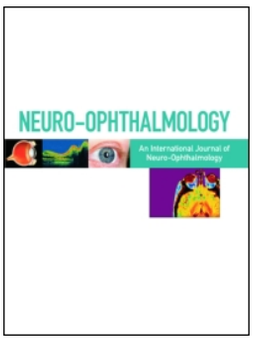
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Correlation between Ocular and Vestibular Abnormalities and Convergence Insufficiency in Post-Concussion Syndrome

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ABSTRACT

The vestibular and oculomotor/visual systems are commonly affected in post-concussion syndrome (PCS). Convergence insufficiency (CI) is the most common ocular abnormality after concussion. Electrovestibulography (EVestG) is a relatively new non-invasive method that measures the peripheral vestibular responses; it has shown abnormal vestibular responses in a PCS. Here, we report the results of investigating the correlation between the vestibular and oculomotor systems in PCS population using EVestG and CI measures. Forty-eight PCS patients were tested using EVestG, out of which 20 also completed the Rivermead post-concussion questionnaire (RPQ). An EVestG feature (Field Potential (FP)-area) was extracted from the stationary part of the EVestG signals. A neuro-ophthalmologist (author BM) measured participants' CI at near vision using cross-cover examination and a prism-bar. Results indicate: (1) vestibular abnormality (i.e. FP-area) and CI values are significantly correlated in PCS ($R = 0.68$, $p < .01$), and (2) there are significant correlations between severity of concussion (i.e. RPQ3) and CI ($R = 0.70$, $p < .01$) and between RPQ3 and FP-area ($R = -0.56$, $p < .02$). To the best of our knowledge, this is the first study that objectively demonstrates a significant positive correlation between the CI and vestibular systems' abnormality. These findings are scientifically important as they help localise the pathology of PCS, and are clinically valuable as they help physicians in their decision-making about PCS diagnosis and rehabilitation strategies.

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Introduction

Annually, an estimated 42 million people worldwide suffer mild traumatic brain injury (mTBI), also known as concussion[1] (mTBI and concussion are commonly used interchangeably). Concussion may occur following a transient alteration of consciousness after a biomechanical force to the head (e.g., sports injuries, falls, motor vehicle accidents, etc.). Concussion can be followed by some transient neurological symptoms such as headache, nausea, vomiting, photophobia, imbalance, double/blurred vision, irritability, etc. Usually, these symptoms recover in 2–4 weeks.[2–4] However, if the symptoms do not recover within an expected period of time, which has been proposed from 2 to 12 weeks but generally accepted as 4 weeks[2], the neurological condition is called post-concussion syndrome (PCS). Patients with PCS may show a cluster of cognitive, somatic and emotional symptoms for an extended period of time after the head trauma. While PCS usually resolves within 3 months after the injury[5], 5% to 15% of the PCS population may carry their symptoms and functional impairment for many more months and even years[6,7]; in that case, they are diagnosed with persistent PCS. Persistent PCS impacts and disrupts patients' personal and social life significantly.[8,9]

Ocular system in PCS

Early and accurate objective diagnosis of concussion and PCS has been the topic of many recent basic science and clinical studies.[10–12] Several studies have shown that eye movements are abnormal in patients with PCS[13–16], and many research groups have tried

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to use these abnormalities to develop algorithms for objective diagnosis of PCS.[17,18]

Patients with concussion can experience acute and chronic visual and ocular symptoms because of the damage to cortical, subcortical or brainstem visual/ocular pathways.[13] Of the four stereotyped eye movements (i.e., saccade, smooth pursuit, vergence, and vestibule-ocular reflex [VOR]) vergence, particularly convergence, is most affected in concussed patients. Despite the fact that convergence insufficiency (CI) seems to be the hallmark of ocular abnormalities in PCS[16,19–22], its use in automated and clinical diagnosis of concussion has been underappreciated. For example, the current two commercially available eye-tracking systems for diagnosis of concussion and PCS have used the accuracy of the coordination of the movements of the two eyes together[17] or the abnormal smooth pursuit.[18] Many concussion clinics are either unaware or unequipped to measure convergence and other eye movement deficits in concussion. Thus, these abnormalities are left unrecognised unless the patients with PCS are examined by the few optometrists, ophthalmologists, or neuro-ophthalmologists who have experience in concussion.

Vestibular system in PCS

There is abundant literature indicating that vestibular system is abnormal in patients with PCS.[23–25] After a head injury, concussed patients often experience dizziness and imbalance. Dizziness is a vague term in neurological symptomatology. Neurologists often try to relate the dizziness to other better understood neurological symptoms such as vertigo, imbalance, syncope, etc. However, sometimes the dizziness in concussion does not fall under any other categories of better-known neurological symptoms. The cause of dizziness in PCS is not completely understood; however, plausible causes are direct damage to the vestibular system, unilateral vestibular nerve injury, structural central nervous system damage[26], or ocular sensorimotor abnormalities such as vergence dysfunction.[27,28] Although the link between visual/ocular and vestibular systems is well established[29], the correlation between vestibular and ocular abnormalities in PCS has not been well studied. This study aims to explore the close relationship between the abnormalities of these two systems in a PCS population.

The vestibular nuclei integrate the efferent signals from the central nervous system and send them to the vestibular periphery (semicircular canals and otolith organs) that modulates hair cell firings and afferent vestibular signals.[30] We study vestibular function with electrovestibulography (EVestG) technology (author BL is consultant to the supporting company, Neural Diagnostics Pty. Ltd.) that measures the spontaneous and activity driven vestibulo-acoustic, predominantly vestibular activity.[31,32] It has been hypothesised that EVestG can pick up and analyse efferent signals showing the status of central and peripheral vestibular systems. Recent studies have shown that EVestG can be used as a diagnostic tool for PCS[25,33–35] and its comorbidities such as depression[36] as well as following its recovery.[37]

Vergence eye movement control in the central nervous system

Recent primate studies on rhesus monkeys have shown that the pathways shown in Figure 1 are involved in controlling vergence (i.e., convergence and divergence). Convergence can occur voluntarily or in response to a near visual stimulus. In the latter, visual information originating from the retina reaches the primary visual cortex (striate cortex) through the lateral geniculate nucleus (LGN) of the thalamus; then, it projects to the extra-striate cortex,

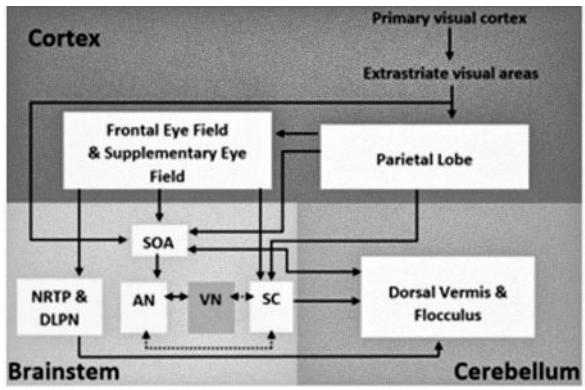


Figure 1. A diagram describes the brain regions and selected pathways involved in generating a vergence eye movement along with associated vestibular connections. SOA, Supraoculomotor area; NRTP, Nucleus reticularis tegmenti pontis; DLPN, Dorsolateral pontine nucleus; AN, Abducens nucleus; VN, Vestibular nucleus; SC, Superior colliculus (Dashed lines refer to indirect connections and solid lines refer to direct connections).

wherein it branches into two brain regions responsible for producing vergence eye movements: (1) The parietal cortex, which in turn sends fibres to the frontal eye field (FEF) areas in the frontal lobe, and the nucleus reticularis tegmenti pontis (NRTP) in the pons. It has been shown in human studies that acquired cerebral lesions, e.g., parietal lobe damage, can cause fusional convergence abnormality.[38,39] Studies in patients who suffered from stroke have shown that damage to the NRTP in the pons caused vergence dysfunction as well[40]; (2) The supraoculomotor area (SOA) in the midbrain, which controls some of the actions of the medial recti muscles. Those are the main generators of convergence eye movements. It is important to note that in order for convergence to occur, eye abduction (i.e., rolling the eyes out) must be relaxed at the same time when adduction (i.e., rolling the eyes in) starts.

While near vision convergence or distance vision divergence activates a complex neural circuit that passes through the occipital cortex, voluntary vergence is initiated at the FEF. The main function of the FEF is to generate saccades, but it also contains neurons that are involved in the control of smooth pursuit and vergence eye movements.[29,41,42] The FEF performs its function through direct projection to the superior colliculus and indirect projection to the superior colliculus via the basal ganglia. The FEF also has direct projections to the pulvinar nucleus, supplementary eye field, thalamus, brainstem reticular formation and cerebellum, which are closely related to different ocular movements.[43]

The SOA is located immediately posterior to the oculomotor nuclear complex in the periaqueductal grey area and provides signals to manage near vision, i.e., controlling accommodation, pupil size, and convergence.[43] Functional MRI studies have shown that during convergence, neural activity increases in vergence-sensitive cells in the SOA and NRTP,[44–46] and more interestingly, signals in the SOA are significantly reduced in patients with traumatic brain injury.[47]

Neural correlate of convergence insufficiency and link to vestibular system

The anatomical connections between the vestibular system and vergence eye movement centres are complex (Figure 1, for details, see (Leigh and Zee 2015)[29]); it includes

(1) The cerebellum is closely interconnected with the vestibular system and FEF/SOA areas have direct and indirect connections to the cerebellar cortex. The cerebellum plays an important role in vergence eye movements[48]
(2) The superior colliculus plays an important role in fine-tuning eye movements as it encompasses a topographical map of the visual field of view.[49] The superior colliculus is closely connected to the FEF and makes connections with the vestibular nuclei through the medial longitudinal fasciculus (MLF). Importantly, vestibular and ocular sensorimotor signals integrate within the superior colliculi. The MLF plays a significant role in ipsilateral eye adduction. Damage to the MLF causes loss of the ability to adduct the contralateral eye, which is known as internuclear ophthalmoplegia (INO).[50]
(3) The interconnection between the vestibular nucleus and nucleolus prepositus hypoglossi is important in vergence eye movements.[51] This was shown by the Gamlin group (1989) after they performed a single-unit neuron study in alert Rhesus monkeys to characterise the vergence eye movement signals carried by abducens (6th cranial nerve) internuclear neurons. They found that activity reduced more for convergence than any other recorded abducens neurons
(4) The SOA along with other brain regions that are engaged in convergence eye movements, such as the pulvinar and extraocular muscles' nuclei (i.e., cranial nerves 3, 4 and 6), have neural connections to the vestibular nuclei and other vestibular-processing brain regions, e.g., the flocculo-nodular lobe and the immediately adjacent vermis part of the cerebellum.[47]

Based on the above brief review, we hypothesise that the vestibular function measured by the features extracted from the EVestG[31] in the PCS population, is correlated with CI.

The highlights of the connections of vestibular and ocular systems and abnormalities in PCS are the:

(1) Prevalence of vestibular dysfunction and CI in concussion: From an epidemiological point of view, there is evidence showing increased prevalence of convergence abnormality in PCS; 90% of patients with concussion have one or more ocular sensorimotor dysfunctions[19] and the prevalence of convergence dysfunction ranges between 40% and 42%[19,52] in PCS versus 0.5–5% in the normal population. Vestibular dysfunction is also common in the PCS population.[25]
(2) Anatomic connections between the ocular and vestibular systems: The vestibular and visual/ocular systems have mutual interactions and anatomical connections in the brain. In particular, the cortical regions responsible for vergence eye movements are closely linked to the vestibular system.
(3) Common symptoms of vestibular and ocular dysfunction in PCS: Dizziness and imbalance are common symptoms of PCS and are likely related to abnormalities in vestibular/cerebellar systems, which are then closely linked with the pathways responsible for ocular sensorimotor functions and particularly with vergence eye movements.

The objectives of this study were to investigate: (1) whether the EVestG extracted features characterising the PCS population[25] are also correlated with CI and its severity, (2) if the EVestG features used to characterise PCS are different between PCS populations with and without CI, and (3) if there is a correlation between CI and the RPQ (post-concussion questionnaire) and a correlation between CI and the EVestG FP-area feature.

Methodology

Participants and assessments

Concussed participants were recruited from the pool of patients visiting their treating neuro-ophthalmologist (author BM); they were diagnosed with PCS, which included the diagnosis of CI, vertigo or imbalance without any other neurological disorders that could explain or confound their diagnosis. Forty-eight individuals (16 males, 44.5 ± 14.9 years) with PCS (Table 1) were tested, out of which 10 (three males, 43.2 ± 18.5 years) had short-term PCS (SPCS-concussion <3 months prior to testing) and 38 patients (13 males, 44.8 ± 13.7 years) had long-term PCS (LPCS-concussion >3 months prior to testing).

The duration between the head injury and the recording date for the majority of the LPCS patients was between 3 months and 5 years with the exception of two patients who remained symptomatic for more than 10 years prior to testing. Some data were adopted from a previous study.[36] The participants were recorded at the Neural Diagnostic Laboratory, Riverview Health Centre, Winnipeg, Manitoba. They completed comprehensive neuropsychological assessments by a neurologist and neuro-ophthalmologist in addition to the RPQ post-concussion questionnaire test and a screening hearing test.

This study was approved by the University of Manitoba Biomedical Research Ethics Board, and

Table 1. Demographics table (*p < .05, **p < .01) *Clinically extraocular motility < 4 prism dioptre (PD) considered normal CI and its measure was set as zero. SD = standard deviation.

Descriptive variables	PCS (*n*= 48)	PCS without CI (*n*= 20)	PCS with CI (*n* = 28)	PCS with no depression	PCS with mild depression	PCS with moderate/severe depression
Sex, female	32	14	18	10	12	10
Age (SD)	44.5 (14.9)	46.0 (12.2)	43.3 (16.4)	38.4 (16.6)	45.7 (15.4)	48.4 (9.7)
Time since injury (years [SD])	2.2 (3.8)	1.7 (1.4)	2.8 (5.1)	0.9 (0.6)	1.7 (1.7)	2.1 (1.5)
CI (SD)	– – -	0*	8.2 (3.2)	7.5 (4.5)	5.2 (4.5)	1.6 (3.0)
FP-area (SD)	32.4 (10.2)	40.0 (8.9)	27.0 (7.1)	25.4 (5.7)	28.6 (5.6)	41.0 (7.7)
RPQ3 (SD)	7.3 (2.6)	5.0 (2.6)	8.4 (1.9)	7.5 (1.9)	7.4 (2.8)	6.7 (3.1)
RPQ13 (SD)	28.3 (11.2)	26.7 (14.3)	29.0 (9.4)	22 (11.4)	28.0 (9.4)	38.5 (6.3)

all the participants signed an informed consent form prior to the experiment.

Convergence insufficiency measurement

The CI was measured at near vision with a cross-cover test using a prism bar by a neuro-ophthalmologist for all participants. Cross-cover test is an objective measurment and the gold standard in measuring ocular misalignment (i.e., strabismus), or vertical/horizontal deviation of the eyes[53] (for details see Appendix A).

EVestG recording

A typical EVestG recording procedure comprises placing active and reference electrodes. Active electrodes (TM-EcochGtrode, Bio-logic, France(Figure 2a) were rested close to the tympanic membrane of each ear (Figure 2b). Identical reference electrodes were placed on the entrance of each ear canal. One common ground electrode (Biopac EL258S) was placed on the forehead. The EVestG was conducted with the eyes closed and head supported to minimise muscle artefacts on a hydraulic chair inside an electromagnetically shielded and sound attenuated (>30 dB) chamber (see Appendix A).

The recording was performed whilst the chair was both stationary and moving. However, we only analysed the stationary segments to minimise the body movement artefacts that resulted from the tilting. Previously, we showed that the analysis of the stationary segment of the data of our results could separate PCS and healthy control populations.[25]

To produce an average FP plot, shown in (Figure 3), the wavelet-based signal processing technique called Neural Event Extraction Routine (NEER)[31] averaged the detected spontaneous and driven FPs buried in the noise.

Neuropsychological assessments

The Rivermead Post-Concussion Questionnaire (RPG) score was used for calculating the severity of the PCS.[54,55] The questionnaire consists of 16 post-concussion symptoms and for each symptom, there is a score from 0 to 4 as an indication of the severity of the symptom. In this study, we divided the RPQ score into two sub-scores to achieve unidimensional constructs[54]: (1) RPQ-3 is the score of the first three symptoms of RPQ (i.e., headaches, dizziness and nausea), which are categorised as the most common symptoms in concussion[54], (2) RPQ-13 is the score of the 13 symptoms, that are common symptoms in prolonged PCS.[54] The RPQ-13 includes four questions focusing on depression and mood as well.

Results

Screening hearing tests showed that all patients had normal hearing. Figure 3 shows an example of an averaged FP extracted from EVestG signal during the background phase. The average extracted amplitude of the FP was normalised to −1 as it can differ according to the electrode contact and placement (e.g., how close it is to the eardrum, etc.) thus facilitating comparison between participants. In our previous study[25], comparison of PCS and healthy control average FPs resulted in a significant ($p < .05$) difference between PCS and control groups in the action potential (AP) area (see Figure 3). AP area represents the area bounded between the baseline and the AP point as shown in Figure 3. In the current study, in addition to the AP area as a characteristic feature for separating PCS and healthy controls, we also

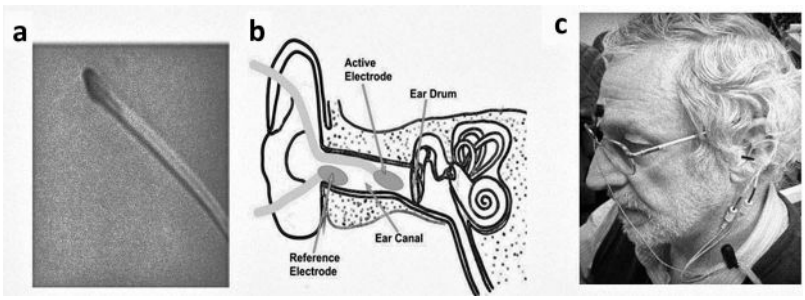


Figure 2. (a) Ear electrode; (b) electrodes placement; (c) participant connection.

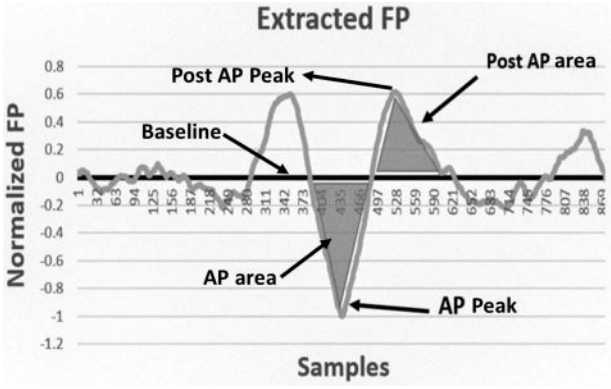


Figure 3. A typical normalised FP extracted from the EVestG signal during the background phase. AP area: the bounded area between the baseline and AP peak. Post AP area: the area bounded between the baseline and Post AP peak. The sum of the AP area and post AP area was used as a characteristic feature and called the FP-area. (Horizontal scale 44.1 samples = 1ms).

calculated the post-AP area (i.e., the area bounded between the baseline and the post-AP peak) of the average FP (see Figure 3). Then, the association between the combined areas and CI was calculated.

Figure 4a illustrates the correlation between the FP-area and the extraocular motility scale for CI including all short- and long-term injuries ($R = -0.68$, $p < .05$). When the PCS population was divided into SPCS and LPCS subgroups, the correlations between FP-area and CI were still significant for the SPCS ($R = -0.94$, $p < .01$) and LPCS ($R = -0.66$, $p < .01$) subgroups (Figure 4a).

Beside the FP, the NEER algorithm also provides the time of occurrence of each detected FP. Vestibular efferent spontaneous activity is usually seen in the range 10–50 spikes/s.[56] Thus, we also looked for modulations of efferent spontaneous FP interval activity in this low frequency range (~10 Hz). Since the average measured time gap that the NEER algorithm detects between two FPs is ~3.3 ms, a 33 FP gap corresponding to about ~100 ms (10 Hz)[57] was used. Therefore, average interval histograms based on the 33rd (IH33) FP gap from the signals of study participants were generated. Figure 5 shows the IH33 histogram of the 'PCS without CI' and 'PCS with CI' datasets with 90% confidence intervals (error bars). The IH33 is shifted to the right in individuals suffering 'PCS with CI' (longer time interval between firing) compared with those experiencing 'PCS without CI' (shorter time interval between firing).

Previous studies have shown that eye movement impairment after a head injury can vary between adolescent males and females[58], with females being more likely to develop eye movement impairments. Therefore, in order to test different factors which might lead to CI development in PCS patients, we divided our PCS data into three subgroups: (a) PCS with and without CI, (b) SPCS and LPCS, and (c) male and female; we considered each subgroup as an independent variable. Then, we used a univariate analysis to test the following measures in each of the sub-groups: (1) CI measured in prism dioptres (PD), (2) EVestG AP area feature, and (3) RPQ3 and RPQ13 scores, respectively.

For univariate tests, Mauchly's test indicated that the assumption of sphericity had not been violated for any of the dependent variables. Univariate analysis showed that the EVestG feature was significantly different ($p = .01$) between the PCS with and without CI

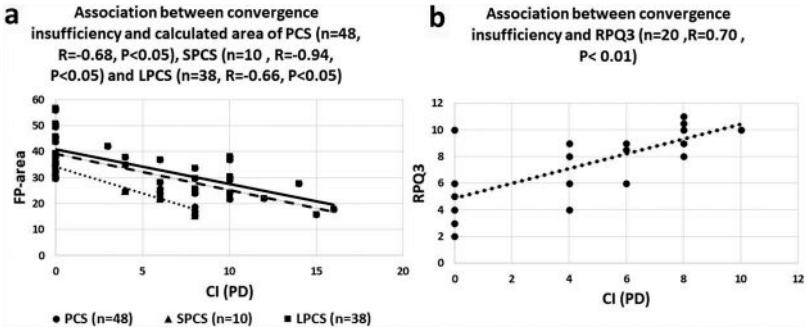


Figure 4. The calculated area of the extracted FP from the EVestG signal during background recording versus the extraocular motility scale (prism dioptre [PD]) for (a) All PCS participants ($n = 48$). PCS (dashed regression line), LPCS (solid regression line) and SPCS (dotted regression line). (b) The calculated score of the first three symptoms of RPQ (RPQ3) versus the extraocular motility scale (PD) for concussed participants ($n = 20$).

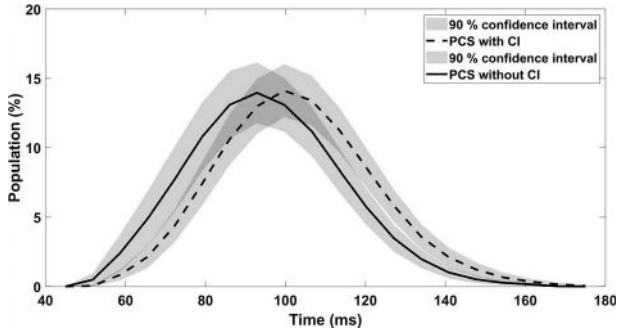


Figure 5. Interval Histogram for an FP gap equal to 33 FPs. The solid and dotted lines represent the PCS without CI (n = 20) and PCS with CI (n= 28) encased by dashed 90% confidence interval lines, respectively.

groups (F (1, 32) = 12.45, η^2 = 0.28). A significant difference (p = .04) was also obtained for the EVestG feature between the SPCS and LPCS groups (F (1, 32) = 4.36, η^2 = 0.12). No main effect for gender on the EVestG feature was obtained. No main effect was found for each independent variable (SPCS/LPCS, gender groups) for the CI measure.

Of the total 48 participants listed in our database, only the last 20 completed the RPQ test as the rest were previously recorded in another study[25] wherein RPQ was not included. For this sample, RPQ3 showed a significant correlation (R = 0.70, p < .01) with CI and FP-area (R = 0.56, p < .02) (Figure 4b). No significant correlation was found between RPQ13 and the CI or FP-area.

Discussion

CI is one of the most common ocular abnormalities in patients with concussion affecting their quality of life, functional abilities and causing difficulty with near vision activities such as reading and working with a cellphone.[16,19–21] In the introduction, we explained the complex neural network and the connections between the vestibular system and the brain centres that control convergence eye movement. PCS can manifest with vestibular, visual and/or ocular sensorimotor dysfunction. It is not clear whether CI in PCS is the direct result of vestibular abnormality or whether the vestibular abnormality and CI are both caused independently by concussion. There are many studies supporting the idea of using ocular sensorimotor dysfunction, and in particular CI assessment, for diagnosis of PCS[20,28]; however, there are some authors who disagree with this idea of using CI as a tool in the assessment of PCS patients due to the lack of objective evidence for a cause and effect relationship between PCS and CI.[59] It is conceivable that the lack of objective evidence for CI abnormality in PCS has been the main reason that, although CI has been shown to be measured automatically[73], it has not been used in automated ocular function tests for diagnosis of PCS, yet. In the current study, we investigated the objective association between CI and PCS using EVestG.

Our study results show a significant association between CI and the corresponding FP-area of EVestG. Our previous study[25] demonstrated that the AP-area could be used as a robust feature for differentiating between PCS patients and healthy controls (i.e., the AP-area of the PCS patients was always narrower than healthy controls). In this study, no significant association was found between CI and the AP-area; however, a significant association was found when the AP-area and the post-AP area were combined (Figure 3). Different areas of FP are the result of the activity within combinations of the vestibular periphery, vestibular nucleus (brainstem) and the efferent vestibular system (EVS). Hypothetically, the AP-area region of the FP is generated as a result of the efflux and influx of sodium and potassium ions through the membrane of the peripheral nerve[60] while the post-area region is generated as a result of the combination of signals in peripheral and brainstem vestibular system.[60–62] By adding the post-AP area, we arguably included an increased representation of the brainstem activity. The significant association of CI and FP only when the post-AP was added to the AP-area suggests a more dominant role for brainstem vestibular abnormality in CI.

In addition, it was shown in a previous study[25] that when the PCS participants were divided into two subgroups of SPCS and LPCS (i.e., short and long-term PCS), the AP-area of the SPCS subgroup was shown to be narrower on average than that of the LPCS subgroup.[25] Herein, splitting the PCS group into the SPCS and LPCS also produced a significant correlation between the FP-area and the CI measure for both subgroups (Figure 4a). As shown in Figure 4a, the SPCS subgroup still showed a smaller FP-area compared to LPCS subgroup. Moreover, the correlation coefficients found between the FP-area and the CI measure were negative; this indicates that a larger FP-area tended to be associated with a smaller CI measure.

This is consistent with our findings from a previous study[25], where the PCS population (mostly LPCS) with a larger FP-area (compared for SPCS) were shown to cluster closer to healthy controls.

The reduction or slowing of efferent activity that was depicted by IH33 changes (i.e., right shift in Figure 5) between 'PCS with CI' and 'PCS without CI' is hypothesised to be related to a reduction or slowing of efferent activity[25,57] and is in agreement with findings in a previous study[47] where activation of the brainstem oculomotor control nuclei was compared between PCS patients and healthy controls. In that study, they used functional magnetic resonance imaging and showed a reduction in the signals in areas that mediate vergence eye movements such as the superior colliculi, oculomotor nuclei, abducens nuclei, and supra-oculomotor area (SOA) in PCS patients.[47]

Our results also showed a significant correlation between CI and RPQ3 scores but not between CI and RPQ13 when the entire PCS population was considered. This finding was consistent with the results in another study where it was shown that the more CI, the higher severity of double vision/blurry vision at near range, headaches and dizziness.[63] This finding was very interesting as a significant correlation between CI and the RPQ3 (headaches, dizziness and nausea) can explain a possible cause for these common symptoms in concussion. These results are also consistent with the results from a previous study[28] in which vestibular/ocular motor screening (VOMS) was used to identify PCS. VOMS measures were positively correlated with the total score of the post-concussion symptom scale ($R = 0.65$, $P < .03$).

Although the cause of CI after concussion is still unclear, the high prevalence of CI among the concussion population is not surprising because: (1) regardless of the site of the head injury, it has been reported that the frontal cortex and subfrontal white matter, the deeper midline structures including the basal ganglia, diencephalon, rostral brainstem, and the temporal lobes, including the hippocampi are the most vulnerable brain regions to be damaged in concussion[64] and as explained in the introduction these areas are closely related to controlling vergence eye movements, (2) our EVestG results showed a difference in the efferent vestibular response of the PCS patients with CI compared with those without CI. Considering the feedback system between the vestibular nuclei (in the brainstem) and the periphery vestibular system (in the inner ear) through the efferent vestibular fibres, we hypothesise that the change in the vestibular response is most likely results from damage/change to the brainstem or peripheral vestibular system. Moreover, the convergence centres, i.e. the FEF is located in the frontal cortex, the relaying centre, i.e., the SOA is located in the brainstem, and (3) from an evolutionary point of view, vergence eye movements developed later in human compared with other types of eye movements.[65,66] As a result, the neural connections of vergence may not be as robust as they are in other eye movements (i.e. VOR or saccade). Therefore, the convergence system may be more vulnerable to physical impact and neural damage in PCS. Given the high prevalence of CI among the PCS population and associated functional impairment and symptoms, screening for CI has become a common practice among some optometrists and neuro-ophthalmologist as a part of a comprehensive clinical PCS examination. The general/standard tool used for screening the vestibular and ocular sensorimotor impairment and symptoms is the VOMS. VOMS has shown good consistency and significant correlation with PCS; however, more research is needed to be conducted on its usage as an appropriate screening tool for vestibulo-ocular symptoms.[27] Therefore, the search for a reliable and valid tool for screening vestibular and ocular impairments and symptoms in PCS is ongoing.

Our results show that EVestG can be used as a complementary clinical tool for detecting a vestibulo-ocular impairment associated with head injury. It might also prove useful in detecting comorbid depressive/mood symptoms and their potential confounding effects.[36,57,67,68] These results should be corroborated by an independent third party.

The main limitations of this study are: (1) The overall sample size was small, and (2) the EVestG signals and convergence measurements of healthy controls were not included in this study. Using EVestG technique can be advantageous to target rehabilitation strategies to reduce the severity of impairment and symptoms and to expedite the

recovery time. However, more research should be conducted to investigate whether the EVestG signal changes after CI recovery.

Disclosure of interest

The author BL is a consultant to the supporting company (Neural Diagnostics Pty. Ltd.).

Glossary

AP: action potential
CI: convergence insufficiency
EVestG: Electrovestibulography
EVS: efferent vestibular system
FEF: frontal eye field
INO: internuclear ophthalmoplegia
LGN: lateral geniculate nucleus
LPCS: long-term PCS
MLF: medial longitudinal fasciculus
mTBI: mild traumatic brain injury
NEER: neural event extraction routine
NRTP: nucleus reticularis tegmenti pontis
PCS: post-concussion syndrome
RPQ: Rivermead post-concussion questionnaire
RPQ3: score of first three symptoms of RPQ
RPQ13: score of last thirteen symptoms of RPQ
SOA: supraoculomotor area
SPCS: short-term PCS
VOMS: vestibular/ocular motor screening
VOR: vestibule-ocular reflex

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