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Vision Concerns After Mild Traumatic Brain Injury

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Opinion statement

Mild traumatic brain injury (mTBI) can manifest with visual dysfunction including deficits in accommodation, vergence movements, versions, and field of vision as well increased photosensitivity and a decline in ocular and overall health. Patients with incomitant strabismus should be referred to an ophthalmologist for intervention. Patients with mTBI who experience photosensitivity, or deficits in accommodation, versions, vergences, or field of vision may benefit from vision rehabilitation. These therapies may include spectacles with tinting and a variety of prism combinations. Patients with chronic visual dysfunction following mTBI may benefit from occupational, vestibular, cognitive, and other forms of physical therapy.

Introduction

Concussion or mild traumatic brain injury (mTBI) commonly results in both acute and chronic visual sequelae [1]. This is likely explained by the fact that a great deal of the brain is dedicated to vision [2]. Visual dysfunction in mTBI can present in a variety of ways [3]. To understand visual dysfunction in mTBI, it is important to understand the mechanism of injury. Injury during mTBI may result from mild direct head trauma or through indirect rotational forces applied to the brain. These mechanical forces can result in stretching of white matter axons and diffuse axonal injury [1, 4]. The damage in mTBI largely occurs at the cellular and subcellular level and is not accompanied by hemorrhage or other abnormalities detectable on head computed tomography (CT) or routine magnetic resonance imaging (MRI) [5••]. The severity of postconcussion cognitive problems has been shown to correlate with the extent of white matter abnormalities on diffusion tensor MRI (DTI) though typically these advanced techniques are not applied to the postconcussion workup [3].

After experiencing mTBI, patients may experience rapid-onset neurophysiological changes. Although most patients demonstrate resolution, approximately 15 % will suffer more lasting deficits [6, 7•]. Animal models

of mTBI have demonstrated that axonal disconnection rarely occurs at the time of injury; rather, axonal stretching leads to an unregulated flux in ion concentrations, which in turn causes a net increase in intra-axonal calcium concentrations [8]. This increase in calcium can activate the protease calpain to facilitate cytoskeletal protein proteolysis including irreversible axonal pathology such as progressive microtubule disassembly [9]. Increased intra-axonal calcium concentration can also stimulate glutamate release and subsequent glutamate-mediated activation of N-methyl-D-aspartate receptors, resulting in inexorable depolarization of neurons [10].

As cells attempt to return to homeostasis through active transport mechanisms, cellular metabolism and glucose consumption are increased to supply the energy demand of various membrane pumps. Such over-activation can result in depleted energy stores, calcium influx into the mitochondria, impaired oxidative metabolism, increased glycolysis with lactate production and ultimately local acidosis and edema [11]. At a critical threshold, axonal swelling becomes so severe that it causes the axons to disconnect in a process known as secondary axotomy [11]. Magnetic resonance spectroscopy, and neuropsychological and electrophysiological data from patients with mTBI demonstrate that the majority of patients return to baseline after 30–45 days [12], whereas in certain patients, secondary axonopathy might continue for years after TBI [13•]. It is important to note that age also plays a profound role in predicting long-term sequelae from mTBI. The developing brain is more vulnerable to concussive forces than the adult brain because of differences in blood-brain barrier integrity, degree of mylenation, and elastic properties [14, 15]. Furthermore, older adult patients may be less tolerant of mTBI.

Visual dysfunction after mTBI can disrupt every aspect of vision including acuity, accommodation, ductions, eye teaming, visual field, photosensitivity, color perception, contrast sensitivity, pupillary function, saccade production, visual memory, reading comprehension, and visual recognition. Different problems stem from damage to the afferent (incoming) visual pathways, efferent (outgoing) visual pathway, and visual association areas (Fig. 1).

In this review, we will describe the acute and chronic visual sequelae of traumatic brain injury (TBI) as well as the current understanding of the underlying pathophysiological causes of such dysfunction. Further attention will then be given to techniques used clinically to identify mTBI visual dysfunction, current treatment options and future treatment prospects. Notably, we will limit our discussion to mild brain injury and not include a discussion of the diagnosis and treatment of direct ocular or orbital trauma.

Afferent visual dysfunction

Acuity, Color, Contrast Deficits

Afferent visual dysfunction after mTBI can present as a decline in visual acuity, contrast sensitivity, and color vision. In most instances, mTBI induces visual deficits that are bilateral. Exceptions to this include traumatic optic neuropathy as well as direct ocular or orbital trauma. Direct trauma can commonly be diagnosed with standard ophthalmoscopic techniques. Retro-bulbar trauma, that is trauma behind the globe but within the orbit, can present in a more subtle way. The clinician should have a high index of suspicion for retro-bulbar trauma in patients who develop new onset proptosis, ptosis, or reduced color vision, visual field, and acuity following a mTBI. In the acute setting, it is imperative to obtain a CT scan that includes the orbits to rule out retro-bulbar hemorrhage. In instances in which a CT scan is nonrevealing, it is important to assess for decreases in visual acuity as well as decline in color discrimination and contrast sensitivity as all can occur in traumatic optic neuropathy [1]. Although nonophthalmologic practitioners including first responders should attempt to assess for these vision changes, a decline in visual acuity after an mTBI should prompt a referral to an ophthalmologist.

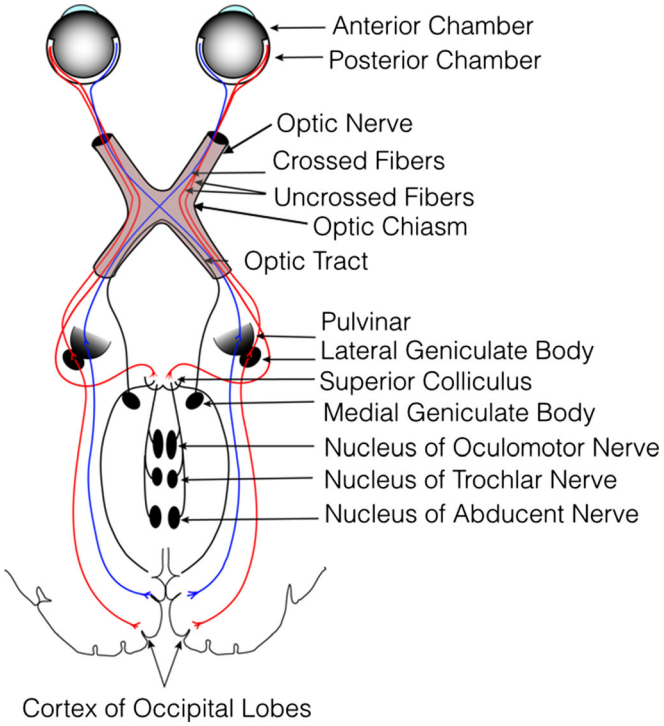

Figure 1. Potential areas of damage with mTBI include any area of the visual system as diagrammed above. The visual system is largely conserved among mammals and includes the eyes including the anterior and posterior chamber, the pathways connecting the retina to the visual cortex and other parts of brain responsible for the oculomotor and nonoculmotor components of vision.

Visual field defects

Visual field defects after mTBI can occur through trauma to the optic nerve, chiasm, optic radiations, or occipital cortex [1]. In these instances, confrontational visual fields can often detect severe defects. Automated perimetry is better suited to detect less obvious visual field defects and to monitor changes over time.

Visual attention deficits

Patients experiencing mTBI may demonstrate visuospatial attention deficits [4]. Visuospatial attention requires alerting and orienting to a visual target followed by visual discrimination and higher order decision making. Deficits can be detected through use of the validated Attention Network Test [16]. During this testing, patients are found to have slowed response to visual cues, a reduced ability to resolve visual conflicts, and an inability to disengage visual attention from one visual target to the next [17].

Visual midline shift

A sense of shifted egocenter after mTBI has been reported and named the visual midline shift syndrome (VMSS) [18]. Here, patients perceive a warping of their visual world so that objects to one side appear abnormally close while objects to the other side appear abnormally remote. This misinterpretation of one's egocenter is thought to be secondary to an abnormal imbalance between the visual pathways supporting detail and those supporting awareness of the overall environment. VMSS can be detected in the clinic by asking a patient to report

when a vertically held object is directly in front of their visual field and an observer determines that the object is being held to one side. VMSS reportedly creates changes in balance and posture that can be detected on pressure sensing treadmills [19]. Changes in cortical signaling in VMSS have been reported through visual evoked potentials [20]. At present, the concept of VMSS needs further research for confirmation as a nosologic entity. However, therapists who diagnose this condition treat it with yoked prisms to shift the patient's sense of midline back toward the patient's true center.

Efferent visual defects

Accommodation Deficits

Patients suffering mTBI may develop deficits in accommodation. These patients can present with both difficulty focusing for near vision and for defocusing for distance vision. In certain instances, mTBI may induce over-activation of the accommodative pathways thereby creating a pseudo-myopia [21]. The underlying mechanism of this spasmodic accommodation remains unclear [3].

Nystagmus

Following mTBI patients may develop nystagmus or rhythmic beating of eyes. Their chief complaint might be oscillopsia. Nystagmus can be clinically detected through visual inspection. Videonystagmography can be employed [22] to quantitate and qualify the various types of nystagmus as well as to detect subtle findings such a torsional nystagmus. Because nystagmus usually involves the labyrinthine apparatus, patients are probably best served by neuro-otologists.

Pursuits

Deficiency of pursuits, a reduction in the ability for the eyes to follow a moving target, and rapid focus at an anticipated location are reportedly common after mTBI [23]. Such dysfunction can result from damage to the oculo-motor pathways. Patients might complain of blurred or double vision. Detection of deficiencies of smooth pursuits may be one of the most sensitive biomarkers for screening patients with suspected mTBI [24]. Although gross defects can be detected by a skilled practitioner, more subtle pursuit abnormalities are better detected by automated eye tracking [25••].

Saccades

Saccades, or rapid re-fixation movements, can be altered after mTBI. Patients may have difficulty releasing attention from one visual target and/or have difficulty locking onto a novel stimulus [25••]. Patients may also demonstrate the inability to inhibit the saccadic system and, thus, be susceptible to visual distraction [26]. Furthermore, patients may demonstrate slowed saccades and ones that undershoot a novel visual target [27]. These hypometric saccades can

be demonstrated with automated equipment [24], which can be used for monitoring therapeutic efficacy post mTBI.

Strabismus

Mild TBI can lead to worsening of pre-existing heterophorias so that they can become manifest heterotropias [28]. In addition, frank strabismus with subjective diplopia can occur after mTBI from isolated or multiple palsies to the 3rd, 4th and 6th cranial nerves [29]. In instances in which the defect is subtle or mTBI has resulted in a unilateral decline in visual acuity, the defect may go unnoticed or may appear to the patient as simply blurring of vision. Blurred vision is especially common if the defect is torsional rather than vertical or horizontal. These subtle defects can be measured with synoptophores and other automated devices for measuring heterophorias [30].

Stereopsis

Reduction in stereopsis, the ability of the brain to judge visual depth by merging the slightly disparate images from each eye, is commonly affected after mTBI [31]. Reduction in stereopsis may occur with strabismus. However, even in the absence of extra-ocular motility defects, damage to the superior colliculus and other areas of the midbrain during mTBI may cause dysfunction in stereopsis [31]. This defect in stereopsis can occur both at distance and near or in some instances may just manifest with objects closer than 6 feet because of inability of the eyes to rotate inward or converge. Convergence insufficiency as well as defects in stereopsis can be quantified with great sensitivity by available tests [29].

Pupillary reaction

Anisocoria and slowed pupillary responses can occur in instances of markedly increased intracranial pressure (ICP), a common problem after severe TBI. Interestingly, it has been reported that patients suffering only mTBI (when ICP is usually normal) not only demonstrate slowed pupil responses but abnormally small excursions of the pupillary sphincter. In fact, automated pupillometry is now being explored as a way of screening for mTBI [32•]. Pupillary abnormalities can also occur in mTBI when traumatic optic neuropathy results [33]. Usually, this condition will be heralded by an afferent pupillary defect in the affected eye as well as reduced visual acuity and visual field.

Higher order deficits

Higher order deficits involve dysfunction in the visual pathways described above (Fig. 1) as well as the nonvisual portions of the brain. These higher order

deficits can manifest in a number of ways including abnormal sensitivity to glare, reading difficulties, decreased visual reaction times, and short- and long-term memory impairment.

Sensitivity to glare

After mTBI patients commonly complain of photophobia and more specifically increased sensitivity to glare [34]. Although an area of active inquiry, some research suggests that deficits in dark adaptation are responsible for this phenomenon [35].

Reading deficits

It seems reasonable to believe that the saccadic dysfunction and difficulty with convergence often associated with mTBI might have an impact on reading, particularly transitioning from one line of text to the next [36]. However, patients also often report losing their place while reading or having difficulty to processing visual information [37] and paraphrasing after mTBI [38, 39], suggesting more than just visuomotor causes for reading deficits. A rapid test of reading speed is the King-Devick test, an automated test validated for identifying concussion in athletes [40].

Reaction time

After mTBI, patients have decreased reaction time to not only visual but also auditory and tactile stimuli [41]. These deficits in reaction to visual stimuli can be measured in an automated fashion using software that was first utilized for monitoring visual dysfunction in children with lead poisoning [42].

Memory impairment

Patients with mTBI may show both acute and chronic memory impairment. In the acute setting, patients with mTBI may not be oriented to person, place, and time [43]. Chronic disorientation often is indicative of more severe TBI. For this reason, the Rapid Screen of Concussion utilizes orientation to differentiate between mild and severe TBI [44]. Subtle memory deficits in mTBI can be detected with automated tests of episodic memory [45].

Therapy

Patients with noncomitant strabismus and complex diplopia patterns are best treated under the auspice of a neuro-ophthalmologist who may guide medical and surgical intervention. Dysfunction in vergence, version, and accommodation are best corrected with orthoptic therapy [46]. When using these techniques in the mTBI population, it is important to remember that such patients

may have additional difficulties secondary to mTBI including induced memory problems, fatigue, depression, and overall physical health.

A particular form of visual therapy, oculomotor vision rehabilitation (OVR), has been offered for treating some oculomotor deficits that commonly occur in the setting of mTBI [36]. OVR utilizes motor training combined with attention training to help mTBI patients overcome visual deficits [47]. Optometrists providing OVR train patients to optimize vergence, accommodation, fixation, and saccades to overcome visual deficits [47]. This is achieved through standardized protocols that present visual targets in a sequence that utilizes motor and perceptual memory as well as optimizes attention in order for patients to maintain binocular vision [48]. This training enables a patient to have a heightened attention to the manifestation of the visual deficit, such as the blurring of an image, and a corresponding motor response that compensates for the deficit. With repetition, this correction becomes reflexive. The effects of OVR on mTBI induced visual dysfunction have been proven to be effective in the laboratory and have proven to have provided robust visual correction in the clinical setting [49]. At the current stage, many of these paradigms are performed with automated, semiautomated, or manual visual stimuli. Manual visual stimuli can be created with the Brock string, polarized hectograms, and anaglyphs used in conjunction with hand-held prisms [49]. Computer based automated targets are available as well [49]. It is likely that in the not too distant future, fully automated systems may be commercially available or alternatively available through open source development [50].

Vergence dysfunction

Standard procedure Oculomotor vision rehabilitation can be beneficial in reducing nonstrabismic vergence dysfunction after mTBI. The goal of visual therapy for vergence dysfunction is to enable the patient to develop adequate fusional reserve in order to maintain binocular vision at both far and near for a period of at least 20 min [49]. During therapy, a patient is presented binocular visual stimuli in a sequential fashion. The visual targets are altered based upon ramp or step, thereby altering, respectively, the vergence or divergence necessary for binocular vision. Initially, small disparity steps are introduced until the patient reports diplopia. Step disparity is then reduced until the diplopia resolves. This exercise is typically performed sequentially. In a similar fashion, small ramp disparities are introduced sequentially until the disparity overcomes the patient's fusional facility, and diplopia is experienced. This disparity is then reduced until the patient is able to adequately fuse. This paradigm is then repeated sequentially [49]. Initially, both ramp and step paradigms are used at various distances (both near and far). When particular target distances produce the greatest visual deficit, these distances should be employed repeatedly for OVR to achieve the greatest benefit. In addition to small step training, large disparity steps may be employed to enhance the patient's fusional vergence. Similarly, large disparity steps can be introduced with opposing accommodative demands in order to optimize fusional facility. After sequential step and ramp training, patients are then asked to sustain vergence at different disparity demands [49].

Contraindications Patients with vestibular dysfunction, especially those who require large magnitude fusional prisms (<10 diopters), may experience motion sickness with strabismus visual therapy. This is due to the increased motion sensitivity inherent in vestibular dysfunction as well as the nonuniform magnification of

	the visual field that occurs with head motion with fusional prisms. Other patients who may be poor candidates for strabismus visual therapy include patients with a comitant strabismus that does not superimpose with use of neutralizing prism. Patients with a comitant strabismus that variably superimposes with prism and finally patients with discordant visual acuity of greater than two lines in best corrected vision between each eye also may not be suitable candidates [49].
Complications	None.
Special points	Patients who have vergence dysfunction as well as a component of vestibular dysfunction may benefit from the use of visual biofeedback with or without overlay prisms [49].
Cost/cost effectiveness	Cost of therapy is variable depending on what percentage of therapy is done in the office and what degree at home by the patient. Therapy is often not covered by medical insurance. Therapy may return a patient to the workforce and, therefore, can have long-term financial benefits for the patient and society at large [49]. Computerized orthoptics programs for home use cost $150 (for example, see www.visiontherapysolutions.net) and has reported to be very effective [51]. In-office therapy can cost the patient and/or their insurance approximately $145 per session with an expectation that sessions occur weekly for 3–6 months (total $1700–$3400); many providers offer some discount if patients pay for multiple sessions in advance. Office-based therapy for convergence insufficiency has been reported to be more effective than home-based computerized exercises [52].

Strabismus

Standard procedure	Strabismus can be treated through surgical and nonsurgical means [53, 54••]. The ultimate goal of therapy is to provide binocular vision at both far and near with good stereopsis or at least to eliminate diplopia. When first evaluating the strabismus of a post mTBI patient, it is important to establish the chronicity and stability of the strabismus and to determine if the strabismus is intermittent or constant. Further, it is important to determine whether the strabismus can be fully suppressed by the patient and is comitant. If a patient can fuse (the synoptophore is particularly helpful for this assessment), prisms, surgery, and and/or vergence training can be applied depending upon the degree of strabismus. There are many surgical procedures to correct strabismus; they involve altering the location of the extra-ocular muscles to relatively weaken or strengthen their effect on the globe so as to allow the globe to reposition.
Contraindications	Patients would not be considered surgical candidates if they posed an unacceptable risk to bleeding, infection, or exposure to anesthesia.
Complications	None for nonsurgical therapy. Surgical therapy may over- or under correct, requiring second procedures.
Special points	Same special points as vergence training listed above.
Cost/cost effectiveness	Surgical therapy has been shown to be highly effective [53, 55].

Deficits of pursuits

Standard procedure The goal of OVR in patients who have deficits in pursuit is to decrease symptoms when tracking a target in motion while maintaining conjugate gaze. A deficit in pursuit should be established by first holding an object at arm's length from the patient and sequentially moving the object from midline to a horizontal position away from midline. A similar test should be done with target excursions in the vertical dimension. In patients with pursuit deficit, one eye may track at a different speed than then contralateral eye, leading to disconjugate gaze. In other instances, the patient may have to employ saccades to track a visual target. In such patients, a visual target should be slowly moved horizontally from midline to ±5 degrees from midline repeatedly followed by a break. This should then be repeated with ±10 degree excursions of the target. Following horizontal excursion, this exercise should be repeated in a similar fashion with vertical excursions of ±5 degree followed by ±10 degrees. In further iterations of the therapeutic paradigm, both target distance and velocity can be altered to further challenge smooth pursuits in patients [36, 37].

Contraindications The above therapy may prove problematic in patients who have increased motion sensitivity or manifest-latent nystagmus, as it may induce nausea in the former and oscillopsia in the latter.

Complications Vision therapy to improve pursuits may be limited in patients with asymmetric visual acuity between the two eyes as similar acuity is necessary for neurosensory bifixational accuracy. It may further be hindered by oculomotor paresis or paralysis as well as impaired fusional vergence.

Special points Patients may be aided in pursuit training by the use of oculomotor auditory feedback as well as foveal visual feedback such as afterimages. These features can be incorporated into a computer based training system [36, 37].

Cost/cost effectiveness Same cost/cost effectiveness of vergence training listed above.

Deficits of saccades

Standard procedure Patients should first be evaluated for saccadic deficits by presenting visual targets in the horizontal, vertical, and oblique directions at excursions of ±10 and ±20 degrees from midline. The exercises should be performed both monocularly and binocularly. A standard paradigm involves presentation of targets sequentially in the four quadrants (up, right, down, left) ±20 degrees from midline. This is repeated at least 10 times and then targets are adjusted to ±10 degrees from midline and a similar repetition is undertaken. Visual targets should first be presented at both distance and near but with time training can be focused on the distance of greatest symptomatology. In addition, excursions less than 10 degrees can be utilized to increase the difficulty level. Automated software can be used to present horizontal, vertical, and oblique steps to enhance saccadic accuracy.

Contraindications Same contraindications as pursuit training listed above.

Complications Same complications as pursuit training listed above.

Special points Same special points as pursuit training listed above.

Cost/cost effectiveness	Same cost/cost effectiveness as vergence training listed above.

Accommodative dysfunction

Standard procedure	The goal of accommodative training is to decrease the patient's symptoms of intermittent blur by improving accommodative amplitude and accuracy. Training incorporates both ramp training in which the accommodative demand is gradually increased as well as step training in which the accommodative demand is rapidly increased. Training should be performed first monocularly, then with optically-imposed vertical diplopia, and then finally binocularly. Target size should be gradually decreased to increase task difficulty as patient's accommodative function improves. For ramp accommodation exercises, a target is brought from arm's length slowly toward the patient until blur occurs. The object is then moved back to arm's length, and the exercise is repeated. Step accommodation should begin by having a patient look at a target 10 feet away for 3 s and then rapidly shift attention to a target 16 inches away for 3 s. Over time, the near target is progressively moved closer to the patient. The exercises can also be performed with overlaying lenses. A concave lens can be introduced, followed by a convex lens to respectively increase and decrease the accommodative stimulus. At subsequent training sessions, the dioptric power of the lenses can be increased to create a greater accommodative demand. If the accommodative deficit is binocular and similar, another therapeutic option would be increased reading power in bifocal lenses.
Contraindications	Training in patients with vestibular dysfunction should be performed with caution as the shift in the accommodative state may disorient and nauseate.
Complications	None.
Special points	Same special points as vergence training listed above.
Cost/cost effectiveness	Same cost/cost effectiveness as vergence training listed above.

Visual field defects

Standard procedure	Humphrey visual fields or an alternate form of automated perimetry should be utilized to map the deficits. Therapy is aimed at increasing awareness of the affected field in order for patients to compensate by scanning routinely into the area of deficit. Certain patient's may also benefit from the use of yoked prisms or field enhancing prisms such as the Fresnel or Peli prisms [56]. Training should involve manual or automated presentation of visual targets in the area of known deficit as well as in the unaffected visual field. Automated programs exist that reportedly expand the visual field through true improvement of cortical function rather than by simply training a patient to be more aware of the missing field or move their eyes more toward the missing field [57].
Contraindications	Same contraindications as vergence training listed above.
Complications	None.
Special points	Patients may benefit from functional visual field software that increases awareness in the affected field that may be used in the clinic and at home by the patient alike [56, 57].

Cost/cost effectiveness True visual field expansion with computerized training systems (for example, see www.novavision.com) is reported to be on the order of a few degrees. The cost to the patient and/or insurance for these systems is approximately $3000 for rental of the computer and software. Peli prisms do not increase the visual field but are rather effective in increasing awareness of the field [56]. The cost to the patient for materials and in-office training sessions is approximately $500 (for example, see www.hemianopsia.org).

Photophobia

Standard procedure Photophobia in patients after mTBI is well documented and typically occurs in absence of ocular inflammation [34]. The use of light-filtering lenses has proven beneficial in not only decreasing symptomatic photosensitivity but also increasing the contrast sensitivity and reading rates of individuals after mTBI [58]. Generally, lenses that block blue-green, such as FL-41 lenses, are of the greatest utility in this setting [34].

Contraindications In certain photophobic patients, light-filtering lenses do not improve visual function. Contrast sensitivity testing and assessment of reading rate in the presence and absence of light-filtering lenses is useful to best identify the optimal light-filtering lens and identify patients who do not improve with such lenses.

Complications None.

Special points Patients may benefit from photochromic lenses to provide differing levels of filtering for indoor and outdoor use. Alternatively they can have a separate set of glasses for indoor and outdoor wear or clip-on tinted lenses that can be applied to the tinted glasses optimized for indoor wear.

Cost/cost effectiveness The addition of tint, photochromic, or static, to a standard eye glass prescription may range in cost everywhere from $10 to $100 per lens. Insurance commonly covers the cost of tinted lenses and even additional clip-on lenses if the necessity of such lenses is documented as secondary to TBI.

Deficits of fixation

Standard procedure At the completion of fixation training, patients ideally can maintain steady fixation at targets midline as well as ±20 degrees vertically and ±20 degrees horizontally. After mTBI, it is not uncommon for patients to display gaze instability, slow drift, and even jerk nystagmus when presented with visual targets. At the beginning of training, patients should fixate on a target at midline for 3 s and then told to close eye for 3 s. The target should sequentially be moved ±20 degrees vertically and ±20 degrees horizontally with 3-s rest periods in between each target. As fixation improves, the required times of fixation can be increased to 10 s or more, and the size of the target can be reduced from large 5-degree targets down to small 1-degree targets. These exercises can be performed monocularly as well as binocularly and should be performed at both distance and at arm's length.

Contraindications Same contraindications as pursuit training listed above.

Complications Same complications as pursuit training listed above.

Special points	Same special points as pursuit training listed above.
Cost/cost effectiveness	Same cost/cost effectiveness as vergence training listed above.

Compliance with Ethics Guidelines

Conflict of Interest

Brad P. Barnett and Eric L. Singman declare that they have no conflicts of interest.

Human and Animal Rights and Informed Consent

This article does not contain any studies with human or animal subjects performed by any of the authors.

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