Article • The Influence of Altered Cervical Input from Whiplash Injury on Post-Concussion Ocular/Visual Signs and Symptoms

Shirley Blanc, OD • Toronto, Ontario, Canada

ABSTRACT

The complex integration between the vision, proprioception, and vestibular systems provides the foundation for postural stability, gait patterns, and many visual functions. Such visual functions include conjugate eye movements, vergence, and stability of gaze as the head, body, and/or target moves. These processes are facilitated by magnocellular pathways in the brain. The magnocellular system is particularly susceptible to dysfunction from a concussion, which can result in a variety of symptoms characteristic of Post-Trauma Vision Syndrome. Whiplash cervical injury without direct head trauma can arguably be considered to be a cause of concussion due to a similar profile of signs and symptoms as traumatic brain injury from direct head impact. Cervical afferent changes that occur from whiplash can produce sensorimotor disruption and thus affect those visual functions that are influenced by proprioceptive input from the neck. As can be seen, rehabilitation of visual function following whiplash injury requires a multi-disciplinary collaborative effort to facilitate the variety of sensory inputs needed for these processes to occur accurately and effortlessly.

Keywords: concussion, convergence, oculomotor, proprioception, sensorimotor, vision, vestibular, whiplash

Whiplash and Concussion

A 1995 report by the Quebec Task Force defined Whiplash Associated Disorders (WAD) as acceleration-deceleration energy transfers to the neck, impacting either bony or soft tissue, that are associated with neck and head pain, visual disturbances, tinnitus, dizziness, and fatigue.[1] Whiplash injury can range from muscle strains and ligamentous sprains to spinal cord contusions and fractured vertebrae.[2] It is interesting to note that the majority of WAD cases with neck pain have no visible pathology;[2] however, evidence of axonal injury has been recently shown using diffuse tensor tractography.[3]

Neck pain and headaches occur in approximately 70% of whiplash patients, with 20-40% of patients having continued symptoms for several years.[2] Cervicogenic headache may be caused by occipital pain from neck movement or from nerve root irritation, inflammation, or compression of the second cervical vertebra (C2 or the axis).[2] Pain in the suboccipital region due to nociceptive fibers in the area of C2 and the first cervical vertebra, the atlas, can cause ipsilateral referred retro-orbital eye pain via the trigeminal nerve.[4,5] Dizziness from a cervicogenic cause may be related to proprioception dysfunction in the cervical spine, poor vertebral artery circulation, or sympathetic system over-activation.[2]

Photosensitivity has also been associated with cervicogenic headache from a whiplash injury.[3] Such injuries can set in motion autonomic dysregulation, particularly over-action of the sympathetic system.[2,6] A study by Edwards et al.[7] showed that upper cervical sensory afferents from muscle spindles have major input into the intermediate nucleus of the medulla (InM). Chemical stimulation of the InM can produce tonic sympathetic discharge, similar to C2 nerve activation.[7] Autonomic

dysregulation from concussion has been shown to result in abnormal pupil dynamics, including reduced average constriction velocity, increased peak dilation velocity, and larger pupil diameter, all sympathetically-driven responses.[8-11] Pupil dynamics are closely related to heart rate variability (HRV), a measure of autonomic function.[12,13] HRV has been shown to be lower in both whiplash patients with chronic pain and in concussion patients compared with healthy controls.[6,14] Thus, it would be logical that disruption of the autonomic system and sympathetic overstimulation stemming from insult to the neck could yield abnormal light adaptation and hence symptoms of light sensitivity.

Neck pain has been linked to visual symptoms. A study by Treleaven's group in 2013[15] compared 70 subjects with chronic neck pain of at least 3 months' duration [traumatic (n = 45) and idiopathic (n=25)], with 70 asymptomatic healthy controls, all between the ages of 18 and 60. Based on symptom questionnaires, the neck pain group had significantly more visual complaints than the control group, with the traumatic group having symptoms of greater magnitude than the idiopathic group. At least 50% of the subjects with neck pain noted the need to concentrate to read, visual fatigue, sensitivity to light, eye strain, and heavy eyes. There was significantly greater magnitude in the WAD group with complaints of blurred vision, words moving, and need to concentrate compared to the idiopathic neck pain group.[15]

The symptom profile, pathophysiological changes, and disturbances in oculomotor, visual, and cognitive function in WAD patients make the case that WAD is a likely cause of, and not merely similar to, concussion in presentation. The CDC defines concussion as a "bump, blow, or jolt to the head or...a hit to the body that causes the head and brain to move rapidly back and forth."[16] Thus, a concussion can occur with or without direct injury to the head. One study involving 183 hockey players stated that all subjects with whiplash neck injuries reported concussion symptoms.[17] A study in 2017 by Beeckmans et al. compared neuropsychological testing of attention, memory, and visuospatial executive functions in patients with whiplash injury, mild traumatic brain injury (mTBI), and healthy controls. They found that the whiplash and mTBI groups did not differ significantly in cognitive function, and both groups had significantly more neuro-cognitive deficits than controls.[18] Cognitive function has been suggested by Mani et al. to influence oculomotor function, particularly noted in anti-saccades and memory-guided saccades in concussed patients.[19] The cognitive deficits and oculomotor dysfunction in WAD patients make the case for a comparable profile to those with concussion from direct head trauma.

Given the cascade of changes that can occur from a whiplash injury, it would appear that an assessment of a patient with concussion-related symptoms should incorporate investigation of the neck as a possible contributing factor.

The Magnocellular Visual System

The magnocellular visual system is required for spatial and motion perception, as well as for motor planning. This requires the convergence of sensory information from vestibular, visual, proprioception inputs. The midbrain is a key area, where pre-conscious feed-forward information from the peripheral retina and somatosensory and auditory-vestibular systems is combined with cortically-driven feedback information. Neck muscle afferents, extraocular muscle input, and retinal information meet in the superior colliculus in the tectum.[20] The execution of motor responses requires multi-sensory input providing information about where we are, where something is relative to us, and temporal information regarding movement

and speed. The vast neurological real estate comprising the magnocellular system might explain the high oxygen and metabolic demand to facilitate these pathways.[21] As a result, the magnocellular system may be particularly susceptible to blood-flow and metabolic changes that occur from concussive injury, including whiplash.[22,23]

Magnocellular system disruption from concussion is demonstrated in various ways when compared with healthy controls: poorer peripheral reaction times, significantly poorer performance in oculomotor function, higher motion coherence thresholds, and convergence insufficiency (CI) demonstrated with abnormal visually evoked potentials (VEP).[24-28] Abnormal cervical input due to WAD can disrupt the magnocellular pathways at the level of the midbrain's sensory integration area and affect these functions.

Gaze Stabilization

In order to keep an image stable on the retina as the head moves, a combination of reflexive eye movements is required: the cervico-ocular reflex (COR), involving rotation of the neck; the vestibulo-ocular reflex (VOR), involving rotation of the head; and the optokinetic reflex (OKR), involving oculomotor responses to moving stimuli in the visual field. Proprioceptors from deep neck muscles and joint capsules from C1 to C3 and the vestibular nucleii are stimulated with head movement, leading to opposing movement of the eyes.[1] The COR and OKR respond best to low head movement frequencies, while the VOR responds to high frequencies.[1] A VOR or COR gain of 1.0 represents perfect accuracy in keeping the eyes steady on a target while the head or neck moves, respectively. Any other value indicates poor gaze stability.

Studies have shown that a whiplash injury results in an increase in gain of the COR as the primary cause of gaze instability, while the VOR and OKR are relatively unaffected.[1,29] It is proposed that the increased COR gain occurs due to reduced range of motion associated with neck pain, resulting in sensorimotor impairment, altered proprioception, and abnormal muscle activation patterns.[1,2,29-32] The testing of COR gain consists of fixed head position and body rotation in complete darkness, with subjects being asked to look at a laser dot straight ahead and the examiner measuring accuracy of gaze. VOR testing involves head and body rotation in a chair while maintaining fixation on a target. Schneider's group showed that the primary factor determining concussion in elite youth hockey players was cervical injury, with VOR relatively unaffected.[33] This seems to support the notion that cervical afferent sensorimotor dysfunction may be a more dominant factor in concussion than visual-vestibular integration.

With age, there is also an increase in the COR gain; however, it is thought to be compensatory for an associated decrease in VOR gain.[34] In the case of whiplash trauma, VOR is unable to compensate for the abnormal increase in COR gain. This may be due to the fact that whiplash trauma results in slower head movements compared to controls;[30] therefore, the VOR cannot be activated in the presence of slower head movement speed. There may also be a possibility of vestibular pathway microtrauma, such as in the flocculomodular area of the cerebellum, preventing adaptation of the VOR.[1] The subsequent imbalance of reflexes results in poor image stability.

Clinically, VOR can be assessed using dynamic visual acuity with the head moving at 2 Hz while looking at a static acuity chart. With head movement of less than 2 Hz, the VOR is influenced by the optokinetic and smooth pursuit system.[35] A decrease in visual acuity of greater than 2 lines is considered abnormal.[36] Active self-generated head motion by the patient runs the risk of generating predictive saccades and smooth pursuits during the

head movement. In contrast, passive motion facilitated by the examiner at the desired frequency and amplitude yields a more unpredictable condition and a more reliable measure of VOR.[37]

Currently, there is no standard clinical test to assess COR. To isolate COR, the head ideally should remain still while the body is rotated to either side with fixation on a target straight ahead. A visual acuity target could be used to assess gaze stability, as COR dysfunction could yield blur as well as dizziness.[38] Also, studies that have measured COR used electrooculography to determine the accuracy of the reflex. A study using commercially available eye movement recording technology to monitor the ability to focus on a static target and facilitating head/body adjustments that would isolate COR may be useful clinically to determine the influence of cervical proprioception on gaze stability.

Convergence

CI occurs in at least 50% of post-concussion patients[39] versus 2-17% in the general population.[40] One study showed a 10-15% prevalence of CI in a cohort of healthy collegiate athletes tested in the pre-season.[41] The integration between proprioceptive, vestibular, and visual inputs in the superior colliculus is important for convergence according to animal studies.[42,43] CI following a concussion has been associated with increased recovery time, poorer neurocognitive function, slower reaction times, and even gait deficits.[44-46] These potential links warrant further investigation to understand how a convergence problem may be related to other widespread post-trauma effects.

A study by Poltavski et al. compared VEP responses in those with CI with and without a history of concussion.[28] The stimuli presented were transient in nature, targeted to the magnocellular peripheral processing system. Results showed that VEP latencies were slower and amplitudes were smaller for patients with CI secondary to concussion versus non-concussed subjects. This study demonstrates the pronounced magnocellular deficit causing the spatial localization problem in the concussed versus developmental case. This is not surprising considering the co-morbid findings that appear to occur in association with CI in the case of concussion.

A study published in the *Clinical Journal of Sport Medicine* by Howell et al. compared subjects with a history of concussion and receded near point of convergence (NPC), concussed subjects with normal NPC (<5cm from the nose), and non-concussed individuals with normal NPC.[46] The subjects were between the ages of 10 and 18 years, with gender evenly split. The tasks involved standing with cognitive task, gait with no cognitive task, and dual gait and cognitive task. The results showed that the post-concussion subjects with CI had significant gait deficits and slower average walking speed compared to both healthy controls and to post-concussion subjects with no convergence problem. Thus, it is plausible that a pronounced magnocellular deficit manifesting as a convergence problem may also impact motor output on a global scale.

Neck pain is often associated with WAD, possibly a cause of reduced range of motion and slower head velocity.[1,29,30] Giffard et al. compared NPC in those with idiopathic neck pain and healthy controls, measured both in neutral position and with neck in torsion, i.e. head still on rotated trunk.[47] Results showed significantly greater left, right, and average torsion differences in NPC for subjects with idiopathic neck pain compared to healthy controls. Although this study did not involve whiplash patients, it is suggestive of abnormal cervical afferent input stemming from neck pain, thus affecting sensorimotor function and convergence ability. A comparable study involving traumatic neck pain would be useful.

A prospective cohort study by Stiebel-Kalish et al. looked at subjective and objective measures of convergence insufficiency in patients with WAD following motor vehicle accident (MVA) compared to healthy controls.[48] They found that the Convergence Insufficiency Symptom Survey (CISS) questionnaire showed diagnosis of CI in 45% of WAD patients compared to 15.4% of controls; however, interestingly, the objective findings indicated that CI occurred in 7% and 7.7% for WAD and control patients, respectively.

The discrepancy in symptoms versus objective findings in this study prompts further investigation, looking at the variation of testing techniques for NPC. In the Stiebel-Kalish study, an accommodative target was used for the non-presbyopes, and a light target was used for the presbyopic subjects. NPC measured with red lens technique has been shown to be more sensitive than an accommodative target or transilluminator in a prospective study of optometry students.[49] Also in the Stiebel-Kalish study, NPC was not repeated with consecutive trials, which might have revealed a receding endpoint. The NPC measurements were taken in a neutral neck position and were not compared with neck torsion posture. The majority of subjects were classified as less than level 2 WAD, and so with more severe whiplash-associated symptoms, a convergence problem may be more prevalent objectively. These variations in conditions highlight the importance of standardization in measuring convergence ability.

It can be hypothesized, then, that the presence of convergence insufficiency following a concussion may in fact reflect a more widespread neurological deficit in spatial processing and motor responses. Also, the worsening of convergence with neck torsion could indicate altered cervical proprioceptive input disrupting sensorimotor function and spatial localization. However, the measuring technique, type of stimulus, and threshold of an abnormal NPC value need to be further standardized for clinical practice.

Saccades

Saccades are important in motion processing, reading, and scanning the environment. They can be voluntary or reflexive in nature. Ciuffreda's group found that approximately 50% of 160 visually symptomatic post-concussion subjects showed oculomotor deficit, particularly in saccades.[39]

A recent meta-analysis and systematic review of oculomotor deficits following acute (<3 months) and chronic traumatic brain injury looked at various oculomotor functions: smooth pursuits, memory-guided saccades, reflexive saccades, self-paced saccades, and anti-saccades.[19] The study found that there are deficits in oculomotor function across the board in TBI cases compared to healthy controls. However, of note, the memory-guided and anti-saccade deficits were the biggest differentiators between injured versus non-injured subjects. This finding highlights the particular vulnerability of cortically-driven saccades with brain injury. Pertaining to the whiplash population, Mosimann et al. found that symptomatic WAD subjects showed increased latency in intentional saccades and a higher percentage error in amplitude in memory-guided saccades compared to recovered WAD patients and age-matched healthy controls.[50]

The *Journal of Neurotrauma* published a study in 2016 comparing acutely injured mTBI subjects with orthopedic controls (excluding the head) and uninjured controls, looking at performance on the King-Devick test, a cognitive test, and a tablet-based anti-point and pro-point task. Results showed that mTBI subjects were slower at the pro-point task compared to orthopedic subjects, and orthopedic subjects were slower than controls, suggesting a sensorimotor deficit. However, the mTBI patients showed cognitive deficits

that were not shown in the other groups and were significantly slower at the anti-point task than both of the other groups.[51] The anti-point task can theoretically be comparable to the anti-saccade task due to the requirement of the frontally driven executive decision-making process to hold back a response and then to make a response in the equal and opposite direction.

These findings appear consistently to suggest that in concussion (including whiplash with persistent symptoms), higher cortically driven cognitive functions such as working memory, response inhibition, and visuo-spatial processing may be particularly affected, consequently impacting saccadic function.

Smooth pursuits

Disruption of the magnocellular system at the level of the superior colliculus by cervical afferent dysfunction associated with whiplash injury can affect smooth pursuit function. The initiation of smoothly following a moving target requires the middle temporal area (MT), the middle superior temporal (MST) area, the parietal and frontal cortical areas,[52] the superior colliculus,[53] and the cerebellum. The cerebellum contributes to both the initiation and continued velocity of smooth eye movement.[54] Velocity of eye movements tend to be slower and less coordinated in whiplash patients.[30]

A study published in 1998 looked at whiplash patients and found that smooth pursuit dysfunction significantly correlated with reduced active cervical range of motion and reduced kinesthetic sense.[55] Kinesthetic sense is the sense of head position in space with eyes closed. An error in this calculation is known as joint position error (JPE). There was no significant correlation with neck pain in this study.

Measuring JPE consists of assessing the accuracy in moving the head in any direction and then back to the center of a target with eyes closed, after aiming at the target initially with a head-mounted laser with the eyes open.[38] JPE was shown in a 2006 study to have a high predictive value but low sensitivity and specificity in correlating with poor balance and smooth pursuit deficit in neck torsion (SPNT). This weak relationship suggested that JPE is valuable but should not be the only measure in identifying oculomotor and postural control dysfunction with persistent whiplash symptoms.[56]

A 2008 prospective trial of smooth pursuits in whiplash patients showed that smooth pursuit gain was not significantly decreased in a neck-neutral position, consistent with the 1998 study; however, there was significant smooth pursuit deficit in chronic patients with neck pain in neck torsion.[57] The 1998 study showed no correlation between smooth pursuit deficit and neck pain. However, it involved testing oculomotor function only in neck-neutral position and not in neck torsion. Treleaven et al. showed that smooth pursuit deficit in neck torsion can be present in whiplash patients with chronic neck pain, especially for those who also experience dizziness, and that this deficit does not seem to be related to level of anxiety. Rather, the smooth pursuit deficit appears to be due to proprioceptive and nocioceptive factors from the cervical spine.[58] In addition, the presence of SPNT deficit initially after whiplash injury does not seem to be predictive of recovery.[57]

Neck pain without history of whiplash injury can also result in smooth pursuit gain deficit in neck torsion compared with healthy controls.[59] This seems to beg the question of whether the presence of neck pain alone in whiplash patients is responsible for decreased smooth pursuit gain in neck torsion. Janssen et al. investigated whether the predictability of a target affects the smooth pursuit gain in various degrees of neck torsion, in healthy controls, and in neck pain patients divided into whiplash and non-whiplash etiology.[60]

The results showed that smooth pursuit gains in neck torsion were decreased in neck pain patients versus healthy controls; however, the WAD patients performed worse compared to both non-WAD neck pain patients and healthy controls when the target moved in a predictable trajectory. This finding suggests that there appears to be more to the picture than simply whiplash-induced neck pain affecting cervical afferent information and causing oculomotor dysfunction in neck torsion, but there are possibly additional cognitive factors dealing with attention and concentration. As mentioned previously in this paper, higher cortical processes may be a significant differentiator in terms of oculomotor deficit after a concussive injury, highlighted particularly with anti-saccades and memory-guided saccades.

Clinically, then, the presence of reduced gain of smooth pursuits in neck torsion with whiplash patients with chronic neck pain, and an associated finding of poor joint position sense, could be valuable information to optometrists in terms of the relative weight placed on altered cervical proprioception input in eye movements. This information would provide the opportunity for optometrists to collaborate with physical therapists to improve sensorimotor function.

Evaluating smooth pursuit in neck torsion consists of the head in neutral position with the body rotated 45 degrees.[61] Determination of gain with examiner observation of the patient following a manual target such as a pen has been shown to be effective when compared with electrooculography.[59] This enables examiners to perform this test accurately without the need for expensive equipment. Decreased gain in neutral position would imply a CNS etiology, not heavily weighted toward cervical afferent misinformation. Moving the target in a predictable versus unpredictable fashion may be a more sensitive probe to uncover gain reduction in whiplash patients with chronic neck pain. The quality of eye movement and symptom provocation would provide valuable information. Future studies looking at attentional and cognitive factors influencing eye movements in the presence of whiplash/concussion patients would be valuable.

Postural control

Postural control and balance rely on central integration of the visual, somatosensory, and vestibular systems.[62] Neck proprioception appears to be more dominant than proprioception of the extremities for postural control when comparing vibration to the neck versus the calf.[63] This may be due to the abundant density of muscle spindles in the neck.[64]

Gaze and ocular proprioceptive input appear to affect postural responses, as demonstrated by Yuri et al. in 1999, where either vibration applied to the neck or galvanic stimulation of the vestibular system resulted in sway that was biased in the direction of gaze with eyes open or closed. Neck vibration alone caused a forward sway, and vestibular stimulation caused a lateral sway.[65] Extraocular muscle vibration can influence full-body responses, where the lateral and medial recti cause lateral sway and the inferior and superior recti cause sway in an anterior-posterior direction.[66] Posturographic analysis has demonstrated a significant increase in anterior-posterior sway with eyes closed in whiplash patients compared to a control group.[67] This seems to correspond to the forward sway seen in neck vibration in experiments. Neck torsion in patients with persistent whiplash may lead to greater balance disturbances and postural deficits.[68]

An error in visual-proprioceptive integration can manifest as a visual midline shift. A visual midline shift is a mismatch between visual spatial perception and the proprioceptive base of support and can result in abnormal embedded motor and postural

patterns.[70] The prevalence of visual midline shift was found to be in 93% of TBI subjects in a retrospective study of 30 TBI and 30 non-TBI patients.[70]

A joint therapy program with optometrists and physical therapists addressing proprioceptive input from the extraocular muscles with oculomotor training, cervical proprioception, and integration of visual, vestibular, and proprioceptive systems for postural stability should be researched further.

Eye-head Coordination

The synchronicity of the eyes and the head moving in tandem, or sequential eye then head movement to a target, can be affected following a whiplash injury. Treleaven et al. showed poorer performance in sequential saccades to a target followed by head movement in whiplash patients compared to asymptomatic controls with no history of neck pain, visual problems, or injury. They also showed that with the head and eyes moving in tandem, whiplash patients had reduced range of motion of the head, but the range of motion was greater without fixation.[32] This study suggests that sensorimotor dysfunction, with respect to the communication between the eyes and the neck, can cause poorer performance in coordinated head and eye movement in whiplash patients.

Treatment Approaches

Given the complex integrative processes that occur between vision, vestibular, and cervical proprioceptive systems, an ideal rehabilitative approach would presumably be a multi-disciplinary effort with optometrists and physical therapists to address each system optimally, as well as synchronized and accurate communication between these systems.

Optometric Intervention

Post-concussion vision syndrome benefits from optometric intervention by way of optical means such as prisms, lenses, and coloured tints, as well as an in-office and home-based therapy program.

Prisms are powerful optometric tools that can provide a more accurate link between vision and proprioception and improve posture, balance, and gait stability, as in the case of visual midline shift. Yoked prisms can bring a patient to centered midline, thereby improving the relationship between vision and proprioception.[71] Since abnormal cervical proprioception from whiplash injury can affect the sense of position in space, altering visual input with prisms can help bring improved synchronicity between vision and proprioception.

Due to the close communication between the extraocular muscles and cervical afferent substrates and the influence of the extraocular muscles on postural stability, oculomotor training should be a component of rehabilitation of whiplash-associated visual symptoms and signs. Post-concussion saccadic training has been shown to improve functional outcomes such as reading speed.[72-75]

The presence of convergence insufficiency in WAD should be addressed by a combination of cervical proprioception training to improve cervical afferent input to the superior colliculus and an in-office optometric program in conjunction with home-based activities, as per the Convergence Insufficiency Treatment Trial (CITT).[76] Although the CITT study dealt with children without a history of trauma, the benefits of the CITT protocol can also extend to the post-concussion population. This was demonstrated in a retrospective study involving a post-concussion cohort in two optometry practices, showing 85% improvement in convergence, as well as symptoms and signs.[77] A possible strategy may be training vergence in a position of neck torsion as well as in neutral position.

Different wavelengths of light can influence the autonomic system. One study showed that

synthesis of melatonin by the pineal gland is affected by short-wavelength light, whereas cortisol production by the adrenal gland is influenced by short- and long-wavelength light.[78] Colour wavelengths have been associated with HRV changes and can alter pupil responses.[79,80] Since the sympathetic/parasympathetic balance is often disrupted following a whiplash injury, a colour spectacle tint or light stimuli of different wavelengths may be potential optometric tools to restore this balance.

Physical Therapy Intervention

With respect to physical intervention, a clinical practice guideline published in 2016 indicated that an exercise program including manual therapy of the neck, neck strengthening, and range-of-motion exercises is beneficial for acute and chronic whiplash and neck-pain patients.[81] A later review article also emphasized the benefit of an exercise program including neck strengthening and stretching for chronic WAD patients.[82]

Conclusion

Not every person with whiplash injury has concussion symptoms, and not every concussion case has a cervical proprioception problem. An optometric assessment of a patient presenting with post-concussion vision symptoms should include tests to determine whether there is a contributing cervical afferent problem. The management of visual symptoms and findings contributed by a cervical problem is best addressed through a multi-disciplinary effort involving both optometrists and physical therapists. Given the benefits of both cervical and visual interventions in concussion injury, a sound approach to deal with many symptoms and signs of whiplash trauma would ideally consist of a joint rehabilitation effort addressing vision, vestibular, and cervical proprioception pathways. Future studies investigating a multi-disciplinary collaborative sensorimotor rehabilitation program consisting of both optometric and physical therapy would greatly benefit this population.

Acknowledgements

Willard Bleything, OD, Fellowship Committee of the Neuro-Optometric Rehabilitation Association, and Cameron Marshall, DC for their editing contributions.

Disclosure

This author has no financial arrangements or commercial associations that may be perceived as conflict of interest in connection with this paper.

References

1. Montfoort I, Kelders WP, Van der Geest JN, Schipper IB, et al. Interaction between ocular stabilization reflexes in patients with whiplash injury. Invest Ophthalmol Vis Sci 2006;47(7):2881-4. http://bit.ly/2PHHcXz
2. Tanaka N, Atesok K, Nakanishi K, Kamei N, et al. Pathology and treatment of traumatic cervical spine syndrome: Whiplash injury. Adv Orthop 2018: Published online 2018 Feb 28. doi:10.1155/2018/4765050.
3. Jang SH, Kwon YH. A review of traumatic axonal injury following whiplash injury as demonstrated by diffuse tensor tractography. Front Neurol 2018;9:57. http://bit.ly/2s3hSCb
4. Ellis BD, Kosmorsky GS. Referred ocular pain relieved by suboccipital injection. Headache 1995;35(2):101-3. http://bit.ly/2PExwgk
5. Tseng SC, Cheng AM, Fu Y. Retrospective study to identify trigeminal-cervical ocular referred pain as a new causative entity of ocular pain. J Pain Res 2017;10:1747–54. Published 2017 Jul 25. doi:10.2147/JPR.S140895.
6. Esterov D, Greenwald BD. Autonomic dysfunction after mild traumatic brain injury. Brain Sci 2017;7(8):100. http://bit.ly/36OBf0D
7. Edwards IJ, Lall VK, Paton JF, Yanagawa Y, et al. Neck muscle afferents influence oromotor and cardiorespiratory brainstem neural circuits. Brain Struct Funct 2015;220(3):1421-36. http://bit.ly/2PDY80X
8. Truong JQ, Ciuffreda KJ. Comparison of pupillary dynamics to light in the mild traumatic brain injury (mTBI) and normal populations. Brain Inj 2016;30(11):1378-89. http://bit.ly/35QWzSQ
9. Truong JQ, Ciuffreda KJ. Objective pupillary correlates of photosensitivity in the normal and mild traumatic brain injury populations. Mil Med 2016;181(10):1382-90. http://bit.ly/2EB5kEJ

10. Ciuffreda KJ, Joshi NR, Truong JQ. Understanding the effects of mild traumatic brain injury on the pupillary light reflex. Concussion 2017;2(3):CNC36. doi:10.2217/cnc-2016-0029.

11. Ciuffreda KJ, Joshi NR. Light on the pupil and concussion: A perspective. Vision Dev & Rehab 2017;3(3):194-6. http://bit.ly/2Q3zBBp

12. Okutucu S, Civelekler M, Sabanoglu C, Aparci M, et al. Assessment of the relationship between dynamic pupillometry and exercise heart rate recovery among healthy subjects. Eur Rev Med Pharmacol Sci 2016;20(7):1344-9. http://bit.ly/2Me4geb

13. Okutucu S, Civelekler M, Aparci M, Sabanoglu C, et al. Computerized dynamic pupillometry indices mirrors the heart rate variability parameters. Eur Rev Med Pharmacol Sci 2016;20(10):2099-105. http://bit.ly/2Z7tW1o

14. Koenig J, De Kooning M, Bernardi A, Williams DP, et al. Lower resting state heart rate variability relates to high pain catastrophizing in patients with chronic whiplash-associated disorders and healthy controls. Pain Pract 2016;16(8):1048-53. http://bit.ly/35F79wf

15. Treleaven J, Takasaki H. Characteristics of visual disturbances reported by subjects with neck pain. Man Ther 2014;19(3):203-7. http://bit.ly/2ECMA82

16. Centers for Disease Control and Prevention [homepage on the Internet]; last updated February 12, 2019. Available from: http://bit.ly/2tdHmgr

17. Hynes LM, Dickey JP. Is there a relationship between whiplash-associated disorders and concussion in hockey? A preliminary study. Brain Inj 2006;20(20):179-88. http://bit.ly/34EFiec

18. Beeckmans K, Crunelle C, Van Ingelgom S, Michiels K, et al. Persistent cognitive deficits after whiplash injury: A comparative study with mild traumatic brain injury patients and healthy volunteers. Acta Neurol Belg 2017;117(2):493-500. http://bit.ly/2Z5Mnn2

19. Mani R, Asper L, Khuu SK. Deficits in saccades and smooth-pursuit eye movements in adults with traumatic brain injury: A systematic review and meta-analysis. Brain Inj 2018;32(11):1315-36. http://bit.ly/2sFM8D7

20. Abrahams VC, Rose PK. Projections of extraocular, neck muscle, and retinal afferents to superior colliculus in the cat: Their connections to cells of origin of tectospinal tract. J Neurophysiol 1975;38(1):10-8. http://bit.ly/2rUfvSl

21. Liu CS, Bryan RN, Woo JH, Liu GT, et al. Magnocellular and parvocellular visual pathways have different blood oxygen level-dependent signal time courses in human primary visual cortex. Am J Neuroradiol 2006;27(8):1628-34. http://bit.ly/2s7gizh

22. Steenerson K, Starling AJ. Pathophysiology of sports-related concussion. Neurol Clin 2017;35(3):403-8. http://bit.ly/2Z3sUn6

23. Vállez G, Doorduin J, Willemsen AT, Dierckx RA, et al. Altered regional cerebral blood flow in chronic whiplash associated disorders. EBioMedicine 2016;10:249-57. http://bit.ly/2SabQKw

24. Womack KB, Paliotta C, Strain JF, Ho JS, et al. Measurement of peripheral vision reaction time identifies white matter disruption in patients with mild traumatic brain injury. J Neurotrauma 2017;34(8):1539-45. http://bit.ly/2Q298nR

25. Clark JF, Ellis JK, Burns TM, Childress JM, et al. Analysis of central and peripheral vision reaction times in patients with postconcussion visual dysfunction. Clin J Sport Med 2017;27(5):457-61. http://bit.ly/2PE0k8H

26. Mani R, Asper L, Khuu SK. Deficits in saccades and smooth-pursuit eye movements in adults with traumatic brain injury: A systematic review and meta-analysis. Brain Inj 2018;32(11):1315-36. http://bit.ly/2sFM8D7

27. Patel R, Ciuffreda KJ, Tannen B, Kapoor N. Elevated coherent motion thresholds in mild traumatic brain injury. Optometry 2011;82(5):284-9. http://bit.ly/2tCEWs5

28. Poltavski D, Lederer P, Cox LK. Visually evoked potential markers of concussion history in patients with convergence insufficiency. Optom Vis Sci 2017;90(7):742-50. http://bit.ly/2sGrov8

29. Ischebeck BK, de Vries J, Janssen M, van Wingerden JP, et al. Eye stabilization reflexes in traumatic and non-traumatic chronic neck pain patients. Musculoskelet Sci Pract 2017;29:72-7. http://bit.ly/2ECkbyG

30. Ischebeck BK, de Vries J, Van der Geest JN, Janssen M, et al. Eye movements in patients with whiplash associated disorders: A systematic review. BMC Musculoskelet Disord 2016;17(1):441. http://bit.ly/2EC4mYY

31. Treleaven J. Dizziness, unsteadiness, visual disturbances, and sensorimotor control in traumatic neck pain. J Orthop Sports Phys Ther 2017;47(7):492-502. http://bit.ly/35A7KiR

32. Treleaven J, Jull G, Grip H. Head eye co-ordination and gaze stability in subjects with persistent whiplash associated disorders. Man Ther 2011;16(3):252-7. http://bit.ly/35CzH9S

33. Schneider KJ, Meeuwisse WH, Palacios-Derflingher L, et al. Changes in measures of cervical, vestibular, dynamic balance, and tasks of divided attention following sport-related concussions in elite youth ice hockey players. J Orthop Sports Phys Ther 2018;48(12):974-81. http://bit.ly/34xrQJi

34. Kelders WPA, Kleinrensink GJ, Van der Geest JN, Feenstra L, et al. Compensatory increase of the cervico-ocular reflex with age in healthy humans. J Physiol 2003;553(Pt 1):311-7. http://bit.ly/2Z5O4ko

35. Lee MH, Durnford SJ, Crowley JS, Rupert AH. Visual vestibular interaction in the dynamic visual acuity test during voluntary head rotation. Aviat Space Environ Med 1997;68(2):111-7. http://bit.ly/2Z5R1S5

36. Marquez C, Lininger M, Raab S. Establishing normative change values in visual acuity loss during the dynamic visual acuity test. Int J Sports Phys Ther 2017;12(2):227-32. http://bit.ly/2Q4D7eO

37. Vital D, Hegemann SC, Straumann D, Bergamin O, et al. A new dynamic visual acuity test to assess peripheral vestibular function. Arch Otolaryngol Head Neck Surg 2010;136(7):686-91. http://bit.ly/2EAVcfq

38. Kristjansson E, Treleaven J. Sensorimotor function and dizziness in neck pain: Implications for assessment and management. J Orthop Sports Phys Ther 2009;39(5):364-77. http://bit.ly/34CFSJH

39. Ciuffreda KJ, Kapoor N, Rutner D, Suchoff IB, et al. Occurrence of oculomotor dysfunctions in acquired brain injury: A retrospective analysis. Optometry 2007;78(4):155-61. http://bit.ly/2XDgx2N

40. Trieu LH, Lavrich JB. Current concepts in convergence insufficiency. Curr Opin Ophthalmol 2018;29(5):401-6. http://bit.ly/34EIJBQ

41. Badovinac SD, Quaid P, Hutchison MG. Prevalence of oculomotor dysfunction in healthy athletes preseason: Implications for concussion in sport. Vis Devel Rehabil 2017;3(2):75-88. http://bit.ly/2BHG12Q

42. Van Horn MR, Waltzman DM, Cullen KE. Vergence neurons identified in the rostral superior colliculus code smooth eye movements in 3D space. J Neurosci 2013;33(17):7274-84. http://bit.ly/34H8S2O

43. Suzuki S, Suzuki Y, Ohtsuka K. Convergence eye movements evoked by microstimulation of the rostral superior colliculus in the cat. Neurosci Res 2004;49(1):39-45. http://bit.ly/2PFInIb

44. DuPrey KM, Webner D, Lyons A, Kucuk CH, et al. Convergence insufficiency identifies athletes at risk of prolonged recovery from sport-related concussion. Am J Sports Med 2017;45(10):2388-93. http://bit.ly/35FGBeu

45. Pearce KL, Sufrinko A, Lau BC, Henry L, et al. Near point of convergence after a sport-related concussion: Measurement reliability and relationship to neurocognitive impairment and symptoms. Am J Sports Med 2015;43(12):3055-61. http://bit.ly/2PGeC8E

46. Howell DR, O'Brien MJ, Raghuram A, Shah AS, et al. Near point of convergence and gait deficits in adolescents after sport-related concussion. Clin J Sport Med 2018;28(3):262-7. http://bit.ly/2Ex9nBY

47. Giffard P, Daly L, Treleaven J. Influence of neck torsion on near point of convergence in subjects with idiopathic neck pain. Musculoskelet Sci Pract 2017;32:51-6. http://bit.ly/2Z89H3q

48. Steibel-Kalish H, Amitai A, Mimouni M, Bach M, et al. The discrepancy between subjective and objective measures of convergence insufficiency in whiplash-associated disorder versus control participants. Ophthalmol 2018;125(6):924-8. http://bit.ly/2Ez5sEC

49. Pang Y, Gabriel H, Frantz KA, Saeed F. A prospective study of different test targets for the near point of convergence. Ophthalmic Physiol Opt 2010;30(3):298-303. http://bit.ly/2reuPc2

50. Mosimann UP, Muri RM, Felblinger J, Radanov BP. Saccadic eye movement disturbances in whiplash patients with persistent complaints. Brain 2000;123(4):828-35. http://bit.ly/2EB7lB2

51. Fischer TD, Red SD, Chuang AZ, Jones EB, et al. Detection of subtle cognitive changes after mTBI using a novel tablet-based task. J Neurotrauma 2016;33(13):1237-46. http://bit.ly/2sLp5GS

52. Born RT, Pack CC, Zhao R. Integration of motion cues for the initiation of smooth pursuit eye movements. In: Hyona J, Munoz DP, Heide W, Radach R. (eds). Progress in Brain Research. Boston, MA: Elsevier Science B.V., 2002:225-37. http://bit.ly/35H87Ir

53. Krauzlis RJ. Neuronal activity in the rostral superior colliculus related to the initiation of pursuit and saccadic eye movements. J Neurosci 2003;23(10):4333-44. http://bit.ly/2Z6vZ5F

54. Moschner C, Crawford TJ, Heide W, Trillenberg P, et al. Deficits of smooth pursuit initiation in patients with degenerative cerebellar lesions. Brain 1999;122(11):2147-58. http://bit.ly/2PCNelM

55. Heikkila HV, Wenngren BI. Cervicocephalic kinesthetic sensibility, active range of cervical motion, and oculomotor function in patients with whiplash injury. Arch Phys Med Rehabil 1998;79(9):1089-94. http://bit.ly/2Z69XA9

56. Treleaven J, Jull G, LowChoy N. The relationship of cervical joint position error to balance and eye movement disturbances in persistent whiplash. Man Ther 2006;11(2):99-106. http://bit.ly/2raSSIR

57. Kongsted A, Jorgensen LV, Leboeuf-Yide C, Qerama E, et al. Are altered smooth pursuit eye movements related to chronic pain and disability following whiplash injuries? A prospective trial with one-year follow-up. Clin Rehabil 2008;22(5):469-79. http://bit.ly/2Z4PZ93

58. Treleaven J, Jull G, LowChoy N. Smooth pursuit neck torsion test in whiplash-associated disorders: Relationship to self-reports of neck pain and disability, dizziness and anxiety. J Rehabil Med 2005;37(4):219-23. http://bit.ly/35FuE8r

59. Daly L, Giffard P, Thomas L, Treleaven J. Validity of clinical measures of smooth pursuit eye movement control in patients with idiopathic neck pain. Musculoskeletal Sci Pract 2018;33:18-23. http://bit.ly/2Exh8YN

60. Janssen M, Ischebeck BC, de Vries J, Kleinrensiink GJ, et al. Smooth pursuit eye movement deficits in patients with whiplash and neck pain are modulated by target predictability. Spine 2015;40(19):E1052-7. http://bit.ly/35GYY2q

61. Tjell C, Rosenthal U. Smooth pursuit neck torsion test: A specific test for cervical dizziness. Am J Otol 1998;19(1):76-81. http://bit.ly/2Z3TWuD

62. Bove M, Fenoggio C, Tacchino A, Pelosin E, et al. Interaction between vision and neck proprioception in the control of stance. Neurosci 2009;164(4):1601-8. http://bit.ly/34AklBr

63. Gomez S, Patel M, Magnusson M, Johansson L, et al. Differences between body movement adaptation to calf and neck muscle vibratory proprioceptive stimulation. Gait Posture 2009;30(1):93-9. http://bit.ly/2sGNE8a

64. Kulkami V, Chandy MJ, Babu KS. Quantitative study of muscle spindles in suboccipital muscles of human foetuses. Neurol India 2001;4:355-9. http://bit.ly/34AN7BL

65. Ivanenko YP, Grasso R, Lacquaniti F. Effect of gaze on postural responses to neck proprioceptive and vestibular stimulation in humans. J Physiol 1999;519(1):301-14. http://bit.ly/2sOeYkA

66. Roll JP, Vedel JP, Roll R. Eye, head and skeletal muscle spindle feedback in the elaboration of body references. Prog Brain Res 1989;80:113-23. http://bit.ly/35Ls8he

67. Bianco A, Pomara F, Petrucci M, Battaglia G. Postural stability in subjects with whiplash injury symptoms: Results of a pilot study. Acta Otolaryngol 2014;134(9):947-51. http://bit.ly/2r6Fy8a

68. Yu LJ, Stokell R, Treleaven J. The effect of neck torsion on postural stability in subjects with persistent whiplash. Man Ther 2011;16(4):339-43. http://bit.ly/2Z8hD56

69. Padula WV, Argyris S. Post trauma vision syndrome and visual midline shift syndrome. Neuro Rehabil 1996;6(3):165-71. http://bit.ly/2sJS9yN

70. Tong D, Cao J, Beaudry A, Lin E. High prevalence of visual midline shift syndrome in TBI: A retrospective study. Vis Devel Rehabil 2016;2(3):176-82. http://bit.ly/2ZgJJLz

71. Padula WV, Subramanian P, Spurling A, Jenness J. Risk of fall (RoF) intervention by affecting visual egocenter through gait analysis and yoked prisms. NeuroRehabil 2015;37(2):305-14. http://bit.ly/2Z5cgn0

72. Thiagarajan P, Ciuffreda KJ, Capo-Aponte JE, Ludlam DP, et al. Oculomotor neurorehabilitation for reading in mild traumatic brain injury (mTBI): An integrative approach. NeuroRehabil 2014;34(1):129-46. http://bit.ly/35HSPmW

73. Yadav NK, Thiagarajan P, Ciuffreda KJ. Effect of oculomotor vision rehabilitation on the visual-evoked potential and visual attention in mild traumatic brain injury. Brain Inj 2014;28(7):922-9. http://bit.ly/3037UMi

74. Ciuffreda KJ, Rutner D, Kapoor N, Suchoff IB, et al. Vision therapy for oculomotor dysfunctions in acquired brain injury: A retrospective analysis. Optometry 2008;79(1): 18-22. http://bit.ly/2ExhD54

75. Thiagarajan P, Ciuffreda KJ. Versional eye tracking in mild traumatic brain injury (mTBI): Effects of oculomotor training. Brain Inj 2014;28(7):930-43. http://bit.ly/2EBZOBK

76. Convergence Insufficiency Treatment Trial Study Group. Randomized clinical trial of treatments for symptomatic convergence insufficiency in children. Arch Ophthalmol 2008; 126(10):1336-49. http://bit.ly/2TTBod3

77. Gallaway M, Scheiman M, Mitchell GL. Vision therapy for post-concussion vision disorders. Optom Vis Sci 2017;94(1):68-73. http://bit.ly/2ECqIcQ

78. Figueiro MG, Rea MS. The effects of red and blue lights on circadian variations in cortisol, alpha amylase, and melatonin. Int J Endocrinol 2010. doi:10.1155/2010/829351

79. Choi CJ, Kim K-S, Kim CM, Kim SH, et al. Reactivity of heart rate variability after exposure to colored lights in healthy adults with symptoms of anxiety and depression. Int J Psychophysiol 2011;79(2):83-8. http://bit.ly/2ECqIcQ

80. Schafer A, Kratky KW. The effect of colored illumination on heart rate variability. Forsch Komplementmed 2006; 13(3):167-73. http://bit.ly/34D2NEO

81. Bussieres AE, Stewart G, Al-Zoubi F, Decina P, et al. The treatment of neck-pain associated disorders and whiplash-associated disorders: A clinical practice guideline. J Manipulative Physiol Ther 2016;39(8):523-64. http://bit.ly/2EC0hnu

82. Anderson C, Yeung E, Tong T, Reed N. A narrative review on cervical interventions in adults with chronic whiplash-associated disorder [published correction appears in BMJ Open Sport Exerc Med. 2018 Aug 21;4(1):e000299corr1]. BMJ Open Sport Exerc Med 2018:4(1):e000299. doi:10.1136/bmjsem-2017-000299.

Correspondence regarding this article should be emailed to Shirley Blanc, OD at sblanc.opt@gmail.com.

Blanc S. The influence of altered cervical input from whiplash injury on post-concussion ocular/visual signs and symptoms. Optom Vis Perf 2019;7(5/6):257-68.