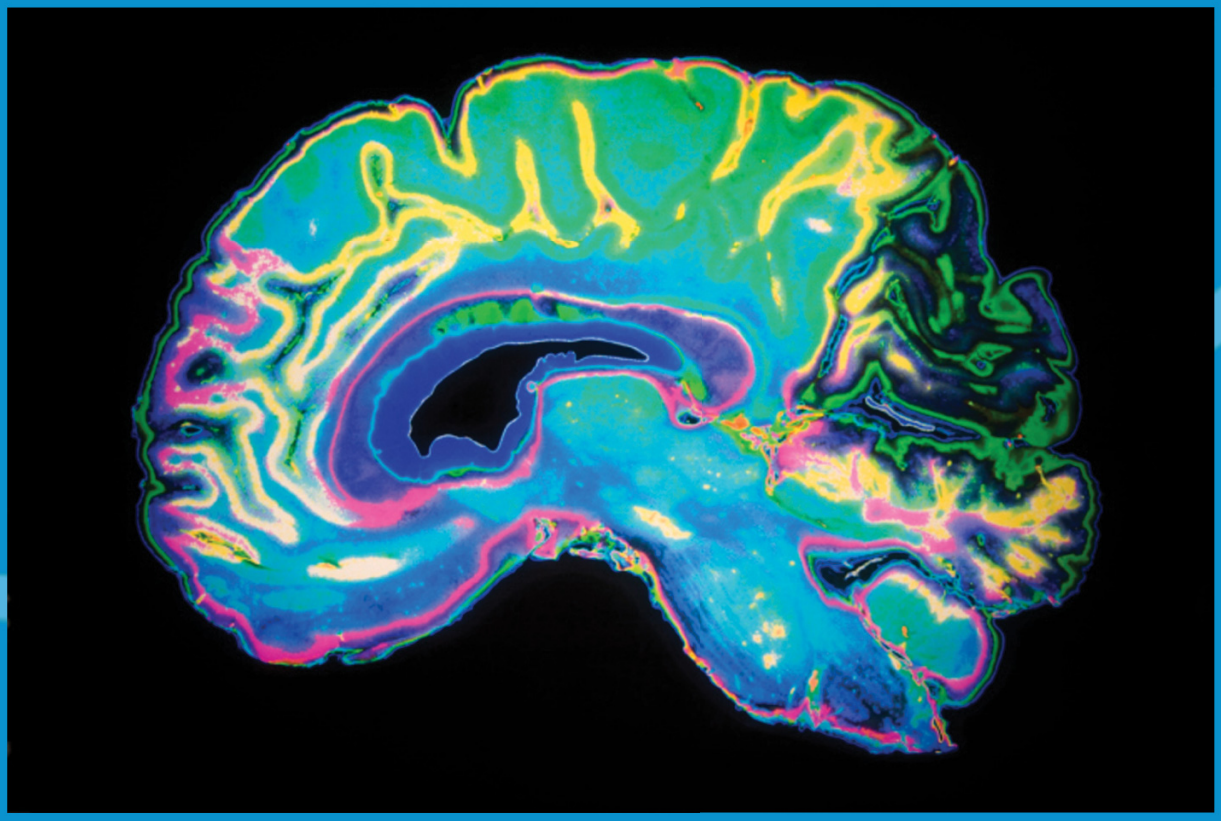
BRAIN INJURY ELECTRONIC RESOURCE MANUAL

VOLUME 1 A : Traumatic Brain Injury

Visual Dysfunction Diagnosis



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American Optometric Association

Vision Rehabilitation Section

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VOLUME I A: Traumatic Brain Injury

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Disclaimer

Recommendations made in this manual do not represent a standard of care. Instead, the recommendations are intended to assist the clinician in the decision-making process. Patient care and treatment should always be based on a clinician's independent professional judgment, given the individual's circumstances, state laws and regulations.

Preface

Traumatic brain injury (TBI) is a subsection of acquired brain injury (ABI). Volumes Ia and Ib cover visual dysfunction secondary only to trauma of the brain from external forces. Subsequent volumes planned will cover visual dysfunctions secondary to acquired brain injury due vasculopathic, mass-effect, infectious, inflammatory and neuro-degenerative diseases, and congenital brain malformations.

The idea for the Brain Injury Electronic Resource Manual (BIERM) came about with the general population's recent increased awareness of brain injury and associated visual dysfunctions. In 2007, much attention was brought to this matter when doctors at the Veterans Affairs (VA) Polytrauma Rehabilitation Center in Palo Alto, Calif., published two manuscripts, one titled "Eye and Visual Dysfunction in Traumatic Brain Injury" and the other titled "Visual Impairment and Dysfunction in Combat-Injured Sevicemembers with Traumatic Brain Injury." These articles played a significant role in the implementation of a VA directive mandating every service member diagnosed with a TBI and treated at any VA Polytrauma Rehabilitation Center to have a very specific and detailed eye exam to rule out ocular trauma and visual dysfunction. The signature injury of the recent and current conflicts in the Middle East was and is TBI, resulting in a huge population base needing to be evaluated. The VA had to set and implement standards for examination and establish VA rehabilitation centers for management of these unique visual dysfunctions.

At about the same time, the media brought to the attention of the general population the importance of identifying and managing TBI secondary to sports injuries. Boxing, football, soccer and any other contact sports were analyzed. Some athletes with concussive syndrome were followed for mild cognitive impairment or even neurodegenerative disease as a result of their head injury. Ocular motility dysfunction was noted to be a major sequela.

Physical and occupational therapists, and optometrists (OD) had long noted visual dysfunction in their stroke patients. A 2008 "Scope of Practice" survey by the AOA found 85.5 percent of ODs saw one or more patients who suffered a neurological insult, a more than 7 percent increase over the previous year's results. One-third of those ODs noted that most of the referrals came from occupational or physical therapists. Interprofessional referrals continued at the same rate in 2011.

Patient examination by neuro-ophthalmologists recognized some of these dysfunctions but failed to support rehabilitation other than the use of prismatic glasses. For many patients, that was all that was needed. Others were left to cope with their visual dysfunctions. Enter optometry: with the unique ability to diagnose AND rehabilitate dysfunctions of the entire visual system. The Vision Rehabilitation Section (VRS) of the American Optometric Association (AOA) decided a resource manual was needed. Members to the committee for brain injury, vision assessment and rehabilitation were co-opted and work on the manual began.

As work progressed, committee members realized the field of brain injury related visual dysfunction assessment and rehabilitation is very well established, but its evidence-based rehabilitative techniques and outcome measures were not readily available. "Evidence-based research" was coined in 1988 when the *Journal of Clinical Epidemiology* first promoted the concept of evidence-based medicine. The term "evidence-based medicine" evolved from that research and in 1996 was officially defined by the Institute of Medicine as "the conscientious and judicious use of current best evidence from clinical care research in the management of individual patients." I stress "clinical care research." As early as 1988, *Optometry: Journal of the AOA* published "The Efficacy of Optometric Vision Therapy," which provided a comprehensive review of "evidence-based research" on all binocular dysfunctions – including oculomotor dysfunction. This was just the beginning of "evidence-based" research on rehabilitation of oculomotor dysfunction, common in the general population as well as the most common sequela of TBI. Much of the literature to date describes assessment and diagnosis, recognition and categorization of visual dysfunction. A

limiting component to all of this research is that patients with TBI often have multiple compounding issues including cognitive deficits and the use of multiple vision affective medications.

Consistent with all professions involved with the management of patients with brain injury is the emergence of new or expanded definitions and terminology. Archaic terms are being replaced with newer, less confusing terms found to better describe clinical findings. Within the profession of optometry, "neuro-optometric rehabilitation," also known as "Brain Injury Vision Assessment and Rehabilitation," is an emerging sub-specialty. As rehabilitation professionals become more aware of this optometric service and its positive impact on the success of overall rehabilitation for the complex and undeserved TBI population, these terms will become more widely used and understood.

In this consensus-based manual, every attempt is made to provide best practices. Some strategies are not verified by sound research, and they are so acknowledged. Some terms are less recognized than others and are noted along with their associated synonyms. All of this points to a discipline that indeed is still young and trying to find its footing to prove that it is sound. Even though research is difficult with this population base, evidenced-based research provides best care information and for the sake of our patients we must all do our part to promote evidence-based research in the area of visual rehabilitation and ultimately provide the best sound care available.

Chrystyna Rakoczy, O.D.

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SECTION 1: Introduction

Mitchell Scheiman, OD

Vision Problems Associated with Traumatic Brain Injury

The treatment of vision disorders related to traumatic brain injury (TBI) is one of the more challenging aspects of optometric practice. Patients who survive TBI generally experience multiple problems, including physical, cognitive, behavioral, motor, and sensory anomalies[1]. Even when vision disorders, such as binocular vision, accommodative, eye movement, and visual processing problems, occur in isolation of cognitive and psychological issues, they tend to be complicated. Cyclovertical and noncomitant deviations, sensory fusion anomalies, unequal accommodation, and visual field loss may accompany the basic vision disorder.
The objectives of this introduction are to review the role of optometry in the management of vision problems after TBI, the prevalence of vision problems after TBI, the impact of these vision disorders on quality of life, and the evidence of treatment effectiveness.

Role of Optometry

Vision problems are common after TBI, and they have a significant negative impact on common activities of daily living, such as reading, writing, shopping, dressing, sports, and driving [2]. Historically, optometrists have not been part of the rehabilitation team in hospitals[3, 4]. This team typically includes various physicians and rehabilitation professionals, such as occupational, physical, and recreation therapists and speech language pathologists. Eye care is usually provided by an ophthalmologist, with a primary emphasis on visual acuity and eye disease[5]. As a result, it is common for some vision problems associated with TBI to be left undetected or untreated, leaving patients with significant functional problems[6-9].
In the past decade, however, optometrists have become more involved in management of vision problems associated with TBI[2, 4, 7, 8, 10-15]. The optometric role includes management of refractive error, binocular vision, accommodative, and eye movement disorders, visual field loss, ocular disease, and other visual manifestations of TBI. Because of the complexity of TBI, it is important for optometrists to work closely with the rehabilitation team. The nature of this interaction varies with the timeframe after the TBI.

Active Rehabilitation Stage

During the active rehabilitation stage, it is critical for the optometrist to work closely with the rehabilitation physicians and occupational, physical, and speech therapists. Because of the high prevalence of vision problems after TBI, these rehabilitation specialists often encounter patients with vision problems that interfere with the rehabilitation process. If left untreated, these vision problems can interfere with progress and act as an obstacle for the patient to resume normal activities of daily living.[16-18] Optometrists with staff privileges and working as consultants at these facilities can provide an important service by identifying and managing the vision problems of patients with TBI in a timely manner. During the early phases of rehabilitation, passive treatment (lenses, prism, and occlusion) is often prescribed to make the patient as comfortable as possible during rehabilitation. The optometrist should also educate the rehabilitation team about the nature of the vision problems. This education should include information about the effect of these problems on various activities of daily living and how the therapists can modify the environment and tasks to enable the patient to function most effectively. During this period of time, it may be difficult for the optometrist to perform active vision therapy. However, some optometrists are prescribing active vision therapy administered in the rehabilitation hospitals by occupational therapists and directly supervised by the optometrist.[23] In this scenario, the optometrist identifies the problem, prescribes and programs the active vision therapy, supervises the therapists who perform the vision therapy, and performs periodic re-evaluations. When the

active phase of rehabilitation ends and the patient returns home, the optometrist can take full control of the patient's functional vision care. The active vision therapy program would then continue within the optometric office.

Post-Rehabilitation Stage

Other scenarios are also possible. Because the majority of optometrists do not have staff privileges at rehabilitation hospitals, they will more likely be involved with TBI patients who do not require hospitalization (mild traumatic brain injury) or patients who have completed active rehabilitation. In other instances, the optometrist may not have the opportunity to examine the patient with TBI for months or even years after the problem first occurs. The patient may turn to the optometrist as a last resort. Often the patient has already been to a number of ophthalmic practitioners and been told either that there is no problem or that treatment is not possible. In these situations, the optometrist is the primary caregiver. If the patient has recently been discharged from a rehabilitation facility, close cooperation with the patient's therapists will still be important.

Ciuffreda et al. [7] summarized the potential role optometrists should play with TBI patients:

> "The provision of full-scope optometric care to this underserved and poorly understood clinic population offers a unique and altruistic opportunity for the profession. By such a comprehensive approach, in conjunction with other members of the rehabilitative team, patients with TBI can more rapidly re-enter society and the work force, and once again be productive members of the community."

Prevalence of Vision Problems after Acquired Brain Injury

Binocular Vision Disorders

Binocular vision deficits have been reported to be among the most common vision problems occurring in both the civilian and the military populations after acquired brain injury [4, 12, 19-25]. Gianutsos et al. [4] performed a vision evaluation on a population of 55 severely brain-injured individuals in a rehabilitation facility for individuals requiring long-term treatment. The most common problems found were binocular vision disorders. Cohen et al. [19] found convergence insufficiency in 38 percent of acute TBI patients and in 42 percent of patients re-evaluated three years after TBI. Suchoff et al.[12] examined 62 brain-injured patients who resided in extended care facilities and found a high occurrence of exodeviations (41.9 percent), including convergence insufficiency and intermittent and constant exotropia. There were also a number of patients with vertical deviations (9.7 percent). Ciuffreda et al. [20] did a retrospective review of 220 patient records with either TBI or cerebral vascular accident (CVA). Vergence deficits (56.3 percent) were most common in the TBI subgroup. Convergence insufficiency was the most common dysfunction found in the TBI subjects (42.5 percent).

Five recent studies [21-25] of military personnel returning from the wars in Iraq and Afghanistan found a similar pattern after acquired brain injury. In these studies, convergence insufficiency was found to be the most common visual disorder after TBI with prevalence from 30 percent to 42 percent.

Accommodative Disorders

The pre-presbyopic, civilian-acquired brain injury population consists primarily of TBI. In this population, accommodative disorders, such as accommodative insufficiency, excess, and infacility, are more common than in the general clinical population.[26] Al-Qurainy[27] reported approximately 20 percent of people with TBI have an accommodative dysfunction. Suchoff et al.,[12] in a study of 62 consecutive patients with TBI, found about 10 percent had accommodative problems. Kowal[28] found 36 percent of 161 head-injured patients had accommodative problems. In the Ciuffreda et al.[20] study of 220 patient records with either TBI or CVA, accommodative dysfunction was found (41.1 percent) in patients with TBI with nearly all showing accommodative insufficiency. Accommodative problems have also been reported in case studies[2, 9, 29, 30].

In recent studies of military personnel with TBI by Goodrich et al.,[22, 25] Brahm et al.,[23] Stelmack et al.,[21] and Capo-Aponte,[24] accommodative dysfunction was found in 22 percent, 42 percent, and 47 percent of the subjects respectively.

Eye Movement Disorders

Suchoff et al. (26) found about 40 percent of the patients had eye movement disorders. In a study of the civilian population with TBI, Ciuffreda et al.[20] found deficits of saccades in 51 percent of patients after TBI and 57 percent in patients after stroke. The three recent studies of vision disorders in military personnel after TBI revealed a high prevalence of pursuit and saccadic dysfunction ranging from 6 percent to 33 percent.[21-23]

Abnormal Spatial Sense

Another common problem associated with acquired brain injury is abnormal spatial sense. Symptoms may include poor balance and posture, bumping into objects, spatial disorientation, lateralward bias in walking, dizziness, and a sense of being "out of synch" with their environment. [31, 32] Ciuffreda and Ludlam describe the problem as a mismatch, or discrepancy, in the incoming perceptual information, between one's subjective versus objective sense of straight ahead.[33] In individuals with normal spatial sense, the subjective and objective spatial sense are identical and objects are perceived to be straight ahead. However, in patients with abnormal visual spatial sense the perception of straight ahead is shifted several degrees to one side. This mismatch may produce visuo-motor problems associated with walking and eye-hand coordination, as well as misperception of the apparent distances of objects in the environment. Ciuffreda and Ludlam attribute these problems with spatial sense difficulty to abnormal egocentric localization.[33] They define egocentric localization as the localization of objects in the environment with respect to one's "self" or "egocenter."[33]

Abnormal spatial sense is common in post-trauma vision syndrome" (PTVS),[34, 35] which is a common visual sequelae of traumatic brain injury originally described by Padula.[34] Padula[34] describes PTVS as a constellation of oculomotor, attentional, and cognitive problems following a brain insult. Signs may include exophoria, convergence insufficiency, oculomotor problems, accommodative dysfunction, visual field loss, reading problems, and visual memory lapses. Symptoms may include diplopia, blur, light sensitivity, concentration difficulties, visual motion sensitivity, and poor spatial judgment/depth perception.[34, 36]

Other Visual/Eye Disorders

In the recent studies of vision disorders in military personnel cited above, visual field disorders were reported in 14 percent to 32 percent of the patients.[21-23, 25] Rutner, Kapoor, Ciuffreda et al.[37] retrospectively reviewed 160 medical records of individuals with TBI to determine the frequency of occurrence of ocular disease in the two major sub-groups of acquired brain injury. Conditions with high relative risk unique to TBI included corneal abrasion, blepharitis, chalazion/hordeolum, dry eye, traumatic cataract, vitreal prolapse and optic atrophy.

Vision Disorders after TBI and Quality of Life

Most symptoms are typical of those experienced by any patient with binocular, accommodative, and eye movement disorders and are often associated with reading or other close work. Common complaints include eyestrain and headaches after short periods of reading, blurred vision, diplopia, loss of place, sleepiness, and difficulty concentrating on reading tasks. These visually related symptoms may have a negative impact on many activities of daily living, including any task requiring reading, driving, sports, and other leisure activities. In addition to these visually related symptoms, there are often other associated symptoms characteristic of patients after TBI. Stelmack et al.,[21] recently reported on the symptoms of 88 patients after TBI. They found a very high prevalence of more than 20 different symptoms in this population, including feeling dizzy, loss of balance, poor coordination, noise sensitivity, poor concentration, difficulty making decisions, fatigue, loss of energy, feeling anxious or tense, feeling depressed or sad, and poor frustration tolerance. Other studies found similar symptoms, including poor balance and coordination, [2, 38] dizziness,[2] and light sensitivity.[2, 39] The combination of visual symptoms and more general symptoms may cause a great deal of difficulty for the patient attempting to return to activities of daily living.

Evidence of Treatment Effectiveness

Today, in this era of evidence-based health care, it is important to integrate the best research evidence with our clinical expertise when treating patients.[40] The phrase "best research evidence" refers to valid and clinically relevant research. Thus, when looking at the literature for support of treatment effectiveness, it is important to understand the value of each study when assessing the implications of the study results. In 1996, the AOA published a white paper providing additional references and background at www.aoa.org/Documents/optometrists/acquired-brain-injury.pdf.

As described above, the literature clearly shows the three most-common visual problems after TBI are convergence insufficiency, accommodative disorders, and oculomotor dysfunction. Fortunately in the past decade a number of high-quality studies were published demonstrating optometric intervention is effective for these three conditions. In regard to convergence insufficiency, the Convergence Insufficiency Treatment Trial (CITT) Study Group published the results of four randomized clinical trials[41-44] demonstrating office-based vision therapy is the most effective treatment for convergence insufficiency. In these studies that also included a placebo control group, home-based pencil push-ups, and home-based computer vision therapy, office-based vision therapy was found to be effective in about 75 percent of the patients. The two home-based vision therapy groups were no more successful than placebo therapy. In addition, the CITT group was able to evaluate the effectiveness of office-based vision therapy for improving accommodative function because many patients in the CITT studies had both convergence and accommodative insufficiency. They found a mean change in accommodative amplitude of 8.5 D and a mean change of 8.4 cycles per minute in accommodative facility after 12 weeks of office-based vision therapy.[45]

Although the CITT studies represent high-level evidence, the patients in these studies did not have any history of TBI. Thus, one could question whether these results can be generalized to the more complicated patient with convergence and or accommodative insufficiency along with TBI.

Numerous investigators have described treatment of binocular vision, accommodative, and eye movement problems associated with TBI using lenses, prism, and vision therapy.[2, 6, 7, 9, 10, 13-15, 29, 30, 34, 46-51] These reports, all of which were retrospective case studies or series of cases, suggest vision therapy can be effective in relieving patient symptoms and improving visual function. These studies do suggest vision therapy may be effective in this population and support the need for large-scale, prospective studies of treatment of vision disorders after TBI.

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SECTION 2: Evaluation and Assessment

A. Assumptions/intent

Section II is intended for optometrists who wish to review evaluation procedures that are high-yield for the TBI population. Most techniques will be familiar, but nuances for examination of the patient with TBI are addressed. In this resource manual, we point out clinical pearls and advanced evaluations that apply to patients with TBI. Information on general eye exam procedures can be found on the AOA website under Optometric Clinical Practice Guidelines: "Comprehensive Adult Eye and Vision Examination" http://www.aoa.org/documents/CPG-1.pdf.

SECTION 2: Evaluation and Assessment

B. Diagnostic Instrument List

In addition to the standard optometric equipment used for routine examination, the following instruments are found to be useful in examining the patient with TBI:

Lea acuity cards: number and symbols
Teller cards
Patti Picks cards
Peli-Robson (considered the standard), MARS Chart or Sloan contrast charts
Farnsworth D-15 or D-24
Worth 4 dot
Stereo chart (Randot)
Vertical and horizontal line near and distance targets
Horizontal and vertical prism bars
3BI/12BO Prism flipper
Flipper set (+/-0.50 to +/-3.50)
Red Amsler Grid
MEM cards and holder
Hand held Risley prism
Monocular Maddox Phoria measure or Maddox Rod paddle
Red lens paddle
Halberg/Jannelli trial lens clips (or other clip)
Modified Thorington
Fixation Disparity testing
DEM for adults
Trial frame
Loose lenses including loose Maddox and prisms
Portable/hand-held slit lamp
Tonopen/Perkins
Streff wedge for binasal occlusion
King-Devick Test
Rotating prisms
Reading cards for near point retinoscopy

SECTION 2: Evaluation and Assessment

C. Examination Approach

Kara Gagnon, O.D., and Chrystyna Rakoczy, O.D

1. Approach

The patient with traumatic brain injury usually presents in one of two ways:

a. The communicative patient who comes in with symptoms and may or may not have had brain imaging or other evaluation for clues to a diagnosis
b. The non-communicative/non-responsive patient who cannot tell you his/her symptoms but may have brain imaging reports for review

The examination approach to each is different. In the case of the communicative patient, we start with the detailed history and symptomatology and work our way through the exam, evaluating responses to achieve the diagnosis. It is important to note the temporal aspect of the symptoms, including patterns, rapidity, chronicity and associations.

In the case of the non-communicative/non-responsive patient, we look at the known history and the nature of the injury via brain image interpretation and deduce the expected symptomatology. The patient can be evaluated for the suspected dysfunctions at a later date when cognitive impairment/communication skills have improved.

Communication with a nonverbal patient may be a challenge. Establish the best method by speaking with a family member or caretaker. Some patients will use "thumbs up" for yes and "thumbs down" for no. Others may use eyebrows lifted for yes and frowned for no. Use a method with which the patient is most familiar.

2. Considerations in Examining the Patient with TBI: Tips for Better Evaluation

When performing the optometric evaluation, the doctor should keep in mind that the patient with TBI will frequently process questions and commands more slowly. Patient responses can be delayed and unreliable, providing inaccurate results with subjective tests. Objective measurements in many cases will provide a better outcome and lead to proper diagnosis.[1]

Points to remember:

- Be prepared.
- Get as much history as possible in advance of the exam to minimize patient stress.
- Consider the patient's cognitive level and attention span.
- Efficiently move from one test to the next to keep patient's interest.
- Break up the exam into segments. Give the patient a short break in between groups of tests as you note your results.
- Do not expect to complete the exam in one visit. Plan to divide it into two to four visits. Be sensitive to patient fatigue.
- Anticipate certain diagnoses based on area of insult and test for them specifically.
- Re-test if needed, on subsequent examination.
- Speak more softly and slowly.
- Limit distractions and interruptions.
- Repeat information and/or write it down for the patient.
- Include interactive activities to keep patient's attention and redirect as needed.

- Prepare the patient for new activities. Present new information in small bits.
- Focus on patient's achievements rather than deficits.
- Do not rush the patient.
- Change the activity if the patient is agitated.
- Encourage eye contact.
- Set goals and expectations/objectives with the patient.
- If a problem arises, offer alternative solutions for the patient and encourage them to make the best choice.
- Note that loud wallpaper or wall colors can be distractive to the patient.
- Do not be fearful of transferring a patient to the exam chair if the caregiver is present.
- **Modify your exam to the patient's level of participation.**[2]

References:
1. Cohen A. Acquired visual information-processing disorders: closed head trauma. In: Press L, ed. *Applied Concepts in Vision Therapy*. St. Louis: Mosby; 1997:154-166.
2. Hibbard MA, Gordon WA, Kenner B. The Neuropsychological Evaluation: A Pathway to Understanding the Sequelae of Brain Injury. In: Suchoff IB, Ciuffreda KJ, Kapoor N, eds. *Visual and Vestibular Consequences if Acquired Brain Injury*. Santa Anna CA: Optometric Extension Program Foundation; 2001

3. Pre-History Observation

Optometric evaluation starts prior to the actual clinical examination. The doctor should be observing the patient with TBI as he/she walks into the exam room and ambulates to the exam chair.[1] The doctor should pay careful attention and look for evidence of motor weakness, imbalances, poor coordination of movement, poor visual spatial judgments, visual field defects, head tilts, and compensatory adjustments.[2]

Observe:

1. Body position: leaning forward, backward, to the side?
2. Centering: walking in the middle of a hallway or veering off to one side?
3. Head turn or tilt?
4. How the patient arrives: unaided ambulation, cane, walker or wheelchair?
5. Patch on one eye?
6. Oriented to time and place, alertness?

Continue your observations throughout the exam. Look for abnormal head tilt or posturing, especially during acuity and ocular motility.

References:
1. Cohen A. Acquired visual information-processing disorders: closed head trauma. In: Press L, ed. *Applied Concepts in Vision Therapy*. St. Louis: Mosby; 1997:154-166.
2. Falk NS, Aksionoff EB. The primary care optometric evaluation of the traumatic brain injury patient. *J Am Optom Assoc* 1992;63:547-53

SECTION 2: Evaluation and Assessment

D. Detailed TBI History and Symptom Review

Chrystyna Rakoczy, O.D., Kara Gagnon, O.D., and Brenda Heinke Montecalvo, O.D.

The descriptions below constitute a detailed review of the TBI specific history and associated symptoms. For optometrists who wish a quick high-yield questionnaire and exam procedures, please refer to the appendix.

1. TBI Narrative (the patient's story in addition to the routine patient and family history)

The TBI narrative includes a recap of pertinent medical events surrounding the injury. Most notably, loss of consciousness (LOC), altered state of consciousness (AOC) and post-traumatic amnesia (PTA) need to be qualified and quantified. In many cases, obtaining the incident history and post TBI treatment when interviewing the patient may be difficult. It may be necessary to access prior medical records, including other rehabilitation center notes, and obtain additional input from caregivers.[1, 2]

The definition of traumatic brain injury is that of a "traumatically induced structural injury and/or physiological disruption of brain function as a result of an external force that is indicated by new onset or worsening of at least one of the following clinical signs immediately following the event:

1. Any period of loss of or a decreased level of consciousness (LOC)
2. Any loss of memory for events immediately before or after the injury: post traumatic amnesia (PTA) is the time interval from when the person regains consciousness until he or she is able to consistently form memories of ongoing events
3. Any alteration in mental status at the time of the injury (AOC)
4. Any neurological deficits that may or may not be transient" [1]

Neurologists use various scales to determine the characteristics of a brain injury. Knowing these values can aid in determining readiness for rehabilitation as well as success of rehabilitation.

The Glasgow Coma Scale[3] (GCS) determines the characteristics of TBI. It is a neurological scale used to assess the conscious state of a person by evaluating: eye opening, verbal responses, and motor responses. These scores are used for initial evaluation as well as monitoring progress and determination of prognosis of long-term sequelae. (See appendix for scale)

General Characteristics of the GCS:

- Patients with a GCS of less than 8 are intubated
- 50 percent of the patients with a GCS equal to or less than 8 at six hours after the injury do not survive
- Initial "post-resuscitation" score is the most accurate predictor of future outcome
- Severe TBI = GCS of 3 to 8 (in a coma)
- Moderate TBI = GCS of 9 to 12
- Mild TBI = GCS of 13 to 15

TBI is categorized into three levels using the GCS, LOC, and PTA:

	GCS	LOC	PTA
Severe	3-8	>24hrs	>7 days
Moderate	9-12	>30min <24hrs	>1day <7days
Mild	13-15	0-30min	<1day

The Rancho Los Amigos Levels of Cognitive Function[2] scale describes a patient's level of cognitive function and is used in stages of recovery of moderate to severe brain injury. It is used in the first weeks and months following injury, because it does not require patient cooperation and is based on observation of responses to external stimuli. There are eight levels ranging from no ability to understand/respond to full cognition. (See appendix for scale)

References:
1. Defense and Veterans Brain Injury Center. http://www.dvbic.org/ Accessed June 15, 2013
2. Rancho Los Amigos National Rehabilitation Foundation. http://www.rancho.org/research/bi_cognition.pdf. Accessed June 15, 2013
3. The Stroke Center. http://www.strokecenter.org/wp-content/uploads/2011/08/Glasgow_coma.pdf. Accessed June 15, 2013

Suggested TBI history questions:

When did the brain injury incident happen?
What caused the injury?
Was the brain injury penetrating or non-penetrating?
Is the Glasgow score known? Rancho level?
Does the patient know if he/she lost consciousness or were disorientated after the TBI?
What was the duration of PTA?
Did the patient report sustaining an eye injury (ocular and/or periocular trauma)?
Has the patient undergone visual rehabilitation? Ask details.
Is this injury under litigation?

Additional optometric history:

In addition to asking the routine optometric history and specifics of the injury, the doctor should investigate prior pre-injury ocular/visual history incidents, diagnoses and treatments, specifically those relating to the oculomotor system. Any history of eye injury, eye surgery, double vision, "lazy eye," amblyopia, strabismus, patching, refractive surgery, vision therapy, face turn, head tilt, ocular allergy, photosensitivity, field deficit, poor reading abilities, and vision therapy should be well documented.

Watch for cues:

During the history, the doctor should pay attention to how the patient responds to the doctor's questions. Can the patient organize his/her thoughts and respond with comprehensible answers? Does the patient have speech issues, can he/she express themselves freely and fluently (do they have aphasia)? What is the attentional level? Does the examiner have to repeat questions and directions continually during the history. Does the patient provide eye contact, etc.[1]? Does the patient have difficulty retaining information[2]? Assess his/her short-term memory by asking what he/she was doing before the exam, such as what he/she ate in the morning, etc.[1] The optometrist should not only ask questions and document the answers, but should also be a detective by observing the patient's interactions,

level of attention, and ability to respond coherently and meaningfully. After all, the history is a critical informative part of the exam.

References:
1. Falk NS, Aksionoff EB. The primary care optometric evaluation of the traumatic brain injury patient. *J Am Optom Assoc* 1992;63:547-53
2. Reitan RM, Wolfson D. Traumatic brain injury. Vol I, Pathophysiology and neuropsychological evaluation. Tucson, AZ: Neuropsychology Press, 1986; 43-59.

2. Review of Visual Symptoms Related to TBI

In any discussion of visual symptoms, a positive response should be followed by these questions:

When did the symptom start? (Chronicity)
Was it sudden or gradual? (Rapidity)
What is the pattern?
Are there any associated symptoms? (Associations)
Can you do anything to help the symptom or make it worse? (Mitigation)

Optometrists are familiar with the appropriate questions to ask in qualifying accommodative, ocular motor, and vergence deficits. Please refer to the following symptom survey lists that should be incorporated into the TBI history intake portion of the exam.

a) Symptoms of Oculomotor Dysfunction Associated With TBI[1, 2]

1. Blurred vision at distance or near (with best prescription)
2. Face or head turn
3. Covering/closing an eye
4. Sensitivity to light (indoors, outdoors, glare, light adaptation)
5. Decreased night vision
6. Headache or brow ache
    - Diagnosis of specific type of headache by medical professional?
    - Does it start during specific tasks: Awake you from sleep? When reading? When concentrating?
    - Localization
    - Frequency/duration
        - Intermittent or constant
        - Time of day it occurs
        - Times per week/month?
    - Relieved by any means
    - Severity and type of pain
    - Associated neurological factors (nausea, vomiting, dizziness, loss of balance)
7. Double vision:
    - If strabismus is obvious, ask if patient sees double. If the patient does not, but has obvious strabismus, the patient is suppressing.
    - Is double vision persistent when covering an eye?
    - Localization:
        - Present at distance or near or both (if both, is it worse at distance or near?)
        - Horizontal, vertical, diagonal, variable
        - Worse in left, right, up or down gaze?
    - Frequency/duration
        - Intermittent or constant

        - Time of day it occurs
        - Times per day, week, month?
    - Relieved by any means?
8. Associated factors such as pain on movement?
9. Wandering eye
10. Missing part of visual field/restricted field of vision, neglect of one side of the body? Is it to the right or left?
11. Issues with reading:
    - Discomfort while reading/pulling or tugging sensation
    - Fatigue and/or strain while reading
    - Skip or lose place while reading
    - Have difficulty finding the next line
    - Words moving/lifting/ or swimming off the page
    - Doubling of words
    - Transient blur while reading, unable to sustain near work/reading
    - Difficulty shifting focus from near to far or far to near
    - Poor reading comprehension
    - Easily distracted/decreased attention span
    - Poor concentration
12. Pain in or around the eyes or pain with movement of eyes
13. Visual disturbances while moving

References:

1. Kapoor N, Ciuffreda KJ. Vision disturbances following traumatic brain injury. *Curr Treat Options in Neurol* 2002, 4(4):271-280
2. Post Trauma Vision Survey used at State University of New York State College of Optometry, Vision Rehabilitation Service, ABI clinic. See Appendix.

b) Symptoms of Possible Ocular Disease Secondary to TBI

1. Flashes of light
2. Floaters
3. Curtain/shade over visual space
4. Dry eye symptoms including any of the following: burning/itching/tearing/redness/matting in morning/foreign-body sensation/mucous discharge
5. Illusions or hallucinations

c) Symptoms of Visual Processing Dysfunction Associated with TBI[1-3]

1. Delayed reaction time
2. Poor organization
3. Over generalizes
4. Difficulty reading
5. Motor-based:

   Poor penmanship
   Knocks cup over while reaching for it
   Projects off-center during mobility activity
   Frequently runs into wall or door jam on one side
   Has bruises on one side of hand and or hip
   Difficult copying words or numbers
   Poor coordination and balance

6. Memory-based:
   Does not remember what is recently viewed
   Cannot finish an idea or sentence
   Cannot retrieve or recall correct words to use in sentence
   Cannot retrieve name of a familiar person
   Poor short-term memory
   Cannot identify mistakes in written or spoken language
   Problems recognizing visual shapes, objects and familiar faces
   Confuses minor likenesses or differences
   Problems understanding instructions
   Cannot remember a series of instructions

7. Orientation- and localization-based:
   Poor sense of direction
   Confusion of right and left
   Difficulty aligning numbers in columns
   Reverses letters and/or numbers, series of numbers or words
   Problems judging distance between themselves and objects
   Lean to one side
   Lateralward bias in walking
   Bumps into objects on one side
   Misses first or last letter of acuity line

Gross level evaluation is to observe patient behavior in the reception area as the patient transfers to the examination room. Note if the patient sits or leans to one side in the chair, veers to the right or left when walking or leans forward or back when sitting. Also observe head position, if it is rotated right, left, forward, back or tilted right or left.

References:
1. Sanet R, Press L. Spatial vision In: Suter, ed. Vision Rehabilitation, Boca Raton: CRC Press, 2011; 104-106.
2. Arcia E, Gualtieri C. Association between patient report of symptoms after mild head injury and neurobehavioral performance *Brain Inj* 1993;7(6):481-489.
3. Lowell L, Cohen A, Kapoor N Optometric management of visual sequelae of frontal lobe related to traumatic brain injury *J Beh Opt* 2010;21:3-11.

d) Symptoms of Egocentric Localization Dysfunction[1]

1. Lean to one side
2. Lateralward bias in walking
3. Bump into objects on one side
4. Miss first or last letters of acuity line

References:
1. Ciuffreda KJ, Ludlam DP. Egocentric localization normal and abnormal Aspects. In: Suter PS, Harvey LH, eds. *Vision Rehabilitation Multidisciplinary Care of the Patient Following Brain Injury.* Boca Raton FL: CRC Press; 2011; 193-209

3. Review of Vestibular TBI Symptoms

As practitioners we should be diligent in recognizing the multi-sensory integration of the vestibular and visual systems. TBI commonly results in imbalance and dizziness along with oculomotor dysfunction, including vertical and horizontal ocular misalignment. Vestibular rehabilitation is a dynamic, multidisciplinary (optometry included) management strategy for central and peripheral vestibular dysfunction.

Asking questions about the vestibular system can at times be confusing, not only for the patient, but also for the doctor. Symptom terminology can encompass many meanings. Background information is included below to help clarify vestibular anatomy and symptom terminology.

The sense of balance (equilibrium) is the result of the neurological interplay between three highly integrated systems: visual, vestibular and somatosensory. Each system has a peripheral and a central component.

Visual system:

- Peripheral: eyes, extraocular motility, accommodative system and optical media
- Central components:
  - Ventral stream: temporal lobe giving us information about WHAT we see
  - Dorsal stream: parietal lobe giving us information about WHERE objects are in space, our spatial orientation, balance posture, movement, and ordination

Vestibular system

- Peripheral: labyrinths, cranial nerve VIII and its root entry
- Central: vestibular nuclei, cerebellum, and integrative pathways

Somatosensory:

- Peripheral: mechanoreceptors and peripheral nerves
- Central: spinal cord, primary somatosensory cortex in the parietal lobe, and the cerebellum

It is important to realize the vestibular system and the interaction between the visual-vestibular systems are commonly affected in the patient with TBI. Insult to these systems results in unfavorable symptoms such as dizziness and disequilibrium. Between 23 percent to 81 percent of mild TBI cases are reported to have symptoms affecting their sense of balance[1]. With these statistics, it is critical that optometrists who are part of the rehabilitative team "understand these symptoms and manage the optometric sequelae of these injuries."[2]

The evaluation of "dizziness" begins first with the optometrist understanding this term and all it encompasses. "Dizziness" in an ambiguous term, and many patients will have difficulty describing this symptom. Below is a table adopted from Weiss[2] to help the doctor understand the nuances.

VERTIGO	Illusion of movement or spinning sensation of themselves or their environment caused by mismatch of the visual, vestibular and somatosensory inputs
DISEQUILIBRIUM/IMBALANCE	Unsteadiness when walking
LIGHT-HEADEDNESS	Sensation of fainting, typically vascular in origin. Symptoms are mild and may be precipitated by stress, anxiety, depression, or panic.
NEAR SYNCOPE	Patient is close to fainting but does not lose consciousness. Associated symptoms include: buzzing in ears, weak legs, constricted fields, pallor, sweating, and nausea.

Other definitions to understand include:

OSCILLOPSIA: illusionary "to-and-fro" movement of the environment;

- Caused by unstable fixation due to neurological or mechanical disorder
- Is of vestibular origin only if exacerbated by head movement

TILT: static rotation of the perceived world or body

- Caused by disturbance in otoliths

When asking a patient with TBI about symptoms, one should incorporate questions specific to probing visual-vestibular dysfunctions. It is important to ask these questions because they will not only guide optometric management, but will also need to be relayed to other rehabilitative team members as these symptoms can affect the progress seen in other therapies.

In any discussion of vestibular symptoms, a positive response should be followed by the following questions:

- When did the symptom start? (Chronicity)
- Was it sudden or gradual? (Rapidity)
- What is the pattern?
- Are there any associated symptoms? (Associations)
- Can you do anything to help the symptom or make it worse? (Mitigation)

1. Do you experience dizziness (light-headedness, vertigo, spinning of yourself or world around you, balance issues)[3]?

If yes, do they occur with particular postures or movements such as: [4]

- When riding in moving vehicle
- Walking down a hall or aisle in a store
- Concentrating at near
- Walking in a crowd
- Approaching busy intersections

2. Are symptoms worse with eyes open or closed?
3. Do you feel like you have balance issues or feel unsteady as if you may fall when walking[4]?
4. Are you bothered by movement in the spatial world (crowded/or congested mall/parking lot, etc.)?
5. Do you have vertigo and the subjective feeling of dizziness induced by sound (Tullio phenomenon)[1]?

References:

1. Alsalaheen BA. Vestibular rehabilitation for dizziness and balance disorders after concussion. *J Neurologic Physical Therapy.* 2010; 34:87-93
2. Weiss LM. Visual-vestibular interaction in the acquired brain injury patient. *Journal of Optometric Vision Development*. 2002; 33:33-41.
3. Falk NS, Aksionoff EB. The primary care optometric evaluation of the traumatic brain injury patient. *J Am Optom Assoc* 1992; 63:547-53.
4. Cohen A. Acquired visual information-processing disorders: closed head trauma. In: Press L, ed. *Applied Concepts in Vision Therapy*. St. Louis: Mosby; 1997:154-166.

SECTION 2: Evaluation and Assessment

E. Examination of Afferent Pathways

Chrystyna Rakoczy, O.D., and Candice Elam, O.D.

Measurements of afferent visual function determine how well a patient sees and assesses the visual pathway from the eye to the visual cortex.[1] Visual function is assessed using the basic psychophysical tests of visual acuity, contrast sensitivity, color vision, and visual fields.
Below are short descriptions of methods for assessing the afferent pathways. The purpose of each test is described.

Information on general eye exam procedures can be found on the AOA website under Optometric Clinical Practice Guidelines: "Comprehensive Adult Eye and Vision Examination" http://www.aoa.org/documents/CPG-1.pdf. In this resource manual, we point out clinical pearls and advanced evaluations that apply to patients with TBI.

1. Acuity

Visual acuity testing, a measurement of an individual's capacity for visual discrimination of fine details of high contrast, [2] has four main limitations: impracticality (requires well-illuminated chart at specified viewing distance for accuracy of measurement), subjectivity (requires patient cooperation), insensitivity (normal visual acuity does not rule out retino-cortical disease that may damage only low-contrast or non-fixational vision), and lack of specificity (subnormal vision does not differentiate between lesions of the optical or retino-cortical components). [5]
A patient with TBI may not be able to participate in some measures of acuity. One needs to consider the cognitive level and any visual-perceptual deficits (such as anomia) the patient may have. The doctor may have to ask the patient to draw or write a letter if he/she seems to see well yet is not calling out the letters correctly. If the patient is non-verbal, ask the patient to simply match a seen distance letter or symbol to a near card with appropriate choices.
Below is a brief description of measures of acuity that can be used with patients who have sustained a TBI.

a) Snellen

The Snellen distance chart displays high-contrast optotypes in lines of diminishing size, with each line defined according to the distance at which letters are read by a person with normal acuity [5]. Disadvantages of the Snellen chart include nonlinear variation in the sizes of letters from line to line and differences in numbers of letters on each line.[2]
While a patient is reading letters off of a Snellen chart, it is important to observe the patient's eyes. Look for loss of fixation and intrusions. If the patient is having difficulty with lines of letters, consider presenting one line at a time or even a single letter at a time. Check acuity at distance and near. Near acuity should be taken at the habitual position (inferior gaze) and then repeated at near in primary gaze (straight ahead). The doctor may also want to use a penlight to highlight reading material at near. Be aware of field loss if the patient is missing the first or last letter consistently. If there is an issue of aphasia, consider giving the patient a choice of two letters to compare with the one presented. For example, present the letter "A" and ask the patient, "Is that an 'A' or a 'T'?"

b) LEA

In individuals unable to read letters, distance visual acuity can be tested with Lea figures.[2] The patient is asked to identify a presented figure in the distance verbally or by pointing to a matching symbol held in his or her lap.[2, 5] Testing can be performed using a folding line chart, light box, crowded symbol book, single symbol test book, or flash cards.

c) Broken Wheel Acuity

The Broken Wheel Acuity Test uses a clinical approach of testing for visual acuity by incorporating the Landolt C presented in a forced choice response. Verbal or non-verbal responses may be used. The simple recognition of the gap in a Landolt Ring is the critical feature to determine the visual acuity at 10 feet.[6]

d) HOTV

In individuals unable to read letters, HOTV letters can also be used to determine acuity. The patient is asked to identify or match the presented letter on the chart with a hand-held card displaying the HOTV letters.[2, 3] Single letters are presented with surround bars allowing a result that closely approximates line letter acuity.[4]

e) Teller

Quantification of visual acuity in individuals who are non-verbal or non-responsive may be performed using preferential looking tests.[1] This type of test is based on the fact that the eyes will be more attracted to a grating pattern of alternating dark and white stripes than to a featureless target.[4] The frequency of the smallest pattern the patient seems to prefer is termed the grating acuity and can be converted to Snellen equivalents.[2] Preferential looking has limited practical value because it requires special equipment, skill of administration, cooperation from the patient, and time to allow enough responses.[5]

f) ETDRS

The Early Treatment for Diabetic Retinopathy Study (ETDRS) chart has become the gold standard for consistency in acuity measurement.[2] Each line of the chart contains five letters; the spacing between the letters and lines is proportional to the letter sizes; the sizes of the letters decrease geometrically; and the ability to recognize each letter is approximately the same.[2, 3] This makes the change in visual acuity from one line to another occur in equal logarithmic steps.[3]

References:

1. Getz LM. et al. Inter-observer Reliability of the Teller Acuity Card Procedure in Pediatric Patients. Investigative Ophthalmology & Visual Science, January 1996; 37(1): 180-7.
2. Liu GT. et al. Neuro-Ophthalmology: Diagnosis and Management. Second Edition. Saunders Elsevier, 2010: 7-11.
3. Miller NR. et al. Clinical Neuro-Ophthalmology: The Essentials. Second Edition. Philadelphia: Lippincott Williams & Wilkins, 2008: 2-8.
4. Repka MX. Use of Lea symbols in young children. *Br J Ophthalmol.* 2002; 86: 489-90.
5. Trobe JD. The Neurology of Vision. Oxford: Oxford University Press, Inc, 2001: 97-101.
6. Richman JE, Petito GT, Cron MT. Broken wheel acuity test: a new and valid test for preschool and exceptional children. *J Am Optom Assoc*. 1984 Aug; 55(8):561-5.

2. Contrast Sensitivity/Low-Contrast Acuity

Contrast sensitivity tests measure discrimination for stimuli of varying spatial frequency and less than 100 percent contrast.[1] These types of tests can uncover deficits in patients who have visual pathway lesions and normal Snellen acuity.[1] Optic neuropathies, media opacities, and macular disease may reduce contrast sensitivity.[2]

a) Pelli-Robson

Contrast sensitivity can be measured using the Pelli-Robson Chart. This chart uses letters of a single spatial frequency but declining contrast.[4] Each line of the chart consists of six letters with the three left-most and three right-most having the same amount of contrast.[3] This method of testing contrast sensitivity is highly reproducible.[3] The Pelli-Robson is considered the standard contrast sensitivity chart. The smaller MARS chart may be more convenient and has been shown to be comparable to the Pelli-Robson.[5]

b) Sloan

Low-contrast Sloan letter acuity testing captures the minimum size at which individuals can perceive letters of a particular contrast level (shade of grey on white background).[2] Sloan charts present gray letters in ETDRS format. This testing evaluates other aspects of visual dysfunction beyond high-contrast visual acuity loss in multiple sclerosis and other neurologic disorders.[2]

References:

1. Burde RM, Savino PJ, Trobe JD. *Clinical Decisions in Neuro-Ophthalmology*. 3rd ed. St. Louis, MO: Mosby; 2002: 8.
2. Liu, GT. Volpe NJ, Galetta SL. *Neuro-Ophthalmology: Diagnosis and Management*. 2nd ed. Saunders Elsevier, 2010: 10-13.
3. Miller NR, Newman NJ, Biousse V, Kerrison JB, eds. *Walsh and Hoyt's Clinical Neuro-Ophthalmology: The Essentials*. 2nd ed. Philadelphia, PA: Wolters Kluwer; 2008 9-12.
4. Trobe JD. *The Neurology of Vision*. New York NY: Oxford University Press; 2001: 102-4.
5. Dougherty BE, Flom RE, Bullimore MA. An Evaluation of the Mars Letter Contrast Sensitivity Test. *Optom Vis Sci* 2005; 82:970-975.

3. Color Vision

Color vision tests can not only identify congenital color vision disturbances, but also may have neuro-diagnostic value [1]. Acquired color vision loss typically is not the same for each eye and may change over time.[3] There are two testing methods for assessing color vision: color confusion tests (Ishihara, Hardy-Rand-Ritter) and color discrimination tests (Farnsworth-Munsell).[1]

a) Ishihara

Ishihara pseudo-isochromatic color plates consist of numbers the patient is asked to identify among different colored dots.[2] The plates were originally designed to screen for congenital dyschromatopsia and may be insensitive to acquired color vision disorders.[1, 2] False positive results may occur if patients have a visual spatial disorder.[1]

b) Hardy-Rand-Ritter

Hardy-Rand-Ritter pseudo-isochromatic plates contain geometric shapes the patient is asked to identify among different colored dots.[1, 2] These plates contain blue and purple figures that screen for tritan defects and may be more helpful in detecting acquired dyschromatopsia due to dominant optic neuropathy.[2]

c) Farnsworth

The Farnsworth D-15 and Farnsworth-Munsell tests can be used to separate the various congenital dyschromatopsias and are more quantitative and sensitive to acquired color vision deficits than color confusion tests.[1, 2] In both tests, patients are asked to arrange colored caps in linear sequence relative to reference caps.[1, 2]

References:

1. Trobe JD. *The Neurology of Vision*. New York NY: Oxford University Press; 2001; 105-6.

2. Liu, GT. Volpe NJ, Galetta SL. *Neuro-Ophthalmology: Diagnosis and Management*. 2nd ed. Saunders Elsevier, 2010: 10-13.
3. Miller NR, Newman NJ, Biousse V, Kerrison JB, eds. *Walsh and Hoyt's Clinical Neuro-Ophthalmology: The Essentials*. 2nd ed. Philadelphia, PA: Wolters Kluwer; 2008; 33-35

4. Amsler Grid

In addition to macular lesions, the Amsler grid can detect primary cortex lesions confined to the central 10 degrees of the visual field when held at about 28 cm where each square will subtend 1 degree of visual angle.[1] The red-on-black Amsler grid is used because of its higher sensitivity for optic nerve disease central/para-central defects.[2, 3]

If the patient has a central scotoma and cannot see the central red dot with primary fixation, place the patient's finger on the dot for reference or use the Amsler grid with the X and tell the patent to look where he or she believes the lines would intersect.

Patients who respond with metamorphopsia should be evaluated for retinal disease. Those who respond with missing corners or blocks should be evaluated for post-retinal afferent pathway disease.

References:

1. Amsler M. Earliest symptoms of diseases of the macula. *Br J Ophthalmol* 1953; 37:521-537
2. Burde RM, Savino PJ, Trobe JD. *Clinical Decisions in Neuro-Ophthalmology*. 3rd ed. St. Louis, MO: Mosby; 2002; 13.
3. Miller NR, Newman NJ, Biousse V, Kerrison JB, eds. *Walsh and Hoyt's Clinical Neuro-Ophthalmology: The Essentials*. 2nd ed. Philadelphia, PA: Wolters Kluwer; 2008; 1-17

5. Confrontation Visual Fields

The disadvantages of confrontation visual field testing include non-standardization, difficulty in detecting subtle defects, and difficulty in monitoring for progression. The advantages include ease of administration anywhere on most patients. Below are detailed descriptions of confrontation tests that are considered high yield for the TBI population. You can view the confrontation fields performed on the YouTube video referenced below.

a) Kinetic Testing:

This method is used for testing peripheral constriction.

Procedure/Instructions:

Sit at arms' length (about 2.5 to 3 feet) in front of the patient. Have the patient cover one eye and fixate with the other eye on your opposing eye. This allows you to compare the patient's visual field to your own. Present two or three moving fingers in the outskirts of the patient's visual field and move toward the center, stopping once the patient reports seeing your fingers move. Repeat in all meridians. This method can also be done with a second person presenting fingers from the far side of (or from behind) the patient's peripheral field.

b) Static Testing:

This method is best for quadrant, hemianopic and altitudinal defects.

Procedure/Instructions:

Sit at arms' length in front of the patient. Have the patient cover one eye and fixate with the other eye on your opposing eye, allowing you to compare the patient's visual field to your own.

- Quadrant/single stimulus: Present one to three stationary fingers in one quadrant at a time about half way between you and the patient and about 30 degrees (shoulder width) off center. Ask the patient how many fingers he or she sees. Patients who are non-verbal can mimic the number of fingers. If the patient keeps

missing in one quadrant or hemifield, switch to kinetic testing moving from non-seeing to seeing across the horizontal and vertical meridians to determine the boarders of the defect.

- Quadrant /double stimulus: present stimuli in two vertically or horizontally opposing quadrants simultaneously.
- Quadrant brightness comparison: with stimuli in opposing quadrants ask the patient if one target is brighter than the other.
- Quadrant color comparison: present red targets in two opposing quadrants and ask the patient if one is a brighter red than the other. Move the duller target into the field of the brighter target and ask if there is a color change.[1,2]

(1) Static Central/Face Identification:

This method can be used for central field testing and is often combined with the red cap test.

Procedure/Instructions:

Your face will be the target. Be mindful that illumination of your face is equal. Sit at arms' length in front of the patient. Have the patient cover one eye and fixate with the other eye on your nose. Ask if any part of your face is missing. Observe that the patient is fixating on your nose. As the patient fixates on your nose, ask if both sides of your face appear equally clear and bright, and if the top and bottom half appear to be equally clear and bright. In a variation for greater sensitivity, present two red caps on either side of your nose and ask if they are equally clear and bright. Repeat with the red caps above and below your nose. One can also hold one red cap on the nose and present the other cap off-center for comparison. If the patient sees the off-center cap brighter than the central cap, this suggests a central scotoma. Desaturation is significant for optic nerve disease.[1,3,4] Note that red caps can vary slightly in intensity, so chose your caps minding their hue and tone.

Sensitivity of confrontation field testing:

Only 10 percent of optic-nerve-related field defects can be detected by finger counting. Using red caps increases the sensitivity to 75 percent for chiasmal defects. Combining confrontation techniques only picks up about 50 percent of defects found by formal perimeter.[1]

References:

1. Miller NR, Newman NJ, Biousse V, Kerrison JB, eds. *Walsh and Hoyt's Clinical Neuro-Ophthalmology: The Essentials*. 2nd ed. Philadelphia, PA: Wolters Kluwer; 2008: 15-16
2. Burde RM, Savino PJ, Trobe JD. *Clinical Decisions in Neuro-Ophthalmology*. 3rd ed. St. Louis, MO: Mosby; 2002: 23-24
3. Liu, GT. Volpe NJ, Galetta SL. *Neuro-Ophthalmology: Diagnosis and Management*. 2nd ed. Saunders Elsevier, 2010: 10-13:14-16
4. Trobe JD. *The Neurology of Vision*. New York NY: Oxford University Press; 2001: 110-13
5. Visual Fields to Confrontation. http://www.youtube.com/watch?v=2-9FVywV2j4 . Accessed Nov. 8, 2013.

(2) Red Cap Desaturation

A comparison of the saturation of colors, between the two eyes, and across the vertical midline can sometimes identify subtle optic nerve dysfunction or chiasmal lesions.[1] A patient with a monocular "red desaturation" may state that with the affected eye the red bottle top appears "washed out," "pink," "orange," "gray" or "brown."[2] When the color is faded, "orange," or "pink," there is a relative reduction in saturation. The color appears to contain less of its spectral hue and more white; however, a response of "gray" or" brown" indicates loss of brightness.[3]

Materials:

-Red stimulus (red cap from dilating drops)

-Occluder

Procedure/Instructions:

Patient is seated wearing appropriate near correction.

A red stimulus is presented to each eye sequentially and the patient reports whether the red color looks "richer" to one eye than the other.[3]
A patient who does notice a difference is asked to describe the difference quantitatively. You may want ask: "If the deep red cap is worth one dollar of redness, how much is the lighter red cap worth?"
Recording:
Record your response as a percent of desaturation. For example, if the patient responds that the lighter cap is worth 80 cents, record "20 percent desaturation" for that eye.

References:

1. Miller NR, Newman NJ, Biousse V, Kerrison JB, eds. *Walsh and Hoyt's Clinical Neuro-Ophthalmology: The Essentials*. 2nd ed. Philadelphia, PA: Wolters Kluwer; 2008: 3.
2. Liu, GT. Volpe NJ, Galetta SL. *Neuro-Ophthalmology: Diagnosis and Management*. 2nd ed. Saunders Elsevier, 2010: 11-14.
3. Burde RM, Savino PJ, Trobe JD. *Clinical Decisions in Neuro-Ophthalmology.* 3rd ed. St. Louis, MO: Mosby; 2002: 7-8.

6. Photo Stress Test:

Purpose:
This test distinguishes between retinal and optic nerve disease. Regeneration of retinal pigments from bleaching is not dependent on neural actions.[1] The disadvantage of this test is its subjective endpoint that may be questionable in patients with cognitive difficulties. This test should be performed last if the patient is very sensitive to light.
Instructions and procedure:
Obtain the best-corrected monocular visual acuity. Have the patient cover one eye. Shine a transilluminator 2 to 3cm from the uncovered eye for 10 seconds. Once the light is removed, note the number of seconds it takes for the patient to read three letters on the line preceding best-corrected acuity (three letters on the 20/30 line with 20/25 best corrected acuity). The normal recovery time is 27 seconds, but can vary with age and technique. Nearly 99 percent of normal eyes recover within 50 seconds.[1] Relative time between the eyes may be a better measure. Optic nerve disease has no delay. Macular disease can take several minutes to recover.[1-4]

References:

1. Miller NR, Newman NJ, Biousse V, Kerrison JB, eds. *Walsh and Hoyt's Clinical Neuro-Ophthalmology: The Essentials*. 2nd ed. Philadelphia, PA: Wolters Kluwer; 2008:4-5
2. Sadun AA. Distinguishing between clinical impairments due to optic nerve or macula disease. *Metab Pediatr Syst Ophthalmol*. 1990; 13:79-84.
3. Glaser JS, Savino PJ, Sumers KD, et al, The photostress receiver test in the clinical assessment of visual function. *Am J Ophthalmol*. 1977; 83:255-260.
4. Liu, GT. Volpe NJ, Galetta SL. *Neuro-Ophthalmology: Diagnosis and Management*. 2nd ed. Saunders Elsevier; 2010: 16

7. Pupil Testing and the Near Reflex

In addition to testing pupils for size, reactivity, and the presence of a relative afferent pupillary defect, it is very important to test the pupillary near response in a patient with TBI to detect the presence of light-near dissociation,[1] a common problem in this population. Conditions where the pupil reacts to near but not to light include Adie's tonic pupil, Argyll Robertson pupils, and tectal (dorsal midbrain syndrome) pupils[1, 2]. Watch for hippus in patients under stress.

References:

1. Savino PJ, Danish-Meyer H. *Neuro-Ophthalmology.* New York, NY: McGraw-Hill; 2003:216-222.
2. Trobe JD. *The Neurology of Vision*. New York NY: Oxford University Press; 2001:207-213.
3. **N**eurologic exam cranial nerves Normal: vergence http://www.youtube.com/watch?v=TFcCHsY1OUo. Accessed Nov. 8, 2013

SECTION 2: Evaluation and Assessment

F. Examination of Efferent Pathways

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Information on basic evaluation of efferent pathways can be found on the AOA website under Optometric Clinical Practice Guidelines: "Care of the Patient with Accommodative and Vergence Dysfunction" http://www.aoa.org/documents/CPG-18.pdf and "Care of the Patient with Strabismus: Esotropia and Exotropia" http://www.aoa.org/documents/CPG-12.pdf. In this resource manual, we point out clinical pearls and advanced evaluations that apply to patients with TBI.

1. Lids

Below are listed several procedures for the evaluation of various aspects of lid position and function. At first the evaluation may seem taxing, but once you know what to look for these observations do not take much time.

a) Position

The position of the eyelids should be assessed to rule out ptosis, entropion/ectropion, eyelid retraction, and apraxia of eyelid opening.[1-3] Comparison of lids with pre-morbid photos may be of value.

Materials:

-Ruler

-Transilluminator

Procedure/instructions:

-Palpebral fissure height: with the patient looking in primary gaze, measure the distance between the upper and lower lid margins at the center of the pupil.

-Margin-to-reflex distance (MRD):

- MRD1: with the patient looking in primary gaze, measure the position of the upper lid with respect to the corneal light reflex.
- MRD2: with the patient looking in primary gaze, measure the position of the lower lid with respect to the corneal light reflex

-Position of the eyelid crease: with the patient looking in primary gaze, measure the distance from the lash margin to the eyelid crease.

Recording:

Record the MRD of the upper and lower eyelids, palpebral fissure height and position of the eyelid crease in millimeters.

Expected values:[1]

Normal MRD1 and MRD2 are 4 to 5 mm

Palpebral fissure height normal range is 9 to 12 mm[1]

Eyelid crease typically measures 10 mm; it will increase with levator dehiscence.[1]

b) Function

Evaluation of voluntary and reflex lid movement will describe their function to protect the globe, help distribute the tear film over the ocular surface, collect tears and propel them to the medial canthus, and protect the cornea from injury and glare.[1]

Materials:

-Ruler

Procedure/instructions:

-Levator muscle function: manually fix the eyebrow to the supraorbital rim to prevent frontalis muscle contribution to eyelid movement, then measure the amount of excursion of the eyelid margin as the patient looks from downgaze to upgaze.[2, 3]

-Examine the lids during eye movements. On horizontal conjugate gaze, half of normal patients will exhibit palpebral fissure widening of the abducting eye; 15 percent will display lid elevation of the adducting eye.[1] Causes for lid lag seen on downgaze may include dorsal midbrain lesion, thyroid eye disease or aberrant regeneration of cranial nerve III.[1]

Recording:

Record levator muscle function in millimeters.

Expected values:

Normal function will measure greater than 12 mm.[1]

2. CN VII Evaluation

a) Observation at Rest/Asymmetry

Procedure/instructions:

Observe the patient at rest and note asymmetry of the face or blink pattern. The paretic side in facial nerve palsy will show a flattened nasolabial fold and widened palpebral fissure.[1]

b) Motor Function

(1) Facial Expression

Procedure/instructions:

-Ask patients to lift their eyebrows and close their eyelids while observing facial movement. Patients with supranuclear lesions preserve the ability to wrinkle their forehead and raise their eyebrow.[1, 4]

-Ask the patient to smile to assess the function of the lower part of CN VII.

(2) Orbicularis Function

Procedure/instructions:

Instruct patients to forcefully close their eyelids while you attempt to open them.[3] Note any asymmetry in the patient's ability to close the lids (lagophthalmos).[1, 4]

3. CN V Evaluation

a) Corneal Blink Reflex

Procedure/instructions:

With the patient viewing in primary gaze, touch the cornea with a cotton wisp or tissue corner.

Recording:
Look for the bilateral blink. Note absence of blinking in either eye, which indicates a lesion of the ophthalmic division of CN V (afferent limb) or asymmetric blinking, which suggests CN VII (efferent) weakness on one side.[1]

b) Naso-lacrimal Reflex

Sensory innervation to the lacrimal gland occurs via the ophthalmic division of the trigeminal nerve.[1] Tear secretion may be altered by supranuclear lesions or lesions along the pathway from the brainstem to the lacrimal gland.[3]
Procedure/instructions:
Measure reflex tearing by instilling topical anesthetic into both eyes, placing Schirmer test strips into the inferior conjunctival sacs of each eye, and stimulating the nasal mucosa using a cotton-tipped applicator soaked in benzene.[1,3]
Recording:
Measure the length of moistened paper after five minutes and record the amount in millimeters.[1]
Expected values:
Normal results are 10 to 30 mm.

References:

1. Liu GT, Volpe NJ, Galetta SL. *Neuro-Ophthalmology: Diagnosis and Management*. 2nd ed. Saunders Elsevier; 2010
2. Howard G. Eye lid retraction. In: Yanoff M, Duker JS, eds. *Ophthalmology.* St. Louis, MO: Mosby; 2004
3. Miller NR, Newman NJ, Biousse V, Kerrison JB, eds. *Walsh and Hoyt's Clinical Neuro-Ophthalmology: The Essentials*. 2nd ed. Philadelphia, PA: Wolters Kluwer; 2008:22.
4. Cranial nerve VII –motor evaluation. http://www.youtube.com/watch?v=W55vFhFWu9g. Accessed Nov. 8, 2013.

4. Ocular Stability, Position, Binocular Alignment, and Motility: CN III, IV and VI Evaluation

The evaluation of ocular stability, position alignment and motility is an evaluation of the cranial nerves III, IV and VI. Though not specific for TBI and maybe a bit humorous, the following video has a very nice review of anatomy and functional assessment of these nerves: http://www.youtube.com/watch?v=FKrCh6BnTR4. Accessed Nov 8, 2013

a) Fixation Stability

(1) Fixation

Purpose:
This test helps to evaluate the ability of the patient to maintain steady fixation on an object. There are several methods for testing:

(a) *Scheiman and Wick*

Materials:
-Fixation target
Procedure:
Ask the patient to fixate on a target during the initial external evaluation or during cover testing in order to evaluate fixation status. Disorders of fixation can be from organic or functional problems.
Instructions:

- Instruct patient to look at a target.

Examiner should be looking for any fixation loss in the 10-second interval.

Recording:

Record whether or not fixation stability was noted OD, OS.

Expected values:

All patients should be able to sustain fixation for 10 seconds. Some exceptions to this are very young, anxious, hyperactive or inattentive patients.

References:

1. Scheiman M, Wick B. *Clinical Management of Binocular Vision: Heterophoric, Accommodative and Eye Movement Disorders.* 3rd ed. Philadelphia, PA: : Lippincott Williams and Wilkins; 2008:58

(b) *Southern California College of Optometry 4+ System*[1]

Materials:

-20/80 target

Procedure:

Present a 20/80 target at 40 cm, monocularly and then binocularly for 10 seconds. Observe and record the quality of steadiness.

Instructions:

-Look at the target.

Recording:

>10 sec =5

10 sec =4+

5 sec=3+

< 5 sec or with proprioceptive support =2+

Almost continuous unsteady fixation=1+

Expected value:

>10 sec =5

References:

1. Griffin JR, Grisham JD. *Binocular Anomalies Diagnosis and Vision Therapy*. 4th ed. New York, NY: Butterworth Heinemann; 2002:38-39

(2) Nystagmus

Purpose:

Assessment of fixation in presence of nystagmus: Patients with TBI have many vestibular issues that can lead to nystagmus. In the evaluation of nystagmus it is important to observe the following:[1]

- posture (head tilt)
- direction of oscillations
- type (pendicular, rotary, jerk)
- amplitude (measured in mm or degrees)
- symmetry between the eyes
- congruency (whether the eyes move in the same direction or opposite)
- occlusion (for latent nystagmus)
- amplitude given various fixation distances (to appreciate visual demand and check for a null point with convergence)

- constancy

For a more in-depth discussion of nystagmus evaluation in the presence of other oculomotor dysfunctions as described below, please refer to the section on Advanced Topics in Volume Ib.

- Assessment of Nystagmus Using Saccadic Testing Measures
- Assessment of Nystagmus Using Pursuit and OKN Testing Measures
- Assessment of Nystagmus Using VOR Testing Measures
- Evaluating Fixation in Acquired Nystagmus

References:
1. Scheiman M, Wick B. *Clinical Management of Binocular Vision: Heterophoric, Accommodative and Eye Movement Disorders.* 3rd ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2008:498
2. Gaze stability and spontaneous nystagmus. http://www.youtube.com/watch?v=D-wP29938as. Accessed Nov. 8, 2013

b) Position/Alignment

(1) Primary Gaze Evaluation

(a) *Observation/Hirschberg/Krimsky Reflex Test*[1,2]

Purpose:

To determine the relative position of the globes; look for deviations

This procedure is used on patients who are less than cooperative. Sit or stand in front of the patient. Place the appropriate prism in front of the deviating eye (BO for eso deviations, BI for exo deviations, BD for hyper, BU for hypo) and compare corneal reflexes of both eyes to determine their relative position. Every mm of deviation is equal to 7 degrees or 15 prism diopters. Increase or decrease the amount of prism until corneal reflexes are equally centered.

References:
1. Prism and cover test: http://www.youtube.com/watch?v=eNpoGOANULI. Accessed Nov. 8, 2013
2. Ophthalmology Lectures: Tropia vs. phorias: http://www.youtube.com/watch?v=FKrCh6BnTR4. Accessed Nov. 8, 2013

(b) *Unilateral Cover Test (Cover/Uncover)*

Purpose:

To establish the presence of heterotropia and to determine the characteristics of the ocular deviation, such as direction, magnitude, laterality, frequency[1]. This test should be performed early on in the exam because many of the procedures throughout the exam will disrupt normal binocular vision. Be sure the unilateral cover test is done prior to the alternating cover test, as you want to obtain the patient's habitual binocular status before you start breaking down binocularity.[1] It is also important to perform this test at distance before near because you do not want to challenge the patient's accommodative system, therefore affecting the distance results. Allow the eyes to relax to their natural position of rest with prolonged occlusion: do not uncover the eyes too quickly. Observe each eye independently for jump. Frequency of tropia is determined by repeating the test a few times. The tropia can be persistent, intermittent and/or alternating. You can repeat this test at the end of the exam to check for decompensation. Responses can be variable.[2]

(c) *Alternating Cover Test: (Distance)*[1-4]

Purpose:

The magnitude of the deviation (tropia or phoria) is determined by this test with use of prism. Because the eyes are continuously dissociated during this procedure, a more accurate assessment of the magnitude of the deviation is obtained because both the tropia and phoria deviations are measured. Be aware that the magnitude can be variable. Allow for adequate occlusion. After the unilateral cover test, if no tropia was observed, the ocular deviation measured in this procedure will be only for phoria. Watch the eye for versional movement, as the occluder is removed. This test is performed the standard way with the exception of giving the patient time to respond.

Unilateral Cover Test at Near and Alternating Cover Test at Near:

These tests are performed at near similar to the way they are performed at distance. The test is routinely done at 16" (40 cms) wearing the habitual near Rx (which may be just distance Rx or with aided Rx, as in the case of a presbyopic patient). The doctor can have the target in the midline or may vary posture of testing and repeat cover test in down gaze—emulating reading posture. The target should be interesting and a good stimulator of accommodation. Remember to always perform the alternate cover test after the unilateral test. Watch for fatigue.

NOTE: If a patient's prescription after refraction for distance and near has changed, these tests should be repeated again with the new distance and near prescriptions.

References:

1. Grunning CF, Hong C, Wong LC. *Vision therapy Diagnostic Procedures Laboratory*. Clinical manual for State University of New York, College of Optometry. 1993.
2. Rosenfield M. *Primary Care Procedures Manual*. Clinical manual for State University of New York, College of Optometry. 1993:107-113
3. Van Rijn U, Tusscher MP, de Jong I, Hendrikse F. Asymmetric vertical phorias indicating dissociated vertical deviation in subject with normal binocular vision. *Vision Res*. 1998;38:2937-8
4. Scheiman M, Wick B. *Clinical Management of Binocular Vision: Heterophoric, Accommodative and Eye Movement Disorders.* 3rd ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2008:58
5. Ophthalmology lecture- tropias and phorias. http://www.youtube.com/watch?v=TxEQWtlXtrI. Accessed Nov. 8, 2013.

(2) Secondary Gaze Evaluation/Ductions/Versions

(a) *Ductions/Versions*

Purpose:

To determine full excursion and rotation of the globes. The globe will rotate around three axes: horizontal axis (elevation and depression), vertical axis (adduction and abduction) and anterior-posterior axis (excycloduction—top of cornea rotates temporally, and incycloduction—top of cornea rotates nasally). Rotation around a combination of axes allows for oblique excursion. This test also allows the doctor to determine full primary action of each ocular muscle and note their restrictions. Ductions are monocular rotations; versions are binocular.

Clinical pearl: On lateral gaze the scleras should disappear. On upgaze, one-third of the cornea should disappear behind the upper lid, and on downgaze, one half of the cornea should disappear behind the lower lid.[1, 2] The excursions can be documented in percent of excursion.[3]

References:

1. Burde RM, Savino PJ, Trobe JD. *Clinical Decisions in Neuro-Ophthalmology.* 3rd ed. St. Louis, MO: Mosby; 2002:203
2. Von Noorden GK, Campos EC. *Binocular Vision and Ocular Motility Theory and Management of Strabismus.* St. Louis, MO: Mosby; 2002:199
3. Ophthalmology lecture- tropias & phorias. http://www.youtube.com/watch?v=dRYBOBSyzAU. Accessed Nov. 8, 2013

(b) *Comitance/Non-comitance*

Purpose:

After completing cover and EOM testing (binocular versions and monocular ductions), any detected tropia, restrictions, and/or diplopia noted indicate the need to determine if the strabismus is acquired or congenital. Testing

for comitancy in many instances will help decipher if the strabismus is due to paralytic or paretic muscles secondary to an acquired pathology or from non-organic causes such as congenital or decompensated tropias.[1] An easy way to test these motor fields is by presenting a light in all nine cardinal positions of gaze to a patient wearing anaglyph glasses (red/green). Record what the patient sees in each position of gaze. The position of the red and green lights relative to each other will be stable if the deviation is comitant.[2, 3]

Congenital strabismus non-related to acquired brain injury will show the same angle of deviation in all positions of gaze. Usually, there is no muscle deficit with congenital esotropia, accommodative esotropias, or decompensated tropias. These non-pathological ocular deviations are said to be comitant. Comitancy is achieved when the angle of deviation remains the same throughout all positions of gaze and with either eye fixating [1].

In contrast, non-comitant strabismus is defined as an angle of deviation that varies (>5 prism diopters) in different fields of gaze with either eye fixating.[1] Typically, the deviation is even greater if the affected eye is fixating (secondary angle of deviation, paretic eye fixating[4]). When there is stroke, trauma, or any neurological insult, the cranial nerves controlling the extraocular muscles can be affected. At onset, one of the first symptoms is diplopia. The patient may adapt to this with a change in head posture, such as a tilt or turn. Over time, the muscles unaffected by neurological damage will compensate and remain in retracted positions. Note that measuring motor fields is more reliable for detecting comitance and non-comitance with recent onset strabismus, before the eye muscles readjust themselves.[1]

The doctor should observe the patient by noting head tilts and turns and pupil or lid abnormalities. Such clinical signs may indicate whether the strabismus is acquired or recent in onset and of emergent neurological concern.[1]

References:

1. Grunning CF, Hong C, Wong LC, Vision therapy Diagnostic Procedures Laboratory. Published by State University of New York , College of Optometry. 1993.
2. Benjamin WJ. *Borish's Clinical Refraction.* 2nd ed. New York NY: Butterworth Heinemann; 2007:380-381
3. Von Noorden GK, Campos EC. *Binocular Vision and Ocular Motility Theory and Management of Strabismus.* 6th ed. St. Louis, MO: Mosby; 2002:182-184
4. Griffin JR, Grisham JD. *Binocular Anomalies Diagnosis and Vision Therapy*. 4thed. New York NY: Butterworth Heinemann; 2002:112-113

(c) *Maddox Rod/Red Lens*

Purpose:

Use of a penlight with a red lens or Maddox rod over one eye in conjunction with the test for comitancy subjectively validates what was seen on version testing. Uncrossed and crossed diplopia can be easily described by the patient by noting the relative positions of the white light and red light or red line.[1] Note: the red lens is most effective if the strabismus is constant, while the Maddox rod is most effective with intermittent strabismus or if the patient is heterophoric.[2]

References:

1. Von Noorden GK, Campos EC. *Binocular Vision and Ocular Motility Theory and Management of Strabismus.* St. Louis, MO: Mosby; 2002:187-189
2. Grunning CF, Hong C, Wong LC. *Vision therapy Diagnostic Procedures Laboratory*. Published by State University of New York College of Optometry. 1993.
3. Cranial nerve palsies. http://www.youtube.com/watch?v=FKrCh6BnTR4. Accessed Nov. 8, 2013

(d) *Parks 3-Step Method (Head Tilt Test)* [1-3]

Purpose:

This test isolates paretic cyclovertical muscles for a non-comitant vertical deviation. The most common cranial nerve injury seen in TBI is CN IV. The doctor may note a slight head tilt away from the injured cranial nerve. Be sure to straighten the head before starting any measurements. Tell patients they may feel as if their head is not

straight. This test is used only to rule in or rule out CN IV palsy. At times a hyper deviation is noted in steps one and two but not in step three. Maddox rod or anaglyph glasses can help patients determine the relative distances of the perceived targets.

References:
1. Grunning CF, Hong C, Wong LC. *Vision Therapy Diagnostic Procedures Laboratory*. Clinical Manual for State University of New York, College of Optometry. 1993.
2. Scheiman M, Wick B. *Clinical Management of Binocular Vision: Heterophoric, Accommodative and Eye Movement Disorders.* 3rd ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2008:407-408.
3. Von Noorden GK, Campos EC. *Binocular Vision and Ocular Motility Theory and Management of Strabismus.* St. Louis, MO: Mosby; 2002:198-99.
4. Cranial nerve palsies. http://www.youtube.com/watch?v=FKrCh6BnTR4. Accessed Nov. 8, 2013.

(e) *Double Maddox Rod for Torsional Misalignment*[1, 2]

Purpose:

The purpose of this test is to isolate the paretic muscle to aid in the differential diagnosis of vertical misalignments (CN IV palsy vs. skew) when comitance has spread over time and in the determination of torsional misalignments in the absence of a vertical misalignment. Any torsional measure exceeding 10 degrees is indicative of bilateral CN IV involvement. A hyper eye that excyclotorts is indicative of an affected CN IV. A hypo eye that excyclotorts is usually indicative of a skew deviation. Note: often cyclotorsion is the cause of non-fusion. Severe cyclotorsion can only be corrected by surgical procedure.

References:
1. Burde RM, Savino PJ, Trobe JD. *Clinical Decisions in Neuro-Ophthalmology.* 3rd ed. St. Louis, MO: Mosby; 2002:190.
2. Scheiman M, Wick B. *Clinical Management of Binocular Vision: Heterophoric, Accommodative and Eye Movement Disorders.* 3rd ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2008

c) *Motility*

Ocular motor deficits are common to the TBI population.[1] Saccades, pursuit and vergence eye movements have distinct neurological pathways; therefore, it is possible to affect one pathway while the other remains intact.[2] However, in many patients who sustained TBI, saccadic, pursuit and vergence dysfunctions are associated with accommodative, binocular, and visual-perceptual dysfunctions.[2]

Examination of oculomotor function requires evaluation of fixation stability and ocular motility (saccades, pursuits and vergence). Oculomotor dysfunction can indicate a neurological problem that may warrant a neurological consultation, so the doctor should refer in cases of suspicion [2]. Evaluation of ocular motility is routinely done through direct observation by the doctor and through standardized testing such as the Developmental Eye Test (DEM)[2].

(1) Saccades

Saccades can be voluntary or involuntary. Saccadic movement is controlled by the frontal lobe, superior colliculi, and the parietal lobe.[1] Additional influences from the occipital lobe are involved in involuntary or stimulus-generated saccades.[1] Chair-side testing of saccades can be done through a standardized test such as the NSUCO (Northeastern State University College of Optometry)/Maples Oculomotor Test method or the DEM[1].

Efficient eye movements are critical for optimal reading function. It is possible for the patient to have normal chair-side saccades but still be symptomatic, and this may correspond to the fact that eye movements made in reading may not correlate to those made during NSUCO testing.[2] Consequently, normal observed eye movements in a patient who is symptomatic should be followed up with a DEM. The DEM provides an objective method of assessing

fixation and saccadic activity during reading and non-reading tests. It is administered individually, free from significant distraction and has two formats: horizontal and vertical.

Both the DEM and NSUCO chair-side saccades have their limitations. With the DEM, it is important to keep in mind factors such as deficits in visual attention function, perceptual skills, integration issues, and verbal skills can influence test results. These issues and ways to account for them are addressed in the manual for "The Developmental Eye Movement Test" © Version 2.0 2009.[4] Objective tests such as the Visagraph or Readalyzer may prove very beneficial with the TBI population.[2] The Visagraph and Readalyzer provide information directly on eye movements as they specifically relate to reading function.[2] They measure the number of fixations, regressions, duration of fixations, reading rate, relative efficiency, and grade equivalence.[2] This technology is expensive, though very useful, and the doctor is able to gather critical information to assess an oculomotor dysfunction with conventional testing [2]. Similarly, chair-side saccade evaluation and assessments rely on an experienced doctor.[2] The NSUCO is dependent on the examiner's observation skills. If the patient passes the test, this does not rule out an oculomotor dysfunction. If the history suggests an eye movement disorder, additional testing, such as the DEM, as well as objective tests with the Visagraph or Readalyzer may prove very beneficial.[2]

An adjunct eye movement test reported to be a tool as a determinant of head trauma and concussion in sports injury is the King-Devick (K-D)[5]. This test uses a visual verbal testing format that is timed and standardized. There are merits of this test as well as the DEM.[6-8]

The easiest way to evaluate voluntary saccades (and with least amount of equipment) is to observe and describe the quality of the saccade: delayed, hypermetric, absent, slow, dysmetric, disinhibited. One method describes observation of the saccade as the patient moves his/her eyes from a visible target displayed in straight-ahead gaze to a target displayed in the near peripheral field. Look for under or overshoot of centripetal and centrifugal saccades that do not extinguish after four to five repetitions.

It is important to observe voluntary and involuntary saccades as well as anti-saccades.[3] Involuntary saccades are observed with the optokinetic drum. The doctor should compare the amplitude and velocity of the quick phases. Note: the patient must have a visual acuity of at least 20/200 to be able to respond to the optokinetic nystagmus drum. Inability to respond to the OKN otherwise indicates pursuit and saccadic dysfunction. Anti-saccades are tested by asking the patient to look from center then away or in the opposite direction of a peripherally displayed target. Inability to perform an anti-saccade can be indicative of frontal lobe and basal ganglion dysfunction.[3]

References:

1. Falk NS, Aksionoff EB. The primary care optometric evaluation of the traumatic brain injury patient. *J Am Optom Assoc* 1992;63:547-53.
2. Scheiman M, Wick B. *Clinical Management of Binocular Vision: Heterophoric, Accommodative and Eye Movement Disorders.* 3rd ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2008:28-31, 48
3. Burde RM, Savino PJ, Trobe JD. *Clinical Decisions in Neuro-Ophthalmology.* 3rd ed. St. Louis, MO: Mosby; 2002:201-202
4. Developmental Eye Movement™ Test (DEM™) http://www.bernell.com/product/1501/417. Accessed Nov. 8, 2013
5. King-Devick Test. http://kingdevicktest.com/. Accessed Nov. 8, 2013
6. Richman MS, Richman JE. Sports concussions raise new awareness of mTBI care. American Optometric Association News; Practice Strategies. Vol. 51; pp 35-38 July 2012.
7. Developmental Eye Movement: http://www.youtube.com/watch?v=WR1YDo2X9Fo. Accessed Nov. 8, 2013
8. Developmental Eye Movement: http://www.youtube.com/watch?v=qoqbWAeXICM/. Accessed Nov. 8, 2013

(2) Pursuits

This test determines if there are any limitations to a patient's eye movements by evaluating his/her ability to accurately follow a target (sensory/motor match). Observe not just the range of motion but, more important, the quality. This test should be performed monocularly and binocularly.

There are several methods for measuring pursuits and all are well-described. NSUCO[1] developed an objective method to measure the quality of the pursuit. Other methods simply employ accurate direct observation: looking for stuttering, ratchet or cog-wheeling movement. The complete inability of a pursuit movement in an adult implies a neurologic dysfunction.[2] Pursuits should always be assessed along with saccades. Note: visual inattention can interfere with the patient's ability to perform pursuits.

References:
1. Scheiman M, Wick B. *Clinical Management of Binocular Vision: Heterophoric, Accommodative and Eye Movement Disorders.* 3rd ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2008:31-32, 48-49
2. Burde RM, Savino PJ, Trobe JD. *Clinical Decisions in Neuro-Ophthalmology.* 3rd ed. St. Louis, MO: Mosby; 2002:203

(3) Vergence: Distance and Near

Vergence testing tells us about the efficiency of the oculomotor system and its ability to compensate for any tendency to ocular misalignment (phoria). It is tested at both distance and near and in the horizontal and vertical planes. For many reasons, testing this population in a phoropter may be difficult. Free space techniques are described below. Note that vergence testing may cause extreme fatigue and may need to be repeated.

(a) *Vertical: Supravergence and Infravergence*

Purpose:
To measure the oculomotor system's ability to overcome a vertical phoria: vertical phoria in normal subjects has been recorded to be ortho to no more than 0.28 prism diopters.[1] This test can be done at distance using a 20/40 target or at near in the reading plane with an accommodative target. Using vertical prism bars in free space allows for more natural posture and also allows the doctor to see how the eyes are moving. Base up or base down can be used as long as the same base direction is used on each eye. Expected values are: 2/1 BD OD, 2/1BD OS or 2/1BU OD, 2/1BU OS[2].

(i) Vertical Amplitude and Balance

Vertical amplitude is calculated by adding the amounts of prism necessary to break fusion on each eye. For example: 5/4 BD OD, 4/3 BD OS 5 + 4=9 prism diopters of vertical amplitude. This number is important in differentiating a congenital CNIV palsy from a traumatic CNIV palsy.[3]

Vertical balance between the eyes is critical in patients with TBI as their neurological systems are compromised and any small misalignment can be difficult to compensate. Each eye should break and recover with the same amount of prism. Often, a slight vertical phoria that is not balanced and not well-compensated will also disrupt a weak horizontal vergence. The patient may exhibit a robust convergence insufficiency leading the doctor to: 1. believe the CI is the only culprit of the patient's inability to read for extended periods of time, and 2. never check the vertical balance. Giving the patient as little as 0.5 diopters of vertical prism to balance the vertical vergence can restore the patient's ability to fuse horizontally without the addition of horizontal prism or CI rehabilitation.

(b) *Horizontal Vergence: Convergence and Divergence.*

(i) Amplitudes

Purpose:
To measure the oculomotor system's ability to overcome a horizontal phoria: this test can be done at distance using a 20/40 target or at near in the reading plane with an accommodative target. Using prism bars in free space allows for more natural posture and also allows the doctor to see how the eyes are moving.
Expected Values:[4] break/recover for adults

Distance: BO: 11±7/7±2

BI: 7±3/4±2

Near: BO: 19±9/14±7

BI: 13±6/10±5

References:

1. Van Rijn U, ten Tusscher MP, de Jong I, Hendrikse F. Asymmetrical vertical phorias indicating dissociated vertical deviation in subjects with normal binocular vision. *Vision Res* 1998;38:2973-8.
2. Von Noorden GK, Campos EC. *Binocular Vision and Ocular Motility Theory and Management of Strabismus.* St. Louis, MO: Mosby; 2002:202-4
3. Burde RM, Savino PJ, Trobe JD. *Clinical Decisions in Neuro-Ophthalmology.* 3rd ed. St. Louis, MO: Mosby; 2002:170
4. Scheiman M, Wick B. *Clinical Management of Binocular Vision: Heterophoric, Accommodative and Eye Movement Disorders.* 3rd ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2008:9

(ii) NPC- Near Point of Convergence [1-3]

Purpose:

This test evaluates a patient's ability to binocularly converge and sustain convergence comfortably and efficiently, while following a moving target to the punctum proximum of convergence. It helps to isolate strabismus, convergence insufficiency (CI) or excess (CE). The test is one of the three components of diagnosis for a CI (receded NPC, higher exo at near than normal and low base-out positive fusional vergence). Suppression indicates a long-standing situation.

NOTE: Use of a red lens for NPC is recommended as it provides a semi-dissociating state. Worsening or weakening of convergence with a red lens is indicative of a fragile fusional system[2]. The doctor is evaluating the patient's ability to **release** (ability to let eyes go from a target at near [at the NPC] to a more distant target) and **re-grasp** (ability of patient to move from the more distant target back to a point near the NPC). The doctor should repeat the entire sequence at least one more time to evaluate if with repetition the NPC recedes.

The norm is a break of 3 inches or closer. Recovery should be at 5 inches or 2 inches farther from the patient's break point.

References:

1. Leslie S. Accommodation in acquired brain injury. In: Suchoff IB, Ciuffreda KJ, Kapoor N, eds. Visual and Vestibular Consequences of Acquired Brain *Injury*. Santa Anna CA: Optometric Extension Program Foundation; 2001:56-57.
2. Scheiman M, Wick B. *Clinical Management of Binocular Vision: Heterophoric, Accommodative and Eye Movement Disorders.* 3rd ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2008:43-44.
3. Griffin JR, Grisham JD. *Binocular Anomalies Diagnosis and Vision Therapy*. 4th ed. New York NY: Butterworth Heinemann; 2002:49-52.

(iii) Vergence Facility [1, 2]

Purpose:

To assess one's ability to make rapid step vergences changes over a period of time: it is a measure of vergence stamina and indirectly one's sustaining ability. This test should be performed to the extent of the patient's abilities. Near point of convergence and vergence ranges may be normal, but facility can still be affected to cause difficulty with change of focus plane. Vergence facility is assessed by noting how many cycles of BI and BO the patient is able to complete in one minute (one cycle equals successful completion of base in and base out).

Materials:

- 12 base out/three base in flipper prism

-20/30 vertical letters target
-reading Rx if used
Procedure/Instructions to patient:
-Look at the letters. I will add a measuring device in front of your right eye, and you may see double. Try to make the letters single and clear. As soon as you do, say "now." I will then change the lens, and you will need to make the letters single and clear again. You will repeat this for 60 sec. With every lens change, try to get the letters single and clear.
Recording**:**
Record the number of cpm in 60 seconds. If the patient cannot clear base-in or base-out prism, note this as 0cpmBI or 0cpmBO
Expected value:
15 cpm

References:

1. Scheiman M, Wick B. *Clinical Management of Binocular Vision: Heterophoric, Accommodative and Eye Movement Disorders.* 3rd ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2008:4-5.
2. Gall R, Wick B, Bedell H. Vergence Facility: establishing clinical unity. *Optom Vis Sci* 1988:75;731-42.

5. Accommodation in Free Space

a) Amplitude[1-2]

Purpose: Monocularly measures the total amount of accommodation. Either push-up or pull-away methods can be used. There is no statistical difference between the two methods. It is important to remember that both methods overestimate the amplitude by 2 diopters. This test is not performed on presbyopes. Values vary by age. Hofstetter's formula for the average accommodative amplitude is: 18.5 - ⅓ age. The formula for the minimum amplitude for age is: 15 - ¼ age. You can also repeat the amplitude test to look for fatigue over time.

References:

1. Scheiman M, Wick B. *Clinical Management of Binocular Vision: Heterophoric, Accommodative and Eye Movement Disorders.* 3rd ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2008:18-20.
2. Leslie S. Accommodation in acquired brain injury. In: Suchoff IB, Ciuffreda KJ, Kapoor N, eds. Visual and Vestibular Consequences of Acquired Brain *Injury*. Santa Anna CA: Optometric Extension Program Foundation; 2001:62.

b) Accommodative Facility

(1) Binocular Accommodative Facility

This test determines how well a patient makes rapid and accurate step changes in accommodation binocularly. The vergence system is also activated during this test: as minus lenses stimulate accommodation and accommodative convergence, negative fusional vergence will need to compensate for the accommodative convergence in order to maintain single vision. This test should be performed before the monocular test of accommodative facility. If passed, there is no need to repeat the test monocularly. If failed, the accommodative response can be tested monocularly as described below. Methods for testing have been described by Grunning,[1] Griffin,[2] and Scheiman.[3] Values are expected to be 8 cpm for binocular. Normative studies indicate the range to be 7 to 13 cpm binocularly.

References:

1. Grunning CF, Hong C, Wong LC. *Vision therapy Diagnostic Procedures Laboratory*. Clinical manual for State University of New York, State College of Optometry. 1993.
2. Griffin JR, Grisham JD. *Binocular Anomalies Diagnosis and Vision Therapy*. 4th ed. New York, NY: Butterworth Heinemann; 2002:45-46.

3. Scheiman M, Wick B. *Clinical Management of Binocular Vision: Heterophoric, Accommodative and Eye Movement Disorders.* 3rd ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2008:46-47.

(2) Monocular Accommodative Facility [1, 2]

This test determines how well a patient makes rapid and accurate step changes in accommodation while maintaining clarity of vision. Note postural changes, attempts on the part of the patient to pull the card closer or move it farther away, squinting, and asthenopic complaints. Expected values for a 13 to 30 year old are monocular 11 cpm. Normative studies indicate a range of 10 to 17 cpm monocularly [1].

Note for 30 to 40 year olds: For this age group, the working distance and the lens power to be used will be different than what is used for <30 years. Below is a chart adopted from Scheiman and Wick[1] to help determine the appropriate flipper lens power to be used. Keep in mind if you determined the amplitude of accommodation using the minus lens technique, the chart below gives the values for the Push-up Technique. Thus, you will need to first compensate for this by adding 2D to the amplitude value found with the minus lens. After converting to the appropriate dioptric value, use the chart below to determine the test distance and flipper power accordingly.

TABLE 1.5. *Amplitude scaled facility*

Test distance = 45% of amplitude*
Lens power range = 30% of amplitude†

Amplitude	Distance from nose (cm)	Test distance (cm)	Flip lens power‡
22.25	4.5	10.0	±3.25
20.00	5.0	11.0	±3.00
18.25	5.5	12.0	±2.75
16.75	6.0	13.5	±2.50
15.50	6.5	14.5	±2.25
14.25	7.0	15.5	±2.25
13.25	7.5	16.5	±2.00
12.50	8.0	18.0	±2.00
11.75	8.5	19.0	±1.75
11.00	9.0	20.0	±1.75
10.50	9.5	21.0	±1.50
10.00	10.0	22.0	±1.50
9.50	10.5	23.5	±1.50
9.00	11.0	24.5	±1.50
8.75	11.5	25.5	±1.25
8.25	12.0	26.5	±1.25
8.00	12.5	28.0	±1.25
7.75	13.0	29.0	±1.25
7.50	13.5	30.0	±1.00
7.25	14.0	31.0	±1.00
7.00	14.5	32.0	±1.00
6.75	15.0	33.5	±1.00
6.50	15.5	34.0	±1.00
6.25	16.0	35.5	±1.00
6.00	16.5	37.0	±1.00
5.75	17.5	38.5	±1.00
5.50	18.0	40.5	±0.75
5.25	19.0	42.5	±0.75
5.00	20.0	44.5	±0.75
4.75	21.0	47.0	±0.75
4.50	22.0	49.5	±0.75

Scoring criteria: patients scoring less than 10 cpm are likely to be symptomatic.
*rounded to nearest 0.5 cm.
†rounded to nearest 0.25 D.
‡range divided by 2.

Example using the Minus Lens Technique:

If the patient's minus lens accommodative amplitudes are 7 D, add 2D resulting in 9D as the overall accommodative amplitude. Refer to amplitude listed in the left column of the chart to get the appropriate testing distance and lens flipper power. For 9D, the chart recommends the use of +/- 1.50 D flippers @ 24.5 cm.

References:

1. Grunning CF, Hong C, Wong LC. *Vision therapy Diagnostic Procedures Laboratory*. Clinical manual for State University of New York, State College of Optometry. 1993.
2. Scheiman M, Wick B. *Clinical Management of Binocular Vision: Heterophoric, Accommodative and Eye Movement Disorders.* 3rd ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2008:581-3.

c) Accommodative Response[1-23]

Dynamic retinoscopy can be used to determine the optimum near lens for brain-injured patients. Using dynamic retinoscopy is valuable for all age groups of the brain injured population. Individuals with brain injury often suffer from extreme visual stress when reading or performing near tasks. Dynamic retinoscopy will determine the optimum near point lens for these individuals that can relieve some of their visual stress.

Dynamic retinoscopies are, with the exception of the Monocular Estimate Method (MEM), binocular procedures usually performed at near viewing distances. As the terms imply, static retinoscopies mean the patient is thought to be passive, and dynamic retinoscopies require varying levels of patient cognitive activity. As such, dynamic retinoscopies can provide the doctor with information and insights regarding the patient's abilities and level of visual processing at the chosen distance.

The most commonly used dynamic retinoscopy with the brain-injured population is book retinoscopy. The optometrist examiner holds reading material at the patient's near working distance (usually 16 inches). While the patient is asked to read the material the optometrist observes the retinal reflex for color, brightness, size and motion. The optometrist places a binocular low plus lens in front of the patient's eyes while reading to try to improve the qualities of the reflex and neutralize the motion.

MEM, Nott, Bell or Stress Point are other forms of dynamic retinoscopy that may be used.

References:

1. Apell RJ. Book retinoscopy. Eastern States Conference transcript, 1958.
2. Apell RJ. Clinical application of bell retinoscopy. *J Am Optom Assoc* 1975; 46:102-03.
3. Apell RJ, Lowry R. Pre-school vision. St. Louis: American Optometric Association, 1959.
4. Apell RJ, Streff JW. The performance skills, optometric childcare and guidance. Duncan, OK: OEP Curriculum II 1962;4.
5. Garcia A, Cacho P. MEM and Nott dynamic retinoscopy in patients with disorders of vergence and accommodation. *Ophthalmic Physiol Opt* 2002; 22:214-20.
6. Gesell A, Ilg FI, Bullis GE. Vision: It's Development In Infant and Child. Santa Ana, CA: Optometric Extension Program, 1998.
7. Getman GN. Techniques and diagnostic criteria. OEP Curriculum II 1959; 4:17-26.
8. Getman GN. The development of binocularity part II. Duncan, OK: OEP Curriculum II 1958; 8:53-59.
9. Getman GN Kephart NC. Book retinoscopy. Duncan, OK: OEP Curriculum II 1958; 10 & 11:65-74.
10. Haynes HM. Clinical observations with dynamic retinoscopy. *Optom Wkly* 1960; 51:2306-09.
11. Hendrickson H. The behavioral approach to lens prescribing. Duncan, OK: OEP Curriculum II 1958; 4:25-31.
12. Kraskin RA. Stress point retinoscopy. *J Am Optom Assoc*. 1965; 36:416-19.
13. Kraskin RA. Lens Power in Action. Santa Ana, CA: Optometric Extension Program, 2003:41-62.
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15. Kruger PB. The effect of accommodative changes on the brightness of the fundus reflex. *J Am Optom Assoc* 1978; 49:47-9.
16. Nott IS. Dynamic skiametry, accommodative convergence & fusional convergence. Am J Physiol Opt 1925; 6:366-74.
17. Pascal JI. Modern retinoscopy. London: Hatton Press, 1930.
18. Steele G. Dynamic Retinoscopy-More than a Snapshot. *J Beh Opt* 2007 18(2):126-8.
19. Streff JW, Claussen VE. Retinoscopy measurement differences as a variable of technique. *J Am Optom Assoc1974*; 47:671-76.
20. Streff JW. Infant retinoscopy. *Optom Vis Dev* 2006; 37:131-37.
21. Tassinari JT. "Monocular estimate method retinoscopy: central tendency measures and relationship to refractive status and heterophoria." *Optom Vis Sci*. 2002 Nov; 79(11):708-14. PMID 12462539.
22. Taub MB, Rowe S, Bartuccio M. "Examining special populations. Part 3: Examination techniques." OT. March 10, 2006. Accessed October 2, 2006.
23. Wallace DK, Carlin DS, Wright JD. Evaluation of the accuracy of estimation retinoscopy. JAAPOS 2006; 10:232-36.

6. Sensory Status

When a binocular dysfunction is suspected, a doctor should probe certain features of sensory fusion such as suppression, simultaneous perception, or flat fusion. Sensory fusion anomalies are more of concern in strabismus. In non-strabismic patients, it is not as severe an issue because typically the suppression scotoma is small and the degree of suppression is not as dense. However, the presence of suppression or reduction in stereopsis may affect prognosis and treatment.

a) Suppression [1-3]

(1) Worth Four Dot

This is an objective test in free space to determine gross fusion and binocular luster. It is also used to differentiate whether the patient has simultaneous perception (diplopia) or flat fusion (second degree fusion) of two similar ocular images. Deep suppression is suspected if still present when the patient is retested in a dark room.

References:

1. Grunning CF, Hong C, Wong LC. *Vision therapy Diagnostic Procedures Laboratory*. Clinical manual for State University of New York, State College of Optometry. 1993.
2. Scheiman M, Wick B. *Clinical Management of Binocular Vision: Heterophoric, Accommodative and Eye Movement Disorders.* 3rd ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2008:16
3. Griffin JR, Grisham JD. *Binocular Anomalies Diagnosis and Vision Therapy*. 4th ed. New York, NY: Butterworth Heinemann; 2002:140-1.

(2) Stereopsis (Randot)

The purpose of this test is to evaluate the presence and degree of stereopsis. The Randot Stereo test uses both global and contour targets [1]. Contour targets such as the Wirt circles laterally displace two similar targets and, therefore, even someone without stereopsis could guess correctly based on monocular cues.[1] In addition, even with a constant strabismus, it is possible to appreciate 70 sec of arc stereopsis,[2] which is considered to be peripheral stereopsis.[1] Therefore, contour stereopsis detects if peripheral stereopsis is present. In contrast, global targets, such as Randot shapes, have no monocular clues, [1] and patients can only appreciate them if they have bi-foveal fusion.[3] Constant strabismics will not appreciate global targets.

References:

1. Grunning CF, Hong C, Wong LC. *Vision therapy Diagnostic Procedures Laboratory*. Clinical manual for State University of New York, State College of Optometry. 1993.
2. Scheiman M, Wick B. *Clinical Management of Binocular Vision: Heterophoric, Accommodative and Eye Movement Disorders.* 3rd ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2008:17-8.
3. Griffin JR, Grisham JD. *Binocular Anomalies Diagnosis and Vision Therapy*. 4th ed. New York, NY: Butterworth Heinemann; 2002:14.

7. Retinoscopy and Refraction:

An accurate refraction is crucial in the acquired brain injury population. This population has a high incidence of accommodative, binocular, vestibular, and proprioceptive issues.[1] Even a small uncorrected refractive error tolerated prior to the acquired brain injury may no longer be tolerated after the brain injury.[1] Small amounts of uncorrected refractive error should be compensated for with a prescription.[1]

Proper measurements of alignment and accommodation require the correct distance prescription.[1] The doctor should do a full refraction with a binocular balance establishing a full plus refraction.[1]

Typically, retinoscopy and refraction are done using a phoropter; however, in some cases, this is not possible. Trial frame/loose lens refraction, or a lens rack, may be necessary if the patient is unable to sit behind the phoropter.[2] Reliable responses may be difficult at times. Auto-refraction results can be used only as a starting point as they do

not account for binocular balance. Objective procedures such as retinoscopy in conjunction with supplementing auto refraction and keratometry may be critical in determining an accurate refractive error.[3]

References:

1. Scheiman M, Wick B. *Clinical Management of Binocular Vision: Heterophoric, Accommodative and Eye Movement Disorders.* 3rd ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2008:585
2. Falk NS, Aksionoff EB. The primary care optometric evaluation of the traumatic brain injury patient. *J Am Optom Assoc* 1992; 63:547-53.
3. Cohen A. Acquired visual information-processing disorders: closed head trauma. In: Press L, ed. *Applied Concepts in Vision Therapy*. St. Louis: Mosby; 1997:154-166.

8. Phorometry[1-2]

All components of phorometry are well described in the references below. There are no differences in performing these techniques on patients with TBI. They are the same as for the non-injured population. Note: not all patients with TBI will be able to keep their head up against the phoropter. Take care to monitor patients' head position as this can influence their response. Allow patients with TBI more time to respond to these tests. When orthophoria is measured, it is still recommended to check both sets of fusional ranges. As little as one prism diopter has been noted to benefit this population. Be sure to speak slowly when giving instructions.

Tests to be performed include:

-von Graefe distance and near, horizontal and vertical phoria

-Risley Prism assessment of positive and negative fusional vergence ranges

-Accommodative amplitude (push up or minus lens techniques)

-Negative and positive relative accommodation

-Accommodative convergence to accommodation ratio

Norms for accommodative amplitude tests are listed below:

Hoffstetter (average) Push-up Technique:

18- ⅓ age (in years) = average amplitude

Minus lens technique:

[18-⅓ age (in years)] – 2 = average amplitude

The expected value for minus lens amplitude is 2 D less than push-up amplitude. Push-up technique tends to overestimate the amplitude.

References:

1. Benjamin WJ. *Borish's Clinical Refraction.* 2nd ed. New York, NY: Butterworth Heinemann; 2007
2. Scheiman M, Wick B. *Clinical Management of Binocular Vision: Heterophoric, Accommodative and Eye Movement Disorders.* 3rd ed. Philadelphia, PA: Lippincott Williams and Wilkins; 2008

SECTION 2: Evaluation and Assessment

G. Examination of Cortical Visual Function

Kara Gagnon, O.D., Chrystyna Rakoczy, O.D., Brenda Montecalvo, O.D., and Michael Peterson, O.D.
Drawings: courtesy of Chrystyna Rakoczy, O.D.

Cerebral blindness is vision loss due to damage to both visual pathways posterior to the lateral geniculate body (LGB). Cortical blindness is bilateral vision loss specifically from damage to the striate cortex. Cortical blindness is considered a type of cerebral blindness. There is no afferent pupillary dysfunction. Pupillary constriction to convergence and accommodation is retained. All oculomotor function is intact. The definition of cerebral blindness does vary on the topic of visual acuity. Some believe the patient's acuity should be "no light perception" bilaterally,[1] whereas others believe any loss of acuity is acceptable as long as the loss is equal in both eyes.

There are many causes of cerebral/cortical blindness. The most common are anoxia, hypoxia and vascular insufficiency secondary to infarct from embolism or hypotension. Another cause may include chiropractic cervical manipulation.[2] Temporary hypotensive situations can cause temporary cerebral/cortical blindness. This usually happens with mild closed head trauma and in patients with temporary vascular insufficiency of the vertebral-basilar system. The blindness clears within hours.[1] Those who get migraines may be predisposed to temporary cortical blindness.[3]

Anton's Syndrome is anosagnosia associated with cerebral blindness in which patients are unaware of their blindness. They actually "make up" their visual environment.

1. Reflex Blink to Visual Threat:

Purpose:
The reflex blink to visual threat is absent in cortical blindness. It requires an intact afferent pathway and occipital, frontal and parietal lobes.[4]
Procedure:
The reflex blink can be elicited in unaffected individuals with a menacing hand gesture, but one needs to be sure not to push air onto the cornea so as not to elicit a corneal blink reflex. Consideration for hemifield deficits: no dazzle or sound need be produced.
Recording**:** Reflex blink to visual threat present or reflex blink to visual threat not present.

References:

1. RM Burde, PJ Savino, JD Trobe, Clinical Decisions in Neuro-Ophthalmology, Mosby 2002 pg 91
2. JW Gittinger, Occipital infarction following chiropractic cervical manipulation. *J Clinical Neuro-Ophthalmology* 1997:123;851-852
3. SH Greenblatt. Posttraumatic transient cerebral blindness. Association with migraine and seizure diathesis. *JAMA* 1973:225:1073-1076
4. GT Liu, M Ronthal. Reflex blink to visual threat. *J Clinical Neuro-Ophthalmology* 1992:12;47-56

2. Optokinetic Nystagmus (OKN)

Purpose:

This test is typically reserved for special cases (for example, an unresponsive patient).[1] It may also be used when there is left brain damage and the patient is unable to communicate.[2] If a patient is able to resolve the stripes on the drum, an involuntary OKN movement is observed.[1] This test grossly assesses if the neurological pathway from the eye to the cortex is intact.[2] OKN testing will depend on the doctor's ability to get the patient to fixate on the drum, and this test may be unreliable if cortical blindness is present.[2]

OKN is the eye movement elicited by the tracking of a moving field.

Patients with horizontal nystagmus, typically seen in unilateral hemispheric lesions, especially parietal or parietal-occipital lesions, show impaired optokinetic nystagmus when the drum is rotated toward the side of the lesion.[3]

Materials:

-OKN drum or OKN tape with large squares, or APP for iPad (iPhone field is too small to produce accurate responses)

Procedures:

Comfortably seat patient. Hold the OKN drum in front of the patient with the bars directed vertically and spin the drum to the left (the patient's right). The examiner should observe a slow pursuit movement of the eyes to the patient's right as a moving bar is followed, then a quick saccade to the patient's left as the patient searches for the next moving bar to fixate on and again follows that bar with a slow pursuit movement to the patient's right. This is then repeated in the other direction, observing the motion of the eyes with the spinning of the drum to the right (the patient's left). Observe the presence of OKN response. Record any asymmetry in direction for each eye. The motion of the eyes should be observed, keeping in mind the latency and velocity of the response, the amplitude of the response, smoothness of movement of the response, the fatigability of the response, and the direction of the response, all of which should be recorded.

To obtain acuity, present stripes of decreasing width until an OKN response is no longer generated. Approximate visual acuity using the testing distance and width of the stripes.[1]

Recording:

Record whether or not nystagmus was noted.

References:

1. Falk NS, Aksionoff EB. The primary care optometric evaluation of the traumatic brain injury patient. *J Am Optom Assoc* 1992; 63:547-53.
2. Swanson M. Eye Quest Magazine. January/February 1995 50-55.
3. Young PA, Young PH. Basic clinical neuroanatomy. Baltimore: Williams & Wilkins, 1997:125.

3. Visual Neglect/Inattention: Testing

Purpose:

Visual spatial neglect refers to when a patient ignores a segment of visual space.[1] Typically, the propensity is neglect for the left side of vision as a result of right parieto-occipital lobe damage.[1] Visual neglect is found in approximately 50 percent of right-sided cerebrovascular accidents.[2] Visual neglect is not the same as a visual field defect. It can be found in conjunction with a visual field defect or may exist in isolation. In contrast to sensory visual field defects, patients with neglect will not move their eyes to visualize the missing field. It is critical for the optometrist to distinguish between visual field defects and visual neglect as prognosis is worse in the case of neglect.[1]

a) Confrontation Extinction Check

Procedures: Confrontation fields are performed in a normal fashion. In addition, the doctor presents two stimuli, one in each hemifield (double simultaneous stimulation). If the initial confrontation field was noted to be full in each eye and now with two targets one hemifield is extinguished then the patient has neglect.
Instructions: How many finger/targets do you see altogether?
Recording: neglect/inattention or no neglect/inattention

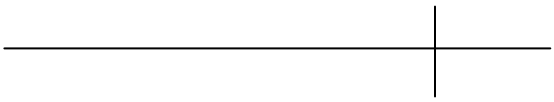
b) Drawings

Materials: pencil and paper
Instructions: copy/draw as instructed below.
Recording: neglect/inattention or no neglect/inattention

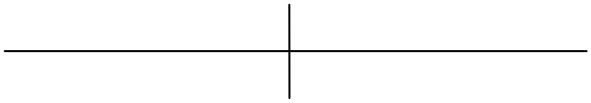
(1) Line Bisection

Visual neglect can be tested during a routine optometric examination by simple line bisection. The patient is asked to mark a line, bisecting it through the center of a line.
See below:

Shows neglect of left visual field:



No neglect:

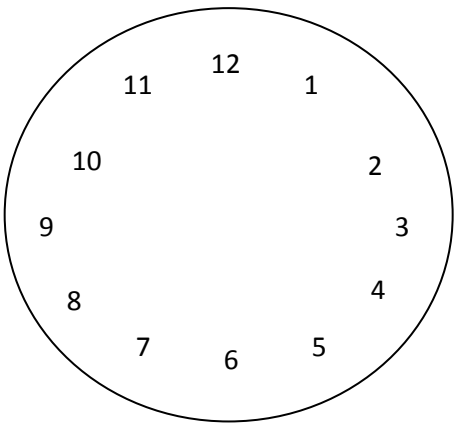


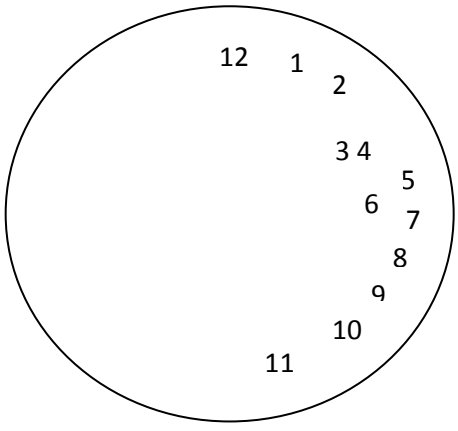
References:

1. Swanson M, Eye Quest Magazine. January/February 1995 50-55.
2. Halligan PW, Marshall JC. Recovery and regression in visuo-spatial neglect: a case study of learning in line bisection. *Brain Inj* 1991; 1:23-31.

(2) Clock

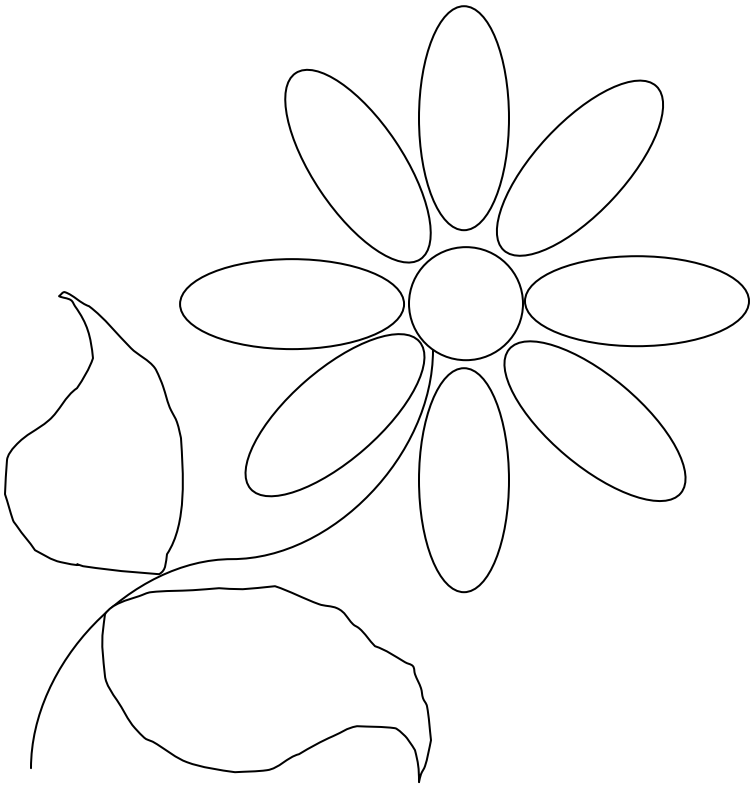
The face of a clock can be drawn for a patient to copy. If the patient has neglect, the numbers on the left-hand side will be copied to the right side of the clock face or will be ignored altogether.[1]

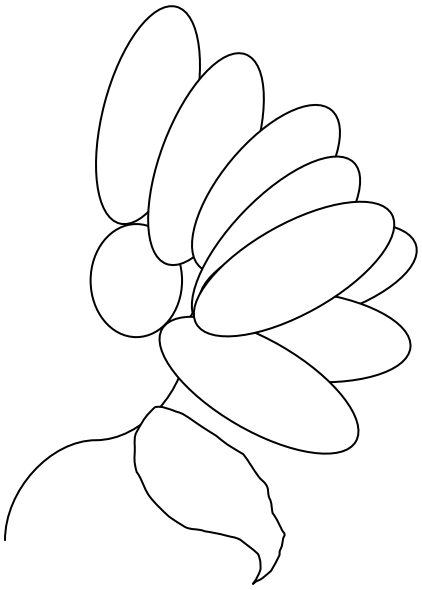




(3) **Flower**

Draw a daisy with five or six petals for the patient to copy. The patient with neglect will leave out the petals on the left or squeeze them to the right side.[1]



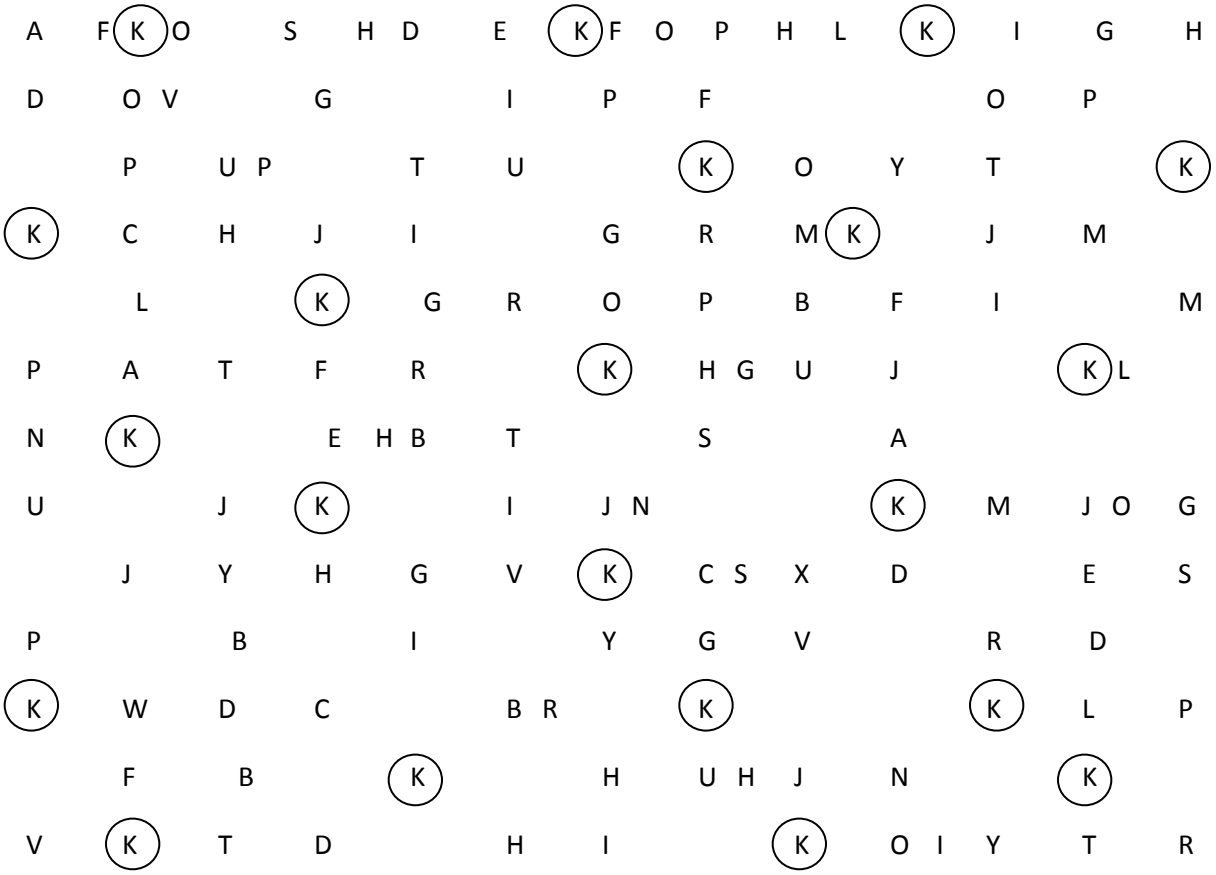


Sample of copied drawing of patient with neglect

(4) **Letter Cancellation**

Patient is presented with a page of random letters and asked to locate all instances of a particular letter. The patient with neglect will identify only letters on the right.[1] (see next page for example)

CANCELLATION OF LETTERS: FIND THE LETER "K" (normal)



CANCELLATION OF LETTERS: FIND THE LETER “K” SHOWING NEGLECT

A F K O S H D E K F O P H L (K) I G H

D O V G I P F O P

P U P T U K O Y T (K)

K C H J I G R M (K) J M

L K G R O P B F I M

P A T F R K H G U J (K) L

N K E H B T S A

U J K I J N (K) M J O G

J Y H G V K C S X D E S

P B I Y G V R D

K W D C B R K (K) L P

F B K H U H J N (K)

V K T D H I (K) O I Y T R

4. Visual Agnosia: Testing

a) Simple Visual Agnosia

The inability to recognize and name common objects presented. Show a pen, cup, or book and ask the patient to identify.[1]

b) Prosopagnosia

The inability to recognize familiar faces: a magazine such as "People" could be used to show faces of well-known TV personalities.[1] Ask the patient to identify a familiar face.

c) Simultagnosia

The inability to analyze complex situations: this can be tested with a Navon figure (see below) where the patient will see the individual letters making up the overall shape but not see the whole picture.[1] Ask patients what they see. A patient with simultagnosia will only see the "As" and not the "H."

A A

A A

A A

A A A A A A A A

A A

A A

A A

d) Central achromatopsia

The inability or difficulty to perceive colors secondary to a cortical lesion: Hardy-Rand Ritter color plates or Farnsworth D-15 can be used to test for this anomaly. Tritan or yellow/blue defects are common if the deficit is incomplete.[1]

References:

1. GT Liu, M Ronthal. Reflex blink to visual threat. *J Clinical Neuro-Ophthalmology* 1992:12;17-18

5. Abnormal Spatial Sense

One of the simplest ways to evaluate egocentric localization at a gross level is to observe patient behavior upon observing the patient in the reception room.[1] Notice how the patient sits in a chair. Look for a head turn/tilt or if the individual is leaning to one side. Does the person "drift" while walking, demonstrate any unsteadiness or use a hand to "feel" the wall when walking down the hall? Also observe head position. Is it rotated right, left, forward, back or tilted right or left?

- "Face-to-face" procedure. While facing the patient at eye level, ask the patient to "point" his/her nose directly at your nose, so that if you both leaned forward the tips of your noses would touch, looking for any tilting, turning or misalignment of the head.[1]

- With patient in a seated position, hold a target at 16 inches to the side and have patient fixate target. As target is moved horizontally at the patient's eye level, ask the patient to report when the object appears straight in front of his or her nose. If the patient's reaction time is delayed, the test can be modified by placing the target directly in front of the patient's nose at 16 inches and asking the patient if the target should be moved to the right or left to align it with his or her nose. Any estimation other than directly in front of the nose is considered a shift of egocenter. Measure the amount of shift in inches at distance tested. The shift can be to the right or left. An upward or downward shift can be observed when the target is moved vertically in front of the patient.

- A third quick assessment screening is to ask patients to quickly touch the tip of their noses with the tip of their fingers. If there is a shift, patients will touch the side of their nose.

References:

1. PS Suter, LH Harvey, Vision Rehabilitation: Multidisciplinary Care of the Patient Following Brain Injury, 2011 CRC Press
2. IB Suchoff, KJ Ciuffreda, N Kapoor, Visual and Vestibular Consequences of Acquired Brain Injury, 2001 Optometric Extension Program Foundation, Inc

SECTION 2: Evaluation and Assessment

H. Vestibular Dysfunction Screening

Kara Gagnon, O.D., and Chrystyna Rakoczy, O.D.

Optometrists can perform many tests for vestibular dysfunction. Below are some that can be easily incorporated into a primary optometric examination. Patients who exhibit abnormal findings on any of these tests should be referred to a neuro-otologist or physical therapist who specializes in vestibular dysfunction.

1. Dynamic Visual Acuity [1-4]

Given that a defective vestibular ocular reflex (VOR) is most evident with patient movement, visual acuity is expected to be worse under dynamic testing conditions vs. static [1].

The dynamic visual acuity test assesses impairments in a patient's ability to perceive objects accurately while actively moving the head [1]. In uninjured individuals, losses in visual acuity are minimized during head movements by the VOR system that maintains the direction of gaze on an external target by driving the eyes in the opposite direction of head movement [2]. When the VOR system is impaired, visual acuity degrades during head movements [2].

DO NOT PERFORM THIS TEST ON PATIENTS WHO HAVE A KNOWN OR SUSPECTED NECK INJURY!

Materials:

-Snellen Chart

Procedure:

The patient is seated wearing distance Rx. The patient is asked to read the lowest line possible on a Snellen eye chart to establish a baseline visual acuity. The doctor then turns the patient's head side to side at a rate of 2 hertz, or 2 cycles per second.[2] As the patient's head is moving, ask him or her to read the distance acuity chart. The patient should be able to read threshold acuity or one line above threshold.[2] Dynamic visual acuity measuring at least two lines worse than static threshold acuity is indicative of a deficient VOR.[3] This test should be abnormal in patients with bilateral vestibular weakness.[3]

Instructions:

-Read the lowest line you can see on the chart. Please do this again, but this time I will turn your head side-to-side from right to left.

Recording:

-Record best-corrected acuity OD, OS, OU

-Record acuity after rotation.

References:

1. Baloh RW, Horubia V. *Clinical Neurophysiology of the Vestibular System.* Philadelphia: FA Davis Company, 1990.
2. Mejia, GA. Vision & balance the optometrist's role in managing patients with dizziness and vestibular dysfunction. *J Behav Optom.* 2008; 19:97-102.
3. Burde RM, Savino PJ, Trobe JD. Clinical decisions in neuro-ophthalmology. 3rd ed. St Louis, MO: Mosby, 2002:197-218.
4. Dynamic Acuity Test. http://www.youtube.com/watch?v=doHHU30U0eE. Accessed June 17, 2013.

2. Oculocephalic Maneuver [1-3]

Purpose:

The oculocephalic maneuver (Doll's head) can be used in several situations: to ascertain a functioning oculo-vestibular system, to rule out cranial nerve palsy and to test the function of oculomotor nuclei in supranuclear gaze

paralysis and ocular motor apraxia. It is considered a postural reflex generated by the vestibular system. With every movement of the head and body, a current is generated in the endolymph of the semicircular canals. This current sends signals to the vestibular nuclei that then relay to the ocular motor nuclei to change the tonus of the ocular muscles. The effect of the labyrinths is both equal and antagonistic: the eyes are held steady when the head is not moving. Once the head position changes, the tonus of the extraocular muscles changes. This prevents the eyes from moving with the head. When the head turns to the right, the eyes seemingly turn left. When the head turns down, the eyes turn up. Do not perform this maneuver if cervical damage is suspected.

Materials:

None

Procedure/Instructions:

Gently and slowly rotate the patient's head from side to side. Observe if the eyes move with the head motion or against it. Repeat by rotating the head up and down.

Recording:

Positive if the eyes move against head movement

Negative if the eyes move with head movement

References:

1. Von Noorden GK, Campos EC. *Binocular Vision and Ocular Motility Theory and Management of Strabismus.* St. Louis, MO: Mosby; 2002:70-71
2. Trobe JD. *The Neurology of Vision*. New York, NY: Oxford University Press; 2001:337-339
3. Burde RM, Savino PJ, Trobe JD. *Clinical Decisions in Neuro-Ophthalmology.* 3rd ed. St. Louis, MO: Mosby; 2002:206.

3. Gaze Stabilization/VOR Cancellation (suppression) [1]

Purpose:

This test allows the doctor to check the stability of ocular fixation as the head is immobilized relative to the body while the body is in motion. This test should not be performed on wheelchair-bound patients.

Materials:

Rotating exam chair

Procedure/Instructions:

Have the patient sit in a rotating chair. Ask the patient to look at his or her own thumbs outstretched. Rotate the chair from side to side. Observe the patient's eyes.

Recording:

Note: fixation, lag of pursuit or saccadic intrusions

Expected results:

Stable ocular fixation

Steady fixation of the eyes on the thumb.

References:

1. VOR Cancellation Test. http://www.youtube.com/watch?v=ExOs7HSHv-c. Accessed June 17, 2013.

4. Sharpened Romberg Test

Purpose:

This test is used as a vestibular screening test. It is not for wheelchair-bound patients.

Procedure/Instructions:

In this test, ask patients to stand with feet heel to toe, with arms crossed across the chest. Ask patients to stand like this with their eyes open. Compare the amount of sway to the same action with eyes closed. More sway with eyes

closed suggests vestibular lesions, most commonly with sway to the side of vestibular weakness. The patient should be able to maintain balance for 30 seconds.
Expect falls toward affected side.
Recording:
Record whether or not patient could maintain posture for 30 seconds.
Note which direction the patient tilted or swayed and how long into the testing it occurred.

References:
1. Weiss LM. Visual-vestibular interaction in the acquired brain injury patient. *J Optom Vis Devel*. 2002; 33:33-41
2. Romberg Test http://www. Youtube.com/watch?v=YBQNwvWgREU. Accessed June 17, 2013
3. Sharpened Romberg Test http://www.youtube.com/watch?v=NS6XtWFbqjc. Accessed June 17, 2013

5. Fukuda Stepping Test

Purpose:
This is a vestibular screening test. This test is not for wheelchair-bound patients.
Procedure:
In this test, the patient is asked to march in place with eyes closed for 50 steps. Patients with vestibular weakness will rotate to the side of the weak labyrinth 30 degrees or more or 1 meter of movement with 50 steps. Expect rotation in the direction of the affected side.
Instructions:
Please close your eyes and march in place for 50 steps.
Recording:
Record whether or not the patient was able to complete the test. Indicate the amount of turn, the direction and after how many steps.

References:
1. Weiss LM. Visual-vestibular interaction in the acquired brain injury patient. *J Optom Vis Devel*. 2002; 33:33-41.
2. Fukuda Step Test. http://youtube.com/watch?v=atXCNq_CgHk. Accessed June 17, 2013

6. Tandem Gait Testing

Purpose:
This is a test of cerebellar function with eyes open and of vestibular function with eyes closed. This test is not for wheelchair-bound patients.
Procedure:
In this test, ask the patient to walk heel to toe in a straight line with eyes closed. Patients unable to take 10 steps without sidestepping exhibit vestibular dysfunction.
Instructions:
Close your eyes. Please walk a straight line with one foot directly in front of the other: the heel of the front foot touching the toe of the back foot.
Recording:
Record whether or not the patient was able to complete the test. Indicate when sidestepping occurred and to which direction.

References:
1. Weiss LM. Visual-vestibular interaction in the acquired brain injury patient. *J Optom Vis Devel*. 2002; 33:33-41.
2. Normal coordination examination: Tandem Gait Test. http://www.youtube.com/watch?v=unBS3eDY9NY. Accessed June 17, 2013.

3. Abnormal Coordination: Tandem Gait Test. Sharpened Romberg Test. http://www.youtube.com/watch?v=7DP_KOpPS7I. Accessed June 17, 2013.

SECTION 2: Evaluation and Assessment

I. Examination of Visual-Processing and Visual Perceptual Performance

Brenda Heinke Montecalvo, O.D., and Kara Gagnon, O.D.

Information on basic evaluation of efferent pathways can be found on the AOA website under Optometric Clinical Practice Guidelines: "Care of the Patient with Learning-Related Vision Problems" http://www.aoa.org/documents/CPG-20.pdf. In this resource manual, we pointed out high-yield testing and clinical pearls that apply to patients with TBI.

Patients who sustain traumatic brain injury can exhibit problems with a variety of visual processing or perceptual skills, thus affecting their ability to return to normal daily living activities.[1-2] The primary care optometrist can effectively screen at chair-side for a few basic perceptual skills, such as visual memory and spatial deficits, at the initial evaluation.[3-4] Chair-side screening can aid in determining if further extended evaluation is warranted, create recommendations for the rehabilitation team and design home visual perceptual activities that can facilitate the patient's rehabilitation. If initial screening indicates perceptual processing problems, a more comprehensive battery of visual perceptual tests can be performed. These tests can be administered by optometrists specializing in vision therapy or other disciplines such as neuro-psychology or occupational therapy. Below is a list of screening techniques and common visual perceptual tests that may be considered to probe visual perceptual processing deficits more specifically. Instructions for testing are included in their purchased package.

References:

1. Scheiman M, Gallaway M. Optometric Assessment: Visual Information Processing Problems. In: Scheiman M, ed. Optometric Management of Learning-related Vision Problems St. Louis: Mosby Elsevier, 2006: 369-414.
2. McKenna K, Cooke D, Fleming J, Jefferson A, Ogden S. The incidence of visual perceptual impairment in patients with severe traumatic brain injury. *Brain Inj* 2006; 20(5):507-518.
3. Cohen A, Rein L. The effect of head trauma on the visual system: The doctor of optometry as a member of the rehabilitation team. *J Am Opt Assoc* 1992; 63(8):530-536.
4. Gianutsos R, Suchoff I. Neuropsychological consequences of mild brain injury and optometric implications. *J Beh Opt* 1998; 9(1):3-6.

1. Chair-side Optometric Visual Perceptual Skill Screening:

The following are chair-side perceptual screenings. These "screening" tools were developed to assist the primary care optometrist to quickly identify possible problems of common visual processing or perceptual difficulties in patients with brain injury. If the results of the "screening" are positive for potential problems, it is recommend the patient return for further evidence-based assessments for accurate diagnosis prior to treatment intervention. (See below for visual processing/perceptual skills and tests)

1. Laterality Screening:[13]
    a. Look to your right
    b. Look to your left
    c. Hold your hands in front of the patient with your palms up. Ask the patient to look at your right hand and then your left hand.
    d. If the patient fails, follow up with the Piaget Right Left Test.[1-3, 15] (see appendix)
2. Visual Memory Screening:

a. Put three large letters (approximately 20/80) on a visual acuity screen. Have the patient read each letter (silent or aloud). Turn off the projector or stand in front of the chart. Have the patient tell you what letters they remember. If the patient is non-verbal, have them point to the choice of correct letters on a presented page.
b. This procedure can be accomplished with objects if letters or numbers are difficult.
c. If the patient fails, follow up with the MVPT-3,[4] TVPS-3,[5] CTIP[6] or refer.

3. Spatial Deficiency Screening: Pen/Cap Test
   a. Hold a large highlighter marker vertically in front of the patient. Hand the patient the cap and ask the patient to look at the tip of the highlighter while placing the cap on it.[7,14]
   b. If the cap is misplaced to the right, left, front or back, the patient has a possible shift in spatial judgment.
   c. Complete more extensive egocentric shift evaluation or refer to a neuro-rehabilitation optometrist for intervention. [8-12]

References:

1. Suchoff I. Cognitive Development: Piaget's Theory. Santa Ana, CA: Optometric Extension Program 1978.
2. Furth H, Wachs H. *Thinking Goes to School: Piaget's Theory in Practice* New York: Oxford University Press 1975.
3. Cohen A. Acquired visual information-processing disorders: closed head trauma. In: Press L, ed. Applied Concepts in Vision Therapy. St. Louis: Mosby, 1997:154-166.
4. Groffman S, Solan H. Spatial orientation tests. In: Groffman S, Solan H, eds. *Developmental & perceptual assessment of learning-disabled children theoretical concepts and diagnostic*. Santa Ana, CA: Optometric Extension Program Foundation, 2001: 116-117.
5. Martin N. Test of visual perceptual skills (3rd ed) Novato, CA: Academic Therapy Publications. 2006.
6. Tombaugh TN, Rees L. Manual for the computerized tests of information processing Ottawa, ON Carleton Univ 2000.
7. Streff J. The value of observation *J Beh Opt* 1994; 1:13-14.
8. Politzer T., Suter P. Vision Examination of Patients with Neurological Disease and Injury. *Vision Rehabilitation* 442-445.
9. Gianutsos R, Suchoff I. Neuropsychological consequences of mild brain injury and optometric implications. *J Beh Opt* 1998; 9(1):3-6.
10. Halsband U, Lange RK. Motor learning in man: A review of functional and clinical studies *J Physiol Paris* 2006; 99:414-24.
11. Ciuffreda KJ, Ludlam DP. Egocentric Localization Normal and Abnormal Aspects Vision Rehabilitation Suter P ed Boca Raton CRC Press; 193-209.
12. Suchoff, I. *Visual Spatial Development in the Child* New York: State University of New York 1975.
13. Ludlam B. Clinical instruction & observations *Pacific University College of Optometry* 1983-1984.
14. Streff J. Invitational Lens Symposium Northeastern State University Oklahoma's College of Optometry Oklahoma 2001.
15. Groffman S, Solan H. Spatial orientation tests. In: Groffman S, Solan H, eds. Developmental & perceptual assessment of learning-disabled children theoretical concepts and diagnostic. Santa Ana, CA: Optometric Extension Program Foundation, 2001: 116-117.

2. Visual Processing or Perceptual Skills and Tests

a) Visual Motor

The influence of motor skills and oculomotor deficits can confound and impact the perceptual assessment. Therefore, visual perceptual tests are assessed with and without these influences. Perceptual tests have been classified as motor influenced and motor-free influenced, so the doctor can decipher between the two types of perceptual tests.[1-2] Brief descriptions follow. More extensive information about these standardized tests is available with instructions for testing in their purchased package. These can be performed as needed by the optometrist or referred to another member of the rehabilitation team.

Visual perceptual functioning requires a high level of cognitive processing and integration. The coding and decoding of visual information can be affected by oculomotor input and graphomotor skills.[2] However, using information

from oculomotor testing, both graphomotor-free tasks (DEM, saccades/pursuits) in comparison to graphomotor-influenced tasks, the doctor can tease out whether oculomotor influence is impacting visual perceptual processing.[2] Below are some high-yield visual processing tests that Cohen[1] recommends for this evaluation.

Wold Sentence Copy Test[4] and Wold Digit Symbol Copy Test[5]: With the norm timed element, there is information about the patient's visual-motor speed. Oculomotor deficits tested through DEM and observational chair-side saccades and pursuits can influence visuomotor and graphomotor functioning.[1] Graphomotor tests "involve sequential processing but not with high cognitive input."[3] Graphomotor (eye-hand coordination) functioning "refers to fine motor tasks that require visual guidance to achieve precision and dexterity."[3] With graphomotor tasks, the visual stimuli are not as complex as with visuomotor integration task[3]. These tests can be administered and compared to the DEM and observational oculomotor tests to decipher whether oculomotor deficits are influential in compromising graphomotor skills.[1]

Visual-Motor Integration (VMI): (Graphomotor influenced). Visual Motor Integration Test for Adults has good reliability and is used to determine level of hand/eye coordination skill.[5]

References:

1. Cohen A. Acquired visual information-processing disorders: closed head trauma. In: Press L, ed. Applied concepts in vision therapy. St. Louis: Mosby, 1997:154-166.
2. Scheiman MM, Gallaway M. Visual information processing: Assessment and diagnosis. In: Scheiman, ed. Optometric management of learning-related vision problems, St. Louis: Mosby-Elsevier, 2006.
3. Groffman S, Solan H. Spatial orientation tests. In: Groffman S, Solan H, eds. *Developmental & perceptual assessment of learning-disabled children theoretical concepts and diagnostic*. Santa Ana, CA: Optometric Extension Program Foundation, 2001: 116-117.
4. Wold RM. *Screening tests to be used by the classroom teacher*. Novato, California: Academic Therapy Publications, 1970.
5. Daly C, et al. Relationship between visual-motor integration and handwriting skills of children in kindergarten: A modified replication study *Am J Occup Ther* 2003;57 (4): 459-462.

b) Visual Motor Speed

Deficits in balance and response speed can be identified in a significant number of individuals even after mild TBI.[1] Dynavision[2], Acuvision, Wayne Saccadic Fixator, Vision Coach, and Wold Visual Motor Test are tests that measure the visual-motor speed or speed of visual process assuming no motor deficits are interfering.[3]

Visual Motor speed and accuracy are the individual's ability to react kinesthetically to an instantly presented visual stimulus. It is often referred to as reaction time. Computerized Test of Information Processing (CTIP) is one of the better tests for visual motor speed or reaction time giving insight into visual-perceptual speed (VPS).[4-7] With the advent of computers, reaction time (RT) is easier to assess with better reliability and repeatability[1]. Several studies have shown that brain injury affects RT, but can vary with severity and level of complexity required for reacting to presented stimulus.[4-7] Tombaugh and Rees[4] developed the Computerized Test of Information Processing (CTIP). The most basic test, Simple RT, is considered a pure measure of speed of information processing.[5] Although little normative data exists, Politzer [7] suggested VPS can be also assessed via two modes: stationary flashing targets and moving targets. Tachistoscopes are used for the first mode and a dynamic acuity tester or a tachistoscope with a variable speed rotating prism mount for the second.[7]

References:

1. Gagnon I, Forget R, Sullivan SJ, Friedman D. Motor performance following a mild traumatic brain injury in children: an exploratory study. *Brain Inj* 1998;12(10):843-53.
2. Klavora P, Gaskovski P, Martin K, Forsyth RD, Heslegrave RJ, Young M, Quinn RP. The effects of Dynavision rehabilitation on behind-the-wheel driving ability and selected psychomotor abilities of persons after stroke. *Am J Occup Ther*.1995 Jun;49(6):534-42.
3. Wold RM. *Screening tests to be used by the classroom teacher*. Novato, California: Academic Therapy Publications, 1970.
4. Tombaugh TN, Rees L. Manual for the computerized tests of information processing Ottawa, ON Carleton Univ 2000.
5. Tombaugh TN, Rees L, Stormer P, Harrison AG, Smith A. The effects of mild and severe traumatic brain injury on speed of information processing as measured by the computerized tests of information processing (CTIP) *Arch Clin Neuropsychol*

2007;22(1):25-36.
6. Trahan DE, Larabee GJ. Continuous visual memory test (CVMT) Lutz: FL: Psychological Assessment Resources 1988.
7. Politzer T., Suter P. Vision Examination of Patients with Neurological Disease and Injury. *Vision Rehabilitation* 442-445.

c) Motor-Free Visual Perception

Motor Free Test of Visual Perception (MVPT-3)[1], Test of Visual Perceptual Skills (TVPS-3)[2], and Developmental Test of Visual Perception (DTVP-A)[4] are newly revised motor-free visual perceptual tests.[1-4] Despite low-to-moderate reliability, these tests are frequently used due to no dependence on verbal or fine-motor skills.[5-10] In the least, these tests help to better understand when the patient is exhibiting difficulty with visual perception. Members of the rehabilitation team more versed in visual perceptual skill assessment and treatment may include: neuro-rehabilitation optometrist, occupational therapist, cognitive therapist or neuro-psychologist.[15]

d) Visual Memory

Visual memory is recollected information about what one has seen. It involves both the mental storage of such information and the ability to retrieve it. Test of Visual Perceptual Skills (TVPS-3)[2], Motor Free Test of Visual Perception (MVPT-3)[1], Developmental Test of Visual Perception (DTVP-A)[3-4] and Continuous Visual Memory Test (CVMT)[11-14] each have a sub-test that measures short-term visual memory and visual sequential memory that can be a factor with the brain-injured population.[1-4] A more involved assessment of visual memory with good reliability is the Continuous Visual Memory Test (CVMT).[11-14] The CVMT gives insight on the patient's ability to put information into long-term memory. The CVMT is specific to asses a variety of components of visual memory with good criterion validity in brain-injured patients.[11-14] Visuo-spatial problems can interfere with test results. The subtests available are recognition memory, delayed recognition memory and visual discrimination. [13]

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e) Visual Processing/Perceptual Speed

Speed of information processing is a major impairment in brain-injured patients.[1-2] Knowing the patient's speed of processing helps the rehabilitation team match the pace of information presented allowing for patients to improve performance.[1-2] On non-aphasic patients, during the optometric examination, delays in response to easily observed visual stimuli such as identifying a large visual acuity letter may indicate a slower than normal visual processing speed. Brain injury also has an impact on attentional and cognitive processes such as encoding, verbal comprehension, and adaptive responding to novel situations, all of which can create a delay in visual processing.[3-4]

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f) Visual Spatial

A spatial centered coordinate system (x, y and z) exists with extensive neural cortical pathways and thus is often affected from brain injury.[1-2] Perceptual spatial orientation deals with information about where one is with respect to objects. Localization deals with information to know where objects are with respect to the individual.[3] For accurate spatial awareness, the patient needs both precise oculocentric localization (OL) and egocentric localization (EL). OL is based on the foveal visual direction.[3] EL is body-based locating objects in the environment.[3] Egocentric shifts can be observed around any of the three orientation axes (x, y, z). It is common to have some rotation in all three axes. Often optometrists concentrate on measuring the "x" axis; however, it is important to evaluate all three for potential misalignment.

New test protocols were developed to measure shifts in egocenter consistently.[4] The Spatial Localization Board, Pointer Straw, and Pen-Cap Egocentric Shift Test all are reported to give the optometrist information on shifts of oculocenter and egocenter.[3-6] The Piaget Right Left Test also provides information on how the patient perceives body awareness, laterality, directionality, and spatial projection.[7]

Pointer Localization Test and the Pen-Cap Test: gives information on where objects may be mislocated in perceived space.[5-8] These tests give information as to misalignment of oculomotor center with monocular measurement or egocentric center with binocular measurement. The patient holds a pointer or cap and is asked to fixate the end of the straw or pen tip. Without changing fixation, the patient is asked to quickly place the pointer in the straw or the cap on the pen. Mislocation on the X, Y or Z axis is documented.[5-8]

X: number of inches to right or left of target fixated

Y: number inches above or below target fixated

Z: number inches in front or behind target fixated

Misalignment can occur on one or more of the visual axis. For the "Y" axis misalignment to be observed, the target needs to be held horizontally with the pointer or cap moving laterally toward the target.

Spatial Localization Board (SLB): The SLB was suggested by Streff as a way of quantifying and recording spatial localization shifts with patients.[9-11] Kraskin utilized similar assessments of a patient's spatial localization in order to determine the amount and direction of yoked prisms to prescribe.[10] The SLB is designed for clinical efficiency to allow the optometrist to quickly assess a patient's spatial localization in real space and time; determine x, y and z

axis spatial warps in nine primary meridians; record patient responses for pre- and post-test data (especially good for documenting egocentric shifts in brain injury) and quantify immediate effects of lenses and prisms. The SLB is a professional instrument with a modern and practical design. It can be used by children and adults as well.[9-11]

Egocentric Shift Test (EST):[3,5,6,12] With patient in a seated position, hold a target at 16 inches to the side and have patient fixate target. As target is moved horizontally at the patient's eye level, the patient is asked to report when the object appears straight in front of his or her nose. If the patient's reaction time is delayed, the test can be modified by placing the target directly in front of the patient's nose at 16 inches and asking the patient if the target should be moved to the right or left to align it with his or her nose. Any estimation other than directly in front of the nose is considered a shift of egocenter. Measure the amount of shift in inches at distance tested. The shift can be to the right or left. An upward or downward shift can be observed when the target is moved vertically in front of the patient. Treatment of egocentric shift can be complicated with minimal results. It is important to consult with an optometric rehabilitation specialist when treating egocentric shift problems.[3, 5,6,12]

Piaget Right Left Test (PRLT): The basic assessment of visual perceptual functioning can be done with the PRLT. This test provides information on how the patient perceives body awareness, laterality, directionality, and spatial projection.[13] PRLT also provides the individual with visual perceptual information important for the spatial centered coordinate system. PRLT is a simple test to administer in a routine primary eye care examination and provides useful information about visual perceptual processing.[14]

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g) Perceptual Organization

"The whole is greater than the sum of its parts," said Wertheimer in 1925[1-2]. Perceptual organization refers to how smaller objects are grouped to form larger ones,[1-2] establishing meaning from overlapping objects in a visual scene.[3] Sub-tests of visual closure and figure ground in Motor Free Test of Visual Perception (MVPT-3)[4], Test of Visual Perceptual Skills (TVPS-3)[5]and Developmental Test of Visual Perception (DTVP-A)[6] give insight into the patient's perceptual organization skill level.

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SECTION 2: Evaluation and Assessment

J. Ocular Health Examination

Candice Elam, O.D.

A complete evaluation of ocular health is required for any person with a history of head trauma. Following is a description of the more common ocular signs associated with traumatic brain injury. Some of the signs are more common than others. All are described here for completeness.

1. Anterior Segment

a) Angle Recession

Blunt trauma to the eye can cause uneven iris insertion, with an area of torn or absent iris insertion, and posteriorly recessed iris revealing a widened ciliary band on gonioscopy.[1] A recessed angle may also cause the scleral spur to appear abnormally white on gonioscopic exam. Glaucoma due to angle recession itself (not from the trauma causing the recession) takes approximately 20 years to develop and is most often unilateral.[1] Monitor patients with angle recession without glaucoma yearly, paying close attention to both eyes because there is a high incidence of delayed open-angle glaucoma and steroid-responsive intraocular pressure in both the uninvolved and traumatized eye.[1]

b) Dry Eye

Interference with normal reflex tearing and/or usage of certain medications can commonly cause dry eye in patients with a history of head trauma. Signs may include decreased tear breakup time, decreased tear meniscus, punctuate corneal or conjunctival staining, excess mucous or debris in the tear film, and filaments .[2] Incidence of dry eye is very high in patients with TBI.

c) Lagophthalmos and Keratitis

Incomplete closure of the eyelids causes corneal drying. The lower third of the cornea will show punctate epithelial defects and conjunctival chemosis and injection may be evident.[3] Corneal infiltrates, ulcers or erosion may also occur. Corneal sensation should be tested prior to instillation of anesthetics. If lubrication fails, eyelid taping, partial tarsorrhaphy, or eyelid gold weights (temporary or implanted) may be indicated.[3] The incidence of lagophthalmos in TBI is high. Light sensitivity is associated.

d) Filamentary Keratitis

Because dry eye is the most common cause of filamentary keratitis, patients will exhibit punctate epithelial defects, conjunctival injection and poor tear film coverage in addition to strands of mucus and epithelial cells attached to the anterior cornea.[4] Filaments can be removed with debridement, but they will recur if the underlying condition is not treated. Bandage contact lenses may be used in addition to long-term lubrication as treatment.[4]

e) Neurotrophic Cornea

Loss of corneal sensation causes fluorescein staining epithelial defects, perilimbal injection, and even corneal ulcers late if epithelial defects do not resolve.[5] Corneal sensation should be tested bilaterally with a sterile cotton wisp. A temporary tarsorrhaphy may be indicated and can be achieved with adhesive tape.[5]

2. Posterior segment

a) Terson's Syndrome

Terson's syndrome includes cases where any type of intraocular hemorrhage (intraretinal, preretinal, or intravitreal) is present after spontaneous or trauma-induced intracranial bleeding.[6] The intracranial hemorrhage may be subarachnoid, subdural, or intracerebral. The pathogenesis of the intraocular bleeding is thought to be due to a sudden increase in intracranial pressure that occurs during an intracranial bleed that causes an acute obstruction to retinal venous circulation resulting in rupture of superficial retinal vessels. Late complications of Terson's syndrome may include epiretinal membrane formation, perimacular retinal folds, and traction or rhegmatogenous retinal detachments.[6] Vision is decreased initially but has good spontaneous recovery.

b) Purtscher's Retinopathy

Purtscher's retinopathy involves bilateral patches or retinal whitening and hemorrhage around the optic disc in patients who suffer severe head or chest trauma, extensive long bone fracture, or certain systemic diseases (acute pancreatitis, pancreatic carcinoma, systemic lupus erythematous, chronic renal failure, thrombotic thrombocytopenic purpura, and following bone marrow transplantation).[6,7] Purtscher's occurs due to occlusion of small arterioles by intravascular microparticles consisting of fibrin clots, fat emboli, air emboli, platelet-leukocyte aggregates, or other particles of similar size.[7] Patients experience acute, painless loss of central vision in one or both eyes. Cotton-wool spots and intraretinal hemorrhages slowly disappear over weeks to months; however, there is no significant recovery of vision due to macular or optic nerve damage.[6, 7]

c) Whiplash Injury

Whiplash maculopathy may occur in adults who experience a whiplash injury to the head.[6] Visual acuity is mildly decreased (rarely worse than 20/30). Fundus examination shows a faint gray haze to the fovea with a small depression; a shallow posterior vitreous separation may be seen.[6] Vision returns to normal within days but the foveal depression persists.

d) Fat Embolism Syndrome

Fat embolism syndrome, seen within a few days of significant fractures of medullated bones, may show distinct posterior segment changes.[6] Bilateral cotton-wool spots and intraretinal hemorrhages are seen – they are typically smaller, less numerous and more peripheral than those seen in Purtscher's retinopathy.[6] The majority of patients with fat embolism syndrome have minor visual complaints or are asymptomatic. Due to the associated systemic manifestations of this syndrome (central nervous system alterations, respiratory compromise, fever, tachycardia, anemia, elevated erythrocyte sedimentation rate, and petechial rash), it is fatal in 20 percent of cases.[6]

e) Valsalva Retinopathy

Intraocular bleeding due to transfer of increased intrathoracic or intra-abdominal pressure into the eye is called valsalva retinopathy.[6, 7] Sub-internal limiting membrane hemorrhage is most common, typically unilateral or bilaterally asymmetric. The hemorrhage is often located in the macula, but typically clears without complications.[6, 7]

f) Papilledema and Raised Intracranial Pressure

Cerebral trauma can damage the arachnoid villi that absorb cerebrospinal fluid causing increased intracranial pressure.[7] This increase in intracranial pressure may lead to compression or dysfunction of the optic nerve, resulting in partial arrest of axoplasmic transport and subsequent papilledema.[6] Optic disc edema from increased intracranial pressure typically presents bilaterally as blurring of the optic disc margins, venous congestion of arcuate and

peripapillary vessels, anterior extension of the nerve head, and hyperemia of the optic nerve head.[6, 7] Retinal striae, choroidal folds, and peripapillary splinter hemorrhages may also be present. Visual acuity and visual fields vary depending on the stage of papilledema from normal to severely impaired.[7]

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SECTION 3: Review of Assessments and Diagnoses

A. Assumptions/Intent

This section presents a review of the more common as well as the lesser known visual dysfunctions following traumatic brain injury. Many of these dysfunctions are also noted after non-traumatic acquired brain injury such as stroke or as the result of neurologic disease. Uncommon visual disturbances are described in detail as the topics are elegant and access to information may be limited for the general optometrist. Common visual dysfunctions are described in context with trauma to the brain.

For additional information, refer to the section on advanced topics or the references cited.

SECTION 3: Review of Assessments and Diagnoses

B. Refractive errors

Michael Peterson, O.D.

TBI has been found to cause disruption in the ability of the eyes to focus for near vision and defocus for distance vision.[1] This can manifest as either an accommodative excess or pseudomyopia. Pseudomyopia is defined as subjectively blurred distance vision that improves with myopic correction in a person with no history of myopia and where cycloplegic refraction reveals much less myopia, emmetropia or even low hyperopia.[2] Although the specific cause of pseudomyopia following brain injury is unknown, one theory proposes an etiology of a lesion that irritates the accommodative portion of the parasympathetic third nerve subnucleus, leading to contracture of the ciliary body.[3] In addition, it is suggested that after a brain injury, individuals suffer from an impairment of learned spatial control of accommodation for viewing distance and near objects.[4]

It is also suggested that the natural history of post-traumatic pseudomyopia may occur in one of three ways: (1) a transient condition that will eventually resolve, (2) a recalcitrant condition that will resolve under cycloplegic examination, but will return immediately after cycloplegia wears off, or (3) less commonly, a condition where the myopia continues to increase over time.[3] Given that post-traumatic pseudomyopia does not follow the same course as functional myopia in regards to reversibility or worsening magnitude, management can be challenging.[3]

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SECTION 3: Review of Assessments and Diagnoses

C. Oculomotor and Accommodative Dysfunction

Matthew Rhodes, O.D, Candice Elam, O.D., and Chrystyna Rakoczy, O.D.

1. Eye Movement Disorders

Eye movement disorders can manifest when the ability to initiate and coordinate conjugate eye movements, or the ability to stabilize eye movements to achieve fixation, has been disrupted. Important categories of versional eye movements that can be affected by traumatic insult to the brain include: gaze stabilization (with consequent fixation instability), pursuits, and saccades.

Gaze stabilization deficits in brain injury can be manifestations of compromised gaze-holding mechanisms, which include the ability to detect and correct retinal image drift, suppress unwanted saccades induced by the vestibulo-ocular reflex during head movement, and resist the eye's tonic pull into primary gaze.[1] Objectively visible drifts, flicks, saccadic intrusions, or nystagmus may be indicators of a fixation deficit and should be observed during fixation exercises.

Damage to the pursuit pathway can result in asymmetric eye motion or decreased tracking velocity accompanied by catch-up saccades. Damage to the saccadic pathway can result in disrupted ability of the eyes to conjugately move from one target to another quickly and accurately and may manifest as increased latency, decreased peak velocity, saccadic targeting inaccuracy, and difficulty performing memory-guided saccades and anti-saccades.

Oculomotor disorders are documented to manifest with high frequency secondary to TBI. Record review study groups reported prevalence ranging from approximately 19 percent to approximately 51 percent in the TBI population.[3-6] It has been suggested the prevalence of saccadic anomalies may vary by mode of injury as indicated by a significantly higher rate of saccadic deficits observed in non-blast-related TBI patients as compared to blast-related TBI patients.[7] Notably, fixation instability involving nystagmus is far less likely to manifest after TBI than after stroke.[3] Emerging data suggest fixation instability, pursuit and saccade deficits are among the oculomotor disorders amenable to rehabilitation.[8]

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2. Binocular Disorders

a) Disorders of Ocular Alignment

(1) Traumatic Cranial Nerve Palsy

With head trauma, distortion of the skull and basal foramina and neural injury due to rotation are the primary mechanisms of direct cranial nerve damage.[1] Subsequent to impact, compression of cranial nerves can occur as subdural, subarachnoid, intra-cerebral bleeding, or cerebral edema ensues. Consequently, head trauma is a common cause of cranial neuropathies.[2]

The prevalence of observed Traumatic Cranial Nerve **(**CN) palsy in TBI populations ranges from about 7 to 9 percent.[3, 4] Multiple cranial nerves may be affected in about one-fourth of these cases. The three cranial nerves directly involved in ocular motility – CN III, IV, and VI – are the most commonly injured cranial nerves.[5, 6] Some reports associate closed head injury (CHI) with oculomotor CN palsy with more severe head trauma than CHI without oculomotor CN palsy as judged by comparing Glasgow Coma Scale Scores, rates of intracranial and craniofacial imaging abnormalities, and short-term neurological outcomes.[7] Furthermore, patients who have closed-head trauma severe enough to cause direct third nerve palsy can have multiple permanent neurological deficits.[8]

During the TBI eye examination, the doctor should carefully elicit any history of diplopia and evaluate signs of diplopia, lid ptosis, fixed or dilated pupil, head turn, head tilt, and facial droop or lagophthalmos. Reported diplopia raises suspicion of one or more paretic oculomotor cranial nerves. If a lid ptosis or dilated pupil is evident, a third nerve (CN III) palsy may be suspected. A head turn may suggest unilateral abducens nerve (CN VI) palsy. A head tilt may be a compensating head position for trochlear nerve (CN IV) palsy. Facial droop or lagophthalmos may be due to compromise of facial nerve (CV VII) function. Even in the absence of these or other signs or symptoms, the high prevalence of cranial nerve compromise after brain injury mandates a thorough neuro-ophthalmological evaluation. For guidance on diagnosis of cranial nerve palsies, refer to the referenced texts below.[9-12]

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(2) Decompensating Phorias

When working with a patient who has a heterophoria, it is important to measure not only the phoria but also fusional vergences to determine if they are adequately compensatory. Exophoria is compensated with positive fusional vergence (PFV); with esophoria, negative fusional vergence (NVF) is the compensating vergence.[1] For hyperdeviations, the compensating reserve is the infravergence of the hyper eye. If the fusional reserves are not able to compensate for the phoria, the patient will become symptomatic or experience reduced binocular vision.

To determine if the compensating fusional vergence is adequate, one may apply Sheard's criterion. This criterion states the compensating vergence should be twice the measured phoria[1]. A second criterion was developed by Percival, who said patients should be functioning in the middle third of their vergence range (Prism needed = 1/3 greater of the two lateral limits – 2/3 lesser of the two lateral limits [where the lateral limits are base-in or base-out])[1].

In symptomatic patients who meet Sheard's criterion, it is important to also analyze recovery values and vergence facility. The recovery value determines a patient's ability to regain single binocular vision.[1] The use of red-green glasses during assessment of vergence ability may reveal reduced values compared to patients with normal binocular vision, particularly when the near point of convergence is tested.[1] Additionally, it can be helpful to measure vergence ranges multiple times using both the step and smooth methods. Having patients blink to break fusion or shake their head to introduce dynamic testing may give more realistic values.[1] If normal break and recovery values are obtained, vergence facility testing with 3^{Δ} base-in/12^{Δ} base-out can be performed to detect issues with vergence latency and velocity.[2]

When both a vertical and horizontal deviation are present, prism correction of the vertical component may have a beneficial effect on the horizontal deviation.[3] If treatment of a patient's horizontal phoria is not proceeding as expected, it may be helpful to re-evaluate for a small vertical deviation that was previously unnoticed. Small amounts of vertical prism can be beneficial for fusion and increase fusional ranges.

Strabismus can occur in otherwise normal adults, or in persons with neurologic or systemic disease, from decompensation of a pre-existing phoria.[4] The strabismus should be comitant – the amount of ocular misalignment should measure the same no matter which eye is fixating a target or which direction of gaze.

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b) Disorders of Vergence

Vergence system abnormalities occur in relatively high frequency in the general population and are the most commonly occurring oculomotor dysfunctions in traumatic brain-injured patients.[1-3] Nuclear and sub-nuclear vergence pathways in the brain are not fully understood, but appear to be susceptible to both localized and diffuse axonal injury.[4] Vergence anomalies known to occur in association with TBI include convergence insufficiency, convergence excess, convergence infacility, divergence insufficiency, and least commonly, divergence excess. Vergence function can be assessed by analyzing the accuracy, speed, and magnitude of the vergence response to accommodative and prismatic stimuli, and by evaluating eye posture at varying focus distances. See the AOA publication "Optometric Clinical Practice Guideline on Care of the Patient with Accommodative and Vergence Dysfunction" for guidance on diagnosis and management of vergence disorders.[5]

The inability to initiate or sustain convergence is the most common vergence anomaly in both TBI and non-TBI populations.[5-6] However, it has been well-established that the prevalence of convergence insufficiency (CI) in the TBI population is markedly higher than in the general population. The majority of published reports of medical record reviews document the prevalence of CI in patients with a history of TBI to be close to 40 percent or higher.[1,3,7,8] Among the available data, there is a similar prevalence between blast and non-blast related TBI's[2] and between civilian and military populations.[1,8,10] This is in contrast to the prevalence in the general non-TBI population, which has been reported to be between 1 percent and 33 percent (the wide range of reported prevalence likely stems from variations in diagnostic criteria) with a median prevalence for both adults and children of about 5 percent.[6]

There is some evidence that the length of time after traumatic insult doesn't strongly influence the presence of TBI-related CI, which suggests that CI associated with TBI is likely to be long-lasting.[7] Symptoms associated with this oculomotor dysfunction, therefore, are likely to persist long after the injury. Importantly, there is evidence that convergence disorders associated with traumatic brain injury are amenable to rehabilitative therapies.[9]

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c) Disorders of Gaze[1-2]

Below is a brief overview for general knowledge of differential diagnosis of gaze disturbances. Congenital causes are not included unless they mimic acquired disease. It is important to note that trauma to the brain and its sequelae can result in almost any ocular motility disorder depending on the part of the brain that is damaged. This does not exclude coexisting disease; therefore, other etiologies are discussed. Some of the diagnoses are complicated beyond the scope of optometry; hence, neuro-ophthalmology or neurology should be consulted.

There are three different types of gaze disturbances: deviations, deficits and vergence dysfunction. Impaired vergence is discussed in the section on binocular vision disorders. In order to make a more exact diagnosis in a patient with gaze disturbance, with possible localization of a lesion, one needs to know if the vestibulo-ocular reflex (VOR) is intact (look for dolls head/oculocephalic movement). Gaze deviations and deficits are bilaterally symmetric and can be divided in two types: those associated with an intact VOR and those associated with an impaired VOR. One other important note is the difference between a deviation and a deficit. In a deviation, the eyes are turned either to the right, left, up or down. In a deficit, the eyes are positioned centrally in their orbits.

(1) Gaze Deviations

Horizontal gaze deviations

Horizontal gaze deviations with an intact VOR most often are localized to a unilateral cerebral hemispheric lesion. Usually the lesion is parietal and the eyes and head are deviated toward the side of the lesion (eg., left parietal lesion, head and eyes deviated to the left). If the lesion is in the right parietal lobe, one can suspect an associated left hemianopic field defect and neglect as well. The patient will also have signs of left hemiparesis. Oculocephalic maneuver may bring the eyes past midline, but typically the larger the lesion the more persistent the deviation.

Horizontal gaze deviations with intact VOR can also be localized to a unilateral thalamic lesion, but the eyes will not have a lateral gaze preference. Thalamic lesions are associated with other neurologic signs such as language and speech impairment, ataxia, quadriparesis, somnolence and ocular presentations of downward gaze deviation and esotropia.

Horizontal deviations with an impaired VOR come from a pontine lesion. The eyes will be turned away from the lesion and toward the hemiparesis. Oculocephalic rotations are non-existent because of damage to the paramedian pontine reticular formation and/or cranial nerve VI nuclei.

Vertical gaze deviations

Vertical deviations are can be upgaze or downgaze and are the only gaze disturbances that are independent of the VOR.

Upgaze deviation can be sustained or ill-sustained. If sustained it may be due to CNS hypoxia or ischemia. If ill-sustained, it may be due to a drug reaction from carbamazepine, phenothiazides or lithium or be psychogenic in nature.

Downgaze deviations are caused most commonly by dorsal midbrain damage. They can also result from orbitopathy as in Graves' disease.

(2) Gaze Deficits

Gaze deficits are of three types: omni-directional, horizontal and vertical. All are dependent on the VOR and are due to structural damage as well as toxic, metabolic or degenerative disease.

Omni-directional gaze deficits

Omni-directional means that the deficit can be horizontal and vertical simultaneously. There are many causes of omni-directional conjugate gaze impairment. Among the causes are: neuromuscular junction, extraocular muscle, cranial nerve, brainstem and cerebral disorders.

If the VOR is preserved, the lesion associated with an acute omni-directional deficit is most likely caused by hypotension or hypoxia and is bilateral affecting the parieto-occipital and frontal areas where pursuits and saccades are generated. In this case, the practitioner will also note an associated oculomotor apraxia: preserved spontaneous saccades, evidence of some voluntary saccadic and pursuit response, yet full oculocephalic excursion. Optic ataxia and simultagnosia may also be associated. This triad, known as Balint's syndrome, is discussed in the section on visual cortical dysfunction. Another area that can produce acute omni-directional gaze deficit is the thalamus affected by vasculopathic, demyelinating or mass effective lesions. As with all thalamic lesions, there will be associated neurologic manifestations. Chronic omni-directional gaze deficits with a preserved VOR are most likely caused by progressive supranuclear ophthalmoplegia, Whipple's, Niemann-Pick, Gaucher's and Creutzfeldt-Jakob-type diseases.

An impaired VOR associated with reduced voluntary ocular motility points to bilateral midbrain and pons structural lesions that can be vasculopathic, demyelinating, mass effective, toxic, and metabolic or CNS degenerative in nature. If CNS signs are absent, the cause may be traumatic extraocular muscle damage. In this case, the defective

eye movements will be asymmetric and the patient will endorse diplopia. If the gaze deficit is chronic, the cause may be due to myasthenia, chronic progressive external ophthalmoplegia, and several dystrophies.

Horizontal gaze deficits

A horizontal gaze deficit with intact VOR can result from an acute unilateral lesion of the ipsilateral parietal or frontal lobe also producing associated contralateral saccadic and pursuit dysfunction (right lesion, right gaze deficit, left saccade and left pursuit dysfunction). The natural history of this deficit shows an ipsilateral gaze deficit that dissipates over days with continued impairment of the contralateral saccades and pursuits. Other causes of a horizontal gaze deficit with preserved VOR are congenital in etiology, but it is important to note they may mimic an omni-directional gaze deficit with viable VOR as described above.

If the VOR is impaired along with a horizontal gaze deficit, the lesion is in the pons. Unilateral versional movements are affected by the ipsilateral pons, but because the midbrain is not involved convergence will be spared. If the medial longitudinal fasciculus is affected, there may be an associated internuclear ophthalmoplegia (INO). An impaired VOR with a horizontal gaze deficit and an INO is called one-and-a-half syndrome.

Vertical gaze deficits

An acute vertical gaze deficit associated with an intact VOR localizes to the thalamus and is usually caused by an infarction. It can also be caused by trauma, hemorrhage, multiple sclerosis, encephalitis and hydrocephalus. Chronic downgaze deficit comes from progressive supranuclear palsy and, like chronic omni-directional gaze deficits with a preserved VOR, are caused by Whipple's, Niemann-Pick, Gaucher's and Creutzfeldt-Jakob-type diseases.

Vertical upgaze palsy with an impaired VOR is caused by damage of the dorsal midbrain. When it is associated with bilateral lid retraction, light-near dissociation, convergence retraction nystagmus and sometimes skew deviation, cranial nerve IV palsy, and convergent retraction, it is called Parinaud's syndrome. If the upgaze deficit is asymmetric it may be because there is asymmetric damage to the supranuclear connections to cranial nerves III. Other diagnoses may need to be ruled out. These include myasthenia and inflammatory and traumatic extraocular myopathy.

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3. Accommodative Disorders

This manual has described four major types of accommodation disorders: insufficiency, infacility, excess, and spasm. All types may be associated with TBI, but the most common type in both TBI and non-TBI populations is accommodative insufficiency, a disorder of the static mechanics of the accommodative system.[1-2] Insufficiency can be identified by measuring amplitude of accommodation that is reduced as compared to age-appropriate values. Static measures of accommodation, such as accommodative amplitude and accommodative interactions, are shown to be reduced in patients with mild TBI.[1-2]

A less common but important accommodative dysfunction in TBI is accommodative infacility, which involves the dynamic aspects of the accommodative response and may be characterized as a deficiency in the latency and speed of accommodation.[3] This impairment of the dynamic flexibility of accommodation often results in symptoms of difficulty changing focus from one distance to another,[4] and diagnosis is generally founded on indirect measures of

power or target distance"[5]. Dynamic measures of dynamic accommodative parameters are shown to be slowed and to exhibit fatigue effects in patients with mild TBI.[1-2] For guidance on the diagnosis and management of accommodation disorders, see the AOA publication "Optometric Clinical Practice Guideline on Care of the Patient with Accommodative and Vergence Dysfunction."[6]

The prevalence of accommodation anomalies in the general population is not well-established. A systematic review of the literature by Cacho-Martinez et al. concluded that despite the range of prevalences reported for the general population, there is a lack of reliable epidemiological studies with unbiased and comparable data on accommodative disorders.[7] However, retrospective review data on rates of accommodative dysfunction in the TBI population support expert consensus that TBI is associated with higher rates of accommodative anomalies than expected in the general population.[8-11] Multiple retrospective studies of military and civilian TBI patient populations reported rates of accommodative dysfunction ranging from 21 percent to 47 percent. [8-11] It is noteworthy that accommodative disorders are more frequently associated with TBI with much higher frequency than with acquired brain injury due to stroke.[10]

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SECTION 3: Review of Assessments and Diagnoses

D. Other Visual Disorders Associated with TBI

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Photography and drawings courtesy of Chrystyna Rakoczy, O.D.

1. Photosensitivity

Photosensitivity, which is broadly defined as intolerance to light, is a very common complaint in brain-injured patients and involves both ocular and non-ocular signs. It often presents as a new, intense, chronic condition that may resolve in a few weeks/months or persist for many years.[1] The photosensitivity can occur despite good ocular health, normal pupil function and visual fields, without defects on neuroimaging, and without any interference from medications or systemic disease. Dry eye and binocular vision dysfunction may exacerbate photosensitivity.[2] Typically, these patients demonstrate increased sensitivity to not only bright illumination, but impaired performance in dim illumination as well.

The mechanism of action is not completely understood, but it is known that the pain is transmitted to the brainstem by the ophthalmic division of the trigeminal nerve.[3] Light sensitivity is also a common symptom in many neurologic disorders, such as meningitis, anterior pathway tumors, thalamic infarcts, migraine, cerebral hypoxia, trigeminal neuralgia and subarachnoid hemorrhage.[1]

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2. Visual fields Deficits

a) TBI Visual Field Deficit in General

Many patients with TBI experience visual field loss from their injuries and can present in various ways. They can vary from small scotomas anywhere in the field, to overall constriction, to congruous or incongruous hemianopias and quadrantonopias, to altitudinal changes, to loss of vision in an entire eye. These defects may be the result of damage anywhere along the visual pathway. A study in 2008 reviewed retrospectively the frequency and types of visual defects in the brain-injured population[1]. Defects were present in 46 percent of the ABI patient sample: 39 percent of the TBI subgroup and in 67 percent of the CVA subgroup[1]. The most frequent defects in the TBI group were scattered (58 percent), followed by homonymous (23 percent)[1]. In the CVA group, the most numerous were homonymous (48 percent), with scattered and non-homonymous accounting for 20 percent each[1]. The high percentage of defects demonstrates that visual field testing is critical in these populations, as well as insuring visual fields are in consideration when evaluating quality of life and developing rehabilitation programs.

Another striking disorder found in brain-injured patients is neglect, or hemi-inattention. With this condition, patients act as if whole regions of space on the side contralateral to their lesions do not exist, even to the extreme where they deny that contralateral limbs are not theirs[4]. As it is most commonly damage to the right hemisphere, it is typically the left side that is neglected. It is important to note that left-sided neglect is different from a left homonymous hemianopia. Patients with neglect will ignore events occurring on the left side, where those with a hemianopia are aware that they are missing events on their left side and can make adjustments[5]. This difference is critical when treatment with yoked or field expanding prisms is considered.

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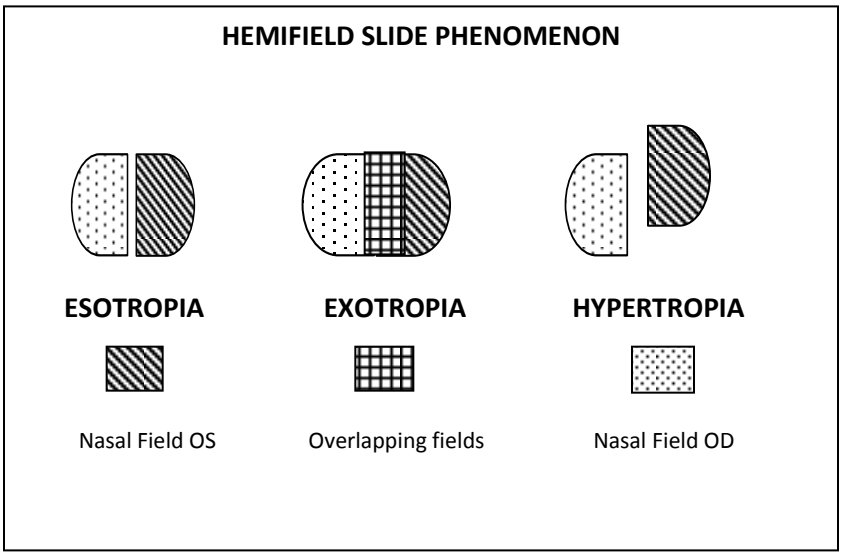
b) Hemi-slide Phenomenon

Lesions of the optic chiasm typically generate a bitemporal hemianopia. The hemi-field slide phenomenon occurs in patients with bi-temporal hemianopic defects who have no overlapping visual fields to link the eyes. This rare condition can result in diplopia, which may be horizontal or vertical in presentation. If it happens with an exodeviation, it can result in horizontal separation of nasal fields, while an exodeviation can cause overlap of nasal fields leading to a non-paretic diplopia.[1] In such cases, a preexisting minor phoria becomes a tropia, thus causing the sensory difficulties.

Hemi-field slide should be suspected in any patient who describes their diplopia as two images that seem to slide in relation to one another.[3] It is possible to test the presence of horizontal deviations by drawing a line of small dots or circles and asking the patient to look quickly at the center of them and report how many are seen.[1]



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3. Cerebral Disorders of Vision

The higher cortical visual areas perform more complex interpretation of the visual information transmitted from the anterior and geniculocalcarine pathways. Damage to these areas is characterized by abnormalities in visual processing or attention, often despite normal visual acuity and visual fields.

a) Occipital Lobe Trauma

(1) Blind Sight (Riddoch Phenomenon)

Blind sight is a subconscious ability to locate light sources and detect moving targets in a field rendered blind by a visual cortex lesion.[1] Patients will verbally claim no conscious awareness of vision, but will avoid obstacles when walking and retrieve objects, thus appearing to have some residual visual functions. Blind sight may be observed during confrontation field testing by comparing responses to moving and still targets.[2]

Blind sight is the result of damage to the occipital lobe, specifically, the primary visual cortex (V1). It is believed patients may have residual vision manifesting from a more primitive visual system, consisting of pathways that bypass V1.[2] Since retino-cortical and retino-mesencephalic visual pathways bifurcate in the posterior optic tract, lesions caudal to that point may spare connections to the midbrain, thalamic, as well as the parietal and temporal lobes. This allows tissues involved in visual functioning to continue working normally, while existing completely dissociated from the dominant stream of verbal consciousness. One example is that pupillary reflexes are spared, which may contribute to the unconscious detection of light.

The most common etiology is a bilateral posterior cerebral artery stroke. Blind sight has been observed in patients with damage to the optic nerves, optic chiasm and retro-chiasmal visual pathway.

(2) Alexia Without Agraphia

Alexia without agraphia is the loss of the ability to read, while retaining the ability to write and speak normally. This condition has also been referred to as "pure alexia" or "word blindness."[2]These patients can spell words aloud, understand them when spoken, and even write words down, but ironically cannot read the words they have written. Numbers, musical notes and familiar symbols may also be unrecognizable.

Alexia without agraphia is caused by lesions affecting the left occipital lobe and ipsilateral splenum of the corpus callosum, most commonly a posterior cerebral artery infarction.[2] The result is a disconnection syndrome with a right homonymous hemianopia, with sparing of key language areas, but a resulting inability to access lexical visual information, which is processed in the right occipital lobe. These findings typically lessen in weeks to months and may disappear completely, except for the hemianopia, which remains.

(3) Cortical Blindness vs. Cerebral Blindness

Cortical blindness is total loss of vision in both eyes from damage to the primary visual cortex (V1).[3] *Cerebral blindness* is blindness to any portion of both visual pathways, posterior to the lateral geniculate neurons[3]. So, by definition, *cortical blindness* is a type of *cerebral blindness*.[3] In both conditions, patients exhibit normal pupillary

functions, normal fundus findings and normal ocular motility, but there is no blink to threat response and no OKN reaction. Because of these findings, it may be mistaken for psychogenic blindness.[4]

The main cause of these conditions is hypoxia or anoxia involving the occipital lobes. Other causes may be basilar artery thrombo-emboli and trauma.

b) Parietal Lobe Trauma

(1) Visual Neglect

Visual neglect is a disabling neurological disorder after unilateral brain damage. It is characterized by a failure to report, respond or orient to novel or meaningful stimuli presented to the contralateral side of the lesion. The condition is multimodal, resulting in loss of auditory, tactile and visual function on the affected side.[6] Patients will act as if the affected side doesn't exist.

Most commonly, damage occurs in the posterior parietal cortex and the temporo-parietal junction. Although it may happen in the left hemisphere, it is believed that true visual neglect occurs in the right parietal lobe. This is because there is redundant visual processing within the right hemisphere, which attends to the left hemifield, as well as some of the right hemifield, making the loss more profound than if the left hemisphere is affected, which does not contain this redundant processing.[2]

In more subtle cases, visual fields may be full to confrontation, but if comparable visual targets are presented simultaneously in both right and left hemifields, the target in the hemifield contralateral to the lesion will be ignored.[5]

Visual neglect goes by many names, including "unilateral spatial inattention," "hemiagnosia," "hemispatial neglect,[3]" "hemiattention" and "unilateral visual inattention."

(2) Anosagnosia

In acute and extreme cases of unilateral neglect, a patient may present with anosagnosia, a condition where the patient will not acknowledge a neurologic deficit or even their own body parts on the side contralateral to a lesion.[3] It can manifest as a failure of awareness of a variety of specific deficits, such as motor (hemiplegia), tactile (hemianesthesia), memory (dementia), language (receptive aphasia) and visual (hemianopia). When the visual system is involved, patients may present with their eyes deviated away from the affected side.

Anosagnosia may result when damage occurs in the non-dominant inferior parietal lobule and/or fronto-temporal-parietal area in the right hemisphere as a result of a cerebral vascular accident or trauma.

(3) Anton's Syndrome

Patients afflicted with Anton's syndrome will deny, at times emphatically, or downplay the extent of their blindness and may even fabricate an imaginary visual environment as an excuse for why they cannot do visually guided tasks.[1] This may be the result of the language control area becoming disconnected and receiving no information regarding the visual status of the brain and, thus, forcing the language center to fill in the gaps and discrepancies in available information by creating an explanation internally as to why a person cannot see.[6] It is found in a small minority of patients with bilateral primary cortex damage following posterior cerebral artery occlusion.

There appear to be three mechanisms that underlie Anton's syndrome:

1. Disruption of the occipital-parietal connections (resulting in unawareness of visual loss)
2. Disruption of the occipital-temporal connections (forgetting or not caring about loss)

3. Disruption of occipital-frontal connections (not being able to keep track of loss)

(4) Optic Ataxia

Optic ataxia, or visuomotor ataxia, is a visually guided inaccurate reaching for objects in extra personal space. Patients reach toward targets at fixation and in the peripheral, as if blind, although they can see and describe the objects they are attempting to reach. This is not associated with any cerebellar dysfunction or motor weakness.[2] In order to reach and grasp external objects, required coordination of several different nervous system functions needs to occur: the brain transforms a target's visual coordinates to body-centered space, plans a trajectory and computes multiple joint torques.[3] This erroneous dynamic spatial processing of proprioceptive information from the hand may be due to an inability to convert visual field coordinates in head-centered spatial coordinates. Alternatively, information from the visual cortex in the occipital lobe may never reach the motor cortex in the frontal lobe. Optic ataxia is caused by bilateral posterior parietal lesions, often the result of an infarct in the parieto-occipital branch of the posterior cerebral artery.

(5) Oculomotor Apraxia

Bilateral parieto-occipital, acute frontal and/or fronto-parietal lesions may cause the eyes to lose the ability to produce voluntary eye movements (pursuits and saccades), which is known as oculomotor apraxia. Reflexive eye movements remain, including reflex saccades, VOR and the quick phases of nystagmus. Patients may use a head thrust, therefore, utilizing the intact VOR to maintain fixation.[2] In acquired oculomotor apraxia, both the horizontal and vertical eye movements may be affected, which differs from the congenital variety in which only horizontal is affected.[3]

Because of the disconnection of the occipital lobe from the frontal eye fields, it appears as if the eyes are unable to move, which is why oculomotor apraxia has been called "spasm of fixation[2]" and "psychic gaze paralysis."

(6) Abnormal Egocentric Localization

Localization is how humans get information to know where objects are in relation to themselves in space and includes "oculocentric" and "egocentric."[7]Oculocentric localization uses the fovea as the center of the spatial coordination system to localize objects in the environment. In contrast to oculocentric, egocentric localization refers to basing the spatial coordinate center on the trunk along the body midline as the reference point, or egocenter.[7]

In abnormal egocentric localization, or visual midline shift syndrome, damage to the right posterior parietal complex or intraparietal sulcus results in visual neglect and individuals may have an anomalous shift (up to 15 degrees) in their subjective sense of straight ahead.[8] There is essentially a mismatch between their subjective and objective visual spaces. These patients may report feeling "unsteady" or "not as grounded" as this systemic directional error involves disrupted connections between the visual, vestibular and proprioceptive inputs.[8]

c) Temporal Lobe Trauma

(1) Visual Object Agnosia

Visual object agnosia is the inability to recognize familiar objects despite adequate visual perception, attention, intellect and language.

Aperceptive visual agnosia—patients with this condition who fail to recognize objects are unable to copy line drawings or match drawings to samples. This deficit of shape and form perception is believed to rest on a failure to perform adequate feature analysis. Appreciative visual agnosia may be caused by trauma, stroke or hypoperfusion occurring in the temporal lobes and/or temporal-occipital cortices bilaterally. It may also be seen in Alzheimer's disease and with carbon monoxide or mercury poisoning.[1]

Associative visual agnosia—some patients, who despite relatively normal vision and apparently normal copying and matching abilities cannot visually recognize common objects, are said to have associative visual agnosia.[1] It appears these patients can copy, but do so by copying just a little part of the object at a time because they can only compare individual features, not the whole target. Lesions are similar in nature to those causing aperceptive visual agnosia and occur in the occipito-temporal region.[2] Usually they are found to be bilateral, but have been seen unilaterally, with a predominance of the left hemisphere.

d) Occipito-Temporal Trauma

(1) Cerebral Achromatopsia

Posterior cerebral artery infarctions damaging the inferior occipital lobe, or the connections between the occipital and temporal lobes, may result in a very rare inability to sort and match colors. It can range from a loss of color brightness to a complete inability to perceive color. This condition may occur in the full visual field if the lesion is bilateral, half the field (most common) or even just parts of the visual field, depending on the extent of the damage. When the full field version occurs, it is often in the setting of a developing or receding hemianopia or resolving cortical blindness.[3] With the bilateral presentation, patients are likely to describe their color deficit as a "graying" or "washing out" of vision, such as switching a color TV into a black and white one.[5] This visual color dysfunction needs to be differentiated from color anomia, where patients cannot name colors, but are able to match hues on testing.

(2) Visual Agnosia

Prosopagnosia— the inability to recognize familiar faces, including one's own, as well as the inability to learn to recognize faces. It may also cause people to be unable to recognize familiar things from each other (e.g., models of cars, species of birds) or get lost in places with which they are accustomed.[1] Usually bilateral damage to the occipito-temporal region is the etiology, although it has been observed in unilateral right-sided lesions, making it seem there is a right cerebral dominance for facial recognition.[2] Interestingly, prosopagnosia has been found without any other associated visual agnosia, suggesting that facial recognition is a modular specific task of the human brain.

Optic aphasia—the inability to name common objects by sight, despite being able to name them by touch or sound. Normally, it is the result of a posterior cerebral artery infarction, damaging the left occipito-temporal junction. It is a visual-verbal disconnection, like other visual agnosias, but patients with optic aphasia (or visual anomia) may have better recognizing capabilities secondary to relatively preserved access to the right perceptual and semantic systems.[5]

(3) Cerebral Akinetopsia

Cerebral akinetopsia, or defective motion perception, is an uncommon complaint and occurs as a result of bilateral damage to the occipto-temporo-parietal junction (V5).[2] V5 is the motion detection center of the brain and, thus, when it is disconnected from the rest of the visual cortex cannot transmit motion information properly. Patients can see stationary objects normally, but do not see moving objects. So, for example, if a person is standing on a sidewalk trying to decide if it is safe to cross the street, there might appear to be a truck off to the person's left and then a moment later, that truck would appear to be directly in front of the person and then, moments later, off to that person's right. Although the truck would have traveled through all those points, a person with cerebral akinetopsia would perceive the truck appeared at these spots sequentially, but have no sense of the truck moving as it went from one point to another.[6] In another example, if a person were pouring liquid into a cup, it would appear as if the liquid was frozen, like a glacier.

e) Parieto-Temporal Trauma

(1) Simultagnosia

Simultagnosia is the inability to detect more than one object at a time when multiple objects are displayed simultaneously, sort of spotlight-like vision. Infarction of the parieto-occipital branch of the posterior cerebral artery supplying the posterior parietal lobe is the main etiology.[2] It was originally called visual disorientation and is the most common element seen in isolation in Balint's syndrome.[2]

f) Parieto-Occipital Trauma

(1) Balint's Syndrome

Balint's syndrome may occur as a result of damage to both posterior parietal, dorsal occipital and/or occipito-parietal regions. In its complete form, Balint's consists of four components:[1]

1. Bilateral visual inattention
2. Ocular motor apraxia
3. Optic ataxia
4. Impaired spatial relations

More minor forms may consist of some combination of the elements above. Balint's syndrome is a variety of combined deficits from lesions of the dorsolateral visual association cortices, including projections to the occipito-parietal cortex.[3] Damage to these areas can disturb multiple aspects of spatial and temporal processing, including perception of visual motion, dynamic stereopsis, self-movement, and coordination of the visual and vestibular systems. These patients may appear disabled and almost completely blind (there is often macular sparing). They bump into objects, often requiring assistance when walking; they do not fixate on novel stimuli and may become focused on single targets.

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4. Visual Illusions and Hallucinations Associated with TBI

At times, patients with TBI may present with symptoms of unusual visual phenomena. It is best to ask the patient to describe what they see in as much detail regarding content and complexity as possible. The patient may even be able to draw what they see or tell you if the phenomenon is monocular or binocular. The practitioner should also ask about associated symptoms such as headache or limb twitching. The patient may know the phenomenon is not real. Schizophrenic and psychotic patients will not be able to tell the difference. Other predisposing conditions such as drug abuse may also be an underlying cause. The ocular health examination will rule out ocular disease as a cause of these phenomena. Visual fields should be checked carefully.[4]

- Positive phenomena:
  - Illusions: (disappear when eyes are closed)
  - Hallucinations: (appear when eyes are open or closed)
- Negative Phenomena:
  - Acuity loss
  - Visual field loss

There are two basic types of visual phenomena/disturbances: positive and negative. Negative phenomena include loss of acuity and visual field defects. Positive phenomena include illusions and hallucinations. The positive phenomena come about from either focal or diffuse damage to the brain. According to Burde and Savino,[1] there are only two phenomena caused by focal lesions of the brain: sparkling lights with zigzags due to contralateral visual cortex disruption and illusions confined to one hemifield caused by a disturbance in the contralateral vision associated areas.[1] All other positive phenomena are cause by more diffuse damage to the cerebral visual areas.

a) Illusions

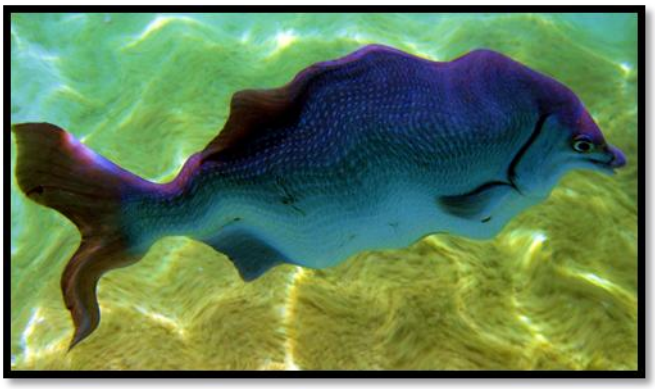
Visual illusions disappear when eyes are closed. They can be caused by optical abnormalities (cataracts, poor refractions, and dislocated lenses) as well as neural pathway disturbances associated with TBI (dysfunction of the visual association cortex, photoreceptor, oculomotor and vestibulo-ocular dysfunction). The neural phenomena are caused by diffuse unilateral occipito-parieto-temporal lesions that interfere with normal visual sensory input.
All are binocular.

(1) Palinopsia

Palinopsia is also known as visual perseveration where a previously seen image is superimposed on a current image viewed either immediately or hours later.[1, 4] Palinopsia is usually associated with right inferior occipito-temporal cortex lesions [1, 2] and right parieto-occipital damage [4] once psychiatric conditions, drug abuse and intoxication have been ruled out.[2] Left lesions may also cause palinopsia, but associated aphasia may mask the presentation.[4]
There are two variants: immediate persistence of an image that has just disappeared and delayed persistence, where the image of an object seen reappears hours later and repeats for days. The image can stay in the same retinal area and will move as the eye moves. Associated findings include homonymous hemianopias that are complete or incomplete, quadrantonopias, macropsias and micropsias.[2] In the photo below, the patient persists in "seeing" a previously viewed fish, obviously out of context.

(2) Cerebral Metamorphopsia

Cerebral metamorphopsia presents as an alteration in shape or elongation of images that may persist for several weeks and is often accompanied by palinopsia.[1] Below is a photo of a fish as it may appear to a patient with cerebral metamorphopsia, similar that which would be seen by a patient with an epiretinal membrane or retinal pucker.



(3) Cerebral Diplopia

Cerebral diplopia is also known as visual allesthesia or altered image position: a patient with partial hemifield defects will see an image repeated in the defective field.[1, 2] This illusion is sometimes seen during epileptic seizures and right lesions predominate.[3] The photo below depicts cerebral diplopia in a patient with a right field defect.

(4) Cerebral Polyopia

Cerebral polyopia or altered motion, which involves moving images with "tails" (think of diplopia but multiple smudged images), is not as common as palinopsia. It is noted with parietal lobe and parieto-occipital CVAs[2], can occur with MS, encephalitis, CVAs and is associated with cerebral achromatopsia, homonymous field defects, and object agnosia.[2,3] The fish below appears to have multiple tails as it swims to the left.

b) Hallucinations

Visual hallucinations have no external stimuli[2, 4] and persist when eyes are open or closed[1]. They are neural disturbances caused by retina, optic nerve and vision-associated areas of the occipital, parietal and temporal lobes. Those associated with occipito-parieto-temporal lesions will persist, are binocular and unformed (circles, squares, lines). Those associated with vertebro-basilar transient ischemic attacks are formed (snowflakes, dogs, sparkles) but last only seconds. Longer hallucinations may show an occipital infarction.

Hallucinations in persons with neurologic conditions are of three types: release, visual seizures and migrainous.[4]

(1) Release

Charles Bonnet Syndrome: hallucinations can occur with visual loss of any type where the acuity in the better eye is 20/60 or worse. It has been proposed that the images are "released" from a visual cortex that is no longer receiving input. Most of these patients are aware the hallucinations are not real. The images can be simple (flashes or points of light) or complex (recognizable objects or images from the past).[2] They can occur with the eyes open, but are typically more intense with the eyes closed.[4] Charles Bonnet-like hallucinations are more common in patients who are older and socially isolated.[2] This syndrome has been compared to the phantom-limb phenomenon, also a release hallucination.

(2) Visual Seizures

Visual seizures are associated with occipital seizures and are accompanied by head or eye deviation and rapid blinking as well as dysphasia, confusion and tonic-clonic limb movement.[2, 3, 4] Patients with these seizures report flashing and steady white and colored lights, stars, wheels or triangles.[4]

(3) Migranous Hallucinations

Migranous hallucinations can occur with and without a headache. They are often confused with ictal hallucinations. Migranous phenomena present as black and white flickering zigzag lines, whereas ictal phenomena present with colored circular patterns.[2, 3]

Migranous hallucination

Ictal hallucination

c) Other phenomena

(1) Altered Color Perception

Altered color perception caused by an inferior occipital lobe lesion may show superior altitudinal achromatopsia. In the photo depiction of altered color perception below, the upper half of the image is washed out.

(2) Altered Spatial Perception

Peduncular hallucinations are caused by an infarct or mass compression of the midbrain. These patients present with hallucinations of vivid life-like images of concrete objects. CNIII dysfunction, ataxia cognitive issues and sleep disorder are frequent companions.[4]

Dysmetria and Upside-Down Vision. Dysmetria is a type of ataxia, characterized by an inability to judge distances in space, which causes under- or over-shooting of intended targets with the hands, legs or eyes. It is caused by bilateral parietal-occipital lesions. Dysmetria with upside-down vision is a condition that can occur if there is an infarct in the middle portion of the posterior cerebellar artery supplying the sensorimotor integration area in the cerebellum.[5] This occurs because the pathways connecting the sensory information (which relay information on the eyes' position) to the area of motor control are damaged[5]. Tilted or upside-down vision caused by infarct of the dorso-lateral medulla (Wallenburg's syndrome) is associated with vestibulopathy.[4] This visual effect will be associated with other signs of vestibular dysfunction, such as skew deviation and ocular tilt or torsion.

(3) Altered Motion

The Pulfrich Effect is a visual condition where the lateral motion of an object is interpreted by the visual cortex as having a depth component. A delay in conduction of impulses between the two eyes caused by unilateral or asymmetric optic neuropathy, or any other condition that affects one optic nerve more than the other,[2] can cause this effect. A pendulum swinging back and forth on a plane will appear as if it is taking an elliptical path.[1]

Oscillopsia is the optic illusion that stationary objects are moving back and forth or up and down. It ranges from a mild blurring of vision to a rapid jumping of vision. Oscillopsia may occur when there is damage to the vestibular system, brainstem or cerebellum.[6] It can also be found with acquired spontaneous nystagmus or impaired coordination of the visual cortex.[6] It may be transient, as in the case of a unilateral peripheral vestibular lesion, or permanent, if there is damage to the central vestibular system or cerebellum[7]. Oscillopsia can also be seen by

patients with VOR dysfunction and can be associated with and large amplitude nystagmus[1]. Often patients with small amplitude nystagmus also experience oscillopsia.

(4) Altered Size

Accommodative-convergence micropsia is a normal physiologic phenomenon when a distant object appears smaller as one focuses up close.

Cerebral micropsia, macropsia and metamorphopsia (Alice in Wonderland syndrome) are associated with migraines, occipital tumors and seizures respectively.[2, 3, 4] In the picture below, note the disproportionately large legs as compared with the small head of the rooster.

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5. Visual Disorders Associated with Vestibular Dysfunction: Skew Deviation

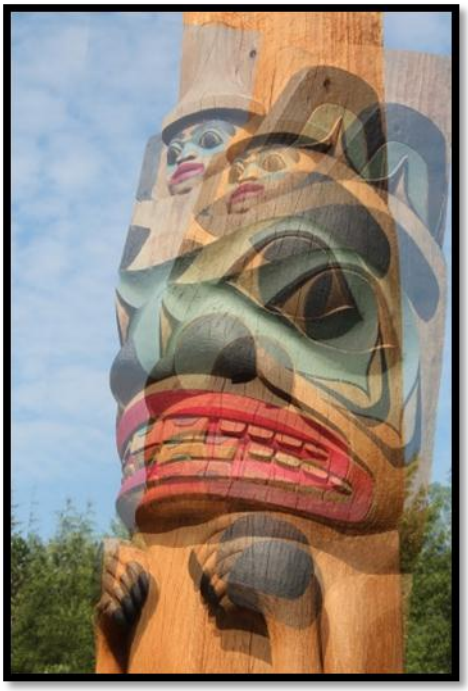
A skew deviation is a transient vertical misalignment caused by damage to the vestibular system, specifically the otolith-ocular pathways in the cerebellum, midbrain or otolith end organ. It presents as a vertical misalignment with excyclotorsion of the ***hypo*** eye. There are many variants of skew deviation, and it can mimic a decompensating CN

IV or palsy; therefore diagnosing a skew can be confusing. One rule, when speaking of skew, is to reference the ***hypo*** eye and not the hyper eye as practitioners usually do.

The hypotropia/phoria can be contralateral to a midbrain lesion (contraversive skew), ipsilateral to a lesion of the lower pons or medulla (ipsiversive skew), and alternating with cerebellar lesions. Misalignments are almost always not comitant. Brainstem skews endure, whereas peripheral vestibular skews resolve in months.

In alternating skew the ***hypo***tropia changes on lateral gaze, with the ***hypo***tropic eye on adduction mimicking a compromised superior oblique. Measuring cyclotorsion will help differentiate the two entities. In a skew, the ***hypo*** eye excyclotorts; whereas in a superior oblique palsy, the hyper eye excyclotorts. In addition, the skew is often accompanied by neurological findings such as ataxia.

The ocular tilt reaction is a triad of ocular torsion, head tilt and hypotropia all to the same side. Ocular tilt can be tonic or phasic. Both can be treated with mild vertical prism, but the phasic ocular tilt may also respond to baclofen.



A patient with skew deviation will see one image that is straight and the other tilted.

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SECTION 4: The Interprofessional Rehabilitation Team for the Brain-Injured Population

Brenda Heinke Montecalvo, O.D., and Chrystyna Rakoczy, O.D.

Rehabilitation requires the collaboration of an interprofessional team of health providers. Research shows patients with TBI receiving interprofessional rehabilitation demonstrate significant gains and maintain the treatment effect after rehabilitation ends.[1] The goals of rehabilitation are to improve cognitive and physical function and modify behavior in an effort to reintegrate the individual into productive family and community life. Successful rehabilitation often engages many disciplines, including physiatry, physical therapy, occupational therapy, audiology, neuropsychology, psychology, and optometry.[2] All work together, with the patient, to improve the rehabilitation outcomes. In complex traumatic brain injury, interprofessional teams can use time more efficiently, coordinate services, integrate health care for a variety of needs, and empower patients as active partners in care.[3] Further, an interprofessional team is able to integrate and synthesize knowledge and contributions of each discipline, resulting in comprehensive understanding and approaches that are more than the sum of the individual parts.[4]

The following is a description of health care professionals who are involved in TBI rehabilitation. Optometrists serving the TBI population will collaborate with many of these individuals. Generally, the optometrist and the occupational therapist (OT) will have the closest relationship. Patients with TBI present with all levels of cognitive, multi-sensory and physical deficits that can and should be addressed during their overall rehabilitation program. The OD/OT relationship ensures all of the patient's rehabilitation therapists and their caregivers are aware of the visual deficits and visual needs specific for each patient. It also facilitates the potential for co-managing the visual rehabilitation needs of patients with BI. Although TBI OTs are well grounded in how vision and visual processing is affected by TBI and its importance to the overall rehabilitative process, they do not have the body of knowledge an optometrist has about the complexities of the ocular motor and visual processing system. OTs should always work with a knowledgeable OD in the rehabilitation of visual dysfunction secondary to brain injury.

Audiologist – One who evaluates hearing defects and aids in the rehabilitation of those with such defects.

Cognitive Therapist – Help the patient overcome difficulties by identifying and changing dysfunctional thinking, behavior, and emotional responses. This involves helping patients develop skills for modifying beliefs, identifying distorted thinking, relating to others in different ways, and changing behaviors. Treatment is based on collaboration between patient and therapist and on testing beliefs. Therapy may consist of testing the assumptions one makes and identifying how certain unquestioned thoughts are distorted, unrealistic and unhelpful.

Dietician Counselor – Assess and provide for the nutritional needs of the patient with a disability. They assist the physician and provide a treatment plan that may include the following: ideal body weight, caloric, and dietary needs, foods that help facilitate swallowing, special diets, dietary modifications that need to be made as a result of the disability, tube-feeding formulas and schedules for patients unable to swallow, and patient and family education on nutrition topics related to diseases.

Neurologist – A physician who specializes in the nervous system and its disorders.

Neuropsychologist – A psychologist who specializes in evaluating (by tests) brain/behavior relationships, planning training programs to help the survivor of brain injury return to normal functioning and recommending alternative cognitive and behavioral strategies to minimize the effects of brain injury. Often works closely with schools and

employers as well as with family members of the injured person.

Occupational Therapy – Occupational therapy is the therapeutic use of self-care, work and play activities to increase independent function, enhance development and prevent disability; may include the adaptation of a task or the environment to achieve maximum independence and to enhance the quality of life. The term occupation, as used in occupational therapy, refers to any activity engaged in for evaluating, specifying and treating problems interfering with functional performance.

Optometrist – A person with doctorate-level education trained and licensed by examination to diagnose and treat the eyes for disease, visual defects, impairments by prescribing medication and or corrective lenses or providing minor eye surgery, visual enhancement, neuro-optometric rehabilitation and optometric vision therapy.

Orientation and Mobility Specialist – Teach the concepts and skills necessary for visually impaired to travel safely and efficiently in their environments. Orientation skills enable people with visual impairments to use sensory information to know their location in different settings, and mobility skills enable them to travel in different areas. People travel to different destinations using different techniques (such as the sighted guide or trailing techniques) or by using assistive devices (such as the long cane and the wheelchair).

Physiatrist – (pronounced fizz ee at' rist) **–** A physician who specializes in physical medicine and rehabilitation. Some physiatrists are experts in neurologic rehabilitation, trained to diagnose and treat disabling conditions. The physiatrist examines the patient to assure that medical issues are addressed and provides appropriate medical information to the patient, family members and members of the treatment team. The physiatrist follows the patient closely throughout treatment and oversees the patient's rehabilitation program.

Physical Therapist – The physical therapist evaluates components of movement, including muscle strength, muscle tone, posture, coordination, endurance, and general mobility. The physical therapist also evaluates the potential for functional movement, such as ability to move in the bed, transfers and walking, and then proceeds to establish an individualized treatment program to help the patient achieve functional independence.

Psychologist – A professional specializing in counseling, including adjustment to disability. Psychologists use tests to identify personality and cognitive functioning. This information is shared with team members to assure consistency in approaches. The psychologist may provide individual or group psychotherapy for the purpose of cognitive retraining, management of behavior and the development of coping skills by the patient/client and members of the family.

Recreation Therapist – Individual within the facility responsible for developing a program to assist persons with disabilities to plan and manage their leisure activities; may also schedule specific activities and coordinate the program with existing community resources.

Rehabilitation Counselor – Also called a vocational counselor. A specialist in social and vocational issues who helps the patient develop the skills and aptitudes necessary for return to productive activity and the community.

Rehabilitation Facility – Agency of multiple, coordinated services designed to minimize for the individual the disabling effects of one's physical, mental, social, and/or vocational difficulties and to help realize individual potential.

Rehabilitation Nurse – A nurse specializing in rehabilitation techniques as well as basic nursing care. Nurses assist the patient and family in acquiring new information, developing skills, achieving competence and exhibiting behaviors that contribute to the attainment of a healthy state.

Speech-language Pathology Services – A continuum of services including prevention, identification, diagnosis, consultation, and treatment of patients regarding speech, language, oral and pharyngeal sensorimotor function.

Social Worker – A professional discipline that seeks to improve the quality of life and well-being of an individual, group, or community by intervening through research, policy, community organizing, direct practice, and teaching on behalf of those affected by poverty or any real or perceived social injustices and violations of their human rights.

Vocational Rehabilitation – Helping those who have disabilities or recovering from them reach their full potential and return to work through patient evaluation and testing, job analysis, patient counseling and understanding related laws.

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SECTION 5: Glossary

Brenda Heinke Montecalvo, O.D.

A comprehensive list of terms and their definitions used in the area of neuro-rehabilitation are listed below. Underlined terms are more common when working with the vision and eye systems of patients with brain injury. The Brain Injury Glossary is provided to assist optometrists in better communicating with and reporting to the various individuals on the brain-injured rehabilitation team.

Abnormal egocentric localization: see egocentric processing dysfunction

Abnormal posturing: an involuntary flexion or extension of arms or legs. This may be caused by TBI as well as stroke, mass effect, vasculopathy and encephalopathy. There are three types: **decorticate –** arms over chest; **decerebrate –** arms extended at sides; **opisthotonus –** head and back are arched backward.

Abstract concept: An idea not related to any specific instance or object and that potentially can be applied to many different situations or objects. Persons with cognitive deficits often have difficulty understanding abstract concepts.

Abstract thinking: Being able to apply abstract concepts to new situations and surroundings.

Abulia: Absence or inability to exercise will-power or to make decisions. Also, slow reaction, lack of spontaneity, and brief spoken responses. It is usually associated with damage to a cerebellar vessel.

Acalculia: The inability to perform simple problems of arithmetic.

Accommodative insufficiency: The inability to sustain focus on near objects in patients younger than 40. Older than 40, the same condition is considered a normal part of the aging process and is called presbyopia. Younger than 40, this condition is very treatable.

Accommodative spasm: A condition in which the ciliary muscle of the eye remains in a constant state of contraction. The ciliary muscle cannot relax when viewing distant objects, causing vision to blur when attempting to view objects from a distance.

Acquired brain injury: see brain injury

Acute care: The phase of managing health problems as conducted in a hospital for patients needing medical attention.

Acute rehabilitation program: Primary emphasis is on the early phase of rehabilitation that usually begins as soon as the patient is medically stable. The program is designed to be comprehensive and based in a medical facility with a typical length of stay of one to three months. Treatment is provided by an identifiable team in a designated unit. See Program/Service Types.

ADL: Activities of daily living. Routine activities carried out for personal hygiene and health (including bathing, dressing, feeding) and for operating a household.

Adaptive/assistive equipment: A special device that assists in the performance of self-care, work or play/leisure activities or physical exercise. *See also adaptive equipment catalog.*

Affect: The observable emotional condition of an individual at any given time. *See also* **frontal lobe.**

Agnosia: Failure to recognize familiar objects although the sensory mechanism is intact. May occur for any sensory modality.

Agraphia: Inability to express thoughts in writing.

Alexia: Inability to read.

Allocentric view: How the individual relates objects to other objects in a multifaceted way. The points that represent object locations in Cartesian space relate to X, Y and Z co-ordinates and to other objects in that space. Locating objects within a framework external to the holder of the representation and independent of his or her position.

Alpha wave: Neural oscillations in the frequency range of 8 to 13 Hz, which is measured by electroencephalography (EEG) and predominantly originate from the occipital lobe during wakeful relaxation with closed eyes. Alpha waves are reduced with open eyes, drowsiness and sleep. Alpha waves do not start to appear until age 3.

Altitudinal visual field defect: A visual field defect in which either the upper or lower half of the visual field is selectively affected. The selective abnormality often creates a horizontal line across the visual field (known as "respecting the horizontal meridian").

Ambulate: To walk.

Amnesia: Lack of memory about events occurring during a particular period of time. See also: anterograde amnesia, retrograde amnesia, post-traumatic amnesia.

Aneurysm: A balloon-like deformity in the wall of a blood vessel. The wall weakens as the balloon grows larger and may eventually burst, causing a hemorrhage.

Aniseikonia: an ocular condition where there is a significant difference in the perceived size of images. It can occur as an overall difference between the two eyes, or as a difference in a particular meridian.

Anomia: Inability to recall names of objects. Persons with this problem often can speak fluently but have to use other words to describe familiar objects.

Anosmia: Loss of the sense of smell.

Anoxia: A lack of oxygen. Cells of the brain need oxygen to stay alive. When blood flow to the brain is reduced or when oxygen in the blood is too low, brain cells are damaged.

Anterograde amnesia: Inability to consolidate information about ongoing events. Difficulty with new learning.

Aphasia: Loss of the ability to express oneself and/or to understand language. It is caused by damage to brain cells rather than deficits in speech or hearing organs.

Apoxia: Lack of oxygen due to being in high altitudes.

Apraxia: Inability to carry out a complex or skilled movement; not due to paralysis, sensory changes, or deficiencies in understanding.

Aprosodia: A condition in which there is a loss of production or comprehension of the meaning of different tones of voice.

Arousal: Being awake. Primitive state of alertness managed by the reticular activating system (extending from medulla to the thalamus in the core of the brain stem) activating the cortex. Cognition is not possible without some degree of arousal. *See also* **brain stem**.

Articulation: Movement of the lips, tongue, teeth and palate into specific patterns for purposes of speech. Also, a movable joint.

Aspiration: When fluid or food enters the lungs through the wind pipe; can cause a lung infection or pneumonia.

Astereognosia: Inability to recognize things by touch.

Ataxia: A problem of muscle coordination not due to apraxia, weakness, rigidity, spasticity or sensory loss. It is caused by lesion of the cerebellum or basal ganglia. Can interfere with a person's ability to walk, talk, eat, and to perform other self-care tasks.

Atrophy: A wasting away or decrease in size of a cell, tissue, organ, or part of the body caused by lack of nourishment, inactivity or loss of nerve supply.

Attention/concentration: The ability to focus on a given task or set of stimuli for an appropriate period of time.

Augmentative and alternative communication: Use of forms of communication other than speaking such as: sign language, yes/no signals, gestures, picture board, and computerized speech systems to compensate (either temporarily or permanently) for severe expressive communication disorders.

Autism: A disorder of neural development characterized by impaired social interaction and communication and by restricted and repetitive behavior. These signs all begin before a child is 3. Autism affects information processing in the brain by altering how nerve cells and their synapses connect and organize.

Balance: The ability to use appropriate righting and equilibrium reactions to maintain an upright position. It is usually tested in sitting and standing positions.

Behavior: The total collection of actions and reactions exhibited by a person. *See also* "Working with Behavior Disorders."

Bilateral: Pertaining to both right and left sides.

Binocular vision dysfunction: A sensorimotor anomaly characterized by the inability to efficiently, accurately and comfortably sustain eye teaming.

Brain injury, acquired: The implication of this term is that the individual experienced normal growth and

development from conception through birth, until sustaining an insult to the brain at some later time that resulted in impairment of brain function.

Brain injury, closed: Occurs when the head accelerates and then rapidly decelerates or collides with another object (for example, the windshield of a car) and brain tissue is damaged, not by the presence of a foreign object within the brain, but by violent smashing, stretching, and twisting of brain tissue. Closed brain injuries typically cause diffuse tissue damage that results in disabilities that are generalized and highly variable.

Brain injury, mild traumatic: A patient with a mild traumatic brain injury is a person who has had a traumatically induced physiological disruption of brain function, as manifested by at least one of the following:

- loss of consciousness (LOC) of 30 minutes or less
- alteration of consciousness (AOC) less than 24 hours
- post-traumatic amnesia (PTA) less than 24 hours
- an initial Glasgow Coma Scale score of 13 to 15 after 30 minutes

Brain injury, traumatic: Damage to living brain tissue caused by an external, mechanical force. It is usually characterized by a period of altered consciousness (amnesia or coma) that can be very brief (minutes) or very long (months/indefinitely). The specific disabling condition(s) may be orthopedic, visual, aural, neurologic, perceptive/cognitive, or mental/emotional in nature. The term does not include brain injuries caused by insufficient blood supply, toxic substances, malignancy, disease-producing organisms, congenital disorders, birth trauma or degenerative processes.

- Any period of loss of or a decreased level of consciousness (LOC)
- Any loss of memory for events immediately before or after the injury. PTA is the time interval from when the person regains consciousness until he or she is able to consistently form memories of ongoing events.
- Any alteration in mental status at the time of the injury (AOC)
- Any neurological deficits that may or may not be transient

Brain plasticity: The ability of intact neurons to take over functions of damaged cells; plasticity diminishes with maturation.

Brain scan: An imaging technique in which a radioactive dye (radionuclide) is injected into the blood stream and then pictures of the brain are taken to detect tumors, hemorrhages, blood clots, abscesses or abnormal anatomy.

Brain stem: The lower extension of the brain where it connects to the spinal cord. Neurological functions located in the brain stem include those necessary for survival (breathing, heart rate) and for arousal (being awake and alert).

Case management: Facilitating the access of a patient to appropriate medical, rehabilitation and support programs, and coordination of the delivery of services. This role may involve liaison with various professionals and agencies, advocacy on behalf of the patient, and arranging for purchase of services where no appropriate programs are available.

Cerebellar syndrome: Characteristic features include a tendency for limb movements to overshoot or undershoot a target, a tremor that occurs during attempted movements, impaired force and rhythm of rapidly alternating movements, and gait ataxia.

Cerebellum: The portion of the brain (located at the back) that helps coordinate movement. Damage may result in ataxia.

Cerebral-spinal fluid (CSF): Liquid that fills the ventricles of the brain and surrounds the brain and spinal cord.

Cerebrovascular accident: The sudden death of some brain cells due to lack of oxygen when the blood flow to the brain is impaired by blockage or rupture of an artery to the brain.

Central scotoma: An area of depressed vision corresponding with the point of fixation and interfering with straight-ahead vision.

Circumlocution: Use of other words to describe a specific word or idea that cannot be remembered.

Chronic: Marked by long duration or frequent recurrence.

Clonus: A sustained series of rhythmic jerks following quick stretch of a muscle.

Closed brain injury: see brain injury

Cognition: The conscious process of knowing or being aware of thoughts or perceptions, including understanding and reasoning.

Coma: A state of unconsciousness from which the patient cannot be awakened or aroused, even by powerful stimulation; lack of any response to one's environment. Defined clinically as an inability to follow a one-step command consistently; Glasgow Coma Scale score of 8 or less.

Communicative disorder: An impairment in the ability to 1) receive and/or process a symbol system, 2) represent concepts or symbol systems, and/or 3) transmit and use symbol systems. The impairment may be observed in disorders of hearing, language, and/or speech processes.

Community skills: Those abilities needed to function independently in the community. They may include: telephone skills, money management, pedestrian skills, use of public transportation, meal planning and cooking.

Comprehension: Understanding of spoken, written, or gestural communication.

Concentration: Maintaining attention on a task over a period of time; remaining attentive and not easily diverted.

Concrete thinking: A style of thinking in which the individual sees each situation as unique and is unable to generalize from the similarities between situations. Language and perceptions are interpreted literally so that a proverb such as "a stitch in time saves nine" cannot be readily grasped.

Concussion: see brain injury, mild traumatic

Confabulation: Verbalizations about people, places, and events with no basis in reality. May be a detailed account delivered.

Confusion: A state in which a person is bewildered, perplexed, or unable to self-orient.

Conjugate movement: Both eyes move simultaneously in the same direction. Convergence of the eyes toward the midline (crossed eyes) is a disconjugate movement.

Convergence: Movement of two eyeballs inward to focus on an object moved closer. The nearer the object, the greater is the degree of convergence necessary to maintain single vision.

Convergence insufficiency: A sensory and neuromuscular anomaly of the binocular vision system, characterized by an inability of the eyes to turn toward each other, or sustain convergence.

Cortical blindness: Loss of vision resulting from a lesion of the primary visual areas of the occipital lobe. Light reflex is preserved.

Computerized Tomography scan/computerized axial tomography: A series of X-rays taken at different levels of the brain that allows the direct visualization of the skull and intracranial structures. A scan is often taken soon after the injury to help decide if surgery is needed. The scan may be repeated later to see how the brain is recovering.

Counter coup injury: Bruising of brain tissue on the side opposite where the blow was struck.

Coup injury: Bruising of brain tissue on the same side as the blow was struck.

Decerebrate posture (decerebrate rigidity): Exaggerated posture of extension as a result of a lesion to the pre-pontine area of the brain stem. Rarely seen fully developed in humans. In reporting, it is preferable to describe the posture seen.

Decorticate posture (decorticate rigidity): Exaggerated posture of upper extremity flexion and lower extremity extension as a result of a lesion to the mesencephalon or above. In reporting, it is preferable to describe the posture seen.

Decubitus: Pressure area, bed sore, skin opening, skin breakdown; a discolored or open area of skin damage caused by pressure. Common areas most prone to breakdown are buttocks or backside, hips, shoulder blades, heels, ankles and elbows.

Diffuse axonal injury: A widespread area of the brain where, due to injury, brain cells die, causing swelling in the brain. This increased pressure in the brain can cause decreased blood flow to the brain, as well as additional injury. The shearing can also release chemicals that can contribute to additional brain injury.

Diffuse brain injury: Injury to cells in many areas of the brain rather than in one specific location.

Diplopia: Seeing two images of a single object; double vision.

Directionality: Understanding, identifying and perceiving one's sidedness and how it relates to object's position in space. Right, left, up down, in, out, under, over are terms of directionality.

Disequilibrium: Loss or lack of stability.

Disinhibition: Inability to suppress (inhibit) impulsive behavior and emotions.

Disorientation: Not knowing where you are, who you are, or the current date. Health professionals often speak of a normal person as being oriented "times three," which refers to person, place and time.

Dorsal stream processing: Primarily associated with visually guided reaching and grasping based on the moment-to-moment analysis of the spatial location, shape, and orientation of objects.

Dorsiflexion: When applied to the ankle, the ability to bend at the ankle, moving the front of the foot upward.

Dynamic retinoscopy: Evaluating the retinal reflex to determine the optimum near prescription while the patient is looking at a near target.

Dysarthria: Difficulty in forming words or speaking them because of weakness of muscles used in speaking or because of disruption in the neuromotor stimulus patterns required for accuracy and velocity of speech.

Dysphasia: A swallowing disorder characterized by difficulty in oral preparation for the swallow, or in moving material from the mouth to the stomach. This also includes problems in positioning food in the mouth.

Edema: Collection of fluid in the tissue causing swelling.

Egocentric processing dysfunction: (aka, abnormal egocentric localization, visual midline shift syndrome) Inability to accurately judge how one is centered to objects viewed. The object may be judged as right, left, anterior or posterior to the actual centered position.

Egocentric view: the internal sense of ones' center and how it is referenced to objects viewed. Locations of objects are represented with respect to the particular perspective of a perceiver.

Electroencephalogram (EEG): a procedure that uses electrodes on the scalp to record electrical activity of the brain. Used for detection of epilepsy, coma, and brain death.

Electromyography (EMG): an insertion of needle electrodes into muscles to study the electrical activity of muscle and nerve fibers. It may be somewhat painful to the patient. Helps diagnose damage to nerves or muscles.

Electronystagmography: a diagnostic test to record involuntary movements of the eye caused by a nystagmus. It can also be used to diagnose the cause of vertigo, dizziness or balance dysfunction by testing the vestibular system.

Emotional lability: exhibiting rapid and drastic changes in emotional state (laughing, crying, and anger) inappropriately without apparent reason.

Epilepsy: a brain disorder involving repeated, spontaneous seizures of any type. Seizures ("fits," convulsions) are episodes of disturbed brain function that cause changes in attention or behavior. They are caused by abnormally excited electrical signals in the brain.

Esotropia: medial deviation of one eye relative to the other fixating eye such that fusion is not maintained.

Evoked potential: registration of the electrical responses of active brain cells as detected by electrodes placed on the surface of the head at various places. The evoked potential, unlike the waves on an EEG, is elicited by a specific stimulus applied to the visual, auditory or other sensory receptors of the body. Evoked potentials are used to diagnose a wide variety of central nervous system disorders.

Exotropia: lateral deviation of one eye relative to the other fixating eye such that fusion is not maintained.

Extended-care facility-basic: residential facility that supplies 24-hour nursing care and supervision and assistance with activities of daily life. See Program/Service Types.

Extended-care facility-skilled: a residential facility for the patient who requires 24-hour nursing care (IV, intramuscular injections, special feeding tubes, skin care, oxygen) and rehabilitative therapy, such as physical therapy, occupational therapy, or speech therapy on a less intensive basis than as an inpatient in a comprehensive rehabilitation center. An extended care facility can be a short-term alternative (a few months) prior to placement at home (with outpatient therapy) or in a nursing home. See Program/Service Types.

Extremity: arm or leg.

Figure-ground: the differentiation between the foreground and the background of a scene; this refers to all sensory systems, including vision, hearing, touch.

Fixation: the ability to point and hold the eyes on an object.

Flaccid: lacking normal muscle tone; limp.

Flexion: bending a joint.

Foley catheter: This is a tube inserted into the urinary bladder for drainage of urine. The urine drains through the tube and collects into a plastic bag.

Frontal lobe: front part of the brain; involved in planning, organizing, problem-solving, selective attention, personality and a variety of "higher cognitive functions."

Frontal network syndromes: multifocal partial lesions that individually are not severe enough to disrupt cognitive function but collectively will undermine internetwork coordination. These include symptoms of apathy, disinhibition, and executive dysfunction.

Frustration tolerance: the ability to persist in completing a task despite apparent difficulty. Individuals with a poor frustration tolerance will often refuse to complete tasks that are the least bit difficult. Angry behavior, such as yelling or throwing things while attempting a task, is also indicative of poor frustration tolerance.

Gait training: instruction in walking, with or without equipment; also called "ambulation training."

GI tube: a tube inserted through a surgical opening into the stomach. It is used to introduce liquids, food, or medication into the stomach when the patient is unable to take these substances by mouth.

Glasgow Coma Scale: a standardized system used to assess the degree of brain impairment and to identify the seriousness of injury in relation to outcome. The system involves three determinants: eye opening, verbal responses and motor response, all of which are evaluated independently according to a numerical value that indicates the level of consciousness and degree of dysfunction. Scores run from a high of 15 to a low of 3. Persons are considered to have experienced a “mild” brain injury when their score is 13 to 15. A score of 9 to 12 is considered to reflect a “moderate” brain injury, and a score of 8 or less reflects a “severe” brain injury.

Global vestibulopathy: dysfunction of the entire vestibular system on either the left or right side.

Head injury: refers to an injury of the head and/or brain, including lacerations and contusions of the head, scalp and/or forehead. See Brain Injury.

Hematoma: the collection of blood in tissues or a space following rupture of a blood vessel.

- Regarding Brain: Epidural – Outside the brain and its fibrous covering, the dura, but under the skull.
- Subdural – Between the brain and its fibrous covering (dura).

Homonymous hemianopsia: loss of half of the field of view on the same side in both eyes.

Hemiparesis: weakness of one side of the body.

Hydrocephalus: enlargement of fluid-filled cavities in the brain, not due to brain atrophy.

Hypertropia: a condition of misalignment of the eyes (strabismus), whereby the visual axis of one eye is higher than the fellow fixating eye.

Hypoxia: insufficient oxygen reaching the tissues of the body.

Impulse control: refers to the individual's ability to withhold inappropriate verbal or motor responses while completing a task. Persons who act or speak without first considering the consequences are viewed as having poor impulse control.

Initiative: refers to the individual's ability to begin a series of behaviors directed toward a goal.

Intracerebral: in the brain tissue.
- Subarachnoid–Around the surfaces of the brain, between the dura and arachnoid membranes.

Intracranial pressure (ICP): cerebrospinal fluid (CSF) pressure measured from a needle or bolt introduced into the CSF space surrounding the brain. It reflects the pressure inside of the skull.

Jargon: spoken language that has a normal rate and rhythm but is full of nonsense words.

Kinesthesia: the sensory awareness of body parts as they move (see Position Sense and Proprioception)

Lability: state of having notable shifts in emotional state (e.g., uncontrolled laughing or crying).

Laterality: understanding and preferential use of limbs of one side of the body.

Locked-in syndrome: a condition resulting from interruption of motor pathways in the ventral pons, usually by infarction. This disconnection of the motor cells in the lower brain stem and spinal cord from controlling signals issued by the brain leaves the patient completely paralyzed and mute, but able to receive and understand sensory stimuli; communication may be possible by code using blinking or movements of the jaw or eyes, which can be spared.

Low vision patient: see visually impaired

Magnocellular cells: responsible for motion detection and word recognition. They are connected to the left hemisphere visual language areas and function in reading.

Memory, episodic: memory for ongoing events in a person's life. It is more easily impaired than semantic memory, perhaps because rehearsal or repetition tends to be minimal.

Memory, immediate: the ability to recall numbers, pictures, or words immediately following presentation. Patients with immediate memory problems have difficulty learning new tasks because they cannot remember instructions. It relies upon concentration and attention.

Memory, long term: in neuropsychological testing, this refers to recall 30 minutes or longer after presentation. It requires storage and retrieval of information that exceeds the limit of short-term memory.

Memory, short term: primary or "working" memory; its contents are in conscious awareness. It is a limited capacity system that holds up to seven chunks of information over periods of 30 seconds to several minutes, depending upon the person's attention to the task.

Meningitis: swelling and inflammation of the membranes covering the brain and spinal cord. This inflammation causes changes and pressure in the cerebrospinal fluid (CSF) that surrounds the brain and spinal cord.

Midline shift: the right or left shifting of the cerebral cortex with in the skull. Not to be confused with visual midline shift syndrome or egocentric shift.

Mild brain injury: see brain injury

Money management: ability to distinguish the different denominations of money, count money, make change, budget.

Motor control: regulation of the timing and amount of contraction of muscles of the body to produce smooth and coordinated movement. The regulation is carried out by operation of the nervous system.

Motor planning: action formulated in the mind before attempting to perform.

Multiple sclerosis: an autoimmune disease where the fatty myelin sheaths around the axons of the brain and spinal cord are damaged, leading to demyelination and scarring as well as a broad spectrum of signs and symptoms.

Muscle tone: used in clinical practice to describe the resistance of a muscle to being stretched. When the peripheral nerve to a muscle is severed, the muscle becomes flaccid (limp). When nerve fibers in the brain or spinal cord are damaged, the balance between facilitation and inhibition of muscle tone is disturbed. The tone of some muscles may become increased and they resist being stretched – a condition called hyper tonicity or spasticity.

Neglect: paying little or no attention to a part of the body.

Neologism: nonsense or made-up word used when speaking. The person often does not realize that the word makes no sense.

Neurological event: anything affecting the neural system.

Neuro-optometric rehabilitation: A complex medical assessment and treatment program involving multiple areas of the eye and visual systems. Its goal is to aid recovery from brain injury and to minimize and/or compensate for any functional alterations resulting from the injury.

Neuro-plasticity: the ability of intact nerve cells to take over functions of damaged cells; plasticity diminishes with maturation.

Non-ambulatory: Not able to walk.

Nystagmus: involuntary horizontal, vertical, or rotary movement of the eyeballs.

Occipital lobe: region in the back of the brain that processes visual information. Damage to this lobe can cause visual deficits.

Ocular motor dysfunction: problems with a person's ability to direct point and move their eyes smoothly and accurately.

Oculocentric process dysfunction: when the object's estimated referenced to the eye center is misjudged up, down, right or left of the true center. Common with nerve palsies, strabismus and high phorias.

Oculocentric view: objects are located with respect to the center of the eye.

Orientation: awareness of one's environment and/or situation, along with the ability to use this information appropriately in a functional setting. See Disorientation.

Paraplegia: paralysis of the legs (from the waist down).

Parietal Lobe: one of the two parietal lobes of the brain located behind the frontal lobe at the top of the brain.

Parkinson's disease: a degenerative disorder affects nerve cells in an area of the brain called the substantia nigra inhibiting the cells to produce dopamine, a brain chemical used to help control the body's movements. Patients can experience problems including tremors, muscle stiffness, and slowed motion.

Parvocellular cells: neurons responsible for color sensitivity and discrimination of fine detail

Perception: the ability to make sense of what one sees, hears, feels, tastes or smells. Perceptual losses are often very subtle, and the patient and/or family may be unaware of them.

Perseveration: the inappropriate persistence of a response in a current task that may have been appropriate for a former task. Perseverations may be verbal or motoric.

Persistent Vegetative State (PVS): a long-standing condition in which the patient utters no words and does not follow commands or make any response that is meaningful. See Persistent Unawareness.

Phonation: the production of sound by means of vocal cord vibration.

Plasticity: the ability of cellular or tissue structures and their resultant function to be influenced by an ongoing activity.

Plateau: a temporary or permanent leveling off in the recovery process.

Post-concussive syndrome (PCS): a set of symptoms a person may experience after a concussion (a mild form of traumatic brain injury). Symptoms may be physical, such as headache; cognitive, such as difficulty concentrating; and emotional and behavioral, such as irritability.

Post-traumatic amnesia (PTA): a period of hours, weeks, days or months after the injury when the patient exhibits

a loss of day-to-day memory. The patient is unable to store new information and therefore has a decreased ability to learn. Memory of the PTA period is never stored; therefore, things that happened during that period cannot be recalled. PTA may also be called anterograde amnesia.

Post-trauma vision syndrome: a group of symptoms occurring secondary to brain trauma injury. The most frequent symptoms of this syndrome include binocular coordination dysfunctions, disorientation, loss of equilibrium, memory problems, cognitive dysfunction, loss of executive function (including reading), and an inability to follow sequential instructions, fatigue, irritability, and sensitivity to light.

Posture: the attitude of the body. Posture is maintained by low-grade, continuous contraction of muscles that counteract the pull of gravity on body parts. Injury to the nervous system can impair the ability to maintain normal posture (for example, holding up the head).

Pre-morbid condition: characteristics of an individual present before the disease or injury occurred.

Prism: a transparent optical element with flat, polished surfaces that refract light. The exact angles between the surfaces depend on the application. The traditional geometrical shape is that of a triangular prism with a triangular base and rectangular sides, and in colloquial use "prism" usually refers to this type. Some types of optical prism are not in fact in the shape of geometric prisms. Prisms are typically made out of glass, but can be made from any material that is transparent to the wavelengths for which they are designed.

Problem-solving skill: ability to consider the probable factors that can influence the outcome of each of various solutions to a problem and to select the most advantageous solution. Individuals with deficits in this skill may become "immobilized" when faced with a problem. By being unable to think of possible solutions, they may respond by doing nothing.

Prognosis: the prospect as to recovery from a disease or injury as indicated by the nature and symptoms of the case.

Prone: lying on one's stomach; the sensory awareness of the position of body parts with or without movement; combination of kinesthesia and position sense.

Pursuits: the ability to follow a slow-moving object with the eyes accurately.

Reasoning, abstract: mode of thinking in which the individual recognizes a phrase that has multiple meanings and selects the meaning most appropriate to a given situation. The term "abstract" typically refers to concepts not readily apparent from the physical attributes of an object or situation.

Reasoning, concrete: the ability to understand the literal meaning of a phrase.

Reasoning, problem-solving: the ability to analyze information related to a given situation and generate appropriate response options. Problem-solving is a sequential process that typically proceeds as follows: identification of problem; generation of response options; evaluation of response option appropriateness; selection and testing of first option; analysis as to whether solution has been reached. A patient/client may discontinue making a cup of coffee because the sugar bowl is empty, even though sugar is readily available in a nearby cabinet. A patient/client may easily navigate his way into a room crowded with furniture, but request staff assistance to navigate his way out.

Reasoning, sequencing: the ability to organize information or objects according to specified rules, or the ability to arrange information or objects in a logical, progressive manner. Nearly every activity, including work and leisure tasks, requires sequencing. For example, in cooking certain foods, it is important that ingredients be added and

mixed in a specified order; in dressing, undergarments must be put on prior to outer garments.

Retrograde amnesia: inability to recall events that occurred prior to the accident; may be a specific span of time or type of information.

Saccades: the ability to accurately shift ones' eyes from one target to another.
Scotoma: An isolated area of diminished vision within the visual field.

Seizure: an uncontrolled discharge of nerve cells that may spread to other cells nearby or throughout the entire brain. It usually lasts only a few minutes. It may be associated with loss of consciousness, loss of bowel and bladder control and tremors. Seizures may also cause aggression or other behavioral change.

Sensation: feeling stimuli that activate sensory organs of the body, such as touch, temperature, pressure and pain. Also seeing, hearing, smelling and tasting.

Sensorimotor: refers to all aspects of movement and sensation and the interaction of the two.

Sensory Integration: combining of two or more sensory processes in a manner that enhances the adaptiveness of the brain.

Sequencing: reading, listening, expressing thoughts, describing events or contracting muscles in an orderly and meaningful manner.

Somatosensory: sensory activity having its origin elsewhere than in the special sense organs (such as eyes and ears) and conveying information to the brain about the state of the body proper and its immediate environment.

Spasticity: an involuntary increase in muscle tone (tension) that occurs following injury to the brain or spinal cord, causing the muscles to resist being moved. Characteristics may include increase in deep tendon reflexes, resistance to passive stretch, clasp knife phenomenon, and clonus.

Spatial ability: ability to perceive the construction of an object in both two and three dimensions. Spatial ability has four components: the ability to perceive a static figure in different positions; the ability to interpret and duplicate the movements between various parts of a figure; the ability to perceive the relationship between an object and a person's own body sphere; and the ability to interpret the person's body as an object in space.

Strabismus: a vision disorder due to a deviation from normal orientation of one or both eyes so that both cannot be directed at the same object at the same time, interfering with fusion.

Tactile defensiveness: being overly sensitive to touch; withdrawing, crying, yelling or striking when one is touched.

Temporal lobes: there are two temporal lobes, one on each side of the brain located at about the level of the ears. These lobes allow a person to tell one smell from another and one sound from another. They also help in sorting new information and are believed to be responsible for short-term memory. Right lobe—mainly involved in visual memory (i.e., memory for pictures and faces). Left Lobe—mainly involved in verbal memory (i.e., memory for words and names).

Top-down processing: the process of selectively filtering inputs of sensory information by attending to relevant stimuli while ignoring irrelevant stimuli, resulting in an internalized model from which to direct a meaningful interaction with the environment. Top-down visual processing is dynamic and always changing. It uses prior

experience, existing knowledge, expectation, and motivation to permit the performance of a broader range of behaviors and faster adaptation to changing environmental conditions, such as navigating.

Tracking, visual: visually following an object as it moves through space. In optometry this is referred to as pursuits.

Traumatic brain injury (TBI): see brain injury

Tremor, intention: course, rhythmical movements of a body part that become intensified the harder one tries to control them.

Tremor, resting: rhythmical movements present at rest and may be diminished during voluntary movement.

Unilateral neglect: paying little or no attention to things on one side of the body. This usually occurs on the side opposite from the location of the injury to the brain because nerve fibers from the brain typically cross before innervating body structures. In extreme cases, the patient may not bathe, dress or acknowledge one side of the body.

Ventral stream processing: allows recognition and discrimination of visual shapes and objects

Verbal Apraxia: impaired control of proper sequencing of muscles used in speech (tongue, lips, jaw muscles, vocal cords). These muscles are not weak, but their control is defective. Speech is labored and characterized by sound reversals, additions and word approximations.

Vertical Strabismus: loss of fusion occurs when one eye deviates upward or downward. Also referred to as Hypertropia

Vestibular: pertaining to the vestibular system in the middle ear and the brain that senses movements of the head. Disorders of the vestibular system can lead to dizziness, poor regulation of postural muscle tone and inability to detect quick movements of the head.

Vision therapy: See visual therapy

Vision rehabilitation: is the process of treatment and education that helps individuals who are visually disabled by any condition, disease, or injury that results in functional limitation attain maximum function, a sense of well-being, a personally satisfying level of independence, and optimum quality of life. Function is maximized by evaluation, diagnosis and treatment, including, but not limited to, the prescription of optical, non-optical, electronic and/or other treatments. In addition to the evaluation, diagnosis and management of visual impairment by an eye care physician (optometrist or ophthalmologist), vision rehabilitation may include, but is not limited to, optometric, medical, allied health, social, educational and psychological services.

Visual ataxia: a problem of extra-ocular muscle coordination not due to apraxia, weakness, rigidity, spasticity or sensory loss. Can interfere with a person's ability to fixate, pursue or saccade and object.

Visual closure: the ability to visualize a complete whole when given incomplete information or a partial picture. This skill helps us understand things quickly because our visual system does not have to process every detail to recognize what we're seeing. Where we are reading, this skill helps us recognize sight words. When driving, it helps in making decisions.

Visual evoked potential (VEP): is caused by sensory stimulation of a subject's visual field and is observed using electroencephalography. Commonly used visual stimuli are flashing lights or checkerboards on a video screen that flicker between black on white to white on black (invert contrast).

Visual field: the portion of the individual's surroundings that can be seen at any one time. The normal extent of field of vision is 50° superiorly, 60° nasally, 70° inferiorly and 90° temporally.

Visual field defect: a loss of part of the usual field of vision, so it does not include blindness of either one eye or both. The lesion may be anywhere along the optic pathway: retina to ophthalmic cortex.

Visual imperception: inability to acknowledge the existence of information or objects presented. Disorders of perceptual functioning commonly associated with unilateral brain injury. The disorder may have a sensory or an attentional basis or both; moreover, the patient is often not aware of the problem.

Visual memory: the ability to create an eidetic image of past visual experiences and or information.

Visual midline shift syndrome: see egocentric localization processing

Visual neglect: hemi-spatial agnosia. Paying little or no attention to objects presented in a specific area of one's visual field. Usually occurs on the left side. This is due to a defect in the parietal area.

Visual perception: the ability to interpret and think about past and present information received from visible light reaching the eye.

Visual processing: an ability to make sense of information taken in through the eyes. This is different from problems involving sight or sharpness of vision.

Visual processing delay: problems with visual processing affect how visual information is interpreted or processed by the brain.

Visual therapy: A sequence of neurosensory and neuromuscular activities individually prescribed and monitored by an optometrist to develop, rehabilitate and enhance visual skills and processing. A vision therapy program is based on the results of a comprehensive eye health and vision examination or consultation and considers the results of standardized tests, the needs of the patient, and the patient's signs and symptoms. The use of lenses, prisms, filters, occluders, specialized instruments, and computer programs is an integral part of vision therapy. Activities paralleling in-office techniques are typically taught to the patient to be practiced at home, thereby reinforcing the developing visual skills

Visually impaired: Non-correctable reduced eyesight. World Health Organization classification. When eyesight in the better eye with best possible refractive compensation in place is:

20/30 to 20/60 is considered mild vision loss, or near-normal vision
20/70 to 20/160 is considered moderate visual impairment, or moderate low vision
20/200 to 20/400 is considered severe visual impairment, or severe low vision
20/500 to 20/1,000 is considered profound visual impairment, or profound low vision
Less than 20/1,000 is considered near-total visual impairment, or near total blindness
No light perception is considered total visual impairment, or total blindness

There are also levels of visual impairment based on visual field loss (loss of peripheral vision).
In the United States, any person with eyesight who cannot be corrected to better than 20/200 in the best eye, or who has 20 degrees or less of visual field remaining, is considered legally blind.

Visualize: the ability to form an image of something that is not present.

Watershed event: a stroke in an area of the brain typically at a border zone between the territories of two major cerebral arteries where blood supply is decreased.

Weight bearing: the amount of weight a patient puts on a leg. It is generally described as a percentage of the body weight because each leg of a healthy person carries the full body weight when walking, in an alternating fashion.

Weight shift: instead of having one's weight evenly distributed, the individual shifts more weight on a constant basis to one side, right, left, anterior or posterior.

Yoked prism: a pair of prism lenses of equal power with their prism base in the same direction; often used to change orientation and perceptual direction of one's visual space

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SECTION 7: Appendix

A. TBI /Visual Symptoms Survey: Requiring Optometric Referral

Post Trauma Vision Survey used at SUNY State College of Optometry, Vision Rehabilitation Service, ABI clinic.
0=symptom not present, 1=symptom is minimally present, 2=symptom is moderately present, 3= symptom is severely present.

Emergent Visual Conditions	0	1	2	3
Flashes of light				
Floaters in field of view				
Restricted field of vision				
"Curtains" billowing into field of view				
Urgent Visual Conditions	0	1	2	3
Inability to completely close eyes				
Difficulty moving or turning eyes				
Pain with movement of the eyes				
Pain in or around eyes				
Wandering eye				
Double vision				
TBI/ABI Optometric Vision Rehabilitation Conditions	0	1	2	3
Blurred vision for distance viewing				
Blurred vision for near viewing				
Slow shift of focus from near to far to near				
Difficulty copying or taking notes				
Pulling or tugging sensation around eyes				
Discomfort while reading				
Unable to sustain near work or reading for periods of time				
General fatigue while work/reading				
Loss of place while reading				
Eyes get tired while reading				
Headaches while reading				
Covering, closing one eye				
Easily distracted when reading				
Decreased attention span				
Reduced concentration ability				
Difficulty remembering what has been read				
Disorientation	0	1	2	3
Loss of balance				
Poor posture				
Face, head turn or head tilt				
Bothered by movement in environment				
Bothered by crowded environments				
Light sensitivity				
A sensation of the floor, ceiling or walls tilting				
Dizziness				
A sensation of the room spinning				
A sensation of not feeling grounded				
Postural shifts/ veering off when walking				

SECTION 7: Appendix

B. Mild TBI Visual Dysfunction Screening: questions and tests

In 2012, the Vision Center of Excellence, which is a Department of Defense and Veterans Affairs (VA) collaborative with the mission to "lead and advocate for programs and initiatives to improve vision health"[1] and advise best clinical practices in vision care of veterans and active-duty sevicemembers, looked to describe quick, high-yield screening and general optometric measures for visual dysfunction secondary to mild TBI/concussion. Federal and civilian subject matter experts from all specialties of care and rehabilitation were identified to meet and reflect collaborative expertise. The result was a document titled "Clinical Practice Recommendations for Visual Dysfunction following TBI."

One of the document's recommendations was to produce a quick mild TBI visual dysfunction screening questionnaire for the general practitioner and general optometrist as well as to identify baseline optometric clinical testing that would best screen for the most common mild TBI related visual dysfunctions. Below are the results of the Delphi Consensus of 16 VA optometrists who provide TBI vision care in the VA Polytrauma System of Care. This article is published in the *Journal of Rehabilitation Research and Development* in August 2013. The Brain Injury Committee of the AOA VRS agrees the survey questions and list of general optometric procedures adequately identifies patients with visual dysfunctions most common in the mild TBI population base.

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Screening Questions
Did you have any neurological problems or symptoms before your TBI (MS, stroke, brain tumor, severe headaches, other)?
When did your TBI occur (on what date)?
Did you lose consciousness during or after your TBI incident?
Were you disoriented or confused during or after your TBI incident?
Do you bump into objects and walls more now than before your injury?
Were your eyes, eyelids, or area around your eyes injured when your TBI event occurred?
Do you cover or close one eye at times since your injury?
Have you noticed a change in your vision since your injury?
Are you more sensitive to light, either indoors or outdoors, since your injury?
Have you had any double vision since your injury?
Have you noticed any changes in your peripheral vision since your injury?
Is your vision blurry at distance or near since your injury?
Have you noticed a change in your ability to read since your injury?
Do you lose your place while reading more now than before your injury?
How long can you read continuously before you need to stop?
Do you get headaches during/after reading more now than before your injury?
Do you have more difficulty remembering what you have read now than before your injury?

Table 2. The 7-mTBI exam procedures testing items accepted by the consensus survey. Each statement ended with the phrase “is/are important to test on every mTBI patient”.

Procedure
(Free Space) Distance Cover Test
(Free Space) Near Cover Test
Versions (EOMs) and/or Pursuits
Accommodation
Saccades
Near Point of Convergence (NPC)
Repeated NPC (any method)

SECTION 7: Appendix

C. Glasgow Coma Scale

Eye Opening Response

- Spontaneous—open with blinking at baseline 4 points
- Opens to verbal command, speech, or shout 3 points
- Opens to pain, not applied to face 2 points
- None 1 point

Verbal Response

- Oriented 5 points
- Confused conversation, but able to answer questions 4 points
- Inappropriate responses, words discernible 3 points
- Incomprehensible speech 2 points
- None 1 point

Motor Response

- Obeys commands for movement 6 points
- Purposeful movement to painful stimulus 5 points
- Withdraws from pain 4 points
- Abnormal (spastic) flexion, decorticate posture 3 points
- Extensor (rigid) response, decerebrate posture 2 points
- None 1 point

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D. Rancho Los Amigos Scale

I. No Response: to sounds, sights, touch or movement.

II. Generalized Response: responds in the same way to sounds, sights, touch or movement. Responses include chewing, sweating, breathing faster, moaning, moving and/or increasing blood pressure.

III. Localized Response: reacts more specifically: turns toward a sound; withdraws from pain; watches a person move around the room; recognizes family and friends; follows simple directions such as "look at me" or "squeeze my hand."

IV. Confused-Agitated: overreacts to what is seen, heard or felt by hitting, screaming, using abusive language, or thrashing about; is highly focused on basic needs such as eating, relieving pain, going back to bed, going to the bathroom, or going home

V. Confused-Inappropriate, Non-Agitated: pays attention for a few minutes; does not know date, place or reason for being in hospital; is restless if tired or if there are too many people around; has poor memory, remembers events prior to the accident better than after the accident; focuses on basic needs of eating, relieving pain, going back to bed, using the bathroom, or going home.

VI. Confused-Appropriate: is confused due to memory and thinking problems, remembers main points of a conversation but forgets and confuses details; pays attention for 30 minutes, cannot concentrate if it is noisy or if the activity involves many steps.

VII. Automatic-Appropriate: follows a set schedule; has problems in new situations; becomes frustrated; acts without thinking first; cannot pay attention in distracting or stressful situations.

VIII. Purposeful-Appropriate: realizes there is a problem in thinking and memory and begins to compensate; is ready for driving or job-training evaluation.

SECTION 7: Appendix

E. Gaze Disturbance Differential

Chrystyna Rakoczy, O.D.

I. Gaze Deviation
   a. Horizontal deviation
      i. VOR intact (positive oculocephalic maneuver)
         1. Unilateral parietal lesion, ipsilateral deviation away from hemiparesis
         2. Unilateral thalamic lesion, no preference
      ii. VOR impaired (negative oculocephalic maneuver)
         1. Unilateral pontine lesion, contralateral deviation toward hemiparesis
   b. Vertical deviation (VOR independent)
      i. Upgaze deviation
         1. CNS hypoxia or ischemia
         2. Pharmacological
         3. Psychogenic
      ii. Downgaze deviation
         1. Thalamic lesion
         2. Hydrocephalus
         3. Extraocular myopathy

II. Gaze Deficit
   a. Omni-directional
      i. VOR intact (positive oculocephalic maneuver)
         1. Bilateral parieto-occipital and frontal lesion, with oculomotor apraxia
         2. Thalamic lesion
         3. Progressive supranuclear palsy
      ii. VOR impaired (negative oculocephalic maneuver)
         1. Midbrain and pontine lesions
         2. CNS disorders
         3. Chronic progressive external ophthalmoplegia
         4. Various dystrophies
         5. Extraocular myopathies
         6. Congenital
   b. Horizontal deficit
      i. VOR intact (positive oculocephalic maneuver)
         1. Acute unilateral cerebral lesion
         2. Congenital
      ii. VOR impaired (negative oculocephalic maneuver)
         1. Pontine lesion
         2. CNS disorders
         3. Extraocular myopathies
         4. Congenital
         5. Myasthenia
   c. Vertical deficit

i. VOR intact (positive oculocephalic maneuver)
    1. Thalamic lesion
    2. Progressive supranuclear palsy
ii. VOR impaired (negative oculocephalic maneuver)
    1. Midbrain lesion
    2. Thalamic lesion
    3. Pineal lesion
    4. Hydrocephalus
    5. Myasthenia
    6. Extraocular myopathy

SECTION 7: Appendix

F. Piaget Test

The basic assessment of visual perceptual functioning can be done with the Piaget Test. This test provides information on how the patient perceives body awareness, laterality, directionality, and spatial projection.[1] The Piaget test is a simple test to administer in a routine primary eye care examination and provides useful information about visual perceptual processing. Below is the procedure for administering the Piaget Test taken from Groffman[2] and Solan [2]:

Instructions:[2]

SET A

1-Show me your right hand
2-Show me your left hand
3-Show me your right leg
4-Show me your left leg

SET B

Examiner sits opposite the patient
1-Show me my right hand
2-Show me my left hand
3-Show me my right leg
4-Show me my left leg

SET C

Place a coin on the table left of a pencil in relation in relation to the patient
1-Is the pencil to the right or to the left
2-Is the coin to the right or left
3-have the patient move around to the opposite side of table and repeat is the pencil to right or left, and how about the coin

SET D

Sit opposite the patient holding a pencil in right hand and a watch on left wrist
1-Is the coin in my right or left hand and is the watch on my right or left hand

SET E

Place three objects in front of the patient (pencil, key, and coin) put pencil to left of key (middle) and coin to right of key.
1-Is the pencil to the right or left of the key
2-Is the pencil to the right or left of the coin
3-Is the key to the left or right of the coin
4-Is the key to the right or left of the pencil
5-Is the coin to the left or right of the pencil
6-Is the coin to the left or right of the key

Norms for the Piaget Right-Left Discrimination (items passed 75 percent age)

Age	*Items passed by 75 percent 0f age*
5	A
6	A
7	A.C
8	ABCD
9	ABCD
10	ABCD
11	ABCDE

References

1. Cohen A. Acquired visual information-processing disorders: closed head trauma. In: Press L, ed. *Applied concepts in vision therapy*. St. Louis: Mosby, 1997:154-166.
2. Groffman S, Solan H. Spatial orientation tests. In: Groffman S, Solan H, eds. *Developmental & perceptual assessment of learning-disabled children theoretical concepts and diagnostic*. Santa Ana, CA: Optometric Extension Program Foundation, 2001: 116-117

SECTION 7: Appendix

H. Convergence Insufficiency Symptom Survey

http://www.aoa.org/optometrists/tools-and-resources/clinical-care-publications/clinical-practice-guidelines