ARTICLE

Basic Neuro-Optometric Diagnostic Tests for Mild Traumatic Brain Injury/ Concussion: A Narrative Review, Perspective, Proposed Techniques and Protocols

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ABSTRACT

Vision problems are common in individuals with mild traumatic brain injury (mTBI)/ concussion. However, a global conceptualization of the diagnostic process remains incomplete and practitioner dependent. Thus, a comprehensive diagnostic test battery is proposed to assist in the management of these patients. This battery includes a range of basic clinical tests of a sensory and motor nature, with all having a clinical and scientific rationale. These tests have been used by the authors for many years, with good success, and furthermore they have been found to be clinically useful and insightful.

INTRODUCTION

A new area of challenge for the neuro-optometrist, as well as the general optometrist and others (e.g., vision therapist, occupational therapist, classroom teacher), is the medical condition of mild traumatic brain injury (mTBI)/ concussion.[1] mTBI has been highlighted by the medical community and the media in regard to the sports arena and military theater,[1] both nationally and internationally. It has been estimated that there are 10 million such head injuries annually in the world,[1] and at least 1.7 million in the United States.[1]

Individuals with this type of brain injury will frequently manifest a constellation of general medical problems, including those of a sensory, motor, perceptual, linguistic, behavioral, attentional, cognitive, and/or physical nature.[1-4] For example, the patient may report sleep problems, risky behaviors, memory loss, and hyperacusis. Diagnostic test batteries are reasonably well established for the wide range of basic medical dysfunctions found in this population.[4]

Similarly, these individuals will frequently manifest a constellation of vision problems, including those of a sensory and motor nature.[1-3,5,6] For example, the patient may report light sensitivity, eye tracking problems, intermittent blur, and diplopia. See Tables 1

Table 1: Oculomotor and visual symptoms in traumatic brain injury (TBI)

- Avoidance of near tasks
- Oculomotor-based reading difficulties
- Eye-tracking problems
- Eye-focusing problems
- Eye strain
- Diplopia
- Dizziness
- Vertigo
- Vision-derived nausea
- Increased sensitivity to visual motion
- Visual inattention and distractibility
- Short-term visual memory loss
- Difficulty judging distances (relative and absolute)
- Difficulty with global scanning
- Difficulty with personal grooming, especially involving the face
- Inability to interact/cope visually in a complex social situation (e.g., minimal eye contact)
- Inability to tolerate complex visual environments (e.g., grocery store aisles and highly patterned floors)

From Ciuffreda KJ, Ludlam DP, Kapoor N. Clinical oculomotor training in traumatic brain injury Optom Vis Dev 2009; 40:16-23; with permission.

Table 2: Oculomotor signs in TBI

- Reduced amplitude of accommodation
- Increased lag of accommodation
- Slowed accommodative facility
- Uncorrected hyperopia/astigmatism (due to inability to compensate accommodatively)
- Receded near point of convergence
- Restricted relative convergence (BO) at far and near
- Restricted overall fusional vergence ranges at far and near
- Abnormal Developmental Eye Movement test results
- Low grade-level equivalent performance on the Visagraph II
- Impaired versional ocular motility

From Ciuffreda KJ, Ludlam DP, Kapoor N. Clinical oculomotor training in traumatic brain injury Optom Vis Dev 2009; 40:16-23; with permission.

and 2. For the neuro-optometrist and others, however, diagnostic test batteries are relatively new, evolving, and expanding in this field,[5,6] and furthermore may differ considerably among practitioners. The development of a comprehensive diagnostic test battery would provide for a better degree of uniformity among practitioners, with the likelihood of improving patient care.

Such a comprehensive, updated, and more complete diagnostic test battery is proposed in the present narrative review. It encompasses a range of basic clinical test procedures for the neuro-optometrist and others involving the sensory and motor systems to help the patient with mTBI/concussion and their vision problems. To a great extent, it reflects the views and perspectives of the authors, who have decades of clinical and research experience in the area.

Pre-Diagnostic Testing

Prior to detailed diagnostic testing, it is assumed that the following two areas will have been thoroughly assessed: *refraction and ocular health*.[1,5,6] Without this information, the clinician may be misled regarding the presence of an "apparent" sensory or motor dysfunction in the subsequent, full, diagnostic test battery. Of course, this would be preceded by a thorough case history.

Refractive assessment performed monocularly and binocularly at distance and at near is the cornerstone for all subsequent testing. This is especially true for the assessment of sensory aspects. For example, presence of excessive retinal defocus and blur due to a poor refraction may lead to an "apparent" vision deficit, such as the finding of reduced contrast sensitivity at the high spatial frequencies.[5,6] Thus, performing both a careful subjective refraction, with objective autorefraction confirmation, is essential. And, if there is a likely accommodative problem, especially in a younger patient, a cycloplegic refraction would be mandated to ascertain the etiology of the problem.

During the refraction, or at other times, the patient may report blur at distance and/or near. Since the autonomic system is frequently dysfunctional in mTBI,[4] the presence of blur could be due to impaired sympathetic and/or parasympathetic pharmacological control

of accommodation. In the former case, over-accommodation in the distance would be predicted, whereas in the latter case, under-accommodation at near would be expected. An interesting and common example is the younger uncorrected, slightly hyperopic (e.g., +1.0D) individual who was able to compensate via accommodation for the hyperopia prior to their injury; however, post-injury they frequently cannot, as this sustained accommodative ability is compromised. They now present themselves as a low hyperope with the symptom of slight blur at near due to impaired accommodative parasympathetic drive.

Ocular health, including the retina and optic nerve, is also critical to assess fully and carefully, as such problems are common in this population.[7] Presence of a retinal disease would likely adversely affect subsequent diagnostic testing, especially sensory and motor aspects. For example, in an older patient with concurrent, advanced bilateral macular degeneration, saccadic tracking and reading would be impaired: they would execute an excessive number of saccades to fixate the target using their eccentric retinal locus.[8]

Thus, in the patient with mTBI, the presence of refractive and/or ocular health problems, if not detected and taken into consideration initially, could lead to a misdiagnosis. Their presence would also have an adverse therapeutic impact, which is beyond the scope of this paper.

Lastly, for all aspects of testing in these patients, there are some general guidelines to yield high quality information.[2,3] First, they fatigue rapidly, even on a simple test such as visual acuity. Brief rest periods are warranted. Second, and related to the above, testing will typically need to be performed over two or three sessions, perhaps each separated by at least two days for full recovery from fatigue. Third, vision testing should not be conducted on the same day as other therapies, if possible, such as cognitive therapy, due to fatigue effects. Lastly, and related to the above, due to cognitive impairment and auditory processing deficits common in the mTBI patient,[1] the test instructions should be articulated slowly and likely need to be repeated once or twice.

Diagnostic Testing

Sensory Assessment (Table 3)

Table 3: Sensory Testing

- distance visual acuity (OD, OS, OU)
- near visual acuity (OD, OS, OU)
- distance dynamic visual acuity (OU)
- near dynamic visual acuity (OU)
- visual field screening (OD, OS, OU)
- contrast sensitivity (OD, OS, OU)
- photosensitivity (PS)
- visual motion sensitivity (VMS)
- critical flicker fusion frequency (CFF)
- coherent motion (CM)
- stereoscopic sensitivity (SS)
- egocentric localization (EL)

Normal sensory function is critical for the detection and processing of visual stimuli in one's environment, as well as for reacting motorically when needed, in an efficient, time-optimal manner. In contrast, presence of a sensory dysfunction would lead to impaired detection and processing of the relevant visual stimuli, thus leading to inappropriate motor responsivity (e.g., ambulatory instability, visuomotor errors). Hence, deficits in visual sensation will also frequently adversely impact on one's fine and gross motor performance.[1,2,5,6]

In this section, the diagnostic sensory assessment in the mTBI/concussion patient will be explored. This will include the basic phenomena and their clinical ramifications.

Best corrected, static, distance visual acuity is typically (~90% of the time) 20/20-20/25, in the absence of an accommodative problem or ocular disease, in the TBI general population.[9] Thus, if found to be considerably worse, the refraction and ocular health should be reassessed. Also, per the cortical phenomenon of binocular summation[10] (i.e., binocular enhancement), suprathreshold binocular visual acuity should be approximately 7% better,

or roughly one-half line of improvement, over that found monocularly. If a summation effect is not evident, the clinician would need to recheck refractive accuracy, as well as ocular/neurological health, and also reexamine the patient for the presence of a binocular anomaly, such as mild suppression, intermittent strabismus, or very mild long-standing amblyopia. Neural processing of contrast-based visual acuity is performed in the visual cortex (V1).[11]

Best corrected, static, near visual acuity in this population is also typically 20/20-20/25,[9] again in the absence of an accommodative problem or presence of ocular disease. If this is not the case, retesting as described above for distance visual acuity should be addressed. Binocular summation should also be present at near. As described above, neural processing of contrast occurs in V1.

Distance dynamic visual acuity (DVA) refers to one's sensation of visual stability and visual clarity while reading a distance visual acuity chart with purposeful and controlled horizontal and vertical head rotation (2 Hz), thus stimulating the vestibulo-ocular reflex (VOR)/vestibular neurological system.[12] The resultant perception of blur and/or oscillopsia, if present, and being greater than two lines worse than the static visual acuity, is suggestive of a vestibular pathway dysfunction. Transient diplopia may also be reported, presumably due to a disturbance of fusion and/or an abnormal vergence-vestibular interaction.[1,5,6]

Near dynamic visual acuity (DVA) refers to the same phenomenon as described above for distance DVA. Testing is similar, but now performed at near. Diagnostic and neural aspects are as described above for distance testing.

Visual field testing in patients with mTBI is especially important, as visual field deficits are common (~40%), especially of the "scattered, diffuse" variety.[13] At a minimum, confrontation testing in the four visual field quadrants should be performed. In addition, the Humphrey Frequency Doubling Technology (FDT) threshold device has been effective in this population,[14] as well as being rapid, having good sensitivity, being easy to use, and also patient friendly. The FDT assesses the visual magnocellular pathway.

Regarding contrast sensitivity (CS), deficits have been found clinically when assessed across the general TBI population: 21% manifested some degree of reduced contrast sensitivity.[9] Therefore, contrast sensitivity should be assessed, especially in those with suggestive symptoms, such as having difficulty reading small, lower contrast text (e.g., newspaper print). Per the neurological phenomenon of binocular summation mentioned earlier, the contrast sensitivity threshold should be approximately 42% better binocularly than that found monocularly. If not, the earlier suggested retesting of refraction and ocular health would need to be performed, as well as assessment for presence of a subtle binocular anomaly. In a recent laboratory study,[15] several deficits in contrast sensitivity were found, including impairment for lower spatial frequency stimuli, thus suggesting cortically-based neurological factors and not optical factors to be involved. Additional testing in this area should be conducted in the future, especially in the mTBI/concussed population per se, which encompasses the largest percentage (~80%) of the general TBI population.[1]

Photosensitivity (PS) refers to the symptom of visual discomfort in the presence of illumination conditions which normally do not provoke such a sensation in others. This is especially true for fluorescent lighting with its inherent 60 Hz flicker. This visual discomfort may be related to the elevated (but high normal) critical flicker fusion (CFF) frequency found in the mTBI population.[1,5,6] PS is present in approximately 10% of the general population, and in about 50% of those with mTBI.[16] PS has been proposed to be due to a baseline light sensor deficit residing in the superchiasmic nucleus and/or a perceptual

Fig. 1: Two examples of Gibsonian optic flow from one's naturalistic environment.

gain problem residing in the lateral geniculate nucleus.[17] About 50% of the mTBI population exhibit visual adaptation to PS, at least to some extent; unfortunately, this desirable neuro-adaptive process may take one year or longer in many cases.[16]

Visual motion sensitivity (VMS) refers to the occurrence of specific visually-related symptoms in response to a moving visual stimulus, especially one encompassing the peripheral visual field, which normally does not provoke the same abnormal sensations in others[1,5,6,18] (Figure 1). Some examples of provocative visual stimuli include: the scrolling of a computer screen, walking in a busy environment such as a supermarket aisle (e.g., "supermarket syndrome") or mall producing Gibsonian "optic flow" patterns,[18] sitting in the back seat of a moving vehicle and gazing out the window, driving, and riding on an escalator. Being exposed to such visual stimuli may lead to the feeling of imbalance, dizziness, disorientation, impaired spatial orientation, and even nausea (e.g., "car sickness").[19] It is estimated that VMS is present in approximately 40% of the mTBI population.[1,18] Two types of diagnostic tests have been used to assess for the presence of VMS, other than case history. The first includes the addition of binasal occluders (BNO) to the patient's distance spectacles to assess for the presence of VMS, for example while ambulating along a long corridor (Figure 2). The BNO reduces the amount of dynamic peripheral visual motion impinging upon the bitemporal retinas arising from the contralateral temporal visual fields.[20] The application of the BNO should reduce the disturbing sense of visual motion. A second approach is the use of either an OKN drum/tape or the examiner's hands to produce visual motion in the periphery, as the patient gazes along the midline at a blank surface located several feet away.[1,5,6,18] The drum or hand motion should increase the sense of visual motion and related symptoms. In the laboratory, virtual reality (VR) systems have been used to produce controlled provocative stimuli, and also to quantify the effects on balance.[19] While cortical areas VS/MT/MST are involved in the early processing of visual motion,[21] recent evidence suggests that the newly-discovered cortical areas V6/V6a are involved in higher-level processing of this specific information.[18] These areas are capable of parsing out visual object motion from one's self-motion across the entire visual field. If their difference is accurate and thus veridical, then visual stability ensues; if not, visual instability and the symptom of VMS will occur.

Fig. 2: Binasal occluders (BNO) attached to a spectacle frame.

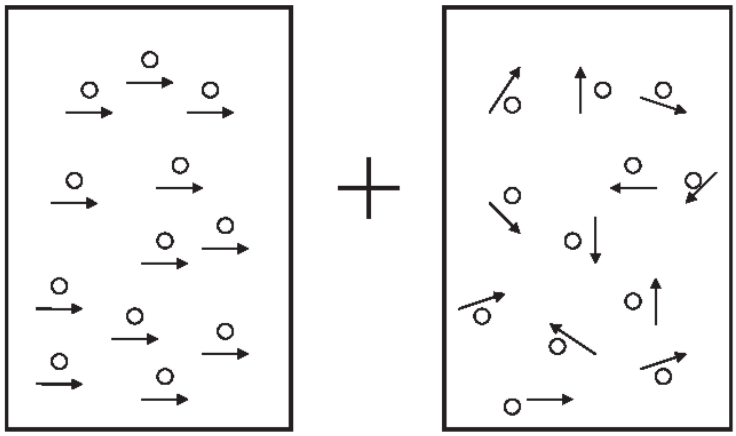

Figure 3: Left shows 100% coherent motion, and right shows lack of coherent motion. Presented on a display monitor along the subject's midline.

The coherent motion threshold (CMT) refers to the smallest percentage of coherently-moving dots whose direction can be reliably detected in a field of otherwise randomly-moving dots (Figure 3). Thus, it tests the ability of the individual to detect motion in the absence of context in a controlled manner. Normal CMTs typically range from about 3.7% to 5.6%,[21] thus demonstrating a highly sensitive visual motion system. Neurologically, coherent motion is processed by the magnocellular pathway including the parietal stream, V5, and MT.[21] The CMT has been assessed in the mTBI population using a two-alternative, forced-choice (2AFC) paradigm with a double-interleaved staircase.[21] CMT was found to be significantly elevated in the mTBI versus normal population (8.8% versus 6.5%). This finding suggested damage to the aforementioned neurovisual pathways. Interestingly, this increased CMT was related to the presence but not graded magnitude of their symptoms, namely dizziness, disequilibrium, vertigo, nausea, and visual motion sensitivity, all occurring in their normal environments.[21]

Critical flicker fusion frequency (CFF) refers to the highest rate of physical light flicker at which an individual does not perceive any flicker (~40 to 60 Hz).[22,23] This temporally-based test assesses the basic overall integrity of the nervous system, which is processed by V5/MT/MST cortical regions.[21-23] Two studies have found CFF to be in the "high normal" range in those with mTBI, and furthermore with this elevated value being related to the presence of VMS and PS in this population.[21-23] The finding of a relatively high CFF value is likely related to these patients frequently having a strong and specific dislike for fluorescent room illumination: they perceive the high frequency fluorescent light flicker (60 Hz).

Stereoscopic sensitivity (SS), or "stereopsis", refers to the smallest horizontal, angular retinal disparity that an individual can detect binocularly, and furthermore can localize in depth relative to a reference target of effectively zero disparity.[10] Stereopsis is best assessed using a random-dot stereogram (RDS) target.[10] Stereopsis is used in conjunction with other monocular (e.g., overlap, linear perspective) and binocular (e.g., vergence innervation, extraocular muscle proprioception) information to judge the relative depth of objects in the environment.[10] Stereoacuity is slightly reduced clinically in mTBI (e.g., 40 versus 20 sec arc in normals).[24] This has been comfirmed in a recent laboratory investigation.[25] Both the dorsal and vertral neural streams are involved in the processing of retinal disparity,[26] with the former assessing the disparity metric, and the latter the disparity sign. Disparity is initially processed in V1, with subsequent signals transmitted to many brain areas.[26] For example, this information travels to V4, and also to the corpus collosum specifically for the processing of midline disparities.[10] It is believed that this reduced stereoacuity is related to damage and dysfunction of these cortical areas,[25] as well as to a small vergence oculomotor error (i.e., fixation disparity related to the typically large near phorias present) resulting in less precise stimulation of corresponding retinal points.[27]

Egocentric localization (EL) refers to one's perception, or "sense", of "straight-ahead."[28] (Figure 4). It incorporates a midline, body-based, *polar* coordinate system, with the individual's sternum functioning as the visual-perceptual, directional origin projecting into visual space. This reference system is used,

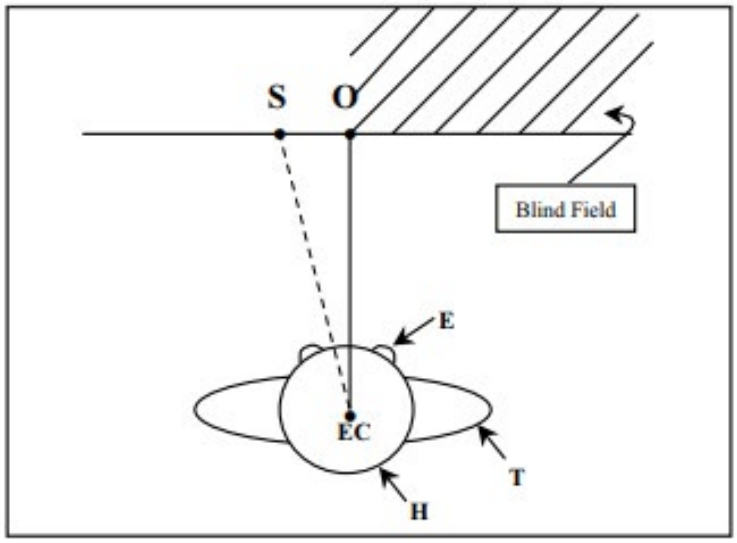

Fig. 4: Abnormal egocentric localization ("sense of straight-ahead") in a patient with right hemianopia, with it being biased to the seeing hemi-field. Symbols: O=objective, veridical straight-ahead along the midline, S=abnormal subjective sense of straight-ahead, E=eyes, T=trunk H=head, and EC=egocenter. Top view.

for example, when pointing at an object. In the laboratory, EL is typically assessed under reduced cue conditions, such as in near total darkness, so that information from the environment does not provide a visual "frame-of-reference" that could contaminate or bias the result.[28-30] However, for simplicity and practicality, it is typically performed in the standard clinic setting, with it still providing valuable diagnostic information.[28] Briefly, while seated, the patient is asked to "gaze" straight ahead (i.e., in primary position) at an uncluttered wall, while the doctor slowly moves a thin wand or pencil first laterally, and then vertically, towards the center of the patient's visual field, and asks when the target is perceived to be straight-ahead.[28] The difference between the objective, or veridical, midline zero position and the subjective, or perceived midline position, represents the amount of polar rotation in EL. A deviation of greater than 1-2 degrees from this objective zero position, either horizontally or vertically or both, would be abnormal, that is the patient would have abnormal[29] EL, or AEL. AEL is estimated to be present in at least 50% of the mTBI population.[1] Individuals with AEL have a range of symptoms, such as difficulty ambulating, a sense of disorientation, and feeling "out-of-synch" with their environment. The neural site involved in EL is the posterior parietal cortex,[30] which provides a properly calibrated map of visual space in normal individuals. This map is frequently distorted and biased laterally by several degrees in mTBI.[28] Vertical biases may also be present, but smaller.[28,29]

Motor Assessment (Table 4)

Table 4: Motor Testing

- near point of convergence (with repetition)
- cover test (distance and near; horizontal, vertical, cyclorotary)
- prism facility (distance and near; fatigue aspect)
- Brock string for vergence
- prisms at near "comfort" test
- amplitude of accommodation (OD, OS, OU)
- lens facility (OD, OS, OU; fatigue aspect)
- lens at near "comfort" test
- DEM/K-D (global saccadic tracking)
- optokinetic nystagmus
- penlight test (PLT) (pupil, ocular motility)

The near point of convergence (NPC) represents the closet point of binocular fixation attained with maximal effort exerted,[27,31] typically being 5 cm in normals, when properly measured to the center of rotation in the eye. This test incorporates all four components of vergence, namely disparity/fusional, accommodative, proximal, and tonic.[27] Several studies have found the NPC to be receded in mTBI,[24,32-34] occurring in approximately 75% of these patients. Thus, it has been proposed to be a good clinical biomarker for the presence of mTBI/concussion,[35,36] given its simplicity, and especially if used in conjunction with other high-yield clinical vergence tests (e.g., distance horizontal prism flipper) to increase diagnostic sensitivity and specificity (e.g., using ROC analysis).[17] A receded NPC would typically adversely affect sustained near vision activities and produce asthenopia. Midbrain control of vergence consists of three cell types:

tonic cells related to vergence angle, burst cells related to dynamic vergence velocity, and tonic-burst cells that carry both signals.[33] There are also many higher-level control areas involved in vergence, such as the parietal cortex.[33] NPC recession suggests primary dysfunction of vergence tonic cells.

The cover test (CT) is used to determine steady-state eye position (horizontal, vertical, and cyclorotational) when fusion (i.e., disparity vergence) is prevented.[27] With either monocular occlusion or vertical prism dissociation, only accommodative (primary), proximal (secondary), and tonic (tertiary) vergence remain, and they interact non-linearly to provide the global vergence phoria eye position.[27] The greater the CT deviation from orthophoria, the greater the overall vergence demand for accurate bifixation/fusion. Clinical findings regarding the CT in mTBI/concussion are equivocal. Typically, large exophoria (e.g., 8 pd) is found,[34] although large esophoria (e.g., 6 pd) is not uncommon (~30%),[37] with presence of either presumed to cause near symptoms, such as asthenopia, diplopia, etc. Also, presence of even small amounts (e.g., 0.5 pd) of vertical hyperphoria can be highly visually-symptomatic in this population[38] due to the occurrence of phoria decompensation[39] and related compromised vergence adaptation.[39] Knowledge of vergence neural control under this condition remains incomplete, but clearly all midbrain disparity vergence cells are deactivated, presumably with analogous accommodative midbrain neural units activated to maintain steady-state accommodation and correlated accommodative vergence during the measurement.[27]

Prism facility testing assesses the overall dynamic interactions of the vergence system, in particular the fusional/disparity component.[27] This test can be performed at distance (6m; 4B0/2BI)[40] and at near (0.4m;12B0/3BI)[41]. The number of alternations executed between the two prismatic demands for which one can rapidly and repeatedly fuse a small, detailed target over a one minute period is recorded, with comparison to normative data. Clinically, abnormality is reflected in a reduced number of cycles/minute. Prism facility is particularly reduced at distance in mTBI/concussion, and thus it appears to be another good "clinical" biomarker for its presence.[40] In addition, this test may be good to assess for "fatigue" of the vergence system in this population.[42] Neurological control likely originates from the midbrain burst cells related to the transient aspect of vergence, predominantly the vergence peak velocity parameter.[33] Furthermore, the basic neurological signal is comprised of a very small pulse controlling the initial vergence dynamics, and a step controlling the final, steady-state vergence angle/position.[33,43] Thus, the pulse component would be primarily involved in this dynamic clinical test. Peak vergence velocity has been found to be consistently and considerably reduced in this population by approximately 50%,[24,33,43] thus resulting in overall slowed vergence responsivity and reflecting a primary pulse deficit.[33,43] And, the presence of increased steady-state vergence variability in the mTBI population implicates the step component.[33,43] Thus, both neural control components are likely abnormal in mTBI.

The Brock string is used clinically to assess global, steady-state vergence responsivity in free space (Figure 5), although it has many other clinical uses (e.g., to assess for binocular suppression, retinal correspondence).[44] Briefly, the patient is asked to converge upon a specified bead target on a long string centered at eye level along the midline, and describe if the specified fixation target, as well as the other bead targets (usually 2-4 more), are perceived as either single or double. The specified bead target should be perceived to be single, whereas the others should be perceived as being

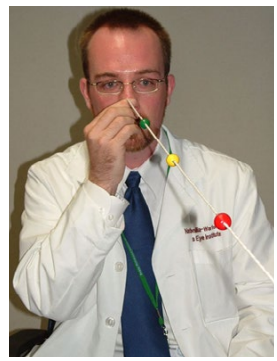

Fig. 5: Individual viewing along a Brock string at the bead target.

diplopic, which is a normal response, since the latter fall on non-corresponding retinal points. Also, the patient is asked if the specified bead target is at the perceived "intersection" of the string, which in normals perceptually forms an X-shaped pattern. This is repeated for the different bead target distances, typically 10 cm to 200 cm. In patients with mTBI, most steady-state and dynamic aspects of vergence are compromised. Thus, slowed, unstable, and inaccurate vergence responses are typically found in this population.[24,33] The neural control is as described earlier for NPC, again likely predominantly involving the tonic vergence neurons in the steady-state assessment.[33,43]

Prisms can be introduced within the near spectacle correction in a trial frame to assess for near vision "comfort", if there is a near vergence dysfunction present, such as a receded NPC or large phoria. Typically, 1-2 pd per eye is given.[1,5,6] The patient is asked if visual comfort is improved, both immediately and after wearing them for 15-30 minutes while reading. Prisms optically reduce the vergence demand that the patient must exert, and thus they typically improve visual comfort, as well as visual "stamina" and visual "efficiency". Again, regarding the neurological aspects, the tonic vergence cells are implicated, in both the normal and mTBI populations.[33,43]

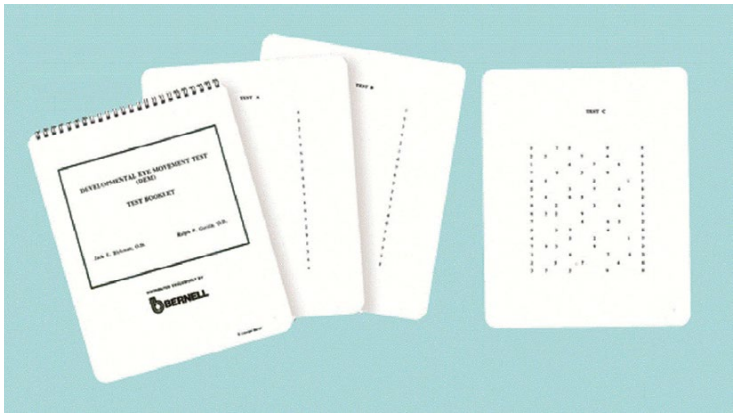

Figure 6: DEM test kit.

Both the Developmental Eye Movement (DEM) test[45] and the King-Devick (K-D)[46] test indirectly assess global saccadic tracking accuracy and directly assess global saccadic tracking time (Figure 6). However, they do not measure the individual saccadic oculomotor components (e.g., latency, peak velocity) as would be the case in the laboratory.[47] Although similar, the DEM test is likely the better of the two, as it disambiguates a saccadic tracking deficiency from a verbal naming deficiency (via the DEM ratio), which is especially important in young children. Essentially, the patient is instructed to read aloud in a given sequence two horizontally-separated columns of numbers, and then the same for several vertically-separated rows of numbers, as accurately and rapidly as possible. The number of naming errors and the total tracking time for completion are quantified and compared to age-related norms. In the patient with mTBI, the DEM results showed an increased number of saccades, increased naming errors, and increased completion time, as compared with adult normative data,[48] likely reflecting higher-level control dysfunction of saccadic programming and sequencing. The DEM test is simple, inexpensive, validated, quantitative, and easy to administer in the mTBI population. Lower-level neural control of saccades includes formulation of the pulse-step signal derived from the paramedian pontine reticular formation (PPRF), whereas higher-level control aspects (e.g., target localization in visual space) primarily involve the frontal eye fields, parietal lobes, superior colliculus, and cerebellum.[47] The latter is likely more involved than the former in this DEM task. Objective testing of eye movements, in general, would be recommended using the Right Eye and Visagraph systems.

The optokinetic nystagmus (OKN) test is used to assess relatively low-level, non-volitional, global "following" oculomotor responses.[47] Basically, a black-and-white, striped drum encompassing much of the central and near peripheral visual field is slowly rotated, with the goal being elicitation of optokinetic-based "jerk nystagmus", i.e., a smooth, slow-phase in the direction of drum rotation, and a "resetting" saccadic phase in the opposite direction, with

continued OKN stimulation. In mTBI, the OKN response may be diminished.[49] The neural control includes the inferior olive, cerebellum, vestibular nuclei, and the oculomotor nuclei, and other sites.[47]

Similar to that discussed earlier for vergence prisms, plus lenses can also be introduced, with or without the prisms, to assess for near vision "comfort", if there is a near accommodative dysfunction present such as esophoria or accommodative insufficiency.[1,5,6] Typically, +0.75 to +1.25 D lenses are added before each eye. Again, the patient is asked if the lenses improve visual comfort, both immediately and after wearing them for 15-30 minutes while reading. Plus lenses reduce the blur-driven accommodative demand that the patient must exert, and thus typically improve visual comfort, as well as visual "stamina" and "visual efficiency." We believe that these near lenses function to "balance" the accommodative and vergence steady-state control systems at near.[1,5,6] While the underlying neurophysiology remains elusive, bioengineering model-based studies suggest that the crosslink gains from accommodation to vergence, and also from vergence to accommodation, are primarily involved.[27]

The lens flipper test is used to assess dynamically accommodative facility at near (e.g., 40 cm, 2.5 D) under monocular and binocular viewing conditions with the near prescription in place. While there are four components to accommodation,[27] namely blur accommodation, vergence-accommodation, proximal accommodation, and tonic accommodation, only the first two are activated interactively during testing, along with accommodative vergence and disparity (fusional) vergence.[27] Essentially, while focusing on a 20/30 line on a near Snellen chart, a lens flipper pair typically of +/- 2D is placed in front of the near spectacles. The number of alternations executed between the two dioptric demands (e.g., 4.5D and 0.5D at 40 cm) for which one can repeatedly and rapidly refocus (and fuse when under binocular-viewing conditions) the target is recorded over a one minute period (cycles per minute, cpm), and compared with normative data. Peak accommodative velocity was found to be significantly reduced (~50%) in all mTBI subjects tested in the laboratory,[50,51] hence resulting in overall slowed dynamic accommodative responsivity. Thus, this laboratory parameter may serve as an objective biomarker for the presence of mTBI/concussion, with good specificity and sensitivity.[36] Clinically, this is reflected in a reduced cpm rate in the mTBI population,[50-52] primarily due to abnormal parasympathetic drive to accommodation. We speculate that neural control involves the midbrain burst cells related to the transient aspect of accommodation, predominantly the peak velocity aspect.

The amplitude of accommodation (AA) refers to the closest point of clear vision attained with maximum effort exerted,[50,51] with this value being age-dependent. This test incorporates all four components of accommodation when using the push-up technique, namely blur, vergence, proximal, and tonic.[27,50,51] The AA has been found to be consistently reduced in mTBI/concussion,[50,51] and as such it may serve as a good clinical biomarker for their presence. A reduced AA would typically cause near visual symptoms, such as blur and asthenopia.[50] Analogous to the midbrain vergence cells, we speculate on the same for accommodation, with the tonic cells predominantly involved in the reduced AA.[51]

The penlight test (PLT)[47] is a simple and rapid way to assess grossly both ocular motility, for example to detect a paresis due to peripheral nerve damage, and the pupillary light reflex (PLR) responsivity, for example to detect an afferent pupillary defect (APD) due to a lesion of the optic nerve. In the former, the penlight is slowly moved along the cardinal positions of gaze that underlie the primary extraocular muscle (EOM) functions to assess for full and equal (i.e., large and parallel) excursions of the two eyes. If one eye fails to respond appropriately,

the abnormal eye, direction, and magnitude are denoted, and further specialized tests are performed (e.g., Hess-Lancaster test[10]). The underlying neurology is extensive: this includes the EOM cranial nerve innervations, as well as the related nuclear and supranuclear controlling sites.[47] In the latter case, the penlight can be used to stimulate each eye separately to detect for presence of strong and normal, direct and consensual pupillary responses. Failure to obtain such responses suggests damage along the basic PLR neural pathway: this includes the photoreceptors, pretectal nucleus, oculomotor nerve, ciliary ganglion, and finally the pupillary sphincter,[17] all being under parasympathetic (for constriction) and sympathetic (for dilation) control.[17] Both the PLT and PLR have been used in the mTBI/concussion assessment. For example, the PLT has been used to detect for EOM paresis due to cranial nerve problems, which are relatively common (6.9%) in this population.[32] And, the PLR test has been used to detect for an APD in these mTBI patients, which is relatively uncommon.[17] However, objectively-based techniques (i.e., infrared pupillometry) are far superior in the diagnostic evaluation, as most of the abnormalities found are of a very subtle and dynamic nature (e.g., reduced constriction peak velocity), and thus not detectable clinically.[17]

CONCLUSION

The patient with a mTBI/concussion frequently exhibits a wide range of visual sensory and motor deficits, which can adversely affect many activities of living (ADL), and hence quality of life.[53] The visual dysfunctions can result from neurological damage within many regions of the brain, with it frequently being of a diffuse nature. It is hoped that the use of the basic visual sensory and motor tests described in this paper can assist and improve the clinical diagnosis in these patients.

REFERENCES

1. Ciuffreda KJ, Ludlam DP, Yadav NK, Thiagarajan P. Traumatic brain injury: visual consequences, diagnosis, and treatment. Adv Ophthalmol Optom. 2016;1:307-333.
2. Suchoff IB, Ciuffreda KJ, Kapoor N (eds.) Visual and Vestibular Consequences of Acquired Brain Injury. Santa Ana, CA: Optometric Extension Program Foundation. 2001.
3. Ciuffreda KJ, Ludlam DP, Kapoor N. Clinical oculomotor training in traumatic brain injury. Optom Vis Dev. 2009;40:16-23.
4. Zasler N, Katy D, Zafonte R. (eds.) Brain Injury Medicine: Principles and Practice. New York, NY: Demos Med Pub. 2007.
5. Ciuffreda KJ, Ludlam DP. Conceptual model of optometric vision care in mild traumatic brain injury. J Behav Optom. 2011;22:10-12.
6. Ciuffreda KJ, Ludlam DP, Yadav NK. Conceptual model pyramid optometric care in mild traumatic brain injury (mTBI): a perspective. Vis Dev Rehabil. 2015;1:105-108.
7. Rutner D, Kapoor N, Ciuffreda KJ, Craig S, Han ME, Suchoff IB. Occurrence of ocular disease in traumatic brain injury in a selected sample: a retrospective analysis. Brain Inj. 2006;20:1079-1086.
8. Ciuffreda KJ. Reading eye movements in patients with oculomotor disturbances. In: Ygge J, Lennerstrand G. eds. Eye Movements in Reading. New York, NY:Pergamon; 1994: 163-188.
9. Lemke S, Cockerham GC, Glynn-Milley C, Cockerham KP. Visual quality of life in veterans with blast-induced traumatic brain injury. JAMA Ophthalmol. 2013;131:1602-1609.
10. Steinman SB, Steinman BA, Garzia RP. Foundations of Binocular Vision. New York, NY: Pergamon. 2000.
11. Niemeyer JE, Paradiso MA. Contrast sensitivity, V1 neural activity, and natural vision. J Neurophysiol. 2017;117:492-508.
12. Herdman SJ, Tusa RJ, Blatt P, Suzuki A, Venuto PJ, Roberts D. Computerized dynamic visual acuity in the assessment of vestibular deficits. Amer J Otology. 1998;19:790-796.
13. Suchoff IB, Kapoor N, Ciuffreda KJ, Rutner D, Han E, Craig S. The frequency of occurrence, types, and characteristics of visual field defects in acquired brain injury. Optom. 2008:79:259-265.
14. Walsh, DV, Capo-Aponte JE, Jorgensen-Wagers K, Temme LA, Goodrich G, Sosa J, Riggs DW. Visual field dysfunctions in warfighters during different stages following blast and non-blast mTBI. Mil. Med. 2015;180:178-185.
15. Spiegel DP, Reynaud A, Ruiz T, Lague-Beauvais M, Hess R, Fariver R. First-and second-order contrast sensitivity functions reveal disrupted visual processing following mild traumatic brain injury. Vis Res. 2016:122:43-50.

16. Truong JQ, Ciuffreda KJ, Han MHE, Suchoff IB. Photosensitivity in mild traumatic brain injury (mTBI): a retrospective analysis. Brain Inj. 2014;28:1283-1287.

17. Ciuffreda KJ, Joshi NR, Truong JQ. Understanding the effects of mild traumatic brain injury on the pupillary light reflex. Concussion. 2017;2:doi.org/10.2217/cnc-2016-0029.

18. Ciuffreda KJ, Yadav NK, Ludlam DP. Binasal occlusion (BNO), visual motion sensitivity (VMS), and the visually-evoked potential (VEP) in mild traumatic brain injury and traumatic brain injury (mTBI/TBI). Brain Sci. 2017; 98:doi:10.3390/brainsci7080098.

19. Slobounov S, Slobounov MS, Newell K. Application of virtual reality graphics in assessment of concussion. Cyberpsychol Beh. 2006;2:188-191.

20. Ciuffreda KJ, Yadav NK, Ludlam DP. Effect of binasal occlusion (BNO) on the visual-evoked potential (VEP) in mild traumatic brain injury (mTBI). Brain Inj. 2013;27:41-47.

21. Patel R, Ciuffreda KJ, Tannen B, Kapoor N. Elevated coherent motion thresholds in mild traumatic brain injury. Optom. 2011: 82:284-289.

22. Chang TT, Ciuffreda KJ, Kapoor N. Critical flicker frequency and related symptoms in mild traumatic brain injury. Brain Inj. 2007; 21:1055-1062.

23. Schrupp LE, Ciuffreda KJ, Kapoor N. Foveal versus eccentric retinal critical flicker frequency in mild traumatic brain injury. Optom. 2009;80:642-650.

24. Szymanowicz D, Ciuffreda KJ, Thiagarajan P, Ludlam DP, Green W, Kapoor N. Vergence in mild traumatic brain injury: a pilot study. J Rehabil Res Dev. 2012;49;1083-1100.

25. Schmidtmann G, Ruiz T, Reynaud A, Spiegel DP, Lague-Beuavais M, Hess R, Farivar R. Sensitivity to binocular disparity is reduced in mild traumatic brain injury. Invest Ophthalmol Vis Sci. 2017;58: 2630-2635.

26. Blake R, Wilson H. Binocular vision. Vis Res. 2011;51:754-770.

27. Ciuffreda KJ. Components of clinical near vergence testing. J Behav Optom.1992;3:3-13.

28. Ciuffreda KJ, Ludlam DP. Egocentric localization: normal and abnormal aspects. In: Suter PS, Harvey LH, eds. Vision Rehabilitation. New York, NY: CRC Press, NY; 2011:193-211.

29. Kapoor N, Ciuffreda KJ, Harris G, Suchoff IB, Kim J. Huang M, Bae P. A new portable clinical device for measuring egocentric localization. J Behav Optom. 2001;12:115-119.

30. Karnath HO. Subjective orientation in neglect and the interactive contribution of neck proprioception and vestibular stimulation. Brain. 1994;117:1001-1012.

31. Ciuffreda KJ. Near point of convergence as a function of target accommodative demand. Opt J Rev Optom. 1974;111:9-10.

32. Ciuffreda KJ, Kapoor N, Rutner D, Suchoff IB, Han ME, Craig S. Occurrence of oculomotor dysfunctions in acquired brain injury: a retrospective analysis. Optom. 2007;78:155-161.

33. Thiagarajan P, Ciuffreda KJ. Effect of oculomotor rehabilitation on vergence responsivity in mild traumatic brain injury. J Rehabil Res Dev. 2013;50:1223-1240.

34. Master CL, Scheiman M, Gallaway M, Goodman, A, Robinson RL, Master SR, Grady MF. Vison diagnoses are common after concussion in adolescents. Clin Pediatr (Phila). 2016;55:260-267.

35. Ciuffreda KJ, Ludlam DP, Thiagarajan P. Oculomotor diagnostic protocol for the mTBI population. Optom. 2011; doi:10.1016/j.optm.2010.11.011.

36. Ciuffreda KJ, Ludlam DP, Thiagarajan P, Yadav NK, Capo-Aponte J. Proposed objective visual system biomarkers for mild traumatic brain injury. Mil Med. 2014;179:1212-1217.

37. Tannen B, Good K, Ciuffreda KJ, Moore KJ. Prevalence of esophoria in concussed patients. J Optom. 2018;doi.org/10/1016/j.optom.2018.02.003.

38. Rosner MS, Feinberg DL, Doble JE, Rosner AJ. Treatment of vertical heterophoria ameliorates persistent post-concussive symptoms: a retrospective analysis utilizing a multi-faceted assessment battery. Brain Inj. 2016;30:311-317.

39. Evans BJW. Optometric prescribing for decompensated heterophoria. Optom in Practice. 2008;9:63-78.

40. Tannen N, Rogers J, Ciuffreda KJ, Lyon E, Shelley-Tremblay J. Distance horizontal fusional facility: a proposed new test for concussion patients. Vis Dev Rehabil. 2016;2:170-175.

41. Scheiman M, Gallaway M. Vision therapy to treat binocular vision disorders after acquired brain injury. In: Suchoff IB, Ciuffreda KJ, Kapoor N. eds. Vision and Vestibular Consequences of Acquired Brain Injury. Santa Ana, CA: Optometric Extension Program Foundation; 2001:89-113.

42. Thiagarajan P, Ciuffreda KJ. Visual fatigue effects on vergence dynamics in asymptomatic individuals. Ophthal Physiol Opt. 2013;33:642-651.

43. Ciuffreda KJ, Ludlam DP, Yadav NK. Convergence peak velocity: an objective, oculomotor-based biomarker for mild traumatic brain injury (mTBI)/concussion. Vis Dev Rehabil. 2018;4:6-11.

44. Ciuffreda KJ, Ludlam DP, Tannen B, Yadav NK. Brock string and the horopter: a perspective. Vis Dev Rehabil. 2016;2:208-210.

45. Garzia RP, Richman JE, Nicholson SB, Gaines CS. A new visual-verbal saccade test: the developmental eye movement test (DEM). J Am Optom Assoc.1990;61:124-135.

46. http://kingdevicktest.com

47. Ciuffreda KJ, Tannen B. Eye Movement Basics for the Clinician. St Louis, MO: Mosby. 1995.

48. Kapoor NK, Ciuffreda KJ. Assessment of neuro-optometric rehabilitation using the Developmental Eye Movement (DEM) test in adults with acquired brain injury. J Optom. 2018;11:103-112.

49. Ron S. Can training be transferred from one oculomotor system to the other? In: Roucoux A, Crommelinck. M. eds. Physiological and Pathological Aspects of Eye Movements. London: Dr. W. Junk Pub; 1982:83-88.

50. Green W, Ciuffreda KJ, Thiagarajan P, Szymanowicz D, Ludlam DP, Kapoor N. Accommodation in mild traumatic brain injury. J Rehabil Res Dev. 2010; 47:183-199.

51. Thiagarajan P, Ciuffreda KJ. Effect of oculomotor rehabilitation on accommodative responsivity in mild traumatic brain injury. J Rehabil Res Dev. 2014;51:175-191.

52. D'Angelo ML, Tannen B. The optometric care of vision problems after concussion: a clinical guide. Optom Vis Perf. 2015; 3:298-306.

53. Sanet RB, Press LJ. Spatial Vision. In: Suter PS, Harvey, eds. Vision Rehabilitation. New York, NY: CRC Press, NY;2011:77-151.

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