

Optometric Management of a Patient with Parietal Lobe Injury



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Abstract

Acquired brain injury (ABI) is an umbrella term referring to sudden-onset, non-degenerative, and non-congenital events, that alter neurological function. Following ABI, any or all of the following aspects of sensorimotor vision function may be impacted negatively: accommodation, vergence, versional ocular motility, visual-vestibular interaction and/or visual field integrity. Therefore, optometric management for those with ABI requires an understanding of the underlying associated neuroanatomy and neurophysiology. This paper further presents a neuro-optometric evaluation and management approach for those with ABI, specifically parietal lobe injury, exemplified by a case report.

Key Words

acquired brain injury, dorsal stream, magnocellular pathway, neuro-optometric vision rehabilitation, Neuro-Vision Rehabilitator, parietal lobes, parvocellular pathway, ventral stream

INTRODUCTION

Acquired Brain Injury

Acquired brain injury (ABI) is an umbrella term for sudden-onset, non-congenital, and non-degenerative conditions that alter neurological function.¹ ABI may occur following external insults resulting in traumatic brain injury (TBI) due to: motor vehicle accidents, gun shot wounds, sports-related injuries, or combat-related insults, to name a few.¹ ABI may also occur due to internal insults, such as: cerebral vascular accident (CVA) (stroke), tumors, aneurysms, vestibular dysfunction, and anoxia or hypoxia due to post-surgical complications.¹

Insult to the brain may be focal or global in nature. A focal insult implies that the presenting signs and symptoms will be related closely to a particular and localized affected area of the brain. Focal neurological insult may be evident with a stroke, aneurysm, or brain tumor. Conversely, a global insult suggests a widespread neurological compromise, not localized to one specific area of the brain. Global neurological insult may be evident with certain types of TBI, depending upon the nature of the injury. For example, some motor vehicle and sports-related accidents involve a coup-contrecoup injury. These coup-contrecoup injuries can present with a global neurological compromise, hypothesized as being related to diffuse axonal injury (DAI).¹

EPIDEMIOLOGY

According to the National Center for Health Statistics, an estimated 1.7 million people sustain a TBI, each year.² Seventy-five percent of these are considered to be mild TBI.³ The common causes of TBI include motor vehicle accidents, falls and

assaults.² Gunshot wounds account for 10% of TBI, however a majority of these result in death.⁴

CVA is an infarct in the brain. It may be hemorrhagic or ischemic in nature and is due to vascular compromise. Approximately 750,000 individuals in the United States suffer a CVA annually.⁵ Despite improved efforts towards prevention, CVA remains the third leading cause of death in the United States and the leading cause of disability among adults.⁵

Persistent and relatively high incidences of TBI and CVA are important because sensorimotor vision deficits affecting activities of daily living (ADLs) often arise following ABI.^{1,6} These visual deficits vary from mild to severe, depending on the etiology, location and severity of the ABI. Hence, a familiarity with the anatomy and function of the lobes of the brain is beneficial to the optometrist.

In addition to the potential vision problems evident following ABI, other aspects of general sensorimotor function, cognitive function, and mood may be altered. Therefore, those with ABI often require interdisciplinary rehabilitative health care for both in- and out-patient stages. The interdisciplinary team members include, but are not limited to: psychiatry, neurology, psychology, social work, neuropsychology, physical therapy, occupational therapy, speech therapy, ophthalmology and optometry.¹

This paper will focus on the neuro-optometric management of ABI, specifically parietal lobe injury, by providing an overview of: DAI, vascular supply to the brain, parietal lobe structures, parietal lobe functions, dual processes of vision, and potential visual signs/symptoms of parietal lobe injuries. A neuro-optometric management

Iskander D, Cohen AH, Kapoor N. Optometric Management of a Patient with Parietal Lobe Injury. J Behav Optom 21;6:143-149.

approach for those with parietal lobe injury will follow as a case report.

BACKGROUND

Diffuse Axonal Injury

Acceleration-deceleration injuries are evident in certain types of TBI including motor vehicle accidents and some sports-related injuries. These often result in coup-contrecoup injuries. The associated pathophysiology of the neurological compromise in such scenarios is hypothesized as being diffuse.⁷

Although axons are generally elastic in nature, sudden forces may cause rapid stretching and shearing of the axons. The information being transferred along the axon can only travel up until the axon thins as a result of stretching. At the point of thinning of the axon, there is a build-up of “transport information.” This then leads to swelling and eventually the tearing of the axon.⁸ The axon distal to the tear shrivels back toward the cell body forming a bulb, with this bulb becoming one of the distinguishing features of DAI.⁸⁻¹¹

Thus, DAI is characterized by axonal stretching, tearing, and bulb formation. This leads to deficits in multiple areas of the brain and subsequently, a global insult to the brain.⁸⁻¹¹

The principal cause of symptoms related to altered sensorimotor, cognitive, and mood changes following TBI was thought to be exclusively due to axonal tearing from the primary injury (i.e., mechanical forces during the trauma). Now, it is believed that there are secondary biochemical cascades occurring between hours and weeks following the primary injury.⁹⁻¹¹ This secondary biochemical cascade essentially causes an increase in intra-axonal calcium concentration.^{11,12} These high levels of intra-axonal calcium disrupt the normal feedback systems within the axon, leading to the damage of the mitochondria. Mitochondrial damage is thought to stimulate the activation of nitric oxide synthases, protease, and lipases, all of which further slow the conductivity of the axons.^{11,12}

Vascular Supply to the Brain

The posterior communicating artery (PCA) supplies the posterior aspect of the brain, mainly the occipital lobe.¹³⁻¹⁵ It then connects with the middle cerebral artery (MCA), that supplies the anterior lateral frontal lobe, parietal and temporal lobe.¹³⁻¹⁵ The anterior cerebral artery (ACA) provides blood to the medial portions of the frontal lobe and the superior medial pari-

etal lobes.¹³⁻¹⁵ In CVA the result is more of a focal deficit rather than a global deficit, which is seen with DAI.

PARIETAL LOBE

Although the frontal and temporal lobes are the most susceptible to DAI, injury of the parietal lobe may impact function significantly as well. The parietal lobe is bordered by the frontal, temporal and occipital lobes. It is comprised of the postcentral gyrus (also known as the anterior parietal lobule), the superior parietal lobule, and the inferior parietal lobule (also referred to as the posterior parietal cortex).¹³⁻¹⁵ Functionally, we can localize the area affected based on the patient’s symptoms. For example, a patient with motor deficits and/or Broca’s aphasia is more likely to have had an infarct in the anterior MCA.^{13,14} Conversely, a patient with sensory deficits and/or Wernicke’s aphasia is likely to have infarcts localized to the posterior MCA.^{13,14}

Parts of the Parietal Lobe and Their Functional Impairments

The potential functional impairments for each part is summarized in the Appendix.

Postcentral Gyrus

The postcentral gyrus is the primary somatosensory area of the brain.

If this area is affected, a patient may have difficulties with proprioception and kinesthesia. While these terms are often used interchangeably, they are not synonymous. They refer to distinct roles in maintaining the sense of equilibrium. Proprioception is the ability to determine the exact location of a particular body part in space. Kinesthesia is the ability to determine that the body part has moved. Additional functional deficits from insult to the postcentral gyrus include agrapism and astereognosis. Agrapism is the inability to write, while astereognosis is the inability to identify objects by palpation.^{16,17} Other responsibilities of the postcentral gyrus include tactile sensation, two point discrimination, and, most important to vision care practitioners, visual attention.^{13,16}

Superior Parietal Lobule

The superior parietal lobule is located directly posterior to the postcentral gyrus. Bilateral lesions at the parieto-occipital junction may result in a syndrome known as Balint’s syndrome. This syndrome is characterized by the inability to voluntarily control gaze or saccades, inability to integrate components of a visual scene (i.e., simultanagnosia), and the inability to

accurately reach for an object with visual guidance (i.e., optic ataxia).^{16,17} Those with Balint’s syndrome are likely to have poor eye-hand coordination, and they may grasp for objects as if they were blind. Unilateral injury to the superior parietal lobule may result in laterality/directionality issues.^{13,16,17} Other responsibilities of the superior parietal lobe include: spatial processing, tactile recognition, proprioception, kinesthesia, and visual guidance to motor actions.^{16,17} However, the evident redundancy of function in the brain may relate to the varied responses to the severity of the symptom manifestation.

Inferior Parietal Lobule

The inferior parietal lobule is sometimes referred to as the posterior parietal lobe, and it is divided into a dominant (left hemisphere) and non-dominant lobe (right hemisphere).

The dominant lobe is typically responsible for perception, interpretation of sensory information, and the formation of the idea of a complex meaningful motor response to sensory and spatial stimuli.¹⁶ This is distinct from the frontal lobes, that are more involved in the actual execution of a given task.¹⁸ The non-dominant lobe is thought to be more responsible for visual-spatial tasks.¹⁶

Typically, those with damage to the non-dominant hemisphere manifest visual spatial neglect or inattention. Visual spatial inattention is a reduced or absent response to visual stimuli presented on one side of visual space, significantly more so than the other, in the absence of significant motor or sensory deficits to corroborate this inattention.^{13,16-19} In severe cases of neglect, the patient often ignores stimuli in multiple sensory domains.¹⁹ For example, a person, whose non-dominant side of the parietal lobe is right, may have an injury of the right inferior parietal lobe resulting in a left-sided inattention for tactile, visual, and auditory stimuli.

Additional deficits of a non-dominant inferior parietal lobule insult include constructional apraxia, asomatognosia, neglect of the contralateral limb, lack of three dimensional sense, geographical agnosia, phonagnosia, dressing apraxia and anosagnosia.

DUAL PROCESSING OF VISION

To explore the visual processing of space and motion, one must refer to the ventral pathway (also referred to as the “temporal” or “what” pathway) and dorsal pathway (also referred to as the “spatial” or

“where” pathway). Goodale and Milner^{17,18} have scrutinized the “what” vs. “where” dichotomy. They place greater prominence on how information is transformed and used distinctly by the two systems. Further, Goodale and Milner suggest that the differentiation should be between visual identification and perception in the ventral pathway and visual control of skilled action in the dorsal pathway.

The ventral pathway receives most of its input through the parvo-retinogeniculate pathway. It begins in the primary visual cortex (V1) and proceeds ventrally to inferotemporal cortex. Its main responsibility is in the identification and recognition of objects. The dorsal pathway receives input through the magno-retinogeniculate pathway. The cortical pathway originates in V1, then to V5, to the middle temporal area, (MT), the prefrontal cortex and to the inferior parietal lobules. The pathway’s main responsibility is motion perception and visual spatial localization.^{17,18,20-22}

As mentioned above, the ventral pathway receives input from parvocellular projections, which are characterized by slower transmission due to their smaller axon diameter.²⁰ Its sustained responsiveness to temporal frequency gives it its poorer sensitivity to high temporal frequency stimuli; in other words it is a poor detector of changes in motion.²⁰ Additionally, parvocellular receptive fields are smaller in size, making them more sensitive to high spatial frequencies. This increased sensitivity to high spatial frequencies provides more information for identifying or recognizing objects.²⁰⁻²²

Magnocellular projections, the main source for the dorsal pathway, have a faster transmission due to their larger axon diameter. However, the transient responsiveness of the dorsal pathway axons makes it better for higher temporal frequencies and more efficient for motion detection.²⁰ Information from this area provides stimulation to the frontal eye fields, optimizing visual-spatial localization.^{18,20}

SENSORIMOTOR VISUAL DEFICITS EVIDENT FOLLOWING ABI

The more common sensorimotor visual deficits evident following ABI may include, but are not limited to, impairments of: accommodation, versional ocular motility, vergence ocular motility, visual-vestibular interaction, and visual field integrity.

Accommodative, Versional Oculomotor, Vergence Oculomotor and Visual Field Disorders

Deficits of the accommodative and ocular motor (versional and vergence) systems have been reported with a relatively high occurrence following TBI and stroke.^{1,6} Given the involvement of the parietal lobe, especially the superior parietal lobule, in ocular motor function (see Appendix), ocular motility and accommodation are often impacted negatively with insult to the parietal lobe. Insult to optic radiations near the parietal lobe may cause an inferior quadrantanopia. Visual field defects will become more congruous when injuries occur more posteriorly along the visual pathway. In terms of visual neglect, injury to the right posterior parietal lobe may result in a neglect/inattention of the left visual field.^{13,16-19}

Visual-Vestibular Interaction

The vestibular system is considered to be the center of balance, but there is no localized primary vestibular cortex. Thus, the neurological control of vestibular function is integrated into many aspects of the brain including, but not limited to the temporal, parietal (specifically the post central gyrus and posterior parietal lobe) and frontal lobes. These projections incorporate somatosensory, vestibular, and visual sensorimotor contributions towards stabilizing balance.^{1,23} Symptoms of vestibular dysfunction, depending upon the specific etiology, may include any or all of the following: dizziness, vertigo, light-headedness, disequilibrium or imbalance. While there are many etiologies to these types of symptoms, it is often beneficial to rule out the contribution of visual deficits to those with vestibular dysfunction, given the interaction between the visual and vestibular systems.

The most dominant connection between the visual and vestibular systems is the vestibular ocular reflex, also referred to as gaze stabilization.^{1,6,24} The reflex is the fastest reflex of the body, with the principal purpose being to maintain a steady image on the retina during head movement.²⁴ Oculomotor deficits may impede the ability to perform the vestibular ocular reflex increasing disequilibrium and discomfort in multiply-visually stimulating environments with or without motion.⁶

TREATMENT AND MANAGEMENT

Management of patients with ABI is interdisciplinary. The complexity of blended treatment can be quite challenging, with no single approach being suitable for all patients. Communication with the interdisciplinary rehabilitation team members is beneficial to optimize the rehabilitative progress. Vision deficits impeding ADLs and other rehabilitative aspects may be shared among optometrists and the other involved rehabilitation professionals.¹ Optometric management of those with ABI involves prescribing spectacles (with tints and/or prism), periodic re-evaluations, and home-based support and/or in-office neuro-optometric vision rehabilitation.

Spectacles, Tints and Prism

Optimizing clarity of vision is the initial step, often with separate single vision spectacles being prescribed for distance and near, as indicated. Multifocal lenses (including bifocal lenses and progressive addition lenses) are contraindicated typically for ambulation in those with ABI. Those with ABI often present with gait and/or vestibular disturbances. These may become exacerbated by the distortions of a progressive addition lens.⁶ Vestibular dysfunction and increased sensitivity to fluorescent lighting tend to favor incorporation of 15-20% blue tints into spectacles. Brown or gray tints may also be used. For patients with general overall photosensitivity, tints may be incorporated into the patient’s spectacles. Most often a 30-40% tint is indicated for indoor wear and 75-80% tint for outdoor use.⁶ Fusional prism in spectacles may benefit patients with diplopia.⁶ Many practitioners prefer a trial to evaluate the benefit of a prism prescription using Fresnel prism application. If the patient responds favorably to the Fresnel prism trial, then prism should be incorporated into the patient’s spectacle prescription.

Visual inattention with or without a visual field defect requires prescribing full-field, yoked prism (ground-in or Fresnel) to benefit the patient.⁶ Those with visual field defect with or without inattention may benefit from field expanding lenses or spotting prisms to increase their awareness of the affected visual field.⁶ However, the prism should be dispensed only after there has been sufficient amount of training with the lens to ensure optimal and efficient use.

Neuro-Optometric Vision Rehabilitation

Neuro-optometric vision rehabilitation is an interactive process that integrates vision with the other senses, typically motor and audition. Therapy encompasses three phases: enhancing the stability of the visual input system; developing visual processing; developing the speed/automation of the visual processing while integrating with motor and audition and maintaining stable output.

Phase one begins working on bottom-up processing that involves building up basic vision skills such as versional ocular motility, accommodation, and vergence. Phase two focuses on more advanced skills such as visual simultaneous memory, visual sequential memory and visual motor integration.²⁰ Phase three works on enhancing top-down processing. Top-down processing integrates higher-level visual skills (e.g., visual speed, visual planning, visual spatial processing, visual figure ground, visual closure) with basic vision skills tasks.²⁰ These activities emphasize the use of stimulus-generated versional and vergence ocular motility associated with higher-level visual tasks such as auditory distractions and balance. This requires cerebral input to direct the gathering and processing of information for the practitioner's multi-sensory approach. This gathering and processing of sensory information is desired to direct visual attention to a given stimulus and vision task. During rehabilitation, both bottom-up and top-down visual types of processing are addressed to improve a patient's ability to attend and orient in familiar and unfamiliar environments.

CASE REPORT: PL

Case History

PL is a 54-year-old male who presented on May 28, 2009 at the Raymond J. Greenwald Rehabilitation Center at SUNY's State College of Optometry. PL was diagnosed with a right parietal glioblastoma. The tumor was removed during a craniotomy on March 19, 2009. Subsequent to the tumor removal, PL underwent radiation and chemotherapy.

His presenting visual symptoms included: lack of vision in the left inferior field; difficulty with depth perception; decreased speed of reading; difficulty maneuvering through busy environments such as crowded streets and shopping malls; difficulty processing information in environments with multiple types of stimuli; left-

Table 1. Neuro-Vision Rehabilitator Modules

Module	Description
Visual Motor Enhancer (VME)	Visual rotator with options that increase/decrease the difficulty by varying the speed, direction of rotation, array of target size/shapes (numbers, letters, hollow, solid, red/blue), and presentation of target (random-jump saccades, sequential- pursuits).
Ocular Vestibular Integrator (OVI)	Projected bull's eyes in the periphery of the screen that illuminates randomly. The patient "shoots out" the targets with Wii remotes.
Dynamic Ocular Motor Processing (DOMP)	Presents procedures that require higher level visual processing with the use of letters, numbers and shapes. This module is designed to generate saccades, enhance visual scanning, visuomotor control, visual sequencing, as well as dynamic sensory binocular fusion.
Visuomotor Integrator (VMI)	Presents procedures that enhance ocular motor control, visually guided graphomotor performance and visual spatial processing.
Fixation Anomalies (FA)	Improves fixation anomalies associated with intrusion fixation, nystagmoid fixation, and crowding phenomenon.

right confusion; disequilibrium; as well as discomfort and insecurity going up and down stairs. In addition to the right parietal glioblastoma, PL's medical history was significant for: carotid artery disease, congestive heart failure, and hypercoagulability syndrome with a history of a deep vein thrombosis. His ocular history was significant for a left optic nerve coloboma and associated reduced best-corrected visual acuity of the left eye. His current medications included: Dexamethasone, Docusate sodium, Omeprazole, Keppra, Oxycodone-acetaminophen, and Folic acid. He reported no allergies to medications.

OPTOMETRIC EXAMINATION

A refractive analysis revealed a best corrected visual acuities of 20/20 in the right eye (OD) and 20/30 in the left eye (OS) at both distance and near. The refraction was OD: +1.25 DS and OS: +0.25-2.50x010 with an add of +2.00 DS OU. There was no restriction of extraocular motilities for either eye, and there were no signs of strabismus or nystagmus evident in primary gaze. However, a convergence insufficiency, deficit of saccades, slower speed of overall visual processing, impaired visual memory, impaired laterality, and visual-vestibular disturbances were evident.

In terms of ocular health, confrontation visual fields revealed a restriction for each eye of the left inferior quadrant and confirmed with automated perimetry. Pupils were equal, round, and reactive to light, with no relative afferent pupillary defect. Anterior and posterior segment assess-

ment revealed bilateral incipient cataracts and left optic nerve coloboma.

Due to PL's demands for near work on a computer and reading material, he was prescribed an occupational progressive lens spectacle. PL was also prescribed separate pairs of spectacles for distance and near. He was advised against using his progressive lens spectacles during ambulation because the peripheral distortions may exacerbate his symptoms of disequilibrium. In addition, PL was prescribed 30-40 sessions of neuro-optometric vision rehabilitation for his visual skills and processing deficits.

NEURO-OPTOMETRIC VISION REHABILITATION

Phase I of NOVR: Visual Input

Visual input refers to the initial visual stimulus that triggers the sensorimotor vision response. For example, an image appears 40 centimeters in front of a patient. He/she must first localize the image accurately and then subsequently apply the requisite amount of accommodation, fixation, and convergence to view the image clearly and singly. All these basic skills are required to maintain a static, single, and clear image of the target. Automaticity of versional ocular motility is required to maintain fixation on a dynamic target. In Phase I, PL worked to advance these skills by utilizing the Neuro-Vision Rehabilitator (NVR).³ The instrument is a state of the art visual therapy system that utilizes a Wii system to apply an interactive approach. While presently there are vision therapy procedures that adequately normalize ocular motor, accommodation and binocular control, the modules of the

Table 2. Pre-Therapy and Post-therapy (30 sessions) Findings

Optometric Evaluation Test	May 2009 (Pre-therapy)	May 2010 (Post-therapy)
Von Graefe phoria (D)	4 exo	3 eso
Von Graefe phoria (N)	12 exo	4 exo
Near point of convergence	4 ⁷ / ₆ " difficulty sustaining fusion, (-) physiological diplopia	3 ⁷ / ₄ " sustained fusion, (+) physiological diplopia
Negative fusional vergence (D)	x/12/6	x/13/12
Positive fusional vergence (D)	x/18/-4	x/25/8
Negative fusional vergence (N)	Not obtained	x/8/6
Positive fusional vergence (N)	x/2/-10	x/6/4
Stereopsis	100" arc	100" arc
Piaget Left-Right Test	Section I pass Section II pass Section III failed- too confusing, could not complete	Section I pass Section II pass Section III pass
Manifest refraction:	OD: +1.25 DS OS: +0.25-2.50X 010 ADD: +2.00	OD:+1.25DS OS:+0.25-2.50x010 ADD:+2.00

instrument (Table 1) were developed to integrate vision, audition, proprioception, balance, and visuomotor control using the uniqueness of Wii remotes, balance boards, and sensors. PL developed accurate ocular motor skills, extended the ranges of extraocular motility, and improved visual scanning skills with the modules as shown in Table 1. Each module has levels that increase in complexity and cognitive demand. Its development stems from a model proposing that repetition, multi-sensory feedback, and active participation in sensory motor tasks are essential for affecting synaptic changes. Further, the neuro-plastic synaptic changes translate into increased information processing and performance.

Phase II of NOVR: Visual Processing

Visual processing requires adequate ranges, speed, stability and fluidity of the accommodative and vergence systems. During the second phase, PL performed therapy techniques involving various sensory feedback cues. Techniques included vectograms (emphasizing the perception of SILO, float, stereopsis), Brock String (increasing awareness of physiological diplopia), Computer Orthopter vergence modules^a (improving stereopsis), and the neuro-vision rehabilitation (enhancing these visual processing skills and integrating vision with other sensory modalities).

All techniques during this phase were geared towards these goals of developing sustained fusional vergence ability.

Phase III of NOVR: Top-Down Processing

By mastering phases I and II, PL no longer exerted as much effort to accomplish relatively simple vision functions such as versional ocular motility, accommodation and vergence ocular motility. Now, PL was able to apply effort towards higher level visual processing such as: visual speed, visual planning, visual spatial processing, visual figure ground, and visual closure. During phase III, both vestibular and multisensory stimulations were incorporated in the majority of the therapy procedures, accomplishing top-down processing. Some examples included the Wii balance boards, head rotations with fusion activities, as well as auditory and visual distracters. PL used the upper level of the Visual Motor Enhancer, Ocular Vestibular Integrator and the Dynamic Ocular Motor processing modules of the NVR. The instrument contains a device that senses the motion of the head. A balance board is used during this third phase of therapy. Other more familiar therapy procedures include transparent eccentric circles (at near and far), red/green filters with yoked prisms, head rotations and computerized programs for visual processing.

REPORTED FUNCTIONAL CHANGES

After 30 sessions, PL's visual function was re-established successfully. Of course, limitations were set by the craniotomy and subsequent damage to the parietal lobe. However, with persistence and repetition, PL was provided with strategies to cope with the presenting vision deficits. Areas such as spatial relationships, visual memory, visual discrimination, visual closure and figure ground were all measured with the Test of Visual-Perception 3rd ed.,^b and were found to be above average. PL's perceived areas of marked improvement included increased attention span, increased memory, improved eye-hand coordination, and improved posture/stability. He reported no further restriction due to his homonymous left inferior quadrantanopia because of his improved scanning and peripheral awareness. Table 2 lists other findings pre- and post-therapy. Overall, PL reported that the vision therapy has improved his ADLs. PL was dismissed after 30 sessions and provided home maintenance vision therapy and has returned to work full time.

CONCLUSIONS

ABI refers to a sudden-onset, non-degenerative, and non-progressive neurological injury. Following ABI, aspects of sensorimotor vision function may be impacted negatively, including: accommodation, vergence, versional ocular motility, visual vestibular interaction, scotopic sensitivity, and visual field integrity. Deficits of vision function evident following ABI may also further impede the person's ability to perform rehabilitative techniques, as well as basic ADLs.

In terms of optometric evaluation and management for those with ABI, a careful primary eye care assessment is needed, followed by certain sensorimotor and higher level visual perceptual tests. Treatment options commence with spectacle prescription with or without tints or prisms and, depending on your patient's needs, neuro-optometric vision rehabilitation.

NOTE

Dr. Allen H. Cohen is the developer of the Neuro-Vision Rehabilitator and has a financial interest in that instrument. However, neither he nor the other authors have a financial or other interest in the remaining instruments or products discussed in this article.

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Date accepted for publication:
October 29, 2010

Appendix. Potential Functional Impairments with Parietal Lobe Injury

IMPAIRED AREA OF THE PARIETAL LOBE	POTENTIAL FUNCTIONAL IMPAIRMENTS	DESCRIPTIONS
Post-central gyrus	Impaired proprioception	Impaired sensation of posture
	Impaired kinesthesia	Impaired appreciation of passive movement
	Tactile sensation deficit	
	Two-point discrimination deficit	
	Astereognosis	Inability to perceive the form of an object by touch
	Agraphism	Inability to write
	Impaired visual attention	
Superior parietal lobule	Optic ataxia*	Inability to accurately reach for objects
	Oculo-motor dysfunction*	Inability to control gaze and/or saccades
	Simultanagnosia*	Inability to integrate components of the visual scene
	Difficulty with visual guidance of hands, fingers, eyes, limbs, and head*	
	Impaired tactile recognition	Limited recognition of objects by touch
	Impaired proprioception	Impaired knowledge of limb position
	Difficulty directing movement in space	
	Left-right confusion	Difficulty distinguishing left from right
Dominant inferior parietal lobule (usually left brain)	Acalculia	Inability to count
	Dysgraphesthesia	Inability to recognize letters or numbers written on one's hand
	Left-right confusion	Difficulty distinguishing left from right
	Finger agnosia	Inability to distinguish fingers on one's hand
	Alexia/Dyslexia	Inability to read
Non-dominant inferior parietal lobule (usually right brain)	Geographical agnoisa	Inability to locate defined places
	Phonagnosia	Inability to identify voices
	Constructional apraxia	Inability to build a whole from its component single parts
	Asomatoagnosia	Lack of awareness of one's body and all body parts
	Anosognosia	Lack of awareness/denial of illness especially hemiparesis/hemiplegia
	Spatial neglect	Having impaired spatial awareness, attention, and representation on the contralesional side of space
	Impaired appreciation of three-dimensional sense	
	Dressing apraxia	Difficulty dressing on the contralesional side of the body

*Components of Balint's syndrome