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Alice Cade & Philip RK Turnbull

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INVITED REVIEW

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Clinical testing of mild traumatic brain injury using computerised eye-tracking tests

Alice Cade and Philip RK Turnbull

School of Optometry and Vision Science, University of Auckland, Auckland, New Zealand

ABSTRACT

Traumatic brain injury (TBI) refers to the alteration of typical brain function that occurs following a blow to the head. Even a mild case of traumatic brain injury (mTBI) can lead to long-term impairment, so accurate and timely detection is vital. Visual symptoms are common following mTBI, so while it may seem to fall outside their typical scope of practice, optometrists are ideally qualified to assess the visual impacts and help with the diagnosis of mTBI. Given that mTBI is challenging to objectively diagnose and has no universally accepted diagnostic criteria, clinicians can lack confidence in diagnosing mTBI, and be hesitant in becoming involved in the management of such patients. The development of easily quantifiable techniques using eye tracking as an objective diagnostic tool provides practitioners with an easier pathway into the field, assigning numerical values to parameters which are difficult to assess using conventional optometric tests. As this evolving technology becomes increasingly integrated into optometric clinical settings, the potential for it to identify deficits accurately and reliably in patients following mTBI, and to monitor both their recovery and the effectiveness of potential treatments will increase. This paper provides an overview of clinical tests, relevant to optometrists, that can uncover oculomotor, attentional, and exteroceptive deficits following a mTBI, so that an optometrist with an interest in eye tracking can play a role in the detection and monitoring of mTBI symptoms.

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Traumatic brain injury

Traumatic brain injury (TBI) is the alteration of typical brain function that occurs when a blunt force to the head damages brain tissue.[1] The consequences of a TBI are dependent on the location and extent of the injury, but can include loss of consciousness, disorientation, memory loss, sensory changes, and, muscle weakness.[1] The resulting mechanical damage to brain tissue may be immediate (primary axotomy), or it may develop over time (secondary axotomy) due to biochemical or metabolic changes affecting nerve fibres and their local environment.[2]

The Glasgow Coma Scale is commonly used to classify TBI via a 15 point scale assessing motor, verbal, and ocular ability.[2] A Glasgow Coma Scale rating of three to eight indicates a severe TBI with a mortality rate of 40%; a rating of 9-12 indicates a moderate TBI with a 10% mortality rate, and a mild TBI (mTBI) is indicated by a Glasgow Coma Scale rating of 13-15 with a mortality rate of 0.1%.[3] The Glasgow Coma Scale does not have sufficient sensitivity to reliably differentiate or identify the milder form of TBI.[1,4] The King-Devick test, another common TBI test, is also flawed as it requires a pre-injury baseline, there is no clear consensus on what represents an abnormal score, and some athletes may 'sandbag' or purposefully perform poorly in their baseline testing.[5–7] Clearly, objective measures of mTBI are needed and eye-tracking is an up-and-coming diagnostic tool that may be useful in identifying mTBI.

This paper describes the epidemiology of mTBI and the types of oculomotor, attentional, and exteroceptive defects that may present following mTBI. Eye movements are neurologically complex actions, involving many areas of the brain, so unsurprisingly functional visual defects are a common finding following TBI, including mTBI.[8,9] Visuomotor defects can result from damage to the primary visuomotor pathway to the occipital lobe, along the parieto-occipital pathway, frontal lobes, cerebellum, or to the oculomotor cranial nerves themselves.[10] Furthermore, eye movements also require integrated action from the proprioceptive,[11] attentional,[12] visual salience,[13] and vestibular[14] brain centres, so injury to any of these areas can also lead to visuomotor abnormalities.[13,15]

Epidemiology

New Zealand has some of the highest rates of TBI and mTBI in the world, with approximately 790/100,000 for all TBIs and 749/100,000 mTBIs in 2011,[16] more than double the rate of Australia, whose most recent estimates were 322/100,000 for all TBIs in 1997.[17] Most TBIs in New Zealand are due to falls (38%) mechanical forces, (21%) transport accidents, (20%) or assaults (17%).[16] Males are disproportionately more affected by mTBI than females (Odds Ratio 1.85, 95% CI 1.66- 2.05),[16] which could be explained in part by sex differentials in risk-taking behaviour.[18] There is also significant ethnic disparity in New Zealand mTBI rates, with incidence rates at 1146, 839 and 236/100,000 for Māori, European and other ethnicities respectively.[16] The cause of this disparity is not clear, but could reflect higher rates of transport accidents, domestic violence, and lower socioeconomic rates in Māori, all of which are associated with higher rates of mTBI.[1,16]

The economic burden of mTBIs is increased by the longer-term effects that mTBI can have on the ability of the patient to work and to maintain their relationships with their family and community.[19] In 2010, the estimated cost to the economy relating to all levels of TBI in New Zealand was estimated to range from $65-138 million dollars annually[20]; patients with mTBI were estimated to cost on average NZ$5,085 during the first year after their injury, with subsequent average annual costs of NZ$6,908 over the course of their lifetime.[20] The personal burden is also

CONTACT Philip RK Turnbull p.turnbull@auckland.ac.nz School of Optometry and Vision Science, University of Auckland, Private Bag 92019, Auckland 1142, New Zealand

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substantial as deficits caused by mTBI can be so significant that those with mTBI symptoms (for example, lapses in concentration and memory, and a reduction in processing speed) cannot be distinguished from those with substance abuse issues.[19] This affects all aspects of life, including the ability to contribute to the workforce.[19]

Oculomotor function after mTBI

Oculomotor dysfunction, or impairment in the brain's ability to coordinate the eyes with accuracy and control, is present in up to 90% of patients following mTBI.[8,10,21,22] This difference means that a test battery including saccades, the head impulse test and pursuits are specific (97.0%) and sensitive (84.9%) in distinguishing healthy controls from participants with mTBI.[23] For 85-95% of patients all symptoms resolve within three months.[1] For the remaining 10-15% of patients, the miserable minority,[24] mTBI symptoms can persist, after which symptoms can be labelled *persistent* post-concussion syndrome.[1,25,26]

Pupillary dynamics

The size and response of pupils are routinely assessed to infer the neural integrity of the visual system.[27] Alterations in size and response provide important data to identify the site and nature of lesions along the complex afferent and efferent pathways of the visual system.[28] Clinical pupillary assessments, however, can be inconsistent and difficult to relate to the clinical exam because of the subjective nature of those assessments, and the small differences in pupillary dynamics. Recently, with the availability of computerised pupillometers, both researchers and clinicians can assess subtle abnormalities in pupil size and dynamics in a more controlled and quantitative manner.[27]

Pupil diameter and dynamics are determined by the interaction between the sympathetic and parasympathetic nervous systems,[29] and most injury occurs in the post chiasmal part of the pupil pathway.[30] Pupillary defects following mTBI tend to relate to the pupillary light reflex, and are commonly bilateral and symmetrical in nature, with no changes in prevalence of relative afferent pupillary defects and anisocoria compared to the general population.[27] Instead, mTBI can negatively affect the maximum diameter, amplitude and velocity of the constriction and dilation of the pupil, which are all difficult to assess without some form of computerised pupillometry.[31,32] Those with mTBI have small increases in constriction latency in response to light of 219 ms (healthy controls = 209 ms) and reduced average constriction velocity (mTBI = 3.6 mm s^{-1}, healthy controls = 4.4 mm s^{-1}) as found in a non-randomised controlled trial.[33] These alterations in pupillary dynamics are likely responsible for the common post-mTBI symptom of photosensitivity, which is present in 50% of those with mTBI (versus 10% of the non-injured population).[27,34]

Saccades

Exploration of the visual environment requires both saccades and fixations. Saccades move the fovea rapidly from one target to another, while fixations keeps the fovea aligned with the target for a certain time, allowing details of the scene to be processed.[35]

Pro-saccades are saccades directed towards a specific target.[35] Pro-saccade accuracy suffers after mTBI, with undershoots or overshoots that require a second saccade back towards the target more common.[12] Post-mTBI saccades also show lower velocities and lower peak accelerations for up to three months after the mTBI.[36] Furthermore, saccade latency, the delay between the appearance of a stimulus and the initiation of a saccade, is increased in patients who have experienced a mTBI (mean = 165.25 ms, SD = 40.72 ms) compared to healthy controls (mean = 149.09 ms, SD = 29.58 ms).[37]

Introducing a no-stimulus or gap interval between the presentation of a central target and the presentation of a peripheral target also reduces saccade latency for those with and without mTBI. If the central and peripheral targets are presented simultaneously participants with a mTBI react more slowly (~220 ms, while control group latency ~195 ms).[38] If there is a 150 ms delay between the central and peripheral target presentation saccade latency drops for both participants with mTBI (~180 ms) and controls (~170 ms).[38] This, along with increased saccadic latency, may suggest that people with acute mTBI may have difficulty quickly disengaging their attention from one target and moving their attention and focus to another target.[37–39] However, this difference in reaction compared with the control group is substantially reduced at one month post-injury.[38]

Anti-saccades are voluntary saccades directed away from a presented target. They require the participant to inhibit their initial reflex to look towards the target and instead look away from the target.[35] Compared to a conventional pro-saccade, this inhibition requires input from additional brain centres,[40] making anti-saccades a useful tool for assessing diffuse brain injury post-mTBI. Nominally, anti-saccadic latency is about 30% higher than pro-saccades, and this longer latency is thought to represent the additional cognitive effort in the parietal cortex,[41] frontal eye fields[42] and supplementary eye fields needed to shift visual attention and inhibit a reflexive saccade towards the target.[42,43] Patients with mTBIs would be expected to show an increased anti-saccade latency of around 40-50 ms over the typical delay of 240 ms in non-mTBI patients.[40,41] These latency effects persist for approximately one week,[40] potentially making anti-saccadic latency a good measure to distinguish if an acute mTBI has occurred. Interestingly, participants with previous mTBIs exhibit a deficit in anti-saccade accuracy (i.e. whether the participant performs the test correctly) for over five years post injury.[44]

Self-paced saccades have been used as a distinguishing marker of TBI.[45] Self-paced or volitional saccades made repeatedly between two targets have no clear consensus regarding what is being tested, be it accuracy, duration, or speed, and how a patient weights these two factors. Therefore, experimental paradigms must be carefully designed, which has not always been the case in the literature. However, a reduction in the number of self-paced saccades in those with mTBI has been found in some studies[46–50] but not all.[37]

Fixations

Fixations keep an object of visual interest steady in the fovea, making it possible to inspect a particular target at length.[51,52] Fixations are not a complete cessation of ocular movement, but rather include micro saccades, slow drifts and tremors, thought to help minimise image fading due to the Troxler effect,[53,54] and help keep the target centred on the fovea.[51] Fixation stability is a measure that can be made in clinic using an eye-tracker, which captures information on the accuracy of

gaze, as well as the micromovements within fixation.[55] As well as subconscious control of stability, fixation reflects the ability to attend a task.[56] Fixation stability in mTBI showed significant differences in the bivariate contour ellipse area, or the area that captures a given percentage of all the gaze points during the test,[36] in a paediatric population with mTBI (95% bivariate contour ellipse area (deg^2): mTBI: 6.16 ± 1.06, control: 5.64 ± 1.06, $p < 0.0001$).[57]

Reading can also be affected by mTBI, as increasing numbers of fixations and pro-saccades are indicators of reading difficulty.[58] Individuals with mTBI had a higher number of fixations per line (mTBI: 6.92 ± 1.69, control: 6.09 ± 1.39, $p < 0.01$), as well as a higher number of backwards eye-movements to re-read information (mTBI: 3.75 ± 1.04, control: 3.05 ± 0.94, $p < 0.01$).[59] Post-mTBI, patients will often complain that they lose their place reading, miss words or lines, read at a lesser speed, or become more fatigued while reading.[8,60] However, data showing increased numbers of fixations and pro-saccades post-mTBI should be considered with caution, as it does not indicate whether the problem is based in the oculomotor system or in higher cognitive centres that process comprehension of the written word.[61]

Smooth pursuits

Smooth pursuit eye movements aim to align a moving visual target with the fovea. Smooth pursuit movements are generated in the ipsilateral parietal and temporal cortices the cortical neurons determine the speed and direction of pursuit movements.[62] Damage to these areas of the brain manifests as saccade-like movements rather than smooth pursuits, while tracking a constant velocity target.[62]

During a smooth pursuit, the initial acceleration of the eye is predictive in nature, as saccadic masking prevents the acquisition of new visual information during the majority of the saccade.[62] However, even once the target is aligned with the fovea, smooth pursuits still need to be predictive in nature, as visual information takes between 80-100 ms to reach visual processing areas,[62] and the target's position must be extrapolated to create a continuous smooth tracking eye movement.[63] Unsurprisingly, with such a cognitive load, those with mTBIs can show reduced capacity for smooth pursuit tasks. They exhibit a gaze error (fixation versus target position) approximately twice as large as healthy controls (TBI: 1.7° ± 1.1° versus controls: 0.8°±0.2°),[57] with significantly reduced horizontal (mTBI = 0.86 ± 0.20, Control = 0.94 ± 0.06) and vertical (mTBI = 0.74 ± 0.24, Control = 0.83 ± 0.16) gains relative to the target velocity.[64] A systematic review of 17 papers investigating pursuits and saccades after mTBI found an overall effect size of Hedge's $g = 0.41$ ($p = 0.001$) under three months post injury, and $g = 0.28$ ($p = 0.001$) over three months post-injury.[21] Saccades, fixations, and smooth pursuits all lend themselves well to computerised eye tracking tasks and could easily be integrated into an mTBI protocol for testing.

Vergence

About half of post-mTBI patients have dysfunctional vergence movements.[65] Convergence insufficiency is the most common singular defect following mTBI,[8,60,65] affecting between a quarter to half of those with mTBI,[22,66,67] compared to a prevalence of 5% in the general population.[68] As well as a neuromotor deficiency,[67] convergence insufficiency following TBI is often attributed to dysfunction within attentional networks.[44,66,69,70] Early work by suggests that diffuse damage in mesencephalic optic supra-nuclear subsystems and their diffuse cortical and sub-cortical connections may play a role in attentional dysfunction.[71] More recent work implicates the dorsolateral prefrontal cortex, a portion of the frontal lobe, part of the parietal lobe, the cerebellum, and the brain stem,[70] areas in which the mechanical forces following head trauma can greatest.[72]

Convergence insufficiency clinically manifests as reduced near point of convergence and following mTBI the near point of convergence decreases from 2.49 ± 1.74 cm in those with normal binocular vision (Age: 22-37 years, average age, 24.9),[73] to between 4.17 ± 2.21 cm (age 18–23 years, average age 20.52)[74] to 12.3 ± 4.7 cm (age: 10-21 years, average age: 14.7)[75] following a TBI.

Vestibulo-ocular reflex

The vestibulo-ocular reflex maintains visual fixation on a target during head movement.[76] This reflex yokes the eyes to a chosen target, meaning that if the eyes focus on a target and the head rotates 25° to the right, for example, the eyes should rotate 25° to the left with a gain of 1.0 in a healthy individual.[77] While the vestibulo-ocular reflex should ideally cancel head movement, in the dysfunctional individual, the gaze point can slide off the object of interest, requiring a corrective saccade.[76]

Traditionally, the vestibulo-ocular reflex is tested using the passive head impulse test.[78] Here the participant fixates their eyes on a stationary target and a clinician rotates the head left or right around the Z axis (positive and negative yaw movement, or turning the head right and left, respectively[79]) through a short arc of 10-20°; this rotation is performed at approximately 100-200° per second.[79]

The head impulse test was created to test the semi-circular canals via the vestibulo-ocular reflex and usual testing consists of a test of yaw, pitch (nodding the head up and down), and roll (laterally flexing the head to the ear) by looking diagonally up and down.[80] Pitch and roll head impulse tests are rarely performed separately.[80] An actively performed head impulse test, where the participant moves their own head, may uncover more signs of adaptation to dysfunction (such as covert saccades, or low latency saccades that occur when the head is still moving,[81]) potentially making an active head impulse test a more appropriate choice when assessing vestibulo-ocular reflex in those with mTBI.[77,82] Yaw head impulse test results, in healthy individuals, have been found to be stable with repeated testing, with active and passive testing showing similar gains (Active: 0.99 ± 0.08, passive: 1.0 ± 0.08).[83] Individuals with mTBI show lower vestibulo-ocular reflex gains in active yaw head impulse tests (TBI: 0.79 ± 0.15, control: 0.87 ± 0.18).[82] The latter study, should however, not be viewed in isolation as it involved a smaller number of participants (n = 24) with no control group.

Computerised eye-tracking has greatly improved the sensitivity of the head impulse test, with some eye-trackers also capable of tracking the head rotation as well, giving precise gain measures making the vestibulo-ocular reflex a good choice for inclusion in a battery of tests for mTBI.

Optokinetic nystagmus

The optokinetic reflex aims to stabilise retinal slip across a wide portion of the retina,[84] and consists of two distinct components – a slow phase in the direction consistent with

the optic flow, and a fast-phase saccadic movement in the opposite direction.[85] A common example of the optokinetic reflex are eye movements that occur when watching passing telephone poles from a moving car or train.

The tracking phase of the optokinetic reflex (in combination with a battery of other tests(saccades, head impulse tests, and pursuits)) is sensitive (84.9%) and specific (97.0%) in distinguishing healthy controls from participants with mTBI.[23] Furthermore, those with mTBI report more symptoms, including dizziness, headache, and nausea, after optokinetic reflex testing compared to healthy subjects.[86,87] Optokinetic reflex testing is therefore a potentially useful tool in diagnosing mTBI, however more research is needed to determine if optokinetic reflex is uniquely useful in identifying those with mTBI therefore optokinetic nystagmus might not yet be a useful addition to a better of tests used in mTBI patients.[23]

Selective attention

During everyday life, many distinct pieces of information, from different sensory organs, converge simultaneously into specific brain areas, some to be attended to and some ignored to build a unified perception of the environment. In order to maintain our attention on everyday tasks, such as reading a book in a public place, we must filter out other stimuli like other's conversations, birds chirping, or vehicles passing.[88] Many neurological structures involved in selective attention are affected by mTBI, such as the visual circuits involved in anti-saccades.[9,35,89] The Stroop Color and Word Test is used to assess information processing under different levels of stimuli compatibility or conflict.[90] In the Stroop Colour and Word Test the participant is presented with a written name of a colour which may be printed in a conflicting colour of ink. There are two types of tests, both of which involve matching from a set of potential colours; either the written colour, ignoring the colour it is written in, or matching the font colour, ignoring the written word.[90]

The Stroop Colour and Word Test examines interference control, a form of cognitive inhibition that allows suppression of the automatic response (for example the colour of the printed word) in favour of the correct response (reading what the word says).[91] A meta-analysis on all types of TBI severity shows the greatest discriminator of TBI deficit was total time on task (Effect size = 1.4 (large), $p < .001$)).[91] In studies using the Stroop Colour and Word Test requiring a participant to read out 100 words whose printed colour was different to the word written, mean total time on task was 149.48 ± 49.42 for those with TBI, compared to 83.64 ± 19.4 for healthy controls.[88]

The SCWT would be a useful addition to a battery of eye tracking tests as its stresses attentional processes, rather than just oculomotor function.[91] To adapt the SCWT to an eye tracking format the coloured words could be presented on-screen, then gaze would be used to match the colours to a palette presented in each corner of the screen, which could give quick and easy scoring and reduce the influence of trial-by-trial feedback and ambiguity that might occur with verbalised responses.

Spatial visual processing

Deficits in spatial visual processing occur in 30% of people following TBI.[92] To successfully interact with the world we must accurately estimate our own body position and motion (exteroception).[93] If spatial visual processing is affected this can lead to feeling out of sync with the environment.[94] People may have difficulty judging distances and maintaining awareness and memory of the position of objects and where they themselves are in space.[8,10,92] There is limited data regarding spatial visual processing in those with mTBI, but magnetic resonance imaging shows functional abnormalities in brain activity in the parietal cortex, right dorsolateral prefrontal cortex, and right hippocampus after mTBI when performing spatial memory navigation tasks.[95]

One spatial visual processing issue post-mTBI that can be easily assessed in clinic, is abnormal egocentric localisation. This refers to the inability of an individual to accurately ascertain what 'straight-ahead' is, relative to their body.[10,96] This is thought to be due to an alteration in their internal spatial map of the world, possible due to right parietal lobe dysfunction.[94,97]

It is possible to assess spatial perception by way of co-opting cerebellar tests such as the finger-to-nose test or by using wearable collars to hide the hands of the patient as they attempt to point to their subjective midline.[98] If participants have a left or right-sided brain lesion they tend to locate their subjective midline towards the side of damage,[99,100] while those with no brain lesion point slightly leftward.[101] Spatial visual processing is relatively simple to test using even low-cost computerised eye-trackers as some eye-tracker now record head position and head rotation as well as the point of regard on screen.[102,103]

Choosing a battery of computerised eye-tracking Tests for mTBI

A patient with a possible mTBI may present with a range of visuomotor and cognitive symptoms, many of which can be investigated using a low-cost eye-tracker. To build a working internal representation of the world around them, a person needs to be able to differentiate self from world motion. As the eyes also move with respect to oneself, this means that accurate fixation, saccades, smooth pursuits, vestibulo-ocular reflex and optokinetic reflex reflexes are required to stabilise the world onto the fovea and localise the body in the environment, before specific objects can be attended to, without the symptoms of asthenopia seen in mTBI.

To determine if someone may have symptoms resulting from mTBI, it would be useful to assess multiple steps along this pathway. Specific tests include assessing pupil constriction latency and velocity, fixation bivariate contour ellipse area, gaze point–to–target error in pursuits, pro- and anti-saccade latency, vestibulo-ocular reflex gain, time on task in Stroop, and the body–to–target error in egocentric localisation (Table 1).

Choosing an eye-tracker

When choosing an eye-tracker there are five important considerations: 1) Spatial accuracy, or the difference between the point on the screen on which the participants gaze falls and what is actually reported by the eye-tracker, 2) Precision, or the difference in sample-to-sample reported position when gaze is fixed on the screen, and 3) the area in front of the screen the eye-tracker can record in, and 4) the sampling frequency, and 5) cost of the hardware, and possibly additional software.[103] An additional consideration is whether restraining or not-restraining the participants head is important. Restraining the participants head can improve tracker accuracy but will not allow for natural head

Table 1. Some of the most informative tests that can be assessed using a computerised eye tracker in a clinical setting, with the expected normal and mTBI values. Note that no one test is likely to be diagnostic by itself, but rather a range of tests may be required to diagnose mTBI with confidence.

Test	mTBI	Healthy	Reference
Pupil constriction latency (ms)	219	209	33
Pupil constriction velocity (mm s-1)	3.60	4.40	33
Fixation BCEA (Deg^2)	6.16	5.64	57
Pursuit gaze point–to–target error (deg)	1.70	0.80	57
Pro-saccade latency (ms)	165.25	149.09	37
Anti-saccade latency (ms)	280-290	240	40,41
VOR gain	0.79	0.87	82
Stroop time on task (ms)	149.48	83.64	88
Egocentric localisation error	Points away from side of lesion	Points leftward	99,100

movements or tests like the head impulse test. For example, a commonly used and expensive eye-tracker the EyeLink (which retails for over USD$10,000) showed large horizontal and vertical gaze offsets in tasks requiring head movements.[103] In recent years several low-cost eye-trackers have been produced for both research and gaming purposes such as the Eye Tribe, Pupil Labs, Gazepoint, and the Tobii 4 C. Previous research was divided around the accuracy and precision of the consumer versus research-grade trackers, with some suggesting they are useful for fixations, gaze analyses, and pupillometry but, not accurate enough or have too low temporal resolution for detailed saccade metrics.[104] Others argue that with careful set-up lower-cost trackers can be a useful, accurate tool.[105] The Gazepoint system, a mid-range system retailing for around NZ$3-4000, can record saccade events, with a lower sampling rate (150 Hz), but does not record head movement.[106] The Tobii eye-tracker (which retails for around €200) maintains comparable accuracy and precision when compared to more expensive trackers.[102] However, if eye tracking data is to be stored, the cost of additional software or licences needs to be factored into the price. The Eye Tribe, being the cheapest tracker, may have similar issues to the EyeLink tracker with participant head movement.[103] The Tobii eye-tracker, on the basis of price, head-tracking, precision, and accuracy, seems to be the best of the lower-cost trackers, even with the addition of the licencing fee. Current research is aimed at developing a battery of reliable, objective optometric tests, with software that will become available for in-office use,[107,108] so that many of the measures can be quantitatively assessed in a clinical setting.

Summary

Control of eye movements relies on multiple areas of the brain, making them vulnerable to damage caused by TBI.[8,10] Those patients suffering from mTBI have significant differences in oculomotor control compared to healthy individuals. This can cause significant disruption to the social and professional life of the patient, but the abnormal cortical function provided opportunity for diagnosis.[1] While many of these oculomotor defects are currently difficult to detect in the clinic, current research is developing a battery of tests to be used with a lower-cost eye-tracker for identifying mTBI that can be used in day-to-day clinical practice.

Furthermore, as the recovery from mTBI is highly variable, reliable, and objective oculomotor function tests are essential for tracking patient outcomes after TBI. Non-invasive eye tracking experiments using these techniques are common in many research fields, including vision science, psychology, automotive sciences, sport and exercise sciences, marketing, and the gaming industry.[105] A lower-cost eye-tracker, with appropriate battery of tests, as described above, may be a useful addition to the clinicians range of assessments when managing the patient with mTBI.

Disclosure statement

No potential conflict of interest was reported by the author(s).

ORCID

Alice Cade http://orcid.org/0000-0002-3050-3958
Philip RK Turnbull http://orcid.org/0000-0002-9892-2964

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