Neuro-ophthalmological consideration in traumatic brain injuries

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Financial Disclosures

• None
Introduction

• Traumatic brain injury (TBI) is a major cause of morbidity and mortality (106 - 790 /100 000 per year).

• TBI can affect multiple aspects of vision; saccades, pursuit, convergence, accommodation, and vestibulo-ocular reflex.
  • Mild TBI (e.g. concussion) frequently leads to disruptions in visual functioning,
  • Moderate or severe TBI often causes structural lesions such as ocular motor palsies, optic neuropathies, and orbital pathologies.

• Neuro-ophthalmological evaluation is critical in the management of all forms of TBI.
• A wide range of visual complaints might follow head trauma as half of the circuits in the brain are involved in vision.
• Examination of the integrity of the visual system helps to both screen and monitor the recovery of patients with TBI.
• TBI can be classified as mild, moderate, or severe.
• Concussion is the most common form of TBI in a subset of patients classified as having mild TBI.
• Diffusion tensor imaging (DTI) identified a high prevalence of injury in the frontal lobes, corpus callosum, and corona radiata, interfering with the cognitive control of vision, in particular of eye movements.
Four Major Ocular Motility Systems

- Saccadic
- Smooth Pursuit
- Vergence
- Vestibulo-ocular reflex
Deficits in visual function in mild TBI
• Eye Movement Abnormalities 15 to 45 Days After Mild TBI:
  • Saccades - 30%
  • Pursuit - 60%
  • Convergence - 50-60%
  • Accommodation - 65%
Saccades

- Rapidly shifting horizontal gaze
- Anatomical pathway:
  - Reflexively generated from the parietal eye field, or intentionally generated in the frontal eye field then sent directly to the contralateral PPRF or via the superior colliculus.
- To assess:
  - The PPRF then generates horizontal saccades Fixate on a peripheral target and then a central object, such as the examiner's nose.
Pursuit

• Follow slowly moving objects

• Anatomical pathway:
  • Descending pathways from tempo-parieto-occipital junction and frontal eye fields connect in the pons and innervate the cerebellum, which then excites the sixth cranial (abducens) nerve nucleus

• To assess:
  • Track a moving object at no more than 30° per second
Vestibulo-ocular reflex

- Stabilizes images by producing eye movements in opposite direction to head movements
- Anatomical pathway:
  - Semicircular canals signal to vestibular nuclei which excite the sixth cranial (abducens) nerve nucleus
- To Assess:
  - Quick head thrusts while fixating
Vergence

• Simultaneous movement of eyes in opposite directions to maintain fusion on objects near or far.
• Anatomical pathway:
  • Cerebro-brainstem-cerebellar pathways. Not well understood
• To assess:
  • measure NPC,
  • assess for phorias with cross-cover test,
  • measure fusional amplitude with base-out prism test
Accommodation

• Accommodative dysfunction is also frequently described in patients with mild TBI.

• Accommodative insufficiency manifests as decreased accommodative amplitude (age dependent)

• Other less frequent abnormalities of accommodation described in patients with mild TBI include slowness in changing from one level of accommodation to another, which manifests as transient blurring.
Ocular motor palsies

• Convergence insufficiency.
• Phorias should be assessed with cover-uncover testing, and often improve over time.
• Cranial nerve abnormalities are unlikely to occur in mild TBI.
• Since mild TBI needs a normal head CT scan, thus cranial nerve abnormalities in the setting of mild head trauma merit further investigation for another cause.
Tests of visual functioning as sideline tests for sports-related concussion
Symptom Checklist

**SYMPTOM EVALUATION**

**3 How do you feel?**

<table>
<thead>
<tr>
<th>Symptom</th>
<th>0</th>
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<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
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<td>Headache</td>
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<td>Neck pain</td>
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<td>Blurred colors</td>
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<td>Balance problems</td>
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<td>Difficulty with vision</td>
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<td>Difficulty concentrating</td>
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<td>Difficulty remembering</td>
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<td>Fatigue or low energy</td>
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<td>Confusion</td>
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<td>Disorientation</td>
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<td>Trouble telling time</td>
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<td>Altered emotional behavior</td>
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<td>Irritability</td>
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<td>Mood swings</td>
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<td>Decrease in stamina</td>
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<td>Total number of symptoms (Max possible 30)</td>
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<td>Symptom severity score (Max severity possible 100)</td>
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**COGNITIVE & PHYSICAL EVALUATION**

**Cognitive assessment**

**Standardized Assessment of Concussion (SAC)**

**Delayed recall (1st memory to test concentration)**

<table>
<thead>
<tr>
<th>What month is it?</th>
<th>0</th>
<th>1</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the date today?</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>What is the day of the week?</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>What year is it?</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>What time is it?</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>What month was I born?</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>What is my eye color?</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>0</td>
<td>1</td>
</tr>
</tbody>
</table>

**Immediate memory score total**

| Total | 0 | 1 |

**Concentration: Delayed Recall (Max possible 42)**

<table>
<thead>
<tr>
<th>0-15</th>
<th>16-29</th>
<th>30-42</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>0</td>
<td>1</td>
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</tbody>
</table>

**Total: 0**
Sideline Testing

Balance Error Scoring System (BESS) or Timed Tandem Gait

King-Devick (K-D) Test of Rapid Number Naming or MULES (Rapid Picture Naming)
King Devick (K-D) test

• The King Devick (K-D) test measures rapid number naming on three test cards; the score for the test is the sum of the three times, in seconds, needed to read the cards.

• The K-D test takes less than 1 min on average to complete and has been studied in several athlete cohorts as a sideline test.

• Performance on the K-D test partly shows how well the patient is able to perform anticipatory saccades; and evaluates visual attention and language, which may be compromised in brain injury.

• The K-D test is a visual performance measure that does not require the clinical expertise to discern normal versus abnormal eye movements.

• The K-D test was shown to take about 5–7 seconds longer to complete in concussed athletes.
Rapid Number Naming (K-D)

Time to read all 3 cards = baseline score

- Objective, takes <1 minute, anyone can do!
- Delay in time has been seen in concussed boxers, collegiate athletes and rugby players
MULES Test of Rapid Picture Naming

Disease-free controls:
38.6 ± 7.3 seconds
(range 29.4 – 53.4 sec)

Other sideline tests

• Standardized Assessment of Concussion (SAC) and the Balance Error Scoring System (BESS), do not assess eye movements and thus the K-D test provides a complementary sideline assessment.

• One study showed that the K-D test took longer to complete thereby showing an abnormality in 79%; when combined with the SAC, abnormalities were captured in 89% of concussed athletes, which increased to 100% with the further addition of the BESS. The BESS and SAC together captured 90%.
Visual function deficits in moderate and severe TBI
Deficits are common in mild TBI

• In the patients with blast-related moderate or severe TBI,
  • 84% had abnormalities in saccades,
  • 46% in pursuit,
  • 48% in convergence, and
  • 62% in accommodation.

• In patients with non-blast related severe or moderate TBI,
  • 48% had abnormalities in saccades,
  • 26% in pursuit,
  • 67% in convergence, and
  • 61% in accommodation.
- Additionally, patients with moderate or severe TBI often have structural lesions

<table>
<thead>
<tr>
<th>Common sites of injury</th>
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</thead>
<tbody>
<tr>
<td>Optic nerve</td>
</tr>
<tr>
<td>Oculomotor nerve</td>
</tr>
<tr>
<td>Trochlear nerve</td>
</tr>
<tr>
<td>Abducens nerve</td>
</tr>
</tbody>
</table>
Traumatic optic neuropathy

• Direct vs indirect
  • Direct traumatic optic neuropathy generally has a worse prognosis, often with immediate irreversible vision loss.
  • Indirect traumatic optic neuropathy has been reported in 0.5–8.0% of cases of head trauma overall.
  • The intracanalicular portion (fixed region) is the most susceptible for shearing at the proximal and distal ends of the bony canal, followed by the intracranial part.

• Initially present with:
  • Normal funduscopic examination, with optic atrophy only apparent after 3–6 weeks,
  • decreased acuity,
  • a relative afferent pupillary defect if the injury is unilateral or asymmetric,
  • decreased colour vision, and
  • possibly field defects.

• Shearing and ischaemia might then be followed by nerve swelling within the tight canal, leading to further injury and potentially to delayed vision loss (10%).

• CT scan is needed to rule out optic canal fractures and hematoma.
Treatment and prognosis of TON

• Controversial!
• Observation, steroids in various doses, and optic canal decompression surgery.
• 20–60% of the untreated group had visual improvement.
• The strongest predictor of visual is the initial visual acuity, with patients who could initially see hand motion or better showing more improvement than those with only light perception or worse.
• Also initial severe degree of a relative afferent pupillary defect was predictive of reduced final visual improvement.
• Steroids generally have not been shown to be of benefit.
• The corticosteroid randomisation after significant head injury (CRASH) study61 showed increased mortality in patients who received high-dose steroids after head trauma versus control patients, suggesting that steroids in traumatic optic neuropathy might make prognosis worse overall.
• The International Traumatic Optic Neuritis Treatment trial was converted from a randomised trial to an observational one because of failure to enrol, but no benefit of surgery or steroids over the untreated group was reported.54 Some specialists have argued that surgery be reserved for those with an optic nerve sheath haematoma, delayed visual loss suggesting a haematoma, or the presence of an optic canal fracture.
• Retrospective uncontrolled reports have shown that extracranial surgery to decompress the optic canal can yield improvement in up to 80% of selected patients,62, 63 although more definitive studies of the role of surgery in traumatic optic neuropathy have not been done.
Optic chiasm and retrochiasmal pathways

• rare,
• usually the result of a severe head impact.
• skull fracture and cranial neuropathy, midline basilar skull fracture, deficits of the pituitary and hypothalamus, with 37–50% of patients developing diabetes insipidus
• Other associated injuries include carotid cavernous fistulae, traumatic carotid aneurysm, and meningitis associated with CSF leakage.
Ocular motor neuropathies

• 3 to 11%
• In one study, patients with TBI who had cranial nerve III nerve palsies had lower GCS scores than did those with trochlear nerve (cranial nerve IV) or abducens nerve (cranial nerve VI) injuries, suggesting an increased severity of injury.
• Sites of injury:
  • exit from the midbrain,
  • the superior orbital fissure,
  • subarachnoid space.
  • at the tentorial edge with uncal herniation due to traumatic oedema or haemorrhage
• 14 month recovery rate of 95% for ptosis, 83% for extra-ocular muscle paresis, and 50% for pupillary involvement. Aberrant regeneration can occur.
Cranial nerve IV injury

• 3–13% of patients with TBI.
• the thinnest CN and has the longest intracranial course.
• 50–60% of patients with traumatic cranial nerve IV injury have recovered at 6 months.
Cranial nerve VI injuries

• 4–6% of patients with TBI.

• Cranial nerve VI has a complex course (figure 6).
  • described in association with petrous bone damage and with flexion-extension injury
  • Delayed abducens injury can occur in the setting of elevated intracranial pressure—eg, due to traumatic haemorrhage or oedema, and has also been described with normal imaging, suggesting additional mechanisms such as ischaemia or local oedema
  • 25% of unilateral cases and 50% of bilateral cases resolve in 6 mo.
Orbital compartment syndrome

- very rare traumatic neuro-ophthalmologic emergency that can lead to loss of vision unless promptly recognized and treated.
- Often due to an arterial retrobulbar haemorrhage after blunt facial trauma.
- Signs of orbital compartment syndrome include vision loss, periorbital ecchymosis, complete ptosis, ophthalmoplegia, a fixed dilated pupil, and tense subconjunctival haemorrhage.
- Loss of vision and ophthalmoparesis are thought to occur as a result of direct compression of nerves and their blood supply.
- Lateral canthotomy can be done at bedside to relieve the pressure and when done urgently can lead to visual recovery
Other injuries

• brainstem injury
• sympathetic pathway injury
• orbital apex,
• superior orbital fissure, and
• cavernous sinus syndromes
Take home messages

• Head trauma requires a careful neuro-ophthalmic examination.
• Tests of saccades, pursuit, convergence, accommodation, vestibulo-ocular reflex, and phorias are often abnormal and therefore important to perform clinically to assess concussive injury, monitor recovery, and provide visual therapy as appropriate.
• Patients with moderate or severe TBI often presents structural lesions, leading to ocular motor neuropathies, optic neuropathies, and orbital pathologies.
• Tests of visual function can be a sensitive means to assess minor head injury.
• Vision-based sideline tests such as the King Devick (K-D) test, which includes rapid number naming, show great promise and are being assessed further in large cohorts of athletes.
• The K-D test can be administered by non-physician observers such as parents of sports participants and could have applications for rapid testing in the military.
Main References


2. Adding Vision to Concussion Testing: A Prospective Study of Sideline Testing in Youth and Collegiate Athletes. Galetta, Kristin M. MS; Morganroth, Jennifer BA; Moehringer, Nicholas; Mueller, Bridget PhD; Hasanaj, Lisena BA; Webb, Nikki MS, ATC; Civitano, Courtney MS, ATC; Cardone, Dennis A. DO; Silverio, Arlene MD; Galetta, Steven L. MD; Balcer, Laura J. MD, MSCE Journal of Neuro-Ophthalmology: September 2015 - Volume 35 - Issue 3 - p 235–241.


Thank you!