REVIEW ARTICLE

The Role of Neuroplasticity in the Neuro-Optometric Rehabilitation of Traumatic Brain Injury

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ABSTRACT

Athletic communities have begun to take precautions to mitigate traumatic brain injury, such as using protective gear, increasing medical oversight, and establishing better return-to-play rules to protect athletes. As awareness increases, there will be a correspondingly higher frequency of people with traumatic brain injury seeking neuro-optometric evaluation and treatment. Therefore, it is imperative for optometrists to understand the signs, symptoms, and treatment of associated visual sequelae of traumatic brain injury. There are many optometric conditions caused by traumatic brain injury, including, but not limited to: convergence insufficiency, accommodative insufficiency, oculomotor dysfunction, visual motion sensitivity and light sensitivity. In particular, the functional vision problems associated with traumatic brain injury can be addressed with neuro-optometric rehabilitation therapy. They are often due to diffuse axonal injury throughout the brain caused by shearing and tearing of the axons. People with diffuse axonal injury tend to have slower processing speeds due to impaired function at the synapse and longer neuronal pathways. Neuro-optometric rehabilitation therapy creates an environment to facilitate neuroplastic changes within the brain, such as axonal sprouting or dendritic plasticity. This article explores possible correlation between improved clinical findings in traumatic brain injury after neuro-optometric rehabilitation therapy and increased neurophysiological changes in brain activity after vision therapy for convergence insufficiency.

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INTRODUCTION

Traumatic brain injury (TBI) is becoming increasingly recognized and addressed, particularly in the athletic community where new safety protocols are being implemented to protect athletes from concussion. The improved awareness is reflected by an increased number of patients seeking care. For example, on the 2014 surveillance report, the Center for Disease Control and Prevention (CDC) reported 2.87 million new cases of traumatic brain injury coming into the emergency department per year, which is a 54% increase since 2006.[1] The significant number of cases in the US creates an increased demand for healthcare. Optometrists can contribute to the need by identifying and treating the visual sequelae of traumatic brain injury.

Traumatic brain injury can be categorized in various ways: mild, moderate, or severe; penetrating or closed-head; focal or diffuse.[2]

This paper will focus on focal and diffuse traumatic brain injury and how neuro-optometric rehabilitation therapy can play a role in the patient's recovery. Although focal and diffuse traumatic brain injury are categorized separately, most traumatic brain injuries are not simply one type and will encompass both focal and diffuse damage.[3] This can be illustrated with the classic example of traumatic brain injury: the coup-contrecoup injury. The specifics of diffuse and focal traumatic brain injury will be discussed in detail later.

Coup-contrecoup Injury

Coup-contrecoup injuries occur when the brain sustains damage from the inner wall of the skull on the same and opposite side as the head collision with a hard surface.[2] The acceleration-deceleration forces (defined as forces causing a change in velocity over time) on the head cause a discrepancy in the coordination of motion with the brain.[4] The brain has a tendency to be inert for longer, thus will collide with the skull when the head moves faster in a direction than it does.[4] Coup-contrecoup injuries may occur when a moving object hits a stationary person, as in some cases of assault or in sports, or when a moving person hits a stationary or moving object, as in sports, car accidents and falls. The greater the acceleration-deceleration force is, the higher the probability and severity of neurologic injury.[4] Depending on the state of the person (moving or stationary), the external force will cause the head to accelerate away from impact or decelerate as it impacts. For example, in American football, a player that is attempting a tackle is moving forward until they collide with the opposing team member. At that point, the head and body of the player quickly decelerates, and the brain strikes the anterior skull. This initial impact and compression of the brain at the site of impact is the coup component of a coup-contrecoup injury and is considered a focal lesion.[2] The contrecoup component is when the brain "rebounds", causing the brain tissue to collide with the opposite side of the skull.[4] Again, the compression of the brain and the sustained damage on the opposite side is also a focal lesion.[2] Meanwhile, diffuse traumatic brain injury can be caused by two types of forces: linear acceleration-deceleration forces, which is in the direction of the force of impact, and rotational forces, which are changes to the direction of the acceleration-deceleration vector.[3,4] When the brain is ricocheted within the skull during a coup-contrecoup injury, diffuse axonal and vascular damage can occur because the brain tissue has different consistencies and strength of connection to other areas of the brain and skull base.[2] When the brain has the effect of linear acceleration-deceleration or rotational forces upon it, certain segments will move slower and cause shearing and tearing damage to the axons and vasculature.[2]

Whiplash-type Injury

Note that traumatic brain injury does not have to be caused by actual impact with some object, structure, or person, although that is the case in most circumstances. A whiplash-type injury may occur due to acceleration-deceleration forces without impact, for instance, shaken baby syndrome.[4] The same concepts of coup-contrecoup injuries apply except the forces are due to shaking and not impact.[4] Whiplash-type injuries are especially susceptible to diffuse-type damage particularly because of the directional inconsistency of the acceleration-deceleration and rotational forces applied on the head and neck.[4]

Focal Traumatic Brain Injury

Focal traumatic brain injury is caused by a mechanical insult to the brain localized in one specific area.[3] Focal lesions include skull fractures, contusions, lacerations, hemorrhages, and hematomas; explanations of each of these are as follows.[3] Skull fractures are a break in the skull, usually from a considerable force, and often do not coincide with serious brain injury.[3] Contusions and lacerations are formed when the brain scrapes against the rough skull walls,

causing damage to the small blood vessels which form small hemorrhages perpendicular to the skull wall.[3] The main locations for focal contusions are the inferior aspect of the frontal lobes, the frontal poles and the inferior aspect of the temporal lobes where the brain comes in contact with bony protuberances.[3] Focal hemorrhages and hematomas are moderate to large bleeds occurring from tearing of blood vessels upon initial head impact.[3] Brain imaging should be considered to rule out any hematomas. Focal traumatic brain injury will affect whichever area has been damaged and the symptoms will be specific to that area's function. For example, if the frontal lobe was damaged in a coup-contrecoup injury, the patient may have difficulties with executive functioning such as planning, voluntary movement and/or impulse control. If the frontal eye fields specifically were compromised, the patient will show deficits in vergence, fixation, saccadic and smooth pursuit control.

Diffuse Traumatic Brain Injury

Diffuse traumatic brain injury occurs when a large area of the brain is affected after impact from external forces.[3] Diffuse lesions include diffuse vascular injury, diffuse brain swelling, excitotoxicity and oxidative stress, and diffuse axonal injury.[3] Diffuse vascular injury occurs when acceleration-deceleration forces shear the capillaries and form many small, petechial hemorrhages.[3] It is common in severe traumatic brain injury.[3] Diffuse brain swelling from edema and vascular congestion will frequently distort, shift or herniate the brain and cause increased intracranial pressure.[3] Excitotoxicity and oxidative stress are evidence of metabolic disruption.[3] Diffuse axonal injury, the most common of the diffuse lesions, is caused by linear acceleration-deceleration forces and rotational forces resulting in shearing and tearing damage to axons particularly in the white matter fiber tracts of the brain.[2,3] In a general sense, diffuse axonal injury will reduce the strength, number and organization of the synapses. It will also reduce the synchrony, firing rates and neuronal dynamics.[5] Which in turn lead to slow responsivity, increased response variability and reduced response accuracy for the patient.[5]

The widespread damage associated with diffuse axonal injury will affect vision via the two information processing streams in the brain: the ventral stream and the dorsal stream.[6] It may also affect the inter-connectivity between the dorsal/ventral streams and subcortical structures.[6] Information is carried through the brain along the ventral stream, which encodes object recognition (colors, shape and texture of objects), and the dorsal stream, which is involved in understanding visual space, motion, perceiving depth (through binocular disparity), discrimination within complex motion situations, and grasping and manipulating objects.[6]

Signs and symptoms of diffuse axonal injury tend to visually manifest as a deficiency of dorsal stream processing because the dorsal stream is so intricately involved with visuo-motor control.[7] These signs and symptoms will be identified during the neuro-optometric evaluation and may present as a top-down processing deficit, a bottom-up processing deficit or a combination of both.[6] Bottom-up processing is the feedforward neuronal projections from lower-order to higher-order cortical areas, while top-down processing is the influence of higher-order cognition and attention on a visual pathway through feedback projections.[6,8] When there is a problem with bottom-up processing, sensory-driven visual functions are affected.[6] For example, the ability to converge to a target moving closer in near point of convergence (NPC) testing can be reduced if the brain does not interpret or respond to retinal disparity properly, leading to under-convergence. Whereas a problem with top-down processing can be elicited with testing that includes attention or planning.[8] For example, as an adjunct to NPC, the clinician can test the patient's ability to regrasp convergence at their recovery distance by doing a test called "reach, grasp, release, regrasp" (RGRR). This

test will measure the patient's break (reach) and recovery (grasp) points in NPC, then have the patient look at a point further than the recovery point (release), effectively diverging their eyes. Then the patient will have to incorporate a planning phase to converge back to the recovery point (regrasp) since there is not a stimulus to follow inwards from the release point. The cognition involved in calculating the distance to the target and generating a voluntary convergence response is indicative of the top-down processing component in this action. If there is no top-down processing deficit, the patient will accurately converge back to their recovery point within one second. If there is a top-down processing deficit, the patient will either take longer than one second to converge to the recovery point or fail entirely to converge to that distance and their regrasp point will be further receded.

Lesions within the dorsal stream may affect the individual's ability to match the speed of a moving target in pursuits, adjusting the amplitude of saccades to a moving target, cause difficulty discriminating a moving target within an array of other moving targets, and cause difficulty understanding and interacting within their space world.[6] Reduced vergence recoveries, receded near point of convergence, oculomotor dysfunction, accommodative infacility and insufficiency, delayed responses to commands, delayed processing speeds and difficulty understanding instructions are common with this type of injury.[8,9,10] For example, a patient may present with a receded NPC with a slow RGRR, undershooting on saccadic testing and a lag in response time (poor motor match) when the rate of saccades is varied. They complain of double vision at near, missing the mug when pouring coffee and losing their place while reading. The slow RGRR and poor motor match indicate a top-down processing deficit and the patient's difficulty interacting accurately with their space-world indicates a dorsal steam processing deficit. See Table 1 for a summary of the most common signs and symptoms associated with traumatic brain injury. Look for a top-down processing component during testing. It may present as motor overflow, a lag in response time, severely receded findings, or inattention.

Some of the most common treatable issues and symptoms reported during a neuro-optometric exam are those associated with diffuse axonal injury. Therefore, it is important to understand how the patient would present and what type of treatments we can offer. We can treat these conditions with a multi-tiered treatment protocol, including an accurate refractive analysis with binocular balance, tints/chromatic filters, optometric phototherapy, prisms (compensatory and yoked), selective occlusion, and neuro-optometric rehabilitation therapy. Neuro-optometric rehabilitation therapy is based on principles of neuroplasticity and is designed to specifically help people with brain injury regain some or all of their previous levels of visual functionality. This paper will discuss the neuroscience concepts supporting neuro-optometric rehabilitation therapy and how neuroplasticity is incorporated in our treatment protocols.

Neuro-Optometric Rehabilitation: A Way to Treat Concussion

The goal of neuro-optometric rehabilitation therapy is to re-calibrate brain processing for performing tasks that were negatively affected after a brain injury. It does this by utilizing a sequence of procedures individualized for each patient based on findings from a comprehensive neuro-optometric exam. The basic structure of neuro-optometric rehabilitation therapy is broken down into three phases. Phase 1 is focused on rehabilitating accommodative and oculomotor skills through bottom-up processing. Phase 2 rehabilitates vergence through bottom-up processing. Phase 3 integrates all visual skills and incorporates top-down processing to re-calibrate the brain. Loading with multi-sensory integration is done throughout all phases. Procedures chosen for the patient all include elements to promote

neuroplasticity to invoke positive changes in neural functioning. Neuroplasticity will be discussed in more detail later.

Studies done by Thiagarajan and Ciuffreda (2012, 2013, and 2014) demonstrate how neuro-optometric rehabilitation therapy can positively impact vergence dysfunction, accommodative dysfunction and versional dysfunction in people with mild traumatic brain injury.[11-14] Each study was conducted as a single-blind, crossover, interventional experimental design where each patient was their own control.[11-14] They had 9 hours of oculomotor training for accommodation, vergence or version and 9 hours of placebo training done in 45 minute sessions, twice a week.[11-14] For accommodation, they trained step accommodative amplitude and step accommodative facility.[12] For vergence, they trained smooth and step convergence and divergence.[13] For versional training, they worked on fixation, predictable saccades and simulated reading.[11] Overall, speed and accuracy improved in oculomotor eye movements (vergence, accommodation and version) after oculomotor training compared to no significant improvement after placebo training.[11-14] Specifically they saw statistically significant increased monocular/binocular accommodative amplitude, improved dynamics of accommodation, reduction in near vision symptoms, and improved visual attention after accommodation training.[12] After vergence training, they saw increased peak velocity for convergence and divergence, increased vergence flipper rate, reduced steady-state response variability of convergence, increased maximum amplitude of convergence, improved relative fusional amplitudes, improved near stereoacuity, improved visual attention, and reduced symptoms.[13] For versional training, they saw reduction in the horizontal fixational error, increased saccadic gain both horizontally and vertically, and reduction in the saccade ratio for the simulated reading, multiple-line paradigm.[11] All these changes are likely due to increased speed of processing resulting from neuroplastic changes in the brain. Neuro-optometric rehabilitation therapy creates an environment that promotes those neuroplastic changes. It does so by utilizing some of the basic tenets of experience-dependent neuroplasticity: repetition, motivation, specificity, appropriate intensity, feedback, and guidance.[15]

Repetition prompts the brain to increase the neural representation for that task with the caveat that large changes only occur for newly learned or re-learned tasks.[15] Much smaller changes occur with the repetition of previously acquired skills.[15] Neuro-optometric rehabilitation utilizes repetition and tries to mitigate boredom by targeting the same skill within many different procedures. For example, Brock string and a vectogram can both work on the vergence system while bringing novelty to the patient experience. Similarly, the specificity of a task plays a role in the effectivity of a procedure.[15] Learning, rather than simple use of a skill, seems to produce more significant results in the brain.[15] For example, if someone has difficulty reading due to deficient convergence or oculomotor skills, simply reading will not produce large neuronal changes but learning how to correctly converge or make a saccadic eye movement more accurately will improve reading and create significant restructuring or strengthening of new pathways within the brain. Additionally, specific skills modulate specific cortical sub-regions.[15] This highlights the importance of honing rehabilitation procedures to a specific damaged region of the brain. The patient's motivation can make or break a training session. The patient's perception of the importance of the exercise will affect the degree of structural change seen in the brain.[15] Motivation promotes engagement in the task which then leads to better results.[15] It is also imperative to regulate the intensity of the task while doing rehabilitative therapy.[15] The task needs to be difficult enough to promote change but cannot over-stimulate or overwhelm the patient, as it may produce counterproductive results.[15] As such, a scale of subjective difficulty

and symptomatology used to monitor the patient's ability to continue can be useful to prevent overstimulation during an activity. Some ways to adjust difficulty level can be to integrate or remove multi-sensory integration, visualization, problem solving, and other forms of loading. Lastly, feedback and guidance during a task can help the patient learn more quickly how to control their eye movements and accommodation.[15] Feedback can be visual, auditory and/or sensory. For example, they may see physiological diplopia and understand that their eyes are not pointing directly at the object that they wish. Auditory feedback for the patient would be providing constructive criticism on a procedure being performed. The patient can also use the proprioceptive awareness of eye movements to identify whether they are converging or diverging. As indicated above, effective neuro-optometric rehabilitation therapy programming will create the environment and set goals which promote neuroplastic changes leading to improved visual function and performance.

Table 1: Common Signs and Symptoms of Traumatic Brain Injury.[10,11]

Common Signs and Symptoms of Traumatic Brain Injury	
Signs	**Vergence** - Convergence Insufficiency - Convergence Excess - Divergence Insufficiency - Divergence Excess - Binocular Instability - Strabismus **Accommodation** - Accommodative Insufficiency - Accommodative Infacility - Accommodative Spasm - Accommodative Excess **Oculomotor** - Deficiency of Saccades - Deficiency of Pursuits - Deficiency of Fixation **Visual-Vestibular Dysfunction**
Symptoms	Light Sensitivity Visual Motion Sensitivity Visual Field Deficit Poor Attention Poor Memory Poor Concentration Problems with Executive Function

Neuroplasticity

Neuroplasticity is the ability to make changes to the neurons within the brain, whether by increasing or decreasing the brain's capability to perform a task.[15] With neuro-optometric rehabilitation therapy, we can utilize the brain's propensity for change under certain conditions to improve the operation of various visual functions. Persons with traumatic brain injury tend to have impaired synaptic function or have longer neuronal pathways to get from one area of the brain to another, and therefore slower brain processing.[16,17] The goal of neuro-optometric rehabilitation therapy is to rehabilitate one or both problems to speed up the person's ability to process and perform a function. Several types of cortical plasticity are involved in this process. These include increased expression of plasticity-related proteins, increased expression of growth promoting genes, neurogenesis, axonal sprouting, dendritic plasticity, angiogenesis, synaptogenesis, unmasking, reactive astrogliosis, and myelination.[18-25] We propose that synaptogenesis, axonal sprouting and dendritic sprouting make the biggest impact on the functional changes seen after neuro-optometric rehabilitation therapy because those are the ones that are making direct changes to the neurons. See Table 2 for a brief summary of each of these cortical plasticity changes. Some of them are direct structural changes like neurogenesis, axonal sprouting, dendritic plasticity, synaptogenesis and unmasking, while the rest are indirect contributors to neuroplasticity.[18-25] They all play a role in speeding up signal transmission throughout the brain. We can correlate these changes with structural functional changes seen on imaging studies such as functional Magnetic Resonance Imaging (fMRI).

Table 2: Summary of Neuroplasticity Changes.[15,22]

Neuroplasticity Changes	These proteins make the environment more adaptive.
Increased expression of plasticity related proteins	The perilesional area becomes growth permissive after insult.
Increased expression of growth promoting genes	Growth of new entire neurons.
Neurogenesis	Note: only been found in hippocampus and olfactory bulb so far.
Axonal sprouting	New axon growth after neuronal loss or axonal damage.
Dendritic plasticity	Increased spine growth on the dendrite with increased stimulation, or decreased spine growth with decreased stimulation.
Angiogenesis	Growth of new blood vessels to increase blood flow. This supports new growth in the damaged area.
Synaptogenesis	Growth of new synapse.
Unmasking	The removal of an inhibition signal to a previously inhibited axon, allowing the availability of a new pathway.
Reactive Astrogliosis	A defensive reaction of astrocytes in response to traumatic event to the brain. They ideally handle acute stress, limit tissue damage and restore homeostasis. If the response persists, it may become maladaptive and cause damage to the brain.
Myelin Remodeling	Three Types: An increase in the thickness or length of existing myelin. The new growth of myelin. The decrease of space between existing myelin.

Research Showing Evidence of Altered Brain Structure Corresponding with Function

The basis for neuroplasticity is that there are some or all the structural changes mentioned above due to a change in environment and demand upon the damaged tracts in the brain. It is impossible at this time to see the microscopic neuroplastic changes in humans, but we can confirm that they occur in animal models. For example, a study by Girgis et. al. demonstrated that there is significant increased axonal sprouting in the corticospinal tract rostral to the site of injury, of trained mice compared to controls.[26] The trained mice underwent therapy involving a reaching task after their corticospinal tract had been lesioned.[26] The trained mice were substantially improved in the reaching task compared to untrained mice.[26] Note that there was insignificant increased axonal sprouting in the untrained group compared to controls.[26] So, damage to the central nervous system automatically increases neuroplastic changes, but therapy increases it even more. Although we cannot do this type of invasive experiment on humans, we can use neuroimaging to correlate functional changes in the areas we expect axonal sprouting, etc. to occur.

Development in technology has allowed us to correlate neuro-optometric rehabilitation therapy clinical changes to actual altered brain structure. Alvarez et. al. (2021) showed evidence of neurophysiological changes after optometric vision therapy for convergence insufficiency in adults.[27] They looked at the fast vergence (fast-fusional) system, responsible for fusing quickly in a changing environment, and the slow vergence (slow-fusional) system, responsible for optimization of prolonged fusion, separately.[27] In general, the visual cortex, cuneus, frontal eye fields, supplemental eye fields, parietal eye fields and cerebellar region were stimulated for fast- and slow-vergence systems.[27] For the fast-fusional system, the right cuneus functional activity was correlated to faster peak vergence velocity after 12 sessions of vergence/accommodative therapy, which means they will be able to converge faster to a close target.[27] For the slow-fusional system, the medial cuneus functional activity was correlated to faster rate of phoria adaptation to a 6BO prism after 12 sessions of vergence/accommodative therapy, meaning the patient can read/do near work for longer periods of time and with more comfort.[27] Overall, this indicates that restructuring of the brain does occur within the cuneus in convergence insufficiency patients after vision therapy. We can extrapolate that this will also occur for traumatic brain injury patients with

symptomatic convergence insufficiency since they have the same clinical improvements after neuro-optometric rehabilitation therapy as the patients in this study. Future studies are also needed to confirm specific structural changes which occur with versional and accommodative training and to correlate neuroplastic changes after neuro-optometric rehabilitation therapy in traumatic brain injury patients.

CONCLUSION

With traumatic brain injury becoming an increasingly recognized problem, it is important to understand how to help people with persistent symptomatology following a traumatic brain injury, as it can have a negatively impact quality of life. Neuro-optometric rehabilitation therapy, based on neuroscience, implements principles of neuroplasticity to increase neuronal processing speeds. From previous fMRI research studies on adults with convergence insufficiency, it can be deduced that traumatic brain injury patients will most likely display structural changes following neuro-optometric rehabilitation therapy on a fMRI. However, further research is needed to investigate the impact of neuro-optometric rehabilitation therapy on neuroplastic changes in patient with traumatic brain injury and associated versional and accommodative dysfunctions.

REFERENCES

1. Publications & Reports [Internet]. Centers for Disease Control and Prevention. Centers for Disease Control and Prevention; 2021 [cited 2021Apr16]. Available from: **https://bit.ly/3VMKzeL**
2. Andriessen TMJC, Jacobs B, Vos PE. Clinical characteristics and pathophysiological mechanisms of focal and diffuse traumatic brain injury. J Cell Mol Med. 2010 Oct;14(10):2381–2392.
3. Mckee AC, Daneshvar DH. The neuropathology of traumatic brain injury. Handb Clin Neurol. 2015;127:45–66.
4. Barth JT, Freeman JR, Broshek DK, Varney RN. Acceleration-deceleration sport-related concussion: the gravity of it all. J Athl Train. 2001;36(3):253–256.
5. Ciuffreda KJ, Ludlam DP, Yadav NK, Thiagarajan P. Traumatic brain injury. Adv Ophthalmol Optom. 2016; 1(1):307-333.
6. Ungerleider LG, Pasternak T. Ventral and dorsal cortical processing streams. In: Werner JS, Chalupa LM, editors. The visual neuroscience. Cambridge: MIT Press; 2004. p. 541-562.
7. Alnawmasi MM, Chakraborty A, Dalton K, Quaid P, Dunkley BT, Thompson B. The effect of mild traumatic brain injury on the visual processing of global form and motion. Brain Inj. 2019 July;33(10):1354-1363.
8. Gilbert CD, Li W. Top-down influences on visual processing. Nat Rev Neurosci. 2013 May;14(5):350-363.
9. Lowell L, Cohen AH, Kapoor N. Optometric management of visual sequelae of frontal lobe-related traumatic brain injury. J Behav Optom. 2010;22:3-11.
10. Ventura RE, Balcer L J, Galetta SL. The neuro-ophthalmology of head trauma. Lancet Neurol. 2014;13:1006–16.
11. Thiagarajan P, Ciuffreda KJ. Versional eye tracking in mild traumatic brain injury (mTBI): Effects of Oculomotor Training (OMT). Brain Inj. 2014;28(7):930–943.
12. Thiagarajan P, Ciuffreda KJ. Effect of oculomotor rehabilitation on accommodative responsivity in mild traumatic brain injury. J Rehabil Res Dev. 2014;51(2):175–92.
13. Thiagarajan P, Ciuffreda KJ. Effect of oculomotor rehabilitation on vergence responsivity in mild traumatic brain injury. J Rehabil Res Dev. 2013;50(9):1223–40.
14. Thiagarajan P. Oculomotor rehabilitation for reading dysfunction in mild traumatic brain injury [dissertation]. New York City (NY): State University of New York State College of Optometry; 2012.
15. Kleim JA, Jones TA. Principles of experience-dependent neural plasticity: implications for rehabilitation after brain damage. J Speech Lang Hear Res. 2008 Feb;51(1):S225+.
16. Han K, Chapman SB, Krawczyk DC. Disrupted intrinsic connectivity among default, dorsal attention, and frontoparietal control networks in individuals with chronic traumatic brain injury. J Int Neuropsychol Soc. 2016;22;263–279.
17. Wen Z, Li D, Shen M, Chen G. Therapeutic Potentials of Synapses after Traumatic Brain Injury: A Comprehensive Review. Neural Plasticity. 2017:1–8.
18. Huang JC. Neuroplasticity as a proposed mechanism for the efficacy of optometric vision therapy & rehabilitation. J Behav Optom. 2009;20:95-99.
19. Pekny M, Pekna M. Astrocyte reactivity and reactive astrogliosis: costs and benefits. Physiol Rev. 2014;94:1077–1098.
20. Burda JE, Bernstein AM, Sofroniew MV. Astrocyte roles in traumatic brain injury. Exp Neurol. 2016 Jan;275(0 3): 305–315.
21. Hylin MJ, Kerr AL, Holden R. Understanding the mechanisms of recovery and/or compensation following injury. Neural Plasticity. 2017 Apr 20, ID 7125057:[12 pages].
22. Lillard AS, Erisir A. Old dogs learning new tricks: Neuroplasticity beyond the juvenile period. Dev Rev. 2011:31:207–239.

23. Wilson NM, Titus DJ, Oliva AA, Furones C, Atkins CM. Traumatic brain injury upregulates phosphodiesterase expression in the hippocampus. Front Sys Neurosci. 2016 Feb 5;10(5):1-13.
24. Maria NSS, Sargolzaei S, Prins ML, Dennis EL, Asarnow RF, Hovda DA, et al. Bridging the gap: Mechanisms of plasticity and repair after pediatric TBI. Exp Neurol. 2019;318:78–91.
25. Griesbach GS, Hovda DA. Cellular and molecular neuronal plasticity. In: Grafman J , Salazar AM, editors. Handbook of Clinical Neurology. Netherlands: Elsevier; 2015. p.681-690.
26. Girgis J, Merrett D, Kirkland S, Metz GAS, Verge V, Fouad K. Reaching training in rats with spinal cord injury promotes plasticity and task specific recovery. Brain. 2007;130(11):2993-3003.
27. Alvarez TL, Scheiman M, Morales C, Gohel S, Sangoi A, Santos EM, Yaramothu C, d'Antonio-Bertagnolli JV, Li X, Biswal BB. Underlying neurological mechanisms associated with symptomatic convergence insufficiency. Sci Rep. 2021;11(1):6545–6545.

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