OCULOMOTOR REHABILITATION IN TRAUMATIC BRAIN-INJURED PATIENTS

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Abstract

Traumatic brain injury producing adverse visuomotor, and more specifically oculomotor, consequences represents an important area for the rehabilitative team, which now frequently includes the optometrist. This paper overviews the general area and reviews the subjective clinical and objective laboratory results in the literature dealing with oculomotor rehabilitation. It then presents a case report with detailed clinical and laboratory findings, including objective pre/post vision therapy versional eye movement recordings that correlate with the overall improvement and success of our patient. The optometrist can play an integral role in the comprehensive rehabilitation of such patients.

Key Words

traumatic brain injury, vision, oculomotor, optometry, rehabilitation

Clinicians providing care for individuals who have suffered traumatic brain injury (TBI) have become increasingly aware of the vulnerability of the visual system in this regard[1-5] (see Table I).

Much attention has been paid to the resultant field defects,[7,8] as well as the diplopia consequent to an intermittent breakdown in fusional ability or in frank constant strabismus.[9-29] While compromised fixation, pursuit, and saccadic eye movements have also been cited,[6,9,10] the literature does not recognize the functional and behavioral consequences of these abnormalities to the same degree as the field defects and the diplopia. Consequently, it is not fully appreciated that eye movement ability and related learned/trained visual search strategies can provide important compensatory avenues for visual field defects and diplopia. For example, individuals with homonymous hemianopsia can be trained to scan into the side of the field cut.[30] Further, in the non-surgical remediation of strabismus, as well as in the treatment of fusional deficiencies by vision therapy and/or prism application, adequacy of oculomotor control has long been considered by many optometrists to be a prerequisite skill.[31] In addition, even in the absence of other vision dysfunctions, abnormal eye movements can adversely effect reading and general perceptual abilities,[32,33] and therefore deserve at least as much diagnostic and therapeutic consideration as in diplopia and visual field defects. For example, Goodwin[34] believes that: "... to perceive an extended visual scene (as in daily viewing), one must 'palpate' the environment by moving the fovea from point-to-point while compiling and storing a central image or engram of the scene." p.321

It is our observation that the majority of patients with acquired TBI, either from

Table I
Clinical Categories of Head Trauma Injury

1. Soft tissue injuries
   a. extraocular muscle avulsion
   b. hemorrhage and edema
2. Orbital fractures
   a. floor
   b. medial wall
   c. lateral wall
   d. roof
3. Cranial neuropathies
   a. oculomotor nerve
   b. trochlear nerve
   c. abducens nerve
   d. sphenocaverous syndrome
   e. orbital apex syndrome
4. Intra-axial brain stem damage
   a. internuclear ophthalmoplegia
   b. horizontal gaze paresis
   c. vertical gaze paresis
   d. Parinaud syndrome
   e. skew deviation
   f. abnormalities of accommodation, convergence, and fusion
   g. cerebellar lesions
   h. vestibular system dysfunctions
5. Cerebral lesions
   a. saccades
   b. pursuit

(Reprinted with permission of the publisher, from Baker and Epstein.[6])

Table II
Some Oculomotor Changes Associated With Head Trauma

1. Downbeat nystagmus
2. Impaired overall oculomotor control
3. Acute vertigo with head movement
4. Reduced fusional vergence ranges (with or without normal nearpoint of convergence)
5. Divergence paralysis
6. Spasm of the near reflex
7. Multiple ocular motor nerve palsies/non-comitant strabismus (fracture of head/orbital bones)
8. Mechanical restriction of upward gaze (blowout fracture)
9. Fourth nerve palsy (blunt frontal area injury)

an external (e.g., accident) or internal (e.g., stroke) source, have compromised fixational, saccade and/or pursuit abilities, and this agrees with the experience of others[1,6,9-10,32-33] (see Table II).

This is not surprising when one considers the length and complexity of the afferent and efferent neural pathways that provide input to ensure efficiency and accuracy of these motor subsystems.[35] Furthermore, either an accompanying intermittent strabismus or compromised fusional and/or accommodative function, all of which are relatively common in this population,[27-28,36] can adversely effect the quality of eye movements.

The diagnosis of abnormal eye movements can include patient complaints, chairside testing, and/or more precise and objective physiological means. Ciuffreda and Tannen have provided a detailed compilation of diagnostic methods for the various eye movement systems.[32] However, our experience with TBI patients has shown that frequently these dysfunctions are either not diagnosed or, if diagnosed, little or no attempt toward remediation has been made. The increasing inclusion of optometrists to the rehabilitation team of these patients[2-3,36] is testimony of the willingness of some optometrists to take this important step beyond diagnosis.

In this paper, we provide a review of previous work regarding the remediation of eye movement dysfunctions in TBI patients. We further present the detailed history, evaluation, diagnosis, management, and clinical and physiological documentation of improved oculomotor control and binocular abilities in one of our patients who had sustained TBI.

Table III
Vision Therapy for Head Trauma

1. Saccades and pursuits
2. Vectograms
3. Brock String
4. Biocular anti-suppression using lenses and prisms
5. Playskool visual motor activities
6. Balance beam sensory-motor skills
7. Spatial orientation/organizational skills
8. Directional activities
9. Parquetry blocks
10. Computer orthoptics visual memory
11. Tachistoscopic procedures

(Compiled from Berne.[38])

REVIEW OF PREVIOUS WORK

Clinical Documentation

There are relatively few clinical reports in which vision therapy has been used to enhance oculomotor abilities in patients with TBI. An early paper by Doden and Bunge[37] indicated that the fusional range could be increased by training in 85% of such patients, but provided little detailed data and related information. Berne[38] reported on three young adult patients from his private practice. They typically complained of blurred vision, diplopia, and headaches, frequently in association with near work. In addition, they experienced reading difficulty, including inability to sustain concentration. All were given six months of one-hour per week in-office therapy consisting of standard optometric vision therapy/perceptual training procedures (eye movements, anti-suppression/fusion, gross motor skills, and perceptual enhancement) (see Table III). No home training was prescribed. Each patient reported marked reduction of symptoms that was correlated with clinical findings (see Table IV). Berne also noted that yoked prisms were especially helpful in some such patients, including one in his own study.

More recently, Hellerstein and Freed[3] reported on one young adult who had sustained TBI two-and-one-half years prior to their evaluation. Residual vision complaints included headache, diplopia, and blurred vision. Home- and office-based vision therapy was conducted over an eight-month period. It included techniques

Table IV
Oculomotor Improvements Following Vision Therapy for Head Trauma

1. Reduced exophoria at near
2. Closer nearpoint of convergence
3. Increased positive fusional range
4. Improved King-Devick saccade test
5. Concurrent elimination of diplopia and/or blur

(Compiled from Berne.[38])

to enhance monocular oculomotor skills, fusion, accommodation, and anti-suppression. Pre/post-vision therapy clinical findings are enumerated in Table V. Clearly, considerable gains were evident with respect to the vergence system, and this correlated with a reduction of symptoms.

And Kerkhoff and Stogerer[39] reported on two TBI patients in their hospital clinic (a third with cerebral vasculitis was also included). These two young adults were seen approximately six months after their TBI. Each had a severe reduction of fusional convergence. Their primary complaint was difficulty with reading. Fusional training was conducted in 50-minute sessions twice a week for six weeks. Training was done with the fusion trainer, loose prisms, and the cheiroscope, with the goal being to increase their fusional convergence ranges. There was a gradual increase in fusion ranges during the treatment phase (13pd in one patient and 4pd in the other). This treatment effect persisted in the two-month follow-up period. There was a concurrent improvement in subjective reading ability. The above clinical studies are consistent with two other recent reports on clinic patients with head trauma.[40,41]

Table V
Oculomotor Improvements Following Vision Therapy for Head Trauma

Test	Pre-VT	Post-VT
Distance cover test ranges	ortho	ortho
Distance vergence ranges		
BO (pd)	x/6/2	16/20/6
BI (pd)	x/12/6	x/10/6
Near cover test (pd)	10(X)T	8-10 exoph
Near vergence ranges		
BO (pd)	diplopia	x/14/3
BI (pd)	diplopia	x/24/17
Nearpoint of convergence (in.)	10/16	5/8
NRA	diplopia	+2 D
PRA	diplopia	-1 D

(Compiled from Hellerstein and Freed.[3])

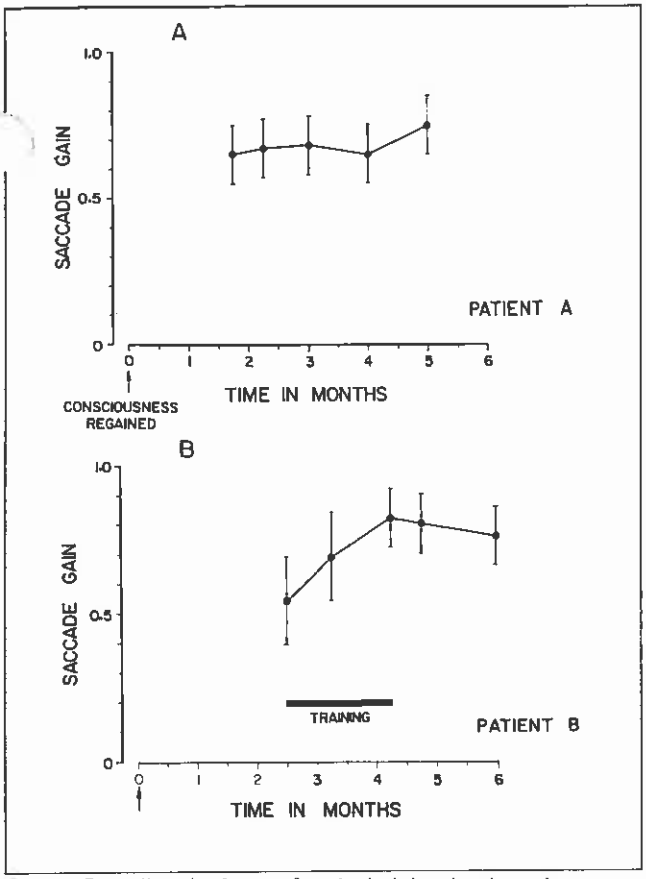

Figure 1. Saccadic gain changes for a brain-injured patient who did not receive training (A) and one who did (B). Each data point is the mean calculated from 15 eye movement responses; bars indicate 1 standard deviation. (Reprinted with permission of the publisher.[43])

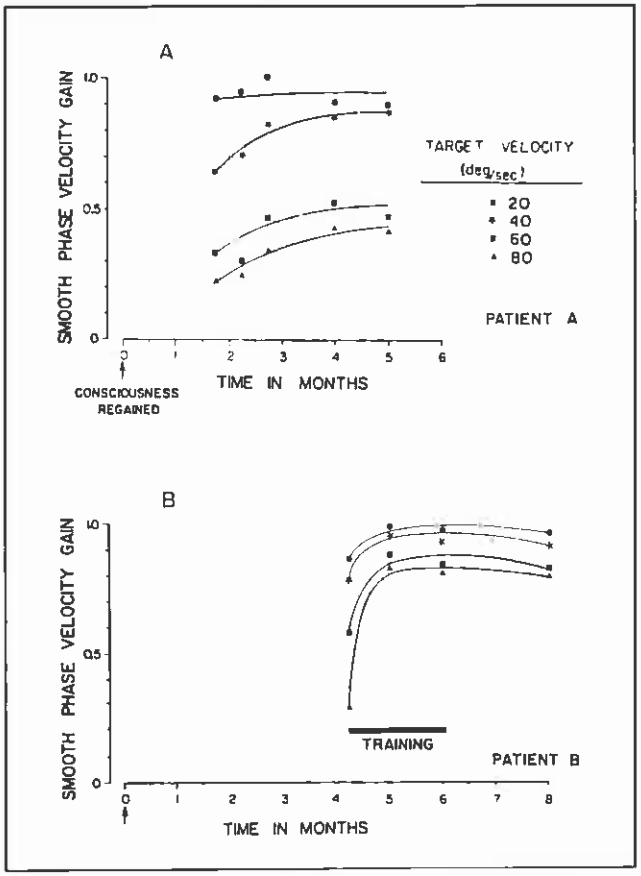

Figure 3. Slow-phase velocity gain of OKN for a brain-injured patient who did not receive training (A) and one who did (B). Each data point is the mean of 15 slow-phase velocities. The curves are calculated regressions for the mean. (Reprinted with permission of the publisher.[43])

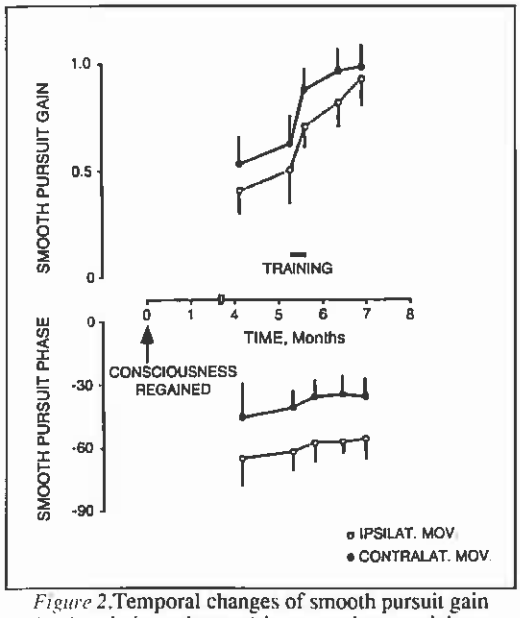

Figure 2. Temporal changes of smooth pursuit gain (top) and phase (bottom) in one patient receiving training. The target movement was 30 degrees at a frequency of 0.5 Hz. Each dot represents 15 measurements, and the bars are one standard deviation. (Reprinted with permission of the publisher.[44])

Objective Laboratory Documentation

Some of the earliest and perhaps most important investigations in this area were conducted by Ron and colleagues.[42-45] He was a neurophysiologist initially interested in brain lesions and neural plasticity in monkeys. Ron later worked in the area of human rehabilitation with emphasis on oculomotor control in patients with TBI. His early work[42] indicated that at least 50% of these patients had an oculomotor disturbance of some type. He therefore asked two critical questions: (1) What is the effect of simple eye movement training in these patients? and (2) Is there transfer of such training from one oculomotor subsystem to another? Ron used objective infrared recording techniques to monitor horizontal eye movements during the first few months following the regaining of consciousness after head trauma. Short periods of simple eye movement training (e.g., simple tracking of various controlled target movements, at times with the addition of auditory biofeedback) were interspersed during this period of natural recovery.[45]

The results for saccades are presented in Figure 1.

Saccadic gain (i.e., the ratio of initial saccadic amplitude to target amplitude for a sudden displacement or step change in horizontal target position) is plotted during this post-trauma period in two patients. Patient A shows the natural recovery function, whereas Patient B demonstrates the effects of natural recovery PLUS the addition of oculomotor training. Clearly, Patient A exhibited little natural recovery. Saccadic gain remained abnormally low throughout this period. In contrast, Patient B exhibited a rapid increase in saccadic gain and considerable normalization during the brief training period. There was subsequent post-training maintenance of the effect. The oculomotor training appeared to enhance both the rate and level of recovery.

The results for pursuit are presented in Figure 2. Clearly, again there was a rapid increase in gain (i.e., eye velocity divided by target velocity) during the very short training period. This continued to normal-

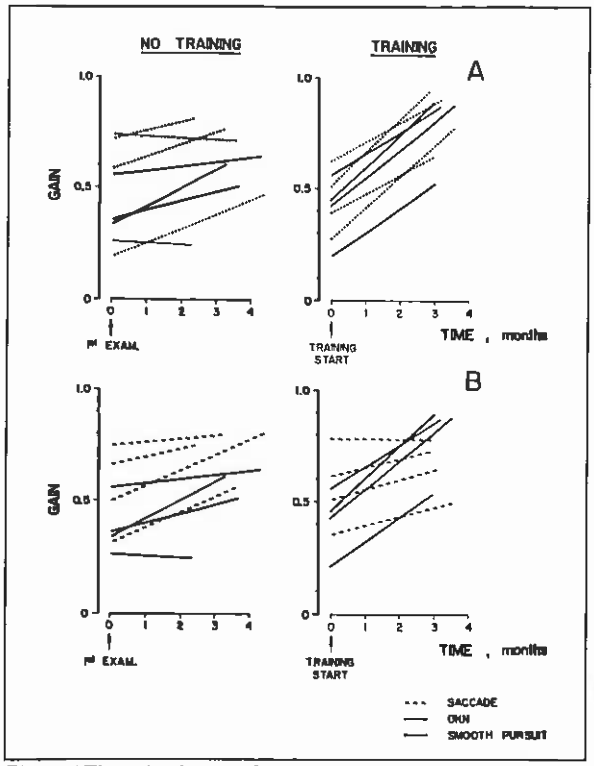

*Figure 4.*The gain change of contralateral saccades, ipsilateral OKN smooth phase, and smooth pursuit when the subjects were trained in optokinetic movements. Each straight line represents one patient through the follow-up period. Comparison of gain trend in the various patients between (A) saccades and OKN smooth phase, and (B) saccades and smooth pursuit. (Reprinted with permission of the publisher.[44])

ize even once the training was terminated. The results for pursuit phase (i.e., the amount of lag or lead of the eye, in degrees, with respect to the predictable sinusoidal target motion) were similar but less dramatic.

The training effects for optokinetic nystagmus are presented in Figure 3 for four different drum velocities. The results were similar to those found for the saccadic and pursuit subsystems described above.

And, lastly, Figure 4 shows Ron's results when the optokinetic subsystem was trained. The results clearly demonstrated a direct effect on the trained optokinetic subsystem, a transfer effect on the untrained saccadic subsystem, and little transfer effect on the untrained pursuit subsystem. The optokinetic-to-saccadic transfer suggested that neural structures common to these two subsystems were positively affected.

Table VI
Oculomotor Therapy Effects in Brain-Injured Patients (n = 22)

1. Faster rate of improvement	
saccades	4.5x
optokinetic nystagmus	3.0x
pursuit	2.5x
2. Higher level of improvement	
3. Some oculomotor subsystem transfer	

(Compiled from Ron.[44])

The key results of Ron's two studies have been summarized in Table VI.

With brief and simple eye movement training, (1) recovery time was shortened, (2) recovery magnitude was enhanced, and (3) some transfer effect was evident. These findings have important clinical ramifications. Clearly, to facilitate remediation and recovery to more functional levels, eye movement training is most efficacious. This is consistent with the general principle of motor rehabilitation training/therapy.[26] And, speedier rehabilitation (e.g., 2.5 to 4.5 times faster than natural recovery: see Table VI) allows the patient to reenter society at an earlier date, thus reducing long-term costs and enhancing self-esteem.

In a more recent case, objective eye movements were recorded before and after simple training of large saccades (eight months, one-hour per day) in a patient manifesting bilateral internuclear ophthalmoplegia after mild brain injury.[46] Conjugacy of saccades gradually occurred during the training period, although at least some improvement would be anticipated during the initial months of natural recovery.

CASE REPORT

History

GC, a 55-year-old black male, was referred by a rehabilitation physician (physiatrist) from the Department of Rehabilitation Medicine of a neighboring hospital. GC was formerly employed as a New York City housing police officer. He presented for a vision evaluation in the Head Trauma Vision Rehabilitation Unit of the SUNY/State College of Optometry, University Optometric Center on March 8, 1994. Case history was significant for blunt head trauma six-and-one-half years earlier. Blows to the occipital area were sustained with a heavy metal object, resulting in severe headache and partial paralysis of the right side of the body. An eight-day hospital stay was required. After being out of work for eight months, he attempted to return, but experienced another incident resulting in injury to the right parieto-temporal region. At that time, a 24-hour hospital stay was required. Since the second trauma, he experienced several episodes of syncope and was forced to cease working completely in March, 1992.

GC's symptoms included constant headaches, intermittent diplopia at distance and near, near blur after five minutes of reading, tearing when reading, occasional black-outs, nausea, and a feeling that the world was "spinning." Personal and family ocular histories were unremarkable. Medical history was significant for hypertension, which was controlled with Inderal. At the time of the initial evaluation, he was taking "pain medication" and Prozac. He had no known drug allergies. He expressed his goal: to maintain clear vision for longer periods of time when watching television and reading.

Examination Results

(Evaluation took place during the course of three visits on 3/8/94, 3/22/94, and 4/5/94, and pertinent findings are presented.)

Current Rx:
OD: + 1.25 - 0.50 X 078 + 2.00 Add
OS: + 1.75 - 0.25 X 162 + 2.00 Ad

Corrected VA (distance):
OD: 20/30
OS: 20/40
OU: 20/30

Corrected VA (near @ 16"):
OD: 20/40
OS: 20/35
OU: 20/35

Clinical Eye Movement Evaluation:
Fixation: obvious discomfort and grimacing with attempted steady gaze on a small detailed target for 10 seconds.
Pursuit: full OD and OS; full and concomitant OU; saccadic intrusions created jerky performance OD, OS and OU; patient complained of obvious discomfort with foreign body sensation on up and down gaze.
Saccades: obvious hypometria OD and OS, however slightly less OU; obvious discomfort during all phases of testing.

Refractive Findings:
Retinoscopy:
OD: + 1.00 - 0.75 X 030
OS: + 2.00 - 1.00 X 160
Subjective:
OD: + 1.00 - 0.50 X 030 OD 20
OS: + 1.50 - 0.25 X 160 OS 20/
OU 20/30

Binocular and Accommodative Findings: (Note that items [a] through [c] below were measured through the habitual Rx; all other findings were measured through the subjective.)

(a) Cover Test (Dist.): orthophoria
(b) Cover Test (Near): orthophoria
(c) Nearpoint of Convergence: 4" break/5" recovery
(d) Distance Phoria (Von Graefe Method):
Lateral: 1-1/2 pd exophoria;
Vertical: 1 pd right hyperphoria
(e) Near (i.e., add determination):
Plus Build Up:
OD: + 2.50 VA 20/25
OS: + 2.00 VA 20/25
NRA/PRA: + 0.75/- 0.75
(f) Near Phorometry and Vergence ranges (with indicated total near prescription):
Lateral: 7 pd exophoria:
Vertical: isophoria
BI: 16/28/10 pd
BO: 32/38/30 pd

Ocular Health Assessment:

(a) Anterior Segment Evaluation: Mild blepharitis OD and OS: mild superficial punctate keratitis superiorly OS: reduced and uneven tear break up time.
(b) Pupils: Equal, round, reactive to light, negative for afferent pupillary defect.
(c) Applanation Tonometry: (Goldmann, 11:45 AM)
OD 10 mm HG OS 8 mm HG
(d) Posterior Segment Evaluation: C/D ratio 0.3 each eye; slight narrowing of arterioles; macula and peripheral retina of each eye within normal limits.

Assessment:

1. Compound hyperopic astigmatism, OU.
2. Presbyopia.
3. Possible binocular instability.
4. Mild blepharitis and tear insufficiency.
5. Oculomotor dysfunction.

Plan:

1. Prescribe indicated bifocal correction. (The patient's current lenses were in poor condition.)
2. Prescribe NV correction for prolonged near tasks.
3. Return for complete binocular evaluation to assess ability to sustain bifixation/fusion at near.
Lid scrubs three times daily with artificial tears as needed (minimum four times daily) OU.
5. Return for automated visual field.
6. Return for objective eye movement evaluation.

Return Visit

GC returned for the recommended testing. Further binocular testing supported the diagnosis of binocular instability and the need for a vision therapy program. Automated visual field testing (Humphrey 3 Zone 120 point screening) revealed a "Swiss cheese" pattern of relative and absolute defects. Approximately 80% of the points were seen in each eye, with 15% recorded as relative defects and 5% as absolute defects in the absence of fixation losses. The areas of defect did not fit the pattern of either neurological or glaucomatous visual fields.

Pre-therapy Objective Eye Movements

Fixation and horizontal eye movements were first recorded objectively on 4/19/94, using the infrared limbal reflection technique.[32] This standard clinical testing system had a linearity of over 5 degrees, a resolution of at least 10 min arc, and a filtered bandwidth from DC to 70 Hz. All eye movements were analyzed directly on the strip chart recorder. The test room was dimly illuminated for all testing unless otherwise specified. To test fixation, the patient gazed at a small (~10 min arc) midline target positioned on the center of a display monitor placed 57 cm, away both monocularly and binocularly. Fixation was also tested in total darkness to the remembered target location. To test saccades, the target moved in a step manner either five degrees to the left or right of center with temporal randomization. To test pursuit, the target moved at various constant velocities (5-50 deg/sec) between these same two endpoints.

The objective eye movement recordings revealed several interesting and clinically-important findings[32] (see Figure 5).

Presence of frequent to-and-fro saccadic intrusions (~1 deg amplitude) and single saccades, with some increased drift, were evident during midline bifixation (see Figure 5A). Results were similar during monocular fixation. With attempted fixation in total darkness, saccadic intrusions were still present, suggesting their generation was not dependent on visual feedback. In addition, in total darkness the

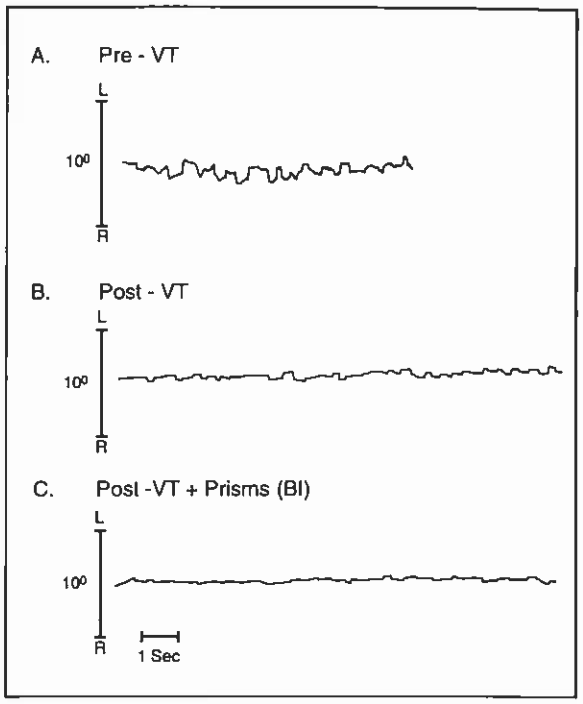

Figure 5.(A) Pre-therapy fixational eye movements of right eye. Post-therapy fixational eye movements of right eye without (B) and with (C) the 5.5 pd base-in prism incorporated into the spectacle correction in each eye. Binocular viewing in all cases.

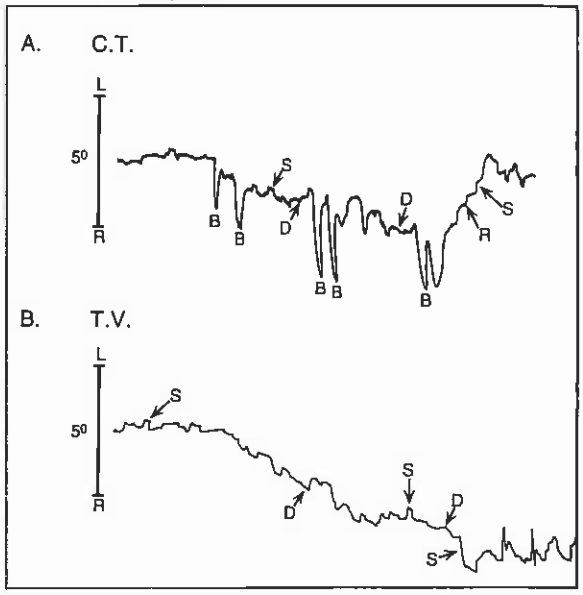

Figure 6.(A) Objective cover test. Right eye fuly occluded showing slow decay to exophoric position. Fellow eye remained fixating the target. B = blinks, D = drift, S = saccades, and R = fusional recovery. (B) Objective tonic vergence. Right eye only recorded and shown in figure. Note similar slow decay time course but to more divergent tonic vergence position. D = drift and S = saccades.

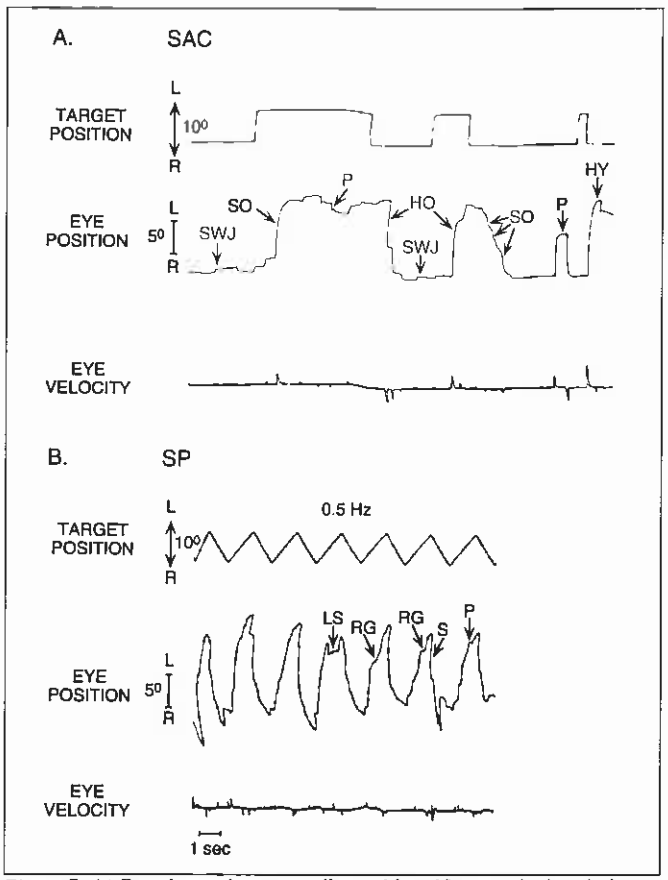

Figure 7.(A) Psuedo-random saccadic tracking. Note marked variation in performance, such as hypometria (HO), hypermetria (HY), slow and overlapping saccades (SO), square-wave jerks (SWJ), and prediction (P). (B) Pursuit to 0.5 cycle per second constant-velocity (i.e., "triangular" movement). Much variation present, such as large saccades (LS), reduced pursuit gain (RG), and prediction (P).

eye slowly drifted exponentially (~10 sec) to the divergent tonic vergence position of 14 pd exophoria (see Figure 6B). This was twice as large as the dissociated position (~7 sec exponential time course) found by the objective cover test (7 pd exophoria) (see Figure 6A), which was in agreement with the clinical phorometric estimate. This indicated that under degraded, dissociated conditions, the eye would shift to this more divergent tonic position. With regard to saccade and pursuit movements (Figure 7), there was considerable variability in both eye accuracy and velocity. Also, the patient was easily fatigued, resulting in a worsened performance, with concurrent grimacing and attempted retreat from the target.

Vision Therapy Program

GC commenced an in-office vision therapy program on 5/3/94 that was supplemented by home therapy.[47,48] Although simply prescribing a spectacle correction with compensatory prism would have been beneficial to GC, the treatment of choice was to build on the patient's existing binocularity by the vision therapy and then consider the necessity of prescribing prism. The one hour of weekly in-office vision therapy was performed over the course of five months for a total of 20 sessions. This was supplemented each day with several 15-minute periods of home therapy. The goals of therapy were to:

a. enhance fixation, saccadic, and pursuit abilities
b. eliminate suppression
c. enhance motor fusion
d. enhance sensory fusion

The 20 vision therapy sessions were divided into three phases. The first seven sessions used techniques involving monocular fixation, saccades and pursuit. The next six sessions stressed anti-suppression and flat fusion abilities. In the final seven sessions, different amounts of compensatory prism were used to determine the near and distance prescription, followed by more advanced binocular enhancement therapy.

During the first phase of therapy (sessions #1-7), monocular eye movements were trained, using techniques common to the general vision therapy practice. Saccades were trained, using the Hart Chart, Acuvision 1000,[a] and various computer-based saccadic programs. Pursuit was trained, using a moving flashlight, pegboard rotator, ball toss, and simple pen tip as different pursuit stimuli. Fixational ability was trained mainly at home by having the patient gaze steadily at a small object at 20 inches for increasing time intervals. Instructions were given to enhance visual and proprioceptive awareness.

The second phase of therapy (sessions #8-13) concentrated on anti-suppression and flat fusion techniques. The Brock String was introduced in the early weeks of training and found to be particularly beneficial during the second phase of therapy. As GC demonstrated an ability to perform Brock String techniques of increasing difficulty in the weekly therapy sessions, he would reinforce the skill at home. With progress, additional anti-suppression and fusion techniques using anaglyphic and polarized targets were added. For example, The GTVT Anti-Suppression Chart,[b] integrates saccadic eye movements with superimposition. These anaglyphic targets were easy and inexpensive to use for home training.

A progress evaluation was done after 13 visits. The patient's symptoms of intermittent diplopia and headaches had diminished substantially. GC exhibited a highly variable exophoria at near following sustained nearpoint tasks. At distance, he manifested an esophoria which increased during concentrated viewing.

The third and final phase of therapy (sessions #14-20) centered around enhancing GC's binocularity with a prismatic correction (see Discussion).

At near, the prismatic correction was probed, using the Keystone Ophthalmic Telebinocular and the Visual Skills Test Set.[c] Using Test #10 (lateral posture for use at nearpoint), increasing amounts of prism were placed in the auxiliary lens holder until GC attained the expected phoria range. This occurred with 6 pd Then Test #11 (fusion for use at nearpoint), was placed in the telebinocular. Six pd BI was used as a starting point to probe for a normal fusional response. The patient reported crossed or exo diplopia (four balls) with 6 pd BI. Increasing the prism amount by one prism diopter at a time resulted in decreasing exo diplopia, until 11 pd BI was reached. Here GC reported immediate fusion (three balls). The 11pd BI was then placed in a trial frame in conjunction with GC's near spectacle correction. GC reported considerable symptomatic relief. Measurement of vectogram ranges demonstrated adequate convergence and divergence with this amount of prism in place.

To determine the distance prism prescription, a modified Hess test was performed. GC was placed in a dimly lit room with red and green filters placed over his distance spectacles. The patient was given a flashlight covered by a red filter and asked to superimpose this light on the green light projected onto a distant wall by the examiner. Similar to the testing for the near prismatic prescription, increas amounts of prism were used to determ the least amount necessary to obtain a

Table VII
Oculomotor Improvements Following Vision Therapy for Head Trauma

1. Fixation: few and smaller saccadic intrusions
2. Saccades: more accurate, regular and appropriate
3. Pursuit: more accurate and regular

superimposition response. Five to seven prism diopters BO were required to compensate for GC's distance esophoria. In two separate trials, a total of either 5pd BO or 7pd BO were placed in front of GC's distance prescription. After reading the letters on the Hart Chart at 10 feet, GC was asked to compare his vision through the two prescriptions. The patient was most comfortable with the lower prism value, i.e., 5pd BO, stating that things looked "right" with them, similar to how things appeared through the prismatic correction for near. Objective and subjective results of compensatory prism lenses were confirmed at the follow-up sessions.

Ultimately two spectacle corrections were prescribed:

DV:	OD	+1.00 sph. c.w. 2.5pd BO
	OS	+1.00 sph. c.w. 2.5pd BO
NV:	OD	+3.25 sph. c.w. 5.5pd BI
	OS	+3.25 sph. c.w. 5.5pd BI

With the use of these spectacles, GC's final sessions included fusion training with vectographic targets, as well as binocular saccadic eye movement techniques.

Follow-up was performed one month after the cessation of training. GC reported that he could now read for at least one-half hour without experiencing headaches, diplopia, or losing his place. This was the first time he was able to do so since the accident six-and-a-half years earlier. Objective eye movement testing was performed at this follow-up visit.

Post-therapy Objective Eye Movements:

Objective eye movement recording with and without the newly prescribed prisms for near was performed at the conclusion of vision therapy on 9/19/94 (see Figure 5). Now habitual binocular fixation showed increased stability. There were fewer and smaller (0.5 deg amplitude) saccadic intrusions, and the drift was markedly reduced and rarely present (see Figure 5B). With inclusion of the 11 pd base-in prisms equally split between eyes at near, the overall stability increased even further. There were yet fewer and smaller (0.25 deg amplitude) saccadic intrusions, with little evidence of increased drift (see Figure 5C). This markedly improved post-therapy bifixation, especially with the prisms, correlated with the patient's more comfortable and considerably extended periods of nearwork. With regard to saccades and pursuit, there was slightly more consistent performance with respect to eye accuracy and velocity. It was now much more difficult to elicit fatigue effects on his eye movement performance, and there was also absence of facial grimacing and target retreat.

DISCUSSION

We have presented the case of an individual who had suffered TBI approximately six-and-one-half years prior to his initial encounter with us. Consequently, any subsequent subjective or objective improvements in vision function could no longer be ascribed to the natural recovery process. It is noteworthy that in spite of two hospitalizations and several eye examinations since the time of his initial injury, the previous eye care professionals were either unaware of GC's ocular and vision conditions, or felt that little could be done to remediate them and alleviate his symptoms. Thus, a significant period of time transpired before anyone considered the degree to which these residual conditions negatively impacted the patient. For example, he had always been an avid reader. Because the injury had caused him to lead a more sedentary life style, reading now became increasingly important to him despite being more difficult than ever to accomplish because of his severe near-point problems. Furthermore, GC had not suffered any cognitive disability and might have been able to return to the work force in a role other than his previous position. However, his lack of visual sustenance at near distances precluded a number of potential opportunities.

We provided full-scope optometric care to this patient. The conditions of mild blepharitis and keratitis were easily treated and resulted in increased general ocular comfort. Careful chair-side testing of fixation, pursuit, and saccadic abilities revealed dysfunctions that were corroborated by the objective eye movement recordings. In addition, although the usual optometric evaluation did not reveal a significant binocular dysfunction, both objective findings and a subsequent in-depth clinical evaluation clearly indicated a binocular instability that became manifest over time. This was in agreement with the patient's observation and provided the basis for a program of vision therapy that substantially benefited him.

The use of prisms alone would have provided some immediate diminution of symptoms. Nevertheless, our strategy was to institute vision therapy first, and then prescribe prisms after it was felt that the therapy had maximally enhanced the patient's binocular sustaining abilities. We have presented two methods for determining the amount of prism that have been found to be efficacious with this population for both distance and near. Furthermore, our experience with GC and a number of other TBI patients has been that there is little or no evidence of prism adaptation. Such lack of prism adaptation is typical in patients with symptoms related to binocular dysfunction[49] and, in fact, is the reason for their success with prism spectacles. We hypothesize that in many of these patients the slow vergence system, which is believed to be involved in the sustained bifixation,[50] has been damaged, perhaps involving the cerebellum.[51] Consequently, the prism is not adapted to or "absorbed"/"eaten up."[52] Therefore, the rationale behind our strategy of vision therapy preceding the prescribing of prisms was based on first maximally improving slow vergence system function. We were then able to determine the amount of prism that compensated for the difference between the level of functioning achieved by the patient and that required for sustained and comfortable, clear and single binocular vision.

We have also presented a growing body of both clinical and objective evidence that vision therapy can be beneficial to many TBI patients. This concords with our patient's outcome. There was significant objective improvement in fixation, and less dramatic improvement in our objective measures of pursuit and saccadic abilities. However, most striking was the patient's report of enhanced quality of life regarding reading, lessening of headaches, and becoming more active in his everyday affairs. These improvements were also noted by other members of his rehabilitative team, namely the physiatrist, psychologist, and occupational therapist. Indeed, the amelioration of GC's ocular and vision conditions allowed him to respond more favorably to his total rehabilitative regimen.

The vision rehabilitation of patients with TBI represents a new and exciting area for optometry. Proper care of such patients demands knowledge and facility in ALL areas of optometry, in particular vision therapy, refraction, and disease detection and treatment. This is clearly demonstrated by our detailed case report. The provision of full-scope optometric care to this underserved and poorly understood clinic population offers a unique and altruistic opportunity for the profession. By such a comprehensive approach, in conjunction with other members of the rehabilitative team,[53-55] patients with TBI can more rapidly reenter society and the work force, and once again be productive members of the community.

Sources

a. International Acu Vision Systems, Inc., 4425 Bayard St., Suite 230, San Diego, CA 92109.
b. GTVT, 2100 196th Street SW, Suite 116, Lynnwood, WA 98036
c. Mast/Keystone, Inc., 4673 Aircenter Circle, Reno, NV 89502

References

1. Cohen AH, Soden R. An optometric approach to the rehabilitation of the stroke patient. J Am Optom Assoc 1981; 52: 795-800.
2. Gianutsos R, Ramsey G, Perlin R. Rehabilitative optometric services for survivors of acquired brain injury. Arch Phys Med Rehabil 1988; 69: 573-78.
3. Hellerstein LF, Freed S. Rehabilitative optometric management of a traumatic brain injury patient. J Behav Optom 1994; 5: 143-148.
4. Hellerstein LF. Vision profile of patients with mild brain injury. J Am Optom Assoc 1995; 66: 634-639.
5. Swanson M. Neuro-rehabilitation. Eye Quest Mag, Jan/Feb 1995; pp. 50-55, 85.
6. Baker RS, Epstein AD. Ocular motor abnormalities from head trauma. Survey Ophthal 1991; 35: 245-267.
7. Trobe JD, Lorber ML, Schlezinger NS. Isolated homonymous hemianopia. Arch Ophthal 1973; 89: 377-381.
8. Teuber HL, Battersby W, Bender MB. Visual field defects after penetrating missle wounds of the brain. Cambridge, MA: MIT Press, 1960.
9. Mitchell R, Macfarlane A, Cornell E. Ocular motility disorders following head injury. Austral Orthopt J 1983; 20: 31-36.
10. Fraco RF, Fells P. Ocular motility problems following road traffic accidents. Br Orthop J 1989; 46: 40-48.
11. Cohen M, Grosswasser Z, Barchadski R, Appel A. Convergence insufficiency in brain-injured patients. Brain Injury, 1989; 3: 187-191.
12. Richard R. Ocular motility disturbances following trauma. Adv Ophthal Plastic Reconstruct Surg 1988; 7: 133-147.
13. Davies CE. Etiology and management of convergence insufficiency. Am Orthop J 1956; 6: 124-127.
14. Carroll RP, Seaber JH. Acute loss of fusional convergence following head injury. Am Orthop J 1974; 24: 57-59.
15. Krohel GB, Kristan RW, Simon JW, Barrows NA. Post-traumatic convergence insufficiency. Ann Ophthalmol 1986; 18: 101-104.
16. Krasnow DJ. Fusional convergence loss following head trauma: a case report. Opt Mtly, 1982; 73: 118-119.
17. Burke JP, Orton HP, West J, Strachan IM, Hockey MS, Ferguson DG. Whiplash and its effect on the visual system. Graefe's Arch Clin Exp Ophthalmol 1992; 230: 335-339.
18. Stanworth A. Defects of ocular movement and fusion after head injury. Brit J Orthop 1974; 58: 266-271.
19. Rutkowski PC, Burian HM. Divergence paralysis following head trauma. Am J Ophthalmol 1972; 73: 660-662.
20. Pratt-Johnson JA. Central disruption of fusional amplitude. Brit J Ophthalmol 1973; 57: 347-350.
21. Wade SL. Loss of fusion following brain damage. Brit Orthop J 1965; 22: 81-83.
22. Hart CT. Disturbances of fusion following head injury. Proc Roy Soc Med 1969; 62: 704-706.
23. London R, Scott SH. Sensory fusion disruption syndrome. J Am Optom Assoc 1987; 58: 544-546.
24. Tenjin S. Study on fusional convergence. Folia Ophthalmologica Japonica, 1981; 32: 800-808.
25. Berrol S. Cranial nerve dysfunction. In: Physical medicine and rehabilitation; traumatic brain injury. Horn LJ, Cope DN, (eds). Philadelphia: Hanley and Belfus, Inc., 1989.
26. Liu D. Post-traumatic visual disorders: Part II. Management and rehabilitation. In: Physical medicine and rehabilitation; neurologic and orthopaedic sequelea of traumatic brain injury. Stone LR (ed). Philadelphia, 1989: Hanley and Belfus, Inc.
27. Mazow ML, Tang R. Strabismus associated with head and facial trauma. Am Orthop J 1982; 32: 31-35.
28. Harrison RJ. Loss of fusional vergence with partial loss of accommodative convergence and accommodation following head injury. Binoc Vis 1987; 2: 93-100.
29. Roca PD. Ocular manifestations of whiplash injury. Annals Ophthalmol 1972; 4: 63-73.
30. Zihl J, Werth R. Contributions to the study of "blindsight." II. The role of specific practice for saccadic localization in patients with postgeniculate visual field defects. Neuro-psychol 1984; 22: 13-22.
31. Birnbaum MH. Optometric management of nearpoint disorders. Boston: Butterworth-Heineman, 1993.
32. Ciuffreda KJ, Tannen B. Eye movement basics for the clinician. St. Louis: Mosby, 1995.
33. Ciuffreda KJ. Reading eye movements in patients with oculomotor disturbances. In: Eye movements in reading. Ugge J, Lennerstrand G. (eds). New York: Pergamon Press, 1994.
34. Goodwin JA. Eye signs in neurologic diagnosis. In: Neurology for the non-neurologist, 2nd ed. Weiner WJ, Goetz CG (eds). Philadelphia: Lippincott Co., 1989.
35. Leigh RJ, Zee DS. The neurology of eye movements, 2nd ed. Philadelphia: F.A. Davis, 1991.
36. Gianutosos R, Perlin R, Mazerolle KA, Trem N. Rehabilitative optometric services for persons emerging from coma. J Head Trauma Rehab 1989; 4: 27-25.
37. Doden W, Bunge H. Fusionsstorungen nach schadelhirntraumen. Monatsblatter fur Augenheilkunde, 1965; 146: 845-853.
38. Berne SA. Visual therapy for the traumatic brain-injured. J Optom Vis Dev 1990; 21: 13-16.
39. Kerkhoff G, Stogerer E. Recovery of fusional convergence after systematic practice. Brain Injury 1994; 8: 23-26.
40. Schlageter B, Gray B, Hall K, Shaw R, Sammet R. Incidence and treatment of visual dysfunction in traumatic brain injury. Brain Injury 1993; 7: 439-448.
41. Suter PS. Rehabilitation and management of visual dysfunction following traumatic brain injury. In: Traumatic brain injury; rehabilitation. Ashley MJ. Krych DK (eds). Boca Raton, Florida: CRC Press, 1995.
42. Ron S, Najenson T, Hary D, Pryworkin W. Eye movements in brain damaged patients. Scand J Rehab Med 1978; 10: 39-44.
43. Ron S. Plastic changes in eye movements in patients with traumatic brain injury. In: Progress in oculomotor research. Fuchs AF, Becker W (eds). Elsevier North Holland, 1981.
44. Ron S. Can training be transferred from one oculomotor system to another? In: Physiological and pathological aspects of eye movements. Roucoux A, Crommelinck M (eds). London: Dr. W. Junk Publishers, 1982.
45. Gur S, Ron S. Training in oculomotor tracking: occupational health aspects. Isr J Med Sci 1992; 28: 622-628.
46. Mueller C, Koch S, Toifl K. Transient bilateral internuclear ophthalmoplegia after minor head trauma. Dev Med Child Neurol 1993; 35: 163-166.
47. Cotter SA. Clinical uses of prisms: a spectrum of applications. St. Louis: Mosby Yearbook, Inc., 1995: 97-105.
48. Griffin JR. Binocular anomalies: procedures for vision therapy, 2nd Edition. Chicago: Professional Press, 1982: 21.
49. North R,. Henson DB. Adaptation to prism-induced heterophoria in subjects with abnormal binocular vision or asthenopia. Am J Optom Physiol Opt 1981; 38: 746-752.
50. Schor C. Influence of accommodative and vergence adaptation on binocular motor disorders. Am J Optom Physiol Opt 1988; 65: 464-475.
51. Milder DG, Reinecke RD. Phoria adaptation to prisms. Arch Neurol 1983; 49: 339-342.
52. Campos EC, Catellani T. Further evidence for the fusional nature of the compensation (or "eating up") of the prisms in concomitant strabismus. Internat Ophthal 1978; 1: 57-62.
53. Cohen A, Rein LD. The effect of head trauma on vision: The doctor of optometry as a member of the rehabilitation team. J Am Optom Assoc 1992 530-536.
54. Suchoff IB. A rationale and clinical model for collaboration between optometry and occupational therapy. J Behavior Optom 1995; 6: 142.
55. Zost MG. Dignosis and management of visual dysfunction in cerebral injury. In:Diagnosis and management of special populations. Maino DM. (ed) St. Louis:Mosby Yearbook, 1995.

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