Brain Injury Rehabilitation: Cortical and Subcortical Interfacing via Retinal Pathways

Deborah G. Zelinsky, OD

Abstract: Most patients with traumatic brain injury (TBI) have visual and nonvisual retinal signal processing problems with concomitant dysfunctional sensory systems integration. These dysfunctions often include problems of spatial orientation, motor planning, and motor control. They are generally not visible on diffusion tensor imaging, diffusion spectrum magnetic resonance imaging, or functional magnetic resonance imaging and are not discernible by neuro-ophthalmological, standard optometric, or ophthalmological testing. In contrast, the neuro-optometrist, while examining the TBI patient's conscious cortical processing, puts great emphasis on unconscious ambient processing to assess the patient's ability to tolerate and adapt to environmental changes beneath conscious awareness. This often overlooked, yet potentially critical, information from both subcortical and cortical components of the visual system, sometimes combined with other sensory signals, is used to evaluate and treat patients with processing dysfunctions.

PM R 2010;2:852-857

INTRODUCTION

In January 2009, this journal presented a Point/Counterpoint addressing the relative merits of behavioral versus pharmacological interventions in patients after traumatic brain injury (TBI) [1]. One author presented a chemical approach to safely managing a multitude of obstacles during rehabilitation, such as pain, sleep disturbances, confusion, and aggression. The other author offered a range of clinical interventions within a behavioral approach to treatment. Both authors acknowledged that impairments in arousal, cognition, and motor control are well-known sequelae of TBI, and both acknowledged that drugs used to lessen agitated behavior usually negatively impact overall function and sometimes worsen impairment problems. This focused review presents a third alternative, a neuro-optometric approach to identifying and managing sensory systems dysfunctions during TBI rehabilitation. Most traumatic brain injury (TBI) rehabilitation is dependent on patient involvement in the conscious (ie, focal) processing of specific tasks while concurrently filtering out unimportant background signals. Often it is that ability to pay attention while filtering multiple stimuli that is dysfunctional, leading to increases in confusion, frustration, agitation, and fatigue. Neuro-optometric rehabilitation complements traditional TBI rehabilitation by altering unconscious and subconscious (ie, ambient) processing to help patients compensate for these higher level processing deficits.

Ambient visual processing concerns information used to maintain balance, while orienting the body to gravity, during movement and navigation. An instability in ambient processing can lead to an imbalance between internal and external sensory signal interaction that can be treated by a range of neuro-optometric interventions. Neuro-optometry is distinct from general optometry and ophthalmology, in which the emphasis is on general eye health and focal processing, that is, how a patient sees details in the environment. It differs as well from neuro-ophthalmology, which primarily diagnoses and treats eye and visual system damage or diseases from systemic conditions originating from problems in the nervous system.

Most TBI patients have visual processing problems [2]. This finding is not surprising because visual pathways exist throughout the brain and are easily disturbed by trauma. There is usually a disruption in ambient processing and an inability to synchronize

D.G.Z. The Mind-Eye Connection, 1414 Techny Road, Northbrook, IL 60062. Address correspondence to D.G.Z.; e-mail: mindeyeconnection@msn.com

Disclosure: nothing to disclose

Disclosure Key can be found on the Table of Contents and at www.pmrjournal.org

Submitted for publication August 30, 2009; accepted June 23, 2010.

PM&R
1934-1482/10/$36.00
Printed in U.S.A.

Vol. 2, 852-857, September 2010
DOI: 10.1016/j.pmrj.2010.06.012

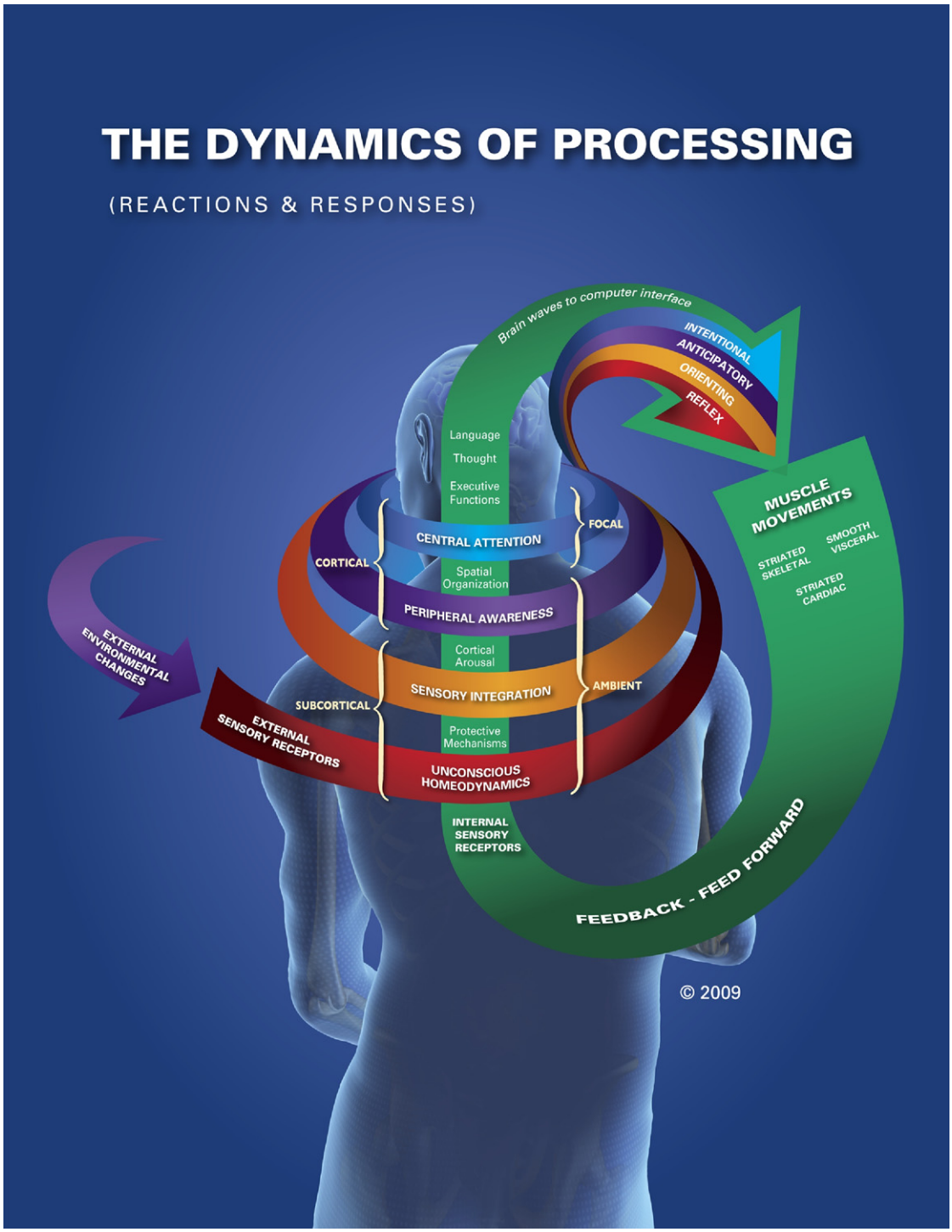

Figure 1. Internal sensory signals are constantly occurring, stimulating internal sensory receptors. Once the internal receptors are stimulated above their range of comfort, sensory stimuli creep into the cortical arousal level, leading to executive functions and eventual movements. Meanwhile, signals from the surrounding environment enter through external sensory receptors, bombarding the sensory systems and diverting a portion of nonconscious attention from central focusing. Neuro-optometrists can intervene at various levels by using lenses, nonyoked prisms, yoked prisms, or filters, thereby controlling incoming signals (with specialized eyeglasses) and measure the induced motor response. By testing an individual's ranges of comfort and tolerance within these interventions, the neuro-optometrist can identify processing deficiencies for further treatment. Reprinted with permission from The Mind-Eye Connection, Northbrook, IL.

nonvisual subcortical signals with peripheral or central eyesight cortical signals, causing nonvisual symptoms such as pain, fatigue, sleep disturbances, confusion, dizziness, and aggression. Neuro-optometric testing can use brain mapping to aid in determining both the area of brain damage and the method and direction of neuro-optometric intervention—the subject of this article.

Visual pathways provide an entrance to brain processes and a means to quantify the interplay of ambient processing with central eyesight in addition to ranges of movement [3]. The retina is an extension of brain tissue, precisely mapped onto the visual cortex, with a parallel retinotopic map on the superior colliculi [4]. In graphic terms, the eye is a 2-way street providing a pathway for looking in as well as looking out, using both afferent and efferent nonvisual fibers, from and to the retina, in addition to the eyesight-related afferent retinal fibers. Its appearance and movements offer information about what is happening in both internal and external environments. For example, light signals entering the eye from the inferior visual field travel through the optic nerve and optic radiations and generally interact with the parietal lobes; light signals from the superior visual field generally interact with the temporal lobes [5]. Signals from the retina are transferred point to point to predictable locations in the brain via predictable bundled retinal fiber pathways [6,7]. Therefore, when light is angled onto the retina in different ways, various regions of the brain are being stimulated, which becomes the basis of neuro-optometric rehabilitation [8-10]. Conversely, even mild damage to the parietal or temporal lobes will have an effect on the balance between the ambient and focal visual pathway signals.

COMPLEXITY OF SYSTEMS

To maintain homeostasis, the body is constantly occupied with interrelated, simultaneous systems, each in its own cycle, beneath conscious control—a process termed homeodynamics (Figure 1) [11]. All systems have individual ranges of tolerance to both external and internal changes. With TBI, internal processes are fragile, and once stimuli exceed individualized tolerance ranges, there may be a decreased ability to reorient and regain dynamic equilibrium on any of the levels [12].

Each level of the hierarchy of brain function, as shown vertically in Figure 1, internally generates movements. These movements are part of a continuous interactive loop, which has to adjust to changes in external stimuli. Patients respond consciously or react unconsciously to and within this unending cycle. Higher processing levels, such as peripheral awareness of and central attention on external surroundings, occur only after cortical arousal. Most postacute rehabilitation is dependent on conscious processing involved in achieving a specific task while simultaneously filtering unimportant background signals.

The traditional interdisciplinary approach facilitates regaining intentional control of conscious movement. In contrast, neuro-optometric rehabilitation often uses lenses, prisms, and filters to alter the direction, amount, and intensity of incoming light signals, which affects unconscious movements by addressing tolerance ranges beneath conscious awareness.

For example, walking through a doorway is not as simple a task as placing one foot in front of the other. A much larger ambient processing first needs to occur through a combination of internal and external signals, culminating in the necessary presequence to movement—assessing the door position, determining how high to lift the foot and how far to place it forward, and the timing of weight shifts—while simultaneously using focal processing to concentrate elsewhere to effortlessly move through the doorway. After TBI, that same task sometimes requires focal processing to replace or aid the now nonautomatic ambient processing.

All of this orchestration of movement is unconscious in a noninjured person. After TBI, this previously unconscious processing becomes disjointed and slowed and can cause confusion or agitation. Because this balanced synchronization relies on peripheral visual signals for external input and proprioceptive and vestibular signals for internal input for body, head, and eye position, neuro-optometric intervention in these areas during TBI rehabilitation can often help the patient regain this nonconscious effortless movement orchestration.

WHY THE EYE: VISUAL AND NONVISUAL PATHWAYS

Conventionally, eye function has been mainly associated with visual acuity, which is a static, conscious, and cortically based process. In actuality, the eyes are connected to eyesight and noneyesight systems through 47 differentiated areas of the brain, with 305 known pathways, including visual and nonvisual retinal pathways involved in some aspect of visual processing [13]. The retinal pathway, in this sense, includes both the afferent pathway, from the retina to either a cortical or subcortical area, and the efferent pathway, from that area, coursing through many locations back to cortical eye fields, leading to an eye movement. Visual pathways offer quantifiable ranges of movement and more direct access into brain activity. During neuro-optometric evaluations, clinicians use stimulation of these pathways to investigate and influence ambient processing and its interaction with central eyesight. This process is complementary to the work of other eye care professionals.

Light signals enter the eye, chemically activating more than 130 million retinal photoreceptors, and visual information is transmitted into 1 million retinal ganglion axons through a cascade of chemical changes culminating in electrical impulses. These exit the optic nerve, 90% of them encountering other

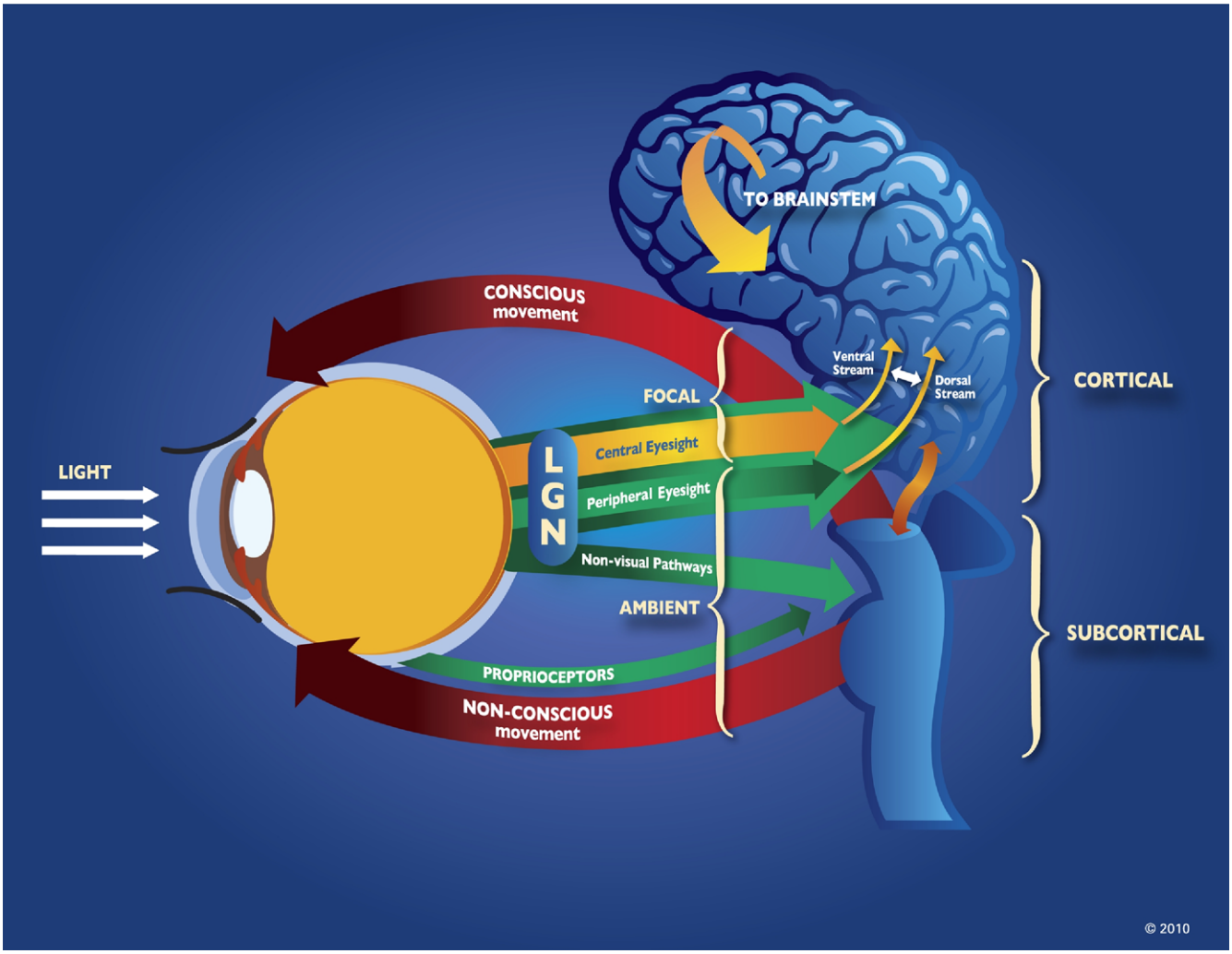

Figure 2. Light entering the eye takes 3 main paths: 1) Central eyesight is the largest, comprising details and colors. 2) Peripheral eyesight includes shapes, location, orientation, speed and size of targets. 3) Nonvisual pathways include connections to and from brainstem and cerebellar nuclei, involved in systems such as auditory, postural, and emotional centers. On entry into the brain through the eye, the mapping is point to point based on spatial location of targets. Signals returning from the brain generating eye movement are sorted based on attention—with information from the target of regard traveling through the temporal lobe (ventral stream) and the background information routed through the parietal lobe (dorsal stream). Peripheral vision (external world) and nonvisual pathways (internal world) combine during ambient processing to contribute to the foundation of spatial orientation. The fibers from all peripheral and nonvisual systems travel through many parts of the brain and together lead to involuntary, nonconscious eye movements. Any damage to these brain structures can disrupt ambient processing which, until stabilized, can cause deficiencies in central eyesight stability and control of conscious eye movements (focal processing). Typical eye tests address conscious eye movements. Neuro-optometry addresses the interaction between ambient and focal processing. For simplicity, Figure 2 shows only retinofugal signals exiting the retina; however, a small percentage of nonvisual retinopetal fibers actually originate in the brainstem and cerebellum and travel to the inner retina, where they effect chemical changes. (See Vereczki V, Koves K, Csaki A, et al. Distribution of hypothalamic, hippocampal and other limbic peptidergic neuronal cell bodies giving rise to retinopetal fibers: Anterograde and retrograde tracing and neuropeptide immunohistochemical studies. Neuroscience 2006;140:1089-1100.) Also, for simplicity, the lateral geniculate nucleus (LGN) is shown close to the retina rather than in its actual position on the caudal portion of the thalamus, and the optic radiations are not shown. Reprinted with permission from The Mind-Eye Connection, Northbrook, IL.

sensory signals in the lateral geniculate nucleus, a relay center located in the thalamus [14], then separate into 3 main segments (Figure 2).

The most common and largest segment is central eyesight, in which signals travel from retina to visual cortex via optic radiations and then to temporal lobes via the ventral stream. The second segment is a group of axons representing peripheral eyesight, wherein signals travel from retina to visual cortex via optic radiations and then to parietal lobes via the dorsal stream. These 2 groups enable

cortically controlled eyesight functions and represent more than 80% of total retinal signals. The third segment, an aggregate of nonvisual fibers, branches to various brainstem and cerebellar nuclei [15] and is used in balance and spatial orientation.

Information passes through these 3 general pathways at different speeds. Nonvisual subcortical signals process more quickly than slower cortical signals [16]. This synchronization is significant, and its disruption often causes disordered cognitive processing [2]. For example, during postacute physical therapy, a patient may be unable to follow the practitioner's directions, which might occur because the patient's focal auditory processing (an example of slower moving cortical signals) of the therapist's voice is being disrupted by faster moving peripheral external stimuli such as people moving, music, or changing light. The patient is not necessarily aware of the processing dysfunction. He or she can then become confused, agitated, frustrated, or angry at the practitioner, or at him- or herself, for no apparent reason, and not be able to perform to full potential because of this sensory processing disruption and concomitant agitation. In this typical scenario, neither practitioner nor patient understands the source of the problem. In preparation for this type of therapy, neuro-optometric intervention can alleviate some of the confusion by stabilizing ambient processing deficiencies and determining areas in the retinal pathways that are dysfunctional because of TBI. Hypersensitivity can be diminished by prescribing customized lenses, not designed for eyesight, but rather for directing the light signals away from those pathways. Thus, the focus of the patient on the practitioner's instructions will not be disrupted.

Peripheral eyesight (the second segment) is a cortical activity involved in ambient visual processing needed to orient the body to gravity during movement and navigation. Patients with TBI usually have disruption in ambient processing that affects their ability to concentrate on slower moving focal processing signals, and they are more likely to be disturbed by the faster moving and now confusing external ambient signals.

Among the aggregate of nonvisual pathways (the third segment) contributing brain function information, the following 4 examples are particularly relevant in rehabilitation. Each can be altered by neuro-optometric intervention. The first, the retino-tectal (or retino-collicular) pathway is involved in maintaining postural stability by reflexively shifting eye position to orient the body. For example, some patients with poor balance might be adapting to the way the floor appears to tilt. Also, a gait disorder after TBI may be altered through the retino-tectal pathway by the use of customized glasses that intentionally make the floor appear tilted, thereby shifting weight bearing and visual perception, until reorientation is achieved. This pathway also has a point-to-point retinotopic representation as a parallel visual back-up navigation system called blindsight, which can be useful when the visual cortex is damaged [17].

A second subcortical retinal pathway, the accessory optic system, is a conglomeration of connections involved in reflexive fine-tuning of eye movements that stabilizes retinal images when head or body position shifts. The accessory optic system contains extensively interconnected nuclei with many efferent and afferent signals. Gaze-stabilization techniques, for example, used to remediate sensory mismatches in visual-vestibular interactions, address head tilt problems by targeting efferent pathways [18].

The third and fourth pathways are intertwined and involved with the autonomic nervous system. The retino-pretectal (third) pathway, from the retina to the Edinger-Westphal nucleus, controls the pupil sphincter via the parasympathetic nervous system. The retino-hypothalamic (fourth) pathway governs many functions, including the pupil dilator muscle, controlled by the sympathetic nervous system. Pupil reactions can be valuable biomarkers in assessing the state of the nervous system. Although the use of medications can sometimes confound pupillary function, relative changes in pupil size can be used by the neuro-optometrist to analyze how these adjustments reflect the patient's inner and outer environments [19]. Also, in TBI patients, the iris muscles governing pupil size can be easily fatigued and may be a hidden cause of discomfort and agitation.

SOME PRACTICAL APPLICATIONS

After TBI, patients often are not referred for visual intervention because their eyes appear normal and there are no direct eye complaints and no visual field losses. However, seemingly nonvisually related problems usually exist that can be identified and treated neuro-optometrically. Visual and nonvisual pathways combine with other sensory signals influencing perception and behavior.

For example, neck and back pain caused by compensatory head rotation often is caused by a patient perceiving an object as being farther away with one eye than the other (a functional visual midline shift). Specialized glasses angling light signals from the side can be used to treat the compensatory behavior by stimulating reflexive head rotation in the opposite direction.

As another example, when a patient complains of dizziness and gaze stabilization techniques have minimal effects, the application of a partial occlusion filter to block entering light signals from striking a hypersensitive area of the retina can alleviate the symptoms by lessening the effect of incoming stimuli.

If a patient complains that eyeglasses produce headaches, even though they provide sharp, 20/20 central eyesight, the problem might be attributable to a delicate balance between ambient and focal processing. A small change in lens prescription can be prescribed to intentionally (but nonconventionally) blur central eyesight slightly, allowing better comfort and more stable ambient processing. Eventually, the lens prescription can be sharpened, once the patient is able to tolerate the change.

Reading comprehension problems after a mild brain injury can be caused by a disruption in ambient visual processing,

which can affect ability to automatically navigate eye movements on a page. The unexpected focal attention on eye movements detracts from the ability to visualize and concentrate. The use of specialized eyeglasses designed to angle light differently can help stabilize ambient processing and allow smoother eye movements and more focal processing on content.

CONCLUSION

The eye is a surprisingly accessible gateway to the nervous system, providing the potential to both measure and affect all levels of the complex processing dynamic, not only those related to central eyesight. This can be particularly useful in treating TBI patients, when agitation, including anxiety, confusion, and loss of concentration can be a secondary response to internal functional impairment, specifically when ambient processing is disrupted or imbalanced. Nonvisual pathways may be malfunctioning, regardless of the patient's visual acuity. By modifying the amount and direction of entering light signals, then measuring motor reactions and responses, the neuro-optometrist can assess the role of visual processing in the maladaption to change and possibly enhance sensory linkage and efficiency. By the use of lens prescriptions, filters, tints, prisms, and other techniques, the neuro-optometrist can remediate or compensate for many visual and sensory misperceptions, freeing the patient's cognitive reserves for the important work of other rehabilitation professionals in restoring functional and normal interfacing of the patient's internal sensory perceptions and external surroundings.

ACKNOWLEDGMENTS

Thanks to Dr. Albert A. Sutton in Tamarac, Florida for introducing me to his interdisciplinary approach to patient care. His mentorship and teaching have been invaluable in opening my eyes to future possibilities in the neuro-optometric profession.

REFERENCES

1. Flanagan SR, Elovic EP, Sandel E. Managing agitation associated with traumatic brain injury: Behavioral versus pharmacological interventions? PM R 2009;1:76-80.
2. Zasler ND, Katz DI, Zafonte RD. Brain Injury Medicine: Principles and Practice. New York: Demos Medical Publishing; 2007, 515-516.
3. Padula W, Argyris S. Post trauma vision syndrome and visual midline shift syndrome. NeuroRehabilitation 1996;6:165-171.
4. Luo L. Developmental neuroscience: Two gradients are better than one. Nature 2006;439:23-24.
5. Swensen RS. Thalamic Organization. In: Review of Clinical and Functional Neuroscience. Dartmouth University Medical School; 2006. Available at: http://www.dartmouth.edu/~rswenson/NeuroSci/chapter_10.html. Accessed July 5, 2010.
6. Falchier A, Clavagnier S, Barone P, et al. Anatomical evidence of multimodal integration in primate striate cortex. J Neurosci 2002;22:5749-5759.
7. Wilson CL, Babb TL, Halgren E, et al. Visual receptive fields and response properties of neurons in human temporal lobe and visual pathways. Brain 1983;106:473-502.
8. Epstein R, Kanwisher N. A cortical representation of the local visual environment. Nature 1998;392:598-601.
9. Downing PE, Jiang Y, Shuman M, et al. A cortical area selective for visual processing of the human body. Science 2001;293:2470-2473.
10. Grill-Spector K. The occipital lobe. In: Aminoff M, Daroff R, eds. The Encyclopedia of Neurological Sciences. Boston: Academic Press; 2003, 653-660.
11. Homeodynamics. In: Mosby's Medical Dictionary. 8th ed. St. Louis, MO: Mosby Elsevier; 2009.
12. Khan F, Baguley IJ, Cameron ID. Rehabilitation after traumatic brain injury. Med J Aust 2003;178:290-295.
13. Klemm WR. Understanding Neuroscience. St. Louis, MO: Mosby; 1996, 150-151.
14. Yücel YH, Zhang Q, Gupta N, Kaufman PL, Weinreb RN. Loss of neurons in magnocellular and parvocellular layers of the lateral geniculate nucleus in glaucoma. Arch Ophthalmol 2000;118:378-384.
15. Hoddevik GH, Brodal A, et al. The pontine projection to the cerebellar vermal visual area studied by means of the retrograde axonal transport of horseradish peroxidase. Brain Res 1977;123:209-227.
16. Liu CSJ, Bryan RN, Miki A, Woo JH, Liu GT, Elliott MA. Magnocellular and parvocellular visual pathways have different blood oxygen level–dependent signal time courses in human primary visual cortex. Am J Neuroradiol 2006;27:1628-1634.
17. Leh, SE, Ptito A, Schönwiesner M, Chakravarty MM, Mullen KT. Blindsight mediated by an S-cone−independent collicular pathway: An fMRI study in hemispherectomized subjects. J Cogn Neurosci 2010;22:670-682.
18. Giolli RA, Blanks RH, Lui F. The accessory optic system: Basic organization with an update on connectivity, neurochemistry and function. Prog Brain Res 2005;151:407-440.
19. Hupe JM, Lamirel C, Lorenceau J. Pupil dynamics during bistable motion perception. J Vision 2009;9:1-19.