Visual Dysfunction in Brain Injury

Debra A. Lehr, OD, FAAO
Learning Objectives

Recognize the signs and symptoms of visual dysfunction related to brain injury

Become familiar with Post Trauma Vision Syndrome (PTVS) and Visual Midline Shift Syndrome (VMSS)

Understand diagnostic and therapeutic strategies available through neuro-optometric rehabilitation
Prevalence of Visual Dysfunction Post-TBI
Retrospective study of 160 TBI patients
(AOA Journal 2007)

90% of TBI patients suffer from visual dysfunctions

Review of Neuro-Visual Processing

70% of all sensory processing in the entire body is directly affected by information coming from the two eyes.

Optic Nerve: 1,000,000 nerve fibers per eye provide pathways for the visual information processing.

8 Cranial Nerves are involved in the visual process.

Visual dysfunction following brain injury may be directly related to structural damage or may be a result of dysregulation between vision and sensorimotor information.
Cranial Nerves related to the visual system

CN II  Optic Nerve (sees)
CN III  Oculomotor (moves eyes/constrict pupil/accommodate)
CN IV  Trochlear (SO: rotates eyes/intorsion, moves eyes down)
CN V  Trigeminal (corneal sensitivity)
CN VI  Abducens (LR: moves eye out)
CN VII  Facial (closes eye lid)
CN VIII  Vestibulocochlear (VOR)
CN XI  Accessory (VOR)

We need the eyelids open and eyes pointed toward the visual target (stimulus) to start the visual process........
Photoreceptors in the Retina

Cones

- 6 Million
- Central 10 degrees - high resolution
- Photopic vision, not very light sensitive (direct light)
- Color vision - trichromatic (photopsin)
  - 3 types of cones: L (red), M (green), S (blue) *variable

Rods

- 120 Million
- Scotopic vision, light sensitive (even scattered light)
- Achromatic (rhodopsin)
Pathway of Light to the Brain

Photoreceptors synapse with bipolar cells.

Bipolar cells synapse with ganglion cells.

Horizontal cells and amacrine cells provide lateral connections in the retina to modify signal activation. *Stimulus analysis begins in the RETINA.*

More photoreceptors connect to a single ganglion cell in the peripheral retina, making the combined distribution more sensitive to light. However, the convergence of many photoreceptors onto a single ganglion cell decreases the image sharpness.
Ganglion Cells in the Retina

1.2 - 1.5 million Ganglion Cells compared to 126 million photoreceptors

Information from photoreceptors via intermediate neurons:

- Bipolar Cells and Retinal Amacrine Cells

Retinal ganglion cell extends via its axon to the brain
The Pathways of Ganglion Cells

Parvocellular or P pathway

Magnocellular or M pathway

Koniocellular or K pathway

Intrinsically Photosensitive ganglion cells

Retino-coliccular pathway
Parvocellular Pathway

Approximately 80% of all retinal ganglion cells

Input from few photoreceptors (cones >> rods)

*Slow conduction velocity*

Responsive to change in *color*

Not as responsive to changes in *contrast*
Magnocellular Pathway

Approximately 10% of all retinal ganglion cells

Input from many photoreceptors (rods > cones)

*Fast conduction velocity*

Responsive to *low contrast* stimuli

*Not sensitive to changes in color*
Koniocellular Pathway

Roughly 10% of ALL rods and 2% of S cones (blue)

Input from moderate number of rods and cones

Moderate spatial resolution

Moderate conduction velocity

Moderate responsiveness to contrast
Intrinsically Photosensitive Ganglion Cells

Approximately 1-3% of retinal ganglion cells

Projects to hypothalamus for circadian rhythm

Projects to midbrain for pupillary control
Retino-Collicular Pathway

R-C ganglion cells project to the Superior Colliculus
Critical for coordination of eye movement with body
and head movement
Ganglion Cell Axons terminate in 4 Nuclei:

- **Lateral Geniculate Nucleus** of the **Thalamus** for visual perception *(Parvo, Magno, Konio)*
- **Superior Colliculus** of the **Midbrain** for control of eye movement *(Retino-Collicular)*
- **Pretectum** of the **Midbrain** for pupillary light reflex *(IP)*
- **Suprachiasmatic Nucleus** of the **Hypothalamus** for control of diurnal rhythms and regulation of melatonin from pineal gland *(IP)*
Let’s Start with Visual Perception via LGN

Bimodal visual processing
- **Ventral Pathway** - Parvocellular GC
  - 4 Outermost Layers of LGN
  - To inferior temporal cortex
  - WHAT is the visual stimulus
- **Dorsal Pathway** - Magnocellular GC
  - 2 Innermost Layers of LGN
  - To posterior parietal cortex
  - WHERE is the visual stimulus
Overview of Visual Processing

**Dorsal pathway**: Where?
To post. parietal cortex
**MOTION** (+ motion illusions)

**Ventral pathway**: What?
To inferior temporal cortex
**Colour and form** (+ illusory contours)

**Bipolar cells**
- Midget
- Amacrine

**Ganglion cells**
- Parvocellular cells (majority, smaller receptive fields, slower, **colour** vision, sustained response)
- Magnocellular cells (larger receptive fields, convergence, faster, more sensitive, detect **motion**, transient response)

**LGN**
- 4 parvocellular layers
- 2 magnocellular layers

**Visual Cortex**
- Spiny stellate neurons
  - 4th layer of sublayer A = 4C-alpha, simple cell?
  - Spiny stellate neurons, 4th layer of sublayer B = 4C-beta, complex cell?
Bi-Modal Visual Process

Ventral Pathway - Parvo cellular - Focal System (Micro)

Detail discrimination

Tells you WHAT an object is

Identification, Attention, Concentration, Conscious

Slow speed in processing
Bi-Modal Visual Process

Dorsal Pathway - Magnocellular - Ambient System (Macro)

Spatial orientation matched with sensorimotor

*Tells you WHERE an object is*

Posture, Balance, Movement, Preconscious, Proactive

Rapid speed in processing
But before we can determine WHAT and WHERE....

We need to:

- Point our eyes in the direction of the visual target
- Maintain a clear, steady image on the retina
- Coordinate the eyes to send a uniform (single) image to the brain
Structures and Pathways of Visual Processing.....
Fixation / Steady Gaze

Maintain gaze on a single location/stimulus (EYE)

Requires Visual Attention

Fixation involves:

Horizontal Gaze Center
- Pons 6th CN nucleus

Vertical Gaze Center
- Rostral Midbrain CN IV Nucleus

Cerebellum
- Integrates head and body position and eye position
Pursuits

Smooth eye movements used to follow a moving target which allows a clear, stationary image on the retina

Duction - Monocular
Versions - Binocular

Limited in speed: 30 degrees/second

Descending pathways from temporo-parieto-occipital junction and frontal eye fields (FEF) connect in the pons and innervate the cerebellum, which then excites the sixth cranial (abducens) nerve nucleus
Saccades

RAPID simultaneous shifting of fixation
700 degrees/second

Voluntary Saccades:
The frontal eye fields unlock fixation and begin deliberate movement to visible targets or to the predicted location

Reflexive Saccades:
The parietal eye fields begin reflexive/stimulus generated movement based on the abrupt appearance of visual targets

The supplementary eye fields assist in initiating and controlling saccades made during motor movement (of head and body)
Fusion and Vergence

Simultaneous movement of eyes in opposite directions to maintain fusion

Convergence:
Adduction of eyes to view an object at near

Divergence:
Abduction of eyes to view an object at distance

Maintaining fusion during vergences involves complex cerebro-brainstem-cerebellar pathways
Vestibulo-Ocular Reflex

VOR stabilizes an image on the retina by producing eye movements in opposite direction to head movement.

Semicircular canals signal to vestibular nuclei which excite the sixth cranial (abducens) nerve nucleus.

Test: Quick head thrusts while fixating.
Extensive Pathways of Visual Processing
Frontal Lobe

Frontal Eye Fields (FEF)

- Anticipation and expectation
- Organization for pursuits and saccades
- Visual decision making referencing fixations and ocular motility
- Spatial organization affecting sequence of ocular motility and visual decision making

Motor cortex

- Orbitofrontal - risk and reward
- Primary motor - interacts with brain stem for adaptability
- Premotor - organization of visual motor sequence
Parietal Lobe

Matching vision, auditory, and tactile sensory information

Posterior Parietal Cortex (PPC)
- Spatial match with FEF and superior colliculus (SC)
- Spatial consciousness affecting position awareness
- Receives input from SC, occipital cortex, FEF, and thalamus
- Provides output to FEF, SC, occipital lobe, and temporal lobe
Remember Visual Perception is Bimodal

- **Ventral Pathway** - Parvocellular GC
  - 4 Outermost Layers of LGN
  - To **inferior temporal cortex**
  - **WHAT** is the visual stimulus

- **Dorsal Pathway** - Magnocellular GC
  - 2 Innermost Layers of LGN
  - To **posterior parietal cortex**
  - **WHERE** is the visual stimulus
Temporal Lobe

Matching visual information with auditory information
Temporal relationships established with FEF spatial context
Receives input from the occipital lobe, FEF and SEF, thalamus, cerebellum, inferior colliculus, parietal lobe
Provides output to FEF and SEF, parietal lobe, midbrain and cerebellum, and occipital lobe
Occipital Lobe

Primary conscious mode of processing the image field

Serves the M-P-K cellular pathways in segregation

Retinotopic mapping of the image field

Facilitates focal vision process

 Receives input from all cortices, midbrain, and brainstem

Provides output to all cortices, midbrain, and brainstem
Brainstem

Connects the cerebrum with the spinal cord
Consists of the midbrain, medulla oblongata, and pons
Brainstem

Motor and sensory neurons relay signals between brain and spinal cord

Coordinates motor control signals sent from the brain to the body

Establishes early reflex patterns for sensorimotor organization

Serves to provide trunk stability and association of trunk-on-body movement
Brainstem

Provides proprioceptive field for sensory organization spatially
Controls life supporting **autonomic functions** of the peripheral nervous system
Receives input from spinotectal tract for sensorimotor information, control of autonomic functions
Provides output to SC of midbrain, FEF, parietal and temporal lobes
Midbrain

Superior Colliculus organizing ambient visual spatial process
Feed-forward to 99% of the cortex
Function for early spatial context for fusion
Match with sensorimotor information
Receives primary input from brainstem, cerebellum, vestibular system, occipital cortex, thalamus
Provides output to occipital cortex, FEF and SEF, parietal lobe, thalamus, cerebellum, and vestibular system
Ganglion Cell Axons terminate in 4 Nuclei:

Lateral Geniculate Nucleus of the Thalamus for visual perception (Parvo, Magno, Konio)
Superior Colliculus of the Midbrain for control of eye movement (Retino-Collicular)
Pretectum of the Midbrain for pupillary light reflex (IP)
Suprachiasmatic Nucleus of the Hypothalamus for control of diurnal rhythms and regulation of melatonin from pineal gland (IP)
Visual Dysfunction in Brain Injury

What part of the brain isn’t involved in visual processing?

Not surprising that 90% of TBI patients suffer from visual dysfunction.
Visual Signs and Symptoms of Brain Injury

Moderate or severe brain injury often presents with structural lesions and more axonal shearing, resulting in ocular motor neuropathies, optic neuropathies, and orbital pathologies.

Mild TBI or concussion require evaluation for deficits in visual function, executive function, visual attention, and visual memory.

Damage to Structures and Pathways

Traumatic Optic Neuropathy
  Unilateral or bilateral presentation
  Reduced VA, + RAPD, dyschromatopsia, VF defect

Chiasm and Retrochiasmal Pathway Trauma
  Bilateral VF defect:
    Chiasm: Bi-temporal hemianopsia
    Retrochiasmal: Homonymous Hemianopsia

Ocular Motor Neuropathies
  Diplopia, ptosis, efferent pupillary defect

Brainstem Injury
  Pupillary and ocular motility dysfunction
Visual Field Defects

- Unilateral
- Bitemporal
- Hemianopsia
- Quadrantanopsia
- Pie in the Sky
  - temporal lobe
- Other Superior field loss
  - AION
- Scattered islands
Unilateral Spatial Inattention ("Visual Neglect")

Inability to be completely aware of visual surroundings

*Cognitive deficit

Unawareness of the deficit is central feature
Line Bisection Cross Out Task
Signs of Post Trauma Vision Syndrome

- Exotropia or high exophoria
- Convergence insufficiency
- Accommodative dysfunction
- Myopic shift
- Dry eye syndromes: altered tear / lid function
- Spatial disorientation or balance issues
- Oculomotor and Visual-Motor dysfunction
- Unstable ambient vision (Magnocellular)
- Visual-perceptual processing dysfunction
Exotropia or high exophoria

Exotropia: Eyes turned outward

Exophoria: “Resting posture”
- Tendency of the eyes to deviate outward
- When dissociated (prisms), the eyes will appear to diverge away from one another
Convergence Insufficiency

• Binocular (eye teaming) problem at near
• Eyes have a tendency to drift outward when reading or doing close work
• Near exodeviation greater than distance deviation
• When the eyes drift out, the person is likely to have double vision
• Associated with headaches, eyestrain, fatigue
Convergence Insufficiency
http://www.convergenceinsufficiency.org/


Accommodative dysfunction

• Reduction of the ability to focus prematurely leading to the need for reading correction or bifocals
  • Accommodation progressively declines throughout our life
  • After age forty, reading lenses or bifocals required
• Spasms of accommodation may cause over focusing, presenting as a temporary increase in myopia
• Impaired ability to interpret spatial relationships accurately and/or precisely coordinate the focus and convergence mechanism
Myopic shift

• Spasms of accommodation may occur causing over focusing and may present as a temporary increase in myopia

• If this additional myopia is “corrected” with increased Rx in glasses/contact lenses, the patient may report more headaches and blur at near
Dry eye syndromes: altered tear / lid function

- Rate of blinking may slow and the completeness of the blinks may decline. The patient may be making only occasional partial blinks. This leaves the lower portion of the cornea to dry and become uncomfortable.
- Quality of tear production may be compromised
- If the cornea is not kept moist, a dry eye or exposure keratitis may develop. It is much like chapped lips and leads to dry, burning, gritty eyes.
Spatial disorientation or balance issues

- Approximately twenty percent of the nerve fibers from the eyes interact with the vestibular system.
- A variety of visual dysfunctions can cause or associate with dizziness and balance problems.
- Pt may misperceive their position in the environment.
- Pt may show a tendency to lean to one side, forward and/or backward.
Oculomotor and Visual-Motor dysfunction

- Limited ability to maintain fixation/steady gaze
- Difficulty smoothly following a moving target
- Unable to shift gaze quickly and accurately from target to target
- Poor VOR
Unstable ambient vision (Magnocellular)

- Spatial orientation not accurately matched with sensorimotor
  
  *Disconnect of WHERE an object is

- Deficits in Posture, Balance, Movement, Preconscious

- Loss in speed of ambient visual processing
Visual-perceptual processing dysfunction

- **Visual-Motor Integration** - Eye-hand, eye-foot, and eye-body coordination
- **Visual-Auditory Integration** - The ability to relate and associate what is seen and heard
- **Visual Memory** - The ability to remember and recall information that is seen
- **Visual Closure** - The ability "to fill in the gaps", or complete a visual picture based on seeing only some of the parts
- **Spatial Relationships** - The ability to know "where I am" in relation to objects and space around me and to know where objects are in relation to one another
- **Figure-Ground Discrimination** - The ability to discern form and object from background
Visual Dysfunction in Brain Injury

The *symptoms* of visual dysfunction align with the signs...

What symptoms or visual complaints do we routinely find following brain injury?
Symptoms of Post Trauma Vision Syndrome

- Avoidance of near tasks
- Oculomotor-based reading difficulties
- Eye tracking and eye focusing problems
- Eyestrain, Diplopia (double vision)
- Dizziness, Vertigo
- Vision-derived nausea
- Increased sensitivity to visual motion
- Visual inattention and distractibility
- Short-term visual memory loss
- Difficulty judging distances
- Light sensitivity
- Inability to tolerate visually complex environments
Visual Midline Shift Syndrome

Mismatch between the perceived egocentric visual midline and the actual physical midline
  Causes an expansion on one side
  Causes a contraction on the opposite side

May be caused by:
  Midbrain (ambient system) dysfunction
  Oculomotor imbalance
  Spatial shifts caused by unilateral hemispheric damage
Visual Midline Shift Syndrome

Signs and Symptoms

Floor may appear tilted
Walls and/or floor may appear to shift and move
Veering during mobility
Person leans away from the affected side
Feelings of imbalance or disorientation similar to vertigo
VISUAL MIDLINE SHIFT TEST

Visual Midline Shift To Right
VISUAL MIDLINE SHIFT TEST

Anterior Shift of Visual Midline
Anterior Visual Midline Shift
Diagnostic Strategies to Detect Visual Dysfunction Following Brain Injury

How do we identify Post Trauma Vision Syndrome?
Development of a mild traumatic brain injury-specific vision screening protocol: A Delphi study

Journal of Rehabilitation Research & Development (JRRD)

Volume 50 Number 6, 2013 Pages 757-768

Fixation

Visual Field Test

*Fixation monitor to assess gaze

Right Eye January

Right Eye February
Saccades

Voluntary Saccades:

Developmental Eye Movement or King-Devick
2 near targets: Beads on a stick
Fingers
Tip of pen
Red/Green Avery Dots
20/40 letters
Infrared technology to track movements (Visagraph III, Readalyzer, etc)

Reflexive Saccades:

Computer generated stimulus eliciting motor response (BVA)
Infrared tracking ocular response to stimulus
King Devick
Developmental Eye Movement Test
Infrared technology to track oculomotor movement
Pursuits

Track a moving target at no more than 30 degrees per second

Targets:

- Penlight/transilluminator
- Tip of pen
- Bead on a stick
- Fingers
- Pencil eraser

Computer generated targets (BVA)
Fusion and Vergence

Free space testing
- Near point of convergence (NPC)
- Handheld prism bar of fusional amplitudes, phorias
- Cover test of phoria at distance and near
- Near point fixation disparity (Vergel)
- Stereopsis, Worth 4 Dot

Phoropter-based testing
- Fusional amplitudes with base in and base out prism
- Computer-based random dot stereopsis testing
Near Point Fixation Disparity
Worth 4 Dot
Accommodation

Near point visual acuity

NRA/PRA: Negative Relative Accommodation
Positive Relative Accommodation

Accommodative amplitude

Binocular cross cylinder

Near point fixation disparity
Accommodative Testing (Visual Acuity)

Accommodation for a near target

Accommodation for a far target
Visual Perceptual Skills

Computer programs (Visual Perceptual Testing)

Motor free testing available
  Visual discrimination
  Visual sequential memory
  Visual memory
  Figure-ground
  Visual spatial relationships
  Visual closure
  Form constancy
Visual Discrimination: Name 10 differences ....
Visual Form Constancy:

Which shape was cut out of the box? The shape can be turned.

1  2
3  4
Figure-Ground: How many horses do you see?
Visual Closure: What is it?
Therapeutic Strategies for Visual Rehabilitation Following Brain Injury

What do we do about visual dysfunction once deficits are identified?
Lubricating Drops (Artificial Tears)

- Lubricating eye drops to reduce visual distortion/discomfort
- Do not use “Get the red out” drops
- Effectivity of brands vary based on individual tear film quality
- Use at least 2 x day, Reminder: Bottle next to your toothbrush
- If using a computer, drops may be required more frequently
  - Review 20-20-20 Rule
Sunglasses and tinted lenses/overlays

GOAL: Reduce glare and decrease photophobia (light sensitivity)

• Outside: Sunglasses with UV protection, hat with a visor

• Inside: Tinted lenses or overlays

• Computer/Electronic devices: Filters/screens/apps are available to reduce blue-violet light absorption
Prescription Glasses

- May be indicated for distance and near (full time wear)
  - More sensitive to mild Rx, discrepancy in binocular balance
- Rx for NEAR work only (reading/computer use/task must be within the length of your arm) - low “Plus” lenses
- Anti-reflective lenses recommended
- Prism to compensate for binocular vision disorder
  - Base In for convergence insufficiency
  - Base Down for near vision correction
  - Field expansion for visual field defect (not for inattention)
In Office Vision Rehabilitation

• Coordinated care with OT for vision rehabilitation
  • Oculomotor skills
  • Binocular vision: Accommodation and vergence therapy
  • Visual perceptual skills

• Coordinated care with PT for vestibular-ocular dysfunction

• Home therapy to reinforce skills acquired in office (HTS)
Workbooks

- Oculomotor Function: Tracking and Saccades
- Visual Perception: Visual Discrimination, Memory, Figure Ground, Motor and Visual Integration
Binasal Occlusion/Glasses Taping (sectional)

- To remove the portion of vision where the images from the two eyes overlap, reducing visual ‘background’ noise
- To help our eyes to work independently, rather than struggling when working together.
  - Our brains tend to focus on the overlapping part of the images that are misaligned, making it difficult to focus on anything else.
- Eliminating portions of our central focal vision stimulates spatial visual system/ambient processing
- Moderate to severe brain injuries
Final thought.....

Potential for mild traumatic brain injury prevention?

An Exploratory Study of the Potential Effects of Vision Training on Concussion Incidence in Football

ABSTRACT Background:

Vision training has become a component of sports enhancement training; however, quantifiable and validated improvement in visual performance has not been clearly demonstrated. In addition, there is minimal literature related to the effects of vision training on sports performance and injury risk reduction. The purpose of the current investigation was to determine the effects of vision training on peripheral vision and concussion incidence.
METHODS:

Vision training was initiated among the University of Cincinnati football team at the beginning of the 2010 season and continued for four years (2010 to 2013). The sports vision enhancement was conducted during the two weeks of preseason camp. Typical vision training consisted of Dynavision D2 light board training, Nike strobe glasses, and tracking drills. Nike Strobe glasses and tracking drills were done with pairs of pitch-and-catch drills using footballs and tennis balls, with instructions to vary arc, speed, and trajectory. For skilled players, “high ball” drills were the focus, whereas for linemen, bounce passes and low pitch drills were stressed. Reaction time data was recorded for each athlete during every Dynavision D2 training session. We monitored the incidence of concussion during the four consecutive seasons of vision training, as well as the previous four consecutive seasons, and compared incidence of concussions (2006 to 2009 referent seasons v. 2010 to 2013 vision training seasons).
RESULTS:

During the 2006-2013 pre- and regular football seasons, there were 41 sustained concussion events reported. The overall concussion incidence rate for the entire cohort was 5.1 cases per 100 player seasons. When the data were evaluated relative to vision trained versus referent untrained player seasons, a statistically significant lower rate of concussion was noted in player season in the vision training cohort (1.4 concussions per 100 player seasons) compared to players who did not receive the vision training (9.2 concussions per 100 player seasons; p<0.001). The decrease in injury frequency in competitive seasons with vision training was also associated with a concomitant decrease in missed play time.
DISCUSSION:

The current data indicates an association of a decreased incidence of concussion among football players during the competitive seasons where vision training was performed as part of the preseason training. We suggest that better field awareness gained from vision training may assist in preparatory awareness to avoid concussion causing injuries. Future large scale clinical trials are warranted to confirm the effects noted in this preliminary report.
AUTHORS:

- Joseph F. Clark, PhD, ATC, Department of Neurology & Rehabilitation Medicine, University of Cincinnati, Cincinnati, Ohio
- Pat Graman, MA, ATC, Department of Education, University of Cincinnati, Cincinnati, Ohio
- James K. Ellis, OD, Department of Sports Medicine, University of Cincinnati, Cincinnati, Ohio
- Robert E. Mangine, MEd, PT, ATC, Associate Athletic Director of Sports Medicine, University of Cincinnati, National Director of Clinical Sports PT Residency, NovaCare Rehabilitation, Cincinnati, Ohio
- Joseph T. Rauch, DPT, SCS, ATC, Departments of Orthopaedic Surgery and Athletics, University of Cincinnati, Cincinnati, Ohio
- Ben Bixenmann, MD, Department of Neurosurgery, University of Cincinnati, Cincinnati, Ohio
- Kimberly A. Hasselfeld, MS, Department of Orthopaedic Surgery Division of Sports Medicine, University of Cincinnati, Cincinnati, Ohio
- Jon G. Divine, MD, Department of Orthopaedic Surgery Division of Sports Medicine, University of Cincinnati, Cincinnati, Ohio
- Angelo J. Colosimo, MD, Departments of Orthopaedics & Athletics, University of Cincinnati, Cincinnati, Ohio
- Gregory D. Myer, PhD, FACSM, Division of Sports Medicine, Cincinnati Children’s Hospital Medical Center, Department of Pediatrics and Orthopaedic Surgery, University of Cincinnati; Sports Medicine Sports Health & Performance Institute, The Ohio State University, Columbus, Ohio; and The Micheli Center for Sports Injury Prevention, Boston, Massachusetts
Thank You!