INTERMITTENT CENTRAL SUPPRESSION caused by CERVICAL TRAUMA WHIPLASH

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Abstract

Whiplash cervical trauma has been shown to cause visual changes. Intermittent central suppression (ICS) has been shown to be associated with reading problems. Three patients were examined before and after whiplash trauma. Vision examination results show that the only significant change in these patients is the development of ICS where none had been before, or a dramatic increase in the number of vectographic tests showing ICS. These three adult patients also complained of reading problems paralleling the complaints of children with ICS.

Key Words

intermittent central suppression, whiplash, vision, reading problems, vision therapy

Suppression is a presumably neurological inhibition of vision in one eye not attributable to pathology or anatomical anomaly. The contralateral eye maintains vision during the suppression period. Interruption of sensation in the non-suppressed eye by mechanical means (occlusion) will force the suppressed eye to see, temporarily eliminating the suppression. Removal of the occluder reinstates the conditions that require or facilitate the suppression.

Constant suppression is considered to almost always accompany long-standing strabismus or amblyopia. Suppression is also diagnosed without strabismus and amblyopia. Non-strabismic suppression usually manifests as an intermittent "on and off" phenomenon. A typical on/off cycle would average approximately three seconds of suppression alternating with two-eyed vision.[1] This type of suppression usually alternates between eyes and involves central, but not peripheral, vision. Designating non-strabismic suppression as Intermittent Central Suppression (ICS) identifies this form of suppression and differentiates it from strabismic/amblyopic/constant suppression.

ICS as a clinical entity can be traced back to the work of Strauss and Immerman.[2] They used stereoscopic targets to diagnose "macular suppression" and to show a correlation to reduced reading performance. However, even as early as 1950, Louis Jaques, Sr. exhorted that correction of "suspension of vision" was the "first and most important" step in correcting vision problems requiring vision therapy.[3] Hussey,[1] along with renaming this form of suppression ICS, showed that common suppression tests, especially those used in strabismus, will in all likelihood *not* diagnose ICS. He described a vectographic target-based test sequence that seems to diagnose the problem more effectively than traditional strabismus suppression tests.

ICS probably interferes with accurate fixation and fusion: repeated suppression-induced vergence fluctuations[4] produce non-registered aiming errors.[1] As each seconds-long suppression resolves, any mis-aim that occurs will create diplopia and super-imposition of letters from the two images, e.g., when reading.

This is usually interpreted during the act of reading as a confused word. Since a one degree aiming error can account for two or three letters of print, even small aiming errors can disrupt accurate reading. Peripheral vision remains intact, preventing frank strabismus. Detection of the mis-aim will trigger a vergence correction, but in another few seconds this sequence starts again with another suppression. This repeated ICS-mediated visual confusion in the central visual area can explain such common reading complaints as recurring variable errors in reading small words, which is frequently attributed to poor visual memory.

Even though documentation of the negative effects of ICS on reading is slowly accumulating,[2,3,5,6,7] one stubborn

question remains. Where does it come from, i.e., what is the pathogenesis? Certainly development might be blamed in some cases; e.g., interference in developing general motoric and/or ocular bilaterality might help explain the alternation seen in ICS. Accommodative and/or convergence fatigue might trigger the development of ICS: an accommodative dysfunction could necessitate increased neural drive to accommodation to retain accurate focus. The parallel neural drive to convergence could easily create a minor image slip that would trigger a suppression to avoid diplopia, similar to the development of a strabismic suppression. Repetition of this event over a period of time might develop into the anomaly of ICS. But another possible cause is trauma.

Trauma (Traumatic Brain Injury/TBI) can cause many different vision anomalies.[8] Trauma-caused anomalies such as visual field defects are well documented.[9,10] Fishman-Hellerstein and Freed[11] provide one case study of mild trauma, producing headaches, diplopia, blurred vision, poor balance and attention problems. Ciuffreda, Suchoff, et al.[12] provide a case study of trauma, producing eye movement errors. Both of these patients showed dramatic improvements in visual abilities and symptoms following optometric rehabilitative interventions.

The cervical trauma of whiplash also can create visual anomalies, including strabismus. In two studies, whiplash is defined as acute cervical trauma following a rear-end automobile collision.[13,14] Typically, whiplash has neither obvious direct head trauma nor loss of consciousness.

Current chiropractic literature is documenting diagnosis and treatment of visual changes, specifically from cervical injury, and suggesting that subclinical cerebral ischemia is responsible for the visual deficits.[15,16] This theory allows an explanation for those symptoms or signs that change with chiropractic cervical manipulation. There is little similar study of whiplash specifically in the optometric literature. However, as stated earlier, Fishman-Hellerstein and Freed[11] and Ciuffreda, Suchoff, et al.[12] have documented that optometric intervention can change both signs and symptoms.

This report presents three case studies strongly suggesting cervical trauma from an automobile accident can cause ICS. Unlike previous trauma reports,[11-14] these patients had been examined by the same doctor both *before* and after whiplash trauma, allowing evaluation of changes. The complaints of these patients post-trauma are particularly interesting to optometrists practicing vision therapy. Strauss and Immerman[2] showed a positive link of ICS to poor reading performance. Hussey[1] found that 2/3 of the ICS patients in his record retrospective study complained specifically about reading in their histories. The three patients in this report presented not for routine exams or because of visual field losses or headaches from the whiplash, but with *post-accident reading problems.*

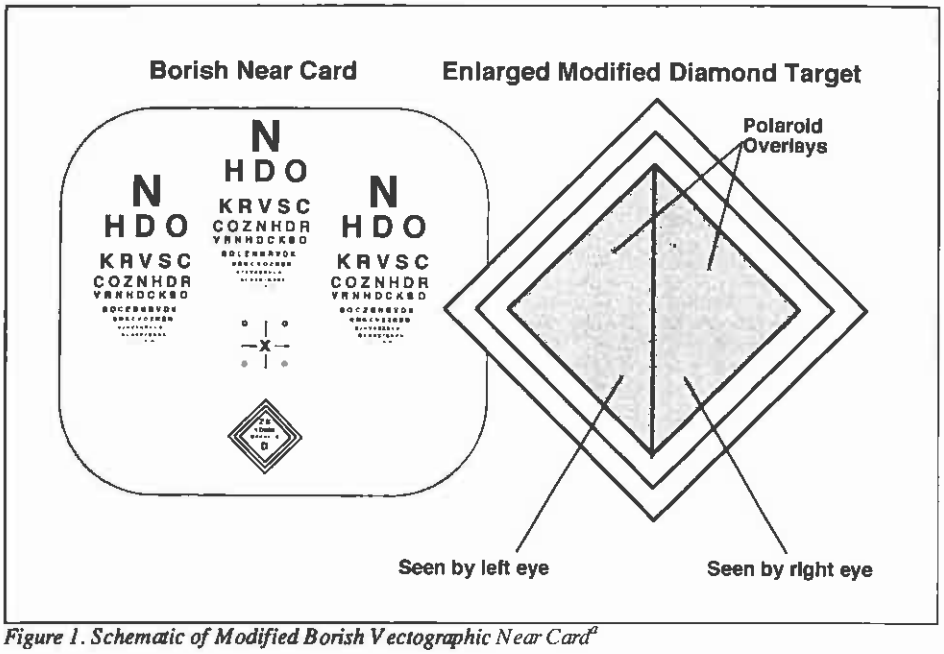

Figure 1. Schematic of Modified Borish Vectographic Near Card[a]

Test Methods

All three patients underwent the same test routine at each vision examination. As previously described,[1,5] the standard analytical test sequence was modified with vectographic targets to maximize the diagnosis of ICS. The test routine proceeds from near testing to distance findings, but is otherwise a fairly standard "21-point" examination sequence. Near vectographic testing uses a Borish Vectographic Near Card[a] modified by the addition of polarizers splitting the diamond target such that the right side of the target is seen by the right eye and the left side of the target is seen by the left eye when viewed through the phoropter Polaroid analyzers (see Figure 1). After three sets of near base-in/base-out ductions, the rotary prisms are removed and the patient is asked if "either side of the diamond changes repeatedly from clear enough to see the letters to completely blackened so the letters can't be seen." If a suppression response occurs, the patient is asked to indicate each change to black and clear for a period of 30 to 60 seconds to assure this is not a single event, but is indeed repetitive. Each repetitive suppression in a typical ICS patient lasts two or three seconds. These suppressions continue during the testing period. Inconsistent suppressions, or large deviations from the "typical" ICS on-off cycle might suggest that accurate diagnosis would benefit from the longer timing period. Timing of the on-off suppression cycle is also useful for determining progress during therapy and then documenting the reduction of the suppression.

After the near sequence is completed, the testing proceeds to distance. The AO Distance Vectographic Test[a] chart provides the same opportunity to test for ICS as the Borish Card did at near. The test lens power is now the distance subjective. The projected AO vectographic chart has a series of polarized targets, each of which also has binocular fixation locks to help maintain accurate fixation. Targets include OD acuity letters, OS acuity letters, OS-OD clockdial cylinder test, OD-OS split anisometropia balance, three "malingering" lines, binocular acuity letters (20/30, 20/25, 20/20, 20/15), fixation disparity cross and stereopic rings. The "malingering" lines are letter rows that have OU - OS - OD polarized acuity letters

repeating across the lines. Unlike the Borish Near Card where a suppression will turn half of the modified diamond black, a suppression of a portion of the projected vectographic chart will cause affected areas of the target to disappear. Therefore, with each target, the patient is asked if any letters, groups of letters, or target line elements repeatedly "disappear completely and come back." "Disappear completely" questioning is easily made age-appropriate.

All of the targets except the binocular acuity letters and stereopsis tests have monocular components and are therefore easily combined with appropriate questioning in order to test for ICS. The aniso and clockdial targets, for example, are split with the right eye letters or line elements on the right side and left eye letters or lines on the left side. So, an alternating ICS can cause an alternation of target elements from side to side. Again, care is taken to time the suppressions over 30 to 60 seconds.

This standard test sequence provides the means to routinely test for ICS in all responsive patients. It works for most children 5 years and older, as well as adults. The routine testing for ICS made this first pre- and post-trauma analysis of visual changes with whiplash possible. Were suppression testing held as "special" or "children's" testing, the ICS documented here would likely have been missed. Conversely, although this report deals with whiplash in adults, most of the other reports on ICS have dealt with reading problems in children. So, this testing should not be held as special testing for trauma patients.

Patient 1: RM

RM, a 55-year-old male, was first seen by the author in 1987 for a routine examination. His hyperopic/astigmatic bifocal prescription was at that time updated to a +2.25 add with other minor changes. RM was taking no medication and denied any health problems. The evaluation indicated no internal or external pathology, eye movement or binocular dysfunction, or ICS on distance or near testing.

In early 1989, RM returned for examination after a car accident. He was taking pain medication and was under chiropractic care. The main reason for returning was that his most recent pair of glasses had been broken in the accident. He was using a previous pair of glasses that were not markedly different from his most recent pair. However, as we progressed through the examination he indicated he was unable to read as well as he had prior to the accident. The parts catalogs required for his work were much more difficult to read accurately. His symptoms were of visual confusion during reading, not blurred vision that might have resulted from an undercorrected near lens prescription.

With the exception of ICS testing, the two sets of examination findings were remarkably similar. Distance refraction, visual acuity, and accommodative and convergence testing revealed minimal, if any, changes. He showed no significant horizontal or vertical phorias. No diplopia or gaze restrictions ever surfaced. RM showed no suppression during the first (1987) examination. Again, test procedures were the same at each examination session. RM's post-trauma changes in findings are summarized in Table 1.

During the second examination, RM showed ICS on the near (Borish Card) fixation disparity target. Since this was different from prior exams, I asked about any reading problems he might be experiencing, and he then reported his difficulty with the parts catalogs. When the distance portion of the examination sequence showed suppression on distance acuities, RM reported, "I've never seen things disappearing like that before." At this second examination, determination of the right eye's refractive status required occlusion of the left eye because of the suppressions, even though this was a vectographic *binocular* test sequence. The subjective and objective findings with RM show a stark contrast between 1987 and 1989 *only* in the ICS responses on vectographic testing. With the association of ICS to reading problems, RM's reading confusion symptoms can reasonably be linked to the development of ICS after the car accident.

Patient 2: PN

PN, a 43-year-old woman, was initially examined in December 1992. Her specific complaint was a variable lack of focusing affecting both distance and near vision. The focusing problem affected her "sometimes close, sometimes far." The eye health examination was within normal limits with the possible exception of mild lenticular hazing. Foveal reflexes were evident in both maculae. Distance acuity corrected to 20/20 OD, OS with Plano and -.25 sphere respectively. The analytical findings agreed with her early presbyopic symptoms. A +1.00 OU add for near resulted in 20/20 acuity for both eyes. PN did show some alternating suppression on the split diamond test, certainly contributing to her perception of variability in focus and possibly explaining the distance focus problem. PN chose to not use lenses or correct the suppression.

Twenty-two months later, PN returned for another examination. PN's car had been rear-ended three months after her 1992 examination. She was still in physical therapy in 1994 and the therapist had suggested her spinal cord had been "stretched" and that might be responsible for some of her symptoms. PN complained that she seemed to be looking through "a film or fibers," which, as we continued, designated as visual confusion. Any spinal manipulation made her eyes worse. She also complained that she noticed her peripheral vision more than before the latest accident. Table 1 summarizes post-trauma changes in PN's findings.

The eye health examination in 1994 was again within normal limits, including the visual fields. Foveal reflexes again were evident and distance acuity was correctable to 20/20 with each eye. Testing again indicated a +1.00 add for near, but at this examination the best near acuity was 20/40, not 20/20 as previously tested (Borish Near Card). Although small changes occurred over the 22 months in the distance refractive status, and base-in ductions were somewhat greater in 1994, the major differences between the two groups of analytical findings were in the amount of suppression in 1994. Whereas suppression occurred only on the Borish Card split diamond in 1992, suppression occurred in 1994 on the Borish Card, and also on the distance vectographic chart acuity letters, the distance alternate letter "malingering" letter line, distance aniso letter grouping and distance fixation disparity cross. The ICS responses on a greater number of vectographic tests suggested an increase in total magnitude of PN's suppression. Distance stereopsis was normal (AO vectographic chart), agreeing with Hussey's previous finding that stereopsis and ICS are independent phenomena.[1]

Table 1. Summarization of Post-Trauma Vision Finding Changes

PATIENT	First Exam Date/ Second Exam Date	Changes in Accommodation, Convergence or Motility Findings	Vectographic Tests Showing Newly Developed Suppression Post-Trauma
RM	1987/1989	Small decrease BI duction recovery. Small decrease BO duction break	OD near fixation disparity, split diamond, dist. fixation disparity; alternation on distance acuities
PN	1992/1994	Small *increase* in BI ductions	Alternation distance acuities; OD distance "malingering" rows, distance aniso chart; OS distance fixation disparity
SS	1979, 1982 1989, 1992 / 1994	Small decrease in duction breaks BO *and* BI *negative* recoveries	Alternation distance fixation disparity and all "malingering" rows; OD distance acuity; OS near fixation disparity

Some caution in interpretation may be necessary since suppression existed at the 1992 examination and therefore the patient "knew what the best answer might be" on those questions probing suppression. Malingering was not detected. Given that caution, the recent trauma (whiplash) apparently greatly increased ICS in PN and increased her visual confusion symptoms. The manifestation of this ICS-induced visual confusion was the "film" through which she seemed to be looking, the disturbing peripheral vision, the variability in vision with spinal manipulation and the reduced near acuity.

Patient 3: SS

Vision care history for Patient SS started in late 1975. At the age of 16 years, SS displayed ICS associated with convergence insufficiency and poor oculomotor control. At that time ICS was diagnosed using a stereoscope and standard Keystone Cards[b] with appropriate searching questions. Five months of vision therapy successfully dealt with those problems. At the conclusion of therapy, SS's mother commented that after therapy she had become a self-motivated and excellent reader.

SS had routine vision examinations in 1979, 1982, 1989, and 1992. From 1982 on, the examinations employed the vectographic binocular refraction techniques described above. Only one examination, 1992, showed *any* suppression. At that examination, the left eye suppressed slightly on the divided diamond test. No symptoms accompanied this suppression.

SS returned in 1994, 25 months after the 1992 examination. She had been rear-ended twice in a three-car accident just three weeks prior to this 1994 examination. SS complained of headaches, slow focus changes from near to far and back, blur if she would lie down, and some light flashes. As we discussed her symptoms and the change in her vision since the accident, SS said, "It feels like before (the therapy of 1975)." Reading was harder than prior to the accident. Her eyes hurt after about 15 minutes of reading. SS stated that since shortly after the accident she had been under the care of a chiropractic physician. Table 1 summarizes post-accident analytical finding changes. Again, the examination routines were the same, and eye health findings continued within normal limits and unchanged throughout the history with SS. Accommodative findings were within normal limits both before and after the trauma.

Post-trauma, ICS was detected on near fixation disparity, OD distance acuity letters, all rows of alternate letter distance "malingering" lines and distance fixation disparity cross. She commented that she "didn't see things disappear" on distance testing during any of the previous examinations. SS had difficulty with near phorias, commenting that the top target "jumps over" the bottom. Suppression precluded testing near fixation disparity and added uncertainty to the distance fixation disparity tests. All phorias were within normal limits and changed insignificantly post-whiplash. However, both base-in and base-out duction recoveries at near were negative. This might be explained by ICS-induced visual confusion preventing SS from detecting the recoveries quickly and accurately. The non-registered aiming error theorized by Hussey[1] with centrally located intermittent suppression could account for this finding.

Summary

These findings should be interpreted with the caution afforded any single case studies. However, all three patients exhibited some striking commonalities. All three cases involved whiplash (cervical) trauma from an auto accident, and either showed ICS where none had been seen before, or displayed an increase in the amount of ICS evidenced during the standard vectographic exam routine. All three patients also complained of visual confusion or changed visual perception coincident with the time of the trauma, and could be explained by our understanding of ICS.

Does this mean whiplash causes ICS? Certainly the case of RM argues strongly for cause and effect. Does whiplash of the severity experienced in an auto crash always cause ICS? That question is probably unanswerable, but the case of PN might indicate that an accident can worsen a pre-existing ICS, or that successive whiplash incidents might have a cumulative effect on the amount of ICS and thereby on the severity of the visual confusion. Is all ICS trauma-based? Most would argue that is unlikely. But, whether the ICS was originally developmental or trauma-based, SS's recurrence of ICS and her description of the symptoms as the same as those she experienced prior to her first treatments of vision therapy implies similar sites of neural malfunction. Burke, et al.[13] suggest the brain stem as a likely site for the damage they see causing oculomotor, convergence, and accommodative difficulties. If accurate, we must then consider the Lateral Geniculate Nucleus (LGN) as a likely site for the sensory disruption defined as ICS, since the LGN lies adjacent to the two sides of the brain stem.[17]

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