

SUDDEN ONSET DIPLOPIA

■ Eric S. Hussey, O.D.

Abstract

Sudden onset diplopia can be the result of underlying systemic or neurological conditions. I present the case of a 32-year-old woman with a previously diagnosed convergence insufficiency, who developed sudden diplopia. Physicians ruled out systemic and neurological conditions, but were unable to resolve the diplopia. Functional optometric examination with vectographic targets showed Intermittent Central Suppression (ICS). I hypothesized that the ICS served to momentarily negate the sensory lock required for binocular fusion in a patient with fragile convergence ability. Correcting the ICS using electronic rapid alternate occlusion on a home therapy basis improved fusional convergence, and the diplopia was significantly lessened. There was no standard direct therapy for the convergence insufficiency. The alternate flicker of rapid alternate occlusion is a strong motion stimulus. Since a strong motion stimulus in isolation eliminated the suppression, this supports the theory that magnocellular defect is responsible for ICS.

Key Words

convergence insufficiency, diplopia, electronic rapid alternate occlusion, fusional convergence, intermittent central suppression, magnocellular pathway, vectograms

INTRODUCTION

Sudden or abrupt onset diplopia can be the harbinger of an underlying serious systemic or neurological condition. Rosner suggests a three-step process to consider the etiology of the diplopia.¹ First, age of the patient: “onset...past the age of four should be viewed with careful suspicion.” Second, the abruptness of the onset: abrupt onset is more likely to be pathological. Third, accompanying symptoms: other signs pointing to CNS involvement are worrisome. This process is proposed as a clinical triage, but does not necessarily preclude optometric testing. If this process does not rule out neurological or systemic causes, Rosner proposes that an immediate appropriate medical referral should be made.¹

The following case demonstrates an abruptly acquired diplopia in an adult that was appropriately investigated for systemic and neurological causes, but responded to treatment as a functional vision problem. Since there was no reported precipitating trauma, it may be more accurate to refer to this condition as sudden *reported* diplopia. This does not preclude the possibility of the condition's gradual development, but still is descriptive of the apparent sudden onset.

HISTORY

Patient LH is a 32-year-old mother of two who works at a local hospital as a recreational therapist. I had provided routine optometric evaluations for her in the 1980s and early 1990s. At those times, I diagnosed myopia and convergence insufficiency. She had no explicit reading complaints and was not interested in therapy to address the convergence insufficiency. I fit LH with soft contact lenses for her myopia.

LH became a member of a managed care Health Maintenance Organization (HMO) of which I am not a provider. Their staff assumed her care. She continued with routine eye examinations there in 1995, 1997 and April, 2000. The resulting records indicate she was still myopic, ocular health was normal, there was no afferent pupil defect, and “muscle balance ortho distance and near with her lens correction.” The only notable medical occurrences through this period were the birth of two children and hypothyroidism treated with Synthroid.

LH presented to the HMO optometric clinic in November of 2000 for examination for sudden onset diplopia of some two months duration. She complained of lateral diplopia with some intermittency, and stated that the diplopic range was from 2 to 10 feet in front of her. When she looked from arm's length to distance, she felt some lag in her convergence but that her vision “slips into place” when she viewed at a greater distance.

The examining optometrist could not elicit diplopia at this examination. Cover testing indicated 3 of exophoria at distance, and “low” exophoria at near. Oculomotor testing in the five cardinal positions revealed “ortho vertically and horizontally.” Neither trials of base-in or base-out prisms ameliorated the diplopia. Static computerized visual fields were normal in all aspects. The examining optometrist noted there was no accompanying arm or leg weakness, and no increase in stress or lack of sleep. Visual acuities remained 20/20 OD, OS with her current soft contact lenses, prescribed from the routine examination of the prior April (subjective: OD-1.25-0.25x179, OS-2.25 sphere).

At the follow-up examination in December 2000, the same optometrist noted

LH now complained of daily headaches. One to 2 of base-in prism at near made things “better;” however, prism was not prescribed. Eye health was still normal. Color vision was normal. The optometrist questioned whether this was a decompensating exophoria. The management plan was to refer her to her primary care physician (PCP) with the request for a complete physical. If there were no apparent causes for the diplopia, “muscle system vision therapy” would be considered.

LH was examined by her PCP in early January, 2001. Her major complaint was diplopia, “pretty much all the time.” At this examination, she described the diplopia as occurring over a time span of 18 months to two years, somewhat at odds with her previous estimate of onset. The PCP noted the diplopia was not associated with headaches (despite the prior complaint of daily headaches), numbness, field loss, weakness, tingling or any seizure disorder. Cranial nerves were intact, although the PCP observed a “visual tracking problem.” A normal thyroid stimulating hormone (TSH) study and the absence of exophthalmos indicated her present medication was appropriately compensating for her hypothyroidism.

The PCP as a first step (similar to the advice of Rosner, above) moved to “rule out a central lesion” with an MRI. The plan was to make a neurological consultation if the MRI was negative. A Gadolinium-enhanced brain MRI scan with T1 and T2 sequences in sagittal, axial, and coronal planes was performed. The radiology report concluded that there was a negative scan, but noted a “small, non-specific” finding.

A neurological referral was made. His report described intermittent and variable horizontal diplopia somewhat associated with fatigue. There were no signs of muscle weakness, chewing problems, ptosis, cranial dysfunction, paresthesias, scotoma, monocular visual impairment, hearing loss, vertigo or ataxia. Eye movements were “conjugate and full.” He also reviewed the MRI. The neurologist concluded that “there is no identifiable CNS lesion, and the small non-specific finding could not explain LH’s symptoms.” Since the history was suggestive of a neuromuscular junction disorder such as myasthenia gravis, he ordered testing for myasthenia gravis with single fiber electromyography. That testing was done

and the findings were negative, thus ruling out myasthenia gravis or other neuromuscular junction disorders.

The HMO optometrist performed a routine optometric examination one week after the neurology examination. LH claimed horizontal diplopia “all the time,” although diplopia again was not elicited during the examination. It was noted that the images overlapped, but there were no other changes. Refraction was similar to prior exams at OD-1.50-0.25x03, OS-2.50 sph; 20/20 OD, OS. This last HMO visit was February 6, 2002.

FUNCTIONAL OPTOMETRIC MANAGEMENT Assessment

LH’s father had followed the testing and had discussed some of the findings with me. I, as an independent optometrist, do not have provider status in LH’s HMO. However, since the HMO offered no further treatment for the diplopia we discussed starting with a functional optometric examination and then, if indicated, a vision therapy program for LH

LH presented to my office in May 2002. Her complaints were blurred vision, double vision, tired eyes and dry eyes. She denied, and PCP records showed, no change in medications. The ocular health examination was within normal limits, both internally and externally. Entrance acuities were 20/20 OD, OS with her current spectacles. Refraction had changed minimally, now OD-1.75 sph (20/20), OS-2.75 sph (20/20). Cover test at this time was orthophoria at distance and near. With prism dissociation the distant phoria was orthophoria, but at near it was 15 of exophoria. I conducted vectographic examinations, which I do as a routine test, and had done in 1993. This method has been previously documented.² A key component involves using vectographic testing at distance and near at various times during the examination as determinants of intermittent central suppression (ICS).

During the present examination I asked that she maintain a “normal amount” of effort to maintain single vision. When she relaxed this amount of effort, diplopia appeared. She was diplopic about 50% of the time during the course of this examination. I hypothesized that her level of effort could explain the absence of diplopia during prior examinations; she

might have been able to maintain the appropriate level over a short period of time. However, if the effort level were automatic and easily sustainable, diplopia would not have been the chief complaint.

LH’s near convergence range had deteriorated since 1993. Base-out vergences at near were now 12/-6 in 1993, but were -6/-8, fatiguing to -10/-12 on repetition in 2002. At distance, the associated phoria (fixation disparity cross target)² was 2 base-in, having somewhat increased from a variable ortho to 1 base-in during my prior exams. Positive relative accommodation was virtually identical to the 1993 finding, suggesting that an accommodative dysfunction was not a contributing factor to the diplopia. Such a dysfunction could lessen the amount of accommodative convergence that she might have relied upon to overcome the convergence insufficiency. No neurological or neuromuscular etiologies had been found to explain the deteriorating—or decompensating—convergence insufficiency. Therefore, I continued with the vectographic ICS testing to explore whether the problem might be one of sensory or fusion control rather than a strictly convergence “muscle” problem.

ICS was not immediately evident during the vectographic testing. However, as the testing proceeded, I found short, infrequent central losses that degenerated to more typical alternate ICS losses on later targets.³ I felt that the increase in ICS over time could be related to LH’s report that the diplopia was associated with fatigue. I concluded that fatigue was the central factor in both the increasing ICS and the diplopia.

I had LH complete a COVD Quality of Life Outcomes Assessment to use as an accepted yardstick to evaluate the subjective effects of therapy.⁴ Her score was 13. Frequent symptoms (score of three each) included double vision and motion sickness. Occasional symptoms (score of two each) were headaches with near work and vision worse at the end of the day. Inability to estimate distance accurately; misplaces or loses papers, objects or belongings; and forgetful, poor memory were seldom occurring symptoms (score of one each). All other items on the symptom list were ranked as zeroes. My primary diagnosis was ICS, and the secondary was convergence insufficiency.

Patient education and treatment rationale

LH and I discussed the findings of the examination. My theory to explain the abrupt appearance of the diplopia was that the ICS worsened over time, probably because of fatigue. The ICS removed the central fixation sensory lock in her already fragile convergence ability. Without a strong sensory lock, the compromised convergence system lapsed into a strabismus. Thus, I propose that the suppression here triggers the diplopia; this is opposite to usual thinking about the two entities, where suppression eliminates the diplopia.

Bielschowsky discussed a model of convergence in convergence insufficiency.⁵ He described voluntary (i.e., volitional) convergence and fusional (i.e., automatic) convergence. Although not explicitly stated, my interpretation of his discussion is that fusional convergence is mediated by intact visual sensation, whereas the voluntary component requires conscious attention to the act of converging. In the Bielschowsky model of convergence, the defect in LH's convergence would be in the fusional or automatic convergence system and, therefore, in the sensory system that triggers fusional convergence. The fact that LH didn't experience diplopia during several examinations speaks to both her availability of volitional convergence, and perhaps also to the fatigue-induced ICS as the trigger for the diplopia. Fusional convergence was apparently close to adequate until the ICS increased. Then volitional convergence could only selectively compensate—such as during an eye examination of relatively short duration. LH's volitional convergence required both more attention than her fusional vergence and could also apparently be easily fatigued.

I proposed to strengthen the central sensory fusional lock for the more automatic fusional convergence by minimizing the ICS. Then, LH should not have to use volitional control to maintain single vision. Reasoning from this hypothesis, I proposed to treat only the ICS directly, as a means to indirectly affect the convergence insufficiency. If we could eliminate the ICS, improve the fusion lock, and thereby eliminate the diplopia, this would support my fusional convergence theory. Success, in turn, would suggest this was a control

problem of a sensory defect rather than a "muscle" problem that would be a product of a decompensating convergence insufficiency.

One possible risk in this approach was evident: It could be reasoned that the convergence insufficiency was the primary problem, which triggered or increased the ICS. If this second theory was accurate, elimination of the ICS without first treating the convergence insufficiency would result in an unsuccessful outcome: the convergence insufficiency would continually reproduce any ICS we eliminated with anti-suppression therapy.

One of the practical advantages to my ICS-producing-the-diplopia theory is that we could use electronic rapid alternate occlusion on a home based therapy mode to treat the ICS.^{6,7} I informed LH that poor outcomes were possible, including no improvement, seizures during treatment and intractable diplopia. After a thorough discussion, LH gave verbal consent, opting to proceed with treatment, essentially as a single clinical trial.

Vision Therapy

This phase of management began during June, 2002. The primary goal was to eliminate the ICS with electronic rapid alternate occlusion. The alternate occlusion unit consists of liquid crystal goggles as are used in virtual reality computer programs. A programmable power unit can be set with both the alternation frequency and use time (Figure 1). We set the alternation frequency using our previously described standard technique of evaluating the ICS "real-time" during cheirosopic tracings.⁸ LH performed these tracings while giving ongoing verbal feedback on her suppressions. The alternation frequency of the liquid crystal goggle was varied until the suppressions were minimized. That alternation frequency was then set in the memory of the control unit for beginning home therapy. The initial alternation frequency was 5 Hz to be used for 45 minutes of reading, three days per week, preferably on alternate days. We conducted a phone interview every one to three weeks, to assess progress and side effects. Then, depending on availability and progress, I proposed in-office visual evaluations every four to six weeks.

Since this was an at-home trial, I had LH keep her own records, self-monitoring and self-reporting the times in minutes



Figure 1. The prototype rapid alternate occlusion goggles (top picture) with control unit (bottom picture) used to set alternation rates.

and frequency of therapy. She reported using the device from two to five times weekly. Because LH's record keeping was not meticulous, I estimated that 30 to 50 sessions of 45 minutes were performed; thus, there were 25 to 35 hours of home VT between June 2, 2002, and March 5, 2003.

Between December 2002 and March 2003, I instructed LH to use a decreasing schedule of therapy in order to gradually taper off the treatment. At office evaluations during July, August, and December 2002, I determined the optimal anti-suppression frequency in the same fashion as described above. These frequencies decreased from the initial 5 Hz to 4.6 Hz. Additionally, phone interviews were made twice in each month from August to November 2002 to monitor progress and side effects from the therapy. No significant side effects were reported at any time during the course of treatment.

The ICS improved along a variable path. At about the two-month mark, some alternation persisted during distance vectographic testing, and LH reported the diplopia had lessened slightly better than 50%. A month later she reported that the diplopia had lessened by 60 to 65%. In different phone interviews she stated there was some variability in her improvement and no side effects. In December 2002 we could find ICS only sporadically on the cheirosopic drawings used to set the alternation frequency. I estimated the ICS

as 95% improved; LH reported diplopia five to seven times daily that “corrects with a blink,” and that about twice weekly she had some difficulty regaining single vision.

An office progress evaluation was done during March, 2003. LH reported an episode of troublesome diplopia that lasted three days since the previous office visit in December, 2002; she felt that during this time she was overly fatigued. Otherwise, she had, at most, one noticeable diplopia event about every two weeks. No ICS was found in the examination, and with this kind of improvement in the diplopia, I estimated this as a 99% functional cure. We discontinued treatment at this point. I recommended an office visit during the following July, but LH was unable to keep this appointment. However, during May, 2003 she completed her post-therapy COVD Quality of Life assessment. Her total score had decreased from the pre-therapy 13 to a score of 3.5. The only symptoms present were double vision, rated between 1 and 2 (between “seldom” and “occasionally”), and occasional carsickness rated 2.

Discussion

It is important to reiterate that all practitioners at the HMO provided care within generally accepted medical guidelines; they acted and tested appropriately within the standard of care. However, after all precautions were taken, the patient’s chief complaint was not resolved. There was no offer for therapy to ameliorate the diplopia. As Sherlock Holmes said to Dr. Watson in *The Hound of the Baskervilles*, “I am more to blame than you, Watson. In order to make my case well rounded and complete, I have thrown away the life of my client.”⁹ Although LH’s condition was not life threatening, it certainly was negatively impacting her quality of life.

My assumption was that the ICS was the primary problem. The working theory I used for treatment was to improve the sensory foundation for fusional convergence by eliminating the ICS. The ICS momentarily removed the fusion lock in a patient with fragile convergence ability. But, the suppression in ICS is not as complete or constant as in strabismus, so that the momentary loss of fusion served to further exacerbate LH’s already flawed convergence ability. Since we were limited by the HMO’s unwillingness to pro-

vide support for therapy, and L.H.’s financial situation, one question was whether it was feasible to use electronic rapid alternate occlusion in a home based program to successfully and safely treat the patient’s primary complaint of diplopia. A second question was whether the treatment would have an effect on the pertinent clinical findings.

The answer to the first question is that the diplopia reduced dramatically and without side effects. From the perspective of the patient, that is the most important point. As the diplopia reduced, the symptoms as measured by the COVD Quality of Life assessment were also reduced.

The response to the second question indicated in the pertinent findings of the office evaluations during the course of treatment is included in Table 1. The near phoria remained unchanged at 15 of exo during this time and the distance associated phoria changed from 2 of exo to 0.5 of eso. In terms of the ability to compensate for the near phoria, the base-out vergence improved from the initial negative break and recovery values to variable, but more respectable positive values, even after four months of no active therapy (Table 1). It is also noteworthy that this increase in base-out vergence was not at the expense of base-in vergence. This supports my premise that decreasing the suppression would enhance fusional vergence. This suggests, at least in the present case, that the CI was not responsible for the ICS, but that the ICS was responsible for the CI.

Although LH received no VT specifically designed to increase base-out vergence, I in no way mean to imply that vergence therapy should be abandoned in all cases. However, it does suggest that, in programming therapy for patients who are

| | Near Phoria | Base-Out | Fatigues to | Base-In | Fatigues to | Dist Associated Phoria |
|------------------|-------------|----------|-------------|---------|-------------|------------------------|
| 5/02 | 15 exo | -6/-8 | -10/-12 | 20/12 | 26/14 | 2 BI |
| 7/02 | ▲ | 24/0 | 24/-6 | 24/6 | | 2 BI |
| 8/02 | | 24/14 | | 24/12 | | 1 BO |
| 12/02 | ▼ | 18/0 | 18/6 | 24/8 | 18/6 | 0.5 BO |
| 12/02 second set | 15 exo | 24/6 | | 18/6 | 24/6 | |

Table 1. Changes in vergence over time during therapy. I do three sets of vergences in succession to ascertain if this simple fatigue affects vergences. The “Fatigues to” columns show the effect of that fatigue, either diminishing the vergence finding or, often with L.H.’s base-in vergences, actually increasing with repetition. An open box means no fatigue effect was seen. At the 12/02 examination, I did a second set of near vergences after the rest of the analytical examination was done just to see if I could elicit any ICS with further fatigue.

being treated for ICS and vergence problems, treating the ICS before vergence could be the sequence of choice. I intend to test this possibility in more patients where divergence or convergence dysfunctions are accompanied by ICS.

This improvement in the sensory lock by elimination of the ICS would seem to follow from, and support the suggested neurological basis for ICS. I have suggested that ICS is a function of a deficient magnocellular (M) visual pathway.^{10,11} These cells have their greatest density at the central retina. I have previously proposed that that ICS can be treated with alternating flicker because flicker is a strong motion stimulus and the M pathway is primarily responsible for motion detection.¹²

Since the M pathway carries motion information, flicker at an appropriate pace can potentially isolate a particular group of similarly responding cells.¹² A repetitive stimulus should strengthen the pathway and its synapses. This was the basis for using the electronic rapid alternate occlusion device. Since I determined the appropriate alternation pace with a central (drawing) target on the cheiroscope for LH, and since the ICS is found centrally, it strongly suggests that the 4.6 to 5 Hz alternation isolates the central M cells. Any decrease in ICS caused by an isolated motion stimulus such as electronic rapid alternate occlusion, therefore, supports my M pathway-based ICS theory.

The fusional convergence improvement with elimination of the ICS also

lends support to some of the explanations for the “dyslexic” visual confusion postulated with ICS. I’ve suggested that ICS eliminates the fusion lock for accurate fixation during reading of the suppressed eye (mis-aim or aiming drift); the central suppression removes sensation momentarily, so that the suppressed eye drifts slightly off target.¹³ The aiming drift off target increases the motion stimulus, finally producing enough motion to trigger resolution of the suppression so both eyes are functioning simultaneously. A confused or diplopic image results, depending on the amount of misalignment. Correction in aim should occur once both eyes are functioning simultaneously. But, the whole process repeats because the M pathway remains deficient or defective producing the intermittency. ICS, then would produce visual confusion and motion as the central vision turns on and off and aim has to correct for any errors that occur during the suppression.

The convergence response of LH supports this thesis that some of the confusion from ICS is the product of the fixation drift and resulting mis-aim of the suppressed eye. However, if the near exophoria is less than L.H.’s, and/or the fusional convergence is more robust than L.H. demonstrated, we might expect small mis-aims and visual confusion rather than frank diplopia.

Conclusions

This case illustrates that although underlying systemic and neurological etiologies must be considered in sudden onset diplopia, when these conditions have been ruled out, the patient’s chief complaint must be addressed. In the present case, ICS, coupled with an inadequate convergence system were underlying causes of the diplopia. Vectographic testing uncovered the ICS. The uniqueness of my therapy was that amelioration of the ICS by home therapy with electronic rapid alternate occlusion improved the patient’s convergence range at near and very significantly improved the patient’s chief complaint and other symptoms.

Dr. Hussey is the primary inventor of the rapid alternate occlusion goggles. No commercial version is yet available. Clinical studies continue.

References

1. Rosner J. Pediatric optometry. Woburn, MA: Butterworth Publishers, 1982:214.
2. Hussey ES. Examination of binocular sensation over time with routine testing. *J Behav Optom* 2000;11(2):31-4.
3. Hussey ES. Temporal characteristics of intermittent central suppression. *J Behav Optom* 2002;13(6):149-52.
4. Maples WC. Test-retest reliability of the College of Optometrists in Vision Development Quality of Life Outcomes Assessment. *Optom* 2000;71:579-85.
5. Bielschowsky A. Lectures on motor anomalies. Hanover, NH: Ophthalmic Publishing Co., Dartmouth Coll Publicat, 1938:102-3.
6. Hussey, ES: Very rapid alternate occlusion as a treatment for intermittent exotropia. *J Optom Vis Dev* Spring 1995;26(1):18-22.
7. Hussey ES, inventor. 1993 Nov 23. Eyeglasses for use in the treatment/diagnosis of certain malfunctions of the eye. US patent 5,264,877.
8. Hussey ES. Use of visual flicker in remediation of intermittent central suppression suggests regionalization of vision. *J Behav Optom* 1999;10(1):3-11.
9. Doyle, AC. The hound of the baskervilles. In: The complete Sherlock Holmes treasury. New York: Avenel Book, 1976. (originally published in Strand Magazine August 1901 to April 1902).
10. Hussey ES. Binocular visual sensation in reading: A unified theory. *J Behav Optom* 2001;12(5):119-26.
11. Hussey ES. The on-switch for seeing. *J Optom Vis Dev* Summer 2003;34(2):75-82.
12. Hussey ES. Speculations on the nature of visual motion with optometric implications. *J Behav Optom* 2003;14(5):115-9.
13. Hussey, ES: Intermittent central suppression: a missing link in reading problems? *J Opt Vis Dev* June 1990;21:11-6.

Corresponding author:

25 W. Nora, #101

Spokane, WA 99205

spacegoggle@att.net

Date accepted for publication:

May 2, 2004

EDITORIAL continued

They had only negative comments about the methods, results, conclusions and motivation to a study by Zaba, Mozlin and Reynolds³ that was published in this journal. Zaba, Mozlin and Reynolds compared the effectiveness of vision screenings to full vision examinations as a result of the Kentucky law that mandates an optometric or ophthalmological examination for children first entering school. That article concluded that:

Vision screenings certainly play an important role in identifying visual dysfunctions in a variety of settings. However, our data strongly indicates that, in the case of youngsters entering school for the first time, vision screenings can identify some youngsters with visual dysfunctions, but can miss a significant number of others.^{3(p125)}

The results of Phase I of the VIP study certainly add more evidence to this conclusion, and hopefully will be read by Drs. Hoskins and Fleming.

References

1. The Vision in Preschool Study Group. Comparison of preschool vision screening tests as administered by licensed eye care professionals in the vision in preschoolers study. *Ophthalmol* 2004;111:637-50.
2. Hoskins HD, Fleming M. Letter to the editor. *J Behav Optom* 2004; 15:17.
3. Zaba JN, Mozlin R, Reynolds WT. Insights on the efficacy of vision examinations & vision screenings for children first entering school. *J Behav Optom* 2003;14:123-6.