

Vision Therapy for Sensory Fusion Disruption Syndrome: Two Case Reports

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ABSTRACT

Introduction: Sensory fusion disruption syndrome (SFDS) is a rare consequence of acquired brain injury that causes diplopia even though there is bifoveal motoric alignment of the two eyes. These individuals frequently note that the images are near each other and may appear as if one image is behind the other. When a constant strabismus is present, the prism that neutralizes the strabismic angle does not eliminate the diplopia. Monocular occlusion is often the palliative treatment utilized. Two cases are presented that have successful outcomes following office-based optometric vision therapy (OVT).

Case Summaries: Case 1 is a 54-year-old female with SFDS who sought relief for her diplopia of four years duration and wished to regain the ability to read for more than ten minutes at a time. An 18-visit office-based optometric vision therapy program led to improved visual function, significant symptomatic relief, and improved activities of daily living but not a complete elimination of her diplopia. Case 2 is an 11-year-old boy with SFDS, fusional vergence dysfunction and accommodative dysfunction following head injury. He gained complete recovery after a relatively short course of eight office-based OVT sessions.

Discussion: These two case reports, as well as the accompanying literature review, suggest a set of guidelines for establishing an appropriate prognosis regarding OVT for SFDS. A novel test for the depth of SFDS is presented along with an original method of arranging conditions for OVT procedures that are intended to alleviate SFDS.

Keywords: acquired brain injury, ambient vision, binocular vision, diplopia, optometric vision therapy, sensory fusion disruption syndrome

Background: Sensory fusion disruption syndrome (SFDS) is the inability to fuse images even though there is bifoveal motoric alignment of the eyes.¹ This syndrome causes diplopia and is an uncommon sequela of acquired brain injury (ABI). SFDS patients present complaining of constant diplopia. They frequently have difficulty pinpointing the spatial characteristics of the diplopic images and will often note that the images are near each other, with one image appearing as if it is behind the other.

The diagnosis of SFDS requires an ABI event and the presence of diplopia when there is motoric bifoveal alignment of the eyes (no strabismus). In cases of SFDS with constant strabismus, prism that neutralizes the strabismus angle(s) does not eliminate the diplopia. Thus, treatment of SFDS cannot be effected with relieving prism or eye muscle surgery. Monocular occlusion is a palliative treatment that is frequently used.¹⁻³ The occluder can be worn during activities (e.g. reading) in which the diplopia is most bothersome. Rummel describes a sector occlusion method in which the occlusion is translucent and placed on a spectacle lens in front of the pupil.³ Long term wear of corrective prism for an associated strabismus has led to spontaneous resolution of the diplopia associated with SFDS.¹ Initially the prism that neutralized the strabismus may not eliminate

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Tassinari JT. Vision Therapy for sensory fusion disruption syndrome: two case reports. *Optom Vis Dev* 2010;41(4):215-221.

the diplopia, over a period of months, the diplopia abated. Another treatment option for SFDS described in-depth by London is monovision.^{1,4} Prism displacement and hypnotherapy have been suggested as compensatory treatments.² A sixth option is optometric vision therapy. This article presents two case reports of successful OVT for SFDS. It will also suggest some prognostic clinical findings regarding OVT for SFDS and introduce a new procedure that allowed temporary extinction of the SFDS diplopia for both patients.

Case 1 – History

YL, a 54-year-old female, presented with a chief complaint of constant double vision that was most problematic while reading. She had difficulty describing the location of the diplopic images relative to one another. Sometimes one was “next to” the other in a random position (above, to the side, etc) and “sometimes I see one behind the other.” In all positions the images were separated by “less than inch but sometimes as much as 1-2 inches”. Her chief complaint of diplopia was noted four years prior to her presentation for the initial evaluation. The diplopia was a result of significant head trauma sustained during the commission of a crime. The head trauma, which resulted from multiple blows using a blunt instrument, caused loss of consciousness for four hours. Secondary visual symptoms included asthenopia, intermittent blur, illusory movement of print while reading, and photophobia while she was both indoors and outdoors. Although reading was important for work (editor) and hobbies, YL could only read for approximately 5-10 minutes before the symptoms associated with the head trauma became intolerable.

YL's past eye history was remarkable for myopia, astigmatism and presbyopia corrected with progressive addition lenses. Her most recent spectacles did not have prism and her progressive addition lenses were mounted in a small frame (fitting height 17mm). Prism was prescribed on three different occasions during the past four years. None relieved the diplopia. Home based convergence therapy was prescribed (pencil push-ups) but YL did not notice any relief in her symptoms after trying the procedure for a few weeks. Her medical history was significant for anxiety, depression and chronic headaches. The headaches were not triggered by reading or other visually demanding tasks, but reading exacerbated them. Her

medical treatments included alprazolam tablets and osteopathic adjustments. The osteopathic treatments were successful at helping to reducing her headaches. YL's osteopath referred for a OVT evaluation.

Case 1 – Diagnostic examination

Visual acuity was 20/20 each eye for distance and near through habitual lenses. The subjective refraction was the same as her current prescription. Her pupil evaluation, color vision, visual fields, anterior segments, and posterior segments were all unremarkable.

Repeated unilateral cover test showed no strabismus in all positions of gaze at distance and near. The alternate cover test demonstrated 3 diopters exophoria at distance and near. The Modified Thorington assessment through a +1.75 resulted in a range of 3 esophoria to 2 exophoria. There was no hyper deviation present. No horizontal or vertical fixation disparity using the Wesson Fixation Disparity card was seen. Using various prismatic powers to try to eliminate her diplopia was unsuccessful.

Sensory fusion testing showed 252 seconds of arc with the Random Dot E test at 100cm. Second degree fusion using a red lens showed normal fusion at near and distance but the SFDS image was also present under normal room illumination. In a dark room, the red lens test resulted in normal fusion at near and constant heteronymous crossed diplopia at far. The target for the red lens test at far was a dot of projected light. Without the red lens, while still in a completely dark room, YL exclaimed that she saw the dot of light as single. This suspension of the SFDS diplopia viewing a projected dot of light at six meters in a dark room was repeatable. At near, viewing a transilluminator light in a completely dark room, the SFDS diplopia was present but the second image was less pronounced.

Vergence skills were assessed with the results noted in Table 1. All vergence tests were based on a report of double vision of the test target. YL was able to differentiate between her SFDS second image (constant) and the induced diplopia of the test.

YL was diagnosed with post-ABI SFDS and fusional vergence dysfunction. The fusional vergence dysfunction is based on the low scores on all six vergence tests (Table 1). Her near prescription (+1.75 add) was placed in a trial frame and YL was given appropriate reading material. She reported a minor improvement in visual comfort but no elimination of

Table 1: Pre-VT and Post-VT (Italics) Vergence Test Results, YL (Case One)

Test	Result	Rating ^a
Nearpoint of Convergence (NPC)	x/30cm/40cm....	very weak
	<i>25cm/3cm/5cm..</i>	<i>strong</i>
Facility (3BI/12BO) ^b	1 cycle/min.....	low
	<i>17 cycles/min</i>	<i>above expected</i>
Negative FV ^c , 6m	x/6/3.....	break – adequate, recovery – weak
	<i>x/8/5.....</i>	<i>strong</i>
Positive FV, 6m	x/15/2.....	break – weak, recovery – very weak
	<i>x/20/5.....</i>	<i>break – adequate, recovery – weak</i>
Negative FV, 40cm	x/10/4.....	break and recovery – very weak
	<i>12/20/12.....</i>	<i>adequate</i>
Positive FV, 40cm	x/12/-4.....	break and recovery – very weak
	<i>x/20/2.....</i>	<i>break – adequate, recovery – very weak</i>

^a Rating is based on Griffin/Grisham for NPC and FV tests.⁵ Facility is based on Scheiman Wick.⁶

^b Facility, vergence facility test administered per Scheiman Wick protocol.⁶

^c FV, fusional vergence range in phoropter with Risley prisms.

the diplopia. A light blue tint was placed in front of the trial frame and she then reported a dramatic improvement in visual comfort. This response to the blue filter led to the initial treatment of prescribing single vision lenses with a light blue solid tint (Blue #1) for reading.

The second component of the treatment plan was OVT. The goal of therapy was to improve her sensory fusion and vergence abilities from their presently deficient levels to at least the expected norms. This improvement in visual function will lead to the main goal of symptomatic relief and increased capability of performing activities of daily living such as reading. The prognosis appeared to be good for at least some improvement in the patient’s symptomology although the degree of improvement was uncertain.

YL agreed to the treatment plan and OVT was administered during weekly one hour office appointments coupled with 20-30 minutes of daily home vision therapy (HVT). Estimated treatment time was 18 office sessions with a progress evaluation planned for every sixth visit. The procedures within and objectives of the OVT program were selected in accordance with treatment procedures described by Scheiman and Wick,⁷ Cohen,⁸ and Griffin and Grisham.⁹ A unique strategy of the OVT program was to arrange conditions such that YL did not see the SFDS diplopia during the VT activity. This treat-

ment strategy was implemented largely by trial and error. It was found that a dark background/dark room incorporated into the therapy strategy was important for arranging the conditions of the OVT procedures. The use of reduced illumination was based on the SFDS diplopia extinction noted while using a darkened room during the assessment.

At YL’s first office OVT session she reported that reading was more comfortable and could be sustained longer (ten minutes) with her new single vision blue-tinted nearpoint glasses. A representative office based procedure completed during her first visit was sliding vergence stepping-stones using the Keystone Telebinocular.¹⁰ While viewing these large in-instrument indirectly illuminated third degree targets, YL obtained normal fusion without SFDS diplopia with the room lights off. She practiced maintaining fusion while the cards were alternately separated to increase divergence and convergence demand. As YL progressed, this procedure was successfully completed with increasing amounts of room illumination. She was also guided toward gaining a self awareness of which type of vergence she was activating; divergence (“looking far”) or convergence (“looking near”).

A HVT procedure given initially incorporated a large red/blue third degree fusion target. YL wore red/blue filters with her habitual PAL glasses. The room lights were off and a single lamp illuminated the VT target. YL noticed the float effect of the targets and reported that the SFDS image was barely noticeable. Her assignment was to move the target randomly in all directions while maintaining the float effect. After two weeks of using this procedure, she reported during office visit number three that the SFDS image was not present while conducting this HVT technique. She continued this procedure at home for eight weeks. It was made more challenging by increasing the room illumination, adding base-in and base-out prism jumps, reducing the size of the target, and assigning third degree targets

with higher convergence and divergence demands.

At the first progress evaluation, YL remarked that her double vision was “definitely better, reading is much better too.” There was also improvement in her vergence ability. Her NPC was now 30/10/15 cm, Positive Fusional Vergence (PFV) (6m) x/20/4, and Negative Fusional Vergence (NFV) (40cm) improved to 15/24/16 (see Table 1 for pre-OVT results).

OVT continued for a total of 18 office visits during which she demonstrated outstanding compliance. During the progress evaluation on visit 18, YL reported that her double vision was significantly reduced and that she could read comfortably and effectively for 45-60 minutes. Her vergence skills were better at this progress evaluation office visit (Table 1). Her PFV recovery (6m) and NFV recovery (40cm) were still somewhat below expected levels. The vergence assessment results were also approximately the same as they were on office visit #12. Moreover, YL’s symptoms and performance gains had changed very little since visit #12. In contrast, there was steady improvement in her symptoms from visit #1 to visit #12 to go along with improved vergence test scores. It was concluded that OVT had been successful and that she had reached a plateau. Therefore, her therapy was discontinued. Three maintenance HVT procedures were assigned. A follow-up progress evaluation three months after the conclusion of OVT showed no decline in vergence ability and no new or worsening of prior symptoms.

Case 2 – History

LS, an 11-year-old boy, presented with his father for an OVT consult because of constant double vision. Intermittent blur and asthenopia while reading were secondary symptoms. Six-months prior, LS fell off his bicycle and hit his head. He was dizzy, confused, had jaw pain and visual disturbances (blur, diplopia, depth confusion) immediately after the head trauma. He was taken to an emergency room where a

physical examination, head CT and jaw x-ray were administered. These were unremarkable. The next day vision returned to normal except for a constant diplopia. The diplopia persisted and LS was evaluated by a neuro-ophthalmologist and two neurologists over a five month period. No therapy was suggested and LS was advised that his symptom would self-resolve. A third neurologist was consulted who then referred LS for an OVT consult. LS was in good general health and doing well in school. His father described him as very well coordinated and an excellent athlete. Prior to the ABI, there were no diagnosed vision or eye problems.

Examination revealed emmetropia with normal visual acuity, color vision, and visual fields. The pupil evaluation, ophthalmoscopy, biomicroscopy, and intraocular pressure were all unremarkable. Monocular ductions were full in all directions. Versions were also unrestricted but the patient noted constant diplopia that varied from a slight horizontal separation to images being one is behind the other. Unilateral cover test showed no movement even after repeated testing. Alternate cover testing was 1 esophoria at far and near. Modified Thorington assessment at near varied between ortho and 2 esophoria. There was no vertical deviation. Fixation disparity was 8 base in. Trial framing BO and BI prism, did not eliminate or decrease the diplopia.

Table 2: Pre-VT and Post-VT (Italics) Vergence Test Results, LS

Test	Result	Rating ^a
Nearpoint of Convergence (NPC)	x/14cm/20cm....	break – weak, recovery – very weak
	<i>x/3cm/6cm.....</i>	<i>very strong</i>
Facility (3BI/12BO) ^b	6 cycles/min.....	well below expected
	<i>18 cycles/min....</i>	<i>above expected</i>
Negative FV ^c , 6m	x/4/1.....	break and recovery – very weak
	<i>x/8/5.....</i>	<i>strong</i>
Positive FV, 6m	x/2/0.....	break and recovery – very weak
	<i>12/16/8.....</i>	<i>blur/break – adequate, recovery – weak</i>
<i>Negative FV, 40cm</i>	<i>x/12/8.....</i>	<i>break and recovery – very weak</i>
	<i>x/20/10.....</i>	<i>break – adequate, recovery – weak</i>
Positive FV, 40cm	x/2/-2.....	break and recovery – very weak
	<i>x/16/8.....</i>	<i>break – very weak, recovery – adequate</i>

^a Rating is based on Griffin/Grisham for NPC and FV tests.⁵ Facility is based on Scheiman Wick.⁶

^b Facility, vergence facility test administered per Scheiman Wick protocol.⁶

^c FV, fusional vergence range in phoropter with Risley prisms.

Table 3: Pre- and Post-VT (*Italics*) Accommodative Test Results, LS

Test	Result	Rating ^a
Amplitude OD/OS (expected = 12.0D)	8.0D / 8.0D..... <i>12.0D / 12.0D.....</i>	very weak <i>adequate</i>
Facility (+/-2.00) OD	3 cycles/min..... <i>12 cycles/min.....</i>	very weak <i>adequate</i>
Facility (+/-2.00) OS	6 cycles/min..... <i>12 cycles/min.....</i>	very weak <i>adequate</i>
Facility (+/-2.00) OU	1 cycle/min..... <i>13 cycles/min....</i>	very weak <i>very strong</i>
NRA	+2.00.....	adequate
PRA	-2.50.....	very strong
MEM Retinoscopy	+0.50DS OD & OS...	normal
Binoc Cross Cyl	+1.00.....	normal

^a Rating is based on Griffin/Grisham guidelines

Vergence and accommodative skills were assessed. LS scored below expected levels on all six vergence tests and on four out of eight accommodative tests. (Tables 2 and 3 summarize the vergence and accommodation test results.) The Developmental Eye Movement- Subtest ‘C’ was administered. He scored above the expected level for his age.

Sensory fusion testing for stereopsis was normal using the Random Dot E test. Second degree fusion testing (red lens) in normal room illumination resulted in an expected response at far and near (both were accompanied by the SFDS second image however). In complete darkness, LS had no second image. This elimination of the SFDS diplopia was repeatable under conditions of darkness with or without the red lens while he was viewing a small circular spot of white light. He was diagnosed with SFDS, fusional vergence and accommodative dysfunctions. Optometric vision therapy was recommended. The secondary symptoms of intermittent blur and asthenopia were given a good prognosis for resolution. The primary symptom of constant diplopia was not given as favorable a prognosis. The possibility of no improvement despite a diligent effort in OVT was discussed with LS and his parents.

LS and his parents agreed to proceed with the OVT consisting of weekly 1-hour in-office sessions with daily HVT. Treatment length estimate was estimated to include 18 in-office sessions with a

progress evaluations every sixth visit. One of the office-based VT procedures assigned on office visit one was for vergence and involved using the Quoits Vectogram.¹⁰ In a dark room, LS fused the back-lit pair of Vectogram targets while wearing polarized glasses. The dark conditions reduced the SFDS diplopia to an intermittent phenomenon. The targets were separated to increase the divergence or convergence demands. He was instructed to maintain fusion and to be aware of any changes in size and location of the image. He ultimately gained a consistent awareness of the Quoits appearing to enlarge

and float away as he diverged to meet the demand and to shrink and float closer during convergence activity achieving S.I.L.O. He was able to consistently diverge to the letter “J” before losing motor fusion and was also able to converge to the number 24. A third goal challenged LS’ vergence flexibility. With the targets set to a divergence demand, LS alternately looked at a very close target (a pencil tip 15 cm from his eyes) and the Quoits target. Thus, he converged in real space then diverged to fuse the separated polarized targets. Vergence flexibility was trained in the other direction. The Quoits were set to a convergence demand and LS alternated between them and looking out a window at a mountaintop several miles away. LS met this goal on visit three by fusing the targets at “J” for divergence flexibility and 22 for convergence flexibility. LS completed this procedure under normal room illumination on OVT visit four. During the OVT, he appreciated a noticeable reduction in SFDS induced diplopia.

HVT included the Brock String procedure as described by Scheiman,¹¹ with a modification in lighting. A gooseneck lamp directed light onto the string in an otherwise dark room. This condition reduced or eliminated the SFDS induced diplopia. In subsequent weeks, increased background room illumination was added. In addition to activating accurate and rapid vergence movements, LS was guided toward an awareness of the way his eyes felt

when looking at near (closest bead on string) versus when looking at the most distant bead. Throughout his OVT program, he was questioned about his awareness of looking near versus looking far during in-instrument procedures.

At the first progress evaluation, LS reported that his double vision was much less and that “I can read...

30 or 40 minutes before I see two or blurry.” The progress evaluation showed significant improvement on all measures as shown in Table 2 and Table 3.

OVT continued for two more office visits after which LS informed his parents that he had no remaining visual symptoms. His parents elected to discontinue therapy based on his asymptomatic status. LS was instructed to continue with home based therapy and watch for a recurrence of symptoms. Three months later he presented for a follow-up evaluation which confirmed that he was symptom-free. This assessment noted his accommodation and vergence skills at or above the expected levels.

Discussion

A literature review of the prognosis for a SFDS cure typically paints a bleak picture. In the first in-depth literature report of SFDS, Pratt-Johnson presented a series of four cases all of which lead to the conclusion that no treatment could be offered.¹² Pratt-Johnson and Tillson reported a second series of four SFDS patients who also showed no improvement.¹³ The authors remarked “we have never seen any recovery from the central disruption of fusional amplitude.”¹³ Stanworth’s case series documented nine SFDS patients, four of whom recovered normal fusion.¹⁴ London and Scott reported four cases of SFDS (along with originating the term ‘Sensory Fusion Disruption Syndrome’) with three having intractable diplopia.¹ The one individual who recovered was a 17-year-old who wore neutralizing Fresnel prisms for esotropia and hypertropia.¹ Initially, the prisms placed the images behind one another but these remained unfused. Four months of constant prism wear lead to normal single vision followed by gradual reduction in the prism amount to the point where no prism was required.¹ Other authors have reported their clinical experience with SFDS and concluded: “it is difficult or impossible to resolve”³ and that “the prognosis in such cases is usually poor”.¹⁵ The lone case report of OVT

Table 4: Clinical findings ascertain prognosis of treating SFDS with VT

Prognosis Better	Result	Rating ^a
Present	<i>Random Dot Stereopsis</i>	Absent
Absent	Comorbid ABI-caused strabismus	Present
Present	ABI-caused vergence skill deficits	Absent
SFDS Diplopia Eliminated	Single Dot of Light at 6m in a dark room	SFDS Diplopia persists

for SFDS found in the literature was of a 61-year-old male who suffered a mild concussion.¹⁵ Eighteen months after the injury, he was diagnosed with an intermittent strabismus and SFDS. Unfortunately after 12 OVT sessions, no resolution of the constant diplopia was noted.

These case reports are the first noting successful OVT for SFDS. They also provide additional support for Scheiman and Wick’s recommendation that “because it is impossible to predict which cases will resolve with treatment, it is appropriate to attempt a trial period of [OVT] treatment.”¹⁵

These two case reports demonstrate the basis of which to establish a favorable prognosis using OVT for patients with SFDS. The patients noted in this paper were uncomplicated ABI cases in that there was no coexistent mobility, cognitive, or language issue. Both patients were highly motivated despite the potentially poor OVT prognosis. Clinicians often are required to make recommendations for OVT in similar complicated cases.

Prognosis Guidelines

First, both patients exhibited random dot stereopsis which has been previously cited as indicating a good prognosis.¹⁵ Second, neither patient had a significant oculo-motor anomalies (a high heterophoria or strabismus). Although SFDS is a sensory fusion abnormality, 17 of the 24 SFDS cases reported in the literature had strabismus caused by ABI.^{1,4,12-16} Among the seven patients noted in various papers without strabismus^{12-14,16,2} had spontaneous resolution of their SFDS.¹⁴ Both of the patients in this article had measurable vergence ranges and there were deficiencies noted in the fusional vergence system. This implies that SFDS may be a consequence of the ABI-caused neurophysiological disorganization that underlies post-ABI binocular dysfunction. It is conceivable that there are two types of SFDS. The primary type is intractable. The second type is

part and parcel of an acquired brain injury related binocular dysfunction. As the binocular system is rehabilitated during OVT, this secondary SFDS improves. Case one in this article, YL, had both types with the secondary form responding to OVT. Case 2, LS, appeared to have the pure secondary form and gained a complete cure.

Another clinical finding that may aid in determining a positive prognosis is the patient having a response to a single dot of white light in a darkened room. As described in case one (YL), this circumstance lead to normal, single, binocular vision. In the second case, the patient also achieved normal single vision/suspension of SFDS diplopia when he was viewing a single dot of light in a dark room. For both patients, the test result was utilized as an indication for OVT that resulted in a reduction (Case 1) or complete elimination (Case 2) of the SFDS diplopia. The implication is that elimination of SFDS diplopia while viewing a single dot of light in a dark room increases the prognosis for successful OVT.

The elimination of SFDS induced diplopia in a completely dark room while viewing a dot of light may provide clues to its etiology. The test conditions remove peripheral binocular fusion clues leaving central fusion to operate practically in isolation. Because central fusion functions in a normal manner in this isolated state, faulty binocular peripheral fusion input is implicated as a potential etiology of SFDS induced diplopia. This connection can also be explained in the context of the Post-Trauma Vision Syndrome as described by Padula.^{17,18} Selective disturbance of the ambient visual subsystem (peripheral spatial orientation, movement detection) which intrudes on the focal visual subsystem (central fusion, form vision) is believed to be at the core of Post-Trauma Vision Syndrome.

The ambient visual subsystem is mediated by structures in the midbrain, which is consistent with reports in the literature that the lesions causing SFDS induced diplopia are probably subcortical and located in the mid-brain.^{1,12,13,16} Thus, faulty precortical spatial

input from the ambient visual subsystem inhibits the ability of the foveae to correspond and diplopia ensues. A seated patient in a dark room viewing a small central target does not require or have access to the influence of the ambient subsystem input and normal fovea correspondence/normal single vision can take place. (Guidelines for prognosis are noted in Table 4.)

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