

“Sensorimotor Vision Disturbances Following Acquired Brain Injury”

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**Overview of acquired brain injury (ABI)**

1. Interdisciplinary health care professionals involved with ABI
2. Vision and ABI:
3. Why is it likely to be affected?
4. What is optometry’s role?
5. Typical vision conditions (along with neurological correlates, associated symptoms, and treatment options) post-ABI

**What is ABI? Any condition/event resulting in a sudden, non-progressive or non-degenerative change in neurological processing, including:**

1. Traumatic brain injury (TBI)
2. Cerebrovascular accident (CVA)
3. Aneurysm
4. Tumor removal
5. Post-surgical neurological complications resulting in anoxia or hypoxia
6. Vestibular dysfunction

**TBI**

Definition: TBI is an acquired brain injury, inclusive of brain injuries caused by anoxia due to near drowning but exclusive of congenital or degenerative disorders and birth trauma (Amendment to U.S. Public Health Service Act under Title VII, 1994)

**TBI: Epidemiology**

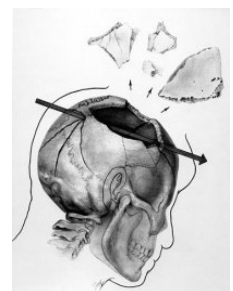
1. According to the NIH’s National Institute of Neurological Disorders and Stroke (NINDS) as well as the Center for Disease Control (CDC), current U.S. civilian statistics are:
  2. Incidence: ~1.4 million people incur a TBI yearly
  3. Prevalence: ~5.3 million people are living with a TBI, with ~2% of the U.S. population requiring long-term assistance for daily activities
  4. Economic Cost (as direct medical cost and indirect as lost productivity): \$56 billion per year
  5. Fatalities: 50,000 people yearly
  6. Hospitalization: approximately 1 million people are treated in ER yearly
  7. Survivors: approximately 230,000 people are released after hospitalization

**Brain Injury Statistics Iraq: A New Perspective**

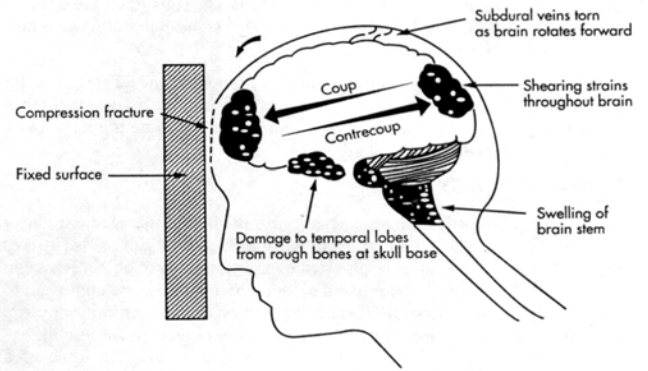
1. As of 01/07, the Department of Defense (DOD) reported:
  - a. 11,852 members of the military had been wounded in explosions involving I.E.D.’s (Improvised Explosive Devices)
  - b. Of the survivors, more than 1,700 are known to have brain injuries impairing behavior and their ability to perform work-related activities.
2. *The NY Times (on 07/26/07) reported that:*
  - a. *when 35,000 apparently healthy returnees from Iraq and Afghanistan were screened, 10 to 20 percent “had apparently experienced a mild TBI during deployment”*
3. The veterans administration has identified Polytrauma rehabilitation centers and secondary rehabilitation centers to evaluate and manage ABI/TBI among returning service members.

**TBI: Pathophysiology Open versus Closed**

1. Open Head Injuries
  - a. Typically cause a more localized or focal insult:
  - b. penetrating injuries (i.e., gun-shot wound, fractured skull, etc.)



2. Closed Head Injuries
  - a. With the cranium intact, typically cause a more diffuse or global insult:
  - b. coup (acceleration) insult
  - c. contre-coup (deceleration) insult
  - d. shearing forces which may lead to:
  - e. breakage of blood vessels (epidural or subdural hematomas)
  - f. diffuse axonal injury (DAI)
  - g. blast injury/shock trauma (evident more recently with returning veterans of the Iraq/Afghanistan wars)



### TBI: Sequence of Brain Damage

1. Primary injury:
  - a. Occurs at moment of injury/insult as:
  - b. Lacerations
  - c. Contusions
  - d. Fractures
  - e. Diffuse axonal shearing
2. Secondary brain injury
  - a. may occur hours to weeks post-injury altering:
  - b. Auto-regulatory physiological mechanisms
  - c. Neurotoxin release
  - d. Cascade of biochemical reactions
3. may result in:
  - a. Hemorrhages
  - b. Hypoxia
  - c. Increased intracranial pressure
  - d. Infection

### TBI: Functional Impact

1. Post concussion syndrome (PCS) encompassing changes in:
  - a. Cognitive abilities
  - b. Sensorimotor abilities
  - c. Emotion
2. Post trauma vision syndrome (PTVS) includes anomalies of:
  - a. Accommodation
  - b. Tear film integrity
  - c. Ocular motility (versional and vergence)
  - d. Visual-vestibular interaction
  - e. Light-dark adaptation (resulting in photosensitivity)
  - f. Visual field integrity

### TBI: Severity      Severity of TBI is determined by the:

1. Glasgow Coma Score (GCS)
2. neuro-imaging
3. loss of consciousness (LOC)
4. alteration of consciousness (AOC) or mental state
5. post-traumatic amnesia (PTA)
6. neuro-psychological testing, when possible (typically performed on those with mild TBI)

**TBI: Severity with Glasgow Coma Scale**

The Glasgow Coma Scale: Responses and Scoring		
Test	Patient's Response	Assigned Score
<b>Eye Opening</b>	Opens eyes on own	4
	Opens eyes when asked in a loud voice	3
	Opens eyes when pinched	2
	Does not open eyes	1
<b>Motor Response</b>	Follows simple commands	6
	Pulls examiner's hand away when pinched	5
	Pulls part of body away when examiner pinches patient	4
	Flexes body inappropriately to pain (decorticate posturing)	3
	Body becomes rigid in an extended position when examiner pinches victim (decerebrate posturing)	2
	Has no motor response to pinch	1
<b>Verbal Response</b>	Carries on a conversation correctly and tells examiner where he is, who he is, and the month and year	5
	Seems confused or disoriented	4
	Talks so examiner can understand victim but makes no sense	3
	Makes sounds that examiner cannot understand	2
	Makes no sounds	1

GCS	Neurological findings	Grade of Severity
15	Inconspicuous	Conscious
13-14	Mild neurological disorder	Cerebral commotion (e.g., TBI grade 1)
9-12	Moderate neurological disorder	Clouded awareness/ cerebral contusion (e.g., TBI grade 1-2)
6-8	Severe neurological disorder	Clouded awareness to unconsciousness (e.g., TBI grade 2-3)
3-5	Most severe neurological disorder	Unconscious, coma (e.g., TBI grade 3)

**TBI: Severity Scale Using Neuro-imaging, LOC, AOC, PTA, and GCS**

Parameter	Mild	Moderate	Severe
<b>Structural Neuro-imaging</b>	<b>normal</b>	<b>normal OR abnormal</b>	<b>abnormal</b>
LOC	0-30 mins	30 mins to 24 hours	>24 hours
AOC	1 min-24 hours	>24 hours	>24 hours
PTA	0-1 day	1-7 days	>7 days
GCS	13-15	9-12	<9

**CVA (Stroke): Epidemiology**

1. 750,000 CVAs per year (based on CDC, American Heart Association, and National Stroke Association)
2. Leading cause of chronic disability in the adult U.S. population
3. Third leading cause of death in the adult U.S. population

### CVA: Pathophysiology

1. Infarcts occurring due to vascular compromise, typically a localized insult
2. Ischemic (vessel blockage) versus hemorrhagic (an internal bleed)
3. Severity of the stroke is dependent upon the lesion's:
  - a. location
  - b. hemisphere
4. Severity of stroke is determined by
  - a. neuro-imaging
  - b. neuro-behavioral responses

### Post-surgical Complications

1. Aneurysm clippings
2. Tumor removals
3. Hypoxia or anoxia occurring during surgical or post-surgical care

### Vestibular Dysfunction

1. Labyrinthitis
2. Acoustic neuroma
3. Gentamycin ototoxicity
4. Benign paroxysmal positional vertigo
5. Meniere's disease
6. Vertigo (often non-specific and may be idiopathic)
7. Disequilibrium (often non-specific and may be idiopathic)

### Interdisciplinary Rehabilitation Team

1. Physiatrist (MDs specializing in physical medicine and rehabilitation)
2. Neurologist
3. Internist
4. Psychiatrist/Psychologist
5. Neuropsychologist (specialize in evaluation and rehabilitation of cognitive impairments)
6. Physical Therapist/Vestibular Therapist
7. Occupational Therapist
8. Speech Therapist
9. And--- Optometrists

### Vision and the Brain

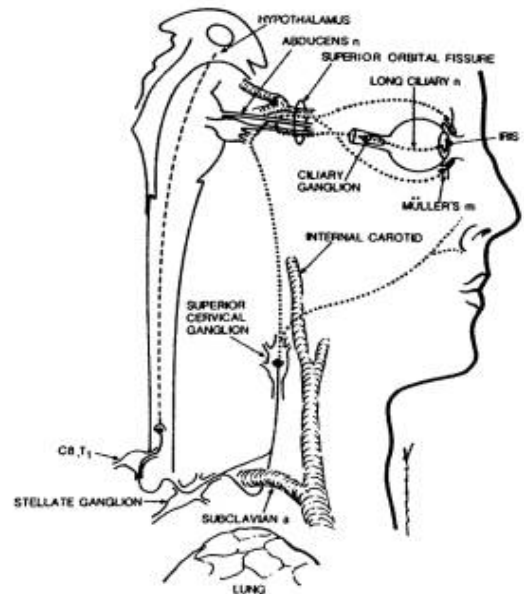
1. Brainstem's vision involvement:
  - a. 50% of the cranial nerves impact vision function:
  - b. CN II, III, IV, V, VI, and VII
2. Primary and associated areas of cortex which relate to these brainstem cranial nerve nuclei

### Role of Optometry

1. To diagnose and/or treat (optically and/or with vision rehabilitation) vision disturbances to optimize vision function for use in their:
  - a. overall rehabilitation regimen
  - b. activities of daily independent living (ADLs) impacting overall quality of life (QOL)

### Typical Vision Deficits Post-ABI

1. Deficits of:
  - a. Accommodation (in pre-presbyopic patients)
  - b. Tear film integrity
  - c. Versional ocular motility
  - d. Vergence ocular motility
  - e. Visual-Vestibular interaction
  - f. Light-dark adaptation (resulting in photosensitivity)
  - g. Visual field integrity



### **Accommodation: Neurological Correlates**

1. Pre-motor neural components:
  - a. Mediated by the autonomic nervous system (ANS)
    - i. primarily the parasympathetic system to stimulate or increase the accommodative response
    - ii. secondarily the sympathetic system to inhibit or reduce the accommodative response
2. Visual cortex to parieto-temporal (PT) area to the Edinger-Westphal (EW) nucleus in the pre-tectum
3. At the EW nucleus, autonomic input is received to form the motor command
4. Combined autonomic and motor neurons travel via the oculomotor nerve from the EW nucleus to the ciliary ganglion to the short ciliary nerve and then to the ciliary muscle
5. End result:
  - a. a change in the contraction of the ciliary muscle
  - b. consequent change in crystalline lens shape and effective state of accommodation

### **Accommodative Deficits: Associated Symptoms**

1. Constant/intermittent blur
  - a. at far or near
2. Intermittent blur due to infacility
  - a. near-far blur
  - b. far-near blur
3. Symptoms associated with near vision tasks (i.e., reading/ using computer/ handicrafts):
  - a. Eyestrain/ eye fatigue
  - b. Browaches
  - c. Dizziness/nausea/motion sickness

### **Accommodative Deficits: Restorative Treatment Options**

1. Equalize accommodative amplitudes
2. Work on improving the weaker aspect of focusing; i.e., if a patient cannot:
  - a. relax the accommodative state, work on near-far focusing
  - b. increase the accommodative state, work on far-near focusing

### **Accommodative Deficits: Restorative Treatment Options**

1. Work on maintaining the ability to:
  - a. rapidly change focus on command and repeatedly over time
  - b. sustain focus for extended periods of time
2. Training may be performed:
  - a. using lenses
  - b. in free space regarding targets at different viewing distances

### **Accommodative Deficits: Compensatory Treatment Options**

1. Lenses may be prescribed for near vision tasks either:
  - a. In lieu of restorative accommodative training
  - b. In conjunction with restorative accommodative training
  - c. Following restorative accommodative training (examples: those who work on computer for 8-10 hours daily may require near vision glasses to prevent eyestrain and headaches regardless of accommodative integrity)

### **Dry Eye: Associated Symptoms**

1. Intermittent lack of visual clarity
2. varies with blinking
3. "gritty", foreign body sensation

### **Dry Eye: Treatment Options**

1. Artificial tears TID/QID OU in conjunction with lid hygiene
2. For moderate to severe dry eye:
  - a. Systane or Restasis
  - b. insertion of punctal plugs

### **Versional Ocular Motility: Neurological Correlates**

1. Integrated pre-motor neural activity occurs in similar areas for vertical saccades, horizontal saccades, and horizontal pursuit:
  - a. frontal lobe

- b. parietal lobe
  - c. basal ganglia
  - d. superior colliculus
  - e. cerebellum
2. Vertical Saccades
    - a. Pre-motor neural area: rostral mesencephalon
  3. Horizontal pre-motor neural components:
    - a. Saccade: excitatory burst neurons in the paramedian pontine reticular formation (PPRF) => project directly to the oculomotor neuron for horizontal saccades
    - b. Pursuit: pursuit neurons in the medial vestibular nuclei and prepositus hypoglossi => project directly to the oculomotor neuron for horizontal pursuit
  4. Horizontal Saccade and Horizontal Pursuit
    - a. Since 2003, a joint premotor path has been proposed in the PPRF to modulate velocity of movement with common:
      - i. inhibitory omnipause neurons
      - ii. saccade/pursuit neurons
  5. Integrated pre-motor neural components for fixation include:
    - a. Frontal eye fields
    - b. Supplemental eye fields
    - c. Parietal eye fields
    - d. Right prefrontal cortex (for attention)
    - e. Right posterior parietal cortex (for attention)

### **Versional Ocular Motility: Associated Symptoms**

1. Reading-related difficulties
2. Slower reading speed
3. Loss of place/skipping or missing lines or words
4. Re-reading/misreading words or lines
5. Print seems to "swim" / "jumble" on the page
6. Difficulty shifting to/tracking objects
7. Dizziness/nausea/motion sickness

### **Versional Ocular Motility Deficits: Restorative Treatment Options**

1. Basic scanning and searching exercises
2. Concentrate on accuracy
3. Gradually build up speed
4. Text size is often not the issue:
  - a. *The space between the lines is often more critical.*

### **Versional Ocular Motility Deficits: Compensatory/Adaptive Treatment Options**

1. Encourage a typoscopic approach (i.e., create an aperture/window highlighting the text of regard while obscuring non-pertinent text)

### **Vergence Ocular Motility: Neurological Correlates**

1. Pre-motor neural innervation:
  - a. Mesencephalic reticular formation with three types of vergence cells:
    - i. Tonic: respond to change in vergence angle
    - ii. Burst: respond to change in vergence velocity
    - iii. Burst-tonic: respond to changes in both vergence angle and velocity
  - b. Medial longitudinal fasciculus
  - c. Cerebellum
  - d. Frontal eye fields
  - e. Role in generating vergence response of the abducens and oculomotor interneurons: not clearly elucidated

### **Vergence Ocular Motility Deficits: Associated Symptoms**

1. Diplopia
  - a. Eliminated with occlusion
  - b. Constant or intermittent
  - c. At far or near
  - d. More evident in one position of gaze than another

2. Eyestrain/fatigue after <10-15 minutes of consecutively performing a visually-related task (i.e., driving, watching movies or television, performing arts and crafts at near, reading sheet music, using computers, studying, reading for pleasure)
3. Closing one eye or squinting when performing a prolonged visual task
4. Avoidance of prolonged vision-related tasks
5. Dizziness/nausea/motion sickness

### Vergence Ocular Motility Deficits: Patient Safety Concerns

1. impaired relative depth perception affecting:
  - a. driving
  - b. ambulation:
    - i. in busy streets/ buildings/ environments
    - ii. up and down stairs/ escalators

### Vergence Ocular Motility Deficits: Restorative Treatment Options

1. If appropriate, application of fusional prism for constant strabismus or associated heterophoria
2. Stabilize vergence in primary gaze (ramp and step) at far and near viewing distances
3. Facility and sustainability of fusional vergence at far and near viewing distances

### Ocular Motor and Vestibular Systems: Neurological Connection=VOR

1. CN III and VI communicate with CN VIII via the medial longitudinal fasciculus to generate the horizontal vestibulo-ocular reflex (VOR, also referred to as gaze stabilization)

### Visual-Vestibular Interaction: Purpose

1. VOR
  - a. Stabilizes the visual world while the head is in motion
  - b. Is utilized in most vestibular rehabilitation
  - c. May be impaired in the presence of ocular motor deficits
2. Improving and stabilizing any ocular motor deficit may facilitate vestibular rehabilitative progress

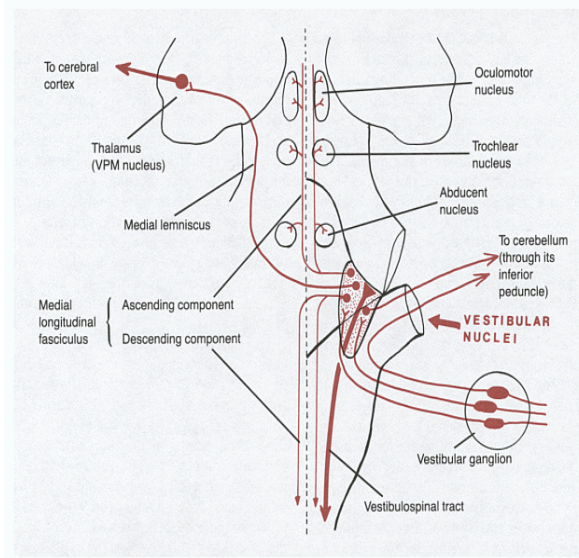
### Ocular Motor Deficits and Vestibular Dysfunction: Associated Symptoms

1. Versional Oculomotility
  - a. slower reading speed
  - b. loss of place while reading
  - c. skipping lines/ word when reading
  - d. the print on the paper seems to "swim"/ "float above the page"
  - e. difficulty tracking objects
  - f. dizzy/nausea/ increased sensitivity to visual motion
2. Vergence Oculomotility
  - a. constant or intermittent diplopia
  - b. eyestrain/eye fatigue after <10-15 minutes of performing a visual task
  - c. closing/squinting one eye when performing a visual task for a prolonged time
  - d. dizzy/nausea/ increased sensitivity to visual motion
3. Same as for vergence and versional ocular motility, with an emphasis on:
  - a. Increased disequilibrium in and sensitivity to multiply-visually stimulating environments (i.e., supermarkets, malls, etc.)
  - b. Dizziness/ nausea/ vertigo/ disequilibrium with vision tasks (i.e., reading, watching television/cinema, ambulation, and using the computer)
  - c. Oscillopsia

### Ocular Motor Deficits and Vestibular Dysfunction: Treatment Options

1. Treatment options are:
  - a. restorative, compensatory, and adaptive
2. Current on-going clinical research with neurotology:

Central connections of the vestibular system



- a. Chandrasekhar S, Kapoor N (2007) Neuro-optometric evaluation and rehabilitation as a useful adjunct in the management of the complex dizzy patient. *Annual meeting of the American Neurotology Society, San Diego, CA, April 28, 2007.*
3. Symptoms being addressed:
  - a. Increased disequilibrium in and sensitivity to multiply visually-stimulating environments
  - b. Difficulty with eye/head dissociation
  - c. Foreground/background discrimination difficulty
4. Same treatment as for versional oculomotor deficits without vestibular dysfunction, except:
  - a. Start at a slower velocity, while patient is seated and minimal targets in the background
  - b. Systematically increase the:
    - c. velocity of the ocular motility
    - d. number of targets in the background
  - e. Build to having the patient marching in place while performing these tasks in front of a multiply, visually-stimulating background
5. Same treatment as for vergence ocular motility deficits without vestibular dysfunction, except:
  - a. After stabilizing fusional vergence in primary gaze under static conditions:
    - i. Stabilize vergence 30 degrees right gaze (ramp, step) and then 30 degrees left gaze (ramp, step)
    - ii. Stabilize dynamic vergence while patients is performing a slow horizontal VOR (approximately 40-60 rotations per minute)
  - b. After stabilizing horizontal fusional vergence and a slow horizontal VOR:
    - i. Stabilize vergence 25 degrees upgaze (ramp, step) and then 25 degrees downgaze (ramp, step)
    - ii. Stabilize dynamic vergence while patients is performing a slow vertical VOR (approximately 40-60 rotations per minute)

### **Photosensitivity**

1. Photosensitivity versus Photophobia:
  - a. Photosensitivity is increased light sensitivity with eyestrain/discomfort in the ABSENCE of ocular inflammation.
  - b. Photophobia is increased light sensitivity with ocular pain in the PRESENCE of ocular inflammation.

### **Photosensitivity: Neurological Correlates**

1. Elevated light sensitivity (to all lights OR specifically to fluorescent lights) despite unremarkable ocular health:
  - a. No ocular inflammation or infection
2. Current hypothesis for neural mechanism:
  - a. Cortical or subcortical substrates resulting in anomalous dark and light adaptation.
  - b. Precise location of neural substrate: not yet localized

### **Photosensitivity: Associated Symptoms**

1. Increased light sensitivity
  - a. General- to all types of light
  - b. Selective- to fluorescent lighting
    - i. more common in the "dizzy" patient
    - ii. may be related to anomalous thresholds to critical flicker fusion

### **Photosensitivity: Compensatory Treatment Options**

1. Incorporation of tints with spectacle correction (30-40% tint for indoors, 80-85% tint for outdoors) for photosensitivity that is:
  - a. general to all lights (using either brown or gray tints)
  - b. specific for fluorescent lighting (using either blue or gray tints)
2. Wearing brimmed hats/caps

### **Impaired Visual Field Integrity: Neurological Correlates**

1. Scattered, non-lateralized
  - a. More typical in TBI
  - b. Less common in stroke
2. Lateralized (i.e., left hemianopia, right hemianopia, superior right quadrantanopia, etc.)
  - a. More typical in stroke
  - b. Less common in TBI
  - c. May occur with or without inattention
3. Scattered visual field defects
  - a. Evident despite intact retina with unremarkable ocular health
  - b. Neural mechanism:

- i. Likely secondary to diffuse neurological damage
- 4. Lateralized visual field defects
  - a. Evident despite intact retina with unremarkable ocular health
  - b. Neural mechanism:
    - i. Secondary to localized lesions (hemorrhagic or ischemic)
      - 1. often left-brain lesions present without inattention
      - 2. often right-brain lesions present with inattention
- 5. Congruous versus non-congruous visual field defects
  - a. Congruous hemianopias/ quadrantanopias → lesions located more posteriorly along the optic radiations.
  - b. Non-congruous hemianopias/ quadrantanopias → lesions located more anteriorly along the optic radiations.

**Impaired Visual Field Integrity: Associated Symptoms**

- 1. Missing any aspect of the visual field
- 2. Awareness of any loss of visual field integrity
- 3. *NOTE: Spectrum of visual field integrity*
  - a. No visual field defect, without inattention
  - b. No visual field defect, with inattention
  - c. Visual field defect, with inattention
  - d. Visual field defect, without inattention
- 4. may or may not be accompanied by:
  - a. “neglect” (i.e., inattention)
  - b. a feeling of unsteadiness when standing or ambulating
- 5. difficulty with:
  - a. reading
  - b. dressing/ eating/ cosmesis
  - c. ambulation on one side more than the other

**Impaired Visual Field Integrity: Patient Safety Concerns**

- 1. veering towards, bumping into, or missing objects on one side of space poses a safety hazard for:
  - a. driving resulting in:
    - i. not seeing cars or pedestrians on one side
  - b. ambulation resulting in bruising, falls, or mild concussions from:
    - i. bumping into walls, doors, and people
    - ii. tripping over shoes, children’s toys, legs of tables/chairs
    - iii. potentially being struck by a motor vehicle when crossing the street
    - iv. strike one’s head on a cupboard door
  - c. shaving resulting in cuts

**Impaired Visual Field Integrity: Compensatory and Adaptive Treatment Options**

- 1. Visual field hemianopic/quadrantanopic defect with inattention:
  - a. application of yoked prisms, mirrors, and field expanding lenses in conjunction with scanning strategies and compensatory/ adaptation approaches
- 2. Visual field hemianopic/quadrantanopic defect without inattention:
  - a. application of sector prisms and spotting prisms in conjunction with scanning strategies and compensatory/ adaptation approaches

**Impaired Visual Field Integrity: NOVA Vision Restorative Program**

- 1. may benefit those with an actual field defect
- 2. more likely beneficial for those with relative (i.e., not ALL absolute) defects on perimetric testing
- 3. no definitive information regarding impact on inattention
- 4. background literature from the 1990s suggested a 5-15 degree improvement into the affected field

“Acquired Brain Injury for the Primary Care OD”

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**Overview of optometric examination**

1. Rationale for non-optometric referrals
2. Two scenarios commonly evident post-ABI:
3. Vertical diplopia/near vision eyestrain/reading problems
  - a. Dizziness
  - b. Overview of Optometric Examination

**When examining those with ABI---**

1. Note that those with ABI typically report sensorimotor symptoms:
  - a. related to sensory stimulation overload with the perception that, in most places (aside from their home and other controlled quiet, dim environments), there are too many:
    - i. visual stimuli/movements
    - ii. sounds
    - iii. smells
  - b. which may be due to difficulty selecting the stimulus from the noise
    - i. often, everything feels like a stimulus for those with ABI
2. Note that they may have sensorimotor deficits such as:
  - a. Hearing deficits
    - i. Loss
    - ii. Hyperacusis
    - iii. Tinnitus
  - b. Speech impairments
  - c. Dizziness/nausea
3. Note that they may have motor disturbances such as:
  - a. Restricted neck movements
  - b. Restricted lateralized mobility of upper and/or lower extremities
  - c. Gait disturbance
  - d. Tremors
4. Note that they may have affect disorders such as:
  - a. Increased lability
  - b. Anger control (with frontal lobe injuries)
  - c. Impulsivity (with frontal lobe injuries)
  - d. Inappropriate behavior (with frontal lobe injuries)
5. Note that they may have cognitive deficits such as:
  - a. Slower speed of processing (in varying degrees), frequently across all modes of processing
    - i. Auditory
    - ii. Verbal
    - iii. Visual
  - b. Impairment of
    - i. short- and/or long-term memory
    - ii. word-retrieval
    - iii. language processing (i.e., as aphasia, expressive/receptive/mixed)
    - iv. organizational abilities
    - v. executive control

**Pearls when examining those with ABI**

1. Minimize movements (i.e., gesticulation, rapid movements in front of or around the patient)
2. Keep the room illumination relatively dim (use incandescent, rather than fluorescent, lighting when possible)
3. Speak clearly and slightly more slowly than you may normally speak
4. Slowly change the prism magnitudes when performing heterophoria/vergence testing
5. Have the patient close their eyes in between tests

## Elements of Vision Exam

1. Case History
  - a. Trauma history: date/nature of neurological insult
  - b. Traditional ocular history PLUS onset/nature of any of the following vision symptoms:
    - i. Blur (constant/intermittent)
    - ii. Reading difficulties (slower speed/loss of place/skipping lines)
    - iii. Diplopia or overlapping images (constant/intermittent)
    - iv. Increased sensitivity in multiply visually-stimulating environments in conjunction with dizziness/nausea/vertigo/disequilibrium
    - v. Increased sensitivity to light (fluorescent versus all types of light)
    - vi. Missing one half of their vision/bumping into objects on one side of space
2. Refractive assessment
  - a. For those with signs or symptoms of gait, vestibular, or cognitive dysfunction, multi-focal lenses are:
    - i. Typically contraindicated for ambulation
    - ii. Appropriate for non-ambulatory tasks
  - b. Prescribe single vision distance correction for ambulation and single vision near correction for prolonged reading/computer use
3. Accommodative assessment for pre-presbyopes
  - a. monocular amplitudes
  - b. monocular (or binocular) lags
4. Versional ocular motor assessment
  - a. monocular and binocular motilities
5. Vergence ocular motor assessment
  - a. cover test at far and near, as well as near point of convergence
6. Relative accommodation, especially when prescribing near vision corrections
7. Visual field assessment
  - a. confrontation visual field testing
  - b. automated visual field testing when possible
8. Ocular health assessment
  - a. monocular color vision
  - b. pupils
  - c. anterior segment evaluation
  - d. tonometry
  - e. posterior segment evaluation with dilation
9. Case Disposition
  - a. 10-15 minutes at the close of the examination to:
    - i. Summarize findings, outlining remarkable ones in terms of:
      1. Diagnosis
      2. Relation to daily living activities
      3. Treatment options
      4. Prognosis
  - b. Report-writing
    - i. Send to referring physician with a copy to the patient

## Rationale for Referral to Other Health Care Professionals

### Referrals from the OD: Internist, Neurologist/Neurotologist, Physiatrist

1. Applicable for:
    - a. patients with ABI who are currently NOT being managed by physicians in a department of rehabilitation medicine
- OR
- b. any patient with ABI reporting:
    - i. recurrent vision symptoms (i.e., constant or intermittent eyestrain/ blur/ diplopia/visually-related headaches)
    - ii. dizziness/vertigo/loss of balance
    - iii. seizure disorder
    - iv. concussion

### Referral to the Internist

1. Recommend that the following be evaluated:
  - a. complete blood work (including, but not limited to: thyroid, HbA1, lipid profile, cholesterol, lyme, lupus)
  - b. blood pressure over time (i.e., at varying times of day and in different body positions)
2. Rationale for recommendations:

- a. Changes in metabolism, as well as blood pressure, may be accompanied by:
  - i. changes in accommodative ability, refractive state, and/or extraocular motility
  - ii. dizziness/vertigo/disequilibrium
- b. Lyme and Lupus both may be accompanied by:
  - i. impaired versional and vergence ocular motility
  - ii. dizziness/vertigo/disequilibrium

#### **Referral to the Neurologist (specializing in ABI)**

1. Recommend that the following be performed to aid in determining and managing the underlying neurological etiology of the patient's:
  - a. recurrent vision symptoms, seizure disorder, and concussions:
    - i. a complete neurological evaluation
    - ii. any indicated neuro-imaging (including electroencephalography for seizure disorder and multiple concussions and MRI of the orbits for recurrent vision symptoms)

#### **Referral to: Neurologist (specializing in Dizziness)/ Neurotologist (ENT specializing in Dizziness)**

1. Recommend that the following be performed to aid in determining and managing the underlying neurological etiology of the patient's:
  - a. vestibular dysfunction:
    - i. a complete neurological evaluation
    - ii. electronystagmography (ENG, also known as caloric testing)
    - iii. audiological testing
    - iv. neuro-imaging of the brain and neck

#### **Referral to: Neurologist or Neurotologist**

1. Rationale for recommendations:
  - a. Etiology of neurological pathology may impact treatment intervention and prognosis with:
    - i. physical therapy
    - ii. vestibular therapy
    - iii. occupational therapy
    - iv. cognitive therapy
    - v. speech and language therapy
    - vi. vision rehabilitation

#### **Referral to the Psychiatrist (specializing in ABI)**

1. Recommend a complete psychiatric evaluation to:
  - a. assess areas of sensorimotor, emotional, cognitive, and social function
  - b. recommend or prescribe treatment interventions for evident deficits
2. Rationale for recommendation:
  - a. determining which (and prescribing) additional types of rehabilitation to benefit the patient
  - b. especially important if the patient presents with any non-vision-related symptoms AND is not currently receiving rehabilitation.

#### **Referral to the Rehabilitative Optometrist (specializing in vision rehabilitation for ABI)**

1. Recommend a sensorimotor vision evaluation to better assess treatment options for the patient's vision symptoms following ABI
2. Rationale for recommendation:
  - a. determining and implementing possible treatment approaches (to address the patient's vision symptoms following ABI will eventually benefit all aspects of rehabilitation and daily life) including:
    - i. optical (lenses, prisms, partial occluders)
    - ii. vision rehabilitation (home-based or in-office)
    - iii. monitor --- no further intervention or rehabilitation indicated at present

#### **Factors Exacerbating Many Symptoms (Including Vision) Post-ABI**

1. Illness (fever/flu/virus) and/or Pain
2. Fatigue (over-exertion/lack of rest OR aerobic respiration)
3. Use of controlled substances (alcohol, nicotine, narcotics, etc.)
4. **Changes in** stress (not stress itself)

#### **Case 1: Vertical Diplopia**

1. CASE 1: History
  - a. In June, 2005, 53-year-old White Female presented with a skull fracture and subsequent neurosurgery (date of injury: 09/29/04) reporting:
    - i. Difficulty viewing objects at near and far with a lack of clarity possibly due to her constant ocular misalignment which is:
      1. More evident when reading than when walking around

- ii. Slower reading speed
    - iii. Frequent errors, loss of place, and skipping lines when reading
    - iv. Difficulty using a computer
  - b. Personal Medical and Ocular History:
    - i. Hypertension
  - c. Medications:
    - i. Atenolol
  - d. Allergies:
    - i. No reported allergies to medications
  - e. Occupation:
    - i. newspaper journalist
- 2. CASE 1-Refractive state
  - a. Acuties with current correction (PALs)
    - i. OD  $-1.00$  sph 20/20 /  $+2.00$  add 20/20 (DV and NV)
    - ii. OS  $-0.50$  sph 20/20/  $+2.00$  add 20/20 (DV and NV)
- 3. CASE 1-Oculomotor Status
  - a. Fixation for 10 seconds in primary gaze:
    - i. OD few saccadic intrusions
    - ii. OS few saccadic intrusions
    - iii. OU immediate diplopia
  - b. Pursuit testing:
    - i. OD full
    - ii. OS full
    - iii. OU non-comitant diplopia, evident increase in left hypertropia in right gaze
  - c. Gross saccade testing:
    - i. OD hypometria evident
    - ii. OS hypometria evident
    - iii. OU not performed due to diplopia which was confusing for the patient
  - d. Additional saccade testing:
    - i. Developmental Eye Movements (DEM) test- unable to complete due to fatigue and diplopia
    - ii. Visagraph-performed at a later visit, confirming deficits of saccades
  - e. Ocular alignment:
    - i. Intermittent left hypertropia (greater at near than far) in primary gaze:
      - 1. DCT 1 XP, 4 left hyperphoria, patient could fuse a 20/30 target at far
      - 2. DBI X/6/4
      - 3. DBO X/8/6
      - 4. LS 6/4
      - 5. LI 2/1
    - 6. NCT 4XP', 14-16 left hyper T', patient was unable to fuse a 20/30 target at near 2/3rds through the examination
    - 7. NPC: oblique diplopia at 2'
    - 8. NRA/PRA: not performed due to diplopia
- f. Ocular alignment:
  - i. Performed Park's three step (even though it was 9-10 months post-injury) to confirm the presence of a left superior oblique (LSO) palsy:
    - 1. Small-angle left hypertropia in primary gaze
    - 2. Increase in left hypertropia in right gaze
    - 3. Increase in left hypertropia with a leftward head tilt
  - ii. \*NOTE: Patients with LSO palsy frequently present with left hypertropia and a possible right head tilt\*
- g. Park's Three Step for LSO Palsy: Step 1
  - i. Distance cover test in primary gaze:
    - 1. left hyplopia (recent onset with intermittent diplopia)
- h. Park's Three Step for LSO Palsy: Step 2
  - i. Compare the magnitude of hypertropia in left versus right gaze
    - 1. For LSO palsy, magnitude of left hypertropia is greater in right gaze relative to left gaze
  - ii. Why is there an increase in left hypertropia upon right gaze more so than left gaze?
  - iii. LSO is the primary depressor of the left eye when in the adducted position
    - 1. Upon right gaze,
      - a. OS is adducted
      - b. LSO is the primary depressor

- c. LSO is impaired and cannot depress OS enough
      - d. OS presents as elevated relative to OD
    - i. Park's Three Step for LSO Palsy: Step 3
      - i. *Compare magnitude of hypertropia with left versus right head tilt*
        - 1. For LSO palsy, magnitude of left hypertropia will be greater with left head tilt
      - ii. Why is there an increase in left hypertropia with the leftward head tilt?
        - 1. With a leftward head tilt:
          - a. Left eye is forced to intort and the right eye is forced to extort to maintain ocular alignment
          - 2. Superior muscles intort and inferior muscles extort
          - 3. One of the intorters for OS, specifically the LSO, is impaired but both of the extorters for OD can function
          - 4. Thus, the left hypertropia is more pronounced with a leftward head tilt
4. CASE 1-Ocular Health Status
  - a. Monocular color vision with the D-15:
    - i. no defects OD or OS
  - b. Automated visual field testing (Humphrey 30-2, Sita Standard):
    - i. no defects OD or OS
  - c. Pupils, tonometry, anterior and posterior segment evaluation without dilation (as this patient is dilated yearly by her ophthalmologist):
    - i. unremarkable OD and OS
5. CASE 1- Assessment and Plan
  - a. Left IV n palsy, Deficits of Saccades (ICD-9-CM: 378.53, 379.57):
    - i. Refer to internist to re-evaluate blood work and blood pressure to insure stable systemic health.
    - ii. Refer to neurologist to re-evaluate neuro-imaging and neuro-behavioral function to insure stable neurological health.
    - iii. Recommend weekly in-office vision rehabilitation to improve and stabilize fusional vergence and versional ocular motility.
  - b. 2. Myopia, Presbyopia (ICD-9-CM: 367.1, 367.4):
    - i. No need to change spectacle prescription; continue:
      - 1. using current PALs for distance vision and ambulation since she:
        - a. is comfortable with the PALs
        - b. has no memory, balance, or visual disturbances
      - 2. removing PALs for prolonged near vision tasks (computer and reading) since her working distance is 18-20" which is perfect for her when uncorrected
6. CASE 1: Vision Rehabilitation
  - a. Recommended a trial of 10-16 sessions of in-office vision rehabilitation
  - b. Areas trained:
    - i. Fixation
    - ii. Small- and moderate-angle saccades
    - iii. Vergence
    - iv. Visual memory (simultaneous, sequential, spatial)
    - v. Visual-motor integration
    - vi. Speed of visual processing
  - c. After 9 sessions of weekly in-office vision rehabilitation with 10-15 minutes of home therapy 4-5 days weekly, she:
    - i. presented with
      - 1. significant symptom reduction
      - 2. normalization of clinical signs
    - ii. was discharged with a tapering of home therapy
      - 1. 3 days/week for 3 weeks
      - 2. 2 days/week for 3 weeks
      - 3. 1 day/week for 3 weeks
      - 4. no home therapy for 2-3 weeks
  - d. She was re-evaluated 3 months post-completion of in-office vision rehabilitation and presented with:
    - i. Persistent symptom reduction accompanied by normalization of clinical signs
  - e. She remains asymptomatic and is monitored twice yearly by our center

## Case 2: Dizziness

### 1. CASE 2-History

- a. On April 21, 2005, 41-year-old White Female presented with a non-specific (etiology: idiopathic) vestibular dysfunction (onset: 10/04) reporting:
  - i. Images bounce or jump when she moves (she feels as though she is on a pogo stick)
  - ii. Blurred vision during ambulation
  - iii. Increased sensitivity in multiply visually-stimulating environments
  - iv. Intermittent horizontally overlapping images (not two distinct objects)
- b. Additional problems:
  - i. Headaches (worse closer to 10/04, but now have lessened in intensity and frequency)
- c. Personal Medical and Ocular History:
  - i. H/o stage 1 site 2 melanoma removed from above her right knee in 2001: there was no evidence of metastasis, and the patient recovered well with no recurrences since 2001.
- d. No medications
- e. Allergies:
  - i. Codeine
  - ii. Sulfa drugs
- f. Occupation:
  - i. Graphic designer

### 2. CASE 2-Refractive state

- a. Visual acuities with her current spectacle correction (+0.50sph OU for prolonged near vision tasks, including computer use):
  - i. OD pl 20/25 / +0.50add 20/20
  - ii. OS pl 20/25 / +0.50add 20/20

### 3. CASE 2-Oculomotor Status

- a. Fixation (for 10 seconds in primary gaze) testing
  - i. No nystagmus OD, OS
  - ii. No fixation losses OD, OS, and OU
- b. Pursuit testing
  - i. Slight jerkiness on pursuit testing OD, OS, and OU
  - ii. No restriction of extraocular motility for either eye
  - iii. Comitancy evident OU
- c. Gross saccade testing:
  - i. OD hypometria evident
  - ii. OS hypometria evident
  - iii. OU rare hypometria
- d. Additional saccade testing:
  - i. Developmental Eye Movements (DEM) test: V=60sec, H=77 sec, E=0
  - ii. Visagraph-performed at a later visit & confirmed deficits of saccades
- e. Ocular alignment and vergence testing (distance performed sc, near performed c +0.50sph OU)
  - i. DCT ortho, iso
  - ii. DBI X/8/4
  - iii. DBO X/6/2
  
  - iv. NCT 2-4XP', iso
  - v. NPC 1"/2"/OS out/+dipl
  - vi. NBI X/18/14
  - vii. NBO X/12/4
  - viii. NRA +2.00
  - ix. PRA -1.75

### 4. CASE 2- Ocular Health Status

- a. Monocular color vision with the D-15:
  - i. no defects OD or OS
- b. Automated visual field testing (Humphrey 30-2, Sita Standard):
  - i. no defects OD or OS
- c. Pupils, tonometry, anterior and posterior segment evaluation without dilation (as this patient is dilated yearly by her ophthalmologist):
  - i. unremarkable OD and OS

### 5. CASE 2- Assessment and Plan

1. Fusional vergence dysfunction, Deficits of Saccades, Deficits of Pursuit (ICD-9-CM: 368.30, 379.57, 379.58):
  - a. Recommend weekly in-office vision rehabilitation.

2. Early presbyopia (ICD-9-CM: 367.4):
  - b. Continue with current reading glasses (+0.50sph OU) for prolonged near vision tasks (including computer use).
  - c. No spectacle correction is indicated for distance vision.
  - d. Multifocal lenses are contraindicated for ambulation.
3. Vestibular Dysfunction (ICD-9-CM: 780.4):
  - e. Monitor with otolaryngologist/neurologist for changes in otolaryngological integrity and neurological health.
  - f. Return to internist for complete blood work and blood pressure assessment to insure stable systemic health.
6. CASE 2: Vision Rehabilitation
  - a. Recommended a trial of 10-16 sessions of in-office vision rehabilitation
  - b. Areas trained:
    - i. Fixation
    - ii. Small- and moderate-angle saccades
    - iii. Vergence
    - iv. Visual-vestibular habituation
    - v. Visual-motor integration
    - vi. Speed of visual processing
  - c. After 10 sessions of weekly in-office vision rehabilitation with 10-15 minutes of home therapy 4-5 days weekly, she:
    - i. presented with:
      1. significant symptom reduction
      2. normalization of clinical signs
    - ii. was discharged with a tapering of home therapy as:
      1. 3 days/week for 3 weeks
      2. 2 days/week for 3 weeks
      3. 1 day/week for 3 weeks
      4. no home therapy for 2-3 weeks
  - d. She was re-evaluated 2 months post-completion of in-office vision rehabilitation and presented with:
    - i. persistent symptom reduction accompanied by normalization of clinical signs (including uncorrected distance acuities)
  - e. She remains asymptomatic and is monitored annually by her private ophthalmologist

### Summary

1. The purpose of today's presentations was to increase familiarity and understanding with respect to the:
  - ⇒ terminology and pathophysiology associated with ABI
  - ⇒ members of the interdisciplinary health care professional team
  - ⇒ reasons why sensorimotor vision function may be impaired post-ABI and optometry's associated role in managing such vision deficits
  - ⇒ typical post-ABI residual vision disturbances (amenable to non-surgical treatment interventions) along with:
    - 1) neurological correlates
    - 2) associated symptoms
    - 3) possible treatment options
  - ⇒ evaluation and management approaches by the primary care optometrist, exemplified with two clinical scenarios (diplopia and dizziness) frequently evident post-ABI
2. Special Thanks To:
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  - d. Optometric organizations for their support re: increasing awareness of vision care and ABI
    - i. New York State Optometric Association
    - ii. College of Optometrists in Vision and Development
    - iii. Optometric Extension Program Foundation
3. Recommended Book: Medical Management of Adults with Neurologic Disabilities by Adrian Christian