Oculomotor Rehabilitation in Acquired Brain Injury: A Case Series

Neera Kapoor, OD, MS, Kenneth J. Ciuffreda, OD, PhD, Ying Han, MD, PhD

ABSTRACT. Kapoor N, Ciuffreda KJ, Han Y. Oculomotor rehabilitation in acquired brain injury: a case series. Arch Phys Med Rehabil 2004;85:1667-78.

Objective: To investigate the effects of systematic, oculomotor rehabilitation on basic versional ocular motility, as well as reading eye movements, in subjects with acquired brain injury, using objective eye movement recording and subjective rating of reading ability.

Design: Case series.

Setting: Clinical research laboratory.

Participants: Two men with acquired brain injury: one with traumatic brain injury and one with stroke.

Interventions: Versional oculomotor training was performed for 1 hour, twice weekly for 8 weeks. There were 2 feedback modes of training: normal internal oculomotor visual feedback alone (4wk), or that feedback in conjunction with external oculomotor auditory feedback (4wk). Testing was conducted before and after training.

Main Outcome Measures: Objective outcome measures included both basic eye movement parameters (fixational accuracy, saccadic gain and latency, pursuit gain, mean saccade frequency ratio for simulated reading), and reading eye movement parameters (words per minute, grade level equivalent, fixations per 100 words, regressions per 100 words, percentage of reading comprehension, duration of fixation in seconds). Subjective outcome measures included the subject's ability to read based on the responses to the reading rating-scale questionnaire.

Results: Both subjects improved objectively in terms of basic versional oculomotor accuracy and reading ability. These findings concurred with their subjective impressions.

Conclusions: This case series provides objective documentation of the positive effects of oculomotor rehabilitation on basic ocular motility and reading ability in selected cases with acquired brain injury, thus suggesting the need for a larger clinical trial in this area.

Key Words: Attention; Brain injuries; Ocular motility disorder; Reading; Stroke; Rehabilitation.

From the Departments of Clinical Science (Kapoor) and Vision Sciences (Ciuffreda, Han), SUNY/State College of Optometry, Raymond J. Greenwald Rehabilitation Center, New York, NY.

Presented in part to the American Academy of Optometry, December 2002, San Diego, CA, and the Association for Research in Vision and Ophthalmology, May 2003, Fort Lauderdale, FL.

Supported by the Jacob and Valeria Langeloth Foundation (project no. 177).

No commercial party having a direct financial interest in the results of the research supporting this article has or will confer a benefit on the author(s) or on any organization with which the author(s) is/are associated.

Reprint requests to Neera Kapoor, OD, MS, Dept of Clinical Science, SUNY/State College of Optometry, Raymond J. Greenwald Rehabilitation Center, 33 W 42nd St, New York, NY, 10036, e-mail: *nkapoor@sunyopt.edu.*

0003-9993/04/8510-8675$30.00/0

doi:10.1016/j.apmr.2003.12.044

ACQUIRED BRAIN INJURY refers to sudden neurologic changes secondary to head trauma, cerebrovascular accident, or postsurgical complications. Patients with acquired brain injury manifest a spectrum of sensory, motor, cognitive, language, and perceptual deficits, many of which are amenable to rehabilitation.[1-5] The natural recovery from an acquired brain injury can be up to 1 year postinjury and incomplete,[6] with many patients eventually being left with multiple residual deficits,[7,8] including reading and visual scanning dysfunctions relating to the oculomotor dysfunction.[7-12] These residual deficits, including vision deficits, may adversely affect their rehabilitation, as well as other avocational and vocational goals.[2,7,10,12-15]

Approximately 8 million head injuries occur annually in the United States, of which 1.5 million are classified as "major" traumatic brain injury (TBI).[16] Approximately 50% of these 1.5 million people do not return to the work force, and the annual cost due to lost productivity is estimated at $4 billion.[17,18] In 1985, the lost earning potential combined with the cost of health care for TBI was estimated at $37.8 billion.[19]

Stroke is the leading cause of chronic disability in the United States. It is the third leading cause of death in the adult population of the United States.[20,21] Each year, approximately 700,000 people, 70% of whom are over the age of 65 years, suffer a stroke. With our ever-improving health care, with respect to preventive, acute, and chronic aspects, more of these people will survive and require extensive and long-term (ie, months and even years) medical and rehabilitative care. The annual combined cost of health care and lost productivity due to stroke is estimated to be between $30 and $40 billion.[21]

Oculomotor Deficits

The prevalence of oculomotor deficits in the acquired brain injury population ranges from 40% to 85%, depending on the literature source.[22-27] Basic oculomotor deficits may include jerk and pendular nystagmus, increased fixational drift, saccadic dysmetria, increased saccadic latency, and reduced pursuit gain, as well as others.[26] These oculomotor signs translate into symptoms while reading resulting in loss of place,[7] reduced speed,[7] and the sensation of visual motion.[28] Because oculomotor dysfunctions are such a common deficit in patients with acquired brain injury,[11,12,17,22-27,29] we designed a computer-controlled, objective, rehabilitative regimen for remediation of oculomotor deficits tailored to this patient population,[30] and then investigated its efficacy and impact on basic, as well as reading, ocular motility.

There are relatively few studies[9,27,31-35] using well-designed training programs and objective assessments of oculomotor performance related to oculomotor rehabilitation in individuals with acquired brain injury. Regarding binocular fixation and associated vergence stability, only 1 detailed case study[9] of a patient with TBI has been reported. That patient's binocular horizontal fixation improved after 3 months of conventional optometric vision therapy that included oculomotor and accommodative components. The patient's fixational stability further improved after the application of base-in prisms in the near-vision spectacle correction, because the prisms optically reduced the near vergence demand.[9]

With regard to basic versional eye movements, Ron et al[31,32] demonstrated that eye movements could be trained and improved, with some degree of transfer from 1 oculomotor subsystem to another, in the TBI population. Further, the oculomotor recovery time was 3-fold faster in those who received formal training versus those who did not.[32] However, details of the training programs featuring only normal internal visual feedback, stimulus characteristics, and results were not provided. Statistical analyses also were not performed on the group data. And, in 1 study,[31] successful results were obtained for normal, internal, visual feedback in conjunction with external oculomotor auditory feedback training, but with no comparison to normal visual feedback and, again, with few details provided.[32] In a short case report,[33] horizontal saccadic accuracy improved in a child with TBI after several weeks of simple, home-based, large-amplitude saccadic training.

Zihl[27,34,35] has conducted considerable research on reading-related eye movements, but only with stroke patients, typically persons with hemianopia. He used specific horizontal and oblique saccadic training with normal internal visual feedback alone, which was directed at modifying the oculomotor patterns (ie, saccadic adaptation[36]) to place an entire word rather than just a fragment within the residual functional hemifield. With several sessions per week, reading ability improved in these patients within a few weeks. Our research group has reported similar positive results using oculomotor training in both basic versional and simulated reading programs in both TBI and stroke populations.[37]

In the present article, we describe 2 cases in detail, 1 with TBI and 1 with stroke, in which basic versional and reading eye movements were successfully trained as part of a larger investigation in this area.

METHODS

Clinical Assessment

As part of a larger study investigating the effects of oculomotor rehabilitation on basic and reading eye movements using objective eye movement recordings, all procedures and all data followed the guidelines and ethical standards of the SUNY Optometry's institutional review board for human subjects. Both subjects in this article were referred to us by medical practitioners, after their medical conditions stabilized. Referral was for the optometric evaluation of any vision involvement in their reading difficulties after acquired brain injury. They presented at their initial visit to us with complete medical documentation and information regarding the diagnosis of the acquired brain injury, care received thus far (acute, subacute, chronic), neuroimaging results (including type, site, and side of lesion), neurologic reports, trauma history, and rehabilitative history. Subsequent to review of the subjects' documentation and histories, both received a detailed vision examination in the Raymond J. Greenwald Rehabilitation Center's Head Trauma Vision Rehabilitation Unit at SUNY Optometry. The optometric evaluation[38] included refractive assessment at distance and near, binocular sensorimotor assessment at distance and near, clinical and laboratory oculomotor assessment, and ocular health assessment (including confrontation visual field testing, biomicroscopy, applanation tonometry, pupillary testing, dilated fundus examination, automated visual fields). The clinical binocular assessment, with the appropriate refractive correction in place, included the following tests[38]: cover test at distance and near, near point of convergence, heterophoria test at distance and near, vergence ranges at distance and near, versional testing at near (ie, fixation, saccades, pursuit), and the Developmental Eye Movement[39] (DEM) test (ie, global saccadic visual scanning eye movement test at near). The DEM test involves the sequential search for numbers, which is scored by recording the following parameters for each of 3 subtests: ability to complete the task, ratio of the number of seconds to complete the task for the horizontal versus the vertical scanning subtests, and number of errors (ie, omissions, repetitions, or substitutions).

Case 1: TBI

The subject with TBI, a man in his early forties, suffered a mild brain injury after a motor vehicle crash in March 2000. He was driving 20 miles per hour when he was struck on the driver's side of his car by an oncoming vehicle driving at approximately 50 miles per hour. The left side of his head struck the window, and he subsequently lost consciousness intermittently for 1 hour. Subsequent to the collision, he had a radiograph, a computed tomography scan of the head and neck, and magnetic resonance imaging (MRI) of the head. The results of the neuroimaging tests and radiographs were negative. However, he still reported intermittent diplopia, impaired short-term visual memory, reduced reading rate, and loss of place while reading. On a "good" day, he reported being able to read comfortably and accurately for 1 to 5 minutes consecutively, while on a "bad" day he was unable to read for longer than 1 minute at a time, if at all. His personal and familial systemic and ocular health was otherwise unremarkable. During the study, he was taking gabapentin for pain management and citalopram for depression, with no known allergies to medications.

The clinical optometric findings revealed that he manifested moderate myopia, with best corrected distance and near visual acuities of 20/25 OD (right eye), 20/25 OS (left eye), and 20/20 OU (both eyes). His current spectacle and contact lens corrections were not altered. Distance and near cover tests showed no evidence of strabismus in primary gaze. Versional extraocular motility testing revealed no evidence of nystagmus, noncomitancy, restriction, or limitation of ocular motility in any position of gaze. However, heterophoria and compensatory fusional vergence measures revealed mild esophoria with poor compensatory fusional divergence in primary gaze at both distance and near. Versional eye movement testing and the DEM test[39] demonstrated moderate deficits of saccades. Confrontation visual field testing with both single and double stimulus presentation was unremarkable. Automated perimetry[a] (30-2 SITA standard analysis) revealed small areas of scattered, low-density, relative visual field defects with no evidence of lateralization for either eye. Pupils were equal, round, and reactive to light, with no evidence of a relative afferent defect. Biomicroscopy revealed clear media, open angles, and no evidence of inflammation in either the anterior or posterior chambers. Intraocular pressure, measured with Goldmann applanation tonometry, was 14mmHg OD and 15mmHg OS. Posterior segment evaluation, with dilation, was unremarkable for either eye.

Case 2: Stroke

The stroke subject, a man in his early fifties, suffered a lesion in the left occipital lobe in September 2000, as revealed by MRI. Consequently, he presented with a right homonymous hemianopia and macular sparing. His vision symptoms included slowness of reading, difficulty with numbers (as one would use for billing and accounts), short span of visual attention, impaired short-term visual memory, difficulty with directions, and mild expressive aphasia. On a "good" day, he reported being able to read comfortably and accurately for 10 to 15 minutes consecutively, while on a "bad" day he was only able to read for 1 to 5 minutes at a time. During the time of the study, his personal systemic and ocular health history was positive for borderline hypertension while his familial health history was unremarkable. There was no evidence of hemiparesis or physical limitation during the time of the study. He was taking warfarin sodium for anticoagulation, with no known allergies to medications.

The clinical optometric findings revealed low hyperopia, astigmatism, and presbyopia. His best corrected distance and near visual acuities were 20/20 OD, 20/20 OS, and 20/20 OU. His current bifocal spectacle correction was not altered. Distance and near cover tests showed no evidence of strabismus in primary gaze. Versional extraocular motility testing revealed no nystagmus, noncomitancy, restriction, or limitation of movement in any position of gaze. Heterophoria and compensatory fusional vergence measures, along with near point of convergence testing, revealed a mild convergence insufficiency in primary gaze. Versional extraocular motility testing and the DEM test[39] demonstrated evidence of moderate deficits of saccades. Confrontation visual field testing with single and double stimulus presentation, and automated perimetry (30-2 SITA standard analysis) revealed a homonymous, right hemianopia with visual neglect. Pupils were equal, round, and reactive to light, with no evidence of a relative afferent defect. Biomicroscopy revealed clear corneas and conjunctiva, open angles, no evidence of inflammation in either the anterior or posterior chambers. Intraocular pressure, measured with Goldmann applanation tonometry, was 18mmHg OD and 17mmHg OS. Posterior segment evaluation, with dilation, was unremarkable for either eye.

Oculomotor Assessment and Training

Each subject underwent objective versional eye movement testing, which included basic eye movements (fixation, saccades, pursuit), reading-related eye movements (single-line and multiple-line simulated reading), and Visagraph reading eye movement assessments. Instruments used for the objective oculomotor testing and subsequent oculomotor training are described in detail in the following section. After the objective oculomotor assessments, each subject received computer-based, oculomotor training twice weekly for an 8-week period. Each training session was 60 minutes in duration, with 36 minutes of actual eye movement training, with rest periods interspersed. At the end of the 8-week training period, objective ocular motility testing was repeated.

In addition to objective assessment of oculomotor function, each subject completed the reading-related questionnaire with their subjective assessment of reading ability before and after the training period. This reading-related questionnaire was used on a series of subjects as part of a larger study on oculomotor assessment of patients with acquired brain injury.[30] The questionnaire consisted of 5 questions that are similar to those posed during routine optometric evaluations, such as how long can one read comfortably. Responses ranged from 1 to 5, corresponding to 0 to 5 minutes, 5 to 10 minutes, 10 to 15 minutes, 15 to 30 minutes, and up to more than 30 minutes, respectively. Additional questions included how the person rates his/her overall reading comprehension, attention when reading in an environment with minimal external sensory stimuli, and attention when reading in an environment with multiple external sensory stimuli. These 3 questions were rated from 1 to 5, corresponding to poor, fair, good, very good, and up to excellent, respectively. Last, the subjects were to describe their reading strategy. This last question presented with 4 possible answers ranging from 1 to 4, corresponding to impulsive and inaccurate, impulsive and accurate, deliberate and inaccurate, and deliberate and accurate, respectively.

Possible scores ranged from 1 to 5, except for the fifth question for which scores ranged from 1 to 4, with the overall optimal maximum score being 24. Higher numbers on the reading-related questionnaire indicated better performance. Fractional values were permitted as responses for the questionnaire. This questionnaire was tested and found to be reliable and valid in a group (N=20) of asymptomatic young adults (age, 24–40y).

Instrumentation and Test Stimuli

For both subjects, we used computer-based protocols to obtain objective eye movement recordings. The protocols combined objective eye movement recording techniques with computer-based stimulus generation, stimulus presentation, and data analysis of basic horizontal and vertical versional eye movements (fixation, saccades, pursuit), as well as reading-related eye movements using simulated single and multiple line dynamic arrays.

In all paradigms, the target consisted of a 0.5°, bright luminous square presented on an otherwise dark, high-resolution, computer monitor (12.5-in screen) positioned 40cm from the subject along the midline. Testing was conducted under binocular viewing conditions with the appropriate refractive correction in place.

Objective assessment of basic binocular horizontal and vertical versional eye movements was performed with the OBER2[b] recording system.[40] The standard OBER2 system consists of a goggle-mounted, infrared limbal reflection eye movement system having a resolution of at least .25°, bandwidth equal to direct current to 120Hz, and a linear range of ±20° horizontally and vertically. Auditory oculomotor feedback hardware related to horizontal and vertical eye position changes was customized and integrated into the standard OBER2 eye movement system, wherein voltage from the eye movement system related to eye position was converted to a correlated tone. We tested 3 types of basic versional ocular motility[30,41]: fixation, nonpredictable and predictable saccades, and simulated reading.

Fixation. The target was presented in 5 positions (0 or midline, ±10° horizontally and vertically), each viewed for a 30-second test duration. The parameter used for analysis before and after training was eye position error measured horizontally, vertically, and radially (ie, the frequency of each magnitude of eye position error summed across all directions).

Saccades: nonpredictable. Within a range of either ±5° or ±10° relative to the midline, the test target was rapidly shifted stepwise with both temporal (ie, timed sequence) and spatial (ie, positional change) randomization for a 30-second test duration, each separately for horizontal and vertical saccades. The parameter used for analysis before and after training for nonpredictable saccades was saccadic latency (ie, reaction time).

Saccades: predictable. Using the same range and directions as for nonpredictable saccades, the target changed position at a constant frequency of .33 cycles per second. The parameter used for analysis before and after training for predictable saccades was saccadic gain (ie, initial saccade magnitude divided by target step amplitude).

Pursuit. With amplitudes of either ±5° or ±10° relative to the midline, the test target moved smoothly at a constant velocity of either 4.1° or 5.4° per second for the 60-second test duration. This was performed with the test target moving either horizontally or vertically. The parameter used for analysis

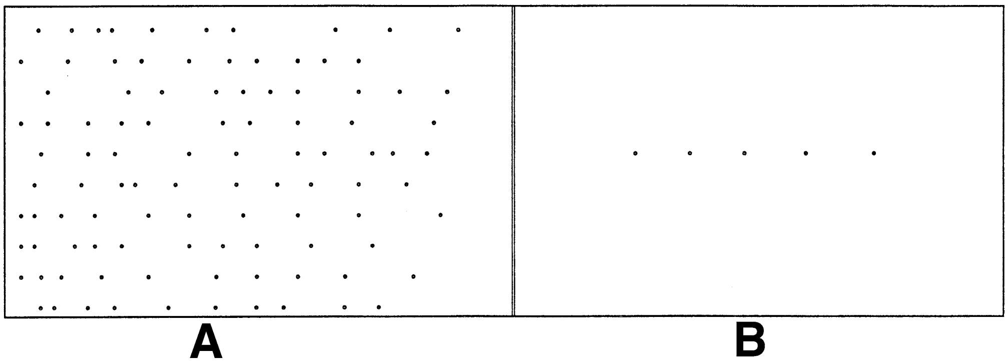

Fig 1. Simulated reading patterns. (A) Multiple-line stimulus pattern. (B) Single-line stimulus pattern.

before and after training was pursuit gain (ie, eye velocity divided by target velocity).

Simulated reading. Two simulated reading patterns were used: a multiple-line (fig 1A) and a single-line (fig 1B) paradigm. The parameters used for analysis before and after training were the mean saccade frequency ratio (ie, the total number of saccades executed divided by the total number of target step displacements; a ratio of 1.0 is ideal) for each stimulus pattern.

The multiple-line, simulated reading test paradigm (see fig 1A) incorporated a randomly spaced (1°–3° step displacements) moving test target in place of actual text equivalent to a 100-word paragraph. The test target moved stepwise every 3 seconds and moved sequentially (ie, from left to right) to positions on the screen as depicted in figure 1A, starting in the upper left corner and shifting across to the right for each simulated "line," then stepped back to the left-most position on the next line and continued to shift sequentially in this manner for a total of 120 seconds. Each of the 12 rows has 10 dot positions. The simulated text subtended 34° horizontally.

In the single-line, simulated reading test (see fig 1B), the target moved stepwise (3.25°) every 3 seconds and sequentially left-to-right to the 5 positions on the screen, stepped back to the left-most position, and then repeated the cycle 5 times for a total of 60 seconds. The simulated "line" subtended 13° horizontally.

In both cases, subjects were instructed to follow the moving target accurately with a single saccade, as one would do during normal reading. This protocol allowed sequential, rhythmic reading eye movement patterns to be trained and reinforced without the cognitive, perceptual, and language load that occurs during actual reading.[42,43] Hence, both simulated reading tests mimic pure oculomotor training tasks. The eye movements tested relate directly to the motor-based aspects of the reading process, yet the tests are free of cognitive load and text information processing.

Visagraph Measurements

Reading eye movements (horizontal position of both eyes) were recorded objectively using the commercially available Visagraph II[c] device,[43,44] which consists of 4 components: (1) an infrared, limbal reflection, horizontal eye movement recording system (resolution, .25°; bandwidth, direct current to 50Hz; linear range, ±20°); (2) hard copy of the test text at 10 graded reading levels (grades 2 to high school), with ten 100-word, standardized paragraphs per level; (3) computer software for automated analysis of the eye movements and subsequent display of the grade-related oculomotor-based performance profiles for each basic reading eye movement parameter,[44] including reading rate, overall reading efficiency, comprehension, span of recognition, duration of fixation, number of progressive and regressive fixations, and number of saccades per line; and (4) hard copy of the reading eye movements along with the tabulated grade-normative oculomotor performance levels. Subjects received 1 practice trial before actual testing, in which they read 2 paragraphs, and the better performance was recorded using the adult level-10 reading material. Analysis of the basic eye movement parameters described above was performed before and after training.

Oculomotor Training

The objective eye movement recording system was identical to that described in the sections above. There were 2 training modes: visual feedback and visual plus auditory feedback (V+A), with the only procedural difference in the training being the nature of the feedback. The auditory oculomotor feedback for V+A incorporated an audible tonal change that related to the subject's eye position.[30,43] The auditory tone generator has a position-to-tone resolution of .25° both horizontally and vertically, and a tonal frequency range from 2000 to 5000Hz. At the initial training session, which introduced both visual and V+A modes, subjects received a verbal description of the tonal expectations for optimal ocular motor responses.[30,43]

The tasks for the 2 training modes were similar to those for the test stimuli (ie, fixation, saccades, pursuit, simulated reading), except for the duration of stimulus presentation.[30] Eye movements were trained for a total of 36 minutes per training session, with interspersed rest periods to prevent subject fatigue. Rest periods of 20 to 30 seconds were provided at regular intervals after every training task (ie, after 60 seconds of saccades, there would be a 20- to 30-second rest period while the next subroutine was loading). Longer rest periods (ie, 2–3 minutes) occurred on demand if needed by the subject. Thus, the total time for the training session, including training and rest periods, was 60 minutes.

RESULTS

Stroke

Testing before and after training for the stroke subject revealed little, if any, change in his clinical optometric data regarding heterophoria and vergence. His DEM clinical findings improved (a lower ratio is better) from a pretraining ratio of 65:50 with no errors to a posttraining ratio of 48:55 with no errors. Objective and subjective oculomotor findings before and after training are presented below.

Figure 2 presents a composite summary of our fixational eye movement findings. In the eye position versus time recordings, 2 improvements occurred after training: horizontal and vertical drift amplitude markedly reduced, and horizontal saccadic in-

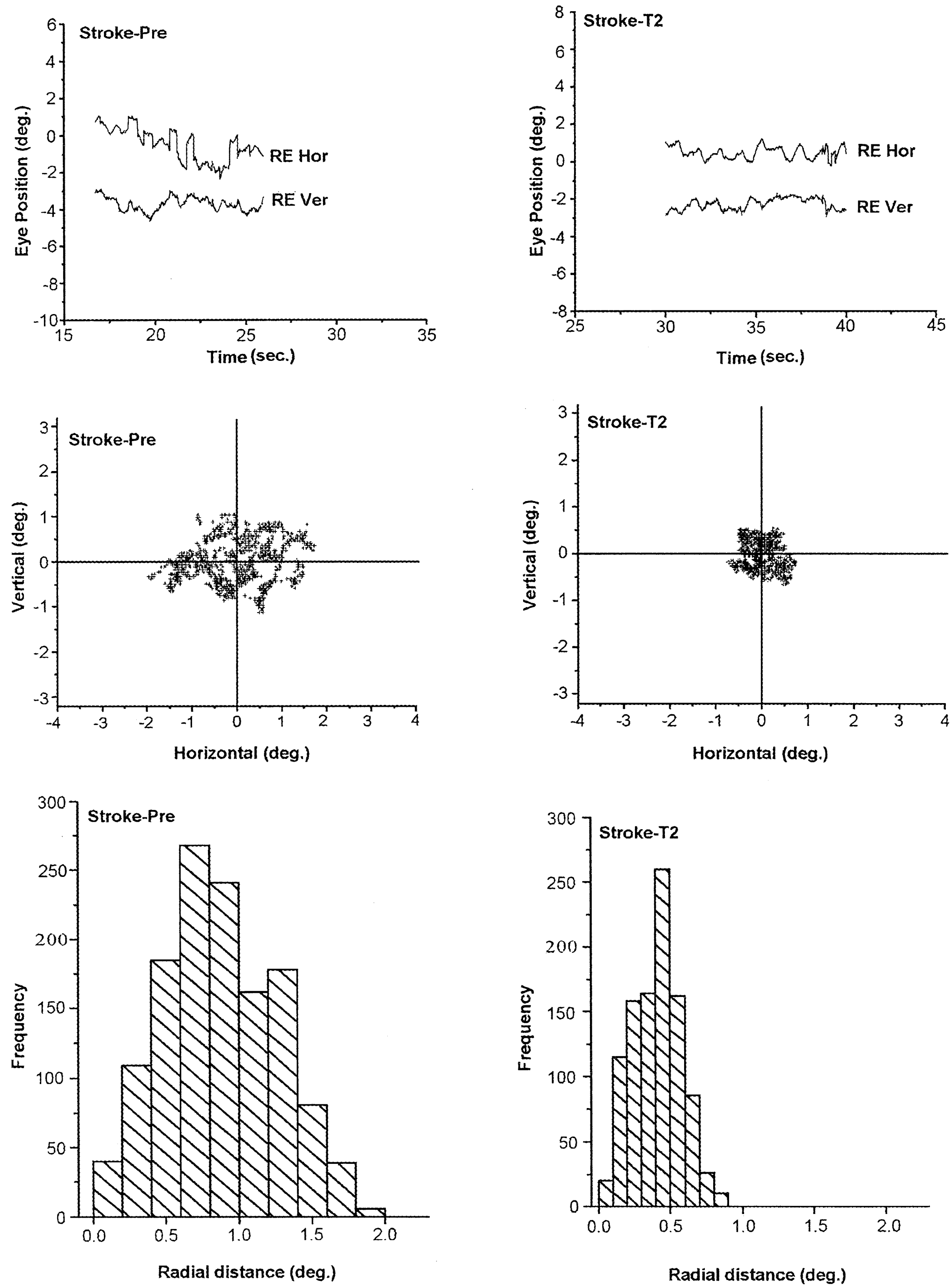

Fig 2. Fixational eye movements in the stroke patient. Left panels, before training (Pre); right panels, after training (T2). Top graphs, horizontal and vertical eye positions as functions of time; middle graphs, 2-dimensional plot of horizontal and vertical fixational eye position scatter/error (ie, the fovea projected into visual space relative to the reference fixation point [0,0]); and bottom graphs, mean radial fixation error. NOTE: 1200 samples from an artifact-free, 10-second portion of the time/eye position traces were used for the 3 fixational error analyses.

trusion frequency and amplitude reduced. These improvements are reflected in the 2-dimensional plots, in which the relatively large and symmetrical fixational errors before training became markedly reduced and near normal limits[41] after training. The frequency distribution of the radial error also became narrower, more peaked, and shifted toward zero after training, which demonstrates a reduction in both mean position error and its variance. The pre- versus posttraining fixational error magnitudes are summarized in table 1. For the horizontal, vertical, and radial dimensions, the training improvements were 63%, 36%, and 55%, respectively. Hence, overall fixational accuracy improved considerably after training.

Table 1: Mean Amplitude of Fixational Error for the Horizontal, Vertical, and Radial Directions Before and After Training in the Stroke and TBI Subjects

Direction From Center	Stroke			TBI		
	Pre	Post	% Improvement	Pre	Post	% Improvement
Horizontal	.83°	.31°	63	.38°	.25°	34
Vertical	.47°	.30°	36	.46°	.28°	39
Radial	.87°	.39°	55	.26°	.17°	35

NOTE: % improvement is (pre−post)/pre×100.
Abbreviations: post, after training (T2); pre, before training.

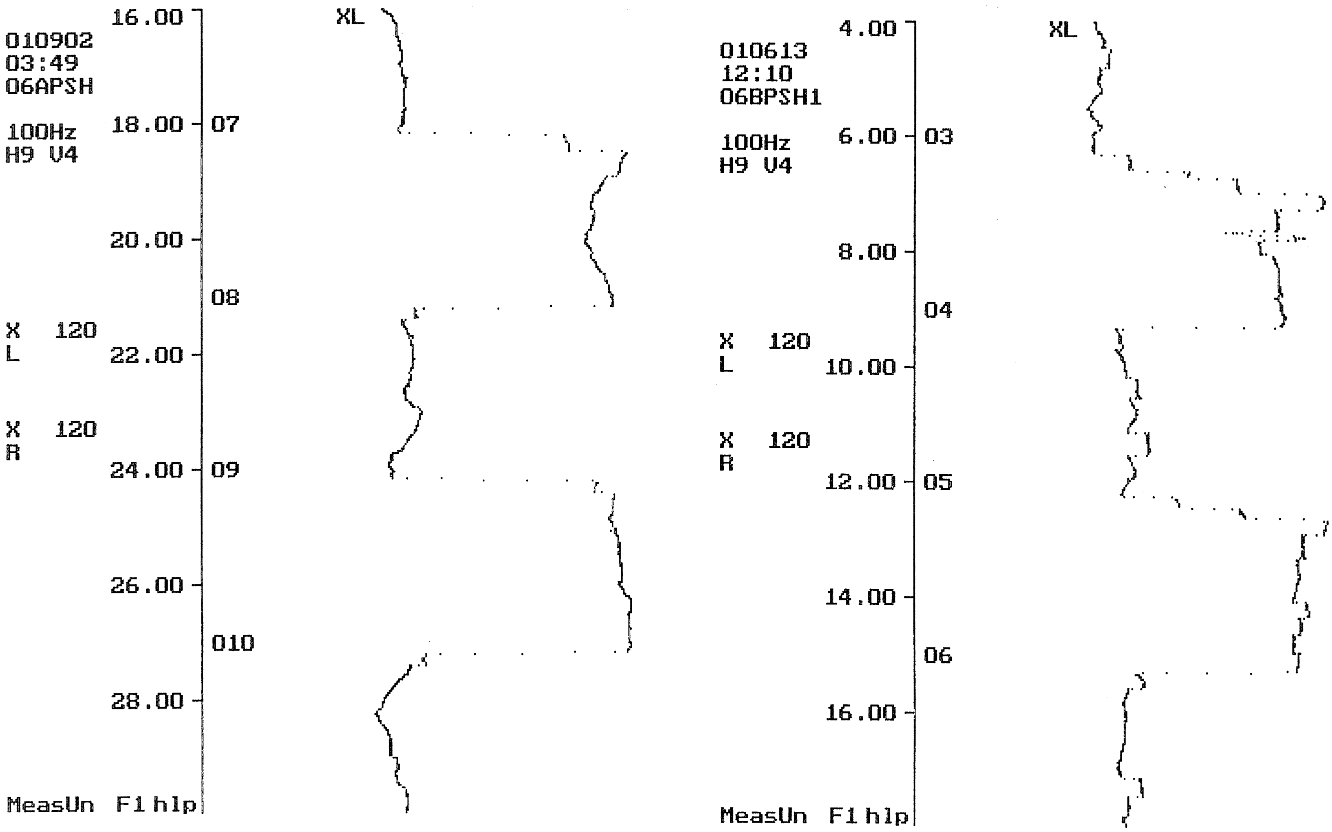

Fig 3. Predictable horizontal saccades (20°) for the left eye (XL) before (right) and after (left) all training in the stroke subject. Leftward is left, rightward is right, time in seconds during the trial is specified to the left of the vertical line, and onset of step target changes is indicated by numbered markers to the right of the vertical line.

Findings for predictable saccades are presented in figure 3 and table 2 (stroke). Before training, multiple saccades were executed into the hemianopic field to attain eventual foveal acquisition of the target. After training, saccadic ocular motility effectively normalized, and target acquisition occurred after only 1 or 2 saccades were executed. Gain of saccades made into the hemianopic field increased from .43 to .95 with training, a range that is considered within normal.[41] This response was consistent. Saccadic gain increased in all directions, except to the left where it was, and remained, normal. Thus, the accuracy of predictable saccades improved markedly with training.

Table 2: Mean Gain of Predictable Saccades (20° amplitude) and Mean Gain of Smooth Pursuit (20° amplitude) Before and After Training for the Stroke Subject and the TBI Subject

Direction	Mean Gain, Predictable Saccades		Mean Gain, Smooth Pursuit	
	Pre	Post	Pre	Post
Stroke				
Left	1.01	1.00	0.94	1.05
Right	0.43	0.95	0.78	1.05
Up	0.71	1.00	0.72	1.06
Down	0.81	1.04	0.99	1.09
TBI				
Left	1.01	0.95	1.00	1.01
Right	1.00	0.93	0.89	0.94
Up	0.80	0.90	1.00	0.93
Down	0.74	1.04	1.03	0.97

Saccadic latency for both horizontal and vertical nonpredictable saccadic ocular motility was within normal limits before training. It remained so during the entire training period.

The pursuit data are in table 2 (stroke). Pursuit ocular motility exhibited variable and slightly reduced gain before training. With training, gain increased and normalized in all directions, especially upward and rightward. The eye movements became less variable and more consistent in overall performance. Thus, overall pursuit ocular motility became considerably more accurate after training.

Simulated reading results are presented in figure 4 for both the multiple- and single-line conditions. Before training, the mean saccade frequency ratio was only slightly elevated in the multiple-line condition, but was markedly elevated in the single-line condition. Thus, the subject was executing up to 5 times more saccades than needed for the step target stimuli and eventual foveal acquisition. After training, however, the mean saccade frequency ratio normalized in the multiple-line condition and nearly normalized in the single-line condition. The mean saccade frequency ratio reduced by 20% and 75% for the multiple- and single-line patterns, respectively, toward a normal value of 1. Thus, saccadic ocular motility for both simulated reading paradigms improved considerably after training.

The reading results are presented in figure 5 and table 3. After training, there was considerable reduction in the number of saccades (ie, both progressive and regressive) as the subject

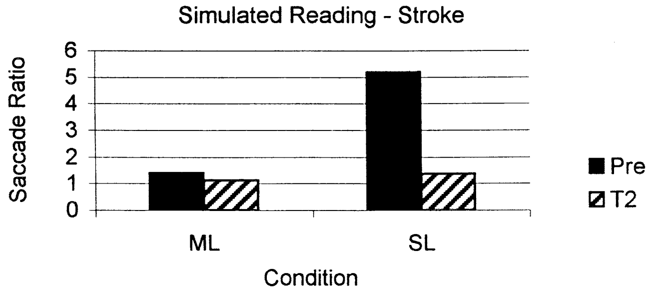

Fig 4. Simulated reading mean saccade frequency ratio before (Pre) and after (T2) training for multiple-line (ML) and single-line (SL) conditions in the stroke patient.

read and shifted his eyes rightward into the hemianopic field. This was reflected by improvement in nearly all of the reading eye movement parameters, with the reading rate increasing by 40%, from 47 to 67 words per minute. Hence, reading eye movements and reading ability improved after training.

Last, the questionnaire results demonstrated considerable improvement in reading and related attentional aspects after training for the subject with stroke. His overall questionnaire score increased from 15 to 21, with the maximum optimal score being 24. This improvement was consistent with the objective ocular motility findings.

Traumatic Brain Injury

Testing before and after training for the subject with TBI revealed little, if any, change in his clinical optometric data regarding heterophoria and vergence. His DEM clinical findings improved from a pretraining ratio of 62:45 with no errors to a posttraining ratio of 42:40 with no errors. Objective and subjective oculomotor findings before and after training are presented below.

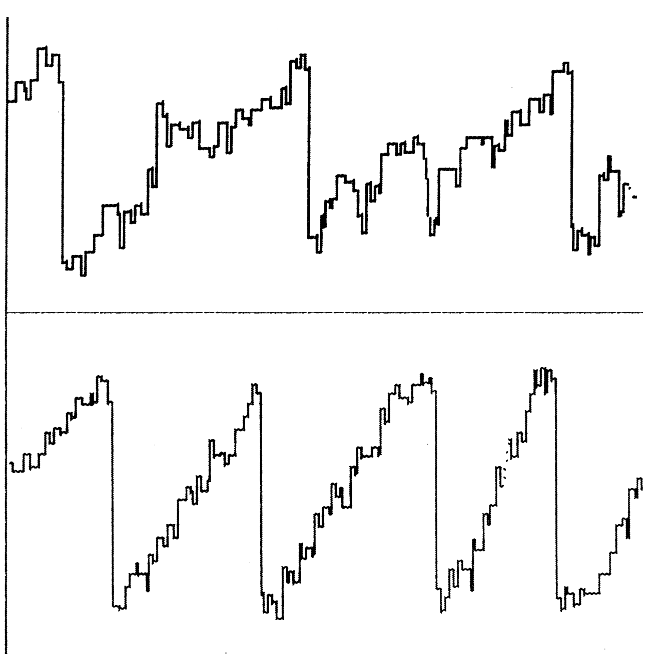

Fig 5. Reading eye movements for adult level 10 paragraphs before (top) and after (bottom) training in the stroke subject with right hemianopia. Up is right, and down is left. Time periods for both eye movement traces are the same.

Figure 6 presents a composite summary of the fixational eye movement findings. In the eye position versus time recordings, the primary training-related improvement was the reduction in amplitude of the obliquely directed, nystagmus-like drift movements (≈2°/s maximum drift velocity), especially in the vertical direction. This improvement is reflected in the 2-dimensional plots, in which the relatively large, vertically biased fixational error before training was markedly reduced, with only a slightly oblique bias now present. In addition, the frequency distribution of the radial error became narrower, more peaked, and shifted toward zero after training, which demonstrates a reduction in both mean position error and its variance. The pre- versus posttraining fixational error magnitudes are summarized in table 1. For the horizontal, vertical, and radial dimensions, improvements were 34%, 39%, and 35%, respectively. Thus, overall fixational accuracy improved considerably after training.

Findings for predictable saccades are presented in figure 7 and table 2 (TBI). Horizontal saccadic gain was within normal limits, but vertical saccadic gain was reduced, before training. After training, however, vertical saccadic gain normalized and was consistently close to 1. This is also evident from the actual eye movement recordings. Hence, the accuracy of predictable saccades improved markedly with training.

Saccadic latency for both horizontal and vertical nonpredictable saccadic eye movements was within normal limits before training. It remained so during the entire training period.

The pursuit results are in table 2 (TBI). Pursuit gain was essentially normal both before and after training.

Simulated reading findings are presented in figure 8. The mean saccade frequency ratio was increased approximately 2-fold in both conditions before training. Thus, the subject was executing twice as many saccades as demanded by the actual step target stimuli for attainment of accurate foveal acquisition. However, with training, the mean saccade frequency ratio normalized toward a value of 1. It reduced by 40% in the multiple-line condition and by 50% in the single-line condition. Hence, saccadic ocular motility for both simulated reading patterns improved considerably after training.

Reading results are in figure 9 and table 3. With training, all parameters improved considerably; for example, reading rate increased from 138 to 177 words per minute (ie, by ≈30%). Thus, reading eye movements and reading ability improved with training.

Last, the questionnaire results revealed considerable improvement in reading and related attentional aspects after the training in this subject with TBI. The overall questionnaire score for this subject increased from 6 to 11, with the maximum optimal score being 24. This improvement was corroborated by the objective ocular motility findings.

Table 3: Visagraph Parameters Before and After Training for the Stroke and TBI Subjects

	Stroke Subject; Adult Level 10		TBI Subject; Adult Level 10	
Parameter	Pre	Post	Pre	Post
Words per minute	47	67	138	177
Grade level	1.0	1.0	3.2	6.0
Fixations/100 words	383	291	148	119
Regressions/100 words	168	117	31	16
Duration of fixation (s)	0.33	0.30	0.29	0.28
Comprehension (%)	90	100	70	90

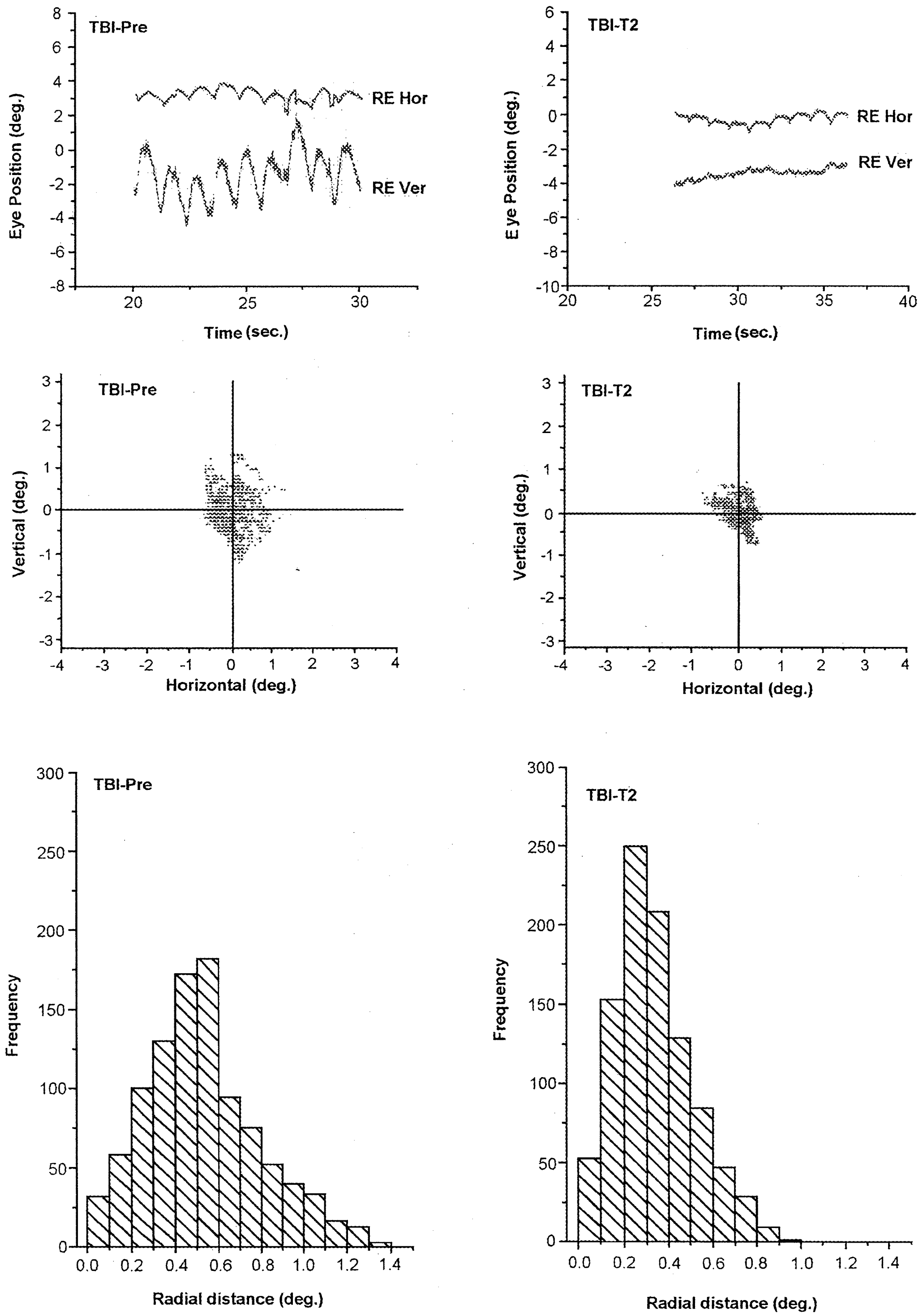

Fig 6. Fixational eye movements in the patient with TBI. Left panels, before training (Pre); right panels, after training (T2). Top graphs, horizontal and vertical eye positions as functions of time; middle graphs, 2-dimensional plot of horizontal and vertical fixational eye position scatter/error (ie, the fovea projected into visual space relative to the reference fixation point [0,0]); and bottom graphs, mean radial fixation error. NOTE: 1200 samples from an artifact-free, 10-second-portion of the above time/eye position traces were used for the 3 fixational error analyses.

DISCUSSION

Oculomotor Neural Centers Involved in Training

Due to the pervasiveness of the primary insult, acquired brain injury affects relatively large regions of the brain. In TBI, this pervasiveness is due to the coup-contrecoup nature of the traumatic event; in stroke, it is due either to the ischemic or hemorrhagic nature of the event directly affecting the site of lesion and indirectly affecting contiguous regions (eg, via compression).[26] Thus, precise and discrete cerebral localization effects of the training as deduced from the oculomotor measures alone are difficult. Furthermore, the overall oculomotor neural network is extensive,[41,45] thus leading to multiple oculomotor and related subsystem deficits from a single cranial insult. Also, many of the same areas of the brain contribute to multiple and diverse oculomotor functions. For example, the frontal, parietal, and cerebellar regions each participate in both saccadic and pursuit neural control. Despite the localization

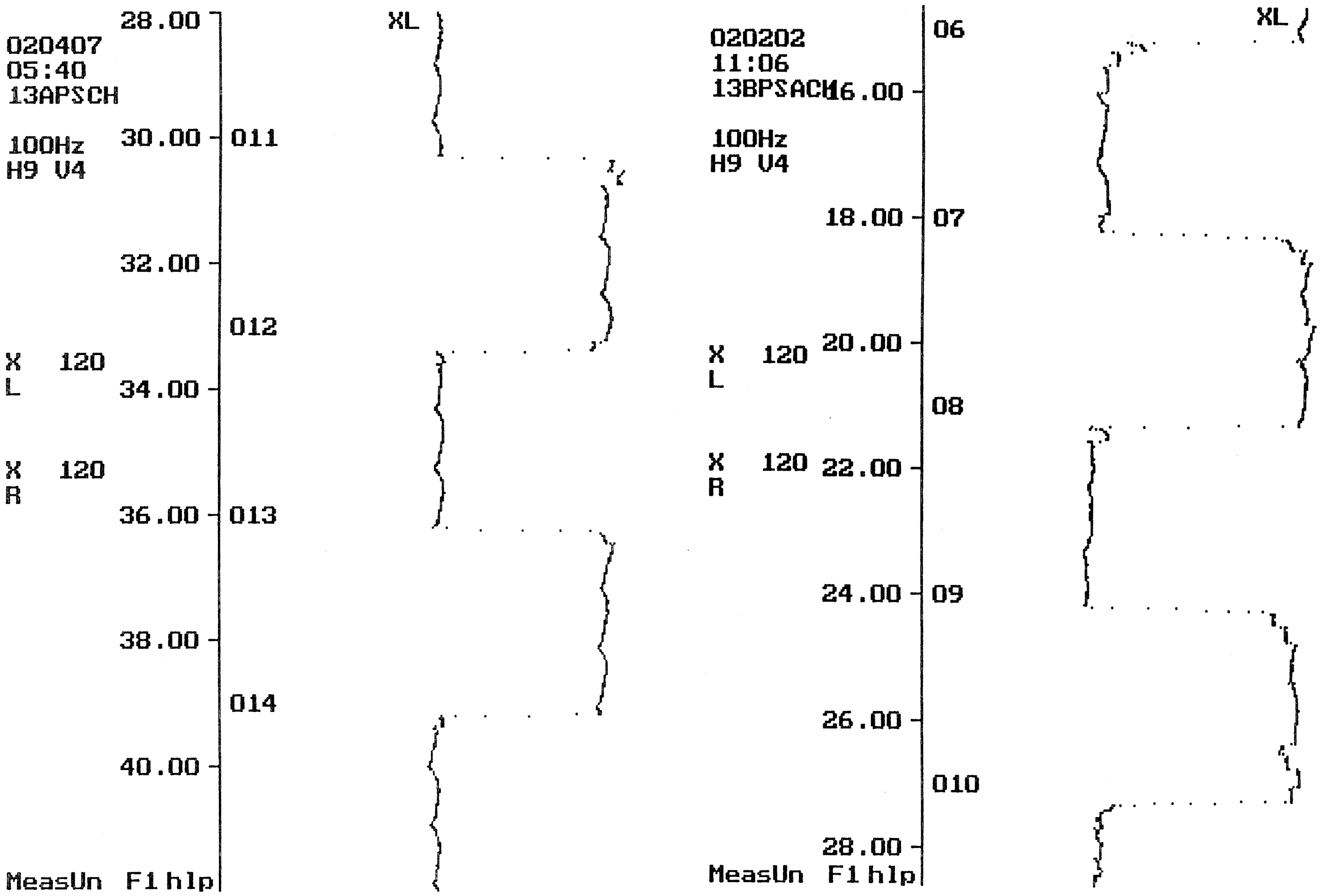

Fig 7. Predictable horizontal saccades (20°) for the left eye (XL) before (right) and after (left) all training in the subject with TBI. Leftward is left, rightward is right, time in seconds during the trial is specified to the left of the vertical line, and onset of step target changes are indicated by numbered markers to the right of the vertical line.

difficulties, based on the results of the present 2 cases and our overall study findings, we believe that the brain's considerable neural plasticity and a person's motor learning can positively affect the versional oculomotor components of fixation, saccade, and pursuit subsequent to the training.[46] This belief was suggested and confirmed by the questionnaire results, as well as anecdotal patient reports. Future studies using high-resolution brain imaging techniques (eg, functional MRI) before and after such training, as well as basic clinical lesion studies,[47] will help to unravel which neural pathways are involved.

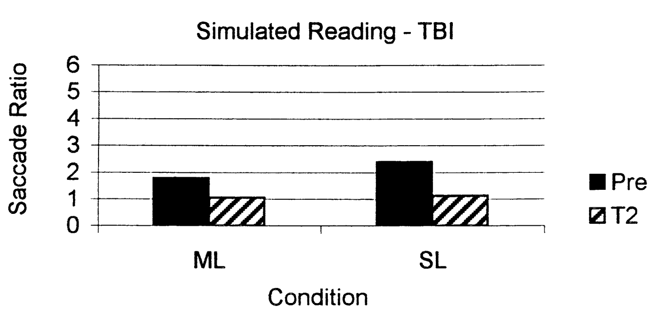

Fig 8. Simulated reading mean saccade frequency ratio before (Pre) and after (T2) training for multiple-line and single-line conditions in the patient with TBI.

Saccadic Gain Adaptation and Oculomotor Plasticity

One of the most striking findings was the rapid, complete, and consistent normalization of saccadic gain involving the process of saccadic adaptation, which reflected the underlying residual neural plasticity of the saccadic system in the stroke patient with hemianopia and visual neglect. Over the course of the 8-week training period, a total of only 64 minutes was specifically devoted to predictable horizontal saccadic ocular motility (10° or 20° step displacements; .33 cycles per second frequency). This appears to be an optimal protocol for saccadic gain adaptation, producing large and positive results after only approximately 300 saccades being executed into the blind hemifield. Our findings are consistent with those of Zihl[27,34,35] in the same population using a more direct, reading-related saccadic ocular motility paradigm, as well as of Ron et al,[31,32] who used a basic oculomotor training paradigm similar to ours in both task and time frame. The results suggest that the medioposterior cerebellar cortex has remained reasonably intact in this patient,[47] and furthermore has exhibited a considerable degree of residual neural plasticity. However, the time course of adaptation may be slower in the patient, because

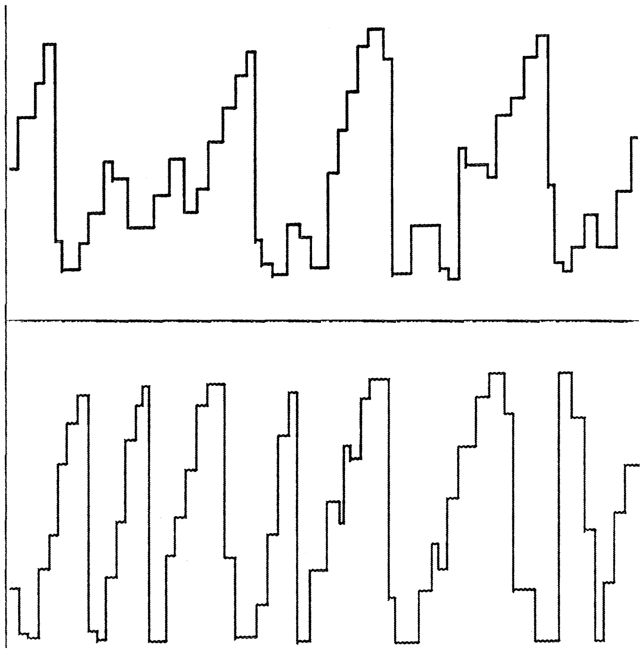

Fig 9. Reading eye movements for adult level 10 paragraphs before (top) and after (bottom) training in the subject with TBI. Up is right, and down is left. Time periods for both eye movement traces are the same.

saccadic adaptation is complete in visually normal subjects with as few as 70 trials.[36]

Saccadic gain adaptation is a subset of general motor learning, which refers to "the process that improves motor performance through practice."[48(p728)] This improvement is believed to occur by increasing synaptic efficiency in a Hebbian manner,[48] with application to human rehabilitation.[49] The practice effect follows a power function, which is optimized using distributed rather than massed trials,[50] as we have used. Motor learning involves 3 stages[46,51]: determining by a trial-and-error process the optimal motor program to accomplish a particular task, repeatedly practicing the optimal motor program/movement to acquire more rapid and precise execution, and attaining automaticity to a level at which the movement essentially becomes "preprogrammed" or "open-loop" in its execution, thus allowing attention to be allocated to other important tasks. Error-related feedback specific for the motor task and optimization of performance is crucial to the learning process.[48,52] Hence, the present use of normal internal oculomotor visual feedback in isolation, or in combination with our laboratory-based external oculomotor auditory feedback, is consistent with this notion.

The DEM versional clinical results improved considerably posttraining. This improvement correlated with the findings for saccadic gain for predictable saccades, multiple-line simulated reading, and single-line simulated reading. Although the DEM results improved with training, heterophoria and vergence are clinical measures that did not improve and, furthermore, would not be predicted to improve because versional, and not vergence, eye movements were being trained with our computer-based protocol.

Role of Attention in Training

Attention refers to "the voluntary control over more automatic brain systems, so as to be able to select and manipulate sensory and stored information briefly or for sustained periods of time."[53(p202)] Traditionally, attention has been broadly categorized into 2 types: (1) *selective attention,* in which relevant stimuli are selected with disregard for irrelevant or competing stimuli, and (2) *divided attention,* in which 2 or more relevant sensory stimului are simultaneously monitored and responded to. In people with TBI or stroke, general attentional and, more specifically, visual attentional difficulties are typically present.[52,54-57] These attentional problems may include difficulty multitasking, distractibility, and inability to sustain attentional focus.[57] In our 2 cases, both general and vision-based attentional deficits were evident, both of which appeared to improve subsequent to training based on the results of the questionnaire and the subjects' anecdotal reports. We also believe that a significant component of both the basic and reading-related training paradigm was its ability to elicit aspects of general attention as well as visual attention. Selective attention was required in the continuous and uninterrupted ocular smooth pursuit of a target, and divided attention was required for the V+A training mode. These findings suggest that both general attention and, more specifically, visual attention may play an important role in the overall oculomotor training process. In future studies, formal testing of specific components of attention should be conducted before and after oculomotor training to assess quantitatively its role and relative importance, as well as to ascertain any correlation with the specific oculomotor parameters that improved.

Training Improvements and Quality of Life

An important aspect of research involving vision rehabilitation is the effect on quality of life (QOL). Attention to QOL represents a practical and common-sense approach; it is also a directive from the National Institute of Health's National Eye Institute (NEI).[58] NEI states that it is important to "evaluate the effectiveness of rehabilitation in the visually impaired," with strategic questions incorporating both "task performance measures" and "quality of life outcome measures."[58(p127)] NEI also states that a goal is to "improve our understanding of the structure/function in the visual central nervous system, neural plasticity, and the performance of everyday tasks, so that visual processing capabilities of the visually-impaired can be optimized."[58(pxvii)] The present testing included all of these aspects. First, both our basic and our reading-related oculomotor findings demonstrated large and consistent improvements in the task performance measures, which suggested considerable residual neural plasticity in the related versional oculomotor pathways.[41,43,46] Second, the questionnaire results incorporated specific QOL measures related to reading and reading-related attentional aspects, and these findings were consistent with the objective oculomotor task performance measures: subjects' subjective rating-scale measures indicated they could read longer, with better understanding, greater attention, and improved strategic approach. And, third, their anecdotal reports were also consistent with both of the above results. For example, both subjects reported that they could read more comfortably and with less visual fatigue, with the stroke subject indicating that the sense of improved attention even transferred to other general tasks, such as watching television and participating in conversations.

CONCLUSIONS

Our detailed and quantitative case presentations provide further evidence of the objectively based, positive effect of oculomotor rehabilitation on oculomotor control. There was also concordant improvement in subjective QOL aspects relating to reading on completion of the training regimen. Future studies using larger sample sizes and assessing attention and other factors should be conducted, because these factors may be important in the training process.

Acknowledgments: We thank Drs. Jan Ygge and Roberto Bolzani for providing the custom software programs for some of the data analysis and graphical display. We also thank Dr. Bin Wang for assistance with the graphs.

References

1. Quinn B, Sullivan SJ. The identification by physiotherapists of the physical problems resulting from a mild traumatic brain injury. Brain Inj 2000;14:1063-76.
2. Richter EF. Interdisciplinary management and rehabilitation of acquired brain-injured patients. In: Suchoff IB, Ciuffreda KJ, Kapoor N, editors. Visual and vestibular consequences of acquired brain injury. Santa Ana (CA): Optometric Extension Program Foundation Pr; 2001. p 10-31.
3. Keren O, Reznik J, Groswasser Z. Combined motor disturbances following severe traumatic brain injury: an integrative long-term treatment approach. Brain Inj 2001;15:633-8.
4. Schmitter-Edgecombe M, Beglinger L. Acquisition of skilled visual search performance following severe closed-head injury. J Int Neuropsychol Soc 2001;7:615-30.
5. McAllister TW, McDonald BC, Flashman LA, Saykin AJ. Executive dysfunction following traumatic brain injury: neural substrates and treatment strategies. Neurorehabilitation 2002;17:333-44.
6. Sbordone RJ, Liter JC, Pettler-Jennings P. Recovery of function following severe traumatic brain injury: a retrospective 10-year follow up. Brain Inj 1995;9:285-99.
7. Hellerstein LF, Freed S, Maples WC. Vision profile of patients with mild brain injury. J Am Optom Assoc 1995;66:634-9.
8. Suter PS. Rehabilitation and management of visual dysfunction following traumatic brain injury. In: Ashley MJ, Krych DK, editors. Traumatic brain injury rehabilitation. Boca Raton (FL): CRC Pr; 1995. p 198-220.
9. Ciuffreda KJ, Suchoff IB, Marrone MA, Ahmann EB. Oculomotor rehabilitation in traumatic brain-injured patients. J Behav Optom 1996;7:31-8.
10. Suchoff IB, Kapoor N, Waxman R, Ference W. The occurrence of ocular and visual conditions in a non-selected acquired brain-injured patient sample. J Am Optom Assoc 1999;70:301-8.
11. Suchoff IB, Gianutsos R. Rehabilitative optometric interventions for the acquired brain injured adult. In: Grabois M, Garrison SJ, Hart KA, Lehmkuhl LD, editors. Physical medicine and rehabilitation: the complete approach. New York: Blackwell Sci; 2000. p 608-21.
12. Suchoff IB, Gianutsos R, Ciuffreda KJ, Groffman S. Vision impairment due to acquired brain injury. In: Silverstone B, Lang MA, Rosenthal B, Faye EE, editors. The Lighthouse handbook on vision impairment and vision rehabilitation. New York: Oxford Univ Pr; 2000. p 517-39.
13. Gianutsos R, Ramsey G, Perlin R. Rehabilitative optometric services for survivors of acquired brain injury. Arch Phys Med Rehabil 1988;69:573-8.
14. Reding MJ, Potes E. Rehabilitation outcome following initial unilateral hemispheric stroke: life table analysis approach. Stroke 1988;19:1354-8.
15. Groswasser Z, Cohen M, Blankstein E. Polytrauma associated with traumatic brain injury: incidence, nature, and impact on rehabilitation outcome. Brain Inj 1990;4:161-6.
16. D'Angelo CM. An overview of enhancement techniques for peripheral field loss. J Am Optom Assoc 1994;64:60-70.
17. Zost MG. Diagnosis and management of visual dysfunction in cerebral injury. In: Maino DM, editor. Diagnosis and management of special populations. St. Louis: Mosby Yearbook; 1995. ch 4.
18. Thurman DJ, Alverson C, Dunn KA, Guerrero J, Sniezek JE. Traumatic brain injury in the United States: a public health perspective. J Head Trauma Rehabil 1999;14:602-15.
19. Max W, MacKenzie EJ, Rice DP. Head injuries: costs and consequences. J Head Trauma Rehabil 1991;6(2):76-81.
20. Kelley RE. Cerebrovascular disease. In: Weiner WJ, Goetz CG, editors. Neurology for the non-neurologist. Philadelphia: Lippincott; 1989. p 52-66.
21. American Heart Association Website. Available at: http://www.strokeassociation.org/presenter.jhtml?identifier=1033. Accessed January 8, 2004.
22. Baker RS, Epstein AD. Ocular motor abnormalities from head trauma. Surv Ophthalmol 1991;35:245-67.
23. Lepore FE. Disorders of ocular motility following head trauma. Arch Neurol 1995;52:924-6.
24. Sabates NR, Gonce MA, Farris BK. Neuro-ophthalmological findings in closed head trauma. J Clin Neurol Ophthalmol 1991;11:273-7.
25. Schlageter K, Gray K, Shaw R, Sammet R. Incidence and treatment of visual dysfunction in traumatic brain injury. Brain Inj 1993;7:439-48.
26. Ciuffreda KJ, Han Y, Kapoor N, Suchoff IB. Oculomotor consequences of acquired brain injury. In: Suchoff IB, Ciuffreda KJ, Kapoor N, editors. Visual and vestibular consequences of acquired brain injury. Santa Ana (CA): Optometric Extension Program Foundation Pr; 2001. p 77-88.
27. Zihl J. Rehabilitation of visual disorders after brain injury. E Sussex (UK): Psychology Pr; 2000.
28. Ciuffreda KJ. Visual vertigo syndrome: clinical demonstration and diagnostic tool. Clin Eye Vis Care 1999;11:41-2.
29. Suchoff IB, Ciuffreda KJ, Kapoor N. An overview of acquired brain injury. In: Suchoff IB, Ciuffreda KJ, Kapoor N, editors. Visual and vestibular consequences of acquired brain injury. Santa Ana (CA): Optometric Extension Program Foundation Pr; 2001. p 1-9.
30. Han Y, Ciuffreda KJ, Kapoor N. Reading-related oculomotor testing and training protocols for acquired brain injury. Brain Res Protocols. In press.
31. Ron S, Najenson T, Hary D, Pryworkin W. Eye movements in brain damaged patients. Scand J Rehabil Med 1978;10:39-44.
32. Gur S, Ron S. Training in oculomotor tracking: occupational health aspects. Isr J Med Sci 1992;28:622-8.
33. Mueller C, Koch S, Toifl K. Transient bilateral internuclear ophthalmoplegia after minor head trauma. Dev Med Child Neurol 1993;35:163-6.
34. Zihl J. Visual scanning behavior in patients with homonymous hemianopia. Neuropsychologia 1995;33:287-303.
35. Zihl J. Eye movement patterns in hemianopic dyslexia. Brain 1995;118:891-912.
36. Semmlow JL, Gauthier GM, Vercher JL. Mechanisms of short-term saccadic adaptation. J Exp Psychol Hum Percept Perform 1989;15:249-55.
37. Ciuffreda KJ, Han Y, Kapoor N. Oculomotor rehabilitation for reading in acquired brain injury. Paper presentation at: the 75th Annual Meeting of the Association for Research in Vision and Ophthalmology; May 4-9, 2003; Fort Lauderdale (FL).
38. Benjamin WJ, editor. Borish's clinical refraction. Philadelphia: WB Saunders; 1998.
39. Garzia RP, Richman JE, Nicholson SB. A new visual-verbal saccade test: the developmental eye movement test (DEM). J Am Optom Assoc 1990;61:124-30.
40. Koenig GS, Price NC, Baird ML, Laukkanen HR, Yolton RL. Use of the Ober2 model B-1200 system for analysis of eye movements made during reading. J Am Optom Assoc 1997;68:155-62.
41. Ciuffreda KJ, Tannen B. Eye movement basics for the clinician. St Louis: Mosby Year Book; 1995.
42. Robinson GM. Rhythmic organization in speech processing. J Exp Psychol Hum Percept Perform 1977;3:83-6.
43. Fayos B, Ciuffreda KJ. Oculomotor auditory biofeedback training to improve reading efficiency. J Behav Optom 1998;9:1-10.
44. Taylor EA. The fundamental reading skill. Springfield: CC Thomas; 1966.
45. Leigh RJ, Zee DS. Neurology of eye movements. 3rd ed. Philadelphia: FA Davis; 1999.
46. Ciuffreda KJ. The scientific basis for and efficacy of optometric vision therapy in nonstrabismic accommodative and vergence disorders. Optometry 2002;73:735-62.

47. Desmurget M, Pelisson D, Urquizer C, Prablanc C, Alexander GE, Grafton ST. Functional anatomy of saccadic adaptation in humans. Nat Neurosci 1998;1:524-8.
48. Lisberger SG. The neural basis for learning of simple motor skills. Science 1988;243:728-34.
49. Byl NN, Merzenick M. Principles of neuroplasticity: implication for neurorehabilitation and learning. In: Gonzalez EG, Myers SJ, Edelstein JE, Lieberman JS, Downey JA, editors. Downey and Darling's physiological basis of rehabilitation medicine. 3rd ed. Boston: Butterworth-Heinemann; 2001. p 609-28.
50. Keele SW. Motor control. In: Boff JK, Kaufman L, Thomas JP, editors. Handbook of human perception and performance. Vol 2(30). New York: John Wiley & Sons; 1986. p 1-60.
51. Abernethy B, Kippers V, MacKinnon LT, Neal RJ, Hanrahan S, Abernethy P. The biophysical foundation of human movement. Champaign: Human Kinetics; 1997.
52. Gottlieb GL, Corcas DM, Jarie S, Agarwal GC. Practice improves even the simplest movements. Exp Brain Res 1988;73:436-8.
53. Park NW, Ingles JL. Effectiveness of attention rehabilitation after an acquired brain injury: a meta-analysis. Neuropsychologia 2001; 15:199-210.
54. Posner MI, Petersen SE. The attention system of the human brain. Ann Rev Neurosci 1990;13:25-42.
55. Mateer C, Mapou R. Understanding, evaluating, and managing attention disorders following traumatic brain injury. J Head Trauma Rehabil 1996;11(2):1-16.
56. Nag S, Rao SL. Remediation of attention deficits in head injury. Neurol India 1999;47:1-10.
57. Hibbard MR, Gordon WA, Kenner B. The neuropsychological evaluation: a pathway to understanding the sequelae of brain injury. In: Suchoff IB, Ciuffreda KJ, Kapoor N, editors. Visual and vestibular consequences of acquired brain injury. Santa Ana (CA): Optometric Extension Program Foundation Pr; 2001. p 32-47.
58. Vision research: a national plan: 1999-2003. A report of the National Advisory Eye Council. Bethesda: US Dept of Health and Human Services, National Eye Institute; 1999. NIH Publication No. 98-4288.

Suppliers

a. Humphrey Field Analyzer; Carl Zeiss Ophthalmic Systems Inc, Humphrey Div, 5160 Hacienda Dr, Dublin, CA 94568.
b. IOTA AB, Sundsvall Business & Technology Ctr, S-851 71, Sundsvall, Sweden.
c. Instructional/Communications Technology Inc, 200-2 2nd St, Huntington Station, NY 11746.