# 29 Evaluating and Treating Visual Dysfunction

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## INTRODUCTION

Complaints concerning vision are common following a traumatic brain injury (1). A traumatic brain injury can cause a wide variety of symptoms related to vision such as headaches, diplopia, vertigo, asthenopia, inability to focus, movement of print when reading, difficulty with tracking and fixations, and photophobia. Persons experiencing these types of visual problems frequently will have other medical sequela related to the TBI.

Epidemiological studies of vision problems relating to traumatic brain injury have been sparse in both literature and research. However, a limited number of studies demonstrate that vision dysfunction is common following a TBI and can affect any part of the visual process (2).

In one study, the most common complaint was blurred vision (46%) followed by diplopia (30%) and headaches (13%). Thirty-five percent of the subjects displayed a visual field defect of which the most common was a compression of peripheral fields followed by homonymous hemianopsia. Thirty-three percent of the subjects experienced cranial nerve palsies of which third nerve palsy (exotropia) was the most common. Ninety-five percent of these subjects had normal funduscopic findings while of those with abnormal funduscopic evaluations, twenty-six percent demonstrated optic nerve abnormality.

Binocular dysfunction is highly prevalent (3) following a TBI and relate to many of the symptoms regarding vision that a person may experience (4). Often, exotropia (an eye deviated outward) or exophoria (tendency for the eyes to deviate) occur following a TBI either through direct cranial trauma or whiplash (5, 6).

Following a TBI, visual sensory deficits can be caused by areas other than the stria of the occipital lobe which is often noted as the primary visual cortex (7). The extent and impact of a TBI on overall sensory function can be quite profound but due to the true nature and primary influence of visual processing, no interference can be as significant as that of the visual system following a

TBI. Mechanisms for sensory deficits require awareness and recognition of the problem by the affected person without which, there is a significant potential for interference in the overall rehabilitation process (8).

The use of vision to lead and guide motor function is often compromised following a brain injury (9) affecting visual perception and visual motor dysfunctions. New evidence has emerged demonstrating that neuromotor function also contributes to spatial awareness and affects egocentric concepts of visual midline (10) beyond the role of acuity. Visual midline is the perceived lateral and anterior-posterior visual concept of egocentric position relative to sensory-motor concepts. The influence of homonymous hemianopsia will also directly affect the perception of visual midline, posture, and balance.

Use of visual evoked potentials (VEPs) have disclosed that dysfunction in visual processing may be a cause of many of the conditions of strabismus following TBI (11). Researchers have also documented through VEP patterns that there are many visual abnormalities significantly related to the clinical disability of persons with TBI (12). The result of such research are leading many to begin questioning the traditional examination methods of examining persons with a TBI relative to diagnosing binocular dysfunction or to assess visual processing of the bimodal system of vision.

Recent studies (11, 13) involving visual evoked potentials (VEPs) have directed consideration that binocular dysfunction and visual midline shift are due to dysfunction of visual processing and not necessarily due to muscle or cranial nerve palsy following TBI. Until recently, research involving the bimodal process of vision had not been considered clinically relevant at the same level as binocular function. In particular, dysfunction of binocularity had been looked at primarily as the cause related to the symptoms. Understanding the true nature of the bimodal visual process may now give the documentation necessary to understand the nature of binocular function in relationship to cortical and subcortical mechanisms that organize vision and sensory motor information. In turn, the visual evoked potential may now give the researcher as well as the clinician the means to join in the study of process related to clinical function. In addition, it may also provide new means of understanding the significant interferences caused by TBI related to a wide variety of visual symptoms following a TBI.

It is common for persons to experience vertigo, dizziness, and other balance disorders following a TBI (14). While studies have emphasized the vestibular system as the primary mechanism for maintaining stability of the visual field, new information regarding the role of the pretectum and superior colliculus offers insight into vision stabilizing the retinal image and establishing peripheral fusion. In addition, the superior colliculus also mediates visual processing affecting posture, movement, and orientation to positional space (15). A new model of processing related to the sensorimotor feedback loop providing a feedforward system to other higher areas of cortical function has been proposed and gives real insight into the role of the motor system affecting sensory perception and stability (16).

## VISION: A BIMODAL PROCESS

Seventy percent of all sensory processing in the entire body is directly affected by information coming from the two eyes (17). These visual influences are directed to the midbrain and the majority extend themselves to the occipital cortex for purposes of seeing (15). As a dominant system in the sighted person, vision is often taken for granted. Fortunately, we do not have to think about the process of using vision as we automatically employ vision in our daily activities. The important dynamics of visual processing are masked behind our intense attention and concentration to the accomplishment of the task at hand. For example, the task of writing involves vision but we do not think about where we are looking when we move the pen on the page. Nor do we think of how we see print as we read. Do we see each individual letter on the page or do we see more globally and embrace whole words and phrases?

Trevarthen (18) and Liebowitz and Post (19) have disclosed that there are two modes of visual processing; the focal process and the ambient process. The focal process delivers information for the purpose of attention, concentration, and higher cognitive processing. Information is sent primarily to the occipital cortex for the purpose of seeing detail. This is the portion of the system which enables us to attend to a task or concentrate. If we had only the focal process, the world we see would break apart into a mosaic of fragments. We would see only the lines, shadows, and shapes on a person's face and although we would see clearly, we would not be able to recognize the inter-relationship of all of these details. We would not be able to recognize the person's face as a familiar gestalt.

Approximately twenty percent of fibers from the peripheral retinas of both eyes are delivered to the midbrain (20). Here, the visual process matches information gathered from the kinesthetic, proprioceptive, tactile, and vestibular systems. This function has been labeled the sensory-motor feedback loop (21). It serves to organize spatial information about balance, movement, and orientation in space. The visual process is very much involved in movement and postural control. The superior colliculus, a critical area in the midbrain system, receives fibers from the optic tract via the superior brachium, occipital cortex via the optic radiations through the lateral geniculate, and from the spinotectal tract connecting it with

sensory motor information from the spinal cord and medulla. It is in the midbrain that sensorimotor matching concepts of midline, body awareness, posture, and orientation are established. Once this is accomplished, a feedforward phenomenon occurs. Information is relayed from the midbrain to the occipital cortex where it is used to preprogram the higher seeing area as to how to organize or look at incoming visual information. This is first done spatially and has been termed the ambient visual process. Following this, focalization on detail will occur. In this manner, the occipital cortex will organize information spatially before it looks at the detail. In other words, it is as if we know where to look before we completely identify the target. Spatial information from this midbrain ambient visual system is also used in other processing levels at the occipital cortex for purposes of bringing together detail information relative to spatial boundaries. In particular, the binocular coordination cells that receive information imaging from both the right and left eye have the primary purpose of beginning to integrate or fuse the two separate images together as one. It is the spatial information from the ambient visual process delivered by the superior colliculus that becomes the binding format of the fusion process (22).

In fetal development, the eye is created from endoderm, mesoderm, and neuroectoderm. Of importance is the fact that neuroectoderm also develops the cortex which allows for recognition that the eyeball is neurologically an end result in part of developing brain tissue. The eyeball is richly endowed with nerve fibers that feed all aspects of the cortex as well as relaying information to midbrain structures. Nerve fibers emanating from the macula or central portion of the eye will align centrally in the optic nerve and optic tract directing themselves to localize in central areas of the visual occipital cortex. Peripheral retinal nerve fibers orient themselves in the optic nerve and optic tract around the central fibers and align themselves in peripheral areas of the occipital cortex.

While the central image from the macula occupies the major portion of our cortical attention and concentration for perception and cognitive processing, how we organize and process visual information is not a simple phenomenon. The dorsal ganglion of the lateral geniculate body seems to function as an integrating center in addition to its role as a relay and distribution center. Therefore the lateral geniculate is important to the relay of visual information to portions of the brain other than the occipital cortex.

It has also been demonstrated that 20% of the peripheral retinal nerve fibers relay information through axons to the thalamus or midbrain for spatial match with kinesthetic, proprioceptive, and vestibular information being received from other sensory-motor systems. The developmental purpose is to organize spatial information to orient higher sensorimotor experiences affecting posture, movement, and balance. Once this has been established, information from the thalamus is sent to higher cortical functions (16). The occipital cortex is not an exception to this process. It receives spatial information in order to organize the detail information processed.

Ganglion cells from the retina emanate through the optic chiasm to the optic tract where they reach three major destinations: 1) the lateral geniculate body for relay to the visual cortex, 2) the pretectal nucleus (pupillary constriction), and 3) the superior colliculus which becomes related to posture, movement, and orientation to positional space (15).

In order to shift the position of the eyes from one point to another, we must spatially orient to the next destination before shifting the eyes (aiming the fovea) to look at detail (23). The ambient process is critical for anticipating change. Without this process, visual information becomes isolated details and there is difficulty shifting visual regard and attention. Also, there is a lack of awareness of body position in a spatial context caused by a shift in visual midline. Due to a mismatch between the ambient visual process, the kinesthetic, proprioceptive, the vestibular input system and the ambient visual process, the concept of visual midline will shift. As will be discussed later in this chapter, if the visual midline shifts, it will reinforce and/or cause postural imbalances.

The ambient process is not a conscious process as is the focal system. We orient this portion of the visual system to monitor and manipulate the spatial environment. The ambient visual process works to integrate sensory-motor information. Without the ambient visual process, an individual would experience fragmented vision as well as difficulty with organizing posture and movement. The bimodal process, however, in many ways has been ignored and/or misunderstood clinically (24).

Ganglion cells traveling from the retinas can be categorized based both on physiology and function into three types; P-cells (parvocellular), M-cells (magnocellular), and K-cells (koniocellular). These cells provide information similar to that which was discussed previously regarding the focal and ambient process. The M-cells and P-cells provide a physiological substrate for the ambient and focal processes. M-cells transmit visual information about shape and movement rapidly, but without detail. P-cells transmit the detail information contained in shapes and are much slower. These cells emanate from the retina through two major brain pathways; the retino-geniculo-cortical pathway and the retino-tectal pathway. The retino-geniculo-cortical pathway contains both P-cells and M-cells and is the most recent in evolution providing a mechanism for focal processing in the cortex. The retino-tectal pathway is mainly derived from M-cells and is more primitive and provides a basis for spatial information particularly related to spatial orientation prior to focalization. For example, a saccadic eye movement (via the

ambient pathway) first requires spatial orientation to establish the direction and trajectory of the eye movement prior to the focalization response. The retino-tectal pathway is most critical in early development of the child and some functions are taken over later in development by the retino-geniculo-cortical pathway (25). However, the midbrain ambient system remains important for spatial orientation and balance. It can be demonstrated clinically that children and adults with oculomotor dysfunction and aspects of dyslexia affecting reading ability demonstrate improvement with pursuit tracking and saccadic fixations as well as with the ability to track a line of print with sensory motor support such as by physically pointing at words.

The P-cell subsystem has been related to the focal visual process whereas the retino-tectal projection of the M-cell subsystem is a substrate for the ambient visual process. Neurons in the retino-tectal pathway are almost completely myelinated at birth whereas those of the retino-geniculo-cortical pathway are not. It is also of interest to know that the superior colliculus has essentially a normal adult organization and neuronal activity at birth whereas the occipital cortex does not (26). These authors have emphasized that while cerebral cortical processes have taken over much of the visual motor functioning, massive innervations still remain between cortical and midbrain structures.

Through development, experience is established to couple and bring balance between the ambient and focal visual process as well as the M-cell and P-cell subsystems. While balance between the spatial (ambient) and detail (focal) visual processing systems provide a means by which organization of time and space become a normal process for humans, injury as a result of a TBI can affect the balance between the systems. A decoupling of the focal and ambient visual process including the magno-parvo cellular systems is suggested as a basis for interference with function and performance. M-cells have larger diameter axons and have demonstrated to be more susceptible to damage in conditions such as ischemia and glaucoma.

More specifically, the decoupling of cerebral blood flow (CBF) and cerebral glucose metabolism (CMGL) has been described. This decoupling is described in terms of ischemia-hyperemia resulting in metabolic imbalances which derange neuronal energy production and cell membrane permeability ultimately causing potential cytoxicity (27) even in a minor whiplash accident. This can be the basis of visual problems following TBI.

A patient with mild TBI is most frequently underdiagnosed or misdiagnosed relative to visual impairments (28, 29). The walking wounded with a negative CT scan or MRI is a potentially classic case of an injured person whose symptoms are frequently dismissed as exaggerated and/or psychosomatic in origin. The many individuals who suffer significant visual symptoms following a brain injury potentially suffer equally from discrimination without identification of the causative factors (29).

The basis of the dysfunction of the visual process is related to the traumatic force imposed upon the central nervous system whether through blunt trauma or inertial acceleration or angular acceleration trauma of whiplash (hyperextension/hyperflexion). This can affect potassium adflux and secondary calcium influx. It is the influx of calcium ions that activate cellular proteate enzymes triggering cytoskeletal disruption and simultaneous neurotransmitter hypercholinergic activity.

It is hypothesized to be the cytotoxic response that initiates cytoskeletal collapse and ultimately deafferentation that is etiologic to the symptoms of post-trauma vision syndrome. In turn, the lifelong established engrams that provided the automaticity of controls affecting the very complex sensory motor components that subserve the visual process become eroded resulting in degradation of our dominant sensory perception, our dominant guidance system: visual information processing (30).

## POST-TRAUMA VISION SYNDROME (PTVS)

Following a TBI, visual symptoms may occur (31) such as diplopia, seeing words and print appearing to move, difficulty shifting gaze, difficulty adapting to environments where there is movement in the periphery (such as in a store), and photophobia (Table 29-1). Characteristically, an examination of the visual system may demonstrate binocular dysfunction such as strabismus, convergence insufficiency, divergence excess, oculomotor dysfunction, and accommodative insufficiency. Some researchers have reported that diplopia, reduced acuities, and poor accommodation are prevalent among those with traumatic brain injuries (28). Others have reported intermittent diplopia present in certain positions of gaze (31). Soden and Cohen (32) as well as Nelson and Benabib (21) have correlated postural adaptation of the body with compensation for vision dysfunction and diplopia produced in certain positions of gaze. In addition, these researchers have found secondary contracture and/or muscle spasms due to poor body posture. A high prevalence of exotropia and exophoria have been reported for persons who have suffered even a mild traumatic brain injury (4, 5, 33).

Studies of binocularity have recognized dysfunction of oculomotor, convergence, and accommodations. A high prevalence of exotropia (5, 6, 34) is evidenced in the literature. While symptoms and characteristics are logically related to the visual dysfunction attributed to nerve palsy (35), the mechanism for the cause of the binocular dysfunctions appear to be more complexly related to the affect of the trauma on the bimodal visual processing systems as related by more recent studies

**TABLE 29-1**
***Post-Trauma Vision Syndrome***

Common characteristics and symptoms associated with post-trauma vision syndrome characteristics:

- Exotropia or high exotropia
- Accommodative dysfunction
- Convergence insufficiency
- Low blink rate
- Spatial disorientation
- Poor fixations and pursuits
- Unstable ambient vision

Symptoms:

- Possible diplopia
- Objects appear to move
- Poor concentration and attention
- Staring behavior
- Poor visual memory
- Photophobia (glare sensitivity)
- Asthenopic symptoms
- Associated neuro-motor difficulties
  - Balance and coordination
  - Posture

on visual evoked potentials (11, 13). In this study, the researchers demonstrated that changes in amplitude under binocular testing were found to correlate statistically with an experimental population (persons with TBI) compared to a control group of which the experimental group of subjects also experienced symptoms and demonstrated characteristics as discussed in the previous paragraph. An increase in amplitudes was produced following treatment with base-in prism and binasal occlusion for the experimental group of subjects. A decrease in amplitudes was found for those in the control group. Changes were also reported in amplitudes and latencies following a TBI.

The researchers have explained that following a TBI, there appears to be interference in the ambient (spatial) organization of the visual field which as a result, affects the focalization process (36). As mentioned previously in this chapter, the spatial match of information between the ambient and sensory motor process can provide a feedforward (37) to the higher cortical processes including the occipital cortex of which the binocular integration cells require the spatial information to bind and establish peripheral vision fusion. A decrease in amplitude of the binocular visual evoked potential as demonstrated by the study emphasizes the role of the ambient visual process in providing this spatial binding for the fusion process. In turn, the decrease in the amplitude provides evidence of the lack of ambient vision feedforward support for higher binocular fusion prior to the focal visual process establishing awareness of detail.

The result of the dysfunction of ambient visual processing becomes characteristic of the binocular dysfunctions such as convergence insufficiency, accommodative insufficiency, exophoria, and may even be the basis for post-traumatic exotropia (38).

The characteristics of dysfunction of the binocular vision process as well as commonly measured comitant and noncomitant strabismus may be more a function of disruption related to ambient and focal visual processing than of specific neuronal interferences. Other studies have demonstrated that lesions in the superior colliculus can produce disturbances of convergence ability (39).

The dysfunction and lack of support through ambient visual processing leaves the person compromised in the attempt to use the focal visual process which isolates detail and does not serve the need for anticipation of change (23). For example, following a TBI, persons will often have to work harder at near vision tasks. The research provides evidence that the ambient process being compromised also interferes with the anticipatory spatial role to provide for release of fixation and detail as well as for change. In turn, the visual process becomes focally bound, isolating to detail rather than utilizing the ambient visual process for release from detail and the anticipation of spatial organization that affects continuity and sequence of visual performance.

This research also demonstrates that the structure of binasal occluders (vertical boundaries in the nasal portion of the visual field) in addition to base-in prism (prism affecting spatial organization), rebalances the ambient visual process. In turn, a reduction in symptoms was noted in these subjects corresponding to implementation of this mode of treatment. Specifically, subjects reported a significant reduction in symptoms (11) as previously mentioned.

The effects of dysfunction between ambient and focal processing as related to common characteristics of binocular dysfunction as frequently seen following a TBI are more common than previously recognized. Binocular dysfunction such as convergence insufficiency is a frequently misdiagnosed condition following a traumatic brain injury. Convergence insufficiency should always be considered in the diagnosis of patients with head trauma.

The dysfunction relative to focal and ambient visual processing has been termed post-trauma vision syndrome (PTVS). Persons who suffer from post-trauma vision syndrome will often have clear sight and their eyes will be healthy. Upon being referred for a routine eye examination, if the examiner is not familiar with PTVS, the examiner will often report that there is nothing wrong with the person's eyes and the symptoms appear to be more psychological in nature. Many persons will spend years suffering from this condition unless it is diagnosed and treated appropriately. Needless to say, this can be the cause of additional inappropriate referral and treatment

at a high cost to families and third party reimbursement systems. Appropriate treatment will not only reduce symptoms, but directly affect attention, concentration, and cognitive processing while preventing secondary emotional complications. The methods of evaluation and treatment through neuro-optometric rehabilitation and neuro-ophthalmological evaluation will be discussed later in this chapter.

## VISUAL MIDLINE SHIFT SYNDROME (VMSS)

Postural orientation is vital for daily function for both adults and children. Postural adaptation that begins in the early stages of development provides a means for the infant to organize space for balance, spatial orientation, and ambulation. For functioning adults and children, anti-gravity alignments are initiated with limbs and/or with head movements. The vestibular system contributes a balance response necessary when the center of gravity has shifted while the cerebellar and midbrain structures through ambient/sensory motor support contribute to the desired qualities of smoothness and coordination for motor planning.

The ambient visual process is primary in the establishment of postural orientation related to boundaries by orienting boundaries in the vertical and horizontal planes with spatial information from sensory motor systems. Early in development, this establishes concepts of visual midline that provide experience for postural orientation (20). Established in relationship with multi-sensory motor cooperation, the ambient visual process together with sensory motor match, feedforwards information to diverse areas of the cerebral cortex including the occipital cortex. This vital component enables anticipation for postural adaptation and movement (37).

Following a TBI, a person may be left with hemiplegia or hemiparesis. The loss of neurological and motor function has traditionally been thought to be the sole cause for the inability to weight-bear on the affected side. Also, in cases of visual field loss such as with homonymous hemianopsia, it has been observed that individuals will begin to lean away from the side of visual field loss even if they do not have a hemiparesis.

Prior to the consideration of vision as a bimodal process, discussion has been centered on a lack of visual cognition and attention leading to neglect (40, 41). These authors have identified the phenomenon where people have limited or no attention to a specific side of their body as well as correlating field of vision. Right hemisphere damage often produces the characteristic left field neglect (42, 43). The spatial component of vision has been primarily considered related to the relationship of magnocellular function with right hemisphere involvement. The complete concept of the ambient process related to midbrain and sensory-motor interaction should also be considered.

Relative to the concept of the bimodal process of vision, inattention is a focal processing phenomenon. The authors of this chapter offer the possibility that the focal visual processing dysfunction of inattention is secondary to the spatial ambient visual processing disorder that is more directly associated with the affect on sensory-motor processing from the TBI.

Studies (10) have demonstrated that the visual midline will shift relative to a hemiparesis and/or homonymous hemianopsia. This may be observed by passing an object in front of a person's face and asking them to report when the object appears to be directly in front of their nose. In the case of a visual field loss such as a homonymous hemianopsia, the person will often report the target in front of their nose when it is directed to the side away from the visual field loss. An interesting observation occurs when working with patients with hemiparesis or hemiplegia. The person will also project the concept of midline in the majority of cases by leaning away from the neurologically affected side. The shift affects weight-bearing on the affected side in most cases and in turn, interferes with aspects of physical rehabilitation. The shifting of perceived visual midline away from the affected side is a compensatory mechanism and essentially reinforces the neglect of visual space caused by the hemiparesis and/or hemianopsia.

The shifting of the visual midline is caused by a distortion (compression and expansion) of the ambient visual process. The midline can shift laterally and/or in the anterior/posterior axis. The perceived visual midline will usually shift away from the affected side. This has been termed visual midline shift syndrome.

## NEURO-OPHTHALMOLOGY

Progress and overall rehabilitation for a person who has suffered a TBI are often interfered with due to visual problems that are sometimes ignored or misdiagnosed (2).

The provision of skilled neuro-ophthalmological services can positively affect the rehabilitation outcome. Referrals for neuro-ophthalmological evaluations are often initiated by physicians or rehabilitation professionals and optometrists practicing neuro-optometric rehabilitation who recognize visual interference with daily living skills and/or perceptual motor function.

The literature lacks good demographic studies on the prevalence of neuro-ophthalmological problems in TBI. Sabates reports that of 181 consecutive patients referred with visual complaints following head trauma, the most common etiology was motor vehicle accident in 57% of the cases. For direct trauma to the head, 15% were represented and 13% were related to injuries

sustained from a fall. He also reports that in over 88% of the eyes tested, acuity of 20/20 was reported. Of this population, 33% suffered cranial nerve palsy.

## NEURO-OPTOMETRIC REHABILITATION

Neuro-optometric rehabilitation represents a new and evolving specialty within the profession of optometry devoted to clinical assessment and rehabilitative treatment of visual binocular/processing disorders following a traumatic brain injury. While there is overlap in the assessment between ophthalmological/neuro-ophthalmological and neuro-optometric rehabilitation, the latter emphasizes rehabilitation of visual processing disorders. The fields of ophthalmology and optometry work in conjunction with individuals who have suffered visual and ocular-visual problems following a TBI.

Neuro-optometric rehabilitation is defined as:

> Neuro-optometric rehabilitation is an individualized treatment regimen for patients with visual deficits as a direct result of physical disabilities, traumatic and/or acquired brain injuries. Neuro-optometric therapy is a process for the rehabilitation of visual/perceptual/motor disorders. It includes but is not limited to acquired strabismus, diplopia, binocular dysfunction, convergence, and/or accommodation, paresis/paralysis, oculomotor dysfunction, visual spatial dysfunction, visual perceptual and cognitive deficits, and traumatic visual acuity loss.
>
> Patients of all ages who experience neurological insults can benefit from neuro-optometric rehabilitation. Visual problems caused by traumatic brain injury, cerebrovascular accident, cerebral palsy, multiple sclerosis, etc. may interfere with performance causing the person to be identified as learning disabled or as having attention deficit disorder.
>
> The visual dysfunctions can manifest themselves as psychological sequela such as anxiety and panic disorders as well as spatial dysfunctions affecting balance and posture. A neuro-optometric treatment plan improves specific acquired vision dysfunction determined by standard diagnostic criteria. Treatment regimens encompass medially necessary noncompensatory lenses and prisms with and without occlusion and other appropriate rehabilitation strategies (23).

The role of the ophthalmologist is to provide excellence of care relative to treatment of disease and trauma affecting the eye orbit and neurology affecting visual function. The ophthalmologist can and should be involved in treatment for acute care as well as long term ophthalmological needs for the person who has suffered a TBI.

The optometrist practicing neuro-optometric rehabilitation can affect visual processing disorders such as PTVS, VMSS, and other binocular spatial disorders causing diplopia, vertigo, etc. as rehabilitation is established. The neuro-optometric rehabilitation assessment includes a careful review of medical and rehabilitative records as well as establishing a history based on visual and spatial dysfunction.

### Evaluation and History

The neuro-ophthalmological and neuro-optometric rehabilitation assessment requires a complete history and differentiation of symptoms and characteristics from premorbid conditions. The symptoms must be assessed relative to the medical history and a complete review of medical records and medications is imperative. It is important to reconstruct the event relative to the history in order to assess injury and lesions relative to inertial force and impact. Understanding the pre-event medical history is necessary relative to determining the nature of injuries.

*Oculomotor and Binocular Function Assessment* An oculomotor evaluation is performed to determine cranial nerve function. One of the most debilitating visual conditions following a TBI is diplopia. Strabismus can often occur following lesions affecting cranial nerve 3 (oculomotor nerve), 4 (trochlear nerve), or 6 (abducens nerve). These lesions can occur from blunt head trauma affecting origin, insertion, and or innervation of the extraocular muscles, trauma to brain stem, midbrain or cortex, vestibular trauma, and/or trauma affecting orbital structure and orbital space occupying lesions (21, 39). Often, as is the case, CT scans and MRI are unable to detect lesions related to these specific oculomotor neurons. The authors have previously suggested a dysfunction of the ambient visual process and the lack of feedforward support for peripheral fusion lock.

The inability to provide spatial information will cause a highly focalized nature of vision. In turn, the visual world becomes fragmented. This causes increased concentration on detail and yields increased fragmentation of the visual world. The result can be diplopia that will be characterized and measured by strabismus (exotropia, esotropia, hypertropia, hypotropia). When an individual has diplopia, the spatial reference by which to use vision to lead posture, balance, and motor function is compromised. In turn, it can affect cognitive-perceptual processing due to the distracting influence of physiological stress. In one study (2), the second most common symptom was that 30% of the subjects experienced diplopia following a TBI.

The most common result of lesions of the cranial nerve following a TBI is third nerve palsy. A third nerve palsy is associated with exotropia as well as ptosis and mydriasis of the pupil on the affected side with complete compromise. In most cases, the affect is incomplete causing

variations from decompensated exophoria to intermittent exotropia. Diplopia depends on visual fatigue and chronic development of suppression. Vertical palsies resulting from the affect of cranial nerve 4 will produce hyperphoria and hypertropia as well as decompensation in specific gaze direction and head tilt. Sixth cranial nerve palsy interferes with abduction of the related eye, causing esotropia.

A variety of cranial nerve and binocular function tests are designed to provoke oculomotor failure causing decompensation of binocularity. These tests are described as follows:

1. Cover Test: The examiner will perform this test with the patient first fixating on a distant target (ten feet or beyond) and then at a near target (40 cm). The Alternate Cover Test is done by passing a cover alternately from one eye to the other and observing the movement of the eye emerging from under the cover. A heterophoria can be elicited. If observed, the examiner will perform a Cover Test by covering and uncovering each eye to discern from a tropia.
2. Krimsky Test: This is similar to the Cover Test, however, compensating prism is introduced to neutralize the motion of the phoria or tropia to determined the extent of deviation.
3. Bielchowsky's Head Tilting test: Cranial nerve and oculomotor function is analyzed by having the patient hold fixation in primary gaze while the examiner rotates the patients head first to the left and then to the right testing 3rd and 6th nerve function. The patient's head is moved in extension and flexion and tilted first to the right shoulder and then to the left shoulder to evaluate 4th nerve palsy affecting over and under-compensating oblique muscles.
4. Lancaster Red/Green Test: The patient is given glasses in which the OD has a red lens and the OS has a green lens. The examiner holds a red flashlight and the patient holds a green flashlight. The examiner shines the red flashlight on a screen and moves it into the nine cardinal gazes of fixation while asking the patient to superimpose their projected green light on the red light. Any deviation in a particular gaze direction will be noted and related to an underactive, overactive, or restricted ocular muscle.
5. Red Lens Test: The examiner holds a red lens before one of the patient's eyes and the patient is asked to fixate on a penlight. If the patient reports seeing two lights, the test is positive for diplopia at the respective working distance.
6. Worth Four Dot Test: The patient is given red/green glasses to wear while looking at the projection of four dots that form a diamond. The top dot is red, the inferior dot is white, and the lateral dots are both green. If the patient is binocular, four dots will be seen. If binocularity is compromised and the patient reports seeing five dots, diplopia has been elicited. If two or three dots are observed, a corresponding suppression is present.
7. Maddox Rod Test: The examiner holds a cylinder before one of the patient's eyes while the patient observes a penlight. The eye behind the cylinder will see a vertical or horizontal line of light depending on how the cylinder is positioned. If the line is reported to either the left or right side of the light, a heterophoria or tropia is present at the respective working distance. If the line appears above or below the light, a vertical deviation has been elicited.

Binocular dysfunctions often present as convergence insufficiency or strabismus of which exotropia is the most common following a TBI. This can cause diplopia. The diplopia can be masked by intermittent suppression or accompanying field loss (i.e. homonymous hemianopsia). Any interference with binocularity will affect stereopsis and depth perception. In addition, oculomotor disturbances will affect pursuit tracking, saccades, and fixations.

For those experiencing blurred vision at near, accommodative insufficiency or accommodative spasms have been found as an interfering mechanism causing the related symptoms. Studies have also related binocularity with the neurological correlates such as cranial nerve palsies (35).

The term divergence paralysis was first described by Parinaud in 1883. Relative to TBI, the literature emphasizes the third nerve palsy as being the cause of the majority of those with exotropia following a TBI (34). Other types of neurological disorders affecting binocularity are sixth nerve palsies causing esotropia and fourth nerve palsies affecting vertical imbalances.

Sensory motor and binocular analysis should be performed to evaluate oculomotor function. Assessment should use quantity of function to analyze any limitations of oculomotor movements as well as quality of function (assessing fixation losses and difficulties encountered with pursuit tracking or saccadic fixations into particular fields). This may relate to oculomotor palsies. The assessment of convergence and accommodation quantitatively as well as qualitatively will enable the clinician to begin to understand levels of visual stress in performing visual skill activity (23).

The refractive sequence will objectively and subjectively analyze refractive error as well as variations in refractions which may relate to an unstable nature of accommodation and/or subluxation of the accommodative lens following trauma to the eye or head. Careful analysis of eye muscle balance through phoria, vergence, and duction testing will provide the clinician with an understanding of states of eye muscle balance and muscle reserves related to dysfunction of ambient and focal

processing. More specifically, a dysfunction of the ambient visual process will cause a lack of spatial support, thus affecting phoria, duction, and/or vergence measurements causing PTVS. In the most severe condition, exotropia can be produced, which is also known as third nerve palsy.

Surgical intervention has been used to affect oculomotor paresis. However, the success from surgery can be compromised if there is interference with the ambient spatial feedforward to binocular cortical cells (23).

The use of botulinum toxin has been found effective in reducing over action and muscle spasticity in affecting general muscle tone in the body as well as in cases of oculomotor spasm (45).

Pupillary assessment can offer an understanding of interruption of afferent and efferent sensory pathways (46). Defects in pupillary function can cause variations in response such as with the Marcus-Gunn pupil (47). Dilation of the consensual response indicates asymmetry in the conduction of afferent sensory fibers anterior to the chiasm and it differentiates a unilateral occurrence.

Wernicke's hemianopic pupil is another method by which to differentiate homonymous hemianopsia of tract lesions from the homonymous hemianopic lesions occurring in optic radiation dysfunction. The pupil sign demonstrates an inappropriate or impaired reaction in both pupils when light is specifically delivered to the amourotic half of either retina (47). Parinaud's syndrome can occur following a TBI and is characterized by displaced pupils, ptosis, nystagmus, oculomotor palsies (limitation of supraduction), papilledema, as well as producing symptoms of vertigo and ataxia (48). Papilledema and optic nerve atrophy are also characteristics that should be carefully evaluated through direct and indirect ophthalmoscopy (48).

## PSYCHOPHYSIOLOGICAL TESTING

Evaluation of visual function is important to identify lesions for the purpose of diagnosis and prognosis of treatment in visual problems following a TBI. Two kinds of examinations, psychophysiological and electrophysiological, allow assessment of the disorders in the entire length of the visual pathway. The following is a description of psychophysiological testing.

### Visual Acuity

Acuities should be functionally based and objectively based. The assessment of visual acuity should be performed both monocularly and binocularly. The standard Snellen Acuity Test can be performed at six meters and forty centimeters. However, other types of tests may be more appropriate if there is vision impairment present. Tests such as the Feinbloom charts and the Sloan Acuity Charts may offer more precise measurements.

Behavioral assessment and functional variations should be noted such as a monocular acuity that is better than a binocular acuity. Often, interferences with contrast sensitivity following post-trauma vision syndrome will affect acuity in varying environmental conditions (49).

Visual acuity and visual field assessments are routinely psychophysiological tests. However, motion perception is also one of the elementary visual phenomena, especially in the affect from neurological trauma.

*Visual Field* Also important is a careful assessment of the visual field (50, 51). Visual field is an island or hill of vision in a sea of darkness (Figure 29-1). When visual defects occur, the corresponding part of the island is lost. The field of vision at any particular sensitivity level is changed.

Abnormalities of any point along the visual pathway affect the visual field, often in a pattern characteristic of a particular disorder process or location. The visual field defects in the visual pathway correlate with the lesion sites and field defects. Homonymous refers to a defect present in both eyes with the same laterality, while heteronymous refers to visual loss that usually respects the vertical meridian. Congruous fields are symmetric in both eyes. The lesions of upper or lower occipital banks produce quadrantanopia defects, while lesions within the temporal and parietal lobes cause field defects that tend not to respect the horizontal meridian.

More sensitive and precise evaluation of visual field can be obtained by automated and computerized threshold perimeter or kinetic testing of Goldmann perimeter. In many instances, the computerized threshold perimetry is a more reproducible test for patients with defects on optic neuropathies, chiasmal disturbances and so on. Computerized threshold perimeter performs static threshold

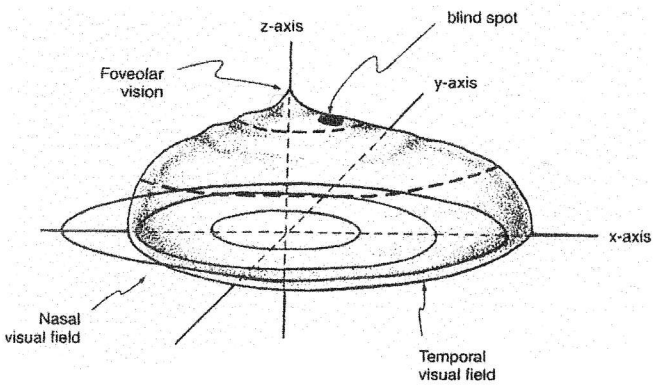


**FIGURE 29-1**

Visual field is an island or hill of vision in a sea of darkness (57)

measurement. New statistical software empowers the user with additional means of refining the assessment and representation of visual field.

The Goldmann perimeter allows interaction with the examiner and may be more appropriate for patients with neurological impairments. It is a substantially more sophisticated method of field assessment. Kinetic perimetry is usually the preferred method. It involves moving the target from a non-seeing area into a seeing area. A contour map of the island of vision can thus be established by using targets of varying size and brightness. Computerized perimetry is currently the more popular test method in visual field testing.

The visual field must be tested for each eye separately. The visual field is a dynamic area that changes according to a large number of variables. It should always keep in mind that all modalities for visual field evaluation are subjective and depend on the patient's level of alertness, cooperation, ability to fixate centrally, and response rapidity.

Visual field assessment should include standard objective instrument measurements of threshold visual fields when possible, but persons with traumatic brain injury may not be able to sustain attention and organization for a purposeful evaluation in automated field tests. Confrontation visual field assessment which will include holding the person's gaze while presenting objects in the peripheral field are effective as well as a saccadic confrontation visual field assessment. The latter can be used effectively for individuals who cannot hold fixation and are peripherally distractible.

The methods for this form of testing are to provide stimuli or targets in various portions of the field. The examiner observes the functional behavior of the patient as they shift their eyes via saccadic movements into the various visual fields. This may be performed monocularly as well as binocularly.

Homonymous deficits often will relate to spatial problems affecting posture and balance. Visual field neglect is a condition in which there is no hemianopsia, but spatial information in the affected field (usually the left visual field) is ignored (24). Dysfunction caused by field loss will be discussed later in this chapter.

*Motion Perception* Motion perception is as elementary a visual perception as form or color senses (52). It includes three-dimension stereo-sense, location, and layer sense. The projected pathway of motion perception is from retinal sensory cells through ganglion cells, lateral geniculate body of thalamus to striate cortex. The abnormalities of motion perception have been seen in optic neuropathy and glaucoma and it was also reported that motion perception decreased or diminished in cerebral traumatic patients with normal visual acuity or color perception.

Three methods for evaluating motion perception are used: 1) using the motion stimuli of the random dots; 2) using the stimuli of sinusoidal grating strips; and 3) using bar target stimuli. The bar targets projected on a monitor can be observed clearly and distinguished easily by subjects. The size and density of bar targets as well as variations in direction of the bars and flickers can be controlled (vertical, horizontal, and oblique).

## ELECTROPHYSIOLOGICAL TESTING

Most electrophysiological tests are evoked responses for which adjusting stimulus conditions and techniques of recording makes possible a representation of the sequence of events along the visual pathway (53–55). Besides international standards of ERG, PERG, EOG, and VEP, it is necessary that each laboratory should have its own normal data to evaluate patient's response recording because the amplitude and implicit time of wave may vary among laboratories.

### Visual Evoked Response (VEP)

Visual evoked response is a measure of the electrical activity through the visual cortex. Certain conditions are required for VEP measurement: visual stimulus, scalp electrodes, amplifier, averaging system, and recording equipment. The electrodes are positioned in the midline at the occipital and posterior frontal region. An electrode placed on the ear provides a ground.

VEP is response to stimulation by flashes or pattern stimuli with alternating checkerboard or stripes on a monitor. The flash VEP is applied in the persons with very poor vision. A full field Ganzfeld stimulator is used for flash stimuli. The pattern stimulus is preferable when the eye is optically correctable, since the occipital cortex is more sensitive to sharp edges and contrast, while it is relatively insensitive to diffuse light.

The VEP is characterized by the waves N, P, N, and P. The amplitude and implicit time of the wave depends on the check size, contrast, and frequencies of the stimulus. The temporal aspect (latency) of the VEP is more reliable with the significance in clinical application of acuity and focalization, however, the amplitude of VEP under binocular assessment has more relevance to ambient spatial relationships.

The VEP traditionally has been used to confirm the diagnosis of neuropathy in the visual system. It assesses disorder of optic nerve fibers, estimates visual acuity in nonverbal and verbal persons, evaluates potentials for reasonable visual acuity in some kinds of patients, and detects and locates visual field. In some cases, the VEP is even more sensitive for detecting compressive lesions than subjective perimetry.

## Multifocal Visual Electrophysiology

### *Multifocal Electroretinogram (mfERG)*

*Multifocal Evoked Response (mfVEP)* In 1992, Sutter and his co-workers reported presenting pseudorandomly multifocal stimulation together with retinal or cortical scaling of the size of the stimulated areas. They were able to stimulate numerous locations of the visual field simultaneously and to extract individual responses from each of them. The electrode permitted the recording of reliable responses from numerous retinal locations such as areas 61, 103, 241 loci of the visual field. This work establishes the multifocal electroretinogram (mfERG) and multifocal visual evoked response (mfVEP) with field topographic mapping of ERG or VEP components.

The mfERG or mfVEP is based on the Wiener kernel expansion and uses deterministic pseudorandom binary m-sequence stimulation. It permits computation of the signal by cross-correlation of the response evoked by m-sequence stimulation with m-sequence itself. Therefore, it is very effective for mapping.

The technique allows computation of the first order or higher order kernel which characterizes the nonlinear interaction between visual events. The amplitude of the averaged quadratic VEPs is in proportion to visual field losses. The mfERG or mfVEP is a powerful method for the investigation of brain activity and mechanisms of human visual perception.

Electrophysiological analysis through use of binocular visual evoked potentials assesses the relationship of support for focal processing by the ambient (spatial) system (10).

The visual evoked potential should be performed with habitual corrective lenses. A P100 cross-pattern reversal analysis is performed. Following the initial response, two diopters of base-in prism in conjunction with binasal occlusion should be introduced before both eyes in addition to the corrective lenses. The amplitude and latency of the P100 should be compared. An amplitude increase following the introduction of base-in prism with binasal occlusion is seen as a test positive for PTVS (11).

*Electro-Oculogram (EOG)* The EOG technique of recording eye movement is widely available and reliable. Since the eye acts as a dipole, the electrical current changes are caused between the cornea and posterior pole of the eyeball when the eye is moving in different directions. The EOG is performed by placing four cutaneous electrodes are placed on the canthal regions of both eyes. An electrode placed on the ear serves as the ground. The potential is recorded when alternate fixations or target movement is performed. Changes in illumination, skin resistance, and speed of the target will affect the results of the EOG. The EOG can yield reasonable recordings of horizontal movements, however, vertical movements may be affected by eyelid blinks and nonlinearities.

Observation of eye movements by the clinicians provides a simple qualitative assessment and in most cases is able to identify most abnormalities. Therefore, the EOG is used to distinguish the affects of specific ocular modalities and to determine the presence or absence of improved motor function during therapy for visual pathways.

## NEURO-MOTOR ASSESSMENT

The neuro-motor assessment can be performed to evaluate the role of the ambient visual process in establishing concepts of visual midline and how it affects posture, balance, and movement. Concerning posture, dysfunction of the ambient visual process will also affect seating position and posture.

The visual midline test may be performed in those individuals that can respond actively (10). Observation of the patient in a seated position as well as during ambulation should be performed to evaluate the correlation of the visual midline shift test to tendencies to drift to one side and/or lean forward or backward during ambulation. Lateral shift of the visual midline will often cause a lean or drift during ambulation. An anterior shift can cause a lean or flexion forward. In more severe cases of the anterior shift of visual midline, toe-walking will develop in an attempt to compensate for the imbalance. A posterior shift of visual midline will cause an extension posture and tendency to lean backward in retropulsion.

## VISUAL-VESTIBULAR INTEGRATION DYSFUNCTION

There are numerous treatises that discuss the anatomy, physiology, and treatment of the separate visual, vestibular, and sensory motor systems, however, until recently, few were able to discuss the functional integration and relationships that exist between these three systems. Dizziness, vertigo, and imbalance are common symptoms following a TBI and often indicate miscommunication between these systems. Inner-ear disorder of the vestibular system is a common diagnosis, however, the relationship to the visual process is often overlooked or misinterpreted.

Coordinated movement involves a complex interaction between sensory afferents from the visual system, somatosensory system, and the vestibular system. In particular, the semicircular canals and utricle/saccule respond to rotational and linear/gravitational information and is transmitted via the vestibule-cochlear nerve through the ascending and descending axon branches of the brainstem vestibular nuclei. At times, vision supercedes or enhances vestibular input. The vestibulo-ocular reflex

(which maintains foveal fixation during head movements), the vestibulospinal pathways (which are responsible for postural control under static and dynamic circumstances), and the vestibulocerebellar pathways (allowing for modulation of motor output via the flocculonodular lobe of the cerebellum) are all integrated at this level of the brainstem.

The vestibular nuclei accept information from the visual system relative to the ambient visual process to support movement and visual interaction of the environment. The subset of the ambient process, the magnocellular system, provides information about perception of contrast and movement involving short wavelength and low and middle spatial frequency input. This affects awareness of depth perception, motion detection, and alterations in brightness. The parvocellular system is mostly responsible for detail discrimination, color perception, and identification of patterns or shapes.

An important differential between magnocellular and parvocellular processing is that the former is responsive to the onset and offset of receptive stimuli, whereas the latter is responsive to longer stimulus duration. Parvocellular input projects to the occipital lobes along the retinal-geniculate-cortical pathway and then may send axons to the temporal brain region. It is the ambient process inclusive of the magnocellular system that supports posture, balance, and spatial orientation. Information synapsing in the occipital lobe will proceed to temporal and parietal lobes for higher level processing while the information from the superior colliculus will integrate with head and body postural reflexes flowing through the vestibulospinal and vestibulocerebellar tracts.

The vestibulo-visual relationship supports a stabilization of vision during movement of the head, neck, or body. Diplopia is the most frequently considered symptom of disruption in this interaction in addition to other interferences with binocularity.

The significance of the vestibulo-oculo-cervical triad is the quality and organization of somatosensory input (20) which occurs in the thalamus and is transmitted to the cerebellum and primary somatosensory cortex within the parietal lobe. Multisensory integration and conscious perception occur as this information is projected into the secondary and association cortices of the frontal, temporal, and parietal lobes where the perception of dizziness, vertigo, or imbalance may be the result.

A complete medical history with careful analysis of the visual process and vestibular system is necessary for diagnosis and prognosis. Too often, functional vestibular rehabilitation for vertigo and dizziness is begun before the relationship to visual processing problems is determined. Under the age of fifty, TBI is the most common cause of dizziness and vertigo from a direct head trauma and whiplash.

Differential diagnosis for dizziness without vestibular dysfunction includes PTVS, hyperventilation, dehydration, orthostatic hypotension, vasovagal syndrome, arteriosclerosis, osteoarthritis, and central nervous system disorders. A Group II Disorder such as aortic, carotid, vertebrobasilar, subclavian, and cerebral artery insufficiency should be ruled out.

Once a Group II disorder has been ruled out, vestibular anomalies such as vestibular neuritis (acute labyrinthitis), benign paroxysmal positional vertigo (BPPV), nucleo-reticular vestibular syndrome, Meniere's Disease, Perilymphatic Hypertension, and Perilymphatic Fistula should be ruled out.

Since destabilization of the ambient process following trauma has been clinically found to cause vertigo and dizziness which are symptoms of PTVS. By treating PTVS, stability is created in ambient visual processing, thereby increasing the efficiency of matching information with vestibular, kinesthetic, and proprioceptive processes. This, in turn, frequently reduces or eliminates symptoms of vertigo and dizziness.

## VISUAL FIELD DYSFUNCTION

A visual field loss is relative to the area of cortical lesion or optic nerve insult. Field defects that occur beyond the optic chiasm will always cause a homonymous hemianopsia not necessarily congruous (56). Depending upon the area of the cortex affected, geniculate lesions are often not suspected and are rarely diagnosed. However, in the case of a homonymous hemianopsia involving primarily the central visual field sparing the peripheral field, the geniculate body should be considered as the source.

Temporal lobe lesions will often produce an incongruous superior quadrantopsia with a sloping margin. In addition to field loss, parietal lobe lesions cause a lack of awareness of the field loss. It can be differentiated by confrontation visual field assessment when the clinician presents simultaneous targets in opposite fields. The parietal lobe lesion will cause an extinction phenomenon yielding a person to be aware of only one target at a time. If the target is moving in both fields, often the extinction phenomenon will not occur. The target must be stationary in order to demonstrate the possibility of a parietal lobe lesion. The common defect is an inferior homonymous quadrantopsia.

Occipital lobe lesions are characterized mostly by their congruous nature. These can encompass the entire hemianopic field or portions thereof depending on the specific location of the lesion. Riddoch's phenomenon described in 1917, involves the inability to see stationary objects presented on the affected side but could see objects that were moving. Recent research related to focal and ambient visual processing suggests that the ambient visual process is a survival system that particularly responds to movement in the peripheral vision. Perhaps an additional

interpretation from the original one would be that areas of the lesion are being circumvented when feedforward occurs from the ambient visual process. However, when stationary targets are presented to those with PTVS in conjunction with a field loss, it will cause them to over-focalize on the sensory component of the target they are looking at. This over-focalization and lack of an ambient feedforward response can possibly cause a condition related to field neglect and/or extinction phenomenon.

Like visual field loss, visuospatial neglect may manifest as a complete loss of visual perception on the side affected or as a relative loss where less is perceived in the neglected field particularly when there are competing stimuli in the opposite hemifield (57). With appropriate treatment, the functional effect of the hemianopsia and/or neglect can be minimized. Patients with visual field loss who do not receive training, rarely use adaptive search strategies (58). Training the patient to scan into the area of loss, in some cases, creates incomplete recovery of visual field (59–62). Zihl and von Cramon (63) have reported extensive field recovery taking place only during periods of appropriate treatment. Patients with visuospatial neglect require additional training to gain skills for compensation for many activities of daily living.

A homonymous hemianopsia can be functionally very debilitating to the patient in rehabilitation. Not only does it represent a complete loss of visual field, but it also affects concepts of midline. The concept of visual midline shift syndrome is an important one to understand relative to visual motor rehabilitation for any person following a TBI, but particularly for those who have suffered a homonymous hemianopsia. When the visual midline shifts and the individual begins to lean in the direction of the midline, the sensory stimulation from the proprioceptive, kinesthetic, and vestibular system will reinforce the lack of weight bearing on the paretic side. In turn, this also reinforces the development of spatial neglect.

To counter the compressions and expansions that distort spatial perception following TBI, prisms can be effectively used. A prism is a wedge of glass or plastic. Optically when one looks through a prism, the image of what is being looked at appears to be shifted toward the apex or thin edge of the prism and away from the base or thick edge. The shift in image occurs because the prism expands space on one side and compresses space on the other. The prism expands and compresses in a three dimensional manner, but for the purpose of this discussion let us consider it only two dimensionally. The prism can be used to alter the distortion of space caused by a visual midline shift if placed in the appropriate direction before each eye. This is termed a yoked prism system. The amount of prism can be varied depending upon the amount of distortion in the visual process. Yoked prisms are an effective means by which to shift the concept of visual midline, but also should be used in conjunction with other methods of field enhancement or to increase awareness in the affected homonymous hemianopic field.

An enhanced sector prism system is used to affect homonymous hemianopsia by increasing awareness of peripheral visual fields on a sensory level, whereas the yoked prism is used to affect VMSS sensory motor level. The enhanced sector prism system (mounted binocularly or monocularly) is positioned to the side of the line of sight in a spectacle frame with the base end of the prism positioned temporally in the direction of the hemianopic field. When looking into the prism(s), the image will be shifted toward the apex enabling the individual to see objects to the side they would normally not be able to see unless they took a significant eye and head turn in the direction of the hemianopic field. Many individuals with a homonymous hemianopsia frequently bump their head and body on objects that they did not see. This causes numerous bruises as well as the possibility of more significant injuries.

The experienced clinician will recognize that individuals who have a visual midline shift syndrome as well as a homonymous hemianopsia will often have difficulty adapting to the use and succeeding with an enhanced sector prism system because the midline reinforces the spatial neglect. Therefore, treatment with yoked prisms for VMSS is necessary prior to or in conjunction with an enhanced sector prism system. By addressing the issue of VMSS, the success of rehabilitation with an enhanced sector prism system will be increased. The combination of yoked prism in the carrier lenses as well as the sector field prism positioned appropriately laterally to the line of sight can be an effective combination. Rehabilitation with yoked prisms to treat visual midline shift syndrome will directly affect field neglect as well as VMSS. Training can then be established to utilize the enhanced sector prism systems (49, 64). In other cases of visual loss including trauma with constricted visual fields, single or multiple prisms are used mounted laterally on each lens (65).

Sector prism systems used to increase a patient's peripheral awareness can be made out of Fresnel press-on prisms (66). Peli (67) reported that the press-on sectoral prisms when applied to the upper and lower part of the spectacle lens causes a refractive exotropia and thus improves simultaneous awareness and greater obstacle avoidance. However, due to the poor optical quality of the press-on Fresnel prisms, sometimes patients experience a decrease in acuity, or have problems with reflections or distortions which negatively affect the patient's acceptance and success (66). Gottlieb, et al. (65) found that press-on prisms sometimes even decreased a patient's tendency to scan into their field of neglect due to their dislike of looking through these prisms and hence amplified avoidance of this area.

Complex visual loss is no longer an insurmountable obstacle in the way of effective rehabilitation and recovery

of function. Using technology along with proven rehabilitation strategies can aid in meeting the goals of the patient and rehabilitation team.

Literature reinforces that enhanced sector prism systems yield fewer mishaps of bumping into objects or people and their fear of collisions notably decrease (68, 69). The primary result of this improved function is an increase in safety and secondarily a decreased risk for insult and injury (70). In the long term, a patient may experience some recovery of vision in the hemianopic field (49). In a study by Gottlieb, Freeman, and Williams in 1992, 27 of the 34 patients who had been fitted with enhanced sector prism systems to use them full or part time over the next 2.5 years. The prism used in this study increased the patient's peripheral field awareness an average of 13.25 degrees.

## VISUAL PHENOMENA ASSOCIATED WITH TBI

Visual phenomena following a TBI can be quite diverse. Sometimes these phenomena are bizarre and assumed to be of psychological nature. Some of these symptoms will be visual hallucinations, flashing lights, palinopsia, variations in homonymous field defects, and scintillating scotomas. It has been reported that visual hallucinations can be associated with temporal and parietal lobe lesions. Following a TBI, individuals may report hallucinations that take several forms. Some individuals will experience movement of the visual field. Sometimes, the entire visual field will appear to shift or move as they move their eyes or body. Others will report seeing movement in their peripheral vision but when they turn to look, they find only stationary objects. Another common hallucination is seeing things or persons in the periphery and/or central vision. Some patients report seeing snakes dropping from the ceiling or wriggling across the floor. Others will perceive strange type of creatures appearing to move and shift.

Many of these phenomena are reported by persons who have PTVS. It has been found clinically that treatment for post-trauma vision syndrome will often stop the perceived hallucinations (23). Although it has been noted in literature that parietal and temporal lobe lesions as well as occipital lobe lesions can be the cause of these hallucinations, it has also been suggested that some of the hallucinations may be due to psychiatric disorders. It is not uncommon for persons following a TBI who are experiencing hallucinations to be placed on psychotropic medications. It is the author's experience that many of the hallucinations will persist even though these medications are being used.

One possible explanation for the existence of hallucinations following TBI is that when there is dysfunction to the ambient visual process, the ability to stabilize the retinal image is compromised. The superior colliculus is responsible to a great extent for providing the spatial matched information for ambient and sensory motor processes to the occipital cortex but more specifically, to provide a stabilization of the peripheral retina. When the ambient visual process is compromised, the visual processing system that remains, the focal process, isolates to detail but does not have a spatial grounding to stabilize the retinal image.

It has been noted clinically that persons experiencing PTVS will perceive words on a page or objects in the field appear to move when they perform movements of their eyes such as saccadic fixations. Saccadic fixations in a field of vertical lines will produce a shimmering movement of the vertical lines. The lines will appear to move in various manners similar to the movement of a snake. It is hypothesized that at least some of the hallucinations experienced following a traumatic brain injury are due to the unstable nature of the ambient visual process in PTVS. The interpretation of 'snakes' and other objects appearing to move are perceived movement of vertical and horizontal lines in their environment due to the unstable nature of the peripheral retina related to the compromise of the ambient visual process. It has been found clinically that by treating individuals for PTVS, the hallucinations will often cease within hours to days.

## BINOCULAR INTERVENTION

Treatment for diplopia varies depending upon the philosophy of the treating doctor as well as the rehabilitation program. Some do not recommend any treatment and choose to wait to determine if time will be the healing factor. Others recommend patching of the deviating eye to eliminate the diplopia, whereas others recommend patching of the fixating eye in order to stimulate fixation in the eye that is deviating. The lack of any treatment approach and often an inappropriate treatment regimen for a person in a state of diplopia can and will interfere with all aspects of rehabilitation.

The visual process is the dominant and primary sensory motor system affecting all aspects of posture, movement, balance, as well as cognitive-perceptual function. Vision works to reinforce all that we do. If vision is dysfunctional, it will interfere with all performance.

Failure to eliminate diplopia can directly affect the outcome of rehabilitation which financially is being undertaken at tremendous costs by insurance programs as well as individual funding from families and relatives. Therefore, in view of the limited amount of time for rehabilitation and the extraordinary amount of money that is often spent to rehabilitate the person, it is logical to attempt to rehabilitate the condition causing diplopia through treatment of PTVS, compensating prism, and/or

ision therapy. It has been suggested that surgical interention, while possible, is at best a challenge for treating iplopia following a traumatic brain injury (44).

## Pathophysiology of Injuries Contributing to Diplopia

ıjury to the visual system can be diffuse and/or focal and an localize to any, or a combination of the ocular structures, cortical areas, midbrain, or nerve nuclei. Brain ıjuries affecting vision typically occur via axonal shearıg, hemorrhage, infarct, inflammation, and/or compresion. Ophthalmoplegias (cranial nerve palsies) may ompletely, or partially involve any single or all three of he nerves controlling ocular motility.

The third nerve controls the medial, superior and nferior rectus muscles, inferior oblique, ciliary body, lev-tor, and papillary sphincter muscles. Clinically, a com-›lete acquired CNIII palsy will present with ptosis, ıypoexotropia, dilated and fixed pupil, paralysis of ıccommodation and limitation of gaze on the affected ide. Partial involvement can be any combination of, but ıot all of the above. Spontaneous recovery, if any, is usuılly within six months.

The sixth nerve controls the lateral rectus muscle. Clinically, the patient will have an esotropia in primary gaze that increases as they attempt to look towards the ıffected side. There is a limitation of gaze to abduction of he affected eye. Spontaneous recovery, if any, is usually within six months.

The fourth nerve controls the superior oblique muscle. Clinically, the patient will present with their head ilted to the contra-lateral side in an effort to offset cyclical, or torsional movement of their eyes. Spontaneous ecovery, if any, usually occurs within six months.

## Vision Rehabilitation

*Vision Therapy.* Vision therapy (also referred to as vision training or orthoptics) is a clinical approach to treat a variety of visual disorders including certain strabismic conditions. The practice of vision therapy uses a variety of non-surgical procedures to modify the visual process affecting function. By first evaluating the bimodal processing of vision, the doctor will establish a plan of treatment designed to affect balance between the focal and ambient visual processes as well as the ability to match information with sensory motor systems. The goal is to improve the visual process and affect binocularity, spatial organization, balance, movement, etc.

Vision therapy will typically involve a series of treatments. During treatment sessions, individually planned activities are conducted under optometric supervision. The specific procedures and necessary instrumentation are determined by the individual patient's needs and the nature and severity of the diagnosed problems. Vision therapy techniques employ the use of lenses, prisms, computers, biofeedback, stereoscopic devices, and a variety of other instruments and techniques designed to affect the visual process.

*Prism Rehabilitation* As mentioned previously regarding VMSS and visual field loss, prisms are ophthalmic tools that refract light while also compressing and expanding it. Prisms are used clinically in a therapeutic manner to reestablish balance of ambient and focal visual relationships causing visual dysfunction or in a compensatory manner to offset the affects of a binocular imbalance.

In binocular and strabismic dysfunctions, use of prism can reduce symptoms, improve function, and hasten recovery. In these situations, prescription of therapeutic prism (an amount, type, and orientation of prism to reduce or neutralize the binocular/vision dysfunction or to stimulate visual function and fusion) can help reduce or eliminate symptoms and aid treatment. To affect spatial imbalance caused by ambient and focal dysfunction (see PTVS and VMSS), yoked prisms are prescribed.

There are times, patients, and situations when remediation of a visual problem is not possible or feasible. In these situations, prescription of compensatory prism (an amount, type, and orientation of prism to reduce pr neutralize the binocular strabismic dysfunction) can help reduce or eliminate diplopia.

Patching. Patching has frequently been used to eliminate diplopia (21, 71). While effectively eliminating diplopia, patching renders the patient monocular. The chief problems of monocular vision are loss of stereopsis, reduction of peripheral visual field, and VMSS (10, 21, 72).

Monocular vision reduces the field of vision by approximately 25%, decreases stereoscopic vision, decreases visual acuity (due to lack of binocular summation), and impairs spatial orientation. Monocular versus binocular individuals will have a disadvantage in visual motor skills, exteroception of form and color, and appreciation of the dynamic relationship of the body to the environment which facilitates control of manipulation, reaching, and balance (44).

Problems arising from acquired monocular vision will manifest as difficulties in eye hand coordination, clumsiness, bumping into objects and/or people, ascending or descending stairs or curbs, crossing the street, driving, various sports and other activities of daily living which require stereopsis and peripheral vision (72).

In the case of diplopia following TBI, a standard recommendation in acute care facilities and rehabilitation hospitals is to patch one eye. As discussed previously in the section about VMSS, this will cause and/or reinforce a shift in concept of visual midline affecting posture and balance as well as having an adverse affect on physical/occupational therapy.

Patching can be performed but should be done with respect to the ambient visual process. A central occlusion patch can be placed on the deviating eye. For example, if there is a left exotropia, vertical adhesive tape can be placed in front of the deviating eye so that it is built from the nasal portion of the eyeglass frame out to just block the center of the pupil or line of sight. In this way, diplopia will be eliminated when the person looks directly at something, but the peripheral field of the deviating eye is respected by not covering it. In turn, it allows the ambient visual process of both eyes to begin to match information with other sensory motor systems. This will support visual midline concept for posture, balance, and ambulation.

Another method involves use of a partial and selective occlusion. The spot patch is a procedure that eliminates diplopia without compromising peripheral vision (73). It is a small, usually round or oval patch made of adhesive tape, blurring film, or other filters. It is placed on the lens of glasses and directly in the line of sight of one eye. The diameter is generally about one centimeter, but will vary on the individual angular subtense required for the particular strabismus, ophthalmoplegia, or gaze palsy. Final size and placement is determined by evaluating different sizes and shapes to arrive at the smallest one which effectively eliminates the diplopia. If there is a paresis, the spot patch should be placed in front of the affected eye.

By eliminating diplopia through central occlusion, the ambient process becomes more effective and supportive. The spot patch is indicated in cases of intractable diplopia where other methods of treatment are either not viable, have failed, or are contraindicated. Examples of such cases include refractory, third nerve ophthalmoplegia, sixth nerve palsy, and inter-nuclear ophthalmoplegia. Central occlusion is effective if the diplopia is constant and the patient exhibits relief and improved general function as a result of eliminating the diplopia.

Determining size, shape, and placement of a central occlusion patch requires measuring the diplopic field, determining limitation of ocular motility, and measuring the angle of strabismic deviation.

## CONCLUSION

Rehabilitation of a person with a traumatic brain injury requires applying the science of understanding neurological dysfunctions as they pertain to interference with motor, sensory, and cognitive processes. Until recently, if a person suffered visual dysfunction in the way of binocular, spatial perception, and/or perceptual motor dysfunction, rehabilitation treatment was limited to patching of the deviating eye causing diplopia or primarily hospital based occupational and physical therapy.

Recent research has demonstrated that often traumatic brain injury affects visual processing which can cause or relate to the performance dysfunction as well as binocular problems. Neuro-ophthalmology has provided an important means of intervention for neurological problems affecting the visual system. Diagnostic assessment through use of electro-physiology analysis in addition to the state of the art assessment through MRI and CT scan enables a neuro-ophthalmologist to function as a critical member of a multi-disciplinary team serving the person with a TBI. A careful evaluation of cranial nerve function and abnormalities is essential for establishing the diagnosis as well as determining appropriate neuro-medical recommendations for treatment. A range of options are available such as utilizing medications, botulinus toxin, and surgery.

Experience has shown that skilled optometrists familiar with the practice of neuro-optometric rehabilitation can be an important contributing member to the rehabilitation team. The skills of the optometrist are in applying the new science and understanding of the bimodal process of vision to the art of visual rehabilitation. The neuro-optometric rehabilitation evaluation involves the careful analysis of binocularity and spatial and perceptual motor function related to dysfunction between the ambient and focal visual processes by utilizing a variety of techniques incorporating lenses, prisms, and sectoral occlusion. The optometrist can affect performance and function at the appropriate critical stages of rehabilitation not being provided in rehabilitation hospitals and long-term care programs. This does not exclude individuals who are still suffering from binocular and spatial-motor disorders after being released from such programs. Neuro-optometric rehabilitation has been found effective in improving binocularity as well as visual spatial-motor dysfunctions affecting balance and posture by treating PTVS and VMSS years after the neurological insult has occurred.

The role of the neuro-ophthalmologist as well as the optometrist practicing neuro-optometric rehabilitation are important in advancing overall rehabilitation for a person with a TBI. The neuro-ophthalmologist and optometrist will provide critical insights into neurological dysfunction as well as the means by which to rehabilitate the high incidence and prevalence of visual dysfunction affecting performance following a traumatic brain injury.

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