CHAPTER

15

Vision Disorders in Mild Traumatic Brain Injury

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OVERVIEW OF CONCUSSION AND VISUAL FUNCTION

Conservatively, it is estimated that at least 40% of the primate brain is primarily visual machinery.[1–5] Recognizing that accommodation, vergences, saccades, orbital sensation, eyelid function, visual fields/acuity, color vision, and pupillary function are subserved by 7 of the 12 cranial nerves, the importance of a detailed visuo-oculomotor examination in mTBI cases becomes apparent. It is therefore prudent to explore visual function carefully in mild traumatic brain injury (mTBI) beyond the ostensible reassurance offered when a patient sees with 20/20 acuity bilaterally. It is entirely appropriate to rule out pathology that might cause gross visual field/acuity and/or visuomotor defects, since these deficits are common in moderate to severe brain injury in which patients suffer loss of consciousness.[6] However, after mTBI, deficits in visual processing as reflected in abnormal eye teaming rather than those from direct injury to the afferent visual pathways or oculomotor nerves are far more common.[7–15] A difficulty that faces examiners in the emergency setting is the often subtle presentation of eye teaming issues, despite the fact they can cause significant symptomatology.[11,16] In addition, patients might not demonstrate certain deficits immediately after an injury due the later onset of inflammatory changes and cerebral perfusion deficits associated with concussion.[17–19] Patients themselves might not even

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notice certain visual deficits until they try to return to their normal routine with visual-intensive tasks such as reading, computer use and driving.

Visual symptoms commonly reported after mTBI include fluctuations in visual acuity at near, headaches with visual-intensive tasks and photophobia.[7,20,21] The prevalence of visual issues in mTBI patients between ages 11 and 17 has been estimated at 69% with overlapping issues including accommodative dysfunction (51%), convergence insufficiency (CI, 49%), and saccadic dysfunction (29%) being reported. Furthermore, 14% of patients were found to have significant dysfunction in all three domains.[22] Dual sensory damage such as vestibulo-ocular dysfunction in children after sport-related injuries was reported to be 63%.[23] Given that persistent (6 months or more after injury) visual symptoms are not uncommon in mTBI patients,[24–27] it is logical to suggest that we need to investigate the oculomotor system of patients with mTBI in more detail than what might be normally offered during a routine eye exam. This is even more vital if we consider that mTBI patients have approximately a 3× higher suicide rate compared to the general population and that more than half saw their primary care physician less than a week before committing suicide.[28] This research speaks to the fact that these patients are reaching out to seek care, but are often underdiagnosed in terms of their underlying functional difficulties.[11]

It is appropriate, then, to refrain from assuming a normal visual status in the absence of appropriate visual testing. Areas vital to assess in mTBI patients include both subjective complaints and objective tests of visual function. Patients will often have difficulty verbalizing their complaints, not only because of cognitive difficulties such as impaired word-finding, memory, and complex attention,[12,29–31] but also because there are only vague terms available to the layperson for the subtle visual problems, such as *eyestrain*. The examiner must ask specific questions and may need to ask them in more than one way; a validated questionnaire is preferable.[32–34] Concerning the physical exam, saccadic testing, vergence amplitude/facility testing, accommodative amplitude/facility testing, and fixation disparity (FD) assessment [i.e., associated heterophoria (AH) testing] are all crucial.[35,36] If such tests are not performed, the assumption of normality might not only be incorrect, but can result in limited gains in other rehabilitative areas, such as vestibular rehabilitation therapy or physiotherapy. This is because patients with visual concerns after mTBI are less sure of their own positioning in space[37] and require correct visual cues to promote better balance.[38]

Once a patient has been shown to exhibit visual processing difficulties, therapy should be initiated in concert with a patient's other providers. As was so elegantly stated, "it takes a village and it begins with each of us."[39] There is a growing body of evidence demonstrating the efficacy of visual exercises for patients with visual concerns that might be seen after mTBI.[14,40–43] The Convergence Insufficiency Treatment Trial,[44,45] for example, proved that in-office therapy with home exercises can effectively resolve CI, albeit in children with no history of mTBI. Sports vision training has been shown to improve stereopsis in athletes,[46] visual search performance in students,[47] and even reduce concussion incidence when used on a team-wide basis;[48,49] these programs are not only likely translatable to mTBI patients, but are also germane, since concussion itself is a risk factor for a second concussion.[50,51] Other studies aimed specifically at mTBI patients have documented improvements in reading, accommodative responsivity,[52] and vergence facility.[53–57]

It should be noted that visuomotor training exercises are very similar to the visual tasks that produce symptoms in patients. Therefore, patients must be apprised with the caveat that visuomotor rehabilitation, like any other form of physical therapy or exercise program, is a process that starts gently, requires adequate engagement of the patient in terms of office visits and homework, and takes time.[58]

SPECIFIC OCULOMOTOR AND VISUAL PROCESSING DIFFICULTIES

Saccadic Dysfunction

Accurate saccadic eye movements underpin the ability to see in a moving environment and are crucial components of reading.[36] It is well-documented that difficulties with driving and reading, two tasks dependent upon saccades, are common complaints for patients suffering mTBI; there is often corresponding saccadic dysfunction demonstrable in these patients including hypometric saccades and reduced ability to inhibit saccades.[21,59–67] Excessive eye movements during reading have been linked to poorer reading skills and poor overall oculomotor function.[36] Notably, patients with reduced oculomotor function are far more likely to reveal a history of concussion.[35] A basic saccadic eye movement is a rapid refixation from one point to another.[68] In order to initiate a saccade, the visual system must not only release fixation from the point of regard, but also preplan where the next fixation will take hold, a process which ultimately requires peripheral awareness. Patients with mTBI demonstrate difficulties with both releasing and capturing objects of regard.[67,69–71] The preplanning phase is dependent upon the interaction between brain regions serving the bimodal vision processes, that is, *focal* (detailed information related to the macular cone-parvocellular ganglion cell pathways) and *ambient* [spatial awareness of the periphery (motion and luminance) related to the rod-magnocellular ganglion cell pathways].[69,72,73] To put this in terms that are likely too simplistic, focal vision is *where* one is looking, whereas ambient vision is the visual area *around* where one is looking. These vision processes appear to be subserved through ventral and dorsal anatomic streams in the brain.[74] In addition, both components ultimately integrate to achieve accurate saccadic performance.[68]

The neurophysiology underpinning oculomotor dysfunction after mTBI has only recently been explored. Studies employing magnetoencephalography (MEG)[75–77] and functional magnetic resonance imaging (fMRI)[78,79] have provided data suggesting aberrant functional connectivities in brains of patients with mTBI, both at rest and during visual tasks. In line with this approach, it has been proposed that mTBI leads to a decoupling of the ambient and focal visual pathways,[80] with subsequent dysfunction of the oculomotor system. Notably, subsequent studies employing diffusion tensor imaging (DTI) and fMRI suggest that human brain regions subserving the generation of saccades (right frontal eye fields, supplementary eye fields, and dorsal striatum) and their inhibition (dorsal striatum, right supplementary eye field, and right inferior frontal cortex) are not identical and even partly dissociable.[81] DTI studies have certainly demonstrated heretofore unknown structural damage after mTBI.[82,83] Therefore, it seems reasonable to suggest that this modality

might serve to clarify the neurologic basis of saccadic dysfunction (and other visuomotor defects) seen in patients.

Screening for saccades can be performed in the office or the field by having a patient look quickly from central gaze to a peripheral target and also by passively rotating the patient's head rapidly to stimulate the vestibulo-ocular reflex (VOR); an excellent visual demonstration of this is provided in a video[84] attached to the review by Ventura et al.[67] Notably, more sophisticated tests of saccadic function such as the King-Devick and Developmental Eye Movement (DEM/DEM-A) tests are becoming standard equipment for providers.[70,85–87] Although clinic-based devices to measure ocular motility during saccades have been available for some time, it should be noted that newer portable devices that passively measure saccades, such as the Saccadometer™, have been introduced[88]; preliminary studies suggest this device might be useful to monitor patients in the field.[89] The available tests for evaluating saccades have age-matched normative data allowing a percentile score to be obtained post-mTBI. Ideally these tests would be offered to those at risk for concussion prior to play/deployment so that premorbid baseline data would be available for each individual where possible.[87] Data culled predeployment would not only be helpful in improving diagnosis of individuals but also in monitoring the progress of these patients through their rehabilitation/deployment.

The pattern of the saccadic dysfunction is important, with horizontal saccadic dysfunction being more suggestive of oculomotor impairment (DEM Type II result),[90] whereas reduction of both horizontal and vertical saccades is more suggestive of a generalized rapid automated naming (RAN) deficit.[90] A patient with a RAN deficit may have other concurrent nonoculomotor concerns such as reduced visual memory,[91] which has been linked to lower reading fluency rates.[92] While reduced saccadic performance has been studied in children with reading difficulties primarily employing DEM testing,[93] a similar evaluation of saccadic function (King-Devick) has been used in the context of concussion. In this case, it was demonstrated that horizontal saccades were impaired and this correlated with deficits in immediate memory recall.[65]

Our understanding of impaired visual processing leading to saccadic dysfunction in patients with mTBI may be supplemented by studies on patients with attention deficit/hyperactivity disorder (ADHD), since abnormal visual processing seems to underlie this disorder.[94–97] Notably, there is a growing body of evidence relating mTBI and ADHD; it is not uncommon for ADHD to develop in patients with mTBI,[98,99] with one study citing a hazard ratio of 1.32.[100] Additionally, a premorbid diagnosis of ADHD leads to poorer recovery in patients with mTBI.[101] Furthermore, a premorbid diagnosis of ADHD may be a risk factor to incur concussion.[102,103] Finally, patients with ADHD demonstrate saccadic dysfunction in ways qualitatively similar to patients with mTBI.[104–106]

Pursuit Dysfunction

A pursuit is a smooth, constant velocity eye movement from one point to another (as opposed to a saccade which is a ballistic jump). Smooth pursuits can be measured in the laboratory or clinic setting using electro-oculography,[107] although novel technologies that are portable, fieldable and relatively inexpensive have also been explored.[108,109] The upper

limit of smooth pursuit velocity for normal humans is over 100°/sec,[110] although this rate declines with aging.[111] Pursuit disruption within the context of concussion has been examined in the past decade,[112–115] where reports suggest that patients with TBI demonstrated decreased target prediction, reduced tracking speed, and less accurate tracking. Smooth pursuits, like other oculomotor functions, are subserved by networks spanning much of the brain, including cortical (V5, frontal, and supplementary eye fields) and subcortical (basal ganglia, thalamus, and cerebellum) structures.[116] A subsequent fMRI study of athletes with acute/subacute concussion showed abnormal activity during tests of smooth pursuits, but did not specifically highlight any one area of the brain,[117] consistent with the idea that concussion causes a more global disruption of networks. MEG has been employed to study patients with concussion-related pursuit dysfunction.[76] These patients appeared to have normal function during visual pursuits until the object of regard was transiently blocked, after which the patients then showed deficits in resynchronizing their gaze when the object reappeared. Specifically, there was abnormal suppression of beta activity in the right parietal cortex and abnormally elevated activity in the left caudate and fronto-temporal cortex. This study highlights the important role that anticipatory control plays in oculomotor function. At present, there are no published studies employing DTI to explore dysfunction of pursuits in patients with mTBI. Notably, DTI has been employed to evaluate parkinsonian patients with disturbed smooth pursuits, and abnormalities in the middle cerebral peduncle (but not in cortical or subcortical white matter) were identified.[118]

Smooth eye movements not only permit tracking a target, but they also permit maintaining a fixed gaze upon a target when that target is stationary while the head is in motion, that is, the VOR, an integration of the oculomotor and vestibular systems. Patients with abnormal VOR screening demonstrate delayed recovery after concussion.[119] Furthermore, primates with lesions to the peripheral vestibular organs demonstrate poorer recovery if they also underwent lesioning of the primary visual cortex (V1).[120,121] Conversely, VOR recovery in monkeys post-labyrinthectomy is significantly better when the animal is in a more brightly lit environment.[121] DTI has demonstrated that the vestibular circuitry runs the length of the brain from brainstem to cortex, with multiple ladder-like crossings in the brainstem and corpus callosum.[122] While patients with mTBI and vestibular symptoms after blunt injury demonstrated abnormal findings in the fusiform gyri and cerebellum on DTI,[123] patients with blast injury and vestibular concerns showed more diffuse axonal injury,[124] highlighting the concern that the mechanism of injury causing mTBI must be a consideration.[125,126] That being said, it must be noted that visuomotor concerns and outcomes appear to be quite similar for patients with mTBI regardless of whether there was a history of blast exposure.[114,127] Concerning other novel imaging modalities, there is a dearth of published studies employing fMRI or MEG to evaluate patients with vestibulo-ocular dysfunction after mTBI.

It is interesting to note that there is significant overlap in the networks subserving saccades and smooth pursuits (e.g., dorsolateral prefrontal cortex, frontal eye fields, and posterior parietal cortex).[128] Despite this, it appears that visual smooth pursuits are not abnormal in patients with ADHD[129–131] in the way they are abnormal in patients with mTBI. If this finding proves to be consistent, one could hypothesize employing it to help differentiate patients with mTBI, ADHD and/or other causes of abnormal visual processing.

Vergence Dysfunction

Vergence function refers to how well the eyes team; this can be described in terms of convergence and divergence amplitude, vergence facility (converging and diverging in sequence) and vertical/torsional vergence ranges. Although, convergence and stereopsis have been shown to be present starting approximately 3–6 months of age,[132] peak normal vergence develops by age 4–5 years[133,134] and, in the absence of injury or disease, should remain stable until the 5th decade of life.[134,135] Isolated measurements of vergence can be accomplished employing graded prisms through which a subject looks from fixed distances; measuring vergence while changing the target distance simultaneously challenges the accommodation system and, therefore, should be avoided if only vergence measurements are sought. Vergence deficits (of amplitude and/or facility) have been shown to be quite common in mTBI,[20,35,136] with some forms of vergence deficit being reported in approximately 45% of adolescent concussions.[22] CI is the inability to move both eyes inwards without undue strain.[137] As with saccadic dysfunction, there is evidence connecting CI and ADHD. First, there is significant symptomatic overlap between the CI and ADHD (i.e., five out of the nine DSM-IV criteria are shared, namely symptoms 1, 2, 4, 6, and 8).[137] In addition, it has been reported that the incidence of CI in the general population is 1.8%–3.3% compared to 15.9% in the ADHD population,[137] although a later study did not support this finding.[138] Finally, children with CI who did not carry an official diagnosis of ADHD scored significantly higher on parental ratings of behavior consistent with ADHD.[139] Academic difficulties are common in children with visual complaints after concussion.[13] Since interventions for CI have been shown to result in academic gains post-mTBI,[140] it could be important to monitor particularly the trajectory of improvement in those mTBI patients who also suffer ADHD. Furthermore, since reduced vergence facility has also been linked to reading inefficiency[36] and training to improve vergence facility has been shown to improve saccadic function,[141] it appears reasonable to inquire about both reading and attentional problems in patients with mTBI.

The midbrain seems to be a key relay station of the pathways subserving vergences via networks to the cerebellum and pons,[142] area V1 (primary visual cortex),[143] middle superior temporal cortex,[144] and frontal eye fields.[142] It is likely that the diffuse axonal injury caused by concussion will affect multiple nodes along this extensive network[145] making the exact location of damage in patients with CI more difficult to pinpoint. One study employing DTI on concussed patients demonstrated abnormal fractional anisotropy values in the right anterior thalamic radiation and the right lateral geniculate nucleus.[123] Serendipitously, the diffuse nature of the vergence network (and therefore its susceptibility to injury) suggests that vergence dysfunction may prove to be a relatively sensitive marker for mTBI.

Accommodative Dysfunction

Accommodative function can be separated into amplitude and facility metrics. Accommodative amplitude can be defined as the monocular ability to sustain a clear image on the retina during fixation at a near target (closer than 20 m). Accommodative facility is the monocular ability to smoothly change focus quickly without undue strain or

delay[36] as fixation targets approach or recede. Accommodative amplitude is measured in diopters [D] [i.e., the inverse of the distance (in meters) from viewer to target] while accommodative facility is measured in cycles per minute (cpm), usually with a ±2D lens monocularly.[36] While the neural network subserving accommodation spans the length of the human brain,[146] the endpoint is mediated via the third cranial nerve through innervation to the ciliary body of the eye, that is, a ring of muscle that controls the thickness of the crystalline lens. Accommodation naturally starts to decrease after age 40 years, a condition called presbyopia. Presbyopia is likely due to the hardening of the crystalline lens.[147–149]

Patients with mTBI commonly demonstrate a degradation of accommodation amplitude and facility; estimated prevalence rates range between 20% and 50% of patients.[20,22,52,61] These deficits might not only include reduced ability to focus at near but also a spasm of the near response.[15,20,22,59,60,67,86,150–153] Subsequently, vision-based rehabilitative strategies for accommodative dysfunction have been shown to be effective both in increasing accommodative function and improving objective VEP data in a small population of patients suffering concussion.[52,154] Positive results of vision-based rehabilitation from larger scale prospective randomized clinical trials studies on pediatric patients with accommodation dysfunction but without a history of mTBI (designed in a manner similar to the CITT study[155]) have also been reported.[156] It may be that mTBI somehow exacerbates the clinical significance of latent hyperopia, although there appear to be no published reports evaluating the prevalence of latent hyperopia in patients with accommodative dysfunction after mTBI. One published report evaluating children (without a history of mTBI) suggests that hyperopia as low as +1.50D in the presence of accommodative dysfunction can impair reading fluency, as assessed by objective infra-red eye-tracking devices.[36] In addition, patients with significant hypermetropia (>+ 4D) more commonly demonstrate accommodation lag.[157]

Accommodative facility can and should be tested monocularly using a ±2D flipper lens; testing binocularly will stimulate both accommodation and vergence and should be avoided if only accommodation facility is sought. Although the average normal monocular accommodative facility is approximately 11 cpm,[158,159] the range is wide, approximately ±6 cpm.[158] Accommodative amplitude is measured in diopters by first calculating the inverse of the distance of the near point for the emmetropized eye; this can then be compared to the age-adjusted normal amplitude of accommodation calculated with Hofstetter's formula (i.e., minimum monocular accommodative amplitude = 15D−0.25 × age).[160] As with vergence and saccadic dysfunction, accommodative abnormalities seem to have a link to ADHD; studies have shown that as little as 2D of excessive accommodative strain can induce symptoms akin to ADHD on the Connor's Rating Scale (CRS).[151] Given that accommodative dysfunction is relatively common in mTBI, it stands to reason that patients with any attention issues arising post-mTBI should be evaluated for accommodative dysfunction prior to diagnosing concomitant ADHD.

Fixation Disparity and Impaired Stereopsis

Recognizing that saccades, pursuits, vergences, and accommodation can be disrupted in mTBI, it is not surprising that visual fusion suffers as well.[161] In this context, deficits in

visual fusion refer to FD slips, or AH.[162] FD slips represent a small misalignment of the visual axes of either eye under binocularly fused conditions such that there is a lack of bifoveal fixation, but maintenance of normal retinal correspondence. This can occur because the disparity is still within Panum's area of fusion, which is approximately 10′ arc at fixation in normal subjects.[163] This small heterophoria, as low as 0.5PD, is too subtle to be measured by those tests use for dissociated heterophoria (DH), such as the cover-uncover test or Maddox-rod test. Whereas AH is a deviation from orthophoria that occurs when fusional contours are absent only from the central visual field, DH is a deviation that occurs when neither central nor peripheral fusional contours are offered,[164] such as during cover-uncover- or Maddox-rod testing. AH has been shown to be a much better indicator of symptomatology compared to DH[165] as the alignment of the visual axis under binocular conditions is more relevant to the habitual oculomotor status. In addition, AH is more useful in determining who might be uncomfortable using 3D-viewing technology.[166] It has been reported that patients with mTBI often demonstrate vertical heterophorias and that correction with prism aids in reducing symptomatology.[167,168] Devices to measure AH, that is, the minimum prismatic correction required to attain alignment recorded in arc minutes or prism diopters, include the Mallett unit, Sheedy Disparometer, and Wesson Fixation Disparity card.[163,169] These tests offer suppression checks, that is, polarized filters over either eye so that different targets can be viewed binocularly, allowing the examiner to determine whether patients are avoiding diplopia through suppression of one visual field, that is, becoming functionally monocular.

Another aspect of visual fusion is stereopsis (depth perception), which can be described as local or global in nature. Stereopsis is measured by standardized tests (e.g., see Refs.[170,171]) and reported in minutes/seconds of arc; normal values have been published.[172] Global stereoscopic targets (i.e., randot displays), which require a larger visual area in order to be seen, can better reveal symptomatology compared to local stereoscopic targets.[173] Notably, stereopsis normally declines as the visual target moves from the fovea to the retinal periphery.[174] In addition, perception of depth appears to require both retinal and extra-retinal inputs during motion of the scene or the observer. Finally, it is noteworthy that studies employing fMRI suggest that depth perception appears to be subserved by the dorsal visual stream in normal subjects, in particular to visual cortical areas V3A, V7, and MT +/V5.[175]

Patients with mTBI demonstrate reduced stereopsis at near[176] and, to a lesser degree, at distance.[177] However, these impairments seem insufficient to explain their relatively common complaint of reduced depth perception. Furthermore, patients suffering concussion report decreased tolerance to aniseikonia (different retinal image sizes due to differing refractive errors between the two eyes)[178] which may play some role in their reduced ability fuse. Although the primary visual cortex (V1) of adult patients suffering from blast-related mTBI demonstrate abnormalities with fMRI,[179] there are no published reports specifically exploring the changes in integrity or connectivity of the ventral and dorsal visual pathways.

Anecdotally, patients with mTBI frequently report intolerance to viewing 3D movies (personal experience, personal communications). Even in normal individuals, observing 3D movies is known to elicit symptoms of imbalance, headache, eyestrain, and motion sickness.[180–182] In addition, these symptoms tend to be more common and more severe in

individuals with a history of susceptibility to motion sickness or migraine.[182] Although there are no studies of the responses of mTBI patients to watching 3D movies, it is likely they would fall into the category of susceptible patients. However, it is unclear whether their reduced stereopsis would play a role in that susceptibility; a large study of normal patients who reported untoward effects of watching 3D movies indicated that there was no correlation between degree of stereopsis and symptoms.[181]

Reduced stereoacuity also seems to a problem shared by patients with mTBI and those with ADHD,[183,184] further supporting the idea that mTBI is associated with abnormal vision information processing. While further research is certainly needed, it seems reasonable to hypothesize that reduced depth perception and binocular fusion may result from impaired integration of central (ventral) and peripheral visual (dorsal) information.

Deficits in Visual Information Processing

Visual information processing (VIP) is multifaceted and includes, among other things (*vide infra*) the ability to explore complex visual stimuli in which there are many equally salient targets of fixation (e.g., crowds of people). VIP appears to depend upon intact central-peripheral (i.e., ventral-dorsal visual stream) integration ability.[185] In addition, the activation of recruited neuronal areas requires a concomitant upregulation of cerebral perfusion pressure.[186] While there are no published reports evaluating the integrity of the dorsal-ventral visual networks after mTBI, it is known that patients with mTBI show measurable deficits with complex visual processing.[187–190] Notably, patients with ADHD also demonstrate abnormal cortical processing of salient visual information when examined with fMRI.[191,192] One could suggest a utility to employing fMRI in the evaluation of patients with mTBI as they scan targets of varying complexity, since they frequently voice complaints of being visually overwhelmed in environments such as a shopping mall.[193]

At present, the substrate for deficits in VIP after mTBI is unknown. There is a growing body of literature indicating that mTBI is associated with abnormal autonomic control of cortical perfusion, even long after the original injury.[194–197] However, it is unknown whether the reduced cerebral perfusion limits VIP or is a consequence of reduced recruitment by impaired visual networks. Concerning those visual processing networks, the dorsal stream is supported by fewer retinal ganglion cells than the ventral one; of approximately 1.2 million retinal ganglion cells, 200,000 are M-cells (motion and luminance detection) with most of the remaining 1 million retinal ganglion cells being P-cells (object identification).[198] The ratio of P-cells to M-cells by eccentricity changes from 15:1 at the fovea to 5:1 at 15 degrees eccentricity[198] reflecting the shift of increased magnocellular-type processing with increasing eccentricity.[199] One could hypothesize that the smaller size of the dorsal pathway is inherently less redundant and, therefore, more susceptible to damage after mTBI. Notably, M-cells also have a higher contrast/gain ratio (aside from increased sensitivity to motion) compared to P-cells.[200] This could support a hypothesis suggesting that defects in the M-cell mediated dorsal visual stream after mTBI contribute to the common complaint of photophobia.[201]

Another aspect of VIP is the figure-ground segregation ability, or the ability to recognize a salient visual target (i.e., figure) buried in a visually noisy background (i.e.,

ground).[202] An example would be a patient shopping in an aisle for a specific item. This visual task is arguably the crux of many other visual functions. For example, physiological diplopia (normal double vision from objects located outside the horopter) is a known cue for vergence and depends upon a subject's ability to discern "foreground" from "background" in order to ultimately control eye movements.[202] Given that saccades have been shown to be impaired after concussion and that figure-ground segregation skills are intimately associated with saccades,[203–206] it stands to reason that figure-ground segregation would be reduced after brain injury. Indeed, stroke patients demonstrate loss of figure-ground segregation and it has been suggested that loss of such skills were rooted in oculomotor dysfunction.[207] There are scattered case reports of patients with closed head injury demonstrating abnormal figure-ground perception,[208] but this aspect of VIP has not been systematically studied in patients with mTBI. It is worth mentioning that lesioning cortical area MT in primates (which has major inputs from the dorsal magnocellular stream) causes a persistent deficit in the ability to identify motion against a complex background.[209] This not only reinforces the importance of the M-system in figure-ground processing, but could support the notion that damage to the dorsal visual stream after mTBI contribute to patients' complaints of discomfiture with moving stimuli.[140]

Disturbance of the figure-ground segregation visual process has been observed in a recently described disorder dubbed "visual midline shift syndrome" (VMSS), in that the patient observes motion or tilting of a surface even in the absence of head or body motion (i.e., independent of vestibular involvement).[80,210] VMSS has been reported in patients with stroke,[211] TBI,[210] and other neurologic conditions[212] as a shift in the patient's sense of bodily midline, or egocenter, such that patients tend to bear weight nonorthogonally to the ground. Notably, it has been reported that patients suffering stroke (CVA)[213] and mTBI[214,215] demonstrate altered postural control, supporting the concept that sensorimotor integration processes for stance and balance are impaired. Stroke patients also demonstrate uneven weight-bearing during ambulation on pressure-sensitive treadmills and respond positively to yoked prisms that revert the visual midline to their body center.[212] VMSS is currently not a widely accepted nosologic entity outside the neuro-optometric community, although there is a case report in the sports medicine literature describing how prismatic correction of VMSS improved an athlete's posture and reduced his lower back pain.[216] Confirmation of the validity and etiology of VMSS is extremely important because this condition may be a significant cause of imbalance and falls after mTBI, independent of mTBI-related pathology to the vestibulo-ocular system.[217] As mentioned previously, the VOR stabilizes the visual scene perceived by a moving patient. Simultaneously, kinesthetic input from the vestibulospinal- and vestibulocollic reflexes steady the trunk and head, respectively.[217] If VMSS is a distinct vision processing disorder, one could hypothesize that it might impede physical rehabilitation directed toward balance disorders or even encourage maladaptive visual fixation techniques that can occur after brain injury.[218]

Clinically, the vestibular system can only be indirectly assessed via the visual system (e.g., saccadic eye movements and nystagmus).[219] Therefore, any oculomotor dysfunction can result in ambiguity as to the cause of abnormal eye movements demonstrated during VOR assessment. This supports the need for evaluating the oculomotor system prior to rendering a definitive diagnosis of vestibular dysfunction for patients with complaints often conflated by patients such as dizziness, vertigo, lightheadedness, imbalance, and

disequilibrium; notably, true vertigo is usually caused by vestibular pathology.[220] Returning to the concept that "it takes a village" to evaluate and treat patients with mTBI, the oculomotor evaluation must be done in the context of a larger, more comprehensive assessment, including explorations of balance, vestibular-cognitive, and neurocognitive function. This suite of tests, called the vestibular-ocular-motor screening (VOMS) approach, appears to be validly sensitive in detecting concussion.[23,221–225] In addition, it has been reported that results of the VOMS may help predict outcomes after mTBI,[225] although conflicting data has been reported in this regard.[114,225]

THE GENERALIST'S APPROACH TO EVALUATING VISUAL DYSFUNCTION IN PATIENTS WITH MILD TRAUMATIC BRAIN INJURY

The subjective visual complaints expressed by patients with mTBI are legion. To guide a patient through this process and facilitate the provider's effort to document these complaints, it seems appropriate to offer a questionnaire for oculomotor dysfunction. One such instrument is the Convergence Insufficiency Symptom Survey (CISS).[226] Although this survey was designed to identify CI, it has also been reported to measure symptomatology in other oculomotor disorders such as accommodative dysfunction.[227] Indeed, this instrument may be more sensitive to global visual dysfunction rather than being specific to CI.[228,229] Given that patients with mTBI who complete vision rehabilitation show marked improvement in CISS scores,[40] it seems reasonable to offer the CISS to these patients early in the diagnostic process so as to proactively guide referrals. The CISS questionnaire is brief, being comprised of 15 questions with each answer being weighted from 0 to 4 (never = 0, infrequently = 1, sometimes = 2, fairly often = 3, always = 4) and scores ranging from 0 to 60; a score over 20 in adults should raise concerns. Notably, other vision-based surveys have been reported and deserve further efforts toward validation.[33,230]

A particular difficulty encountered when recording visual dysfunction after mTBI is that the list of ICD-10™[231] codes lags behind the science. For example, there are no codes for some of the proposed nosologic entities, such as VMSS or even confirmed ones such as indirect traumatic optic neuropathy[232] or central visual processing disorder. Notably, while there are assigned codes for both central and acquired *auditory* processing disorders, there are no codes for central *vestibular* processing disorder. Table 15.1 provides a list of diagnostic codes for conditions known to occur after TBI.

CONCLUSION

Visual dysfunction after mTBI is pervasive and long-lasting, albeit often amenable to treatment. It is the responsibility of the medical community to educate providers, offering better means of detection, and avenues of therapy. It is time to recognize that the term "mild TBI" is oxymoronic and a misnomer,[233,234] considering the fact that the impact on quality of life can be pervasive and chronic.

TABLE 15.1 Diagnoses of Visual Dysfunction After TBI

Diagnosis	ICD-10 Code
Strabismus (eso-, exo-, hyper-, hypo-, cyclo-tropias)	H50.xx
Monofixation syndrome	H50.42
Spasm of conjugate gaze	H51.0
Convergence insufficiency	H51.11
Convergence excess	H51.12
Other specified disorders of binocular movement	H51.8
Paresis of accommodation	H52.52
Spasm of accommodation	H52.53
Diplopia	H53.2
Anomalous retinal correspondence	H53.31
Fusion with defective stereopsis	H53.32
Simultaneous visual perception without fusion	H53.33
Suppression of binocular vision	H53.34
Visual field defects	H53.4x
Color vision deficiencies	H53.5x
Glare sensitivity	H53.71
Heterophoria (unspecified)	H55.50
Nystagmus	H55.50
Saccadic eye movements (deficiency)	H55.81
Other irregular eye movements	H55.89
Neurologic neglect syndrome (incl. visuospatial neglect)	R41.4
Visuospatial deficit	R41.842
Visual agnosia (incl. topograph-agnosia)	R48.3
Abnormal oculomotor study	R94.113
Injury of optic nerve and pathways	S04.0xxx
Injury of oculomotor nerve	S04.1xxx
Injury of trochlear nerve	S04.2xxx
Injury of abducens nerve	S04.4xxx

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