

# EVIDENCE-BASED MANAGEMENT OF VISION DEFICITS FOLLOWING TRAUMATIC BRAIN INJURY

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## Objectives

- To identify:
  - the more common vision conditions evident following mild and moderate traumatic brain injury (TBI) and associated common ophthalmic terminology
  - evidence-based medicine support related to sensorimotor vision function following TBI in terms of:
    - elements for a high yield vision screening
    - occurrence
    - treatment efficacy
  - the underlying neurology of common vision conditions following mild and moderate TBI, as well as the primary vision symptoms and possible treatment options

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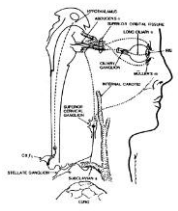
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## Vision and the Brain

• 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



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# TYPICAL VISION CONDITIONS AND ASSOCIATED VISION TERMINOLOGY

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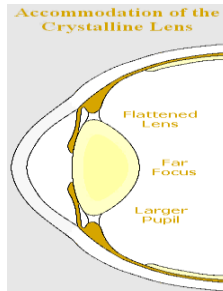
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## Terminology

- **Accommodation:** the ability to change focus and maintain a clear image of an object (when looking from far to near and vice versa), using the eye's crystalline lens-based mechanism.



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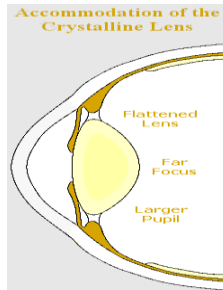
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## Terminology

- **Accommodative amplitude:**
  - the closest point of clear vision
  - typically performed monocularly.



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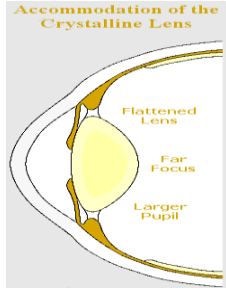
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### Terminology

• **Accommodative facility:**

- the ability to maintain clarity of vision when looking from near to far/far to near repeatedly, accurately, and on command.
- may be performed monocularly or binocularly.




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### Typical Vision Deficits Following TBI

Deficit of:	Primary Associated Symptom:
Accommodation	Constant/intermittent blur
Versional Ocular Motility	Slower, less accurate reading /difficulty sustaining gaze, shifting gaze, or tracking targets
Vergence Ocular Motility	Constant/intermittent eyestrain / diplopia <i>eliminated with monocular occlusion</i>
Visual-Vestibular Interaction	Disequilibrium exacerbated in multiply, visually-stimulating environments
Visual processing	Slower speed/impaired visual memory and visual-spatial processing
Visual Field Integrity	Missing a portion of vision /overall restriction
Tear Film Integrity	Distorted clarity/gritty sensation, which <i>varies with blinking</i>
Light-Dark Adaptation	Elevated light sensitivity

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### Terminology

• **Versional ocular motility:**

- refers to the conjunctive movement of the eyes to follow targets moving laterally, vertically, or obliquely in one plane, with no change in depth (i.e., 2-dimensional eye movements in the x-y plane)
- may be tested monocularly or binocularly.
- includes
  - fixation
  - saccades
  - pursuit

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## Terminology

• **Fixation:** an eye movement in which the eyes are fixed on a target to maintain the target's image on the fovea and may be tested monocularly or binocularly.

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## Terminology

• **Saccades:** are rapid, step-like conjugate eye movements which redirect the line of sight from one position (or object) in space to another and may be tested monocularly or binocularly.

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## Terminology

• **Pursuit:** is a slow, continuous conjugate eye movement used when the eyes follow a slowly-moving object and may be tested monocularly or binocularly.

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## Typical Vision Deficits Following TBI

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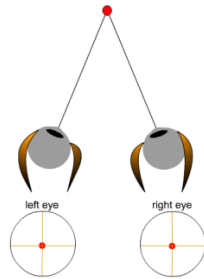
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### Terminology

- **Vergence:** the disjunctive movement of the eyes to track targets moving in depth (i.e., along the z-axis)
- **Fusion:** single, cortically-integrated vision under binocular viewing conditions




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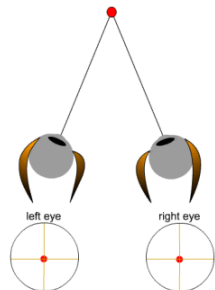
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### Terminology

- **Heterotropia (ie., strabismus):** the position of the eyes when fusion is not disrupted (i.e., under normal binocular viewing conditions)
- **Heterophoria:** the position of the eyes when fusion is disrupted




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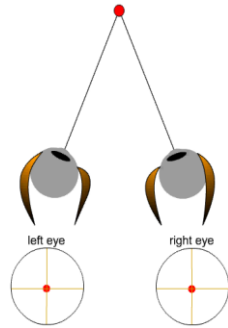
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## Terminology

- **Near point of convergence:** the closest point of binocular, fused, single vision.




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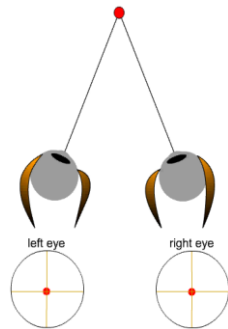
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## Terminology

- **Vergence facility:** the ability to maintain single vision when looking from near to far/far to near repeatedly, accurately, and on command. This is performed under binocular viewing conditions.
- **Stereopsis:** relative depth perception




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## Typical Vision Deficits Following TBI

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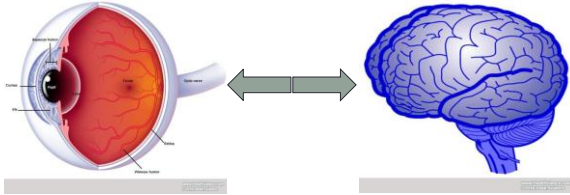
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## High Yield Vision Screening




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### High Yield Vision Case History/ Testing from 2013 JRRD

- Goodrich et al., 2013 JRRD article citation:
  - Goodrich, Martinsen, Flyg, Kirby, Asch, Brahm, Brand, Cajamarca, Cantrell, Chong, Dziadul, Hetrick, Huang, Ihrig, Ingalla, Meltzer, Rakoczy, Rone, Schwartz, and Shea (2013). Development of a mild traumatic brain injury-specific vision screening protocol: A Delphi study. JRRD Vol 50 (6): 757-768.
- Polled 16 experts (who work with the VA Medical Center and/or Army) in vision and TBI about their high-yield:
  - case history questions
  - testing procedures

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### High Yield Case History Vision-related Questions

#### Functional Vision and Reading-related

- Have you noticed a change in your vision since your injury?
- Are you more sensitive to light, either indoors or outdoors, since your injury?
- Have you had any double vision since your injury?\*
- Have you noticed any changes in your peripheral vision since your injury?\*
- Is your vision blurry at distance or near since your injury?
- Have you noticed a change in your ability to read since your injury?
- Do you lose your place while reading more now than before your injury?
- How long can you read continuously before you need to stop?\*
- Do you get headaches during/after reading more now than before your injury?
- Do you have more difficulty remembering what you have read now than before your injury?

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### Typical Vision Deficits Following TBI

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### High Yield Vision Screening Test Elements

- \*Case history
- \*Extraocular motility - assessment of eye:
  - Alignment (i.e., presence of heterotropia or heterophoria)
    - distance cover test
    - near cover test
  - Movement-range and quality
    - pursuit (may be performed monocularly or binocularly)
    - fixation and saccades (may be performed monocularly or binocularly)
  - Focusing and teaming ability
    - accommodation
      - near point of convergence (performed mid-way through and again towards the end of the exam to assess fatigue impact)

Goodrich, Martinsen, Flyg, Kirby, Asch, Brahm, Brand, Cajamarca, Cantrell, Chong, Dziadul, Hetrick, Huang, Ihrig, Ingalla, Meltzer, Rakoczy, Rone, Schwartz, and Shea (2013). Development of a mild traumatic brain injury-specific vision screening protocol: A Delphi study. JRRD Vol 50 (6): 757-768.

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### High Yield Vision Screening/Bedside Assessment

- Case history
- Optic nerve function
  - visual acuity
  - confrontation visual fields
  - color vision testing
- Extraocular motility function
  - fixation, saccades, and pursuit (may be performed monocularly or binocularly)
  - near point of convergence (performed binocularly)
  - stereopsis (performed binocularly)
- Optic nerve function: pupils

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## OCCURRENCE OF SENSORIMOTOR VISION PROBLEMS FOLLOWING TBI

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### Clinical Research on Vision and TBI

#### PATIENT PROFILE

Parameter	TBI (n=160)
Age range (years)	8 to 91
Mean age (years)	44.9
#of males	73
# of females	87
Years post-injury (range)	0.1-42.0
Mean years post-injury	4.5

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### Clinical Research on Vision and TBI

#### OCCURRENCE OF OCULAR MOTILITY (INCLUDING ACCOMMODATIVE) DEFICITS

**RESULTS** re: occurrence of sensorimotor vision conditions in a selected, visually-symptomatic TBI sample (n=160)

Vision Anomaly	TBI (%)	Most common anomaly
Accommodation	41.1	Accommodative insufficiency
Versional	51.3	Deficits of saccades
Vergence	56.3	Convergence insufficiency
Strabismus	25.6	Strabismus at near
CN palsy	6.9	CN III

Ciuffreda KJ, Kapoor N, Rutner D, Suchoff IB, Han ME, Craig S (2007). Occurrence of oculomotor dysfunctions in acquired brain injury: a retrospective analysis. Optometry, 78(4): 155-61.

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### Clinical Research on Vision and TBI

#### OCCURRENCE OF OCULAR MOTILITY (INCLUDING ACCOMMODATIVE) DEFICITS

- Beneficial to know that over 50% in a visually-symptomatic TBI sample may present with deficits of:
  - vergence ocular motility
  - versional ocular motility
  - most common anomalies for TBI were convergence insufficiency and deficits of saccades

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### Clinical Research on Vision and TBI

#### OCCURRENCE OF OCULAR MOTILITY (INCLUDING ACCOMMODATIVE) DEFICITS

- **RECENT STUDIES** –reporting on non-selected patient samples with TBI
- Regarding the military, studies by Drs. Gregory Goodrich and Glenn Cockerham support a high occurrence of vision dysfunctions in TBI

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### Clinical Research on Vision and TBI

#### OCCURRENCE OF OCULAR MOTILITY (INCLUDING ACCOMMODATIVE) DEFICITS

- › Goodrich et al. 2007 RESULTS re: the occurrence of sensorimotor vision deficits on patients at their center whether patient symptomatic or not and comparison blast TBI versus non-blast-related TBI

Common Deficits	Total TBI sample (n=46)	Blast-related TBI (n=21)	Non-Blast-related TBI (n=25)
Convergence	30.4%	23.8%	36%
Accommodation	21.7%	23.8%	20%
Saccades/pursuit	19.6%	4.8%	32%

Goodrich GL, Kirby J, Cockerham G, Ingalla SP, Lew HL; JRRD 2007; 44 (7) :929-936

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### Clinical Research on Vision and TBI OCURRENCE OF OCULAR MOTILITY (INCLUDING ACCOMMODATIVE) DEFICITS

› Cockerham et al. 2009 RESULTS re: compared the occurrence of vision dysfunctions for in-(n=108) versus out-(n=125) patient TBI.

Common Vision Deficit	In-patient TBI (n= 1 08)	Out-patient TBI (n= 1 25)
Strabismus	32%	8%
Accommodative insufficiency	31%	47%
Convergence insufficiency	40%	48%
Pursuit/saccade deficits	29%	23%
Diplopia	19%	6%

Cockerham GC, Goodrich GL, Weichel ED, Orcutt JC, Rizzo JF, Bower KS and Schuchard RA; JRRD 2009 46(6): 811-818

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### Clinical Research on Vision and TBI OCURRENCE OF OCULAR MOTILITY (INCLUDING ACCOMMODATIVE) DEFICITS

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

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

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### Clinical Research on Vision and Concussion: Saccadic Function in Sideline Testing for Concussion

- At least 5 papers since 2010 have been published demonstrating that the King-Devick test is effective at identifying neurologic deficits following concussion:
  - saccadic eye movement deficits
  - associated impairment of visual attention
- The simplicity, rapidity, validity, and reliability of the test lend itself for use as part of sideline testing for concussion

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## EFFICACY OF TREATMENT FOR SENSORIMOTOR VISION DEFICITS FOLLOWING TBI

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### Clinical Research on Vision and ABI TREATMENT OF OCULAR MOTILITY (INCLUDING ACCOMMODATIVE) DEFICITS

- Kapoor N, Ciuffreda KJ, Han Y. *Arch Phys Med Rehab* 2004.
  - two case reports (one TBI and one CVA) showing pre-/post-therapy objective eye movement recordings and subjective symptoms.
- Ciuffreda KJ, Han Y, Kapoor N, et al. *NeuroRehabilitation* 2006.
  - a series of 9 TBI and 5 CVA showing pre-/post-therapy objective eye movement recordings and subjective symptoms.

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### Clinical Research on Vision and TBI TREATMENT OF OCULAR MOTILITY (INCLUDING ACCOMMODATIVE) DEFICITS

- Doble JE, Feinberg DL, Rosner MS, Rosner AJ (2010) Identification of binocular vision dysfunction (vertical heterophoria) in traumatic brain injury patients and effects of individualized prismatic spectacle lenses in the treatment of postconcussive symptoms: a retrospective analysis. *Physical Medicine and Rehabilitation* 2010 Apr;2(4):244-53.
- 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.

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### Clinical Research on Vision and TBI:

TRAINING OF OCULAR MOTILITY  
(INCLUDING ACCOMMODATIVE) DEFICITS

- Ciuffreda KJ, Rutner D, Kapoor N, Suchoff IB, Craig S, Han ME (2008). Vision therapy for oculomotor dysfunctions in acquired brain injury: a retrospective analysis. Optometry, 79:18-22.
- 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).

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### Clinical Research on Vision and TBI:

TRAINING OF OCULAR MOTILITY  
(INCLUDING ACCOMMODATIVE) DEFICITS

- Ciuffreda et al. 2008 METHODS: The **visually-symptomatic TBI patient sample** included those who had started and completed their vision therapy during the proposed time period (10/2000 through 10/2003):
- 33 (out of 160) persons with TBI
- symptoms and signs were reported, with some patients having multiple symptoms or signs.

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### Clinical Research on Vision and TBI:

TRAINING OF OCULAR MOTILITY  
(INCLUDING ACCOMMODATIVE) DEFICITS

- Ciuffreda et al. 2008 RESULTS:
- Success was determined as reduction of at least 1 primary symptom and normalization of at least 1 clinical sign.
- 30/33 of those with TBI improved with vision therapy.

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

Sub-group	Total completing vision therapy	Total improving after vision therapy	Number of sessions			
			10-14	15-20	21-25	26-30
TBI	33	30	4	7	10	12

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**Clinical Research on Vision and TBI**  
OCULAR MOTILITY  
(INCLUDING ACCOMMODATIVE) DEFICITS

- Beneficial to know that vision therapy can reduce vision symptoms and normalize clinical vision signs in those visually-symptomatic persons with TBI

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**TYPICAL VISION DEFICITS FOLLOWING TBI:**

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- Primary vision symptom
  - Generalized neurological correlates
  - Possible treatment options

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Typical Vision Deficits Following TBI	
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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

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### 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 motor command

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### 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

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### 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 (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)

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### 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



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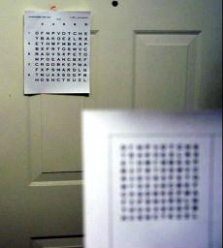
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## Accommodative Deficits: Restorative Treatment Options

- 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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## Typical Vision Deficits Following 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
  - parietal lobe
  - basal ganglia
  - superior colliculus
  - cerebellum

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### 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

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### 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)

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### 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)




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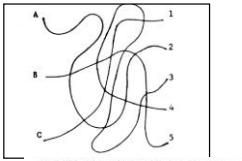
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## Versional Ocular Motility Deficits: Restorative Treatment Options



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abcdefghijklmnopqrstuvwxyz

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Zuth pirm nur&e &f st&k.Nileg myt  
l&T&T&s nom raus zab tuin lug&ts.  
Marb sewt rot&f puje. Yon&k nesud  
voz &lle. X&rt ch&od l&ugh& turh sn&f  
tre& ge& f&ru. V&b. rep&s H&le k&wj.  
D&gh meulb fw&e ig &ida. Ubc th&ey  
bo&f y&ed neoph w&ik. W&len kig pe&b  
nod t&nc &erb. R&it reb&g f&l &bt  
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- 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.*

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## Versional Ocular Motor Deficits: Restorative Treatment Options



ABCDEFGHIJKLMNOPQRSTUVWXYZ  
abcdefghijklmnopqrstuvwxyz  
In ch&ko evi nomd z&by ihpug nare.  
Zuth pirm nur&e &f st&k.Nileg myt  
l&T&T&s nom raus zab tuin lug&ts.  
Marb sewt rot&f puje. Yon&k nesud  
voz &lle. X&rt ch&od l&ugh& turh sn&f  
tre& ge& f&ru. V&b. rep&s H&le k&wj.  
D&gh meulb fw&e ig &ida. Ubc th&ey  
bo&f y&ed neoph w&ik. W&len kig pe&b  
nod t&nc &erb. R&it reb&g f&l &bt  
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## Versional Ocular Motor Deficits: Restorative Treatment Options



ABCDEFGHIJKLMNOPQRSTUVWXYZ  
abcdefghijklmnopqrstuvwxyz  
In ch&ko evi nomd z&by ihpug nare.  
Zuth pirm nur&e &f st&k.Nileg myt  
l&T&T&s nom raus zab tuin lug&ts.  
Marb sewt rot&f puje. Yon&k nesud  
voz &lle. X&rt ch&od l&ugh& turh sn&f  
tre& ge& f&ru. V&b. rep&s H&le k&wj.  
D&gh meulb fw&e ig &ida. Ubc th&ey  
bo&f y&ed neoph w&ik. W&len kig pe&b  
nod t&nc &erb. R&it reb&g f&l &bt  
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61	
Typical Vision Deficits Following TBI	
Deficit of:	Primary Associated Symptom:
Accommodation	Constant/intermittent blur
Versional Ocular Motility	Slower, less accurate reading /difficulty sustaining gaze, shifting gaze, or tracking targets
Vergence Ocular Motility	Constant/intermittent eyestrain / diplopia <i>eliminated with monocular occlusion</i>
Visual-Vestibular Interaction	Disequilibrium exacerbated in multiply, visually-stimulating environments
Visual processing	Slower speed/impaired visual memory and visual-spatial processing
Visual Field Integrity	Missing a portion of vision /overall restriction
Tear Film Integrity	Distorted clarity/gritty sensation, which <i>varies with blinking</i>
Light-Dark Adaptation	Elevated light sensitivity

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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

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### Vergence Ocular Motility: Neurological Correlates



- Pre-motor neural innervation (continued):
  - medial longitudinal fasciculus
  - cerebellum
  - frontal eye fields
- Role in generating vergence response of the abducens and oculomotor interneurons: not clearly elucidated

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### Vergence Ocular Motor Deficits: Compensatory Treatment Options



- To compensate for constant diplopia, decompensated phoria, or fixation disparity, incorporate:
  - fusional prism, if possible


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### Vergence Ocular Motor Deficits: Compensatory Treatment Options



- To compensate for constant diplopia, decompensated phoria, or fixation disparity, incorporate:
  - 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)


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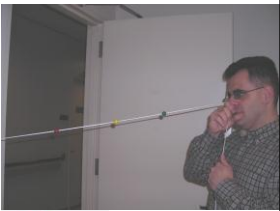


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### Vergence Ocular Motility Deficits: Restorative Treatment Options



- 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

Scheiman M, Mitchell L, Cotter S, Cooper J, Kulp M, Rouse M, Borsting E, London R, and Wensveen J, for the Convergence Insufficiency Treatment Trial Study Group (2005) A randomized clinical trial of treatments for convergence insufficiency in children. Arch Ophthalmol 123:14-24

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## Vergence Ocular Motility Deficits: Restorative Treatment Options



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



Scheiman M, Mitchell L, Cotter S, Cooper J, Kulp M, Rouse M, Borsting E, London R, and Wensveen J, for the Convergence Insufficiency Treatment Trial Study Group (2005) A randomized clinical trial of treatments for convergence insufficiency in children. Arch Ophthalmol 123:14-24

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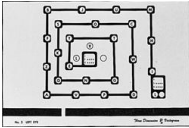
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## Vergence Ocular Motility Deficits: Restorative Treatment Options









Scheiman M, Mitchell L, Cotter S, Cooper J, Kulp M, Rouse M, Borsting E, London R, and Wensveen J, for the Convergence Insufficiency Treatment Trial Study Group (2005) A randomized clinical trial of treatments for convergence insufficiency in children. Arch Ophthalmol 123:14-24

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69

## Typical Vision Deficits Following TBI

Deficit of:	Primary Associated Symptom:
<b>Accommodation</b>	Constant/intermittent blur
<b>Versional Ocular Motility</b>	Slower, less accurate reading /difficulty sustaining gaze, shifting gaze, or tracking targets
<b>Vergence Ocular Motility</b>	Constant/intermittent eyestrain / diplopia <i>eliminated with monocular occlusion</i>
<b>Visual-Vestibular Interaction</b>	Disequilibrium exacerbated in multiply, visually-stimulating environments
<b>Visual processing</b>	Slower speed/impaired visual memory and visual-spatial processing
<b>Visual Field Integrity</b>	Missing a portion of vision /overall restriction
<b>Tear Film Integrity</b>	Distorted clarity/gritty sensation, which <i>varies with blinking</i>
<b>Light-Dark Adaptation</b>	Elevated light sensitivity

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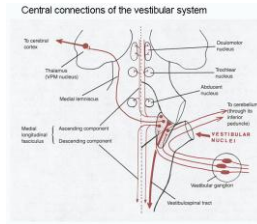
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### Visual-Vestibular Interaction

- 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)




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### 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
  - **any visual information mismatch can exacerbate vestibular problems (dorsal and ventral streams of processing)**
- Improving and stabilizing any ocular motor deficit may facilitate vestibular rehabilitative progress

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### Visual-Vestibular Dysfunction: Associated Symptoms

- Similar for vergence and versional ocular motility, emphasizing:
  - 1) \*increased dizziness and/or disequilibrium in/sensitivity to multiply-visually 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




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### Visual-Vestibular Dysfunction: Associated Symptoms

- Similar for vergence and versional ocular motility, emphasizing:
  - 1) difficulty with eye/head dissociation
  - 2) difficulty with eye/head dissociation
  - 3) foreground/background discrimination difficulty




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### 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

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### 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, work on stabilizing:
    - vergence 30 degrees right gaze and then 30 degrees left gaze
    - dynamic vergence while the patient is performing a slow horizontal VOR

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### Vergence Ocular Motility Deficits and Vestibular Dysfunction: Treatment Options

- After stabilizing horizontal fusional vergence and a slow horizontal VOR, work on stabilizing:
  - vergence 25 degrees upgaze and then 25 degrees downgaze
  - dynamic vergence while the patient is performing a slow vertical VOR

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




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### Typical Vision Deficits Following TBI

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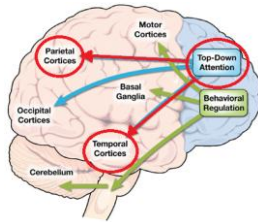
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### Visual Processing: Neurological Correlates

- 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?")




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### Visual Processing: Neurological Correlates



- 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)

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### Visual Processing: Neurological Correlates



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

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Visual Processing:  
Neurological Correlates

- 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)

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Visual Processing:  
Neurological Correlates

- Dorsal stream purpose:
  - uses precise 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

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Visual Processing:  
Dorsal-Ventral Stream Mis-communication

- Example: Patient presents with blur, eyestrain, and difficulty tracking when reading/ambulating/using a computer:
  - often unremarkable (involves more ventral processing):
    - visual acuity
    - perimetry

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

- Example: Patient presents with blur, eyestrain, and difficulty tracking when reading/ambulating/using a computer:
  - impaired ocular motility and accommodation (involve more dorsal processing):
    - vergence (and accommodation)
    - pursuit
    - saccades
    - fixation
- Sometimes, it is that the patient has difficulty computing the location of the object in space (dorsal), which impedes basic ocular motility and accommodation

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### Typical Vision Deficits Following TBI

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### Impaired Visual Field Integrity: Scattered/Non-lateralized

- Scattered, non-lateralized
  - more typical in TBI
  - less common in stroke
- Evident despite intact retina with unremarkable ocular health
- Neural mechanism:
  - likely secondary to diffuse neurological damage

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### Impaired Visual Field Integrity: Lateralized Visual Field Defects

- Lateralized (i.e., left hemianopia, right hemianopia, superior right quadrantanopia, etc.)
  - more typical in stroke
  - less common in TBI
  - may occur with or without inattention
- Evident despite intact retina with unremarkable ocular health
- Neural mechanism:
  - secondary to localized lesions (hemorrhagic or ischemic)
    - NOTE: right-brain lesions present often with inattention

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

- Visual field hemianopic/ quadrantanopic defect with inattention:
  - apply yoked prisms, mirrors, and field expanding lenses in conjunction with scanning strategies and compensatory/ adaptive approaches

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

- Visual field hemianopic/quadrantanopic defect with inattention:
  - refer to these links to see examples of:
    - yoked prism readers, sector prisms on glasses, and button (or spotting) prism on glasses  
[http://www.chadwickoptical.com/index\\_files/otherhemianopicsproducts.htm](http://www.chadwickoptical.com/index_files/otherhemianopicsproducts.htm) (accessed on 03/07/14)
    - the Peli prism system  
[http://www.chadwickoptical.com/index\\_files/pelilensforhemianopia.htm](http://www.chadwickoptical.com/index_files/pelilensforhemianopia.htm) (accessed on 03/07/14)

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


- Visual field hemianopic/quadrantanopic defect without inattention:
- application of sector prisms and spotting prisms in conjunction with scanning strategies and compensatory/adaptive approaches




Gottlieb Visual Field Awareness system



Fresnel "Gottlieb" style spotting prism



Chadwick Hemianopsia lens



Fresnel version

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### Typical Vision Deficits Following TBI

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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

Cockerham GC, Lemke S, Glynn-Milley C, Zumhagen L, and Cockerham KP. (2013) Visual performance and the ocular surface in traumatic brain injury. Ocul Surf Jan 11 (1): 25-34

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### Dry Eye: Etiology

- NEW THEORY (Cockerham et al., 2013 in Ocular Surface)- Dry eye:
  - is not apparently associated with medications or prior issues
  - still requires assessment and should almost be viewed as a new condition
  - may persist for months or years post-TBI

Cockerham GC, Lemke S, Glynn-Milley C, Zumhagen L, and Cockerham KP. (2013) Visual performance and the ocular surface in traumatic brain injury. Ocul Surf Jan 11 (1): 25-34

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### Dry Eye: Compensatory Treatment Options

- Artificial tears TID/QID OU in conjunction with lid hygiene
- For moderate to severe dry eye:
  - liqigels
  - insertion of punctal plugs

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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

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## Photosensitivity: 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 for fluorescent lighting (using either blue or gray tints)
- Wearing brimmed hats/caps

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## Summary

- Today's presentation was prepared to increase the ability to understand and identify:
  - the more common vision conditions evident following mild and moderate traumatic brain injury (TBI) and associated common ophthalmic terminology
  - evidence-based medicine support related to sensorimotor vision function following TBI in terms of:
    - elements for a high yield vision screening
    - occurrence
    - treatment efficacy
  - the underlying neurology of common vision conditions following mild and moderate TBI, as well as the primary vision symptoms and possible treatment options

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## Typical Vision Deficits Following TBI

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## Summary

- To find an **optometrist specializing in neuro-optometric rehabilitation** near you, please refer to the websites below:

- [www.covd.org](http://www.covd.org)
- [www.nora.cc](http://www.nora.cc)

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## Special Thanks To:

- Drs. Kenneth J. Ciuffreda, Allen Cohen, Irwin Suchoff, Barry Tannen, and Nathan Zasler
- Faculty/Staff/Administration at SUNY-Optometry, especially the Raymond J. Greenwald Rehabilitation Center, for their excellence in clinical assessment, management, and contribution to clinical care and research
- Optometric organizations for their support regarding increasing awareness of vision care and TBI:
  - American Optometric Association
  - College of Optometrists in Vision and Development
  - Optometric Extension Program Foundation
  - Neuro-Optometric Rehabilitation Association

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THANK YOU!

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104

**Accommodation:  
Relevant Publications**

- Ciuffreda KJ, Kapoor N, Rutner D, Suchoff IB, Han ME, Craig S (2007). Occurrence of oculomotor dysfunctions in acquired brain injury: a retrospective analysis. *Optometry*, 78: 155-61.
- Ciuffreda KJ, Rutner D, Kapoor N, Suchoff IB, Craig S, Han ME (2008). Vision therapy for oculomotor dysfunctions in acquired brain injury: a retrospective analysis. *Optometry*, 79:18-22.
- Green W, Ciuffreda KJ, Thiagarajan P, Szymanowicz D, Ludlam DP, Kapoor N. (2010) Accommodation in mild traumatic brain injury. *Journal Rehabilitation Research and Development* 47(3):183-200.
- Greenwald, BD, Kapoor N, Singh AD. (2012) Visual impairments in the first year after traumatic brain injury. *Brain Injury* 26(11):1338-59.

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105

**Clinical Research on Vision and Concussion:  
Saccadic Function: Sideline Testing for Concussion**

- Galetta KM, Barrett J, Allen M, Madda F, Delicata D, Tennant AT, Branas CC, Maguire MG, Messner LV, Devick S, et al. (2011). The King-Devick test as a determinant of head trauma and concussion in boxers and MMA *Neurology*. Apr 26; 76(17):1456-62.
- King D, Clark T, Gissane C. (2012) Use of a rapid visual screening tool for the assessment of concussion in amateur rugby league: a pilot study. *J Neurol Sci* 320: 16-21.
- King D, Brughelli M, Hume P, and Gissane C. (2013) Concussions in amateur rugby union identified with the use of a rapid visual screening tool. *J Neurol Sci* 326: 59-63.

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### Clinical Research on Vision and Concussion: Saccadic Function: Sideline Testing for Concussion

- Galetta MS, Galetta KM, McCrossin J, Wilson JA, Moster S, Galetta SL, and Balcer LJ, Dorshimer GW, and Master CL. (2013) Saccades and memory: baseline associations of the King-Devick and SCAT2 SAC tests in professional ice hockey players. *J Neurol Sci* 328: 28-31.
- King D, Brughelli M, Hume P, and Gissane C. (2014 epub ahead of print) Assessment, management and knowledge of sport-related concussion: systematic review. *Sports Med* Jan 9

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### Versinal Ocular Motility: Relevant Publications

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