did well with near correction. Accommodation returned to normal within 3 to 14 months in all 5 patients.

Conclusion: An isolated transient loss of accommodation unrelated to any other ocular or systemic manifestations may occur in children and young adults and may be considered a specific idiopathic entity.

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Loss of accommodation is the most frequent significant disorder that affects the eye. In most events, it may be considered to be part of the normal aging process rather than a disease. Accommodation capacity decreases gradually beginning at early adulthood and worsens to the level of absolute failure at about the age of 50 years in a process known as presbyopia.1,2

When a sudden loss of accommodation occurs in a child or young adult, generally, a search for a specific pathologic cause is conducted. There are numerous possible etiologies, including systemic, neurological, and local ocular diseases. Some recognized causes that may affect accommodation include head trauma,3 encephalitis and meningitis,4 midbrain disease,5 oculomotor nerve palsy, tonic pupil,6 pharmacological and toxic agents,7 ocular and orbital trauma,8 uveitis,9 cataract,10 lens subluxation, laser or cold applications to the retina or sclera,11 viral diseases,12,13 diabetes,14 botulism,15 diphtheria,16 and functional causes.17,18 With some of these, the relationship is obviously causative and the mechanism of accommodative loss is clear, eg, injury to the short ciliary nerves. In other instances, the exact mechanism of accommodative failure is unknown and only a temporal association is noted, eg, nonspecific viral syndrome. This list of possible causes is extensive, but generally a comprehensive history-taking and thorough ophthalmological and neuro-ophthalmological examination will be sufficient to disclose the origin of accommodation paralysis.

Occasionally, a child or young adult may have an abrupt loss of his or her near vision owing to complete accommodation paralysis without any apparent systemic, neurological, or ocular causes.19-22 A case series of 5 such young patients with an isolated, sudden, complete, and transient loss of accommodation is reported in this study to describe a specific benign (though idiopathic) entity.

Methods

Presented herein are 5 young patients who were referred for a neuro-ophthalmological consultation to exclude neurological causes of their rapid loss of accommodation. Four patients were referred by community ophthalmologists and 1 was referred from an orthoptic clinic. I examined all of the patients in a consulting neuro-ophthalmology clinic from 1997 to 2006 and followed up the patients for 4 to 15 months until they recovered and regained their normal accommodation capacity. Age at initial examination ranged from 8 to 21 years.

Detailed general, medical, and ophthalmological histories were taken in all patients or from their parents, with particular emphasis on any neurological disease or symptoms, chronic

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systemic diseases, and a febrile disease or other acute onset of disease at the time of examination of the near vision loss. Patients were asked about the use of local ocular or systemic medications and about any possible contact with chemicals as well as any history of ocular or head trauma.

Detailed ophthalmic histories and thorough ophthalmological examinations were conducted. Specifically, each patient was examined for visual acuity for distance and near, subjective refraction, and objective cycloplegic retinoscopy. Accommodation capacity was evaluated by measuring near point of accommodation as well as by assessing the patient's ability to accommodate through negative sphere lenses on a distant target. Eye movements and alignment were evaluated; the evaluation included assessment of near point of convergence and measurement of convergence amplitude for near and distance with base-out prism insertion. Alignment was measured with the alternate cover test at 4 m and ½ m. Pupils were checked for size, equality, and reaction to light and near target. A dilated pilocarpine, 0.125%, test was conducted and irises were inspected with a slitlamp for vermiform movements to rule out Adie tonic pupil. Lenses were inspected for any sign of cataract, phacodonesis, or subluxation, and eyes were examined for anything that would suggest ocular trauma. Any sign that suggested active or past uveitis or pars planitis was recorded. Four patients underwent magnetic resonance imaging (MRI) of the brain and orbits and 3 patients were examined by a pediatric neurologist.

**REPORT OF CASES**

**CASE 1**

A generally healthy 10-year-old boy was referred for a neuro-ophthalmological consultation owing to difficulty with near vision. For a few days, he could read by increasing the distance of his reading material, but later he could not read at all. He had no other general or ocular complaints. His parents did not reveal any recent acute febrile or other disease, head or eye trauma, or the use of medication or eye drops.

The ocular examination revealed a visual acuity of 20/20 in both eyes for distance with no correction. The patient had near vision of J16 at 33 cm and J5 at 1 m. With a +2-diopter (D) sphere lens, he could read J1 at 33 cm. With a −1–D sphere lens, his distance vision was blurred. Cycloplegic retinoscopy results were +0.75 D in both eyes. Ocular movements were full and he was orthophoric. His convergence was preserved; he was able to overcome 35 prism D (Δ) of base-out prism on a near target. Pupils were round, equal, and reactive to light and a near target. Results of a pilocarpine, 0.125%, test for tonic pupil were negative. Anterior segments revealed normal-appearing irises and lenses with no phacodonesis, subluxation, or cataract. Fundus examination results were normal bilaterally. Brain MRI results were normal.

The diagnosis was accommodation paralysis from unknown etiology. The patient was prescribed +2.5–D reading glasses. He was examined every 3 months and was doing well with the glasses. Seven months after his reading difficulties started, he realized that he did not need his reading glasses anymore. Two months later, he could read at J1+ with no difficulty and his near point of accommodation was 10 cm (corresponding to 10 D of accommodation).

**CASE 2**

An 11½-year-old girl arrived at her ophthalmologist’s office complaining of difficulty with reading and seeing from near for the last 4 months. When her symptoms began, she had a febrile illness, which was not specifically diagnosed and resolved within 1 day. She was not known to have any general medical problems or any other ocular problems. When she was first seen in the neuro-ophthalmology clinic, she was already using multifocal glasses prescribed to her by the ophthalmologists who referred her. She also underwent a brain MRI, the results of which were interpreted as normal. She did not use any medications or eye drops and denied head or ocular trauma.

Her first neuro-ophthalmological examination revealed uncorrected distance visual acuity of 20/40 in both eyes, with +1 D sphere she could see 20/20 bilaterally. She could read J16 at 33 cm and, with the addition of +3–D sphere lenses, J1+ at 33 cm. Cycloplegic retinoscopy results were +1 D in both eyes. She had full-range eye movements, including good convergence, and she was orthophoric on the alternate cover test. Her pupils were round and equal with brisk reaction to light and near target. Pilocarpine, 0.125%, test results were negative for tonic pupil. Anterior segments and fundus examination results were normal.

The patient was examined every 3 months for more than a year with no change in her status. Fourteen months after her symptoms began, she stopped using her multifocal glasses. Her eye examination at that time revealed an uncorrected visual acuity of 20/20 for distance and J1+ at 33 cm. She was able to accommodate to a distance of 12 cm in her right eye and 13 cm in her left.

**CASE 3**

An 8-year-old girl was referred for a neuro-ophthalmological consultation. For 4 months, she complained about being unable to read. Since the age of 4 years, she was known to have accommodative esotropia and she used +4–D sphere glasses, which fully corrected her hypermetropia. Her mother denied any general medical problems or a history of head or ocular trauma; the patient did not use any medications or eye drops.

The girl’s pediatric ophthalmologist detected very low accommodative amplitude and prescribed her bifocal glasses (+4 D for distance and +7 D for near). She was also seen by a pediatric neurologist, who did not find any neurological symptoms other than her accommodation paralysis. He ordered a brain MRI and its results were interpreted as normal.

Her first examination in the neuro-ophthalmology clinic revealed a distance visual acuity, with +4–D sphere correction, of 20/25 in both eyes. Without her glasses, she could not see the 20/200 line of the Snellen chart. Near vision with full hypermetropic correction was J10 at 33 cm. With the addition of +3 D sphere over her +4–D sphere glasses, she could read J1 from 33 cm. The addition of −1.5 D sphere to her +4–D sphere distance glasses entirely blurred her distance vision. Cycloplegic retinoscopy results were +4 D in both eyes.

She had full-range eye movements but moderate convergence amplitude (25Δ for a near target). She was or-
throphoric with her hypermetropic correction. Pupils were round and equal, with brisk reaction to light and moderate reaction to a near target. Anterior segments were normal. Fundus examination results were also normal.

The patient was followed for an additional 4 months with no change in her status. On her last examination, 8 months after the beginning of her symptoms, her uncorrected visual acuity was 20/20 for distance and J1+ at 33 cm. Taking into consideration her +4-D hypermetropia, this means she could generate at least 7 D of accommodation.

CASE 4

During the 2 months before examination, a 21-year-old woman could not read and had difficulty with near vision. She denied any recent or chronic general disease and past ocular problems. On examination, her visual acuity was 20/20 with +0.75–D sphere correction. Near vision was J14 at 33 cm and J3 at 1 m. With +2.5–D near correction, she could read J1+ at 33 cm. Convergence was well preserved. Dynamic retinoscopy was performed while the patient was converging to fixate on a target held at a distance of 33 cm, but no myopic shift was observed. Her cycloplegic refraction was +0.5 D sphere bilaterally. Pupils were round, equal, and reacted briskly to light and near. Pilocarpine, 0.125%, test results were negative.

The patient was prescribed +0.75–D distance correction and +3–D near correction. On examination 6 months later, she had an uncorrected visual acuity of 20/15 for distance and J1+ for near at 33 cm. She could accommodate and overcome a −7-D lens for distance, and her near point of accommodation was 10 cm.

CASE 5

Case 5 was an 18-year-old woman with no general medical problems or history of eye disease. During the last week, she noticed that she had to hold reading material far from her eyes. She was using +2–D sphere reading glasses prescribed to her by an optometrist. On examination, her visual acuity was 20/20 for distance, but she could not overcome more than −2–D sphere lenses for a distant target. She could, with much effort, read J2 at 33 cm, but her near point of accommodation was 40 cm (which corresponds to 2.5 D of accommodation). Results of dynamic retinoscopy while the patient was fixating on a near target was −1 D in both eyes. Cycloplegic retinoscopy revealed emetropia in both eyes. She had full-range eye movements and her convergence amplitude was 20Δ for distance and 35Δ for a near target. Her pupils were not dilated, reacted normally to light and near, and did not constrict after application of pilocarpine, 0.125%, drops. Anterior segments and fundi were normal. Brain MRI results were normal.

She was prescribed +2–D sphere reading glasses. She did not show up for her follow-up examination, but in telephone questioning 5 months later, she reported that she did not have any more reading difficulties and did not need her reading glasses any more.

I have described 5 young patients who had complete and rapid loss of their accommodation. They were identical in their symptoms, insignificant systemic and ocular medical history, onset and evolution of disease, and ophthalmological examination findings. Hence, it would be reasonable to consider this condition a specific medical entity.

The importance of describing such a set of events as a specific entity is that diagnosing it as a benign, particular, and recognized entity will prevent the need for exhausting and expensive investigations in the future and will make it possible to treat those patients properly with glasses for near correction and to reassure them about the benign and transient course of their disease.

Some features that can lead to the diagnosis of this entity include (1) the patient being a child or young adult, (2) complete or nearly complete bilateral symmetric paralysis of accommodation, (3) a relatively sudden onset, (4) preserved convergence, (5) equal and normal reacting pupils, (6) negative pilocarpine, 0.125%, test results, (7) normal ocular examination results, except for accommodation paralysis, (8) no systemic or neurological disease, (9) relief of patient’s difficulty with near glasses correction, and (10) recovery of normal accommodation within a few months. Theoretically, the cause of loss of accommodation should be looked for at the different levels of the system that control and perform this task, namely, the crystalline lens, ciliary body, short posterior ciliary nerves, ciliary ganglion, oculomotor nerve, third nerve nucleus, and the supranuclear control of the near reflex. In fact, the literature is abundant with reports in which loss of accommodation is related to diseases that affect the accommodation system at its different levels, but few references about cases of isolated, sudden, bilateral, severe, and transient loss of accommodation that seems to be unrelated to any other ocular, neurological, or systemic diseases can be found; in these cases, however, no attempt was made to describe them as specific entities. Von Noorden et al described patients who had associated accommodation and convergence insufficiency; they concluded that it was distinct and separate from the ordinary convergence insufficiency.

In the ophthalmological and optometric literature, some relatively large series of young patients with low amplitude of accommodation relative to their age can be found. This type of accommodative weakness is chronic and partial and does not seem to apply to the 5 patients presented herein; the entity I described is much more remarkable as an episode, with abrupt onset, total loss of accommodation, and complete and sudden healing.

No cause could be found for the loss of accommodation in the presented case series. If the source were supranuclear, one would expect that the other 2 components of the near reflex, namely convergence and miosis, would also be involved. As there was normal convergence amplitude and brisk pupillary constriction to a near stimulus in all 5 patients, it is not reasonable to assume that the cause of the loss of accommodation was at the supranuclear level.

The possibility that loss of accommodation in these patients was infranuclear is also very unlikely, as it is not usual for any recognized disease to simultaneously affect...
either the 2 oculomotor nerves or the ciliary nerves to the same extent and to fully preserve all the other functions of the nerves (eye movements, eyelid elevation, and papillary constriction). Therefore, it seems that the loss of accommodation does not originate at the infra-nuclear level either. The possibility that the source of accommodation loss was the result of a disease that directly affected the ciliary body or the lens was not supported by the ocular examination results, which did not reveal any disease of the end organs.

Another possibility that should be considered is the Adie tonic pupil syndrome. This syndrome is generally idiopathic, yet it is accepted as a specific separate entity. This syndrome is mainly regarded as pupillary dysfunction but can affect accommodation as well. Bell and Thompson⁶ found that most patients with Adie syndrome had a moderate accommodative paresis and that there was a strong tendency for the ciliary muscle to recover with time; moreover, 2 of their patients were described as having pupil-sparing Adie syndrome in 1 eye. Could the 5 patients described herein represent a variant of Adie syndrome that affects the ciliary body bilaterally without involving the pupils?

Some authors have proposed that loss of accommodation is a functional manifestation of a psychogenic disease.¹⁹,²⁰ This may be an appropriate explanation of the complete symmetry of the disease between 2 eyes in this case series, though psychogenic problems were not encountered in these 5 patients. Malingering, as 1 variety of a functional visual failure, does not appear to be the preferential alternative to be considered here; unquestionably, the patients (most of them children) were not expected to know and be able to imitate so perfectly the rules of optics as revealed by their examination (ie, improvement of their visual acuity when reading distance was increased, failure to overcome a negative sphere lens for distant vision, and normal near vision with the near correction). Functional accommodation loss that results from the lack of accommodative effort could be considered here; yet, an effective near attempt was shown in all 5 patients by the normal convergence amplitudes that were measured. In 2 patients, dynamic retinoscopy was performed while the patients were effectively converging on a near target; yet, no myopic shift was observed; this means that there was an accommodative near effort that could yield convergence but not accommodation.

The issue of whether an MRI scan is required for a patient with accommodation loss should be considered. It is reasonable to search for the cause of accommodation loss at the upper midbrain where the center of the near reflex and third nerves nuclei are located. However, if a patient fits the clinical criteria as described in this study, an MRI scan may be unrewarding. It is unreasonable to propose that a brainstem lesion would cause an isolated total and bilateral accommodation loss, as there is no anatomic place that can explain such an event. In summary, though there is no apparent etiology to suitably explain the loss of accommodation described herein, it seems that the incidents are distinct enough to recognize them as a specific medical entity.

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REFERENCES