Learning Objectives

To increase familiarity and understanding with respect to:

1) why sensorimotor vision function may be impaired following concussion/mild traumatic brain injury (mTBI)

2) the members of the inter-professional health care management team

3) the vision provider’s associated role in managing such vision deficits

VISION AND CONCUSSION/mTBI

TBI/Concussion

According to the Centers for Disease Control and Prevention (CDC):

“A TBI is caused by a bump, blow, or jolt to the head or a penetrating head injury that disrupts the normal function of the brain. Not all blows or jolts to the head result in a TBI. The severity of a TBI may range from “mild” (i.e., a brief change in mental status or consciousness) to “severe” (i.e., an extended period of unconsciousness or memory loss after the injury).”

According to the CDC, current U.S. civilian statistics from 2006-2010 reveal that:

- ~2.5 million people present at hospital (emergency room, hospitalization, fatality) with TBI in 2010, with most being concussions or other forms of mild TBI
- for non-fatal TBI-related hospitalizations:
  - gender bias towards males evident
  - those aged 65 years and older have the highest hospitalization rates
  - children aged 0-4 years have the highest rates of ED visits
  - the greatest reason was:
    - a fall for 0-4 years of age and > 45 years of age.
    - motor vehicle accident for > 45 years of age.


According to the CDC, current U.S. civilian statistics from 2006-2010 reveal that:

- TBI-related deaths:
  - due to any cause: three-fold gender bias towards males evident
  - highest rates for persons 65 years and older.
  - Leading cause varied by age.
    - Falls for those >65 years of age.
    - Assaults for children ages 0-4 years of age.
    - Motor vehicle crashes:
      a) for children and young adults ages 5-24 years of age
      b) were the second leading cause of TBI-related deaths, accounting for 26% in 2006-2010


According to the CDC, current U.S. civilian statistics from 2006-2010 reveal that the common causes of TBI (in order) are:

1. falls (40%)—bimodal age distribution for TBIs being due to falls: for persons under 15 years of age (55%) and over 65 years of age (~81%)
2. unintentional blunt trauma (15%)—TBI was due to blunt trauma in ~24% of persons under 15 years of age
3. motor vehicle accident (14.3% of TBI), which were the third leading cause of TBI among all age groups
4. assault (10.7%)—~75% of all assault-related TBI occurred in persons 15 to 44 years of age.


According to the CDC, current U.S. civilian statistics regarding concussion and sports/recreational injuries reveal that:

- ~248,418 persons under 19 years of age were treated in U.S. emergency departments (EDs) in 2009
- from 2001-2009, rate of ED visits associated with concussion/sports injuries in those under 19 years of age rose by 57%


Between January 2000 through December 8, 2015, 339,462 servicemembers were identified as having suffered a TBI, based on the most recent update from the Defense and Veterans Brain Injury Center (web-link: http://www.dvbic.org/dod-worldwide-numbers-tbi, accessed on 01/25/16). From January 1, 2000 through December 8, 2015, ~82.5% of these TBIs were classified as mild TBIs or concussions.

- Operation Enduring Freedom (Afghanistan)
- Operation Iraqi Freedom (Iraq, primarily)
- Operation New Dawn (working with Iraqi forces to develop their internal security and infrastructure)

With the cranium in tact, typically cause a more diffuse or global insult:

- Coup (acceleration) insult
- Contre-coup (deceleration) insult
TBI/Concussion: Closed Head Injuries

• With the cranium in tact, typically cause a more diffuse or global insult:
  • Percussive event/blast injury/shock trauma (evident more recently with returning veterans of the Iraq/Afghanistan wars)

Shearing forces which may lead to:
  • Breakage of blood vessels (epidural or subdural hematomas)
  • Diffuse axonal injury (DAI)

TBI/Concussion: Sequence of Brain Damage

• Primary injury:
  • Occurs at moment of injury/insult as:
    • Lacerations
    • Contusions
    • Fractures
    • Diffuse axonal shearing

Secondary brain injury (from where functional changes associated with the TBI/Concussion arise)

May Occur Hours to Weeks Post-Injury and Alter:

<table>
<thead>
<tr>
<th>May Result In:</th>
<th>May Occur Hours to Weeks Post-Injury and Alter:</th>
</tr>
</thead>
<tbody>
<tr>
<td>hemorrages</td>
<td>auto-regulatory physiological mechanisms</td>
</tr>
<tr>
<td>hypoxia</td>
<td>neurotoxin release</td>
</tr>
<tr>
<td>increased intracranial pressure</td>
<td>cascade of biochemical reactions</td>
</tr>
<tr>
<td>infection</td>
<td>build-up of the protein tau</td>
</tr>
</tbody>
</table>

TBI/Concussion: Severity

• Severity of TBI/Concussion is determined by:
  • Glasgow Coma Score (GCS)
  • Neuro-imaging
  • Loss of consciousness (LOC)
  • Alteration of consciousness (AOC) or mental state
  • Post-traumatic amnesia (PTA)
  • Neuro-psychological testing, when possible (typically performed on those with mild TBI)

TBI/Concussion: Commonalities

• Commonalities between mild/moderate TBI and concussion are evident in the:
  • Presenting categories of symptoms
  • Parameters of the grading scales


TBI/Concussion: Functional Impact

- Encompasses possible changes in:
  - Cognitive abilities
  - Mood/affect
  - Sleep
  - Sensorimotor abilities — including vision

TBI/Concussion: Typical Signs Observed and Symptoms Reported

<table>
<thead>
<tr>
<th>Signs Observed</th>
<th>Symptoms Reported</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poor recall of events prior to or subsequent to a hit or fall</td>
<td>Headache or head pressure</td>
</tr>
<tr>
<td>Appears dazed or stunned</td>
<td>Nausea and/or vomiting</td>
</tr>
<tr>
<td>Forgets an assignment or position/unsure of game, score, or opponent</td>
<td>Confused, problems concentrating, or problems with memory</td>
</tr>
<tr>
<td>Clumsy movements</td>
<td>Balance problems, dizziness, or vision problems (commonly blur/diplopia)</td>
</tr>
<tr>
<td>Slow to answers questions</td>
<td>Feel sluggish, foggy, or groggy</td>
</tr>
<tr>
<td>Loses consciousness (even if brief)</td>
<td>Increased sensitivity to light and/or noise</td>
</tr>
<tr>
<td>Mood, behavior, or personality changes</td>
<td>Just not &quot;feeling right,&quot; or &quot;feeling down&quot;</td>
</tr>
</tbody>
</table>


TBI/Concussion: Informational Web-based Documents from the CDC


TBI/Concussion: Typical Vision Deficits/Primary Associated Symptoms Evident Following TBI/Concussion

<table>
<thead>
<tr>
<th>Deficit of:</th>
<th>Primary Associated Symptom:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accommodation</td>
<td>Constant/intermittent blur</td>
</tr>
<tr>
<td>Tear Film Integrity</td>
<td>Distorted clarity/irritity sensation, which varies with blinking</td>
</tr>
<tr>
<td>Versional Ocular Motility</td>
<td>Slower, less accurate reading, difficulty sustaining gaze, shifting gaze, or tracking targets</td>
</tr>
<tr>
<td>Vergence Ocular Motility</td>
<td>Constant/intermittent eyestrain / diplopia eliminated with monocular occlusion</td>
</tr>
<tr>
<td>Visual-Vestibular Interaction</td>
<td>Vestibular symptoms exacerbated in multiply, visually-stimulating environments</td>
</tr>
<tr>
<td>Light-Dark Adaptation</td>
<td>Elevated light sensitivity</td>
</tr>
<tr>
<td>Visual processing</td>
<td>Slower speed/impaired visual memory and visual-spatial processing</td>
</tr>
</tbody>
</table>

**"The above is also referred to as "Post-trauma vision syndrome" (PTVS)"
TBI/Concussion: Informational Web-based Documents from the CDC's Heads Up Site


Inter-Professional Neuro-Rehabilitation Team

- Usual team leaders—one of the following:
  - Physiatrist (MDs specializing in physical medicine and rehabilitation)
  - Neurologist
  - Neuropsychologist (specializing in evaluation and rehabilitation of cognitive impairments)
  - Sports medicine (coaches, physical education teachers, and engaged parents/caregivers are also involved and frequently refer to an internist or team leader)

  - Internal medicine- internists, physician’s assistants, and registered nurses
  - Psychologist/psychiatrist

Inter-Professional Neuro-Rehabilitation Team

- Typical therapies
  - Physical Therapist
  - Occupational Therapist
  - Vestibular therapy (depending upon the rehab department) by one of the following:
    - Physical Therapist
    - Occupational Therapist
  - Cognitive rehab—one of the following:
    - Speech/Language Therapist
    - Neuropsychologist
  - Vision therapy—one of the following:
    - OT
    - PT
    - Optometrist

Inter-Professional Neuro-Rehabilitation Team

- Management approaches:
  - Refer for/provide EBM services within that particular center/rehab department/hospital
  - Refer to/recommend needed services (regardless of location) as long as they are EBM
  - Refer for/provide any potentially beneficial services (even if not EBM) within that particular center/rehab department/hospital
  - Refer to/recommend needed services (regardless of location and even if not EBM)

Vision and the Brain

- Starting with the brainstem:
  - 50% of the cranial nerves impact vision function directly or indirectly:
    - Direct:
      - CN II, III, IV, and VI
    - Indirect:
      - CN V and VII

  - Moving on to cortex:
    - Primary and associated neurons relating to these brainstem cranial nerve nuclei traverse all 4 cortical lobes (not just occipital).

Role of Optometry/Ophthalmology

- To diagnose and/or treat (optically and/or with vision rehabilitation) vision disturbances to optimize vision function for use in a patient’s:
  - Overall rehabilitation regimen
  - Activities of daily independent living (ADLs), thereby impacting overall quality of life (QOL)

- Finding an optometrist specializing in neuro-optometric rehabilitation near you:
  - www.covd.org
  - www.nora.cc
Behavioral and Visual Considerations with Concussion

What to be Aware of When Examining Those with Concussion/mTBI

- Those with concussion/mTBI typically report sensorimotor symptoms:
  - 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:
    - visual stimuli/movements
    - sounds
    - smells
  - NOTE: the sensory stimulation overload may be due to difficulty selecting the stimulus from the noise - i.e., often, everything feels like a stimulus

What to be Aware of When Examining Those with Concussion/mTBI

- Those with concussion/mTBI may have sensorimotor deficits such as:
  - Hearing deficits
    - loss
    - hyperacusis
    - tinnitus
  - Speech impairments
  - Dizziness/nausea

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    - loss
    - hyperacusis
    - tinnitus
  - Speech impairments
  - Dizziness/nausea

What to be Aware of When Examining Those with Concussion/mTBI

- Those with concussion/mTBI may have cognitive deficits such as:
  - Slower speed of processing (in varying degrees), frequently across all modes of processing
    - auditory
    - verbal
    - visual
What to be Aware of When Examining Those with Concussion/mTBI

- Those with concussion/mTBI may have cognitive deficits such as:
  - Impairment of:
    - Short- and/or long-term memory
    - Word-retrieval and/or language processing (i.e., as aphasia, expressive/receptive/mixed)
    - Organizational abilities/prioritization
    - Executive control

Factors Exacerbating Symptoms (Including Vision) Following Concussion/mTBI

- Illness (fever/flu/virus) and/or Pain
- Note for those with vestibular dysfunction, changes in barometric pressure exacerbates their symptoms
- Fatigue (over-exertion/lack of rest OR aerobic respiration)
- Use of controlled substances (alcohol, nicotine, narcotics, etc.)

Pearls When Examining Those with Concussion/mTBI

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

Visual Appearance of Text to Our Patients

Ideal, Clear and Single Vision

How it May Look:

- “Is that old man in the corner?”
- “It’s the one who used to live over there.”
- “It’s the old man who used to live in the corner.”
- “It’s the old man who used to live over there.”
- “It’s the old man who used to live in the corner.”
- “It’s the old man who used to live over there.”

Optometric Examination: High Yield History and Testing

Elements of High Yield Vision Exam

- Very good paper supporting high yield case history questions and diagnostic tests for mild TBI:

Optometric Examination: High Yield History and Testing

Elements of High Yield Vision Exam

- **Case History**
  - Trauma history: date/nature of neurological insult
  - Traditional ocular history PLUS onset/nature of any of the following
    **Vision symptoms:**
    - blur (constant/intermittent)
    - reading difficulties (slower speed/loss of place/skipping lines)
    - diplopia or overlapping images (constant/intermittent)
    - increased sensitivity in multiply visually-stimulating environments in conjunction with dizziness/nausea/vertigo/disequilibrium
    - increased sensitivity to light (fluorescent versus all types of light)
    - missing one half of their vision/bumping into objects on one side of space
    - visual perceptual processing (visual recognition, visual memory, visual spatial relations, visual spatial memory, speed of visual processing)


Elements of High Yield Vision Exam: Management of Refractive Status

- **For those with signs or symptoms of gait, vestibular, or cognitive dysfunction:**
  - Multi-focal spectacle lenses are:
    1) typically contraindicated for ambulation
    2) appropriate for non-ambulatory tasks
  - Prescribe single vision distance correction for ambulation and single vision near (or flat-top/round-segment computer top/reading bottom bifocal) correction for prolonged reading/computer use

Elements of High Yield Vision Exam

- **Accommodative assessment for pre-presbyopes**
  - Monocular amplitudes
  - Monocular (or binocular) lags
  - **Vergence ocular motor assessment**
    - Cover test at far and near, as well as near point of convergence (NPC)
  - **Versional ocular motor assessment**
    - Monocular and binocular motilities
    - Relative accommodation, especially when prescribing near vision corrections

  **denotes high yield diagnostic tests in those with TBI/ concussion**


Elements of High Yield Vision Exam

- **Visual field assessment**
  - Confrontation visual field testing
  - Automated visual field testing when possible
- **Ocular health assessment**
  - Pupils
  - Anterior segment evaluation
  - Tonometry
  - Posterior segment evaluation with dilation
  - As indicated:
    - contrast testing
    - color vision
    - OCT

Elements of High Yield Vision Exam

- **Case Disposition**
  - 10-15 minutes at the close of the examination to:
    - Summarize findings, outlining remarkable ones in terms of:
    - diagnosis
    - relation to ADLs
    - treatment options
    - prognosis
  - Report-writing
  - Send to referring physician with a copy to the patient
OPTOMETRIC EVALUATION:
ADDITIONAL TESTING
PERFORMED BY NEURO-OPTOMETRY

- Sensorimotor vision testing:
  - Cover test, NPC, phoria and vergence ranges
  - Accommodative amplitudes, facility, and lag
  - Keystone Visual Skills
  - Cheiropic tracing
  - Van Orden Star
  - Vectograms
  - King-Devick or DEM
  - Visagraph

- Visual-perceptual testing:
  - Visual memory
  - simultaneous (Tachistoscope)
  - sequential (Visual Span)
  - Speed of visual processing
  - Visual-spatial processing
  - block design
  - visual spatial relations
  - visual spatial memory

**For patients with vestibular symptoms:**
- Dynamic visual acuity (DVA) — an assessment of VA performed while the head is in motion
  - re-check monocular/binocular acuity at far or near while head is slowly (50–60 rotations per minute, if possible and patient has no neck issues) moving horizontally
  - if DVA is more than 2 lines poorer than static VA (SVA), then there is a visual-vestibular problem

- For vestibular patients:
  - Check for opticokinetic response (if possible or warranted) — helpful to rule out malingering for visual-vestibular symptomatology

EVIDENCE-BASED MEDICINE ON OCCURRENCE OF SENSORIMOTOR VISION DEFICITS IN TBI
Clinical Research on Vision and TBI:
OCCURRENCE OF OCULAR MOTILITY (INCLUDING ACCOMMODATIVE) DEFICITS


- PURPOSE: To report the percentage of ocular motor (including accommodative) diagnoses evident in a selected, visually-symptomatic out-patient sample with TBI (n=160) and CVA (n=60) of patients seen 10/01/2000 to 10/07/2003.

Patient Profile

<table>
<thead>
<tr>
<th>Parameter</th>
<th>TBI (n=160)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age range (years)</td>
<td>8 to 91</td>
</tr>
<tr>
<td>Mean age (years)</td>
<td>44.9</td>
</tr>
<tr>
<td># of males</td>
<td>73</td>
</tr>
<tr>
<td># of females</td>
<td>87</td>
</tr>
<tr>
<td>Years post-injury (range)</td>
<td>0.1-42.0</td>
</tr>
<tr>
<td>Mean years post-injury</td>
<td>4.5</td>
</tr>
</tbody>
</table>


Results of Ciuffreda et al. (2007)

<table>
<thead>
<tr>
<th>Ocular motor dysfunction</th>
<th>TBI (%)</th>
<th>Most common anomaly (TBI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accommodation</td>
<td>41.1</td>
<td>Accommodative insufficiency</td>
</tr>
<tr>
<td>Versional</td>
<td>51.3</td>
<td>Deficits of saccades</td>
</tr>
<tr>
<td>Vergence</td>
<td>56.3</td>
<td>Convergence insufficiency</td>
</tr>
<tr>
<td>Strabismus</td>
<td>25.6</td>
<td>Strabismus at near</td>
</tr>
<tr>
<td>CN palsy</td>
<td>6.9</td>
<td>CN III</td>
</tr>
</tbody>
</table>

*NOTE: regarding accommodation: 51 (out of 160) persons with TBI were pre-presbyopic and were included in the accommodative statistics.


Clinical Research on Vision and TBI: OCCURRENCE OF OCULAR MOTILITY (INCLUDING ACCOMMODATIVE) DEFICITS

- GOODRICH ET AL. JRRD 2007

<table>
<thead>
<tr>
<th>Total TBI sample (n=46)</th>
<th>Blast-related TBI (n=21)</th>
<th>Non-Blast-related TBI (n=25)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Convergence</td>
<td>30.4%</td>
<td>23.8%</td>
</tr>
<tr>
<td>Accommodation</td>
<td>21.7%</td>
<td>23.8%</td>
</tr>
<tr>
<td>Saccades/pursuit</td>
<td>19.6%</td>
<td>4.8%</td>
</tr>
</tbody>
</table>

**SO WHAT?**

- In a visually-symptomatic TBI/concussion sample, over 50% may present with deficits of:
  - versional ocular motility (51.3%)
  - vergence ocular motility (56.3%)
- Most common anomalies were convergence insufficiency and deficits of saccades

Recent Studies — Reporting on non-selected patient samples with TBI

- Regarding the military, studies by Drs. Gregory Goodrich and Glenn Cockerham also support a high occurrence of vision dysfunctions in TBI => Goodrich GL, Kirby J, Cockerham G, Ingalla SP, Lew HL; JRRD 2007; 44 (7) :929-936
- Investigated the occurrence of sensorimotor vision deficits on patients at their center whether symptomatic or not and then compared blast-related TBI to non-blast-related TBI

Clinical Research on Vision and TBI: OCCURRENCE OF OCULAR MOTILITY (INCLUDING ACCOMMODATIVE) DEFICITS

- GOODRICH ET AL. JRRD 2007

Common Deficits

<table>
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<th>Non-Blast-related TBI (n=25)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Convergence</td>
<td>30.4%</td>
<td>23.8%</td>
<td>36%</td>
</tr>
<tr>
<td>Accommodation</td>
<td>21.7%</td>
<td>23.8%</td>
<td>20%</td>
</tr>
<tr>
<td>Saccades/pursuit</td>
<td>19.6%</td>
<td>4.8%</td>
<td>32%</td>
</tr>
</tbody>
</table>
Clinical Research on Vision and TBI

OCCURRENCE OF OCULAR MOTILITY (INCLUDING ACCOMMODATIVE) DEFICITS

- RECENST STUDIES—reporting on non-selected patient samples with TBI


    - Compared the occurrence of vision dysfunctions for in- (n=108) versus out- (n=125) patient TBI.

Clinical Research on Vision and TBI

OCCURRENCE OF OCULAR MOTILITY (INCLUDING ACCOMMODATIVE) DEFICITS

- Beneficial to know that when evaluating patients in a non-selected, TBI sample, over 20% of patients may present with symptoms/signs of deficits of:

  - Convergence (36-48%)
  - Accommodation (20-47%)
  - Saccades/pursuit (23-32%)

Clinical Research on Vision and TBI

TREATMENT OF OCULAR MOTILITY (INCLUDING ACCOMMODATIVE) DEFICITS


  - Identified a series of patients with TBI who had vertical deviations. They gave a survey pre- and post-prescribing prism glasses and found a 71.8% reduction in symptoms with the prism glasses.

Clinical Research on Vision and TBI

TRAINING OF OCULAR MOTILITY (INCLUDING ACCOMMODATIVE) DEFICITS


  - PURPOSE: To report the efficacy of conventional in-office vision therapy in in our selected, visually-symptomatic, out-patient sample with TBI (n=33) and CVA (n=7).
Clinical Research on Vision and TBI: TRAINING OF OCULAR MOTILITY (INCLUDING ACCOMMODATIVE) DEFICITS

- Ciuffreda et al., Optometry, 2008

METHODS: The patient sample included those who had started and completed their vision therapy in the proposed time period (10/2000 through 10/2003):

- 33 (out of 160) persons with TBI
- Symptoms and signs noted for each subgroup, with some patients having multiple symptoms or signs.
- Success = reduction of at least 1 primary symptom and normalization of at least 1 clinical sign.

RESULTS:

30/33 of those with TBI improved with vision therapy

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>Total completing vision therapy</th>
<th>Total improving after vision therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>TBI</td>
<td>33</td>
<td>30</td>
</tr>
<tr>
<td></td>
<td>10-14</td>
<td>15-20</td>
</tr>
<tr>
<td></td>
<td>21-25</td>
<td>26-30</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Vision Symptom</th>
<th># TBI reporting the symptom</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ocular motility difficulty when reading</td>
<td>27</td>
</tr>
<tr>
<td>Eyestrain</td>
<td>18</td>
</tr>
<tr>
<td>Diplopia (at near more so than far viewing distances)</td>
<td>18</td>
</tr>
<tr>
<td>Headaches</td>
<td>11</td>
</tr>
<tr>
<td>Visual fatigue</td>
<td>5</td>
</tr>
<tr>
<td>Near blur</td>
<td>3</td>
</tr>
<tr>
<td>Sliding together of text words</td>
<td>1</td>
</tr>
<tr>
<td>Increased sensitivity to visual motion</td>
<td>1</td>
</tr>
<tr>
<td>Avoidance of near tasks</td>
<td>1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Vision Sign</th>
<th># TBI reporting the sign</th>
</tr>
</thead>
<tbody>
<tr>
<td>Receded near point of convergence</td>
<td>23</td>
</tr>
<tr>
<td>Abnormal DEM test</td>
<td>23</td>
</tr>
<tr>
<td>Reduced near positive relative vergence (NBO) range</td>
<td>16</td>
</tr>
<tr>
<td>Reduced near vergence ranges</td>
<td>9</td>
</tr>
<tr>
<td>Binocular suppression during testing</td>
<td>3</td>
</tr>
<tr>
<td>Impaired versional ocular motility</td>
<td>2</td>
</tr>
<tr>
<td>Nausea during testing</td>
<td>1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th># of vision symptoms</th>
<th>Pre-Therapy (# TBI reporting symptoms)</th>
<th>Post-Therapy (# TBI reporting symptoms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>11</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>11</td>
<td>1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th># of vision signs</th>
<th>Pre-Therapy(# of TBI manifesting vision signs)</th>
<th>Post-Therapy(# of TBI manifesting vision signs)</th>
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<td>3</td>
<td>12</td>
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Kapoor N, Thiagarajan M, Clark JF, et al. Beneficial to know that vision therapy (for those with TBI) can: reduce vision symptoms, normalize vision signs using standard clinical testing.

In the future, investigate efficacy of treatment (with vision therapy) for accommodative/ocular motility conditions using:

- Larger sample sizes
- Multicenter
- Non-selected patient samples (civilian and/or military) should involve larger samples of:
  - TBI
  - Concussion

The Ciuffreda et al. 2007 retrospective, Goodrich et al. 2007, and Cockerham et al. 2009 papers:

- Concurred (in general) with a report of an elevated occurrence in TBI (relative to a normal, non-neurologically-impaired reference population) of ocular motor deficits (convergence insufficiency, deficits of saccades, accommodative insufficiency)

- Two case reports (one TBI and one CVA) showing pre-/post-therapy objective eye movement recordings and subjective symptoms.

- A series of 9 TBI and 5 CVA showing pre-/post-therapy objective eye movement recordings and subjective symptoms.


TYPICAL VISION DEFICITS FOLLOWING CONCUSSION/mTBI

Optical Management Options
- Place patient with single vision spectacle lenses due to lens design and try to address small magnitudes of:
  - Astigmatism
  - Hyperopia
  - Anisometropia-contact lenses can be helpful
  - Esophoria/Exophoria/Hyperphoria/Fixation disparity
- Increased sensitivity to light is often present in this patient population. Consider prescribing a light tint and/or anti-reflective coating
- *When these optical issues are not compensated for, visual and visual-vestibular symptoms are worse*

Rehabilitation Treatment Options
- Habituation/Adaptive
- Compensatory
- Restorative
- Some combination of the above

Common Vision Deficits/Their Primary Associated Symptoms Evident Following Concussion/mTBI

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Accommodation: Neurological Correlates
- Pre-motor neural components:
  - mediated by the autonomic nervous system (ANS)
  - primarily the parasympathetic system to stimulate or increase the accommodative response
  - secondarily the sympathetic system to inhibit or reduce the accommodative response

Accommodation: Neurological Correlates
- Retinal cones stimulated by defocus blur
- Summated blur signals transmitted through magnocellular layer of lateral geniculate nucleus (LGN) to primary visual cortex (V1)
- Contrast-related neurons in V1 alter signal and send signal to parieto-temporal (PT) area to the Edinger-Westphal (EW) nucleus in the pre-tectum
- At the EW nucleus, autonomic input (from parasympathetic fibers) is received to form the combined motor command
Accommodation: Neurological Correlates

- Combined autonomic and motor neurons travel via the oculomotor nerve from the EW nucleus to the ciliary ganglion (where sympathetic fibers join CN III without synapsing) to the short ciliary nerve and then to the ciliary muscle.
- End result:
  - a change in the contraction of the ciliary muscle
  - consequent change in crystalline lens shape and effective state of accommodation

Accommodative Deficits: Associated Symptoms

- Constant/intermittent blur at far or near
- Intermittent blur due to infacility
  - Near-far blur
  - Far-near blur
- Symptoms associated with near vision tasks (i.e., reading/using computer/handicrafts):
  - Eyestrain/eye fatigue/browaches
  - Dizziness/nausea/motion sickness

Accommodative Deficits: Compensatory Treatment Options

- Lenses may be prescribed for near vision tasks either:
  - In lieu of restorative accommodative training
  - In conjunction with restorative accommodative training
  - Following restorative accommodative training

Accommodative Deficits: Restorative Treatment Options

- Equalize accommodative amplitudes
- Work on improving the weaker aspect of focusing; i.e., if a patient cannot:
  - Relax the accommodative state, work on near-far focusing
  - Increase the accommodative state, work on far-near focusing

Accommodative Deficits: Restorative Treatment Options

- Work on maintaining the ability to:
  - Rapidly change focus on command and repeatedly over time
  - Sustain focus for extended periods of time
- Training may be performed:
  - Using lenses
  - In free space regarding targets at different viewing distances

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Dry Eye: Etiology

OLD THEORY: Dry eye in TBI is related to:
• Poor lid hygiene
• Side effects of anti-depressant, anti-hypertensive, and anti-anxiety medications
• Prior refractive surgery, contact lens wear, facial nerve, and/or meibomian gland dysfunction

NEW THEORY (Cockerham et al., 2013 in Ocular Surface):
• Is not apparently associated with meds or prior issues
• Still requires assessment and should almost be viewed as a new condition
• May persist for months or years post-TBI


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Versional Ocular Motility: Neurological Correlates

• Integrated pre-motor neural activity occurs in similar areas for vertical saccades, horizontal saccades, and horizontal pursuit:
  • Frontal lobe (frontal eye fields, supplemental eye fields, dorsolateral prefrontal cortex, and cingulate eye field)
  • parietal lobe
  • basal ganglia
  • superior colliculus
  • cerebellum

Versional Ocular Motility: Neurological Correlates

• Vertical Saccades pre-motor neural area:
  • rostral mesencephalon

• Horizontal pre-motor neural components:
  • saccade: excitatory burst neurons in the paramedian pontine reticular formation (PPRF) => project directly to the oculomotor neuron for horizontal saccades
  • pursuit: pursuit neurons in the medial vestibular nuclei and prepositus hypoglossi => project directly to the oculomotor neuron for horizontal pursuit

Versional Ocular Motility: Neurological Correlates

• Integrated pre-motor neural components for fixation include:
  • frontal eye fields
  • supplemental eye fields
  • parietal eye fields
  • right prefrontal cortex (for attention)
  • right posterior parietal cortex (for attention)
Versional Ocular Motility: Associated Symptoms

- Reading-related difficulties
  - Slower reading speed
  - Loss of place/skipping or missing lines or words
  - Re-reading/misreading words or lines
  - Print seems to “swim” / “jumble” on the page
- Difficulty shifting to/tracking objects
- Dizziness/nausea/motion sickness

Versional Ocular Motility Deficits: Compensatory/Adaptive Treatment Options

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

Versional Ocular Motility Deficits: Restorative Treatment Options

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

Examples of versional oculomotor restorative treatment techniques:

- Small-angle (i.e., Ann Arbor/Michigan tracking, pencil/paper tracings and mazes, Pegboard Rotator, Groffman computer scan/search/coding/perceptual speed)
- Medium-angle (i.e., Hart Chart, Pegboard Rotator, Keystone Rotator, Groffman computer pegboard/visual motor integration/visual tracings)
- Large-angle (i.e., 4-corner saccades, Keystone Rotator)
**Common Vision Deficits/Their Primary Associated Symptoms Evident Following Concussion/mTBI**

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**Vergence Ocular Motility: Neurological Correlates**

- Pre-motor neural innervation lies in the mesencephalic reticular formation, with three of the better-studied types of vergence cells being:
  - tonic: respond to change in vergence angle
  - burst: respond to change in vergence velocity
  - burst-tonic: respond to changes in both vergence angle and velocity

**Vergence Ocular Motility Deficits: Associated Symptoms**

- Diplopia/ overlapping images/eyestrain *which is eliminated with monocular occlusion*
  - Constant or intermittent
  - At far or near
  - More evident in one position of gaze than another
  - After a brief (<10-15 minutes) time period of performing a visually-based task
  - Closing one eye or squinting when performing a prolonged visual task
  - Avoidance of prolonged vision-related tasks
  - Dizziness/nausea/motion sickness

**Vergence Ocular Motor Deficits: Compensatory Treatment Options**

- To compensate for constant diplopia, decompensated phoria, or fixation disparity, incorporate:
  - fusional prism, if possible
  - varying degrees of occlusion may be required if fusion is not achievable:
    - selective (to insure peripheral fusion, while inhibiting central simultaneous perception)
    - graded (i.e., using Bangerter foils or other such translucent materials to blur/degrade image)
    - complete (i.e., with an opaque eyepatch)

**Vergence Ocular Motor Deficits: Restorative Treatment Options**

- Once person presents with fusion (even if intermittent), then:
  - Stabilize vergence in primary gaze (ramp and step) at far and near viewing distances
  - Work on facility and sustainability of fusional vergence at far and near viewing distances
Vergence Ocular Motor Deficits: Restorative Treatment Options

- Integration with other modalities is important, including:
  - Visual motor integration
  - Auditory visual integration

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Vestibular Function

- Afferent systems
  - Vestibular
  - Somatosensory/proprioception
  - Visual—which is a significant contributing component!
- Efferent systems
  - Musculoskeletal
  - Ocular motor

Visual-Vestibular Interaction

- 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

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

Videonystagmography (VNG)

- A series of objective recordings to evaluate/aid in diagnosing patients who report dizziness or other balance problems
  - Uses goggles that can detect the smallest eye movements using infrared cameras
  - Computer analyzes video images to track movements of the pupil
Hearing Evaluation

- Certain vestibular conditions are accompanied by auditory symptoms
- Need to know if patient experiences:
  - Hearing loss (unilateral, asymmetric bilateral, symmetric bilateral, progressive, or fluctuating)
  - Aural fullness (constant or intermittent, unilateral, asymmetric bilateral, symmetric bilateral)
  - Tinnitus (constant or intermittent, unilateral, asymmetric bilateral, symmetric bilateral)

Visual-Vestibular Dysfunction: Associated Vision Symptoms

- Similar for vergence and versional ocular motility, emphasizing:
  1) Vestibular symptoms (often accompanied by nausea, vomiting, and/or headache), such as:
     a) Dizziness
     b) Lightheadedness
     c) Vertigo
  2) *Increased dizziness and/or disequilibrium in/sensitivity to multiply-visualy stimulating environments. Examples of stimulating environments/tasks include:
     a) Supermarkets
     b) Malls
     c) Motion sickness in a moving vehicle
     d) Scrolling on a computer
     e) Watching television or movies

Visual-Vestibular Dysfunction: Associated Vision Symptoms

- Similar for vergence and versional ocular motility, emphasizing:
  3) Difficulty with eye/head dissociation
  4) Foreground/background discrimination difficulty

NOTE: ask about hypertension, heart problems, sinus problems, tinnitus, changes or asymmetry in audition, as well as aural fullness or pain.

Versional Ocular Motility Deficits and Vestibular Dysfunction: Treatment Options

- Same as for versional oculomotor deficits without vestibular dysfunction, except:
  - Start at a slower velocity and lower number of repetitions of saccades and pursuit, while patient is seated and minimal targets in the background
  - Systematically and gradually increase the:
    - velocity of the ocular motility
    - number of targets in the background
  - Build to having the patient marching in place while performing these tasks in front of a multiply, visually-stimulating background

Vergence Ocular Motility Deficits and Vestibular Dysfunction: Treatment Options

- Same as for vergence ocular motility deficits without vestibular dysfunction, except:
  - After stabilizing fusional vergence in primary gaze under static conditions, then stabilize:
    - vergence 30 degrees right gaze (ramp, step) and then 30 degrees left gaze (ramp, step)
    - dynamic vergence while the patient is performing a slow horizontal VOR (approximately 40-60 rotations per minute)
Vergence Ocular Motility Deficits and Vestibular Dysfunction: Treatment Options

- After stabilizing horizontal fusional vergence and a slow horizontal VOR, then stabilize:
  - vergence 25 degrees upgaze (ramp, step) and then 25 degrees downgaze (ramp, step)
  - dynamic vergence while the patient is performing a slow vertical VOR (approximately 40-60 rotations per minute)

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Photosensitivity: Neurological Correlates

- Elevated light sensitivity (to all lights OR specifically to fluorescent lights) despite unremarkable ocular health:
  - No evident ocular inflammation or infection

- Current hypothesis for neural mechanism:
  - Cortical or subcortical substrates resulting in anomalous dark and light adaptation and associated filtering/processing deficits when in very bright or very dim lighting
  - Precise location of neural substrate: not yet localized

Compensatory Treatment Options

- Incorporation of tints with spectacle correction (30-40% tint for indoors, 80-85% tint for outdoors) for photosensitivity that is:
  - General to all lights (using either brown or gray tints)
  - Selective/Specific for fluorescent lighting (using either FL41, blue or gray tints)
  - Wearing brimmed hats/caps
Visual Processing: Neurological Correlates

- More than just occipital cortex
- Multiple areas of the brain with two principal, parallel interacting pathways:
  - Ventral stream (a.k.a. the "what is it?")
  - Dorsal stream (formerly the "where is it? or "how is it?")

**Ventral Stream**

Pathway
- V1 in the occipital cortex moving anteriorly through V2, the ventral posterior aspect of V3, V4, and finally reaching the posterior inferior temporal lobe for processing
- Changes in ventral stream processing directed by the ventral lateral prefrontal cortex (VLPFC)

**Purpose**
- Uses a representational system that is rich and detailed, but not precise metrically, for:
  - form perception
  - object identification (i.e., examines the visual array and identifies different objects in the scene)

**Dorsal Stream**

Pathway
- V1 in the occipital cortex moving anteriorly through V2, the dorso-medial area of V3, the middle temporal area (V5/MT), and finally reaching the parietal lobe for final processing.
- Changes in dorsal stream processing directed by the dorso-lateral prefrontal cortex (DLPFC)

**Purpose**
- Uses egocentric coding of location & orientation of goal object for:
  - spatial representation via the inferior parietal lobule
  - visually-guided action and motion perception of objects, as well as ocular and limb motility, in the superior parietal lobule

**Visual Processing: Dorsal-Ventral Stream Mis-communication**

- Example in which patient presents with blur, eyestrain, and difficulty tracking when reading/ambulating/using a computer:
  - Unremarkable (involves more ventral stream processing):
    - visual acuity
    - perimetry
  - Impaired ocular motility (involves more dorsal stream processing):
    - vergence, accommodation
    - pursuit
    - saccades
    - fixation
- Sometimes, the patient has difficulty computing the location of the object in visual space (dorsal stream issue), which impedes basic ocular motility (i.e., vergence, accommodation, pursuit, saccades, fixation)

**Summary**

- The purpose of today's presentation was to increase familiarity and understanding with respect to:
  1. Why sensorimotor vision function may be impaired following concussion/mTBI
  2. The members of the inter-professional health care management team
  3. The vision provider’s associated role in managing such vision deficits. To find an optometrist specializing in neuro-optometric rehabilitation near you:
    - www.covd.org
    - www.nora.cc
Summary

The purpose of today’s presentation was to increase familiarity and understanding with respect to the:

4) behavioral and visual considerations when evaluating patients with concussion/mTBI
5) high-yield vision examination for patients with mTBI/concussion and evidence-based medicine support regarding the occurrence of and treatment for vision problems
6) typical residual vision disturbances (amenable to non-surgical treatment interventions) evident following concussion/mTBI along with:
   - neurological correlates
   - associated symptoms
   - possible treatment options

Some (not all!) Evidence-Based References For Vision and Rehab

Accommodation: Relevant Publications


Versional Ocular Motility: Relevant Publications

**Versional Ocular Motility: Relevant Publications**

**Visual-Vestibular Dysfunction: Relevant Publications**

**Visual Processing Relevant Publications**

**Dry Eye: Relevant Publications**
Photosensitivity: Relevant Publications


Seminal References


