Advances in Ophthalmology and Optometry 1 (2016) 307–333

ADVANCES IN OPHTHALMOLOGY AND OPTOMETRY

Traumatic Brain Injury

Visual Consequences, Diagnosis, and Treatment

Kenneth J. Ciuffreda, OD, PhD[a,*], Diana P. Ludlam, BS, COVT[a], Naveen K. Yadav, BS Optom, MS, PhD[b], Preethi Thiagarajan, BS Optom, MS, PhD[a]

[a]Department of Biological and Vision Sciences, Brain Injury Research Unit, SUNY/State College of Optometry, 33 West 42nd Street, New York City, NY 10036, USA; [b]Chicago College of Optometry, Midwestern University, 555 31st Street, Downers Grove, IL 60515, USA

Keywords

• Traumatic brain injury • TBI • mTBI • Vision • Concussion • Visual diagnosis • Visual remediation • Vision therapy

Key points

- Mild traumatic brain injury (mTBI) represents a major public health problem in the United States and worldwide.
- The visual problems found in individuals with mTBI encompass a wide range of basic clinical vision areas, as well as more specialized oculomotor and non–oculomotor-based aspects.
- These vision problems can be remediated by a range of visual interventions: lenses, prisms, occluders, tints, and vision therapy.
- There are several critical areas for future study of the vision sequelae in mTBI, including the search for clinical and objective biomarkers for mTBI, visual assessment and therapeutic intervention in the pediatric and severe brain injury populations, and development of a symptom and quality-of-life questionnaire.

INTRODUCTION

Traumatic brain injury (TBI) is a major public health, social, economic, political, medical, and optometric concern in the United States [1,2]. It will likely continue to be so in the foreseeable future due to the past wars in Iraq

The authors have nothing to disclose.

*Corresponding author. *E-mail address*: KCIUFFREDA@SUNYOPT.EDU

2452-1760/16/$ – see front matter
http://dx.doi.org/10.1016/j.yaoo.2016.03.013

and Afghanistan and the related high occurrence of TBIs [3], as well as the recent focus on sports-related concussions [4] (see Sections Current Relevance and Future Avenues to Investigate). The costs in the United States are estimated to be more than $75 billion annually [5], with TBI being the nation's leading cause of death and disability [6]. TBI has been estimated to affect 1.7 million people in the United States annually [5], with motor vehicle accidents and falls being 2 of the most common causes [7]. TBI affects 10 million worldwide [8]. At the early acute phase of the injury (ie, the first few hours), a TBI is traditionally classified as either mild (mTBI), moderate, or severe, with approximately 75% being of the mild variety [9]. This categorization has traditionally been based on the Glasgow Coma Scale and other metrics [9,10]. However, the term "mild" is misleading, as the multilevel and long-term impact on many of these patients is anything but "mild," as described in this article.

What is TBI? It can be defined as "any structural damage caused by an external force to the brain and its associated structures, such as the cranium, resulting in physiologic disruption of brain function" [7]. It is of a sudden onset, nongenetic, noncongenital, nondevelopmental, and nondegenerative nature. Its immediate effects include one or more of the following general signs and symptoms: loss/altered state of consciousness, memory loss, intracranial lesion, and neurologic deficits [10].

A patient with mTBI presents with a constellation of general dysfunctions (Fig. 1). This is not surprising given the global nature of the 2-phase brain insult that is typical in mTBI [11]. In the first or *primary* phase, there is the immediate, biomechanically based, and typical coup-contrecoup injury (Fig. 2). This initially occurs to the cranial area and underlying brain tissue in the region of the direct external force (ie, the coup). Then, due to the differential deceleration/acceleration inertial forces between the rigid/fixed cranium and the 2.5-pound jellolike brain mass, there is injury to the opposite brain pole region (ie, the contrecoup). In addition, there are concurrent rotational, translational, and screw movements of the brain within the cranium, thus causing further brain contusion and damage (eg, stretching), especially to the white matter fiber tracts, a key problem in mTBI (Fig. 3). Also, there is concomitant flexing and twisting of the highly susceptible midbrain region, especially in children, with this being a primary oculomotor control area. This primary phase is then followed by the *secondary* phase of the brain injury occurring from days to months afterward, with it being of a biochemically based nature. It results in a cascade of events at the cellular level (Fig. 4), thus producing cell damage and death, and related toxic events, to the brain and its environment. The degree of cellular insult during this secondary phase is predictive of the patient's recovery; the more the damage, the poorer the recovery [12]. Together, the comprehensive and global effects of the primary and secondary injury phases will produce abnormalities in the sensory, motor, perceptual, cognitive, attentional, behavioral, pharmacologic, somatic, and linguistic domains in many patients with TBI. For example, an individual with mTBI might exhibit cognitive and attentional deficits, difficulty sleeping, inappropriate behavior,

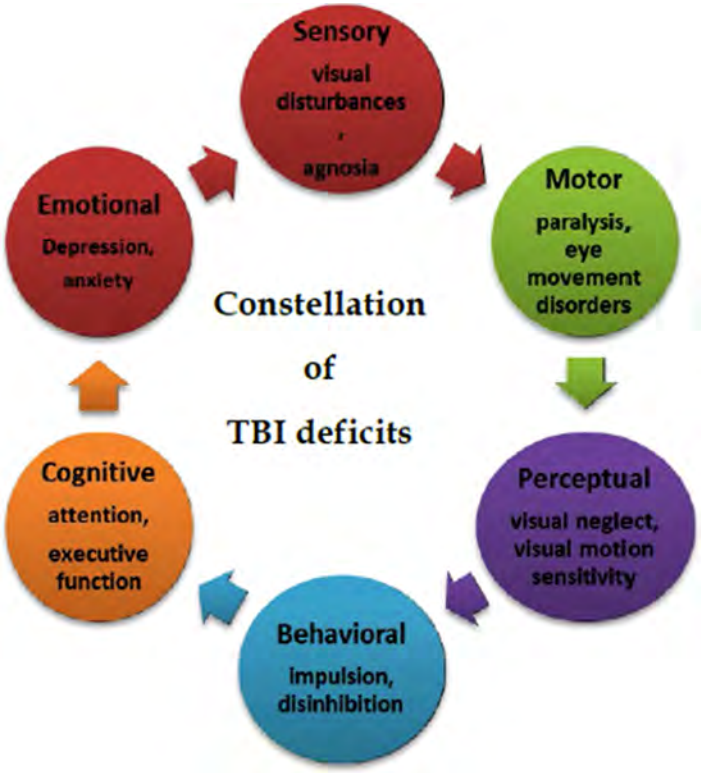

Fig. 1. Spectrum of general deficits in TBI. (*From* Thiagarajan P. Oculomotor rehabilitation for reading in mild traumatic brain injury. [PhD dissertation]. NYC: SUNY/Optometry; 2012.)

auditory information-processing problems, hyperacusis, and unsteady gait. These must be considered in the overall diagnosis, prognosis, and therapeutic recommendations. Each of these potentially abnormal areas should not be assessed in isolation by the physiatrist and others on the overall rehabilitative team. These global and integrative problems also must be considered along with the individual's short-term and long-term vocational and avocational goals in mind, including those related to vision.

Similarly, and more specifically, those with mTBI present with a constellation of visual dysfunctions, and with related symptoms (Box 1) and signs (Box 2), for the same reasons. This is not surprising, as most of the 12 cranial nerves and 30 to 40 areas of the brain involve vision and visual information processing [2]. Injury to these nerves and brain areas will result in a range of sensory (eg, visual field loss, photosensitivity), motor (eg, inaccurate saccades, vergence deficits, and variable accommodation), perceptual (eg, difficulty with figure-ground discrimination, hypersensitivity to visual motion), and attentional (eg, visual attention deficits, integrated auditory-visual processing) problems [1,2,13–15].

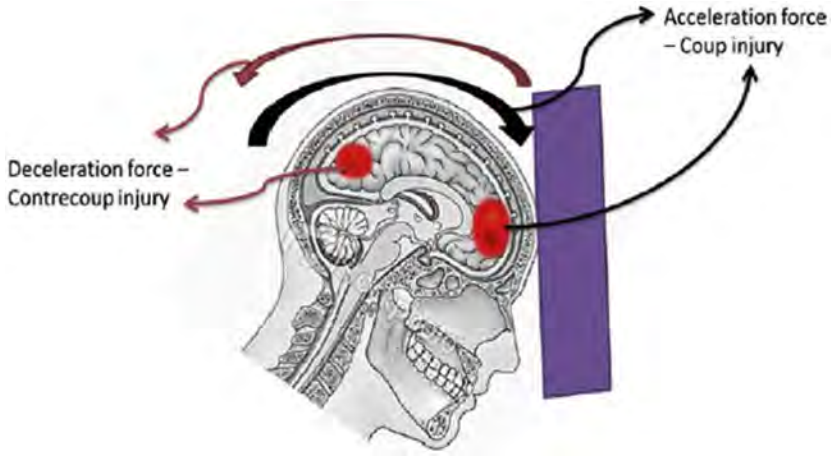

Fig. 2. Coup-contrecoup mechanism of head injury. (*From* Thiagarajan P. Oculomotor rehabilitation for reading in mild traumatic brain injury. [PhD dissertation]. NYC: SUNY/Optometry; 2012; with permission.)

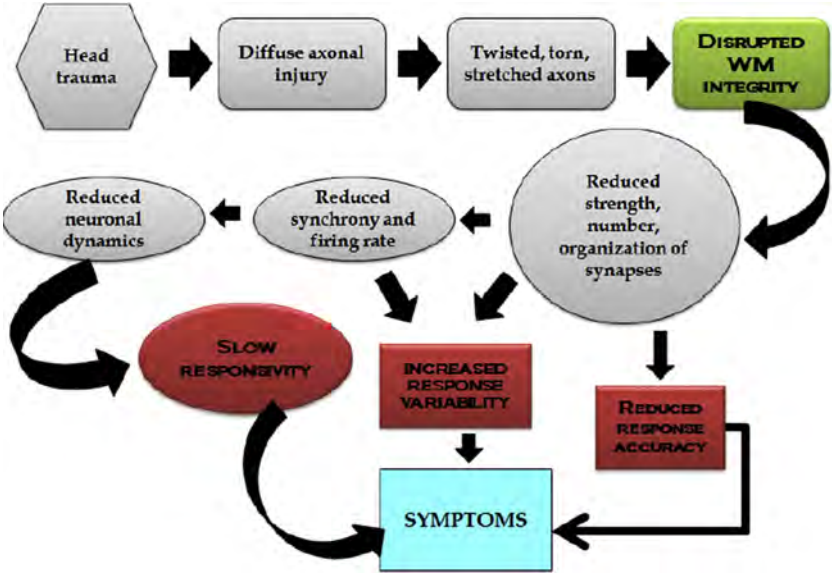

Fig. 3. Proposed mechanisms contributing to the slow responsivity and symptoms in TBI. WM, white matter. (*From* Thiagarajan P. Oculomotor rehabilitation for reading in mild traumatic brain injury. [PhD dissertation]. NYC: SUNY/Optometry; 2012; with permission.)

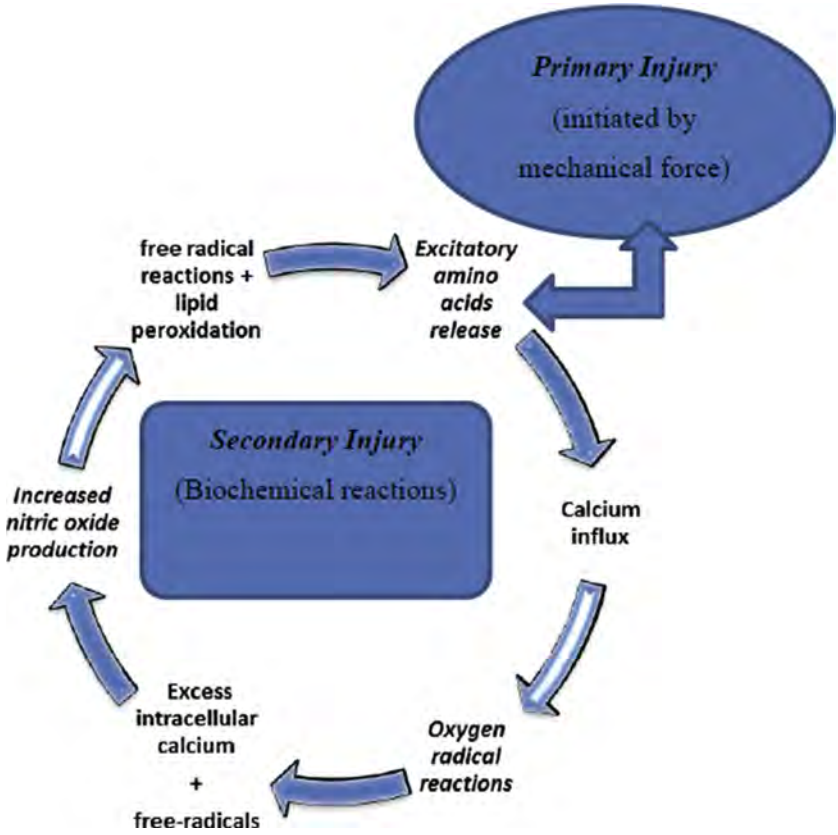

Fig. 4. Cascade of biochemical reactions in TBI. (*From* Thiagarajan P. Oculomotor rehabilitation for reading in mild traumatic brain injury. [PhD dissertation]. NYC: SUNY/Optometry; 2012; with permission.)

Thus, by the global nature of the injury process, and the complex and interactive nature of the brain areas, visual problems would be a likely sequelae after an mTBI in many individuals.

One of the problems that many of those eye care practitioners who wish to be involved in the vision care of the patient with mTBI (and TBI in general) have is that they perceive it to be too complicated and simply overwhelming, and ask, "Where do I start?" Or they perform only selected and common, relatively low-yield tests (eg, visual acuity and refractive correction at distance) from the entire, optimal, and essential clinical armamentarium [13], and then deem the person to be "normal" despite their persistent visual and related symptoms. Clinical testing of these patients is not simple, but it is not that difficult, *if* one has a conceptual model, or "roadmap," of the symptoms and signs that may be anticipated, and their interactions, as well as the possible therapeutic aids, in conjunction with some specific instructional and test condition

Box 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 (eg, minimal eye contact)
- Inability to tolerate complex visual environments (eg, 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; with permission.

Box 2: Oculomotor signs in TBI

- Reduced amplitude of accommodation
- Increased lag of accommodation
- Reduced relative 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; with permission.

guidelines. With this goal in mind, we have developed a "conceptual model" of vision care in patients with mTBI [14,15], both with respect to the clinical diagnosis and realm of therapeutic interventions.

Thus, in the next section, we discuss the visual problems and diagnosis, and forms of available remediation, as well as related clinical issues, in patients with mTBI within the context of the 4 tiers of our conceptual model.

TRAUMATIC BRAIN INJURY: THE VISUAL CONSEQUENCES, DIAGNOSIS, AND TREATMENT

Our conceptual model of vision care in mTBI, with its implications for related basic and clinical research, is presented in Fig. 5 and Box 3 [14,15]. Four tiers exist: the basic vision examination and related vision problems, the binocular/oculomotor-based examination and related vision problems, the non–oculomotor-based examination and related vision problems, and the non–vision-based problems. In each case in Box 3, the upper line (A., B., C., etc) represents the diagnostic condition or problem, and the text below it shows the possible remediations. See Table 1 for possible neurologic substrates for each dysfunction [10].

Tier 1

This entails the basic vision examination, which serves as the "foundation" for the subsequent 3 clinical tiers, as well as for any subsequent related clinical

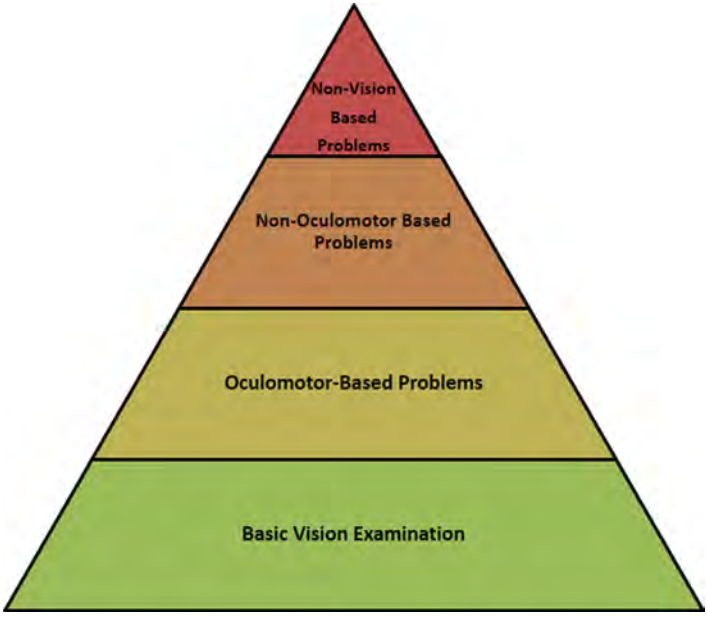

Fig. 5. Conceptual model pyramid showing the 4 tiers of vision care in mTBI. (*From* Ciuffreda KJ, Ludlam DP, Yadav NK. Conceptual model pyramid of optometric care in mild traumatic brain injury (mTBI): a perspective. Vis Dev Rehabil 2015;2:105; with permission.)

Box 3: Four-tiered conceptual model of vision care in mild TBI

I. Basic vision examination
  A. Refractive status
    Distance and/or near lenses
  B. Binocular status
    Vision therapy, near lenses, and/or prisms
  C. Ocular health status
    Treatment and/or medical referral
II. Oculomotor based-vision problems
  A. Version
    Vision therapy
  B. Vergence
    Vision therapy, near lenses, and/or prisms
  C. Accommodation
    Vision therapy and/or near lenses
III. Non–oculomotor-based vision problems
  A. Abnormal spatial localization
    Yoked prisms
  B. Photosensitivity
    Tints and/or wide brimmed hats
  C. Motion sensitivity
    Binasal occlusion, tints, and/or motion desensitization
  D. Vestibular dysfunction
    Vision and/or vestibular therapy
  E. Visual field defect
    Visual scanning training and/or prisms
  F. Visual information processing dysfunction
    Visual information processing and perceptual therapy
IV. Non–vision-based problems
  A. Depression
    Counseling and/or medications
  B. Fatigue
    Nutritional counseling, exercise, and/or adaptive strategies
  C. Cognitive impairment
    Cognitive therapy
  D. Behavioral problems
    Counseling and/or medications

E. Postural problems
Yoked prisms, physical and/or occupational therapy
F. Attentional problems
Cognitive/attentional therapy and/or medications
G. Neurologic problems
Referral to a neurologist

From Ciuffreda KJ, Ludlam DP. Conceptual model of optometric vision care in mild traumatic brain injury. J Behav Optom 2011;22:12; with permission.

and basic human research in the area of TBI. If each subcomponent of this "foundation" tier is not assessed comprehensively and carefully, then any subsequent testing is held in possible question and with uncertainty. This tier involves the basic refractive status, the general binocular/oculomotor status, and the ocular and general health status, which are the 3 subcomponents of *any* vision assessment irrespective of the diagnosis.

Within each of the 3 subcomponents of this first tier, there are unique problems relevant to the patient with mTBI, as well as the patient with TBI in general.

Refractive status

After an mTBI, there can be found either increased myopia or new/increased hyperopia in a patient, which on first blush seems to be contradictory: how can

Table 1
Some possible neurologic substrates for visual dysfunctions in traumatic brain injury

Dysfunction	Substrates
Altered refractive state	Parasympathetic and sympathetic systems
Abnormal version	Frontal eye fields, supplementary eye fields, posterior parietal cortex, superior colliculus, visual cortex, pontine reticular formation, cerebellum
Abnormal accommodation	Visual cortex, parasympathetic system, sympathetic system, cerebellum, midbrain, parietal-temporal area, Edinger-Westphal nucleus
Abnormal vergence	Cerebellum, pretectal nucleus, superior colliculus, supraoculomotor area, oculomotor nucleus, nucleus tegmenti ponti
Photosensitivity	Brainstem trigeminal nucleus, nociceptors of the trigeminal subnucleus caudalis
Motion sensitivity	Middle temporal areas, vestibular apparatus
Vestibular defects	Vestibular labyrinth, vestibular nerve, vestibular nuclei, cerebellum
Visual field defects	Retina, optic nerve/tract/chiasm, lateral geniculate nucleus, visual cortex, temporal/parietal lobes
Visual information processing/perception/ visual attention	Right parietal lobe, right prefrontal lobe, right cingulate nucleus, thalamus, striatum

From Thiagarajan P. Oculomotor rehabilitation for reading in mild traumatic brain injury. [PhD dissertation]. Santa Ana (CA): SUNY/Optometry; 2012; with permission.

you have both? The former can be explained by an abnormally functioning sympathetic system, common in mTBI, so that the pharmacologic control system of the crystalline lens cannot reduce/"relax" accommodation fully and sufficiently with distant gaze, and thus increased myopia and blur become manifest. In contrast, the latter case can be explained by an abnormally functioning parasympathetic system, which can occur in mTBI. Thus, the ability to increase accommodation to compensate for any residual, uncorrected hyperopia is compromised (eg, slowed, delayed, ill-sustained), and hence the latent hyperopia becomes manifest, perhaps with intermittent blur reflecting the ability to compensate only partially. In addition, 2 other problems/concerns may arise based on the optical correction. First, the use of progressive addition lenses is usually not recommended, especially in those with visual motion sensitivity (VMS) and/or vestibular dysfunctions (see "aging" in the "Current relevance" section). Due to their complex design optics, the phenomenon of "swim" is present in the more peripheral regions of the lenses, thus producing apparent motion of the visual field in a counterphase manner with any head movement. A better option is to prescribe separate single-vision spectacles for each of the patient's working distances. Second, and related, these patients can be highly sensitive to very small changes in their spectacle correction, as general sensory hypersensitivity is common in the mTBI population. Thus, minor changes in their correction may result in large perceived effects, sometimes being adverse in nature.

Binocular status

This has been a primary focus of attention in mTBI for decades [1,2,16]. In general, this includes the areas of binocularity (eg, phoria, tropia) and ocular motility (eg, saccades, paresis). Aspects of binocularity and ocular motility are abnormal in many individuals with mTBI. This has been well documented both in the clinic and in the research laboratory. Near plus lenses and prisms, as well as related vision therapy (ie, oculomotor rehabilitation), have been advocated and used successfully in the treatment and remediation of these problems, respectively. These are considered more fully in the section on Tier 2.

Ocular and general health status

This too is a critical aspect in the care of patients with mTBI, as they exhibit a high prevalence (up to 10 times greater "relative risk") of certain ocular disease-related conditions as compared with the age-matched, patient without TBI [17]. These abnormal conditions include corneal abrasion, blepharitis, chalazion/hordeolum, dry eye, traumatic cataract, vitreal prolapse, and optic atrophy. Some of these diagnostic conditions would likely involve comanagement by an optometrist and an ophthalmologist. In addition, it may require specialized testing, such as contrast sensitivity, A-scan and B-scan ultrasonography, electroretinography, and the visual-evoked response, as well as ocular surgery. Inclusion of direct ocular insult per se in conjunction with the head insult occurs in many with mTBI/TBI, and, hence, the aforementioned ocular consequences are readily explained, and in fact are to be expected in many.

Tier 2

This second tier specifically addresses the "oculomotor" system in detail, namely version, vergence, and accommodation. For each of these oculomotor subsystems, a range of key findings is briefly discussed based on objective laboratory measurement and clinical testing. The oculomotor system is critical to our understanding of the challenges that these individuals have, as 90% of them reporting "visual symptoms" in a clinic mTBI population manifested 1 or more oculomotor deficits [18].

Version

Aspects of versional eye movements have been found to be abnormal in approximately 50% of a sample clinic population of adults with mTBI and visual symptoms [18].

Fixation. Fixational eye movements were found to exhibit increased positional errors and considerable variability (up to 3°) compared with the norm of 0.25°. Fixational accuracy improved by 35% following a brief 9-hour period of oculomotor rehabilitation [10] (Fig. 6).

Saccades. Saccadic accuracy (ie, gain) was reduced by 10% to 20%. Thus, relatively large saccadic positional errors frequently are present, which require execution of 1 or 2 additional saccades to attain foveation. Such inaccuracy was also found during both actual and simulated reading, the presence of which acts to impede the reading rate process and reduce the reading rate. Following oculomotor rehabilitation, accuracy improved significantly [10]. However, both saccadic latency and saccadic peak velocity were normal. The former suggests absence of a visual processing delay in the neural

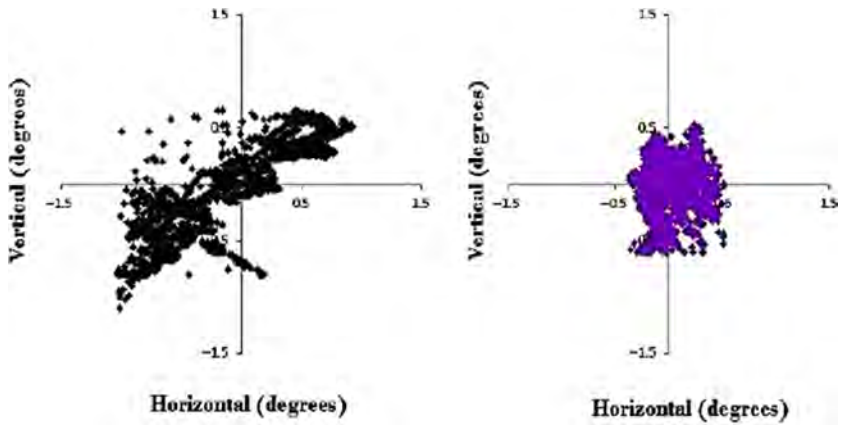

Fig. 6. Two-dimensional (horizontal and vertical) plot of binocular central midline fixation before (*left*) and after (*right*) oculomotor training in a typical adult patient with mTBI. (*From* Thiagarajan P. Oculomotor rehabilitation for reading in mild traumatic brain injury. [PhD dissertation]. NYC: SUNY/Optometry; 2012; with permission.)

pathway, whereas the latter suggests normal midbrain saccadic controller signal processing and formulation.

Pursuit. The findings here were similar to those mentioned previously for saccadic accuracy before and after oculomotor therapy; that is, the reduced gain significantly increased [10].

Reading. The most common vision symptom in the mTBI population is a "reading problem" [18–20]. Reading rate was reduced by approximately 25%, and the number of fixations (ie, progressive left-to-right saccades) was increased by approximately 20%, per the objective Visagraphic reading eye movement assessment. Both parameters improved following only 9 hours of oculomotor-based therapy over an 8-week period (Fig. 7) [10]. This suggests a primary *oculomotor basis* for their common symptom of having a "reading problem." Both reading comprehension and regressive eye movements were normal.

Vergence

Aspects of vergence eye movements have been found to be abnormal in approximately 55% of a sample clinic population of adults with mTBI and visual symptoms [18].

Clinically. The following clinically based parameters were found to be abnormal: near point of convergence (NPC) break and recovery, positive fusional vergence

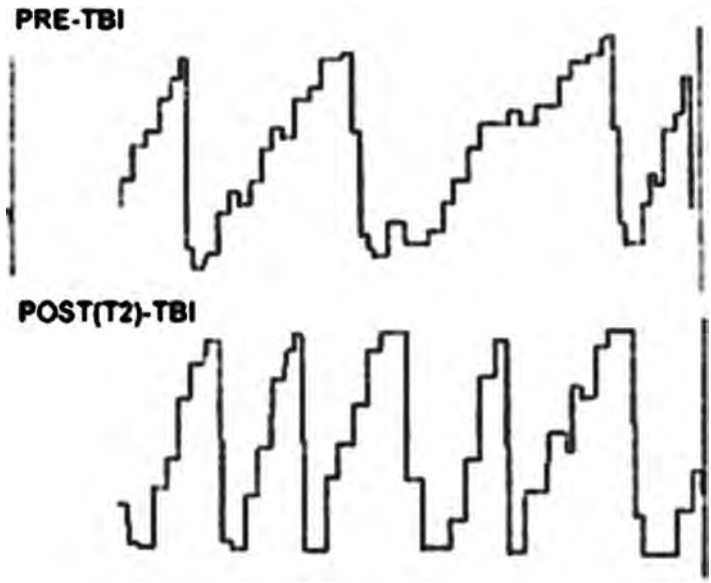

Fig. 7. Horizontal reading eye movements in an adult patient with mTBI before (PRE) and after (POST) oculomotor training. (*Adapted from* Han Y, Ciuffreda KJ, Kapoor N. Reading-related oculomotor testing and training protocols for acquired brain injury in humans. Brain Res Brain Res Protoc 2004;14:10; with permission.)

break and recovery, negative fusional vergence break and recovery, and vergence dynamic facility. These parameters significantly improved with oculomotor rehabilitation [19,21]. In addition, near symptoms were reduced, and visual attention was improved, following the therapy. A likely key parameter that could serve as a high-yield, "clinical biomarker," for mTBI is the NPC break value [22].

There are 2 other aspects that are important and deserve special mention. First, in our clinic population, approximately 25% of the visually symptomatic patients with mTBI presented with strabismus of some type [18]. Thus, this oculomotor deviation should be remediated to ensure an improved level of binocularity, as well as a more acceptable cosmetic component. Second, vertical deviations, especially phorias, are common in those with mTBI based on our experience and those of others. Of particular importance is that a small hyperphoria (eg, 0.5–1.0 prism diopters) that is frequently found in the visually normal, asymptomatic, non-mTBI population may be problematic in those with mTBI, as their vertical vergence compensatory ability appears to be compromised, as is true of their overall vergence function. Vertical prism and/or vertical vergence training should be prescribed here. Related is our approach to this critical measurement. Because the dynamics (ie, response completion time and peak velocity) of vertical vergence is approximately 5 to 10 times slower than its horizontal counterpart, the vertical measuring prism should be introduced proportionately slower for accurate vertical vergence range assessment, primarily to prevent underestimation [10]. Further, to assess the phoria magnitude itself requires complete fusional dissociation/disruption of binocular vision for at least 10 seconds [10].

Laboratory. The following laboratory-based parameters were found to be abnormal using an objective recording technique: peak convergence and divergence velocity were reduced, and steady-state variability was increased. All improved significantly with only 9 hours of oculomotor rehabilitation [21]. In addition, near symptoms reduced, and visual attention improved, following the therapy [21]. A likely key parameter that could serve as a high-yield, "objective biomarker" for mTBI is convergence peak velocity [22].

Accommodation

Aspects of accommodation have been found to be abnormal in approximately 40% of a sample clinic population of adults with mTBI and visual symptoms [18].

Clinically. The following clinically based parameters were found to be abnormal: the amplitude of accommodation and the accommodative dynamic facility, both monocularly and binocularly. Furthermore, they all significantly improved following only 9 hours of therapy [23]. In addition, near symptoms were reduced, and visual attention was increased, following the therapy [23]. A likely key parameter that could serve as a high-yield "clinical biomarker" for mTBI is the binocular amplitude of accommodation [22].

Laboratory. The following laboratory-based parameters were found to be abnormal using an objective recording technique: peak accommodative

velocity for both increasing and decreasing rapid step responses [23]. Furthermore, these parameters significantly improved following a 9-hour period of oculomotor therapy [23]. A likely key parameter that could serve as a high-yield, "objective biomarker" for mTBI is accommodative peak velocity [22].

Tier 3

This third tier encompasses the *non*–oculomotor-based vision problems commonly found in individuals with mTBI, and typically TBI in general. Presence of any 1 or a combination of these problems can confound the vision examination procedures and findings in tiers 1 and 2, as well as adversely affect the overall prognosis and therapeutic progress.

Abnormal egocentric localization

Abnormal egocentric localization (AEL) refers to a rotational (ie, polar), and *not* lateral, displacement of one's perception, or sense, of "straight-ahead." This results in a mismatch between the veridical, or objective, direction of straight ahead and the patient's anomalous, or subjective, direction sense [24,25]. AEL is estimated to be present in at least 30% of those with mTBI/TBI. Symptoms include difficulty with ambulation (eg, they feel "out of synch" with their environment) and mislocalization of objects. This is due to alteration, or directional bias, in their spatial map of the external world, presumably due to right parietal lobe involvement. Yoked prisms (ie, prisms with their bases in the same direction, such as "bases left") can reduce this perceptual mismatch and improve spatial abilities [25].

Photosensitivity

Photosensitivity (PS) is one of several "hypersensitivities" found in those with mTBI. It refers to the sensation of "discomfort" in the presence of light levels, or types of lighting (eg, fluorescent), that do not invoke such a sensation in others. There may be, in part, a pupillary basis for PS [26,27]. PS, or light sensitivity, is found in 50% of those with mTBI, as compared with 10% in healthy individuals [28]. Tinted lenses of various types (ie, gray, colored, or polarized) and different magnitudes of transmission can be very helpful, as well as the wearing of a wide-brimmed hat to occlude the offensive overhead illumination. Prescription of lesser magnitudes of tinted lenses has been found to gradually promote visual adaptation to the PS over the long term (ie, 1 year or more) [28].

Visual motion sensitivity

VMS is another of the sensory-perceptual "hypersensitivities" found in this population. VMS refers to visual motion hypersensitivity, especially in the retinal periphery, akin to "Gibsonian optic flow." That is, as individuals traverse their complex environment (ie, walking down a busy street or grocery aisle), the correlated dynamic stream of retinal-image motion reduces their stability. This causes the individual to feel unstable and fear bumping into objects, and even falling, frequently accompanied by nausea. The use of binasal occluders (BNO) adhered to the spectacles (Fig. 8) has been demonstrated to be

Fig. 8. Schematic representation of BNO on a patient.

helpful in many, as it reduces the amount of peripheral retinal-image motion bitemporally [29–31]. The positive effects of BNO in those with mTBI *and* VMS have been documented objectively using visual-evoked potential (VEP) testing [29,30]. Use of spectacle tints to reduce the intensity of the offensive motion, as well as desensitization procedures to adapt to the retinal motion, also may prove beneficial. Computerized dynamic posturography (CDP) could be used to assess objectively the effect of a therapeutic intervention. VMS is estimated to be present in at least 40% of this population.

Vestibular dysfunction

Vestibular dysfunction refers to disturbance of the balance system. Symptoms may include dizziness, blur, and nausea, as well as a sense of instability, with related possible falls. It is estimated to be present in up to 80% of this population. Presence of spectacle blur, for example, due to a faulty spectacle

correction, may exacerbate the primary vestibular deficit (see the section "Current relevance"). The abnormal vestibular system can be remediated, at least in part, by using such techniques as the "Brock string" to incorporate vergence interaction with the vestibular system, a critical aspect, especially at near bifixation distances. As described previously in "Visual motion sensitivity," CDP could be a useful assessment tool here also. Others, such as the vestibular, physical, or occupational therapist, also can perform this therapy.

Visual field defects

Visual field defects refer to regions of the individual's visual field that are either absent or sensorially depressed due to the effects of an mTBI on the primary visual brain tracts. Deficits of all types may be present, ranging from hemianopia to small, scattered regions of reduced sensitivity [32]. Symptoms may include bumping into objects and difficulty with ambulation. Visual field deficits have been found in 35% of patients with mTBI in a sample clinic population having a range of visual symptoms [32]. One can use specific programmed, visual scanning techniques to improve object detection and ambulation. Sector prisms also may be used and incorporated into the periphery of the spectacle correction to function as a "spotting" system, as needed, for detection of objects and people in the abnormal visual field. Increased head movement also may be encouraged.

Visual information processing dysfunctions

A wide range of visual processing and perceptual deficits are found in this population. These include processing delays and processing errors, sometimes in conjunction with similar and related problems of the auditory system (ie, "dual-sensory impairment") [33]. An interesting perceptual disturbance found in many involves "figure-ground" discrimination/disambiguation [2]. For example, the patient will have difficulty locating an object embedded in a complex array, such as a desktop or refrigerator, despite the object being well above their visual threshold. Various forms of perceptually based vision therapy can be helpful to these patients, such as parquetry blocks and maze tracing. Although the prevalence of such deficits remains unknown in this population, it is likely quite high, perhaps 50%. This area of clinical and laboratory study remains relatively neglected in these patients.

Before moving on to Tier 4, a brief discussion of the clinical practice of "vision therapy" is warranted, as this has been mentioned several times in tiers 1 to 3. In the present context, the terms "neuro-optometric rehabilitation" (NOR) or "neuro-visual rehabilitation" (NVR) are more descriptive and broader in nature than "vision therapy" [34]. What exactly do we mean by this? NOR/NVR refers to "a series/sequence of scientifically proven, brain-based, sensory-motor-perceptual procedures and techniques that remediate visual dysfunctions/deficits, which improves one's visual efficiency and visual comfort" [34]. It "addresses the oculomotor, accommodative, visuomotor, binocular, vestibular, perceptual/visual information processing and specific ocular/neurologic sequelae of the acquired brain injury population" [34]. It

includes "…corrective lenses, prisms, tints and coatings, selective occlusion, and visual therapy" [34]. It incorporates the psychological principles of perceptual and motor learning underlying brain plasticity [35]. See Fig. 9 for a proposed model of oculomotor-based vision therapy incorporating these principles. Furthermore, its efficacy is demonstrated by objectively based improvements in visual system responsivity and related visual performance [35].

Tier 4

This last tier encompasses the *non*–vision-based problems commonly found in individuals with mTBI, and TBI in general. With the possible exception of postural problems, there is little that the eye care practitioner can directly do to help the patient. However, the practitioner can help *in*directly by first identifying the specific problem, at least tentatively, and then making the proper referral. This is important, as presence of 1 or more of these non–vision-based problems can have a profound effect on the patient's vision-based problems as related to prognosis and remediation effects: their presence will likely have a substantial, negative effect on the vision care rendered, as described in the following sections.

Depression

The depressed patient may forget appointments, not perform the therapy faithfully or accurately, and not wear their spectacle correction as instructed.

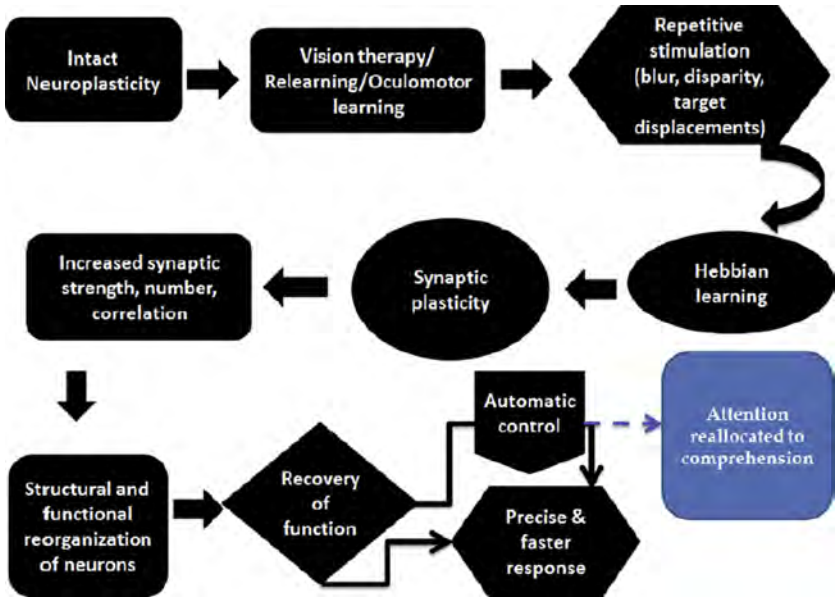

Fig. 9. Proposed underlying basis for oculomotor-based vision therapy/vision rehabilitation and neurovisual rehabilitation in general. (*From* Thiagarajan P. Oculomotor rehabilitation for reading in mild traumatic brain injury. [PhD dissertation]. NYC: SUNY/Optometry; 2012; with permission.)

Fatigue
The fatigued individual may not be able to perform any testing or therapy for more than a few minutes. Thus, such a patient will require more frequent rest intervals, and furthermore possibly not be able to complete the daily therapy as required for a high level of success.

Cognitive impairment
Presence of a cognitive impairment may result in an inability to follow instructions carefully and in the proper sequence (eg, executive function disability).

Behavioral problems
The patient with a behavioral problem (eg, impulsivity) may be disruptive during testing, as well as make unreasonable demands on the doctor and staff.

Postural problems
The problem of postural control and stability may be helped, at least in part, by a prescription of yoked prisms, which alter the visual input in a controlled manner based on their characteristic nonuniform magnification properties, and thus influence one's postural stance and center of gravity and can be used therapeutically [25]. This can frequently be done in conjunction with the physical and/or occupational therapist.

Attentional problems
With presence of an attentional deficit, instructions may be misunderstood and need to be repeated, and stated more slowly, and hence the testing may be difficult to complete fully.

Neurologic problems
Presence of a neurologic dysfunction (eg, uncontrolled muscle spasms, tremors) could adversely affect visual motor abilities, such as eye-hand coordination and identification-based cognitive therapy using visual scanning, as well as any oculomotor-based vision therapy.

CURRENT RELEVANCE

In addition to the more obvious areas involved in the etiology of concussion/mTBI/TBI that have been, and will remain, important (eg, motor vehicle accidents and falls), there are several other crucial areas of particular "current relevance" (see sections "Introduction" and "Future avenues to investigate"). Some are suggested and briefly described in the following sections.

Military encounters

Military encounters involving the United States (eg, the Iraq and Afghanistan wars) over the past nearly 2 decades have brought the neurologic condition of concussion/TBI to the forefront of the eye care and medical communities [13], as well as to the military and to society at large [3]. Over the past 15 years, at least 250,000 military members have sustained a concussion and at least 60,000 more a TBI [36]. The resultant visual sequelae are manifold, including the visual sensory, motor, and perceptual domains [1,2,13–15]. For example, some of the most

common and persistent symptoms include photosensitivity, oculomotor and related reading problems, and visual-spatial deficits [1,2,13–15]. These head and *brain* insults frequently occur in conjunction with a host of related eye, or *ocular*, trauma-induced insults (eg, a corneal burn), especially in the military [37]. This compounds the overall visual loss and deficit in many cases, as well as increases the time and complexity of any vision rehabilitation.

Both the short-term and long-term consequences are, and will continue to be, overwhelming to the military and Department of Veterans Affairs medical complex economically, socially, educationally, vocationally, and politically. Although the number of service members incurring a concussion/TBI is likely to decline substantially in the foreseeable future due to reduction in the intensity of our military engagements, many cases will still be diagnosed. However, and of perhaps greater concern, are those individuals already in the long-term, chronic phase of their TBI (ie, post 45 days following insult) and in need of remediation for the residual basic vision and visual information-processing deficits. For example, in a recent pilot study (n = 31) of blast-induced mTBI, on average 4 years after injury, more than two-thirds (68%) remained visually symptomatic, with this number increasing to nearly 90% (87.5%) for those incurring more than 1 brain insult [38]. Thus, there will be continued and increasing need over the next several decades to perform extensive/intensive vision remediation of all sorts, along with long-term follow-up (ie, years). In addition, there will be a need to develop a validated concussion/TBI-specific quality-of-life (QOL) symptom questionnaire to assess the immediate effects, as well as the natural recovery changes and any remediation effects. Last, efforts should be made to develop and institute "reeducation" programs to allow these neurologically compromised individuals to be reintegrated into the military, and later civilian workforces, to once again become productive members of society.

Sports

Interest and research in the area of sports concussion has "exploded" over the past decade in the National Football League (NFL). It is likely that the recent resurgence of interest in sports-related concussion evolved from the Iraq and Afghanistan wars [3], where TBI/concussion was the "signature injury," as well as the "invisible injury" in many cases. However, sports-related interest dates back to at least 1905, where concussion was referred to as the football "death harvest" [39]. Concussion in sports has become, and will remain over the foreseeable future, a major medical and public health concern. It is estimated that there are 1.6 to 3.8 million sports-related concussions in the United States annually [4]. This is particularly relevant for "contact" sports, especially football, soccer, and rugby.

There are many relevant and critical questions that need to be addressed based on sound scientific principles and techniques over the next decade in this area:

a. What can be done to minimize and even prevent the occurrence of a concussion? Factors such as improved helmet design in conjunction with changes in the rules of engagement are likely choices.

b. What is the best test, or test battery, to detect a concussion during a game? Some suggestions have been the subjectively based King-Devick Test (K-D) to assess overall global saccadic eye movement sequencing and the Sports Concussion Assessment Tool (SCAT3) to assess cognitive and balance performance [40], as well as objectively based pupillary dynamics [22]. Simple, objective testing of saccadic latency has also been proposed and tested in collegiate boxers [41]. This will be a rapidly developing and increasingly important area of study.
c. What can be done if a concussion is detected, or even suspected, in terms of ''recovery''? Some suggestions have been both physical and cognitive rest for a week or more. This critical area needs further investigation, likely involving objectively based assessments, such as saccadic latency testing and perhaps even brain imaging in selected cases.
d. What tests can be administered to determine ''return-to-play''? The SCAT3 test [40] and objective pupillometry [22] have been proposed, as well as objective testing of saccadic eye movements for the parameter of latency [41], as mentioned previously. This remains an area in need of extensive and intensive further investigation, possibly including brain imaging using diffusion tensor imaging for white matter tract assessment in selected cases, especially for well-paid National Football League–level players in whom any unnecessary ''down'' time is costly.

Aging

The United States has an increasing elderly population. For example, the 65-year and older population was 43 million in 2012, and it is projected to be 84 million by 2050. Similarly, this same elderly population comprised 13% of the total population in 2010, and it is projected to be greater than 20% by 2030 [42].

With advancing age, there are increased balance and postural control problems, for a number of reasons. For example, saccular function of the vestibular system reveals distinct aging effects [43], as well as the occurrence of aging of the overall vestibular system [44]. In the latter case, the visual-vestibular-ocular reflex exhibits considerably earlier aging effects when compared with the general vestibular-ocular reflex (VOR) [44], and hence a critical and direct *visual*-vestibular neurologic connection is compromised at an earlier stage in the aging process.

With increased balance problems in the elderly, there is a greater likelihood of a fall, which in many cases may involve the head. For example, falls are responsible for 60% of all TBIs in the 65-year and older population [45]. Furthermore, patients older than 75 years have the highest rates of TBI-related hospitalizations and deaths [45]. These statistics have important ramifications for the physiatrist, and others, involved in the overall medical and rehabilitative care of the geriatric patient.

Last, and most importantly for eye doctors and related personnel (eg, vision nurses, vision therapists, and occupational therapists), presence of visual impediments, such as cataract and refractive blur, will *further* increase the probability of a fall in this already ''at-risk'' elderly population, thus making visual

detectability more difficult, especially for low-contrast objects. Either of the aforementioned visual problems could adversely affect general contrast perception. These statistics and related problems have important clinical implications for those involved in their vision care, especially eye care practitioners. First, the visual impediments must be remediated, which would include proper refractive correction and cataract surgery [46], respectively, and other approaches (eg, sector prisms for visual field loss) [14,15]. In addition, preventive strategies should be developed to reduce the risk of falls and related TBI in this elderly population. This might include strategically placed, nighttime lighting in the home, as well as high-contrast and well-illuminated signage in the workplace and public areas.

Air pollution

One area that is critically important, and not in the limelight, is that of air pollution. The effects of such particulate matter on the brain appear to be substantial, especially in young children [47] and women [48]. These pollutants enter the nasal passages, then lungs, and on to the circulatory system and brain. They appear to have a neuroinflammatory effect resulting in neurodegenerative changes in the brain that adversely affect its development; that is, it produces a chronic encephalopathy phenomenon, and thus an insidious source of mTBI. These pollutants cause reduction in brain volume equivalent to 1 year of aging in persons older than 60 years [47,48], and hence there is accelerated brain aging. Such adverse effects are restricted to the white matter throughout the brain [47], with apparent greater effect in women [48]. These effects are also of special significance in children, with their continually developing brain. For example, 40% of autopsied children exposed to high levels of air pollution in Mexico City exhibited frontal lobe tau material, and approximately 50% had brain plaques. In contrast, none of the control child population exposed to low levels of air pollution in Mexico City manifested either of these brain abnormalities [47].

Clearly, this is an area of high neurobiological and neurodevelopmental concern, especially in the large "megacities," such as Beijing, Mexico City, and Delhi, where air pollution is rampant. In addition, cognitive deficits also have been uncovered in such exposed populations. These and related factors need to be considered educationally, both by the parents and the school system.

FUTURE AVENUES TO INVESTIGATE

There are a wide range of important avenues for future exploration in the area of mTBI/TBI and related visual dysfunctions (see "Current relevance"). Some are suggested and briefly described in the following sections.

Natural history of traumatic brain injury

This information would be useful as a guide in the patient's prognosis, as well as in the potential future need of a vision-based therapeutic intervention. For example, if a patient with mTBI fully recovers over the first 6 to 9 months of potential

"natural recovery," and now performs visually the same as he or she did before injury, then the patient would not require any vision rehabilitation. The patient could then return to the workforce. This would have a large and positive economic impact. Such information would also yield critical information regarding human brain plasticity.

However, a study of this type may be difficult to implement for 2 reasons. First, it would demand that subsequent to the acute and subacute phases of the brain injury, no direct vision (ie, vision therapy) or indirect vision-involved (eg, attentional and/or cognitive therapy) intervention would be permitted over a long time-course (eg, years) of follow-up assessment, as any intervention might contaminate the recovery findings and their interpretation. However, most patients would find this to be unacceptable, as they all want to "get better" and "recover" as quickly as possible. Second, in those with self-reported "recovery" and/or having received either a cursory vision or physical examination indicating "normal vision," this so-called "recovery" may reflect, at least in part, an adaptive strategy to circumvent and prevent recurrence of their residual visual symptoms. For example, if a patient found that he or she developed either a visually based headache or experienced visual fatigue after reading for 20 minutes, the patient would then conservatively cease reading after 10 minutes or so. This may be interpreted by the patient as "recovery," but not really physiologically and neurologically. Thus, a careful and thorough case history is essential.

Quality-of-life assessment questionnaire

The impact of a head injury on one's QOL is well known, but typically only documented *anecdotally* in the clinic domain. That is, in the vast majority of those individuals with persistent visual disturbances, their QOL is reduced in a number of ways. For example, they frequently report reading problems, visual memory deficits, blur, VMS, and so forth [1,2,13–15]. Presence of such symptoms and related visual deficits will adversely affect their ability to function optimally and efficiently for a range of vocational and avocational, visually based, tasks and endeavors, such as reading a book, ambulating in a busy corridor, and reading instructions on a medicine bottle. Unfortunately, as of yet, no such validated and comprehensive QOL questionnaire exists specifically to address the visual sequelae of TBI, especially in a detailed, quantitative manner. However, a useful vision-based QOL questionnaire that can be used in mTBI is available [34]. In addition, other questionnaires have been used with reasonable results that were not specific to TBI. Although they are not formally validated for TBI, this information may still provide important insights to the clinician and others.

Clinical trials

Again, and quite unfortunately, there is a paucity of well-conceived clinical trials for the range of visual deficits and therapeutic interventions in this population. For example, there has been only 1 randomized clinical trial in mTBI assessing the use of vision therapy to remediate oculomotor and related visual

dysfunctions [10,20,21,23], as described in "Tier 2" of the section "Traumatic brain injury: the visual consequences, diagnosis, and treatment." The results were excellent, including the 3-month and 6-month follow-up findings. Such clinical trials should be performed and expanded to include other forms of visual intervention (eg, yoked prisms or binasal occlusion) in larger sample sizes (eg, n = 100) with longer follow-up times if possible (eg, 1–2 years). The present objectively based laboratory results are convincing and favorable, and furthermore are consistent with the clinical findings in the mTBI/TBI population [1,2], so this area shows much promise.

Other populations

Most studies have focused on the adult mTBI population, which represents the largest TBI category and is likely the easiest to assess. However, there are several other distinct and underrepresented TBI subpopulations that both need and deserve to be investigated further, described as follows.

a. Head injuries are common in the pediatric population [49,50]. Despite this, little is known about the prevalence of mTBI/concussion in this group, as well as the related vision remediation aspects. This is unfortunate, as one would assume that brain plasticity is more robust in these younger patients than in the adult and geriatric populations. Furthermore, this information has critical educational implications for these children and their parents, as well as their teachers and school systems; for example, the development of return-to-learn school criteria [51].
b. Relatively little is known about the visual deficits and their remediation in the populations with moderate and severe TBI [52], which represent a relative minority (approximately 25%) but important sector of the population with TBI. These more impaired individuals can still receive and benefit from much of the full armamentarium of visual interventions, such as vision therapy, tints, and occluders, although perhaps in a more limited fashion depending on the severity of their medical, physical, and cognitive status. The QOL also should be assessed in this population [53].
c. Blast versus non–blast-induced TBI has important implications for the military. This has been a growing area of investigation due to the recent wars (see ''Current relevance''), yet it still remains understudied (see ''Traumatic brain injury: the visual consequences, diagnosis, and treatment''). Somewhat remarkably, the range of visual symptoms and visual deficits in those with blast versus nonblast head injury of the mTBI variety, in the *absence* of either associated ocular (eg, corneal laceration) or facial trauma (eg, blow-out fracture), is relatively similar.
d. A most interesting and socially important group is those who are either incarcerated [54] or have experienced spousal/parental abuse [55], which frequently involves blows to the head, and hence possible concussion. In the former, especially adolescents, history of a concussion will likely have an adverse effect on learning, reading, and general behavior and attention, with clear educational and vocational ramifications [56]. Self-reported concussion/mTBI in this population ranges from 20% to 70%. The likely high prevalence of oculomotor problems and related reading deficits, as well as attentional dysfunctions, in this group would result in a reduced QOL, increased educational problems, and greater overall general frustration, with perhaps these being factors in the high recidivism rate in the incarcerated population [57].

Further development of objective tests for detection of concussion/mild traumatic brain injury

This is currently one of the most aggressive areas of research in the concussion/mTBI field. It will likely continue to be so over the next decade (see "Current relevance"), with its great economic and social impact. Everyone is searching for "the" test, or short test battery, that can detect and confirm the presence of a concussion/mTBI in those more subtle and vague cases, such as those that may occur at the football sidelines, with a high degree of sensitivity and specificity. We, and others [41], have sought to develop instrumentation and protocols for this purpose, including assessment of the brain's visual cortex (eg, VEP amplitude and latency), pupillometry (eg, peak dilation velocity), and the oculomotor system (eg, peak vergence velocity) for this purpose [22]. Some of these tests have resulted in providing a very high yield (>90% true positives) for detecting concussion/mTBI using clinical laboratory techniques that take only a few minutes to implement [29,30]. More tests need to be developed that are rapid, simple, noninvasive, and automated, all with reasonable cost. Such tests would be especially important before and after a visual intervention, military deployment, or sports season.

Objective visual biomarker(s) for concussion/mild traumatic brain injury

Related to the section "Further development of objective tests for detection of concussion/mild traumatic brain injury," the development of appropriate instrumentation and related efficient test protocols should be advanced to uncover reliable, vision-based, objective biomarkers for detecting the presence of concussion/mTBI. This would be especially important in the emergency room, sports field, and military theater across the acute, subacute, and chronic phases of concussion/mTBI. Again, as in the previously mentioned section, it should involve a rapid and noninvasive approach to be most useful across all domains.

Automation

Related to the preceding 2 sections, objective procedures for both assessment and as potential visual biomarkers for concussion/mTBI should incorporate automated approaches [58]. This would make for a more rapid, less costly, and bias-free determination. It has been attempted in the military for oculomotor assessment [59]. Automation would allow testing and analysis to be performed by a trained technician or therapist rather than demanding the use of more highly trained and costly eye care practitioners or medical personnel.

SUMMARY

The topic of TBI has been an area of interest for millennia, probably as far back as the first engagements of rival tribes of cavemen. However, only over the past 2 decades has TBI come so dramatically to the forefront of both the public and professional eye. This is due to the relatively recent US military encounters, with its heightened awareness and visibility of this "invisible injury," namely concussion/mTBI, as well as sports-related concussion headlines and concern over both its prevention and detection. This latter point is particularly important

for young children playing contact sports, in which the desire to win is frequently aggressively promoted by both the coach and parents.

With the aforementioned, dual-pronged interest and motivation, as well as concerns about the elderly population and its increased frequency of falls and related head injuries, the specific aspects of TBI and its "visual consequences, diagnosis, and treatment" have been in the limelight. This has resulted in increased and intensive clinical and basic research, with this trend likely to continue into the foreseeable future.

There have been many recent advances in several avenues of this area of vision in TBI: improved detection and specification of the signs and symptoms, and their quantification; novel and objective approaches in the diagnosis, including the electro-physiological and brain-imaging domains as well as dynamic posturography; and further investigations including clinical trials related to therapeutic interventions using both subjective and objective outcome assessments.

These advances will continue into the future, with a push to find objective "biomarkers" for detecting the presence of concussion in its acute phase. These biomarkers will likely include both the noninvasive visual (eg, pupillometry dynamics, saccadic latency) and the invasive nonvisual (eg, blood serum) domains acting in concert to result in scientifically based, high-yield detectability.

Ultimately, the goal of study and investigation into the area of "vision and TBI" should result in improved clinical outcomes and quality of life for the patient.

References

[1] Suchoff IB, Ciuffreda KJ, Kapoor N, editors. Visual and vestibular consequences of acquired brain injury. Santa Ana (CA): Optometric Extension Program Foundation, Inc; 2001.

[2] Suter PS, Harvey LH, editors. Vision rehabilitation. Boca Raton (FL): CRC Press; 2011.

[3] Warden D. Military TBI during the Iraq and Afghanistan wars. J Head Trauma Rehabil 2006;21:398–549.

[4] Giza CC, Kutcher JS. An introduction to sports concussions. Continuum 2014;20:1545–51.

[5] Centers for Disease Control and Prevention. Available at: www.cdc.gov. Accessed June 01, 2016.

[6] Faul M, Xu L, Wald MM, et al. Traumatic brain injury in the United States: emergency department visits, hospitalizations, and deaths, 2002-2006. Atlanta (GA): National Center for Injury and Prevention and Control, Center for Disease Control and Prevention; 2010.

[7] VA/DOD, Clinical practice guidelines for management of concussion/mild traumatic brain injury. Washington, DC: Department of Veterans Affairs, Department of Defense; 2009.

[8] Humphreys I, Wood RL, Phillips CJ, et al. The costs of traumatic brain injury: a literature review. Clinicoecon Outcomes Res 2013;5:281–7.

[9] Kushner D. Mild traumatic brain injury: Toward understanding manifestations and treatment. Arch Intern Med 1998;158:1617–24.

[10] Thiagarajan P. Oculomotor rehabilitation for reading dysfunction in mild traumatic brain injury [PhD dissertation]. NYC (NY): SUNY/Optometry; 2012. Available at: https://dspace.suny-connect.suny.edu/bitstream/handle/1951/60654/Thiagarajan_thesis.pdf?sequence=1.

[11] MacFarlane MP, Glenn TC. Neurochemical cascade of concussion. Brain Inj 2015;29:139–53.

[12] Greve MW, Zink BJ. Pathophysiology of traumatic brain injury. Mt Sinai J Med 2010;76:97–104.

[13] Ciuffreda KJ, Ludlam D, Thiagarajan P. Oculomotor diagnostic protocol for the mTBI population. Optometry 2011;82:61–3.
[14] Ciuffreda KJ, Ludlam DP. Conceptual model of optometric vision care in mild traumatic brain injury. J Behav Optom 2011;22:10–2.
[15] Ciuffreda KJ, Ludlam DP, Yadav NK. Conceptual model pyramid of optometric care in mild traumatic brain injury (mTBI): a perspective. Vis Dev Rehabil 2015;2:105–8.
[16] Candler R. Some observations on orthoptic treatment following head injury. Br Orthopt J 1944;2:56–62.
[17] Rutner D, Kapoor N, Ciuffreda KJ, et al. Occurrence of ocular disease in traumatic brain injury in a selected sample: a retrospective analysis. Brain Inj 2006;20:1079–86.
[18] Ciuffreda KJ, Kapoor N, Rutner D, et al. Occurrence of oculomotor dysfunctions in acquired brain injury: a retrospective analysis. Optometry 2007;78:155–61.
[19] Thiagarajan P, Ciuffreda KJ, Ludlam DP. Vergence dysfunction in mild traumatic brain injury (mTBI): a review. Ophthalmic Physiol Opt 2011;31:456–68.
[20] Thiagarajan P, Ciuffreda KJ, Capo-Aponte JE, et al. Oculomotor rehabilitation for reading in mild traumatic brain injury (mTBI): an integrative approach. NeuroRehabilitation 2014;34:129–46.
[21] Thiagarjan P, Ciuffreda KJ. Effect of oculomotor rehabilitation on vergence responsivity in mild traumatic brain injury. J Rehabil Res Dev 2013;50:1223–40.
[22] Ciuffreda KJ, Ludlam DP, Thiagarajan P, et al. Proposed objective visual system biomarkers for traumatic brain injury. Mil Med 2014;179:1212–7.
[23] Thiagarajan P, Ciuffreda KJ. Effect of oculomotor rehabilitation on accommodative responsivity in mild traumatic brain injury. J Rehabil Res Dev 2014;51:175–91.
[24] Kapoor N, Ciuffreda KJ, Suchoff IB. Egocentric localization in patients with visual neglect. In: Suchoff IB, Ciuffreda KJ, Kapoor N, editors. Visual and vestibular consequences of acquired brain injury. Santa Ana (CA): Optometric Extension Program Foundation; 2001. p. 131–44.
[25] Ciuffreda KJ, Ludlam DP. Egocentric localization: normal and abnormal aspects. In: Suter PS, Harvey LH, editors. Vision rehabilitation. Boca Raton (FL): CRC Press; 2011. p. 193–211.
[26] Capo-Aponte JE, Urosevich TG, Walsh DV, et al. Pupillary light reflex as an objective biomarker for early identification of blast-induced mTBI. J Spine 2013;(S4):004; http://dx.doi.org/10.4172/2165-7939.
[27] Thiagarajan P, Ciuffreda KJ. Pupillary responses to light in chronic non-blast-induced mTBI. Brain Inj 2015;29:1420–5.
[28] Truong JQ, Ciuffreda KJ, Han MH, et al. Photosensitivity in mild traumatic brain injury (mTBI): a retrospective analysis. Brain Inj 2014;28:1283–7.
[29] 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;27:41–7.
[30] Yadav NK, Ciuffreda KJ. Effect of binasal occlusion (BNO) and base-in prisms on the visual-evoked potential (VEP) in mild traumatic brain injury (mTBI). Brain Inj 2014;28:1568–80.
[31] Corwin DJ, Wiebe DJ, Zonfrillo MR, et al. Vestibular deficits following youth concussion. J Pediatr 2015;166:1221–5.
[32] Suchoff IB, Kapoor N, Ciuffreda KJ, et al. The frequency of occurrence, types, and characteristics of visual field defects in acquired brain injury: a retrospective analysis. Optometry 2008;79:259–65.
[33] Lew HL, Pogoda TK, Baker E, et al. Prevalence of dual sensory impairment and its association with traumatic brain injury and blast exposure in OEF/OIF veterans. J Head Trauma Rehabil 2011;26:489–96.
[34] Available at: COVD.org. Accessed June 01, 2016.
[35] Ciuffreda KJ. The scientific basis for and efficacy of optometric vision therapy in non- strabismic accommodative and vergence disorders. Optometry 2002;73:735–62.

[36] Helmick KM, Spells CA, Malik SZ, et al. Traumatic brain injury in the US military: epidemiology and key clinical and research programs. Brain Imaging Behav 2015;9:358–66.
[37] Cockerham GC, Goodrich GL, Weichel ED, et al. Eye and vision function in traumatic brain injury. J Rehabil Res Dev 2009;46:811–8.
[38] Magone MT, Kwon E, Shin SY. Chronic visual dysfunction after traumatic brain injury. J Rehabil Res Dev 2014;51:71–80.
[39] Moroon JC, Mathyssek C, Bost J. Cerebral concussion: a historical perspective. Prog Neurol Surg 2014;28:1–13.
[40] Ventura RE, Jancuska JM, Balcer LJ, et al. Diagnostic tests for concussion: is vision part of the puzzle? J Neuroophthalmol 2015;35:73–81.
[41] Pearson BC, Armitage KR, Hormer CW, et al. Saccadometry: the possible application of latency distribution measurement for monitoring concussion. Br J Sports Med 2007;41:610–2.
[42] Available at: https://www.census.gov/population/projections/data/national/2014.html. Accessed June 01, 2016.
[43] Maleki M, Jafari Z, Zarrinkoob H, et al. Effect of aging on saccular function. Med J Islam Repub Iran 2014;28:117–9.
[44] Hsieh LC, Lin HC, Lee GS. Aging of vestibular function evaluated using correlational vestibular autorotation test. Clin Interv Aging 2014;9:1463–9.
[45] Filer W, Harris M. Falls and traumatic brain injury among older adults. NC Med J 2015;76:11–114.
[46] Arand V, Buckley JG, Scally A, et al. Postural stability changes in the elderly with cataract simulation and refractive blur. Invest Ophthalmol Vis Sci 2003;44:4670–5.
[47] Calderon-Garciduenas L, Torres-Jardon R, Kulesza RJ, et al. Air pollution and detrimental effects on children's brain: the need for a multi-disciplinary approach to the issue complexity and challenges. Front Hum Neurosci 2014;8:1–7.
[48] Chen JC, Wang X, Wellenius GA, et al. Ambient air pollution and neurotoxicity on brain structure: evidence from women's health initiative memory study. Ann Neurol 2015;78:466–70.
[49] Tannen B, Darner R, Ciuffreda KJ, et al. Vision and reading deficits in post-concussion patients: a retrospective analysis. Vis Dev Rehabil 2015;1(3):206–13.
[50] Master CL, Scheiman M, Gallaway M, et al. Vision diagnoses are common after concussion in adolescents. Clin Pediatr (Phila) 2016;55(3):260–7.
[51] Master CL, Gioia GA, Leddy JJ, et al. Importance of 'return-to-learn' in pediatric and adolescent concussion. Pediatr Ann 2012;41:1–9.
[52] Ciuffreda KJ, Ludlam DP, Kapoor N. Clinical oculomotor training in traumatic brain injury. Optom Vis Dev 2009;40:16–23.
[53] Tulsky DS, Kisala PA, Victorson D, et al. TBI-QOL: Development and calibration of item banks to measure patient reported outcomes following traumatic brain injury. J Head Trauma Rehabil 2016;31:40–51.
[54] Davies RC, Williams WH, Hinder D, et al. Self-reported traumatic brain injury and post-concussion symptoms in incarcerated youth. J Head Trauma Rehabil 2012;27:E21–7.
[55] Farrer TJ, Frost RB, Hedges DW. Prevalence of traumatic brain injury in intimate partner violence offenders compared to the general population: a meta-analysis. Trauma Violence Abuse 2012;13:77–82.
[56] Donders J, Strom D. The effect of traumatic brain injury on children with learning disability. Pediatr Rehabil 1997;1:179–84.
[57] Williams WH, Mewse AJ, Tonks J, et al. Traumatic brain injury in a prison population: prevalence and risk for re-offending. Brain Inj 2010;24:1184–8.
[58] Singman EL. Automating the assessment of visual dysfunction after traumatic brain injury. Med Intsrum 2013;1:3.
[59] Capo-Aponte JE, Tarbett AK, Urosevich TG, et al. Effectiveness of computerized oculomotor vision screening in a military population. J Rehabil Res Dev 2012;49:1377–98.