The neuro-ophthalmology of head trauma

Rachel E Ventura, Laura J Balcer, Steven L Galetta

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Department of Neurology, New York University School of Medicine, New York, NY, USA
(R E Ventura MD, Prof L J Balcer MD, Prof S L Galetta MD)

Correspondence to:
Dr Steven L Galetta, Department of Neurology, New York University School of Medicine, New York, NY 10016, USA
steven.galetta@nyumc.org

Traumatic brain injury (TBI) is a major cause of morbidity and mortality. Concussion, a form of mild TBI, might be associated with long-term neurological symptoms. The effects of TBI and concussion are not restricted to cognition and balance. TBI can also affect multiple aspects of vision; mild TBI frequently leads to disruptions in visual functioning, while moderate or severe TBI often causes structural lesions. In patients with mild TBI, there might be abnormalities in saccades, pursuit, convergence, accommodation, and vestibulo-ocular reflex. Moderate and severe TBI might additionally lead to ocular motor palsies, optic neuropathies, and orbital pathologies. Vision-based testing is vital in the management of all forms of TBI and provides a sensitive approach for sideline or post-injury concussion screening. One sideline test, the King-Devick test, uses rapid number naming and has been tested in multiple athlete cohorts.

Introduction

Traumatic brain injury (TBI) is increasingly recognised as a major cause of morbidity and mortality worldwide, with estimates of its incidence ranging from 106 to 790 per 100 000 people per year.[1,2] Because around half of the circuits in the brain are involved in vision, many aspects of the visual system are vulnerable to moderate, severe, or mild TBI.[3] Not surprisingly, a wide range of visual complaints might follow head trauma, including photophobia, double vision, blurred vision, loss of vision, and visual processing problems. The topic of sports concussion, a mild form of TBI, has received a lot of media attention as a result of increased recognition of potential long-term deleterious effects of repeated concussive events, such as depression, altered cognition, and neurodegenerative diseases (eg, chronic traumatic encephalopathy and Alzheimer's disease).[4,5] Improvement of the ways in which we screen for concussion is imperative, particularly since objective findings can be subtle and athletes often under-report their symptoms.[6] Examination of the integrity of the visual system helps to both screen and monitor the recovery of patients with TBI.

TBI and concussion

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[7–9] (see panel 1 for definitions).The differences between combat-related and civilian TBI have not fully been elucidated, although it should be noted that mechanisms of blast-related head injury are complex and might include other injuries related to burns, toxic inhalation, and radiation exposure.

Many clinical features of post-concussive syndrome mirror those of frontal and temporal lobe syndromes, including problems with executive function and difficulties with attention, memory, and concentration,[10] and frontal and temporal lobe damage has been shown in neuropathological[11] and imaging[12] studies. Investigators of studies using diffusion tensor imaging (DTI) to assess microstructural white matter changes in mild TBI identified a high prevalence of injury in the frontal lobes, corpus callosum, and corona radiata, and diffusely in deep white matter.[13,14] The cognitive control of vision, in particular of eye movements, needs coordination of reflexive and voluntary activity, including frontoparietal circuits and subcortical nuclei;[15] these pathways are vulnerable to injury in mild to severe TBI.

Deficits in visual function in mild TBI

Saccades, antisaccades, and cognitive function

Saccadic eye movements involve a wide variety of cognitive processes and findings from several studies have shown abnormalities in saccade generation after all forms of brain trauma. The generation of saccades involves weighing aspects of the stimulus as well as processes incorporating goals and intentions.[16] Visuospatial information is relayed from the occipital lobe and then processed in the posterior parietal cortex. Saccades can then be reflexively generated from the parietal eye field or intentionally generated in the frontal eye field.[16,17] The dorsolateral prefrontal cortex plays a part in inhibition of reflexive saccades, in prediction of when anticipatory saccades are needed, and in short-term spatial memory[18] (table 1, figure 1). One frequently used test to assess executive function is the assessment of antisaccades. Antisaccades are examined by having the patient try to look away from a stimulus; this action needs inhibition of reflexive saccades and generation of voluntary saccades. Saccades can also be used to test for memory (eg, with the memory-guided saccade test) or to assess attention (eg, with the gap-saccade test; panel 2).

In a study in patients with mild TBI within 10 days of their injury, antisaccades were assessed with video-oculography and found to be impaired, with prolonged saccadic latencies, high directional errors, and poor spatial accuracy, although simple reflexive saccades were normal.[19] These patients also had impairments in memory-guided saccades.[19,20] This work was then extended to patients who had post-concussive syndrome with symptoms 3–5 months after their injury in comparison with patients who had good recovery. Those with post-concussive syndrome did worse in antisaccades, in memory-guided saccades, and in a self-paced saccade test in which patients were instructed to look back and forth between two points as rapidly as possible.[21] Kraus and colleagues[22] identified similar

results in patients with chronic TBI of duration greater than 6 months, and showed that eye movements were more sensitive than neuropsychological testing for persisting neurological abnormalities in these patients. Drew and colleagues[23] noted that patients with acute mild TBI had deficits in a gap saccade test, in which the patient fixates on one target and then has to orientate to a peripheral target after a variable amount of time. The patients with mild TBI had longer saccadic reaction times compared with healthy controls when the temporal gap between the initial target and the new target was short but not when it was long, which is consistent with abnormalities in disengagement of attention.

Patients with mild TBI with acute or chronic symptoms, therefore, seem to have impairments in executive function, attention, and memory that can be associated with impaired saccadic function.[19–23] Deficits in subcortical visual pathway function were also seen as manifested by slowed self-paced saccades.[21] Patients who have recovered well from their TBI and do not have ongoing symptoms do not seem to have continued saccadic dysfunction,[21] although further studies are necessary to characterise the exact evolution of these eye movement abnormalities. Tests of saccades recorded with video oculography enable examiners to provide an objective correlate to concussive and post-concussive symptoms.

Deficits in saccades can also be identified with a cursory clinical examination, though detecting abnormalities of individual eye movements at the bedside or on the sideline can be challenging and requires clinical experience (figure 2). In one prospective study of patients with blast-induced mild TBI, six (30%) of 20 patients had saccadic dysfunction versus none of 20 in the control group.[24]

Smooth pursuit

Predictive visual tracking can be used to assess higher cognitive functioning, because it requires attention, anticipation, working memory, as well as smooth, and at times saccadic eye movements to maintain gaze on a fixed target (figure 3).[25] Smooth pursuit of a predictive target is programmed by the cerebellum and is based on retinal and higher cortical input, including input from the prefrontal cortex.[17,26] Suh and colleagues[27,28] had controls and patients with mild TBI track a target moving in a circle at a fixed rate, with or without temporarily extinguishing the target. The patients with TBI had decreased target prediction with increased eye position error and variability of eye position, with more robust findings when the target was temporarily extinguished, which requires an increased degree of predictive tracking.[27,28] These smooth pursuit deficits correlated with scores of attention and executive function on the California Verbal Learning Test.[28] Maruta and colleagues[29] correlated variability of eye position in the circular tracking test in patients with chronic TBI to changes in the white matter tracts using DTI fractional anisotropy (FA). They reported that gaze error variability corresponded with abnormal mean FA values in the right anterior corona radiata, the left superior cerebellar

Panel 1: **Glossary of terms**

Concussion
A traumatically induced transient disruption of brain function;[9] a subset of mild traumatic brain injury.

Mild traumatic brain injury
Traumatic brain injury has been deemed mild when there is a normal CT, Glasgow coma scale score (GCS) of 13–15, loss of consciousness of less than 30 min, alteration of mental state of less than 24 h, and post-traumatic amnesia for less than 24 h.[8]

Accommodation
The process by which the eye changes the shape of the lens to maintain focus on an object as its distance varies.

Accommodative amplitude
Reciprocal of the focal length in metres. It can be subjectively measured by correcting for distance vision and then having the subject move a near reading card towards the eyes until the text is no longer in focus, then measuring the reciprocal of the distance in metres. Alternatively, the card could be initially placed close to the eyes and then pulled away until the patient sees it clearly. Typically, the 20/30 line on a near card is used. For instance, a patient who has blurry vision at 10 centimetres has 1/(0.10 metres) or 10 dioptres of accommodative amplitude.

Convergence fusional amplitude
The amount of prism glass that can be placed in front of the eyes at a particular distance before double vision is seen. It can be measured with a base out prism (eg, with the thicker edge facing outwards so the light is bent towards the nose) over one eye when the patient focuses on a target.

Phoria
Misalignment of the eyes that occurs only some of the time, such as when one eye is covered and the synchronisation between eyes is broken. The presence of phoria can be tested for with the cross-cover test, in which the subject focuses on an object while each eye is alternatively covered while the uncovered eye is observed for movement. Convergence insufficiency often leads to near phorias because the eyes cannot move in tandem to align on a target.

Exotropia
A type of eye misalignment in which one eye is manifestly deviated outward. It can be congenital or acquired, constant, or intermittent.

Exophoria
Eye misalignment in which the eyes are deviated outward from each other in the absence of binocular fusion.

Dioptres
A unit of measurement of the optical power of a lens, which is equal to the reciprocal of the focal length in metres. One prism dioptre is a deflection of light by a prism one centimetre on a plane placed at a distance of one metre.

Dysmetria
A lack of accuracy in movement, typically with overshoot or undershoot of a limb or the eye, characteristic of cerebellar dysfunction.

	Purpose	Anatomical pathway	Clinical tests to assess
Saccades	Rapidly shifting horizontal gaze	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. 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	Descending pathways from tempero-parieto-occipital junction and frontal eye fields connect in the pons and innervate the cerebellum, which then excites the sixth cranial (abducens) nerve nucleus	Track a moving object at no more than 30° per second
Vestibulo-ocular reflex	Stabilises images on the retina by producing eye movements in opposite direction to head movements	Semicircular canals signal to vestibular nuclei which excite the sixth cranial (abducens) nerve nucleus	Quick head thrusts while fixating
Vergence	Simultaneous movement of eyes in opposite directions to maintain fusion on objects near or far	Cerebro-brainstem-cerebellar pathways. Not well understood	Measure NPC, assess for phorias with cross-cover test, measure fusional amplitude with base-out prism test

PPRF=paramedian pontine reticular formation. NPC=near point of convergence.

Table 1: **Major eye movements**

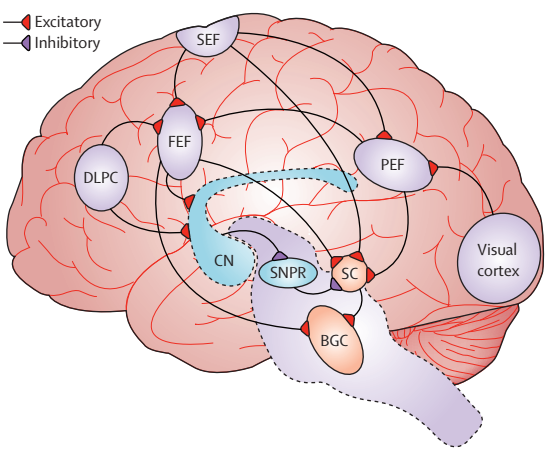

Figure 1: **Major cortical areas in control of eye movements and visual processing, with projections illustrating saccade generation**
Saccades are initiated by signals sent from the frontal, parietal, or supplementary eye fields (FEF, PEF, SEF) to the superior colliculus (SC), which then projects to the brainstem gaze centres (BGC). In parallel, the FEF also initiates saccades via direct connections to the BGC. In the indirect pathway, the substantia nigra pars reticulata (SNPR) inhibits the SC, preventing saccade generation. To turn off this inhibition, the frontal eye fields are activated prior to a saccade, which then inhibits the SNPR via the caudate nucleus. The saccade pathways are a multidistributed network, but the FEF primarily generates voluntary or memory guided saccades, the PEF reflexive saccades, the SEF saccades in coordination with body movements and successive saccades. The dorsolateral prefrontal cortex (DLPC) modulates antisaccades, the inhibition of reflexive saccades, and the advanced planning of saccades. The cerebellum also modulates saccades (projections not shown).

peduncle, and the genu of the corpus collosum, confirming that deficits in their visual test reflected injury in areas frequently compromised in TBI.[29] Deficits in pursuit in mild TBI have been documented in an outpatient setting using clinical measures. In one prospective study, for instance, 12 (60%) of 20 patients with mild TBI versus none of the controls had an abnormality in pursuit.[24]

Vergence

Deficits in vergence have been long described in association with head injury.[30,31] Convergence is the simultaneous adduction of the eyes to maintain binocular fusion on near targets. Three main examination findings suggest the diagnosis of convergence insufficiency, not all of which need to be present in a patient.[32] The first is an impaired near point of convergence (NPC), which can be assessed by moving a target towards the patient's nose and measuring the distance at which the patient can no longer maintain fusion or the distance at which the patient develops an exotropia (figure 4). A normal NPC is 5–10 cm and anything above suggests convergence insufficiency. The Nearpoint Fixation Disparity Test is another subjective near point test that is being explored as a method to assess convergence insufficiency. Other findings observed in convergence insufficiency include near exophorias of 10 dioptres or more, and reduced convergence fusional amplitudes (see panel 1) measuring less than 15 dioptres. The prevalence of vergence dysfunctions ranges from 47% to 64% of patients with mild TBI.[24,33] Capo-Aponte and others[24] also performed a convergence insufficiency symptoms survey in patients with blast-related mild TBI versus controls, and identified significant differences in symptoms such as headaches, sore eyes, words coming in and out of focus, and loss of place while reading. As such, reading is another important test in patients with mild TBI. Other symptoms that can be associated with convergence insufficiency include double vision, sleepiness, and loss of concentration. Both convergence insufficiency and accommodative insufficiency after mild TBI might improve with vision therapy.[34] A small proportion of healthy individuals have convergence or accommodative insufficiency, emphasising the importance of baseline assessment.

Panel 2: **Tests of eye movements that have been used to assess high cognitive functions.**

Attention

Gap saccade test

The participant fixates on one central target on a computer screen, which then disappears and a peripheral target, either to the left or right, appears after a variable period of time. The deviation of the participant's eyes when the second object appears is measured using infrared tracking devices.

Visually cued saccades

The participant fixates on a central target while a cue indicates the possible location of an upcoming target; then a congruent or incongruent target to the cue appears, generating saccades. The deviation of the eyes is measured.

Smooth pursuit of a predictive target

The participant tracks an object on a known trajectory (eg, a circle); the object might at times be transiently obscured at any point in its trajectory. Eye velocity and gaze positional errors are recorded using visual tracking devices.

Executive function

Antisaccades

The participant is asked to look away from a presented object.

Memory

Memory-guided saccades

The participant focuses on a central target while a peripheral target appears briefly; after a variable delay, usually between 500 and 1500 ms, the participant is asked to fixate by memory on where the peripheral target was located.

Memory-guided sequences

The participant is presented with targets on a screen and then asked to memorise the sequence in which they appear. The participant generates saccades in the memorised order.

Smooth pursuit of a predictive target

See above.

Accommodation

Accommodative dysfunction is also frequently described in patients with mild TBI. Accommodative insufficiency manifests as decreased accommodative amplitude (panel 1). Accommodative amplitude is age dependent and the minimal amount expected can be estimated by the formula $15-(0\cdot25\times\text{age in years})$. So, a 20-year-old patient might be expected to have about 10 dioptres of accomodative amplitude. Capo-Aponte and colleagues[24] prospectively studied a population with blast-related mild TBI and showed that 13 (65%) of 20 patients versus 3 (15%) of 20 controls had abnormalities in accommodative amplitude. Other less frequent abnormalities of accommodation described in patients with mild TBI include accommodative insufficiency and slowness in changing from one level of accommodation to another, which manifests as transient blurring.

Figure 2: **Saccades**

Saccades can be tested by having the patient (A) look at a target in the periphery and then (B) look at a target directly in front of their face. Saccades can be assessed for speed and accuracy. This test is shown in an accompanying video.

See Online for video

Figure 3: **Pursuit**

Pursuit can be tested by having the patient fix and follow a slowly moving object, such as the end of a reflex hammer. Please see accompanying video.

Vestibulo-ocular reflex and nystagmus

Peripheral and central mechanisms of vertigo have been frequently reported in mild TBI, with peripheral causes associated with an impaired vestibulo-ocular reflex

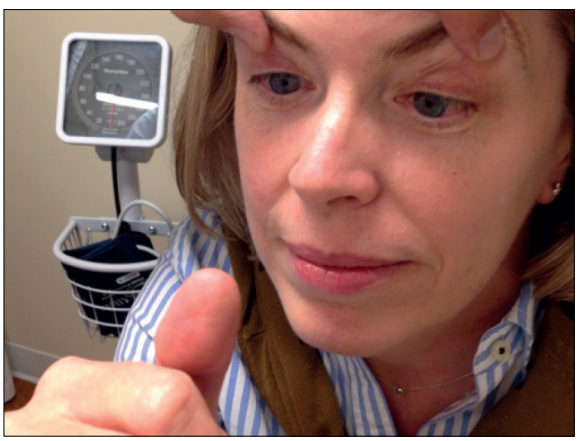

Figure 4: **Convergence**
Covergence can be assessed by moving the patient's thumb towards their nose and measuring the distance at which fusion is broken or the distance at which an exotropia is developed. Note the constricted pupils with the near response. This test is shown in an accompanying video.

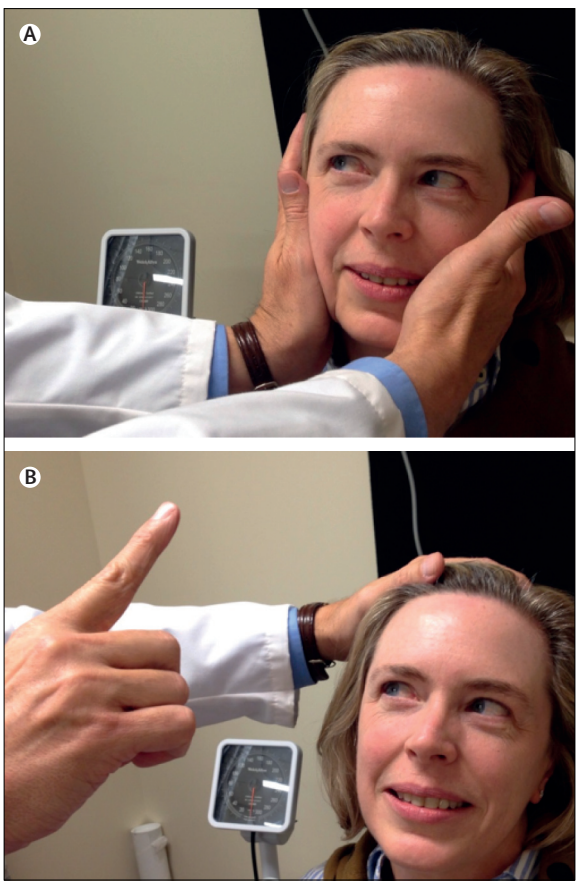

Figure 5: **The vestibulo-ocular reflex and oculocephalic movements**
(A) The vestibulo-ocular reflex is tested by rapidly thrusting the patient's head from side to side with both of the examiner's hands, while asking the subject to fixate on the examiner's nose. (B) Oculocephalic movements can also be assessed by moving the patient's head quickly from side to side with one hand, while they fixate on the examiner's finger. Normally the eyes will rotate smoothly in an opposite direction to the head turn. Please see accompanying video.

(figure 5). Central mechanisms of vertigo are sometimes associated with cerebellar abnormalities, such as impaired pursuit or ocular dysmetria. Spontaneous nystagmus is associated with both peripheral and central causes of vertigo from head trauma. Nystagmus of peripheral origin is usually horizontal or horizontorotary, is commonly suppressed by visual fixation, has a latency before onset, and is frequently transient.[35] Direction changing nystagmus is associated with central causes. Central nystagmus can occur in any direction, including vertical or rotational, is often enhanced by visual fixation, does not usually have a latency, and is sustained with prolonged eccentric gaze.[35]

Ocular motor palsies

Phorias have been described in patients with mild TBI, mostly near phorias in association with 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 unless there is a pre-existing structural abnormality.[36] For example, in one case report, a third-nerve palsy after mild head trauma was associated with an underlying posterior communicating artery aneurysm[37] and, in another report, a fourth-nerve palsy after mild head trauma was associated with an arteriovenous malformation compressing the nerve.[38] However, when Coello and colleagues[39] reviewed 16 440 patients described as having mild head injury, that they classified as Glasgow coma scale (GCS) score of 13–15 irrespective of imaging findings, they reported that seven had oculomotor nerve palsies (none of whom had normal head CT scans), seven had trochlear nerve palsies (three of whom had normal head CT), and eight had abducens nerve palsies (four of whom had normal head CT scans). In view of the fact that the most widely accepted definition of mild TBI needs a normal head CT scan,[8] the incidence of cranial nerve abnormalities in mild TBI in this study is very small but not zero, and whether further study with MRI would have identified an underlying associated abnormality is unclear. 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

Because many aspects of vision relate to high cognitive functioning, which is characteristically compromised in mild TBI, and because many of the pathways in the brain are devoted to afferent and efferent vision, tests of visual function are promising candidates as rapid sideline tests. Rapid sideline tests seem imperative in view of the increasing recognition of the prevalence of sports concussions and their possible deleterious effects, including second-impact syndrome. This is a rare but potentially catastrophic second concussion before symptoms of the first have cleared. 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.[40–43] Performance on the K-D test partly shows how well the patient is able to perform anticipatory saccades; the origin of anticipatory saccades has been localised to the dorsolateral prefrontal cortex, an area susceptible to injury in TBI.[15,18] The K-D test also evaluates visual attention and language, which may be compromised in brain injury. The K-D test is a visual performance measure and one advantage is that it does not require the clinical expertise to discern nomal versus abnormal eye movements. The K-D test was shown to take about 5–7 seconds longer to complete in concussed boxers, mixed-martial-arts fighters,[41] collegiate athletes,[40] professional ice hockey players,[42] and rugby players,[43] and was shown to be a sensitive measure of detecting concussions in these athletes. Other sideline tests, such as the 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. In our study of athletes after a concussion who received a battery of sideline tests, 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.[44] The BESS and SAC together captured 90%, missing abnormalities in two of the 20 patients in the cohort.[44]

Visual function deficits in moderate and severe TBI

Deficits are common in mild TBI

Patients with moderate and severe TBI might have similar deficits to those seen for mild TBI. Investigators of one retrospective study specifically excluded mild TBI to tally these deficits in patients with moderate or severe combat-related TBI, 48 of whom had blast-related TBI and 23 of whom had non-blast related TBI.[45] 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. However, the authors did not directly compare the moderate or severe subgroups with the mild subgroup to determine whether any differences could be identified. Additionally, patients with moderate or severe TBI often have structural lesions (table 2).

Increased light sensitivity or chronic glare is a common complaint in patients with TBI in general, and it is possibly due to abnormalities in dark adaptation,[46] meningeal irritation, or migraine, or driven through central pathways such as thalamic pathways.

	Common sites of injury
Optic nerve	Intracanalicular portion
Oculomotor nerve	At the exit from the midbrain; superior orbital fissure
Trochlear nerve	Midbrain site of exit; midbrain parenchymal contusions along its extra-axial course through the cisterns
Abducens nerve	Along the skull base, the petrous apex, and at the point of entry into the extradural space

Table 2: **Nerve injury after head trauma**

Traumatic optic neuropathy

Traumatic optic neuropathy can be classified into a direct form due to penetrating injury to the nerve and an indirect form due to transmission of forces to the optic nerve from a distant site. Injury can occur anywhere along the optic nerve—eg, the optic nerve head, intraorbital, intracanalicular, or intracranial portion. Direct traumatic optic neuropathy generally has a worse prognosis than indirect traumatic optic neuropathy, often with immediate irreversible vision loss. Indirect traumatic optic neuropathy has been reported in 0·5–8·0% of cases of head trauma overall,[47–50] although it should be noted that most of these studies are heavily skewed towards moderate or severe head injury because the patients included are usually admitted to hospital or referred from rehabilitation units. Anterior indirect traumatic optic neuropathy can be caused by avulsion injury, which is the sudden rotation of the globe from blunt trauma, leading to separation of the optic nerve on exiting the globe. Most indirect traumatic optic neuropathies are posterior to where the central retinal artery enters the nerve and thus are associated with a normal retinal circulation.[47] Posterior axonal injury does not cause any acute effects on the optic disc either, so posterior indirect traumatic optic neuropathy can present with a normal funduscopic examination, with optic atrophy only apparent after 3–6 weeks.[51] The ophthalmic examination should be done early in the assessment of the patient and before eyelid swelling causes the lid to close completely, thereby precluding such examination. The only ophthalmic examination findings in posterior indirect traumatic optic neuropathy might be decreased acuity, a relative afferent pupillary defect if the injury is unilateral or asymmetric, decreased colour vision, and possibly field defects. Posterior indirect traumatic optic neuropathy is often due to a frontal or midfacial blow, and the injury is severe enough to cause loss of consciousness in 40–72% of patients.[47]

Most posterior indirect traumatic optic neuropathy occurs in the intracanalicular portion of the optic nerve.[52] The intracanalicular portion is the only fixed region of the nerve, and the nerve is particularly susceptible to shearing at the proximal and distal ends of the bony canal. Shearing and ischaemia might then be followed by nerve swelling within the tight canal, leading to further injury and

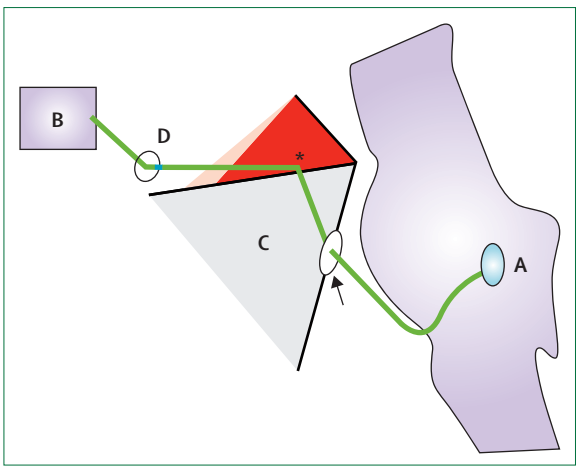

Figure 6: **Schematic diagram of the course of cranial nerve VI**
The cranial nerve VI (in green) is represented from its nucleus in the pons (A) to the eye (B). The nerve exits the pons and then ascends in the subarachnoid space before running through the dural hole (arrow) to enter the extradural space. It then ascends over the ridge of the petrous bone (C) to reach the petrous apex (asterisk), where it is tethered to dura. It then changes direction by 120° and passes under the petrosphenoid ligament (shown in pink) in Dorello's canal (shown in red). After Dorello's canal, it enters the cavernous sinus (not shown) where it travels with the sympathetic fibres along the internal carotid artery, then passes through the superior orbital fissure (D) to innervate the lateral rectus. Adapted in part from Arias.[84]

potentially to delayed vision loss.[53] Delayed vision loss was described in about 13 (10%) of 127 patients with traumatic optic neuropathy in one study.[54] Indirect traumatic optic neuropathy is sometimes accompanied by a fracture within the optic canal and thus CT imaging should be done as part of the initial assessment. The intracranial portion of the optic nerve is the second most common site of traumatic optic neuropathy, and this region can be injured by compression against the falciform dural fold, where the nerve enters the fixed opening of the optic foramen.[52]

Treatment for indirect posterior traumatic optic neuropathy is controversial.[55–57] Strategies used include steroids in various doses, optic canal decompression surgery, and observation. The natural history of traumatic optic neuropathy has not been adequately studied, but in several observational studies[54,58,59] and in one small randomised controlled trial[60] 20–60% of the untreated group had visual improvement. These results vary depending on how long after optic nerve injury the patients are studied; later enrolments will miss those patients that have already improved. The biggest predictor of visual improvement in these studies is the initial visual acuity after the injury (the exact timing is variable and not available in some cases), with patients who could initially see hand motion or better showing more improvement than those with only light perception or worse. Additionally, visual recovery in the first 2 days portended improvement in final visual acuity, and an initial severe degree of a relative afferent pupillary defect was predictive of reduced final visual improvement.[60] There is no large, randomised controlled clinical trial of the effects of steroid treatment on traumatic optic neuropathy, but steroids generally have not been shown to be of benefit.[55,57] The corticosteroid randomisation after significant head injury (CRASH) study[61] 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. Likewise, no randomised controlled clinical trials assessing surgery in traumatic optic neuropathy have been done; only observational studies are available. 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

Traumatic chiasmal syndrome is rare, occurring in three of 326 patients with TBI in one retrospective series,[49] and usually the result of a severe head impact. In the largest case series of 19 patients with traumatic chiasmal syndrome, 68% of patients had accompanying skull fracture and more than half had accompanying cranial neuropathy.[64] Chiasmal syndrome has been associated with frontal blows and a midline basilar skull fracture.[65] It is often accompanied by deficits of the pituitary and hypothalamus, with 37–50% of patients developing diabetes insipidus, according to the few case series.[64–67] Other associated injuries include carotid cavernous fistulae, traumatic carotid aneurysm, and meningitis associated with cerebrospinal fluid leakage. Chiasmal syndrome is accompanied by a bitemporal haemianopia. Optical coherence tomography (OCT) has been used to show the loss of the retinal nerve fibre layer in the nasal sectors of both eyes in one case with traumatic chiasmal syndrome.[68]

Traumatic lesions to the optic tract, radiation, and occipital lobe can also occur. Findings from retrospective reviews showed that 9–14% of patients with TBI have retrochiasmal visual field defects.[49,69] In a retrospective review of 103 patients with a homonymous haemianopia due to TBI, 11% had lesions in the optic tract, 23% in the optic radiation, 13% in the occipital lobe, and 60% had multiple lesions.[70] The more severe the injury, the more probable that retrochiasmal visual pathway injury might occur, probably because of shearing. In one autopsy

study of 45 patients with fatal closed head injuries, 87% had axonal injury in the optic chiasm, tracts or radiations.[71] In one case report of a post-traumatic optic tract injury, gradient-recalled echo positivity was noted in the tract, suggesting a mechanism of focal haemorrhage in that case.[72]

Ocular motor neuropathies

The reported incidence of oculomotor nerve (cranial nerve III) injury in TBI ranges from 3 to 11%.[48,49] 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.[36] Sites of injury include the exit of cranial nerve III from the midbrain, at the superior orbital fissure, or along its course in the subarachnoid space.[36,73] In one case report of a traumatic cranial nerve III injury, diffusion tensor tractography showed a discontinuation in the nerve at its exit from the midbrain, suggesting that this technique might hold promise to confirm traumatic axonal injury not otherwise apparent with standard MRI sequences.[74] Uncal herniation due to traumatic oedema or haemorrhage can lead to compression of cranial nerve III at the tentorial edge, by the posterior cerebral artery, or the parahippocampal gyrus, or the nerve can be contused over the skull base. Findings from one retrospective review of 26 patients with traumatic cranial nerve III palsy showed a 14 month recovery rate of 95% for ptosis, 83% for extra-ocular muscle paresis, and 50% for pupillary involvement.[75] Aberrant regeneration can occur.

Cranial nerve IV injury has been described as occurring in 3–13% of patients with TBI.[48,49] Of the ocular motor nerves, cranial nerve IV is the thinnest and has the longest intracranial course.[76] Cranial nerve IV injury is associated with dorsal midbrain parenchymal contusions and haematomas probably due to impact of the midbrain against the tentorium.[77–79] Traumatic cranial nerve IV palsies have also been shown with injury in the cisterns along the extra-axial course of the nerve—eg, in association with haemorrhage in the superior cerebellar, quadrigeminal, or ambient cisterns.[79–81] 50–60% of patients with traumatic cranial nerve IV injury have recovered at 6 months.[82,83]

Cranial nerve VI injuries have been shown in 4–6% of patients with TBI.[48,49] Cranial nerve VI has a complex course (figure 6). Traumatic cranial nerve VI palsies have been described in association with petrous bone damage and with flexion-extension injury. Flexion-extension forces in cervical injury are believed to cause vertical movement of the brain.[85] This movement leads to displacement, stretch, and contusion injury of the sixth nerve in the vicinity of Dorello's canal, at the fixed dural entry point and at the apex of the petrous ridge.[86] Pathological examination of ten individuals with fatal severe head trauma showed that cranial nerve VI injury was greatest at the site of the dural entry point and the petrous apex, and identified injury at the site of anastomosis with the sympathetic plexus along the internal carotid artery.[87] 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.[86,88] Investigators of a prospective natural history study noted that seven of 25 patients with unilateral cranial nerve VI palsies and three of eight patients with bilateral palsy spontaneously recovered at 6 months.[89] Predictors of non-recovery include inability to abduct past midline and bilateral palsy.[90]

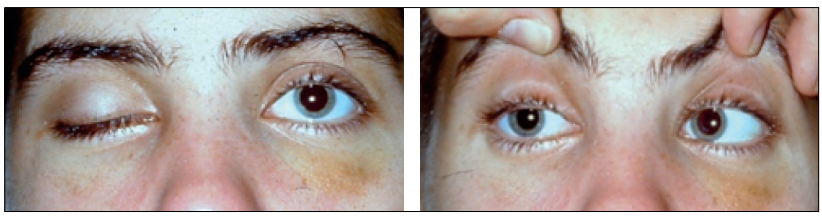

Figure 7: **Orbital apex syndrome**
This woman aged 24 years was involved in a car accident and had a right orbital apex syndrome with a fracture of the superior orbital fissure. She had complete ophthalmoparesis in the right eye with no light perception vision, also with greatly reduced vision in the left eye due to optic nerve injury.

Brainstem injury

In addition to the cranial nerve palsies, brainstem injury in TBI can cause several neuro-ophthalmic findings, including pupillary and motility disturbances. Brainstem injury often occurs in association with severe blows to the back of the head.[91] Traumatic internuclear ophthalmoplegia due to medial longitudinal fasciculus injury has been described as occurring bilaterally in 3 (1·6%) and unilaterally in 1 (0·5%) of 181 patients with TBI who were referred for neuro-ophthalmological assessment.[92] When 410 inpatients with internuclear ophthalmoplegia were reviewed, 20 (5%) had brain trauma as the cause.[84] Dorsal midbrain syndrome is also rare in patients with TBI, occurring in 2 (0·3%) of 326 of patients in one retrospective series.[49] Dorsal midbrain syndrome is characterised by paralysis of upgaze, pseudo-Argyll Robertson pupils, convergence retraction nystagmus, and eyelid retraction.

Sympathetic pathway injury

In one study, signs of Horner's syndrome were detected in three (0·9%) of 326 of patients with TBI.[49] Signs of Horner's syndrome associated with a traumatic cervical artery dissection can occur with penetrating and non-penetrating trauma.

Orbital apex, superior orbital fissure, and cavernous sinus syndromes

The orbital apex syndrome includes damage to the optic nerve, cranial nerve III, cranial nerve IV, cranial nerve

Search strategy and selection criteria

We identified articles with PubMed searches for combinations of "trauma" or "brain injury" or "head injury" or "concussion" and "neuro-ophthalmology" or "vision" or "saccade" or "pursuit" or "vergence" or "accommodation" or "vestibular-ocular reflex" or "optic neuropathy" or "third nerve palsy" or "fourth nerve palsy" or "sixth nerve palsy" or "Horner's" or "orbital apex syndrome" or "carotid-cavernous fistula" or "sideline test." We reviewed articles published from 1985 to April 10, 2014, but focused on those articles published after 2005. We identified further articles by reviewing reference lists within other articles, irrespective of year of publication. Prospective, randomised, controlled studies were preferred but scarce, so caveats regarding methods are mentioned. Primary references cited within textbooks, when available, were also reviewed irrespective of date of publication. Only publications written in English were reviewed.

VI, and the ophthalmic branch of the trigeminal nerve (figure 7). Superior orbital fissure syndrome includes lesions anterior to the orbital apex and leads to several cranial neuropathies without optic nerve involvement. The cavernous sinus syndrome usually spares the optic nerve but otherwise includes elements of the orbital apex syndrome with the addition of the maxillary division of the trigeminal nerve and sympathetic fibres—all of which might be due to blunt or penetrating trauma, possibly in the setting of skull fractures.[93]

Direct carotid cavernous sinus fistulas usually occur during trauma and can cause features of a cavernous sinus syndrome. Carotid cavernous sinus fistulas occur when there is a tear in the wall of the cavernous segment of the internal carotid artery, producing a connection between the carotid and venous channels of the cavernous sinous. Patients with carotid cavernous sinus fistulas might become symptomatic immediately or there might be a delay of days to weeks. Carotid cavernous sinus fistulas can cause proptosis, conjunctival chemosis, ocular pulsations, bruits, dysfunction of one or more ocular motor nerves or muscles, and vision loss due to an optic neuropathy or retinal or choroidal injury. Endovascular or surgical treatment is often curative.[94]

Orbital compartment syndrome

Orbital compartment syndrome is a very rare traumatic neuro-ophthalmologic emergency that can lead to loss of vision unless promptly recognised and treated.[95] It occurs when there is a sudden rise in intraorbital pressure to greater than systemic pressure, causing a fall in perfusion; it is 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.[95]

Conclusions and future directions

Head trauma has a multitude of effects on the visual system, necessitating a careful neuro-ophthalmic examination. Individuals with mild TBI or concussion might have deficits in executive function, visual attention, and visual memory, all of which can be assessed with tests of visual tasks including saccades or pursuits and can be quantitated with video-oculography. 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 or more substantial axonal shearing, leading to ocular motor neuropathies, optic neuropathies, and orbital pathologies.

Tests of visual function can be a sensitive means to assess minor head injury, providing promising sideline screening methods for sports-related concussion. 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 on the sidelines, such as parents of sports participants,[96] and could have applications for rapid testing in the military.

As understanding of the long-term repercussions of repeated head trauma grows, the need for objective and sensitive means to assess even subclinical brain injury becomes crucial. Findings from electrophysiological studies suggest that there are subtle, long-lasting changes in evoked response potentials in those with previous TBI who are doing visual memory tasks, even when the patients with TBI do as well as controls on those clinical tasks.[97,98] A combination of visual processing tasks, neuroimaging, serum biomarkers, and electrophysiological recordings might allow us to assess subclinical injury associated with head trauma. Further research is needed to identify exactly which patients with subclinical injury are at risk of chronic traumatic encephalopathy and should therefore be barred from further collision activity. Tests of visual pathway structure and function therefore not only help in the immediate assessment and management of TBI but could possibly be used to help to predict those at risk of long-term cognitive sequelae, while also providing outcome measures for treatment trials.

Contributors

REV and SLG made an outline of the manuscript. REV wrote the first draft. REV, LJB, and SLG edited the text.

Declaration of interests

LJB has received consulting fees for the development of visual outcome measures for multiple sclerosis clinical trials from Vaccinex, Biogen-Idec, Questcor, and Novartis. SLG has received honoraria for consulting from Biogen-Idec, Genzyme, and Vaccinex. REV declares no competing interests.

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