From Braille to Quilting: A Neuro-Optometric Rehabilitation Case Report

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ABSTRACT

Background: Acquired brain injury is most likely caused by trauma or by a cerebrovascular accident. No matter the cause, it can have a dramatic impact on the patient’s quality of life, and can respond to optometric neuro-rehabilitation. The middle cerebral artery (MCA) is the largest cerebral artery and is the vessel most commonly affected by cerebrovascular accident (CVA). The MCA supplies most of the outer convex brain surface, nearly all of the basal ganglia, and the posterior and anterior internal capsules. An MCA from brain infarction or ischemia typically results in the sudden onset of focal neurologic deficits.

Case Report: A 66 year old female with a history of aphasia and right hemiparesis secondary to a stroke of the MCA presented for neuro-optometric services. She was diagnosed with right homonymous hemianopsia, right visual field neglect, visual midline shift syndrome, intermittent diplopia and convergence insufficiency. Lenses, prisms and a program of comprehensive optometric vision therapy were used in conjunction with home vision therapy, occupational, physical and speech therapy. This regimen resulted in successful visual rehabilitation.

Conclusions: A neuro-optometric evaluation of every patient with a history of CVA is warranted. In conjunction with treatment by other disciplines, a comprehensive vision rehabilitation program which includes lenses, prisms and optometric vision therapy procedures can result in improved quality of life.

Keywords: Middle cerebral artery stroke, Neuro-optometric rehabilitation, Post trauma vision syndrome, Visual midline shift syndrome, Visual field loss

Introduction

Acquired Brain Injury

Acquired Brain Injury (ABI) usually affects cognitive, physical, visual, emotional, or social functioning and can result from traumatic brain injury (TBI) (i.e. accidents, falls, assaults, etc.) or non-traumatic brain injury (i.e. stroke, brain tumors, infections, poisoning, hypoxia, ischemia or substance abuse). Damage to the brain can be focal or diffuse. People with a brain injury may have difficulty controlling, coordinating and communicating their thoughts and actions, but they usually retain their intellectual abilities.

Visual problems resulting from acquired brain injury are often overlooked during initial treatment of the injury. Frequently these problems are hidden and neglected, which can lengthen and impair the overall rehabilitation program. Brain injury has dramatically varied effects and no two people can expect the same outcome or resulting difficulties.

The brain controls every part of human life: physically, intellectually, behaviorally, socially and emotionally. When the brain is damaged, some other aspect of a person’s life may also be adversely affected. Even a mild injury can result in a serious disability. While the recovery from the injury depends largely on the nature and severity of the injury itself, appropriate evaluation and treatment will play a vital role in determining the level of recovery. Even without a proper vision assessment and treatment, a gap can
occur in rehabilitative services, resulting in deficient treatment and frustration for the patient, family and the rehabilitation team.

Many visual problems can arise from brain injury and stroke, including: visual field loss, intractable double vision, visual/balance disorders, strabismus, reduced visual acuity at far and near, ocular motility disorders, binocular vision dysfunctions, accommodative disorders, difficulty in visual perception and deficits in visual motor integration.

**Stroke**

Hemorrhagic stroke occurs when a blood vessel bursts inside the brain. The brain is very sensitive to bleeding. Damage can occur very rapidly, either because of the presence of the blood itself, or because the fluid increases pressure on the brain causing harm by pressing it against the skull. Bleeding irritates the brain tissue causing swelling. The surrounding tissues of the brain resist the expansion of the bleeding, which is finally contained by forming a mass (hematoma). Both the swelling and hematoma will compress and displace normal brain tissue. Most often, hemorrhagic stroke is associated with high blood pressure which stresses the artery walls until they break.1

**Blood Supply to the Brain**

Two main pairs of arteries supply the brain: the two internal carotid arteries and the two vertebral arteries.2 (Figure 1) An important imaginary line divides the cerebrum into a front (anterior) and a back (posterior) area. The internal carotid artery supplies the frontal area and divides into the anterior and middle cerebral artery. The middle cerebral artery supplies the lateral surface of the cerebrum above the dotted line in Figure 1, whereas the anterior cerebral artery supplies the medial aspect of the cerebral hemispheres above the dotted line.

The middle cerebral artery (MCA) more specifically supplies most of the outer convex brain surface, nearly all the basal ganglia, and the posterior and anterior internal capsules. The MCA generally arises as a single trunk 18-26 mm long with a diameter of approximately 3 mm. The first branches consist of 15-17 small lenticulostriate arteries that supply the putamen and pallidum or the lentiform nucleus, internal capsule, and caudate nucleus of the basal ganglia. After the lenticulostriate branches, the MCA generally bifurcates, forming superior and inferior divisions. The superior branch supplies the prefrontal and orbitofrontal cortex, and the inferior branch supplies the anterior, middle and polar temporal regions. Infarcts that occur within the vast distribution of this vessel lead to diverse neurologic sequelae.3-9

Main trunk occlusion of either side yields contralateral hemiplegia, eye deviation toward the side of the MCA infarct, contralateral hemianopsia, and contralateral hemianesthesia. Eye and head deviation toward the side of the lesion is probably due to damage of the lateral gaze center (Brodmann area 8) or it can represent classic neglect, particularly when the right MCA is involved.3,5,9 Trunk occlusion involving the dominant hemisphere causes global aphasia (an almost total reduction of all aspects of spoken and written language, in expression as well as comprehension) whereas involvement of the nondominant hemisphere causes impaired perception of deficits (anosognosia) of speech. Superior division infarcts lead to contralateral motor deficits with significant involvement of the upper extremity and face and partial sparing of the contralateral leg and foot.3,9

Inferior division infarcts of the dominant hemisphere lead to Wernicke aphasia. Such infarcts on either side yield a superior quadrantanopsia or homonymous hemianopsia, depending on the extent of infarction. Right inferior branch infarcts also may lead to a left visual neglect. Finally, resultant temporal lobe damage can lead to an agitated or confused state.1

**Neuro-Optometric Rehabilitation**

Neuro-optometric rehabilitation, which includes optometric vision therapy, can aid in the resolution
of both TBI and stroke victims. It is defined as an individualized treatment regimen for patients with visual deficits as a direct result of physical disabilities or acquired brain injuries. These deficits include acquired strabismus, diplopia, binocular dysfunction, anomalies of accommodation, paresis/paralysis, and oculomotor dysfunction. Other areas that can be treated include visual spatial dysfunction, post-trauma vision syndrome, and visual midline shift syndrome, visual field loss, visual perceptual and cognitive deficits, and traumatic visual acuity loss. These visual dysfunctions increase the likelihood of psychological sequelae such as anxiety and panic disorders, as well as spatial dysfunctions affecting balance and posture.

A neuro-optometric rehabilitation treatment plan can improve specific acquired vision dysfunctions determined by standardized diagnostic criteria. Treatment regimens encompass non-compensatory lenses and prisms with or without occlusion and other appropriate rehabilitation strategies.

Case
A 66 year-old, Caucasian, female homemaker was referred by an occupational therapist for neuro-optometric rehabilitation due to suspected vision problems. The patient was discovered 10 weeks previous by her family to be poorly responsive, with decreased movement of her right side. A computed tomography (CT) scan of the brain demonstrated early changes in the distribution of the left middle cerebral artery. There was also evidence of a previous stroke affecting the right parieto-occipital region. A neurologist’s examination revealed right hemiplegia and nonfluent global aphasia secondary to the left cerebrovascular accident. Specifically, the patient had an acute ischemic infarction of the left middle cerebral artery distal to the bifurcation (and signs of a previous stroke with an old infarction in the right parieto-occipital lobe). She was examined by a physiatrist who reported right homonymous hemianopsia, right hemiparesis, and spasticity in the right upper extremity. She also demonstrated a right Babinski sign and right facial weakness.

Medical History
The patient had a history of coronary artery disease and heart attack three years prior to this event, and a two vessel stent insertion had been performed at that time. Other medical conditions included hypertension, hyperlipidemia, urinary incontinence, gastroesophageal reflux disease, hip replacement, and, most recently, shingles. She had been a 40 plus pack per year smoker, but had quit ten years prior to the stroke. She did not report alcohol use. Her mother died from stroke (age; 82 years) and her father (57 years) and sister (68 years) from acute myocardial infarction. She was allergic to morphine and was on the following medications: Endocet (Oxycodone), Lidocaine 2% gel (Xylocaine), nortriptyline 20 mg PRN (Pamelor), omeprazole 20 mg bid (Prilosec), dipyridamole 75mg tid (Persantine), lovastatin 80 mg qhs (Mevacor), atenolol 25 mg daily (Tenormin), captopril 12.5 mg bid (Captopren), hydroxyzine 25 mg (Atarax) and, as needed aspirin 81 mg daily.

Although a recommendation was made for the patient to enter a subacute facility upon discharge from the hospital four weeks following the stroke, her family’s decision was to take her home. She was seen by an ophthalmologist one month following discharge, who had found her vision to be 20/200 OU and referred her to the Braille Institute. She had completed physical, occupational and speech therapy as an inpatient. Her outpatient care was in the process of being transferred to another facility for insurance reasons.

Vision Assessment
The vision examination was performed one week after the patient was seen by the ophthalmologist, approximately eight weeks after the stroke occurred. The patient was aphasic and subjective responses were minimal and slow. She did not appear to have difficulty understanding and following instructions as long as they were repeated slowly. She complained of decreased visual acuity and intermittent diplopia. (The patient was unsure whether diplopia was horizontal or vertical or whether it was greater at distance or near). Mild symmetrical proptosis OD and OS without ptosis was noted. Pupils were round and reactive, and the Hirschberg reflexes were symmetrical. Applanation tonometry was 15 mmHg OD, OS. The patient was dilated and binocular indirect ophthalmoscopy revealed a healthy fundus with a cup to disc ratio of 0.25 round OD, OS. The visual field revealed a right homonymous hemianopsia with slight macular sparing in the right eye. (Figure 2)

The objective vision examination (autorefraction and retinoscopy) revealed hyperopia and astigmatism: OD: + 4.75 – 1.00 x 050 and OS: + 2.50 – 1.00 x 165 Add + 2.50 OU. Distance visual acuity was
estimated at 20/200 by the standard Snellen chart and the Tumbling E's. Speech was slow and labored. The patient could not recognize any letters beyond the 20/200 size even if asked to copy the letters. Near visual acuity was approximately 20/40, OU with the Tumbling E’s chart for which she used her hand to show the orientation.

Figure 2: Visual field loss with right homonymous hemianopsia and slight macular sparing OD.

Figure 3: Flower drawing (incomplete, displaced, compressed on the left) and Line Bisection Test (displaced to the left) - pretherapy.
The complete examination was spread over several weeks due to the patient's difficulty with extended testing. Other results from the examination are as follows:

(Measured with spectacles):
cover test: ortho, distance; 8–10 exophoria near; near point of convergence was receded at 15 cm break and 20 cm recovery. Maddox rod near horizontal phoria was 8BI with no response at distance. Near vertical phoria measured ortho in primary gaze was slightly inconsistent. She was unable to see on the right due to the visual field loss and was unresponsive to the left field with either eye. Worth 4 Dot: 4 dots in primary gaze at near. The Padula test (Appendix A) revealed a midline shift of 15-20 degrees to the left but none anterior or posterior. Saccadic fixator targets were consistently missed on the right side. Flower drawing was displaced to the right. The right side was missing while the left side was compressed. (Figure 3) The Line bisection test showed significant displacement to the left. (Figure 3)

Performance Testing

Response to yoked prisms (15°) in different orientations was evaluated while the patient was seated. Marsden ball games were used to assess eye hand coordination with the same yoked prisms in different orientations. A dramatic subjective improvement was noted with 15° BD OU. Bi-nasal occlusion testing was attempted on a near prescription with 2 BI prisms OD and OS (recommended treatment for post-trauma vision syndrome). Bi-nasals were discontinued after 1 week at the patient's request. Since the patient was very sensitive to light, a trial pair of blue tinted glasses (#2) were given to wear over her current glasses. These were prescribed after she indicated a subjective improvement (the tint allowed her to keep her eyes open and the light was not as bothersome). Prior to determining the final prescription, the tint was adjusted according to her preference.

Computer saccades (Computer Orthoptics) with a large target were performed as baseline testing and the patient scored 73.8% with 3.74 seconds per response. Wolrd sentence copy and tachistoscope testing demonstrated that the patient was unable to recognize letters/numbers. Visagraph assessment (Figure 4), with non standard reading material (symbols), showed poor performance with a significantly increased average span of recognition and very poor eye movement skills. The patient needed assistance while walking. She leaned significantly to the left and veered to the left while walking without assistance.

The TVPS was completed over several weeks due to very slow responses. She scored the following: Visual Discrimination scored 7/16 (Visual Perceptual age or VPE- 4yrs 5mo-9mo, poor); Visual Memory 11/16
Optometric Vision Therapy

Optometric vision therapy of approximately 27 sessions was recommended with an option to discontinue after 12 visits if no progress was evident. The goal of the therapy was to minimize the effect of visual field loss by using compensation strategies, decrease visual field neglect, improve visual midline shift, convergence insufficiency, visual motor integration and speed of visual information processing. With the patient’s motivation and commitment to complete home therapy assignments, the prognosis was considered to be guarded to fairly good. The family was concerned about her vision, but also her ability to live independently at home while her husband was at work.

Therapy was initiated ten weeks after the stroke first occurred. The TBI Non-Strab Vision Therapy (OEP) Curriculum framework was used extensively while incorporating inclusive therapy techniques suggested by the NORA (Neuro-Optometric Rehabilitation Association) clinical skills program. Numerous improvisations had to be made even on the first day of therapy when eye control as witnessed by the yoked prism ball game probe, Hart chart (near-far) rock and C-P saccades (central, peripheral saccades) were deficient. As the patient was unable to read or recognize letters or numbers, letter flash cards with picture association to recognize small words were used as well as the multi-matrix and tracking tube. Dr. Harry Wachs techniques involving parquetry blocks were also used to make therapy interactive. Materials needed for this activity include colored wooden or plastic blocks (squares, triangles, and diamonds) and clear plexiglass sheets. It is suggested that a dot or sticker be placed at the top of both plexiglass sheets to serve as a “clue” in orientation. Initially one has to make sure that the patient can identify the color, name of the shape, and number of sides. Next, with the therapist seated directly across from the patient, the therapist builds patterns with specific shaped blocks in specific orientations. Activities such as “direct matching,” “flipped over” (top to bottom, bottom to top), flipped side to side (left to right, right to left), rotations, and off-center patterns are done to improve a patient’s visual analysis ability, develop eye-hand coordination, fine motor control, visual-motor analysis and visualization abilities.

The tracking tube is made of clear plastic and is 48 inches long and 1 5/8 inches in diameter with a row of colored letters and numbers on opposite
sides. This technique helps to enhance visual tracking skills in patients with visual neglect and visual field loss. A portion of the tube (12 inches) centrally can be covered by an opaque material to challenge these patients to train tracking skills on the affected side.

Sector peripheral prism $^{24} (15 \Delta)$ and then Peli prism $^{25} (12 \Delta)$ was used for about 12 weeks to help minimize the impact of visual field loss, but as her scanning ability improved she did not find them to be as useful. The patient preferred scanning techniques $^{26-28}$ such as hallway saccades, the Wayne saccadic fixator, and multi matrix.

Hallway saccades are done by arranging letters or numbers sequentially on the wall of a passage or hallway at eye level and have the patient call them out with each step as they look at a target straight ahead and at the letters or numbers on the wall alternately, in the direction of the neglect. The Wayne Saccadic Fixator has specific programs with additional central cueing to help fixation or stick-ups to expand the peripheral targets. Multi-matrix is an activity that helps build speed and automaticity in sequential and spatial information processing.

She was unable to appreciate physiological diplopia or do the Brock string but was able to appreciate SILO on the Keystone fusion games and vectograms. $^{29}$

Several alternatives to traditional therapy had to be made by using different shapes, and font size changes instead of alphabets for tracking, etc. Syntomics or light therapy was also incorporated towards the middle of the treatment plan. Syntomics involves the manipulation of specific frequencies of visible light to treat specific dysfunctions. $^{30}$ These treatments can result in the expansion of constricted functional visual fields, improvement of mental functioning, and elimination of symptoms. Therapy consists of 20-minute light treatments, 3 to 5 days a week. Eighteen treatments usually result in recovery. $^{30}$

Yoked prisms (15 diopters) to re-orient her visual midline in all orientations were used with ambulation as well as therapy activities. Base-right yoked prisms were emphasized. Fresnel prism base right (OD) was also used during therapy activities to enhance scanning and evaluate the need for prism sector glasses. Color fields were performed and showed restriction in all three colors tested: red, blue and green. This is performed as follows: Red, blue and green targets are brought in one at a time from the periphery while the patient is fixated at a central target of the same color. The patient indicates when he or she first recognizes the hue and intensity that matches the central target color. These points are connected later to give the extent of the color fields.

Physical and occupational therapy had been discontinued nine weeks after the initiation of vision therapy. It was noted that there was an overall improvement in balance but residual visual deficits affecting gait remained.

An emphasis throughout the program was to have the patient reinforce specific activities at home. Her daughter, who devoted all her time to help in the rehabilitation program, was instrumental in giving feedback and also took it upon herself to improvise by enlarging certain font sizes given for tracking, etc., so that her mother could complete the assignments. Although there were periods of frustration initially, as therapy progressed larger stacks of home activities were returned each week. Progress was evident as seen in Table 1.

Line bisection showed significant improvement (Figure 5) and the drawing copy test showed more balance and good representation of both sides of the picture. (Figure 5) Computer saccades could now

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<tr>
<th>Procedure</th>
<th>Initial Examination</th>
<th>Progress Evaluation @ eight months</th>
</tr>
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<tbody>
<tr>
<td>NPC (blur/break/recovery)</td>
<td>x/15/20</td>
<td>6/4/6</td>
</tr>
<tr>
<td>Wayne Saccadic fixator: Program #121</td>
<td>11 Consistently missed right targets</td>
<td>30</td>
</tr>
<tr>
<td>Computer vergence</td>
<td>Unable to perform</td>
<td>BO: 47.5 with SILO, BI: 27.5,</td>
</tr>
<tr>
<td>Computer saccades</td>
<td>Large size 73.8% at 3.74 sec/response</td>
<td>Current: Large size: 88.7%, 1.89 sec/response.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Medium size: 87.9%, 2.42 sec/response,</td>
</tr>
<tr>
<td>Visual midline shift</td>
<td>20 degrees shift to the left</td>
<td>Current: 5 - 10 degree shift to the left</td>
</tr>
<tr>
<td>Motor field</td>
<td>Left eye fixating - 8 Exophoria</td>
<td>Left eye fixating: 2 exo, 1 left hyper.</td>
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<th>Procedure</th>
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Table 1: Examination information
be done even with the small targets that the patient was not able to see previously (62.8% 3.03 sec/response). Functional visual field – REACT (Reaction Time Measure of Visual Fields)²⁶,²⁸,h was tested and showed appropriate compensation and scanning for right visual field loss (a significant improvement). A stimulus is displayed along different meridians in a series of discrete trials until the patient responds to it. The examinee presses a response button as soon as they become aware of a moving (kinetic) stimulus. Usually the stimulus is a two digit number which increases in one hundredth of a second. Once the patient responds, the time is displayed. If no response is recorded, after two seconds the screen clears and a new trial begins. The ability to compensate for visual field loss can be measured with this tool in brain injured patients who might have difficulty with conventional methods of evaluating peripheral visual fields. She was responsive to subtleties and subjectively a mild left hypertropia was elicited during her progress testing. Two diopters Fresnel prism base down left eye was therefore prescribed in her distance glasses and later incorporated permanently.

The most significant improvement was the patient’s presentation thirty weeks into vision therapy of patches of quilt that she had been working on (Figure 6). Although her progress was slow, it was a major step forward in building confidence that the patient could now hold a needle (even though she could not thread a needle and used a magnifier). This was her favorite hobby and it was a great relief and happiness to her family. She did not need assistance in walking and did not veer to the left as she had

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**Figure 5:** Flower drawing and Line Bisection test (balanced and good representation of both sides) – post-therapy.

**Figure 6:** Thirty weeks into therapy she presented with these patches of quilt. Her family members were relieved that she had resumed her favorite hobby.
previously. This was a tremendous boost in her self confidence and she was even able to cross the street by herself, which astonished her family. Her daughter related that she was fairly independent around the house as well. She did not need personal assistance to dress herself, did the laundry and helped in the kitchen except cooking.

The patient was discharged from therapy due to her progress (and a reduced enthusiasm in completing home therapy assignments). She was next seen three months later for her comprehensive eye examination. The distance glasses were updated as follows. OD +5.00 -1.00 x 045 20/40- 1°BU Add +3.00 20/20- OS +2.50 -0.25 x 150 20/40- 1°BD Add +3.00 20/20-

The patient’s husband had bought her a view magnifier but it provided little help. He realized that she still had cognition problems, and that her progress had stopped when she stopped therapy. He expressed an interest in resuming therapy for vision information processing.

Conclusions

Even with extensive neurological deficits resulting from a cerebral artery occlusion, successful outcome can be achieved with vision rehabilitation. One should be cognizant of the extent of the vision, cognitive as well as associated sensori-motor and physical impairment in such individuals. These patients should have an appropriate evaluation to quantify the deficits and the clinician should be prepared to improvise specific activities to facilitate further improvements. Hence, a comprehensive vision rehabilitation program in conjunction with management from other disciplines will enable an acquired brain injured patient to achieve an improved quality of life.

References

Appendix A

Visual Midline Shift Syndrome

Definition: Postural problems causing a person to displace the body mass by leaning to one side or in an anterior or posterior misalignment due to neurological dysfunctions such as cerebrovascular accidents, traumatic brain injury, multiple sclerosis, etc.

Neurology: Dr. Padula describes vision to be comprised of two separate visual processes, focal and ambient as the basis for neuro-optometric rehabilitation. He places a heavy emphasis on ambient vision in particular and has explained this process as follows:

He postulated that ambient vision becomes part of the sensory-motor feedback loop at the level of the midbrain. Not all of the visual fibers emanating from the two eyes go to the occipital lobes, but instead deliver signal to various areas of midbrain and the superior colliculus. At these levels, information is matched with other sensory-motor systems from the kinesthetic, proprioceptive, vestibular and tactile systems. The cerebellum modifies this information before a feed-forward system delivers input to higher organization centers in the cortex for stabilization and anticipation of action. It is this ambient process that creates stability and enables us to anticipate change related to movement, posture and balance and to experience time relationships, past, present and anticipation of future.

The following example is given to illustrate this further: A person who has spent his entire life matching his ambient visual information with balance and sensory-motor information from the two sides of the body, creates a level of normalcy which becomes experience. The person then relies on this information for orientation in space. If however, after a neurological event such as a cerebrovascular accident, a person has paralysis of one side and is left with a hemiparesis or a hemiplegia, the information received from the kinesthetic and proprioceptive systems from one side of the body is different from that of the other. The ambient visual process creates a relative balance between the mismatch of information received at the midbrain and thus expands and contracts space internally in its attempt to understand the dysfunction. The contracted side relates to a compression of space internally, whereas the extended side related to an expansion of internal space. The ambient process, through a feed-forward mechanism to the occipital cortex, then projects this expansion and contraction of space externally and causes distortion of the higher sensory spatial environment. This is reported by some individuals as the tilting of the floor in one direction or the other. This can usually be seen by examining the leaning posture and mobility of a hemiparetic patient. The contracted side relates to a compression of space, whereas the extended side relates to an expansion of internal space.

This mismatch of information and resulting distortion of space has been termed the Visual Midline Shift Syndrome (also known as the perceptual shift syndrome).

Testing for lateral Visual Midline Shift: It can be assessed using a wand or a pencil with the patient not having the examiner facing or have any frame of reference to assist in determining the midline. The vertical target is held approximately 16 inches in front of a person’s face and moved left to right across the field. The patient is to track the wand left to right and tell the examiner exactly when the wand appears to be directly in front of the nose. If a person consistently stops the wand when it is either to the right or left side of their structural midline, it is an indication that the person has shifted their concept of visual midline in that direction. A left hemiparetic patient will shift the conceptual midline to the right. While standing and walking, it may be observed that the patient has difficulty transferring weight to the left side. The right visual midline shift is a reinforcement mechanism to enable the patient to have some aspect of balance even though it is abnormal.

Testing for anterior/posterior Visual Midline Shift: The second phase of the testing may be done by holding the wand horizontally and passing it vertically in front of the person’s face. The person should be instructed to tell the examiner when the wand appears to be at eye level. If the wand was reported at eye level when it was actually positioned below the eye level, it indicates an anterior shift in the perception of visual midline and persons with this distortion will show a flexion posture or a tendency to lean forward while seated or walking. If the wand was reported at eye level when it was actually above the eye level, it indicates a shift in his visual midline posteriorly. Frequently, persons with this shift in visual midline will show extension in posture and thrust their weight backward when walking or seated in a chair.
Therapeutic Rx of Yoked Prism

In a patient with a left hemiparesis, the base of the prisms on both eyes should be positioned on the left in order to move the visual midline back toward the left side. (The apex of the prism expands space while the base end of the prism compresses space) When there is an anterior or posterior shift, the effect of the prisms compresses and expands space around a near-to-far axis (Z-axis). The base down prism will expand near space and compress far space countering the distortion of the downward tilt of the floor seen with persons who learn forward in flexion and vice versa.

Visual Midline Shift  Prism Orientation
Right  Base Left
Left  Base Right
Anterior  Base Down
Posterior  Base Up

Dr. Padula emphasizes that treating a visual midline shift dysfunction should not be considered a cure but a means of rehabilitation that can often influence and maximize potentials beyond the more conventional approaches for treating these disorders. Midline shifts have been detected to some degree in subjects with seemingly normal vision, including athletes. He also states that rehabilitation by the neuro-optometric approach should only be offered when performance is affected and interference in function occurs.

Reference

Appendix B
Equipment Manufacturers

a. Wayne Engineering
8242 N. Christiana Ave.
Skokie, IL 60076
319-266-1721
http://www.wayneengineering.com

b. Bernell Corporation
Bernell VTP
4016 N Home Street
Mishawaka, IN 46545
800-348-2225
http://www.bernell.com

c. Computerized Binocular and Perceptual Therapy System
HTS, Inc.
800-346-4925
http://www.visiontherapysolutions.net/

d. Optometric Extension Program Foundation
1921 E. Carnegie Ave., Suite 3-L
Santa Ana, California, USA, 92705-5510
949-250-8070
http://www.oepf.org

e. Reading Plus (Taylor Associates)
200 E. Second St., Suite 2
Huntington Station
NY 11746
800-732-3758
http://www.readingplus.com

f. Psychological and Educational Publications
PO Box 520
Hydesville, CA 95547
800-523-5775
http://www.psych-edpublications.com

g. Professional Development Programs/PDP Products
1675 Greeley Street South, Suite 101
Stillwater, MN 55082 651-430-8865
http://www.pdp.pro.com

h. Life Science Associates
1 Fenimore Road
Bayport, NY 11705-2115
718-457-7483
http://lifesciassoc.home.pipeline.com
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