The prevalence of cranial bone and upper cervical mobility restrictions in post-concussion syndrome

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Abstract

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Introduction: The Centre for Disease Control in the United States estimates 1.7-3.8 million sport concussions annually with twenty percent presenting persistent symptoms requiring targeted clinical assessment. Few studies examine manual therapy in concussions. Our purpose was to investigate cranial bone and upper cervical mobility restriction prevalence in post-concussion syndrome.

Methods: Twenty-one adults with post-concussion syndrome (PCS), 11 with history of concussion (CHx) and 12 controls (Ctl) participated. An osteopath assessed cranial bones and C0-C1-C2 mobility using a standard protocol to determine number of restrictions (NR). An athletic therapist assessed participants on Post-Concussion-Symptom-Scale (PCSS), King-Devick (KD), Tandem Gait Test (TGT), Sensory Organisation Test (SOT), and Vestibulo-Oculo-Motor-Screening (VOMS). Assessments were blinded to group assignment. We used a one-way ANOVA to assess group differences and a Pearson Correlation to assess relationships between variables.

Results: NR was statistically different between groups ($F_{(2,41)}$= 6.231, p= .004). PCS (8.24±4.25) had a higher NR compared to the Ctl (2.92±3.8) (mean difference 5.321±1.512, p= .003). The NR demonstrated a relationship with PCSS symptom severity (r^2=0.333, p= .027) and VOMS vestibular score (r^2=0.305, p= .044). Although not significant, there was a trend with number of symptoms (r^2=0.283, p= .062), visual (r^2=0.267, p= .079) and total score (r^2=0.293, p= .054). There was no relationship between NR and KD, TGT and SOT.

Conclusion: NR was significantly higher in the PCS group compared to the Ctl group. NR was associated with the PCSS and VOMS, but not the KD, TGT, SOT. Cervical and cranial mobility restrictions should be investigated in concussions with prolonged recovery.

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The prevalence of cranial bone and upper cervical mobility restrictions in post-concussion syndrome

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List of Abbreviations

#Sx	Number of Symptoms
AT	Athletic Therapist
CGH	Cervicogenic headache
CHx	Concussion history group
CRI	Cranial rhythmic impulse
Ctl	Control group
HCP	Health care professional
HI	Head injuries (number of)
HPA	Hypothalamus-pituitary-adrenal axis
ICD-10	International Classification of Disease
ImPACT	Immediate Postconcussion Assessment and Cognitive Testing
KD	King-Devick test
mTBI	Mild traumatic brain injury
NR	Number of restrictions
PCH	Post-concussion headache
PCS	Post-concussion syndrome
PCSS	Post-Concussion Symptom Scale
PF	Predisposing factors (number of)
PrefR	Preferential visual reflex
SBS	Spheno-basilar-synchrondrosis
SCAT	Sport Concussion Assessment Tool
SD	Standard deviation
SOT	Sensory organization test
SSR	Somatosensory reflex
SxSev	Symptom severity
TGT	Tandem Gait test
TScore	Total score
TVest	Total vestibular score
TVis	Total visual score
VestR	Vestibular reflex
VisR	Visual reflex

VMS	Visual motion sensitivity
VOMS	Vestibulo-Ocular-Motor Screening test
VOR	Vestibular-Ocular Reflex
WHO	World Health Organization

1. Introduction

1.1 Background

The Centre for Disease Control in the United States estimates that 1.7-3.8 million concussions occur in sport annually.[1] In 2007, Statistics Canada estimated the annual incidence of mild traumatic brain injury (mTBI) to be 600 per 100,000 people.[2] However, to date, no single clinical or medical test can clearly diagnose a concussion or confirm recovery. A concussion is induced by biomechanical forces and results in a complex pathophysiological process affecting the brain.[3] The mechanism of injury produces a neuro-metabolic cascade within the neurons and a disruption in the cerebral blood flow autoregulation resulting in a concussion.[4] Within the neuron, there is an ionic imbalance, rapid adenosine triphosphate depletion and mitochondrial dysregulation.[4] Researchers have observed decreases in cerebral blood flow associated with a disruption in autoregulation and changes in the basic properties of the cerebral vasculature.[5] Blood flow to the brain is essential for oxygen and glucose delivery, and necessary for the brain to restore physiological homeostasis.[4] Metabolic disruption and cerebral blood flow delivery may potentially mediate some of the symptoms in acute sport-related concussion and post-concussion syndrome.[4] These changes have mostly been studied in acute sport-related concussions, but decrease in cerebral blood flow has also been observed in certain areas of the brain of asymptomatic athletes for up to seven months post-concussion.[5] Recent research is highlighting autonomic dysfunction as a potential major contributing factor in the symptomatology of mTBI.[6] The autonomic nervous system is involved in vascular and cardiac regulation, blood pressure regulation, gastrointestinal response, contraction of the bladder, focusing of the eyes, thermoregulation, and innervates cardiac muscle, smooth muscle and various endocrine and exocrine glands. These systemic complications have been studied through changes in heart rate variability, pupillary dynamics, eye pressure and arterial pulse wave in those with mTBI, mainly in acute concussion. More research is needed to study the prolonged effects of autonomic dysfunction and persistent symptoms of a concussion.[6]

Most concussions resolve clinically in a short 10 to14 day period. However, approximately 20% of cases will present persistent non-specific post-traumatic symptoms, beyond the expected recovery time frames (>10-14 days in adults).[3,7] Ellis et al. describe the lack of a clinically accepted definition for post-concussion syndrome.[7] A common definition for PCS is the presence of three or more symptoms for one month post-injury including: headache, dizziness, fatigue, irritability, insomnia, and concentration or memory difficulty.[7,8] In post-concussion syndrome, whether these symptoms are directly related to metabolic, blood flow and

autonomic disruptions is still poorly understood. Every individual who suffers a concussion is unique, they can have a different number of symptoms and experience them to a different degree. A suspected concussion can include one or more of the following clinical domains: symptoms (somatic, cognitive and/or emotional symptoms); physical signs (loss of consciousness, amnesia, neurological deficits); balance impairments (gait unsteadiness); behavioural changes (irritability); cognitive impairment (slowed reaction times); sleep/wake disturbances (drowsiness, somnolence).[3]

Clinical symptoms are thought to reflect a functional disturbance rather than a structural brain injury.[3] Furthermore, research suggests that the brain injury does not necessarily cause the symptoms to persist beyond 1 month, therefore alternative explanations must be considered.[9] Due to the variety and different degrees of symptoms, a thorough clinical evaluation requires several different tests to establish which systems are potentially affected and thereby contributing to the persistent symptoms. These assessment tools can thereafter help guide the health care professional throughout the rehabilitation and help establish more objective recovery landmarks.

1.2 Clinical tests

Many tools are currently used by health care professionals to assess the multiple facets of a concussion. The clinical evaluation tools used in this study included the Post-Concussion Symptom Scale (PCSS), a neurological assessment via the cranial nerves, the King-Devick test (KD), the Tandem Gait Test (TGT), a Sensory Organization Test (SOT) using the Neurocom and the Vestibulo-Oculo-Motor Screening test (VOMS).

The post-concussion symptom scale (PCSS) has been established as a valid and reliable tool to determine the presence and severity of the symptoms present following a concussion.[3,10,11] The King-Devick test is a vision-based rapid number naming task. The test has been validated, demonstrating high specificity to distinguish between concussed and non-concussed athletes across multiple different sports.[12] Simple and time-efficient, the KD has been demonstrated to be reliable when administered by both health care professions,[13] and parents.[14] The visual system involves 70-80% of the brain's neurological pathways, as information travels from the eyes to the visual cortex where countless connections are made in the frontal, parietal and temporal lobes.[12] These areas are responsible for planning, initiation and execution of coordinated saccades, essential for reading and rapid number naming. These complex circuits also involve cognitive processing such as memory, attention and language function. The KD requires eye movement (saccades, convergence and accommodation), attention and language function. These tasks involve the integration of function of the brainstem, cerebellum and

cerebral cortex.[12]

The TGT is one of the tests used to assess balance after a concussion.[15] The TGT is a quantifiable dynamic motor performance task that involves balance and coordination to determine neurological function.[15,16] To maintain postural equilibrium, the central nervous system must process and integrate afferent information from the somatosensory, visual and vestibular system and execute the appropriate and coordinated musculoskeletal responses.[16] The TGT has been shown to be a valid and reliable test, of dynamic assessment of sensorimotor function.[16,17]

Postural control is maintained through the combined afferent information generated by the somatosensory (through proprioception), visual and vestibular systems.[18] A change in overall balance could be driven by a suppressed visual or vestibular system functioning or an inefficient integration of the vestibular information.[18] The SOT was designed to objectively identify abnormalities in the participant's ability to use these three sensory systems that contribute to postural control.[19] As described by McDevitt et al., the SOT done on the Neurocom measures the vertical ground reaction and shear forces produced from the body's center of gravity moving around a fixed base of support.[19] The test systematically disrupts the sensory selection process by altering available somatosensory and/or visual information while measuring the ability to minimize postural sway in the anterior-posterior direction. Concussed individuals have produced lower scores on the Neurcom as compared to non-concussed individuals. The Neurcom has also shown good specificity and sensitivity in detecting concussed from non-concussed individuals.[19]

The VOMS test is used to establish any problems associated with the vestibular and oculomotor systems associated with saccades and the vestibular-ocular reflex (VOR). The test has been shown to be reliable and valid and is easily done in a clinical setting.[20] The vestibular system includes the peripheral system, the central system, the oculomotor system and postural muscles. Together they are responsible for maintaining balance, postural control and gaze stability.[21] The peripheral vestibular apparatus is housed in the temporal bone. Within the bony labyrinth, the semi-circular canals are responsible for angular head accelerations, the otolith organs for linear head accelerations and all is surrounded by lymphatic fluid. Information is primarily processed in the brainstem via afferent fibers of the VIII cranial nerve. The second-order sensory neuron information is processed via the III, IV and VI cranial nerves that supply the oculomotor muscles.[21] The VOR maintains fixation of an image on the fovea of the eye during head motion. The VOR is essential for activities such as reading, driving and feature detection. The oculomotor system will fulfill this role when the object of interest is moving, such

as when tracking a moving object. For the oculomotor system to function properly there has to be an inhibition of the VOR. Therefore, the visual-vestibular system interaction is essential for an individual to move around in their environment without provocation of symptoms.[21] These are only some of the clinical tests currently found in the literature.

1.3 Clinical research

Ellis et al. described an evolving clinical approach, where research done on individuals suffering from PCS has led to early identification and targeted rehabilitation to address the pathophysiological mechanisms that govern persistent symptoms of a concussion.[7] For example, recent studies have shown that athletes who present vestibulo-ocular dysfunction take twice as long to recover from a concussion and are more likely to develop PCS.[7] Historically, individuals who suffered from concussion were thought to require more physical and cognitive rest,[3] but recent research suggests interventions beyond rest and different rehabilitation therapies have enhanced clinical recovery and successful return to play.[7, 22-25] However, in a more recent masterclass article by Schneider, on the need for a multifaceted approach, she described several research challenges in the area of concussion rehabilitation.[26] Schneider highlighted that to date there is a limited number of quality studies evaluating the efficacy of treatment strategies for the persistent symptoms of a concussion. Challenges include: different treatments may be more appropriate at different times in the rehabilitation; that a different number of treatments may be needed for each subtype of ongoing alterations in function; and the lack of a validated measure of recovery. Despite the lack of evidence, Schneider suggests treatment interventions should include: cervical spine treatment, vestibular rehabilitation, sleep management, low level aerobic exercise, headache management, psychological interventions, cognitive rehabilitation, and vision therapy.[26]

In 2016, Quatman-Yates et al. performed a systematic review of the possible physical rehabilitation interventions used to address persistent symptoms of a concussion.[27] Out of a possible 3437 titles and abstracts screened, 8 were retained for evaluation. The inclusion criteria included: a physical rehabilitation intervention, published in English in a peer-reviewed format, with human participants. The interventions investigated by the included studies were categorized into 3 types: physiological, vestibulo-ocular, and cervicogenic. The results of this systematic review indicate that several physical rehabilitation options with minimal risk for negative outcomes are available for treating patients experiencing persistent post-mTBI symptoms. These options include: vestibular rehabilitation, manual therapy, and progressive exercise interventions.[27]

Leddy et al. evaluated the effectiveness of a physiological intervention with sub-symptom

threshold exercise training to address the prolonged symptoms of a concussion.[22] The case series had 12 participants (6 athletes/6 non-athletes), mean age 27.9 years (SD 15.3, range 16–53), with symptoms of at least 6 weeks, but no longer than 52 weeks. Their outcome measures were: concussion symptom scale, exercise duration, blood pressure, heart rate, perceived exertion, and oxygen consumption. They achieved statistically significant improvements in symptoms and exercise time, higher peak heart rate and blood pressure during exercises. Athletes recovered faster than non-athletes. No adverse events were reported. Several studies have addressed persistent symptoms with exercise intervention.[27] In a more recent study, Leddy et al. did a random controlled trial on 103 adolescents (aged 13-18) to evaluate the efficacy of early progressive sub-symptom threshold aerobic exercise on concussion recovery.[23] The intervention group recovered faster (13 days) than the placebo group (17 days) which was deemed significant. There was no significant presence of prolonged recovery, individuals were admitted to the program within 10 days of sustaining a sports-related concussion. Although results are promising, further studies are needed to assess efficacy surrounding prolonged recovery and intervention parameters.

Jensen et al. compared manual therapy with the use of a cold pack for the treatment of post-concussion headache (PCH) following a concussion using a randomized controlled trial.[24] They found that 18 of the 19 participants who had suffered a concussion an average of 359 days prior, had painful upper cervical joint restrictions when compared to an uninjured control group (n = 19). They randomly assigned to two treatment groups: manual therapy group or the application of a cold pack. Results demonstrated a 57% reduction in pre-injury pain scores after 2 manual treatments 1 week apart for the manual therapy group, reduction in analgesic use, and 52% reduction in dizziness and visual disturbance ratings.[24] However, Jensen highlighted that the pain relief may have been temporary. Quatman-Yates suggest additional studies are necessary to investigate efficacy, timing, dosing and other intervention parameter for cervicogenic interventions.[27]

Schneider et al. aimed to determine whether a combination of vestibular rehabilitation and cervical spine manual therapy decreased the time until medical clearance compared with the local standard of care using a randomized control trial.[25] The standard care was given to all participants and included cervical range of motion, stretching and postural education. In addition to standard care, the intervention group also received cervical manual therapy and/or vestibular rehabilitation. Their physiological, vestibular and cervicogenic intervention was done on 31 athletes (15 treatment group, 16 control group), with median age 15 years (range 12–30). Time from injury was a mean of 53 days for the treatment group, and a mean of 47 days for the

control group. Their outcome measures were the number of days until medical clearance to return to sports, pain, Balance Confidence Scale, Dizziness Handicap Index, Sport Concussion Assessment Tool 2 (SCAT2), dynamic visual acuity test, head thrust test, modified motion sensitivity test, functional gait assessment, cervical flexor endurance, and joint position error test. Seventy-three percent of the treatment group, compared to 7.1% of control group, was medically cleared for return to play within 8 weeks. Their analysis indicated that patients in the treatment group were 3.91 times more likely to be medically cleared by 8 weeks. No adverse events were reported. In this case, not only was the sample size low, the intervention group received both vestibular and cervicogenic interventions and the control group received the same physiological intervention as the treatment group. This combining of intervention types makes it difficult to identify direct association between vestibular interventions and the outcome measures versus direct associations between cervicogenic interventions and the outcome measures. Furthermore, 26.7% of the participants were not medically cleared after 8 weeks of intervention.[25]

The prevalence and devastating impact of concussions on individuals is a public health concern. Research has improved our understanding of the underlying mechanisms and has improved concussion identification even though much is still unknown. Several clinical tests exist for different health care professionals and to help target rehabilitation. These strategies will enhance return to learning and return to physical activity following a concussion. However, in the literature, few studies have been done on evidence-based physical rehabilitation. Although results are promising, many have limitations such as absence of control groups, poor study design, and low sample numbers. Benefits have been seen with sub-maximal symptom-limited threshold aerobic exercise which has changed the previous concept that rest is the primary rehabilitation strategy. Manual therapy of the cervical spine and vestibular rehabilitation have also offered benefits in rehabilitation strategies. No research has been done using cranial manual therapy to address the persistent symptoms of a concussion. The literature demonstrates that cranial and upper cervical spine manual therapy has shown positive results for many concussion related symptoms such as headaches, dizziness, vision function and on the autonomic function.

1.4 Manual therapy: a novel approach

Manual therapy, such as osteopathy, that addresses mobility restrictions in cranial bones, and upper cervical spine, appears to be an effective treatment[28-32] to address symptoms commonly seen in concussions. Case studies have reported positive outcomes of osteopathic treatment for concussions[33,34,35], yet no empirical data specific to concussions has been

published. Osteopathy is a strictly manual form of therapy that aims at restoring mobility and function to the structures of the human body to stimulate the body's capacity for self-regulation.[36] The cranium, when observed as a vault, is a model of tensegrity. Derived from the word tension and integrity, tensegrity describes structures that are inherently stable as a result of balance between compression and tensional forces.[37] The cranium is composed of an outer structure of malleable, curved bones which are the compression element.[38] The sutures, which are held apart by the dura mater layer of the meninges, link the bones together and serve as the tensional forces. With Newton's 3rd law of motion: action and reaction are equal and opposite, if a load is applied to the structure, there will be a uniform change in the whole shape and the tension and compression will be distributed evenly.[37] The cranial vault is therefore, both a stable and compliant structure.[37]

Cranial bones exhibit viscoelasticity that improves their malleability and ability to protect the internal structures. When subjected to external trauma due to impact forces, cranial bones exhibit high bending forces.[38] In addition, the cranial sutures hold the bones of the skull together while allowing for mechanical stress and deformation. In adults, sutures serve as shock absorbers to dissipate stress transmitted to the skull.[38] Recent advances in micro-computed tomography has shown that sutures remain partly open even beyond the 7th decade, with varying degrees of connectivity across the suture gaps.[38]

Both Maloul et al in 2013 and Yu et al. in 2004 demonstrated the biomechanical suture force absorption abilities in the cranium on cadavers.[38,39] They deduced that sutures have the greatest absorption ability when subjected to parallel forces, but not as much with perpendicular forces and even less in shear forces received at a 45-degree angle. They also described that sutures with high degrees of inter-digitation, such as the sagittal suture, are more effective to withstand load. The falx cerebri is a meningeal fold of the dura mater that encloses the superior sagittal sinus from the ethmoid to the occiput along the sagittal and metopic sutures, and is attached to the endocranial surface of the ethmoid, frontal, parietal and occipital bones.[40] The tentorium cerebelli is a meningeal fold of the dura mater that encloses the transverse and superior petrosal sinuses, attaches to the sphenoid anteriorly, wraps around the trigeminal nerve, the pituitary gland, the optic nerve and the endolymphatic sac, and lines the endocranial surface of the temporal and occipital bones.[40] The dura mater is innervated by the trigeminal nerve.[40] Hernandez et al. demonstrated, using a cranial sensor cap and mouth guard sensors on football players, that at the moment of impact in a concussion mechanism of injury, the falx cerebri is stretched and kept under tension, and the greatest fluctuation in movement is seen either in the corpus callosum, at the center of the brain, or on the periphery along the cranial

bones, depending on the direction of impact.[41]

Ommaya et al. also demonstrated biomechanically that with rotational forces, as seen in traumatic brain injuries, the greatest biomechanical displacement is at the base of the skull at the cranio-cervical junction (C0-C1-C2).[42] The mechanical impact and spasms of the multiple muscle attachments on the temporal, occiput, atlas and axis could maintain a loss of mobility and affect the underlying structures and thereby contribute to concussion symptoms.[8,43,44] The cranio-cervical junction gives passage to multiple anatomical structures such as: the carotid and vertebral artery, and the inferior jugular vein could affect blood flow, the trigeminal nerve(V) which innervates the dura mater and blood vessels and could trigger headaches[45], the Xth cranial nerve plays a role in autonomic function and could trigger nausea; the XIth cranial nerve innervates the trapezius and the sterno-cleido-mastoid and could put these muscles under tension; and the XIIth cranial nerve contributes to tongue control and is involved in speech and swallowing.[40] Considering the anatomo-physiological relationships, and the impact of the concussion mechanism of injury on the falx cerebri and the cranio-cervical junction, cranial bone viscoelasticity and suture shock absorption, mobility restrictions could affect the physiology and function of the underlying anatomical structures.

1.5 Manual therapy: reliability and validity

Little research is done on the reliability and validity of manual therapy, which poses a challenge for physical rehabilitation studies. Schoetker-Koeniger et al. attempted to evaluate the validity of a general active cervical spine range of motion as observed by a health care professional (HCP). When compared to the golden standard, an ultrasound, they determined that the HCP's visual evaluation was moderate.[46] More specifically, Ogince et al. investigated the reliability of the cervical flexion-rotation test with 2 evaluators, as well as the ability to identify a relationship between a positive test result and presence of cervicogenic headache (CGH).[47] Experienced physical therapists with a high degree of manual skill assessed 3 groups, an asymptomatic group, a migraine with aura group, and a CGH group using a strong methodology. The kappa was 0.81 indicating excellent agreement, and evaluators agreed on presence of mobility restriction and absence or presence of CGH 98.3% of the time. There was no significant correlation between the presence of a mobility restriction of C1-C2 and the headache severity index. Ogince et al. determined that the cervical flexion-rotation test is reliable and established that it is valid and sensitive as a diagnostic test for CGH.[47] Concurrently, Hall et al. studied the reliability of the manual mobility assessment of cervical segment restrictions from C0-C4 in individuals presenting CGH.[48] Two evaluators assessed 60 participants who fit in either the control group or CGH group. Kappa coefficients ranged from 0.61 to 0.71 for all 4 segments C0-

C1, C1-C2, C2-C3, and C3-C4. They also determined that the dominant symptomatic segment for CGH was C1-C2 in 63% of participants.[48] Both of these studies highlight the importance of a structured manual assessment and its contribution to diagnostic directed intervention for different symptomology.

The benchmarks for training in osteopathy released by the World Health Organization in 2010 considers cranial osteopathy as an important skill for the profession.[36] Guillaud et al. performed a systematic review in 2016 on the reliability of clinical diagnosis and efficacy of cranial osteopathy. Out of a possible 1280 possible articles, 9 were retained for evaluation. Eight of the articles demonstrated a high risk of bias with misreported or selected data reporting, inappropriate methods for a reliability study and lack of blinding of the examiners.[49] They concluded that the evidence supporting the reliability of diagnosis and the efficacy of treatment in this field appears scientifically weak and inconsistent.[49] Guillaud used a modified version of the quality appraisal tool for studies on diagnostic reliability (QUAREL) to assess the risk of bias for the reliability studies. QAREL is an 11-item checklist that cover 7 key domains: the spectrum of participants; the experience of the evaluators; evaluator blinding; effects of order of assessments; the suitability of the time-interval between repeated measurements; appropriate test application and interpretation; and appropriate statistical analysis.[49,50] Ericsson et al. describe a general consensus amongst researchers, in the field of expertise development, that it takes approximately 10,000 hours of intense deliberate practice to become an expert within a chosen domain.[51] These elements could influence the quality of the research.

Halma et al. performed an intra-rater reliability study of the cranial rhythmic impulse (CRI), the spheno-basilar synchrondrosis (SBS) strain patterns, and quadrant restrictions on 48 adult participants divided into 3 groups and assessed by 2 blinded osteopaths.[52] Cohen's kappa for the CRI was 0.23 with a 64% percent agreement; 0.67 for the SBS strain patterns, with a 74% percent agreement; and 0.33-0.52 for quadrant restrictions with a percent agreement of 69-83%. Halma used a strong methodology that screened participants, limited the number of mobilisations, and for blinding purposes, used an opaque sheet to separate the participant from the evaluator. This methodology was considered outstanding by Guillaud et al. in order to isolate the evaluator from tactile, visual, auditory, and olfactory cues.[49] Fraval et al. performed an inter-rater reliability study of the temporal bone and occipital condyles manual assessment on infants of 6 months of age or less. Their study demonstrated a 95.7% agreement on presence of mobility restriction and an overall Pearson correlation coefficient of 0.58 for the right temporal bone and 0.71 for the left temporal bone.[53] However, they did not calculate the interclass coefficients, the optimal statistic method for ordinal variables of reliability. In their assessment,

they used different degrees of restriction from 0-3: not restricted, mildly, moderately and severely restricted. The use of degrees could have influenced the reliability results as degrees of restriction are difficult to perceive.[53]

1.6 Clinical research on concussion symptoms

Different studies used cranial manual therapy to address symptoms we commonly see in individuals who suffer from post-concussion syndrome. In 2015, Cerritelli et al. conducted a 3-armed random control trial to assess the effect of cranial manual therapy on chronic migraines as defined by the ICHD-II criteria (lasting 15 days or more per month for at least 3 months).[28] One hundred and five participants were randomly assigned to the intervention, sham and control group. Participants in the first two groups received 8 treatments over a period of 6 months. According to the headache impact test, the intervention group demonstrated a significant decrease in migraines, for days of migraine, intensity, and functional disability. The intervention group also had a significant decrease in medication intake.[28] Rolle et al. also conducted a single-blind random control trial with placebo group on forty-four participants experiencing headaches. After four cranial manual treatments over four weeks, participants in the experimental group had a decrease in headache frequency and drug use.[29] The trigeminal nerve leaves the brain stem, reaches the axis (C2), re-enters the skull to sits within the greater wing of the sphenoid bone adjacent to the temporal bone, and its 3 branches penetrate different parts of the sphenoid bone to reach their targeted distribution. The ophthalmic portion (V-1) innervates the dura-mater portion of the meninges, as well as the blood vessels which can trigger headaches. Mobility restrictions of the cranio-cervical region, sphenoid and temporal could therefore trigger headaches.[44,45]

Sandhouse et al. conducted a pilot study to assess the effect of cranial manual therapy on vision function.[30] Using a random-controlled trial with fifteen participants assigned to the intervention group and fourteen to the sham, they demonstrated significant changes in visual acuity, near point convergence, local stereo acuity, and pupillary size within the intervention group, compared to the control group. They described, as potential underlying mechanisms to explain the changes, the correction of the spheno-basilar synchrondrosis influencing the ocular muscle attachments on the eyes and the sphenoid bone, altering the shape of the eyes affecting axial length. Axial length and ocular mobility could affect distance visual acuity, local stereo acuity, and near point convergence. The pupillary size could be affected by the parasympathetic innervation of the eye via the oculomotor nerve and ophthalmic branch of the trigeminal nerve that passes through the superior oblique fissure of the sphenoid.[30] Sandhouse et al. believe that manual correction of the sphenoid could have released bony and fascial restrictions placed on

these nerves.[30]

Fraix et al. conducted a pilot study to assess the effect of cranial manual therapy on patients suffering from dizziness for more than 3 months.[31] Fraix assessed 16 participants using the SMART balance master on the Neurocom, the Dizziness Handicap Inventory and a self-assessment inventory. There were significant improvements in all the outcome measures.[31] The temporal bone houses the vestibular apparatus, the semi-circular canals, the vestibulocochlear nerve, and the endolymphatic sac. A restriction of the temporal bone could potentially contribute to symptoms of dizziness, nausea and balance issues.[44] As a preliminary exploration, this study demonstrated that cranial manual therapy can have a positive impact for individuals suffering from long term dizziness.

Finally, Ruffini et al. assessed the influence of cranial manual therapy on cardiac autonomic modulation in healthy subjects compared to a sham and control group.[32] With sixty-six patients in a cross-over single-blind study, they used an electrocardiogram before, during and after the intervention. They established statistical significance and demonstrated that cranial manual therapy can increase parasympathetic, and decrease sympathetic function compared to a sham intervention and control group.[32] They used manual techniques to release mobility restrictions of cranial bones, cranial sutures and cranial-sacral techniques as described by Magoun (1976).[54] Correction of the SBS, temporal, parietal and frontal bones could release tension within the falx cerebri and tentorium cerebelli which surrounds the trigeminal nerve, the hypothalamus, and the pituitary gland involved in cardiac autonomic modulation.[40] The trigeminal and hypothalamus-pituitary axis contribute to the parasympathetic and sympathetic function, such as respiratory and cardiac centers, cause disruption in an individual's sleep patterns and fatigue, and contribute to emotional problems, as seen in concussion.[6]

1.7 Purpose statement

The purpose of this study was to investigate, through a manual therapy evaluation, whether there are cranial and upper cervical mobility restrictions in individuals suffering from post-concussion syndrome, and whether these restrictions are linked to the clinical concussion tests performed by health care professionals. We hypothesized that individuals with post-concussion syndrome (PCS) would present more cranial bone, and upper cervical spine mobility restrictions than the control group (Ctl) and the concussion history group (CHx). We hypothesized that the mobility restrictions would be correlated with the clinical test results of the Post-Concussion Symptom Scale, the King-Devick test, the Tandem Gait Test, a Sensory Organization Test, and the Vestibulo-Ocular-Motor Screening test. Our independent variable was the presence or not of post-concussion syndrome. The dependent variable was the

presence of mobility restrictions and the results on the clinical concussion tests. The primary outcomes were the group comparisons and the associations between the mobility restrictions of the cranial bones, atlas and axis and the clinical concussion test results.

This study will provide insight on possible mechanisms that could contribute the persistent symptoms of a concussion and prolonged recovery. This study could suggest a possible mechanical anatomical link between bone restrictions and the persistent symptoms sustained during the mechanism of injury. Results could also provide insight on early detection after the concussion is diagnosed to target rehabilitation strategies and potentially improve recovery outcome.

2. Methods

2.1 Design

A correlation study design was used to investigate associations across the participants. A quasi-experimental design was used to determine group differences. The Human Research Ethics Committee at Concordia University approved this study (Ethics certification number: 30008220). Participants gave written informed consent prior to the study and were able to withdraw from the study at any time.

2.2 Setting

The clinical tests and manual evaluations were performed in a laboratory setting.

2.3 Preliminary step

As a preliminary step to our study, we established the inter-reliability of the manual mobility tests of the frontal, parietal and temporal bones, as well as the 5 spheno-basilar synchrondrosis strain patterns in an adult population. According to the Landis and Koch classification,[55] we established a moderate inter-rater reliability for a lateral strain of the spheno-basilar synchrondrosis strain pattern (0.481), and a substantial inter-rater reliability for flexion/extension (0.749), torsion (0.673), side-bending rotation (0.714) of the spheno-basilar synchrondrosis strain patterns, as well as for the temporal (0.666), parietal (0.774) and frontal (0.807) bones. The average pairwise percent agreements between the different evaluators ranged from 81.0 to 93.7 for all the possible mobility restrictions. The results demonstrate consistency and gave us confidence to proceed with this assessment protocol for the current study.

2.4 Participants

Recruitment was conducted by an athletic therapist within Quebec's different sports organizations, colleges, universities, health professional associations, and social media, from

November 2017 to May 2019. The recruiter was not blinded to the group attribution and sought out participants that fit the criteria for one of the three groups. The inclusion and exclusion criteria can be found in Table 1. Group 1 (PCS) was composed of participants with post-concussion syndrome (3 or more symptoms, 1-month post-injury), group 2 (CHx) was composed of participants who had previously sustained at least one concussion and fully returned to physical activity, and group 3 (Ctl) was the control group was composed of participants who have never sustained a concussion. A total of 48 participants were recruited: 22 PCS, 11 CHx, and 15 Ctl.

2.5 Procedures

Participants were greeted by the research assistant who explained to them the procedures, asked them to fill out a demographics and medical history questionnaire, and sign an informed consent form. Each participant was assigned an alphanumeric code. All participants went through a three-part evaluation; a manual evaluation, a clinical concussion evaluation, and a repeat of the manual evaluation. All results were noted on the assessment forms found in appendix 2. The research assistant led the participant through each stage, giving them instructions to limit communication where appropriate between the participant and the evaluator. The evaluators were blinded to the participant's group assignment.

In part 1 of the evaluation, an osteopath, according to the World Health Organization education standards, completed the manual evaluation. The osteopath performed passive mobility tests of both occipital condyles of C0 (2), C1 (1), C2 (1), the spheno-basilar synchrondrosis strain patterns (5), temporal bones (2), parietal bones (2), and of the frontal bone (1), for a total of 14 passive mobility tests. In part 2 of the evaluation, a certified athletic therapist (AT), according to the Canadian Athletic Therapists Association, completed the clinical tests, which included the Post-Concussion Symptom Scale, active range of motion of the cervical spine, evaluation of the cranial nerves, the King-Devick test, the Tandem Gate Test, a Sensory Organization Test using the Neurocom, and the Vestibulo-Ocular-Motor Screening test. In part 3 of the evaluation, the osteopath repeated the evaluation done in part 1. Each of these tests and evaluations is described below.

2.6 Part 1 and 3: Manual evaluation

The research assistant instructed the participant to remove any jewelry, to lie supine on the table, head in neutral resting on a pillow, hair free of hair ties, hands by their side with their eyes closed. The osteopath was then invited to enter the room. The osteopath was seated on a stool at the head of the participant, behind an opaque curtain blinded to the participant, with their hands under the sheet in contact with the participant's head and did not have any verbal

communication with the participant. The manual evaluation lasted approximately 15 minutes. For each of the 14 mobility tests, absence of compliance of movement in one direction or both direction along the mobility axis of each bone was noted as restricted. There is no normative data for these tests, except for the C1 flexion/rotation test where 45 degrees is considered normal. Results were written on an evaluation form that was collected by the research assistant after each evaluation. After the manual evaluation, the osteopath left the room.

Description of the mobility tests:

A) Unilateral flexion/extension of the occiput performed on the left and right condyle:[48] With index fingers in contact with the occiput and thumbs over the temporal bones just above the ears and the head rests in the palms, the participant's head was rotated 30-45 degrees in one direction. An anterior glide of the occipital condyle from neutral was performed. Then a posterior glide of the occipital condyle from neutral was performed. The glides were repeated on both sides.

B) Flexion rotation of the atlas (C1) on the axis (C2):[47] With hands on either side of the head and index fingers on the posterior arc of C1, the neck was brought into full flexion to restrict rotation of the lower cervical vertebrae. The head was brought into rotation, normal is considered 45 degrees.

C) Axis (C2):[48] With the pulp of the index and major fingers in contact with C2, from neutral, C2 was translated from left to right and then right to left. The test was repeated with C2 in flexion and in extension.

D) Spheno-basilar synchrondrosis (SBS):[54] Initial position: with hands on either side of the head, index on the greater wing of the sphenoid, 3rd and 4th finger over the ear, 5th finger on the lateral portion of the occiput and thumbs resting on either side of the sagittal suture. The SBS was mobilized along its physiological axis (sphenoid: transverse axis in front and above the sella turcica; occiput transverse axis above the foramen magnum, level with the jugular processes) in the 5 strain patterns of the SBS. 1) Flexion: Push the 2nd and 5th fingers caudal. Extension: Pull the 2nd and 5th fingers cephalic. 2) Torsion (right): turn the body to the left (pronation/radial deviation of the right forearm and supination/ulnar deviation of the left forearm). Torsion (left): opposite motion. 3) Side bending rotation (right): Bring the 2nd and 5th fingers on the left together and tilt the right side of the skull caudally. Side bending rotation (left): opposite motion. 4) Vertical strain (high): Impose an ulnar deviation of the wrists. Vertical strain (low): Impose a radial deviation of the wrists. Lateral strain (right): Bring the 2nd and 5th fingers on the right anterior. 5) Lateral strain (left): opposite motion.

E) Temporal bone:[54] One hand was placed under the occiput as a reference point, the other hand on one temporal bone with a butterfly grip (thumb and index bridging the zygomatic process while the ring and little finders bridge the mastoid process). The temporal bone was mobilized on its physiological axis (from the jugular surface to the petrous apex) in a motion called external rotation (to carry the superior border of the petrous portion anterolaterally towards the exterior of the skull) and from the neutral position in the opposite direction (internal rotation).

F) Frontal bone:[54] Both hands were placed on the frontal bone with index fingers on either side of the frontal crest, majors along the axis of the frontal bone, 4th and 5th fingers on the external pillars and thumbs crossed on opposing parietal bones behind the bregma. The frontal bone was mobilized on its physiological axis (vertical from the centre of the orbit through the frontal eminences slightly tilted backwards) in a motion called external rotation (bringing the external pillars anterior and the frontal crest cephalic) and from the neutral position in the opposite direction (internal rotation).

G) Parietal bone:[54] Both hands were placed on the parietal bones with thumbs on the opposite parietal bones just anterior to lambda on either side of the sagittal suture, index fingers on the antero-external angles, majors over the parieto-squamous suture, and 4th and 5th fingers on the postero-external angles. The parietal bones were mobilized along their physiological axis (antero-posterior from the coronal border of the parietal bone just lateral to the bregma, in a postero-lateral direction passing through the parietal eminences) in a motion called external rotation (bring the thumbs posterior and laterally, and simultaneously bring the angles anterior) and from neutral in the opposite direction (internal rotation).

2.7 Part 2: Clinical concussion evaluation

The research assistant invited the athletic therapist (AT) to enter the room. Test instructions and evaluations were performed by the AT and results were noted on an assessment form. Communication was kept to a minimum between the evaluator and the participant, additional instructions were given by the research assistant. After all the clinical tests were performed, the AT left the room.

A) The Post-Concussion Symptom Scale (PCSS):[3,10,11]

The participants were asked to report symptoms based on the severity of each symptom that day on a Likert scale from 0-6. They were scored on a total of 22 symptoms and for a maximum severity score of 132.

Equipment: PCSS questionnaire.

B) Cervical spine range of motion:[46]

Equipment: cervical inclinometer.

The participant was seated and the AT standing. The participant was asked to perform all cervical motions one after another: flexion, extension, right and left rotation, and right and left side bending. A demonstration was offered by the AT. The range of motion in degrees using inclinometer was noted.

C) Cranial nerves:[56]

The following cranial nerves were tested with the participant seated and the AT standing. An inability to perceive a sense or to do the action required is noted as a positive finding.

Instructions:

1) The trigeminal (V) nerve is a sensory nerve responsible for facial sensation over. It has three branches that each cover a different region of the face: forehead, cheek and jaw. With the participants eyes closed, the AT gently touches each region of the participant's face. The participant reports if they feel the touch and if it perceived as equal on both sides.

2) The facial (VII) nerve is a motor nerve responsible for facial expression. The participant is asked to smile and frown.

3) The acoustic (VIII) nerve is a sensory nerve responsible for hearing. With the participant's eyes closed, the AT rubs their fingers together at different distances from the participant's ears. The participant reports which noise they perceive as closest.

4) The glossopharyngeal (IX) is a motor nerve responsible for swallowing and voice. The vagus (X) nerve is a sensory and motor nerve responsible for swallowing and the gag reflex. The test is the same for both nerves. First the participant is asked to open their mouth, with a penlight, the AT checks to see that the epiglottis is centered at the back of the throat. Then the participant is asked to open their mouth, stick out their tongue and say "ah".

5) The spinal (XI) nerve is a motor nerve responsible for neck strength. The participant raises their shoulders in elevation. The AT puts their hands over the shoulders. The AT asks the participant to maintain their shoulders in elevation as the AT pushes down on the shoulders with the instructions "don't let me move you".

6) The hypoglossal (XII) nerve is a motor nerve responsible for tongue movement and strength. The participant is asked to stick out their tongue and move their tongue from left to right.

D) The King-Devick test:[12]

Equipment: Stopwatch; 1 King-Devick demonstration card, 3 King-Devick test cards

Instructions: Participants were asked to read the numbers on each card from left to right as quickly as possible, without making any errors. Following the completion of the demonstration card, participants were then asked to read each of the three test cards in the same manner. The

times required to complete each card was recorded in seconds using a stopwatch. The sum of the three card times scores constitutes the summary score for the entire test, the King-Devick time score. The number of errors made in reading the test cards was also recorded; misspeaks on numbers were recorded as errors only if the participant did not immediately correct the mistake before going on to the next number.

E) The Tandem Gait Test:[57]

Test for balance, speed and coordination.

Equipment: 3.8 mm tape 3 m long, a stopwatch.

Instructions: The participant was instructed to walk along a 38 mm wide, 3m long sports tape. The participant started with both feet together at the start of the line, alternated one foot in front of the other, heel to toe, along the line as quickly and accurately as possible, turn 180° behind the end of the line, and returned to the start. The best time of 4 trials back and forth along the tape was recorded as the official score. Participants failed the test if they did not maintain approximation between their heel and toe, deviated from the track, or did not turn behind the end of the line.

F) The Sensory organization test (SOT):[19]

The SOT testing was performed using the Neurocom. The SOT was designed to objectively identify abnormalities in the participant's ability to use the 3 sensory systems that contribute to postural control: somatosensory (proprioception), visual and vestibular. The Neurocom device calculated the SOT composite scores as a weighted average of all 6 conditions to determine the overall level of performance as a percentage from 0–100, with better performance represented as a higher score and a fall scored as 0. The Neurocom software also calculated the sensory ratios, which estimated the participant's ability to utilize each type of sensory input to maintain balance. The somatosensory ratio is the quotient of condition 2 over condition 1. The visual ratio is the quotient of condition 4 over condition 1. The vestibular ratio is the quotient of condition 5 over condition 1. The visual preference ratio is the sum of conditions 3 and 6 divided by the sum of conditions 2 and 5. This ratio represents the degree to which a participant relies on visual input to maintain balance even when the visual input is unreliable.

G) The Vestibulo-Oculo-Motor Screening (VOMS):[20]

At the beginning of the test, baseline symptoms on a Likert scale of 0-10 was noted for headache, dizziness, nausea and fogginess. After each test, the participant was asked to rate each of the 4 symptoms once again from 0-10. Also, abnormal findings such as presence of saccades, overshooting or undershooting a distance, divergence or convergence of the eyes, inability to keep eyes fixated on a moving target or inability to follow the instructions were noted.

For each of the 5 components, the evaluator was standing 3 feet from the participant who was seated.

Equipment: Tape measure (cm); Metronome; Target with a 14-point font print.

Instructions:

1) Smooth Pursuits: Tested the ability to follow a slowly moving target (IV and VI cranial nerves). The evaluator held a fingertip at a distance of 3 feet from the participant. The participant was then instructed to maintain focus on the target as the evaluator moved the target smoothly in the horizontal direction 1.5 feet to the right and 1.5 feet to the left of midline. The test was then repeated with the examiner moving the target smoothly and slowly in the vertical direction 1.5 feet above and 1.5 feet below midline. For both tests, one repetition was completed when the target moved back and forth to the starting position, and 2 repetitions are performed.

2) Saccades: Tested the ability of the eyes to move quickly between targets. The participant and the evaluator are seated.

Horizontal Saccades: The evaluator held two single points horizontally at a distance of 3 feet from the participant, and 1.5 feet to the right and 1.5 feet to the left of midline so that the participant's gaze was 30 degrees to left and 30 degrees to the right. The evaluator instructed the participant to move their eyes as quickly as possible from point to point. One repetition was completed when the eyes move back and forth to the starting position, and 10 repetitions were performed.

Vertical Saccades: The test was repeated with the two points held at a vertical distance.

3) Convergence – Measures the ability to view a near target without double vision. The participant was seated and wearing corrective lenses (if needed). The participant focuses on a small target (approximately 14-point font size) at arm's length and slowly brought it toward the tip of their nose. The participant was instructed to stop moving the target when they saw two distinct images or when the evaluator observed an outward deviation of one eye. Blurring of the image was ignored. The distance in centimeters between target and the tip of nose was measured and recorded. This was repeated 3 times with measures recorded each time. Abnormal: Near Point of convergence ≥ 6 cm from the tip of the nose.

4) Vestibular-Ocular Reflex (VOR) Test: Assesses the ability to stabilize vision as the head moves. The evaluator held a target of approximately 14-point font size in front of the participant in midline at a distance of 3 feet.

Horizontal VOR Test: The participant was asked to rotate their head horizontally at an amplitude of 30 degrees to each side, while maintaining focus on the target, following the beat of a metronome to ensure the speed of rotation is maintained at 180 beats/minute (one beat in each

direction). One repetition was completed when the head moved back and forth to the starting position, and 10 repetitions were performed.

Vertical VOR Test: The test was repeated with the participant moving their head vertically.

5) Visual Motion Sensitivity (VMS) Test: Tests visual motion sensitivity and the ability to inhibit vestibular-induced eye movements using vision. The participant stood with feet shoulder width apart. The evaluator stood next to and slightly behind the participant for safety but allowed the movement to be performed freely. The participant held their arms outstretched and focuses on one of their thumbs. Maintaining focus on their thumb, the participant rotated, together as a unit, their head, eyes and trunk, at an amplitude of 80 degrees to the right and 80 degrees to the left. A metronome was used to ensure the speed of rotation was maintained at 50 beats/min (one beat in each direction). One repetition is complete when the trunk rotates back and forth to the starting position, and 5 repetitions were performed.

Part 3: Repeat part 1.

2.8 Statistical analysis

A one-way ANOVA and Tukey post hoc test was used to assess PCS, CHx, and Ctl group differences of the continuous variables (NR, PCSS, KD, TGT, SOT, VOMS, HI, PF). A Pearson Correlation was used to analyze the relationships between the continuous variables across the whole sample. Pearson Correlation classification was used to establish the degree of relationship between two continuous variables (very high: ±0.9-±1; high: ±0.70-±0.90; moderate: ±0.50-±0.70; low: ±0.30-±0.50).[58] A Pearson Chi-square 2-tailed association test was used to assess PCS, CHx, and Ctl group differences and relationships for the dichotomous variables (C2, C1, C0, SBS, temporal, parietal, frontal bone restriction). A p-value less than .05 determined statistical significance, and all tests were 2-sided. All data analysis was conducted using SPSS statistics computing program version 25.0 (IBM Inc, Armonk, NY).

3. Results

3.1 Participants

Forty-eight participants were assessed and 4 were removed from the sample before data analysis. Three participants had played collision sports therefore did not meet the exclusion criteria for the control (Ctl) group, and 1 was removed from the post-concussion syndrome (PCS) as they were diagnosed and medicated for anxiety prior to the concussion. The 44 participants were aged between 18 and 32 years old, 24 females and 20 males. There were 21 in the PCS group, 11 in the CHx group, and 12 participants in the Ctl group. The PCS group averaged 2.33 concussions and the CHx group averaged 1.09 concussions.

3.2 Outcome measures for the mobility restrictions

For the total number of restrictions, there was a statistically significant difference between groups as determined by one-way ANOVA ($F_{(2,41)}$= 6.231, p= .004) as demonstrated in Fig 1. A Tukey post hoc test revealed a statistically significantly higher number of restrictions in the PCS (8.24±4.25) compared to the Ctl (2.92±3.8) (mean difference 5.321±1.512, p= .003). There was no statistically significant difference between the Ctl (2.92±3.8) and the CHx (5.91±4.41) (mean difference 2.992±1.744, p= .212), and between the PCS (8.24±4.25) and the CHx(5.91±4.41) (mean difference 2.329±1.555, p= .303).Complete ANOVA results for group statistics can be found in table 3, descriptive statistics in table 4, and Tukey post hoc multiple comparisons tests in table 5 in appendix 1.

We used the Pearson Correlation Coefficient to analyze relationships between the number of restrictions and the other continuous variables (number of symptoms, symptom severity, number of head injuries, number of predisposing factors, King-Devick test, Tandem Gait Test. We used the Pearson Correlation Coefficient to analyze relationships between the number of restrictions and the VOMS (visual score, vestibular score, and total score) and the Neurocom scores (Somatosensory, Visual, Vestibular, and Preferential).

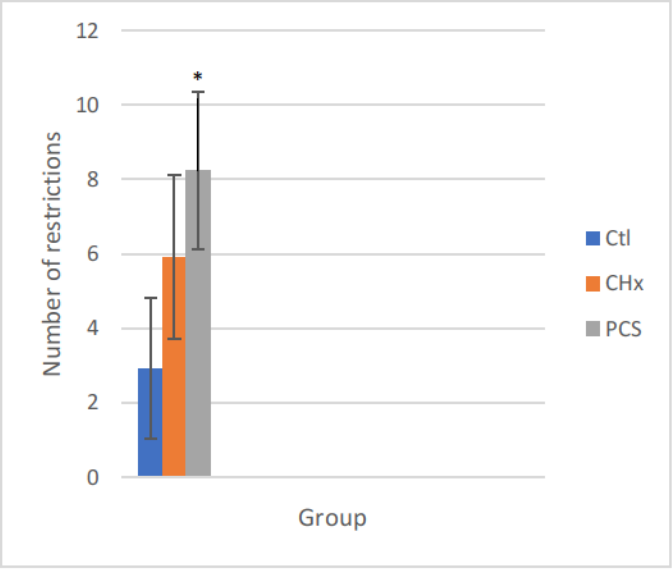

Figure 1. Mean number of restrictions by group.

There was low relationship between NR and SxSev (r^2=0.333, p= .027), HI (r^2=0.396, p= .008), PF (r^2=0.338, p= .025) and TVest (r^2=0.305, p= .044) as seen in Fig. 2. Although not significant, there was a relationship trend between NR and the #Sx (r^2=0.283, p= .062), TVis

(r^2=0.267, p= .079) and TScore (r^2=0.293, p= .054). There was no NR relationship with KD (r^2=0.211, p= .170), TGT (r^2=-.0.76, p= .624), SSR (r^2=0.000, p= 1.000), VisR (r^2=-0.123, p= .428), VestR (r^2=0.058, p= .708), PrefR (r^2=0.108, p= .487). Fig 2 represents trend lines between NR and other variables.

3.3 Prevalence

The characteristics of the participants medical histories by group can be found in Table 2 of appendix 1. The prevalence of mobility restrictions by structure within each group can be found in Figure 4. There were 53 occiput restrictions (32 PCS, 13 CHx, 8 Ctl), 12 atlas restrictions (9 PCS, 3 CHx, 0 Ctl), and 20 axis (12 PCS, 5 CHx, 3 Ctl) restrictions. There were 97 SBS strain pattern restrictions (63 PCS, 21 CHx, 13 Ctl): 17 flexion/extension (11 PCS, 4 CHx, 2 Ctl), 12 torsions (10 PCS, 1 CHx, 1 Ctl), 24 side-bending-rotation (14 PCS, 7 CHx, 3 Ctl), 25 vertical strains (16 PCS, 6 CHx, 3 Ctl), and 19 lateral strain (12 PCS, 3 CHx, 4 Ctl). There were 42 temporal bone restrictions (25 PCS, 11 CHx, 6 Ctl), 30 parietal bone restrictions (22 PCS, 5

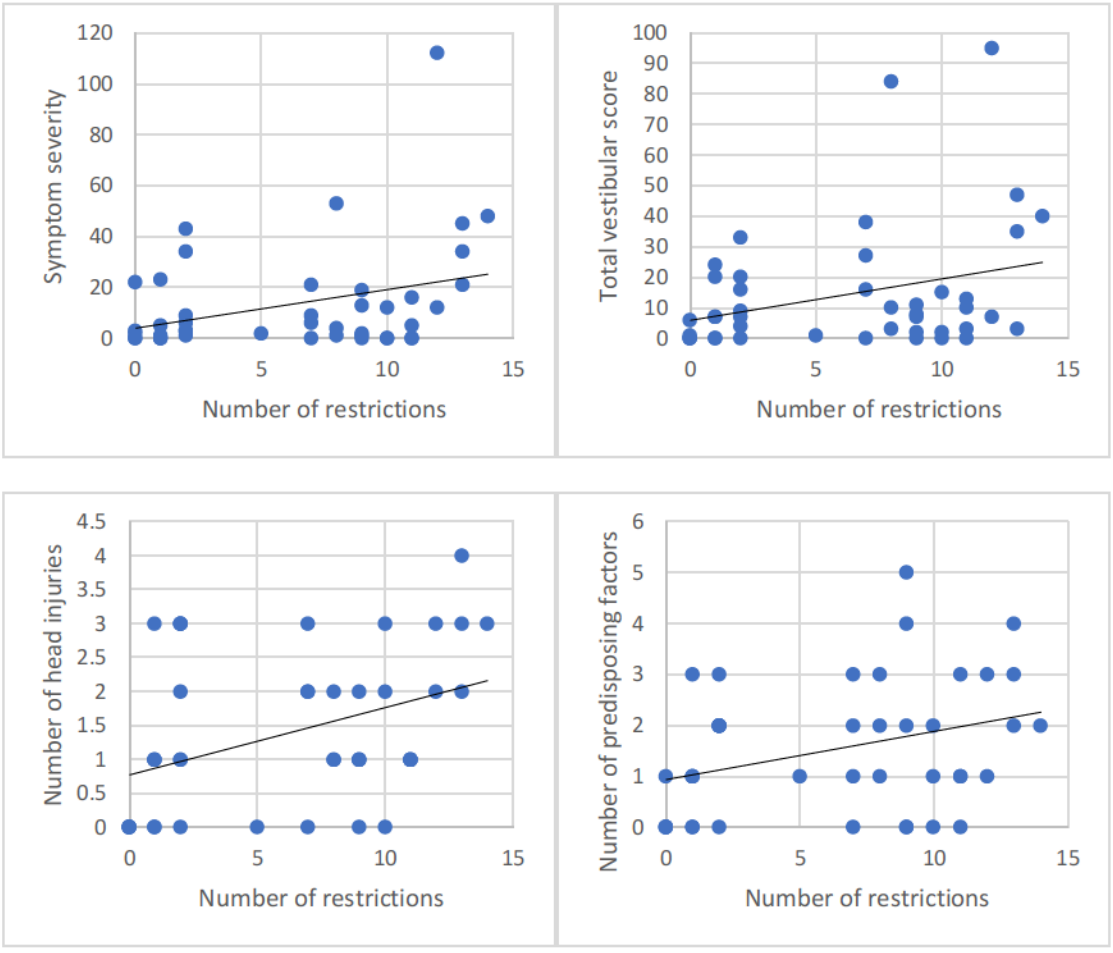

Figure 2. Pearson correlation of the number of restrictions with symptom severity, total vestibular score, number of head injuries, and number of predisposing factors.

CHx, 3 Ctl), and 19 frontal bone restrictions (12 PCS, 7 CHx, 2 Ctl). The total number of restrictions present among participants was 273 out of a possible 616: 173 in the PCS group, 65 in the CHx group and 35 in the Ctl group.

A Pearson Chi-square was used to assess group differences in the presence or absence of mobility restrictions for each bone. There was a significant difference between groups for C0 ($X^2(2) = 14.633$, $p = .001$) and C1 ($X^2(2) = 7.071$, $p = .029$). The PCS group presented a greater of restricted C0 and C1 than the CHx and Ctl groups. There was no significant difference between groups for C2 ($X^2(2) = 3.182$, $p = .204$), SBS ($X^2(2) = 2.365$, $p = .307$), temporal ($X^2(2) = 5.928$, $p = .052$), parietal ($X^2(2) = 5.752$, $p = .056$), and frontal ($X^2(2) = 5.483$, $p = .064$). When the 5 strain patterns of the SBS were assessed separately, there was a significant difference between groups for torsion of the SBS ($X^2(2) = 8.386$, $p = .015$), and a vertical strain ($X^2(2) = 8.187$, $p = .017$). The PCS groups presented a greater number of restricted torsion and vertical strain than the CHx, and Ctl groups. There was no significant difference between groups for flexion/extension of the SBS ($X^2(2) = 4.140$, $p = .126$), side-bending rotation ($X^2(2) = 5.836$, $p = .054$), and lateral strain ($X^2(2) = 3.277$, $p = .194$). Results can be found in table 6.

A Pearson Chi-Square was used to assess the association in the presence or absence of mobility restrictions between each bone. There was a significant association between C0 and C1 ($X^2(1) = 4.243$, $p = .039$), SBS ($X^2(1) = 15.121$, $p \leq .000$), temporal ($X^2(1) = 16.343$, $p \leq .000$), parietal ($X^2(1) = 8.599$, $p = .003$), and frontal ($X^2(1) = 8.599$, $p = .003$). When C0 was restricted, C1, SBS, temporal, parietal and frontal were restricted as well. There was no significant association between C0 and C2 ($X^2(1) = .670$, $p = .413$). There was a significant association between C1 and SBS ($X^2(1) = 4.872$, $p = .027$), and temporal ($X^2(1) = 7.243$, $p = .007$). When C1 was restricted, SBS, and temporal were restricted as well. There was no significant association between C1 and C2 ($X^2(1) = 2.994$, $p = .084$), parietal ($X^2(1) = 3.709$, $p = .054$), and frontal ($X^2(1) = .313$, $p = .576$). There was a significant association between SBS and temporal ($X^2(1) = 32.874$, $p \leq .000$), parietal ($X^2(1) = 17.297$, $p \leq .000$), and frontal ($X^2(1) = 12.368$, $p \leq .000$). When SBS was restricted, temporal, parietal and frontal were restricted as well. There was no significant relationship between SBS and C2 ($X^2(1) = 3.240$, $p = .072$). There was a significant association between the temporal and C2 ($X^2(1) = 6.631$, $p = .010$), parietal ($X^2(1) = 23.151$, $p \leq .000$), and frontal ($X^2(1) = 17.577$, $p \leq .000$). When the temporal was restricted, C2, parietal and frontal were restricted as well. There was a significant association between the parietal and frontal ($X^2(1) =$

5.439, p= .020). When the parietal was restricted, the frontal was as well. There was no significant association between C2 and parietal ($X^2(1) = 2.087$, p= .149), and frontal ($X^2(1) = .695$, p= .405). The association between the presence of mobility restrictions between the different bones can be found in table 7.

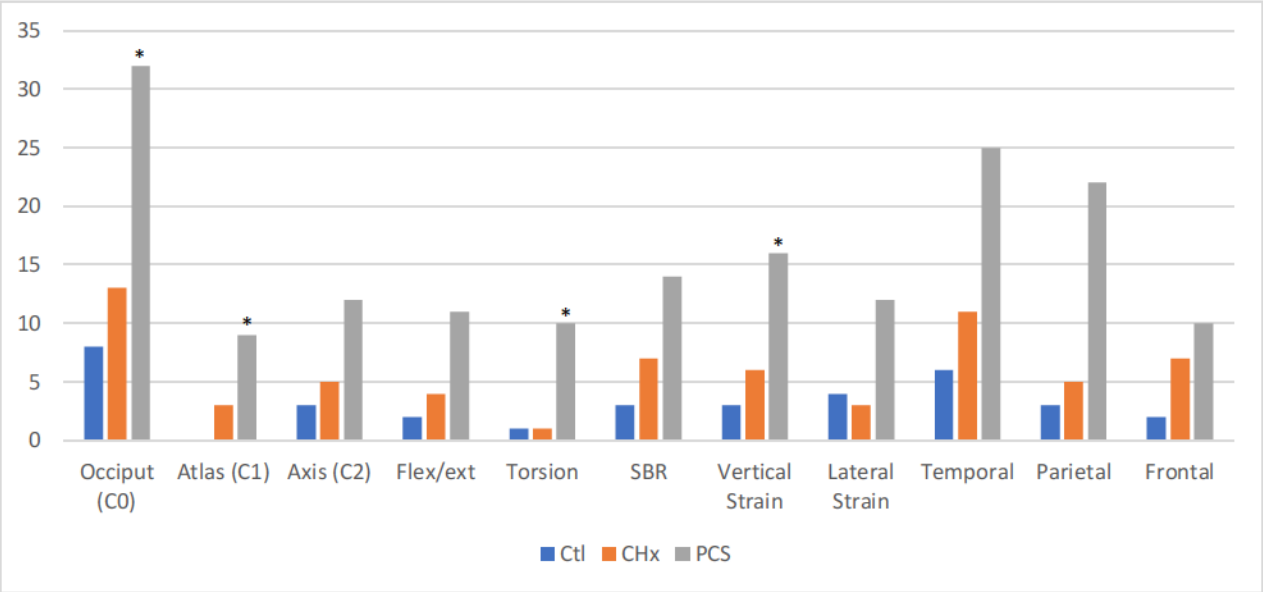

Figure 3. Prevalence of mobility restrictions by structure distributed by group.

3.4 Group Statistics

As a secondary analysis, the medical histories and clinical tests were assessed for group differences. For the total number of symptoms, there was a statistically significant difference between groups as determined by one-way ANOVA ($F_{(2,41)}$= 23.710, p≤ .000). A Tukey post hoc test revealed a statistically significantly higher number of symptoms in the PCS (10.9±5.8) compared to Ctl (1.75±3.19) (mean difference 9.155±1.627, p≤ .000), and between the PCS (10.9±5.8) and CHx (1.36±2.11) (mean difference 9.541±1.673, p≤ .000). There was no statistically significant difference between the Ctl (1.75±3.19) and CHx (1.36±2.11) (mean difference 0.386±1.877, p= .977). For the symptom severity, there was a statistically significant difference between groups as determined by one-way ANOVA ($F_{(2,41)}$= 8.833, p= .001). A Tukey post hoc test revealed a statistically significantly higher symptom severity in the PCS (25.43±25.36) compared to Ctl (2.92±6.22) (mean difference 22.512±6.549, p= .004), and between the PCS (25.43±25.36) and CHx (2.0±3.82) (mean difference 23.429±6.736, p= .003). There was no statistically significant difference between the Ctl (2.92±6.22) and CHx (2.0±3.82) (mean difference 0.917±7.555, p= .992).

For the King Devick test, there was no statistically significant difference between groups as determined by one-way ANOVA ($F_{(2,41)}$= 1.721, p= .192). For the Tandem Gait test, there was no statistically significant difference between groups as determined by one-way ANOVA ($F_{(2,41)}$= 1.232, p= .302).

All Vestibulo-Oculo-Motor scores were statistically significant different between groups. The total visual score was a statistically significant difference between groups as determined by one-way ANOVA ($F_{(2,41)}$= 7.656, p= .001). A Tukey post hoc test revealed a statistically significantly higher total visual score in the PCS (27.81±29.16) compared to Ctl (3.41±4.93) (mean difference 24.393±7.535, p= .007), and between the PCS (27.81±29.16) and CHx (3.0±7.07) (mean difference 24.810±7.750, p= .007). There was no statistically significant difference between Ctl (3.41±4.93) and CHx (3.0±7.07) (mean difference 0.417±8.692, p= .999). The total vestibular score was a statistically significant difference between groups as determined by one-way ANOVA ($F_{(2,41)}$= 9.735, p≤ .000). The Tukey post hoc test revealed a statistically significantly higher vestibular score in PCS (26.48±24.36) compared to Ctl (2.58±3.15) (mean difference 23.893±6.314, p= .001), and between PCS (26.48±24.36) and CHx (4.0±7.08) (mean difference 22.476±6.494, p= .004). There was no statistically significant difference between the Ctl (2.58±3.15) and CHx (4.0±7.08) (mean difference 1.417±7.283, p= .979). The VOMS total score was statistically significant different between groups as determined by one-way ANOVA ($F_{(2,41)}$= 8.819, p= .001). The Tukey post hoc test revealed a statistically significantly higher total VOMS score in PCS (54.76±53.3) compared to Ctl (6.0±8.01) (mean difference 48.762±13.786, p= .003), and between PCS (54.76±53.3) and CHx (7.0±14.06) (mean difference 47.762±14.179, p= .005). There was no statistically significant difference between Ctl (6.0±8.01) and CHx (7.0±14.06) (mean difference 1.000±15.902, p= .998).

For the sensory organization test, there was no statistically significant difference between groups as determined by one-way ANOVA. The somatosensory reflex composite score was not significant between groups ($F_{(2,41)}$= .700, p= .502). The visual reflex composite score was not significant between groups ($F_{(2,41)}$= .726, p= .490). The vestibular reflex composite score was not significant between groups ($F_{(2,41)}$= 2.814, p= .072). The visual preference reflex composite score was not significant between groups ($F_{(2,41)}$= .686, p= .509).

For the total number of head injuries, there was a statistically significant difference between groups as determined by one-way ANOVA ($F_{(2,41)}$= 56.405, p≤ .000). A Tukey post hoc test revealed a statistically significantly higher number of head injuries in PCS (2.33±.86) compared to Ctl (0±0) (mean difference 2.333±.223, p≤ .000), between PCS (2.33±.86) and CH (1.09±.3) (mean difference 1.242±.229, p≤ .000), and between Ctl (0±0) and CHx (1.09±.3)

(mean difference 1.091±.257, $p \leq .000$). For the number of predisposing factors, there was a statistically significant difference between groups as determined by one-way ANOVA ($F_{(2,41)}$= 15.756, $p \leq .000$). A Tukey post hoc test revealed a statistically significantly higher number of predisposing factors in PCS (2.29±1.01) compared to Ctl (.25±.45) (mean difference 2.036±.363, $p = .000$), and between Ctl (.25±.45) and CHx (1.45±1.37) (mean difference 1.205±.419, $p = .017$). There was no statistically significant difference between the PCS (2.29±1.01) and CHx (1.45±1.37) (mean difference 0.831±.373, $p = .079$). ANOVA descriptive statistics can be found in table 3, results for group statistics table 4, and Tukey post hoc multiple comparisons tests in table 5.

3.5 Correlation statistics

Other relationships were found among the variables. The #Sx demonstrated a high relationship with SxSev (r^2=0.890, $p \leq .000$), moderate with HI (r^2=0.621, $p \leq .000$), and PF (r^2=0.649, $p \leq .000$), and a low relationship with KD (r^2=0.448, $p = .002$), and SSR (r^2=0.346, $p = .021$). SxSev demonstrated a moderate relationship with HI (r^2=0.500, $p = .001$), PF (r^2=0.541, $p \leq .000$), and KD (r^2=0.606, $p \leq .000$), and a low relationship with TGT (r^2=0.423, $p = .004$), and SSR (r^2=0.313, $p = .039$). The PF demonstrated a moderate relationship with the HI (r^2=0.553, $p \leq .000$), and a low relationship with KD (r^2=0.455, $p = .002$). The KD demonstrated a low relationship with the TGT (r^2=0.494, $p = .001$).

All three VOMS score had relationships with several variables. TVis demonstrated a high relationship with #Sx (r^2=0.754, $p \leq .000$), SxSev (r^2=0.856, $p \leq .000$), a moderate relationship with SSR (r^2=0.559, $p \leq .000$), and a low relationship with HI (r^2=0.452, $p = .002$), PF (r^2=0.392, p=0.009), KD (r^2=0.430, $p = .004$), and TGT (r^2=0.431, $p = .003$). TVest demonstrated a high relationship with #Sx (r^2=0.738, $p \leq .000$), SxSev (r^2=0.819, $p \leq .000$), a moderate relationship with SSR (r^2=0.553, $p \leq .000$), and a low relationship with HI (r^2=0.484, $p = .001$), PF (r^2=0.388, $p = .009$), KD (r^2=0.374, $p = .012$), and TGT (r^2=0.366, $p = .015$). TScore demonstrated a high relationship with #Sx (r^2=0.754, $p \leq .000$), SxSev (r^2=0.845, $p \leq .000$), a moderate relationship with SSR (r^2=0.558, $p \leq .000$), and a low relationship with HI (r^2=0.470, $p = .001$), PF (r^2=0.396, $p = .008$), KD (r^2=0.414, $p = .005$), and TGT (r^2=0.410, $p = .006$). These results can be found in table 8.

Table 9 shows a very high relationship within all the VOMS scores: TVis with TVest (r^2=0.974, $p \leq .000$); TScore with TVis (r^2=0.994, $p \leq .000$); and TScore with TVest (r^2=0.992, $p \leq .000$). Table 9 also shows a moderate relationship between VisR and VestR (r^2=0.640, $p \leq .000$), and a low relationship between SSR and VisR (r^2=0.435, $p = .003$).

4. Discussion

4.1 Mobility restrictions

We found a significant difference in the number of mobility restrictions between groups. The PCS group had a significantly higher number of mobility restrictions than the control group. Although not significant, the number of mobility restrictions was higher in the PCS group compared to the CHx group, and between the CHx group compared to the Ctl group. In our study, we found an average number of mobility restrictions of 2.92 in the control group, 5.09 in the concussion history group, and 8.23 in the post-concussion group out of a possible 14 restrictions. There is no previous literature on the prevalence of cranial mobility restrictions. Tiwari et al. reported characteristics of cervical spine impairments in children and adolescents post-concussion.[59] Among the 73 participants from 8-18 years of age, Tiwari et al. found that 71% of them presented a mobility restriction of the C0-C1 and C1-C2 upper cervical segments. Our study reported 63% prevalence of C0, C1, C2 segment mobility restrictions in the post-concussion group. Tiwari's study included a higher number of participants, and upper cervical segment restrictions could be more prevalent than our study revealed. Tiwari's participants also represented a younger population, where the cranio-cervical musculature is less developed, potentially rendering this area more vulnerable to injury. There was a significant relationship between the number of restrictions with symptom severity, vestibular score, number of head injuries, and number of predisposing factors. Across the whole sample, those who presented a greater number of mobility restrictions also scored higher on the PCSS score, VOMS scores, and had a greater number of previous head injuries and predisposing factors.

To our knowledge, our study is the first to investigate cranial mobility restrictions in an adult concussion population. The literature in this field is limited, therefore we can only speculate as to what our results mean. Research has demonstrated a reduction in mobility of the cranio-cervical junction[42] and the maintained tension of the falx cerebri[41] following a concussion. Both mobility restrictions of the cranio-cervical junction and tension of the falx cerebri, through their anatomo-physiological relationship could contribute to the persistence of cranial and upper cervical mobility restrictions in a post-concussion syndrome population. We do not know if the mobility restrictions were sustained in the mechanism of injury or if they developed over time. The presence of mobility restrictions in a population that has previously sustained concussion also suggests that mobility restrictions could persist beyond recovery time or develop over time.

Our results also suggest a relationship between the number of mobility restrictions and the PCSS and VOMS scores. The literature supports the involvement of the cervical spine as a

contributing factor to the persistent symptoms of a concussion.[8,24,25] Our results demonstrate that mobility restrictions could be one of the elements that contribute to the persistent symptoms of a concussion and influence the underlying structures responsible for visual and vestibular function. Our results cannot allude to a cause and effect relationship, further studies are required on the relationship between specific mobility restrictions and the underlying physiology.

We did not find a significant relationship between the number of restrictions and the King-Devick test, the Tandem Gait test and the Sensory Organization test. Our results demonstrate that while the number of restrictions may be related to post-concussion syndrome, the number of restrictions does not influence, or are not influenced by, the KD, TGT, and SOT. Our results did not reveal group differences for the KD, TGT, and SOT. The balance component of a concussion assessed using the TGT and SOT typically resolves within 3-5 days following the concussion,[60] and the KD has demonstrated high specificity in identifying a concussion at the moment of injury.[12] These KD, TGT, and SOT are typically used to assess a concussion in the acute phase and there is a lack of literature to support their use in post-concussion syndrome.

We found a significantly higher number of C0, C1, torsion (SBS) and vertical strain (SBS) restrictions in the PCS group than in the concussion history group and in the control group. Through their anatomo-physiological relationship, mobility restrictions of the sphenoid, occiput and atlas could contribute to the persistent concussion symptoms (PCSS), as well as visual and vestibular function. Most of the cervical rotation occurs at the cranio-cervical junction between C0-C2, and C0-C1 is often associated with rotational mechanism of injury seen in traumatic brain injuries[42]. The cranio-cervical junction includes multiple muscles insertions connecting the upper cervical vertebrae to the occiput, temporal, and temporo-mandibular joint. The passage of cranial nerves V, X, XI, XII, the presence of the superior cervical ganglion at C2, and an abundance of vasculature in this area. The vagus nerve (X) is responsible for nausea and contributes to the parasympathetic control of the heart, lungs and digestive tract; the accessory nerve (XI) supplies the sternocleidomastoid and the trapezius muscle and could trigger neck pain and tension; and the hypoglossal nerve (XII) is a motor nerve that supplies the tongue and is involved in speech and swallowing. The superior cervical ganglion at C2 is responsible for several sympathetic innervations such as the pineal gland (circadian rhythm sleep patterns), the blood vessels, the eyes (lachrymal glands, pupillary dilation), and the peripheral vestibular system (balance and dizziness). The cranio-cervical junction is also the location for the passage of the jugular vein, the vertebral artery, and the carotid artery and could affect blood flow. Marshall et al., in a literature review, proposed the involvement of the cervical spine as a contributing factor to post-concussion syndrome.[8] Marshall describes two possible mechanisms

for symptoms of headaches and dizziness related cervical dysfunction: pain and proprioception.[8] A convergence phenomenon of a continuous afferent pathway from C2 dorsal root to the trigeminal sensory nucleus could trigger a headache in the upper cranium and forehead.[8] The trigeminal nerve sits within the greater wing of the sphenoid bone adjacent to the temporal bone, and its 3 branches penetrate different parts of the sphenoid bone to the their targeted distribution.[40,45] As some PCS related symptoms (headache, nausea, dizziness, neck pain, sleep related problems) may not necessarily be specific to the brain injury aspect of the concussion[9] and could be tied to cervical and cranial mobility restrictions, future research on the anatomo-physiological impacts of specific mobility restrictions is warranted.

Research on acute phase concussion pathophysiology suggests ion imbalance, metabolic disruptions, blood flow abnormalities and autonomic dysfunction as the main culprits, and generally demonstrate a return to baseline control levels within 2-4 weeks following the injury.[8] Research on concussion pathophysiology in the chronic stage is sparse.[8] Research also suggests that the brain injury does not cause the symptoms to persist beyond 1 month, therefore alternative explanations must be considered.[9] When compared on an individual basis, each bone demonstrated a higher number of mobility restrictions in the PCS group than in the CHx group and the Ctl group. Mobility restrictions of the cranio-cervical joints can cause headaches and the pain usually starts at the occiput.[9] Additional analysis, across the whole sample, was conducted in the scope of this study and revealed significant associations between the SBS, the temporal, the parietal, the frontal bone, with the occiput (C0), and atlas (C1). Only the temporal bone was associated with the axis (C2). There is reported clinical success using cranial osteopathy for migraines[28], headaches[29], vision (acuity, accommodation and convergence),[30] dizziness[31], and on autonomic function.[32] If the cranial mobility restrictions we found associated with long term concussion symptoms are the result of the mechanism of injury, or develop over time, there could be an explanation for the persistent symptoms of a concussion.

The temporal bone had a significant relationship with all the cranial and cervical bones. The temporal bone houses the vestibular apparatus, the endolymphatic sac, and the vestibulocochlear nerve.[40] The carotid artery passes anteriorly through the foramen lacerum where the sphenoid meets the temporal bone. Where the occiput meets the temporal bone, the jugular vein, and IX, X, XI cranial nerves pass through the jugular foramen, at the cranio-cervical junction.[40] The temporal bone is also linked to the sphenoid, and occiput by the tentorium cerebelli. Through these structural links, a restriction of the temporal bone could be responsible for dizziness and balance issues through changes in orientation in the semi-circular canal or pressure on the endolymphatic sac and vestibulocochlear nerve. Similarly, a mobility restriction

of the temporal bone could influence blood flow through the carotid artery and jugular vein. Finally, a restriction of the temporal bone as its base could influence the cranio-cervical junction provoking headaches and neck pain. These anatomo-physiological relationships could influence certain PCSS symptoms, such as dizziness, balance, headaches and neck pain, and the vestibular and total score of the VOMS.

The sphenoid bone houses the passage of the oculo-motor cranial nerves (II, III, IV, VI), the optic chiasm, the trigeminal nerve, gives insertion to the oculo-motor muscle attachments, and the pituitary aspect of the hypothalamus-pituitary-adrenal axis (HPA). The HPA axis plays a role in autonomic function through regulation of homeostatic systems in the body.[61-64] Together the pituitary gland and hypothalamus are responsible for controlling blood pressure, thyroid gland, metabolism, body temperature, pain relief, thirst, fatigue, and sleep circadian rhythms. HPA integrates the physical and psychosocial influences in order to allow an organism to adapt effectively to its environmental use of resources and optimize survival.[61-64] The pituitary gland sits in the sella turcica of the sphenoid bone and its stalk above is surrounded by the tentorium cerebelli. The trigeminal nerve sits within the greater wing of the sphenoid, is surrounded by the tentorium cerebelli, innervates the dura mater and blood vessels, and its ophthalmic branch is responsible for pupillary reflex. An irritation of the trigeminal nerve has been known to provoke headaches.[8] Understanding of this anatomical link could explain the influence that mobility restrictions of the SBS could have an impact of vision, headaches, and autonomic function. The restrictions could be responsible for the persistence of certain PCSS symptoms such as headaches, pressure in the head, sensitivity to light, vision, sleep, fatigue, as well as the visual and vestibular scores of the VOMS. Further studies could investigate the impact of spheno-basilar synchrondrosis mobility restrictions on the HPA axis and its influence on the autonomous nervous system.

Hernandez et al. investigated the mechanism by which skull movement, produces brain deformation that penetrates deep in the brain structures.[41] They determined that coronal and horizontal head rotation accelerations stiffen the falx cerebri in the center and at the periphery respectively. Both of these lateral displacements of the falx cerebri cause high strains in the corpus callosum, deep in the brain.[41] The corpus collusum connects the left and the right brain hemispheres. Hernandez also described the influence of the impact on the thalamic region, near the corpus collosum, which they explain may simply be due to proximity,[41] however this could influence the HPA axis. The falx cerebri is a meningeal fold that connects the frontal, sphenoid, parietal, and occipital bones via the dura-mater where cerebrospinal fluid circulates and houses the sagittal sinus vein. The falx cerebri, combined with the force of impact on the cranial bones,

could have an impact on the underlying structures. The tentorium cerebelli works with the falx cerebri to maintain tension and compression within the cranium. The tentorium cerebelli is a meningeal fold that links the sphenoid, temporal and occipital bone via the dura-mater, encloses the transverse and superior petrosal sinuses, and surrounds the trigeminal nerve, the endolymphatic sac and the pituitary stalk. The mechanism of impact on the skull could have an influence on the deep structures of the brain, including the HPA axis which could explain the effects on autonomic function. Future studies could investigate the anatomo-physiological implications of an impact to the skull on specific structures and how they could influence blood flow, vision, the vestibular system, and muscle tension. If structure governs function, the anatomy could explain the relationships between the number of restrictions and the PCSS and VOMS scores.

Our study has provided insight on the validity of the cranial mobility tests. Our results contribute to the external validity of the sample as they concur with the literature for post-concussion syndrome on medical histories, positive findings in the PCSS and VOMS, as well as negative findings for the KD, TGT, and SOT. However, our study does not provide insight on internal validity of the sample because our results cannot inform to the cause and effect relationship between the mobility restrictions and post-concussion syndrome. We cannot allude to validity of the cranial bone mobility test itself. Future research could investigate the sensitivity and specificity of the cranial bone mobility tests. We also believe advances in technology will be able to provide insight as to the physiological implications of mobility restrictions, for example in relation to cerebral blood flow.

4.2 Clinical concussion tests

There was a significant group difference for number and severity of symptoms with the PCS exhibiting a higher number of symptoms and greater severity than the concussion history and the control group. While there was a strong correlation between the number of symptoms and symptom severity among the subjects, the higher number and worse symptom severity was negatively associated with scores on the TGT, KD, the VOMS, and SSR across the whole sample. Harrold et al. found similar results as our study, in an adult population averaging 30±16 years of age, n=426.[65] They found that those with higher symptom severity had a higher number of symptoms ($r = 0.85$, $p< 0.0001$), longer KD times ($r = -0.23$, $p= 0.0003$), and longer TGT times ($r = 0.48$, $p= 0.0006$).

The Tandem Gait Test presented no significant difference between groups. The tandem gait test assesses dynamic balance, speed, coordination and requires sensory integration from the visual, vestibular and proprioceptive systems.[66] Balance issues typically resolve within 3-5

days following the concussion,[60] therefore we would not expect to see group differences in a post-concussion population. Research is demonstrating different norms within different populations.[66,67] The Sports Concussion Assessment Tool (SCAT, 2013) suggests a cutoff value of 14 seconds for a positive result.[3] Oldham et al. established norms for healthy collegiate-level NCAA athletes, with a mean age of 20 years old. All results, regardless of sex, collision vs non-collision sport, and history of previous concussion ranged from 10.10-11.43 seconds.[66] In a more recent study, Galea et al. established normative data within different age categories in a general population of healthy adults between the ages of 18 and 55,[67] results differ greatly from previous research.[66] Galea established a mean time of 18.49 seconds between the ages of 18-24 category.[67] Our Tandem Gait test times were longer than the norm presented by Oldham et al., which could reflect the level of sport participation. No baseline data was available on our participants, however 50% of the Ctl group, 18% of the CHx, and 24% of the PCS group had results above those suggested by Galea et al. These elevated percentages compared to Galea, as well as the inconsistencies between studies, support the fact that TGT results following a concussion should be compared to baseline results.

There was no significant difference between groups on the King-Devick Test. The King-Devick test is a vision-based rapid number naming task that uses both cognitive function and vision.[12] A recent study performed by Vartiainen et al. suggest that post-injury KD results be compared to either individual pre-season performance or with normative data. Vartiainen established a norm of 36.5-43.9 seconds, n=185.[68] In 2015 Galetta et al. performed a meta-analysis and systematic review of the literature on the King Devick test.[17] Studies with larger sample sizes (n=217), average age of 20.3 years old, male and female college football and basketball players[69] had an average of 38.5 seconds. Another group (n=152), of male and female college football and basketball players (average age 19.6 years) averaged 36.3 seconds.[70] No baseline data was available on the participants of our study for comparison; 62% of the PCS group, 55% of the CHx group, and 42% of the Ctl group had results above the range suggested by Vartiainen et al. on the KD. These elevated percentages suggest that results are population specific and supports comparing KD results to an individual's baseline data. Considering the absence of group differences might suggest that the King-Devick test is valid in the acute stages rather than in the chronic stages. There was a low correlation between the KD and TGT results in our study. Our results are similar to those found by Harrold et al. where they found that longer KD times were associated with longer TGT times ($r = 0.43$, $p = 0.002$).[65]

There was a significant difference in the Vestibulo-Oculo-Motor Screening test between groups. Higher VOMS scores were found in the PCS group compared to the concussion history

group and the control group. Eagle et al. assessed differences in established concussion-specific evaluations, Immediate Postconcussion Assessment and Cognitive Testing (ImPACT), Post-Concussion Symptom Scale (PCSS), and Vestibular–Ocular-Motor Screening (VOMS), between individuals with no history of concussion and individuals who had a history of sport-related concussion and been cleared to return to play.[71] The concussed group average 263.83±228.92 days since the last concussion and averaged 2.71±1.52 previous concussions. The concussed group reported more vestibular/ocular symptoms after horizontal/vertical saccades, horizontal/vertical VOR and VMS. The VOMS evaluation requires a higher level of sensory integration, with deficiencies still present after symptom resolution and clearance to return-to-play.[71] Given the group differences seen in our study, VOMS results could be a useful tool to monitor concussion recovery beyond symptom resolution to help determine return to play status.

Our results revealed a correlation between the total visual, total vestibular and total score on the VOMS test with the King-Devick test and the TGT. To our knowledge this is the first study to demonstrate a correlation in an adult population. Russel-Giller et al. found that KD testing times correlated with all VOMS items ($r(69) = 0.325\text{-}0.585$, $p < 0.01$) in a youth population averaging 14 years of age. Russel-Giller suggests that prolonged KD testing times could be related to subtypes of vestibular/ocular motor impairment other than visual saccadic abnormalities.[72]

Postural control is maintained through the combined afferent information generated by the somatosensory (proprioception), visual and vestibular systems.[18] A change in overall balance could be driven by a suppressed visual or vestibular system functioning or an inefficient integration of the vestibular information.[18] The SOT was designed to objectively identify abnormalities in the participant's ability to use these 3 sensory systems that contribute to postural control. Previous research has supported the use of the SOT in differentiating concussed individuals from healthy controls, in the acute stages following the injury.[11] However, balance issues typically resolve within 3-5 days following the concussion.[60] The sensory organization test done on the Neurocom in our study did not present a significant difference between groups. Our results suggest that the participants may not have had issues with postural control and that they were able to integrate the visual and vestibular information available, even though the VOMS symptom provocation was positive.

The number of predisposing factors and previous head injuries must be taken into consideration during the initial assessment to avoid complications that may lead to a prolonged recovery. There was a significantly greater number of predisposing factors and head injuries in

the PCS group compared to the CHx group and Ctl group. The results also demonstrated a moderate correlation between the number of head injuries and the number of predisposing factors, although we cannot allude to the type of association. The number of head injuries and predisposing factors was also associated with several of the variables: number of restrictions, PCSS, VOMS, as well as the predisposing factors with KD. Among the predisposing factors, there was a greater number of participants with medication intake (PCS= 9, CHx=0, Ctl=3), diagnosed migraine or headache (PCS= 10, CHx=2, Ctl=0), sleep-related problems (PCS= 14, CHx=1, Ctl=0), and anxiety/depression (PCS= 5, CHx=2, Ctl=2), in the PCS group compared to the CHx group and Ctl group as seen in Fig. 1. These results are consistent with the literature.[3,7] Headache is the most common symptom reported in concussions.[7] Sleep-related problems are a common symptom of concussion.[7] Sleep may be disturbed from autonomic dysfunction, emotional symptoms, neck pain, and are not exclusive to concussion, but are a predisposing factor to developing post-concussion syndrome.[6,7] This may allude to the possibility that with a growing number of concussions, the presence of predisposing factors may prolong concussion recovery.

4.3 Strengths and limitations

Some of the strengths of this study are the methodology, the consensus training elaborated in the preliminary reliability study to achieve consistency within this study, and the experience of the health care professionals. The methodology was rigorous, detailed, and strongly enforced. The manual evaluation was double-blind, the evaluator had no contact with the participant (aside from the hands in contact with the head), and no knowledge of group attribution. The clinical evaluator was single-blind, had no knowledge of group attribution, had minimal communication and interaction with the participant, limited to the test instructions and noting the data. Both evaluators had no access to the results throughout the entire data collection period. A research assistant, not blinded to group attribution, did all the recruiting, interviews over the phone, scheduling of participants, and directed the participant on the day of the assessment. All 3 members of the research project did multiple hours of consensus training practicing the tests and applying established criteria for positive and negative findings. Finally, both evaluators had over 5 years of experience in the field and were meticulous about consistency. All these factors follow Guillaud's suggestions for successful clinical research.[44]

There are limiting factors to this study. The study was intended to compare the PCS group to a control group. Hits to the head is a difficult element to control and in future studies greater care needs to be taken in the recruitment interview to screen for history of concussion, sports practiced and impacts to the head for concussion-related cranial mobility studies. Given

that concussions have multiple facets and are very complex to study, phone interviews upon recruitment need to be more detailed to see if the participant fits the intended profile. The medical questionnaire filled out the day of the assessment could have been more detailed to include more information concerning concussion timeline and mechanism of injury. This information could have offered insight on anatomical reasoning behind different mobility restrictions and positive clinical findings for further analysis. Also, the medical questionnaire and post-concussion symptom scale were self-reported. Questions could have been misunderstood or details could have been omitted that could have furthered the analysis. Finally, the sample number was not as large as intended. With the large variety of clinical profiles possible with concussions, this could have influenced the results as some tests neared significance.

5. Conclusion

Our research found a greater number of cranial and upper cervical mobility restrictions in the PCS group compared to the Ctl group. There was a correlation between the number of restrictions and the PCSS, the VOMS scores, the number of head injuries and the number of predisposing factors. This is the first study to investigate the prevalence of cranial mobility restrictions in a concussion population. Currently, the literature only describes different case studies on osteopathic manual therapy for concussions and lack empirical data. Current research does not provide insight on the anatomo-physiological relationships, the impact of a specific mobility restriction, specific techniques nor their efficacy, nor at which point in the rehabilitation these therapies should be used or with which treatment parameters.

Current research supports positive outcomes of cervical manual therapy on concussion recovery, and cranial manual osteopathy as a treatment for migraines, headaches, dizziness, vision function, and autonomic function. As some PCS related symptoms (headache, nausea, dizziness, neck pain, sleep related problems) may not necessarily be specific to the brain injury aspect of the concussion and could be tied to cervical and cranial mobility restrictions, these restrictions should be investigated within the concussion assessment. Research in acute stages could help determine if the mechanism of injury causes mobility restrictions or if they develop over time, and investigate the sensitivity and specificity of the cranial mobility tests in assisting in the identification of a concussion. With advances in technology, research could investigate the physiological implications of cranial and upper cervical mobility restrictions. With answers to these questions, future research could study cranial manual therapy as an alternative treatment approach for concussions. The question also remains, could the persistent mobility restrictions of the cranial bones and upper cervical left untreated become a predisposing factor for prolonged recovery in subsequent concussions? Also, do mobility restrictions influence the

anatomo-physiological relationship involved in blood flow, vision, the vestibular system, and the cranio-cervical junction musculature? More studies are required to answer these important questions.

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Appendix 1:

Table 1: Inclusion and exclusion criteria by group.

Inclusion Criteria		
Post-Concussion Syndrome Group (PCS)	Concussion History Group (CHx)	Control Group (Ctl)
- Healthy physically active adults between ages of 18-35 years old. - Having sustained a concussion 1 month ago or greater. - Having 3 concussion symptoms or more still present	- Healthy physically active adults between ages of 18-35 years old. - Having recovered from at least 1 previous concussion - Participation in collision or non-collision sports	- Healthy physically active adults between ages of 18-35 years old.
Exclusion Criteria		
Post-Concussion Syndrome Group (PCS)	Concussion History Group (CHx)	Control Group (Ctl)
- motor vehicle accident in last 5 years (unless this current concussion is from MVA) - previous skull fracture - chronic neck pain - severe learning disabilities - psychiatric disorders - neurological conditions - who are currently under prescribed medication that may cause dizziness, influence motor control or mimic concussion symptoms - having received cranial and/or upper cervical manual therapy or this concussion	- motor vehicle accident - previous skull fracture - chronic neck pain - severe learning disabilities - psychiatric disorders - neurological conditions - who are currently under prescribed medication that may cause dizziness, influence motor control or mimic concussion symptoms.	- previous concussion, or sub-concussive impact (hit to the head) - any participation in a collision sport where hits to the head are frequent - motor vehicle accident - previous skull fracture - chronic neck pain - severe learning disabilities - psychiatric disorder - neurological conditions - who are currently under prescribed medication that may cause dizziness, influence motor control or mimic concussion symptoms.

Table 2. Characteristics of participant's medical histories.

	Ctl group	CHx group	PCS group
Diagnosis of learning disability	0	1	0
Diagnosis of attention deficit	1	2	3
Diagnosis of anxiety or depression	2	2	5
On prescription medication	3	0	9
Diagnosis of migraine/headache	0	2	10
Sleep related problems	0	1	14
History of concussion	0	11	17
Total number of predisposing factors	3	12	45
Total number of concussions	0	16	48

Table 3. Group descriptive statistics for continuous variables.

Variable	Group	N	Mean	Standard Deviation	95% Confidence Interval	
					Lower bound	Upper bound
NR	Ctl	12	2.92	3.8	.502	5.33
	CHx	11	5.91	4.41	2.94	8.88
	PCS	21	8.24	4.25	6.30	10.17
	Total	44	6.20	4.66	4.79	7.62
#Sx	Ctl	12	1.75	3.19	-.28	3.78
	CHx	11	1.36	2.11	-.05	2.78
	PCS	21	10.9	5.8	8.27	13.54
	Total	44	6.02	6.45	4.06	7.98
Sx Sev	Ctl	12	2.92	6.22	-1.03	6.87
	CHx	11	2.0	3.82	-.57	4.57
	PCS	21	25.43	25.36	13.89	36.97
	Total	44	13.43	21.14	7.0	19.86
KD	Ctl	12	44.42	8.24	39.19	49.65
	CHx	11	43.58	8.52	37.86	49.31
	PCS	21	50.75	15.0	43.92	57.58
	Total	44	47.23	12.27	43.50	50.96
TGT	Ctl	12	18.19	2.89	16.35	20.02
	CHx	11	16.13	1.95	14.82	17.44
	PCS	21	16.54	4.20	14.63	18.46
	Total	44	16.89	3.45	15.84	17.94
TVis	Ctl	12	3.42	4.93	.29	6.55
	CHx	11	3.0	7.07	-1.75	7.75
	PCS	21	27.81	29.16	14.53	41.08
	Total	44	14.95	23.83	7.71	22.20
TVest	Ctl	12	2.58	3.15	.58	4.58
	CHx	11	4.0	7.09	-.76	8.76
	PCS	21	26.48	24.36	15.39	37.57
	Total	44	14.34	20.69	8.05	20.63

TScore	Ctl	12	6.0	8.01	.91	11.09
	CHx	11	7.0	14.06	-2.44	16.44
	PCS	21	54.76	53.30	30.50	79.02
	Total	44	29.52	44.49	16.0	43.05
SSR	Ctl	12	.97	.02	.96	.99
	CHx	11	.97	.02	.95	.98
	PCS	21	1.0	.10	.95	1.04
	Total	44	.98	.07	.96	1.0
VisR	Ctl	12	.78	.16	.67	.88
	CHx	11	.85	.09	.79	.91
	PCS	21	.81	.16	.74	.89
	Total	44	.81	.15	.77	.86
VestR	Ctl	12	.58	.12	.50	.65
	CHx	11	.72	.12	.64	.80
	PCS	21	.64	.16	.57	.72
	Total	44	.64	.15	.60	.69
PrefR	Ctl	12	.95	.09	.89	1.01
	CHx	11	.99	.10	.92	1.06
	PCS	21	1.0	.12	.94	1.05
	Total	44	.98	.11	.95	1.02
HI	Ctl	12	0	0	0	0
	CHx	11	1.09	.30	.89	1.29
	PCS	21	2.33	.86	1.94	2.72
	Total	44	1.39	1.17	1.03	1.74
PF	Ctl	12	.25	.45	-.04	.54
	CHx	11	1.45	1.37	.54	2.37
	PCS	21	2.29	1.01	1.83	2.74
	Total	44	1.52	1.30	1.13	1.92

Table 4. One-way ANOVA between and within groups for the continuous variables.

Variable		Sum of Squares	df	Mean Squares	F	Sig.
NR	Between Groups	217.524	2	108.762	6.231	.004
	Within Groups	715.635	41	17.455		
	Total	933.159				
#Sx	Between Groups	958.372	2	479.186	23.710	.000
	Within Groups	828.605	41	20.210		
	Total	1786.977	43			
SxSev	Between Groups	5786.736	2	2893.368	8.833	.001
	Within Groups	13430.060	41	327.562		
	Total	19216.795	43			
KD	Between Groups	501.401	2	250.701	1.721	.192
	Within Groups	5973.418	41	145.693		
	Total	6474.820	43			

TGT	Between Groups	29.024	2	14.512	1.232	.302
	Within Groups	483.053	41	11.782		
	Total	512.078	43			
TVis	Between Groups	6639.754	2	3319.877	7.656	.001
	Within Groups	17778.155	41	433.614		
	Total	24417.909	43			
TVest	Between Groups	5927.732	2	2963.866	9.735	.000
	Within Groups	12482.155	41	304.443		
	Total	18409.886	43			
TScore	Between Groups	25597.168	2	12798.584	8.819	.001
	Within Groups	59503.810	41	1451.312		
	Total	85100.977	43			
SSR	Between Groups	.007	2	.004	.700	.502
	Within Groups	.218	41	.005		
	Total	.225	43			
VisR	Between Groups	.032	2	.016	.726	.490
	Within Groups	.901	41	.022		
	Total	.933	43			
VestR	Between Groups	.116	2	.058	2.814	.072
	Within Groups	.842	41	.021		
	Total	.958	43			
PrefR	Between Groups	.017	2	.009	.686	.509
	Within Groups	.512	41	.012		
	Total	.529	43			
HI	Between Groups	42.856	2	21.428	56.405	.000
	Within Groups	15.576	41	.380		
	Total	58.432	43			
PF	Between Groups	31.714	2	15.587	15.756	.000
	Within Groups	41.263	41	1.006		
	Total	72.977	43			

Table 5. Post hoc comparisons using Tukey post hoc test for multiple group comparisons.

Variable	(I) Group	(J) Group	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
						Lower Bound	Upper Bound
NR	Ctl	CHx	-2.992	1.744	.212	-7.233	1.248
		PCS	-5.321*	1.512	.003	-8.998	-1.645
	CHx	Ctl	2.992	1.744	.212	-1.248	7.233
		PCS	-2.329	1.555	.303	-6.110	1.452

	PCS	Ctl	5.321*	1.512	.003	1.645	8.998
		CHx	2.329	1.555	.303	-1.452	6.110
#Sx	Ctl	CHx	.386	1.877	.977	-4.177	4.950
		PCS	-9.155*	1.627	.000	-13.111	-5.199
	CHx	Ctl	-.386	1.877	.977	-4.950	4.177
		PCS	-9.541*	1.673	.000	-13.610	-5.472
	PCS	Ctl	9.155*	1.627	.000	5.199	13.111
		CHx	9.541*	1.673	.000	5.473	13.610
SxSev	Ctl	CHx	.917	7.555	.992	-17.454	19.287
		PCS	-22.512*	6.549	.004	-38.438	-6.586
	CHx	Ctl	-.917	7.555	.992	-19.287	17.454
		PCS	-23.429*	6.736	.003	-39.809	-7.048
	PCS	Ctl	22.512*	6.549	.004	6.586	38.438
		CHx	23.429*	6.736	.003	7.048	39.809
KD	Ctl	CHx	.835	5.038	.985	-11.417	13.086
		PCS	-6.332	4.368	.325	-16.954	4.289
	CHx	Ctl	-.835	5.038	.985	-13.086	11.417
		PCS	-7.167	4.493	.259	-18.091	3.757
	PCS	Ctl	6.332	4.368	.325	-4.289	16.954
		CHx	7.167	4.493	.259	-3.757	18.091
TGT	Ctl	CHx	2.056	1.433	.333	-1.428	5.540
		PCS	1.642	1.242	.391	-1.378	4.663
	CHx	Ctl	-2.056	1.433	.333	-5.539	1.428
		PCS	-.413	1.278	.944	-3.520	2.693
	PCS	Ctl	-1.642	1.242	.391	-4.663	1.378
		CHx	.413	1.278	.944	-2.693	3.520
TVis	Ctl	CHx	.417	8.692	.999	-20.720	21.553
		PCS	-24.393*	7.535	.007	-42.716	-6.069
	CHx	Ctl	-.417	8.692	.999	-21.553	20.720
		PCS	-24.810*	7.750	.007	-43.656	-5.963
	PCS	Ctl	24.393*	7.535	.007	6.069	42.716
		CHx	24.810*	7.750	.007	5.963	43.656
TVest	Ctl	CHx	-1.417	7.283	.979	-19.127	16.294
		PCS	-23.893*	6.314	.001	-39.246	-8.539
	CHx	Ctl	1.417	7.283	.979	-16.294	19.127
		PCS	-22.476*	6.494	.004	-38.268	-6.685
	PCS	Ctl	23.893*	6.314	.001	8.539	39.246
		CHx	22.476*	6.494	.004	6.685	38.268
TScore	Ctl	CHx	-1.000	15.902	.998	-39.669	37.669
		PCS	-48.762*	13.786	.003	-82.285	-15.239
	CHx	Ctl	1.000	15.902	.998	-37.669	39.669
		PCS	-47.762*	14.179	.005	-82.241	-13.283
	PCS	Ctl	48.762*	13.786	.003	15.239	82.285
		CHx	47.762*	14.179	.005	13.283	82.241
SSR	Ctl	CHx	.006	.030	.976	-.068	.080
		PCS	-.024	.026	.672	-.087	.042
	CHx	Ctl	-.006	.030	.976	-.080	.068
		PCS	-.029	.027	.539	-.095	.037
	PCS	Ctl	.024	.026	.672	-.042	.087

		CHx	.029	.027	.539	-.037	.095
VisR	Ctl	CHx	-.075	.062	.458	-.225	.076
		PCS	-.034	.054	.807	-.164	.097
	CHx	Ctl	.075	.062	.458	-.076	.225
		PCS	.041	.055	.740	-.093	.175
	PCS	Ctl	.034	.054	.807	-.097	.164
		CHx	-.041	.055	.740	-.175	.093
VestR	Ctl	CHx	-.142	.060	.057	-.287	.004
		PCS	-.065	.052	.431	-.191	.061
	CHx	Ctl	.142	.060	.057	-.004	.287
		PCS	.077	.053	.329	-.053	.207
	PCS	Ctl	.065	.052	.431	-.062	.191
		CHx	-.077	.053	.329	-.207	.053
PrefR	Ctl	CHx	-.042	.047	.644	-.155	.071
		PCS	-.045	.040	.506	-.147	.053
	CHx	Ctl	.042	.047	.644	-.071	.155
		PCS	-.003	.042	.996	-.105	.098
	PCS	Ctl	.045	.040	.506	-.053	.144
		CHx	.003	.042	.996	-.098	.105
HI	Ctl	CHx	-1.091*	.257	.000	-1.716	-.465
		PCS	-2.333*	.223	.000	-2.876	-1.791
	CHx	Ctl	1.091*	.257	.000	.465	1.717
		PCS	-1.242*	.229	.000	-1.800	-.685
	PCS	Ctl	2.333*	.223	.000	1.791	2.876
		CHx	1.242*	.229	.000	.685	1.800
PF	Ctl	CHx	-1.205*	.419	.017	-2.223	-.186
		PCS	-2.036*	.363	.000	-2.918	-1.153
	CHx	Ctl	1.205*	.419	.017	.186	2.223
		PCS	-.831	.373	.079	-1.739	.077
	PCS	Ctl	2.036*	.363	.000	1.153	2.918
		CHx	.831	.373	.079	-.077	1.739

Table 6. Pearson Chi-Square correlation group statistics of the cranial bones and upper cervical vertebrae. PCS n=21, CHx n=11, Ctl n=12, total n=44.

	Group	Not restricted	Restricted	Pearson Chi-square	Sig. (2-tailed)
C0	Ctl	7	5	14.633*	.001
	CHx	1	10		
	PCS	1	20		
		9	35		
C1	Ctl	12	0	7.071*	.029
	CHx	8	3		
	PCS	12	9		
		32	12		
C2	Ctl	9	3	3.182	.204
	CHx	6	5		
	PCS	9	12		
		24	20		
SBS	Ctl	6	6	2.365	.307
	CHx	4	7		
	PCS	5	16		
		15	29		
Flex/Ext	Ctl	10	2	4.140	.126
	CHx	7	4		
	PCS	19	11		
		27	17		
Torsion	Ctl	11	1	8.386*	.015
	CHx	10	1		
	PCS	11	10		
		32	12		
SBR	Ctl	9	3	5.836	.054
	CHx	4	7		
	PCS	7	14		
		20	24		
Vertical Strain	Ctl	9	3	8.187*	.017
	CHx	5	6		
	PCS	5	16		
		19	25		
Lateral Strain	Ctl	8	4	3.277	.194
	CHx	8	3		
	PCS	9	12		
		25	19		
Temporal	Ctl	8	4	5.928	.052
	CHx	5	6		
	PCS	5	16		
		18	26		
Parietal	Ctl	9	3	5.752	.056
	CHx	8	3		
	PCS	8	13		
		25	1		
Frontal	Ctl	10	2	5.483	.064

	CHx	4	7		
	PCS	11	10		
		25	19		

Table 7: Pearson Chi-Square correlations between mobility restrictions of the cranial bones and upper cervical vertebrae (n=44).

	C0	C1	C2	SBS	Temporal	Parietal	Frontal
C0	1	4.243*	.670	15.121*	16.343*	8.599*	8.599*
Sig. 2-tailed		0.039	0.413	0.000	0.000	0.003	0.003
C1	4.243*	1	2.994	4.872*	7.243*	3.709	0.313
Sig. 2-tailed	0.039		0.084	0.027	0.007	0.054	0.576
C2	.670	2.994	1	3.240	6.631*	2.087	0.695
Sig. 2-tailed	0.413	0.084		0.072	0.010	0.149	0.405
SBS	15.121*	4.872*	3.240	1	32.874*	17.297*	12.368*
Sig. 2-tailed	0.000	0.027	0.072		0.000	0.000	0.000
Temporal	16.343*	7.243*	6.631*	32.874*	1	23.151*	17.577*
Sig. 2-tailed	0.000	0.007	0.010	0.000		0.000	0.000
Parietal	8.599*	3.709	2.087	17.297*	23.151*	1	5.439*
Sig. 2-tailed	0.003	0.054	0.149	0.000	0.000		0.020
Frontal	8.599*	0.313	0.695	12.368*	17.577*	5.439*	1
Sig. 2-tailed	0.003	0.576	0.405	0.000	0.000	0.020	

Table 8. Pearson Correlations between the continuous variables (number of symptoms, symptom severity, number of restrictions, number of head injuries, number of predisposing factors, King-Devick and Tandem Gate Test) (n=44).

	NR	#Sx	SxSev	HI	PF	KD	TGT
#Sx	0.283	1	.890*	.621*	.649*	.448*	0.215
Sig. 2-tailed	0.062		0.000	0.000	0.000	0.002	0.161
SxSev	.333*	.890*	1	.500*	.541*	.606*	.423*
Sig. 2-tailed	0.027	0.000		0.001	0.000	0.000	0.004
NR	1	0.283	.333*	.396*	.338*	0.211	-0.076
Sig. 2-tailed		0.062	0.027	0.008	0.025	0.170	0.624
HI	.396*	.621*	.500*	1	.553*	0.189	-0.173
Sig. 2-tailed	0.008	0.000	0.001		0.000	0.219	0.263
PF	.338*	.649*	.541*	.553*	1	.455*	-0.034
Sig. 2-tailed	0.025	0.000	0.000	0.000		0.002	0.827
KD	0.211	.448*	.606*	0.189	.455*	1	.494**
Sig. 2-tailed	0.170	0.002	0.000	0.219	0.002		0.001
TGT	-0.076	0.215	.423*	-0.173	-0.034	.494*	1
Sig. 2-tailed	0.624	0.161	0.004	0.263	0.827	0.001	
TVis	0.267	.754*	.856*	.452*	.392*	.430*	.431*
Sig. 2-tailed	0.079	0.000	0.000	0.002	0.009	0.004	0.003
TVest	.305*	.738*	.819*	.484*	.388*	.374*	.366*
Sig. 2-tailed	0.044	0.000	0.000	0.001	0.009	0.012	0.015
TScore	0.293	.754*	.845*	.470*	.396*	.414*	.410*
Sig. 2-tailed	0.054	0.000	0.000	0.001	0.008	0.005	0.006
SSR	0.000	.346*	.313*	-0.011	0.195	-0.026	0.184
Sig. 2-tailed	1.000	0.021	0.039	0.945	0.205	0.865	0.233
VisR	-0.123	-0.089	-0.248	-0.066	0.040	-0.305*	-0.188

Sig. 2-tailed	0.428	0.567	0.105	0.669	0.798	0.044	0.221
VestR	0.058	0.043	-0.048	0.157	0.159	-0.139	-0.118
Sig. 2-tailed	0.708	0.780	0.759	0.309	0.303	0.370	0.447
PrefR	0.108	0.010	0.009	0.001	0.058	0.016	-0.036
Sig. 2-tailed	0.487	0.949	0.954	0.993	0.707	0.918	0.818

Table 9. Pearson correlation between the continuous variables of VOMS and Neurocom scores (n=44).

	TVisual	TVestibular	TScore	NC:SSR	NC:VisR	NC:VestR	NC:PrefR
TVisual	1	.974*	.994*	.559*	-0.108	0.015	0.073
Sig. 2-tailed		0.000	0.000	0.000	0.483	0.922	0.636
TVest	.974*	1	.992*	.553*	-0.120	-0.054	0.173
Sig. 2-tailed	0.000		0.000	0.000	0.439	0.727	0.262
TScore	.994*	.992*	1	.558*	-0.112	-0.019	0.112
Sig. 2-tailed	0.000	0.000		0.000	0.469	0.904	0.469
SSR	.559*	.553*	.558*	1	.435*	0.213	0.089
Sig. 2-tailed	0.000	0.000	0.000		0.003	0.165	0.564
VisR	-0.108	-0.120	-0.112	.435*	1	.640*	-0.080
Sig. 2-tailed	0.483	0.439	0.469	0.003		0.000	0.606
VestR	0.015	-0.054	-0.019	0.213	.640*	1	-0.253
Sig. 2-tailed	0.922	0.727	0.904	0.165	0.000		0.098
PrefR	0.073	0.173	0.112	0.089	-0.080	-0.253	1
Sig. 2-tailed	0.636	0.262	0.469	0.564	0.606	0.098	

Appendix 2:

Participant code: _____
Date: _____
Time: _____

Post-Concussion Symptom Scale

Symptom	None	Mild		Moderate		Severe	
Headache	0	1	2	3	4	5	6
"Pressure in head"	0	1	2	3	4	5	6
Neck Pain	0	1	2	3	4	5	6
Nausea or vomiting	0	1	2	3	4	5	6
Dizziness	0	1	2	3	4	5	6
Vision problems	0	1	2	3	4	5	6
Balance problems	0	1	2	3	4	5	6
Sensitivity to light	0	1	2	3	4	5	6
Sensitivity to noise	0	1	2	3	4	5	6
Feeling slowed down	0	1	2	3	4	5	6
Feeling like "in a fog"	0	1	2	3	4	5	6
"Don't feel normal"	0	1	2	3	4	5	6
Problems concentrating	0	1	2	3	4	5	6
Problems remembering	0	1	2	3	4	5	6
Fatigue or low energy	0	1	2	3	4	5	6
Confusion	0	1	2	3	4	5	6
Drowsiness	0	1	2	3	4	5	6
Trouble falling asleep	0	1	2	3	4	5	6
More emotional	0	1	2	3	4	5	6
Irritability	0	1	2	3	4	5	6
Sadness	0	1	2	3	4	5	6
Nervous or Anxious	0	1	2	3	4	5	6
Total number of symptoms (maximum possible 22)							
Symptom severity score (maximum possible 132)							

Participant code: _____
Date: _____
Time: _____
Clinical evaluator code: _____

Cervical Range of Motion

Direction	Degrees
Flexion	
Extension	
Right Side bending	
Left side bending	
Right Rotation	
Left Rotation	

Cranial nerves- Neurological evaluation

Cranial Nerves	**V**	**VII**	**VIII**	**IX/X**	**XI**	**XII**
1: Normal						
2: Dysfunctional						

The King-Devick Test

	Time (seconds)	**Errors (number)**
Card 1		
Card 2		
Card 3		
Total		

Trial 1:
2---5------8----0------7
3----7---9-----4----6
5-----3-----1-----6----4
7---9----7----3-----5
1----5---4------9------2
6----5-----5------7-----3
3-----1-----8----6------4
5-----3-----7---5-----2

Trial 2:
3 7 5 9 0
2 5 7 4 6
8 4 7 6 3
7 9 3 9 0
4 5 2 1 7
5 3 7 4 8
7 4 6 5 2
9 0 2 3 6

Trial 3:
5 4 1 8 0
4 6 3 5 9
7 5 4 2 7
3 2 6 9 4
2 4 5 1 3
9 3 4 8 5
5 1 6 3 1
4 3 5 2 7

Participant code: _____
Date: _____
Time: _____
Clinical evaluator code: _____

Tandem Gait test

Repetition	Time (seconds)	Errors (#)	Best time
1			□
2			□
3			□

Vestibular/Ocular-Motor Screening

Test:	Headache (0-10)	Dizziness (0-10)	Nausea (0-10)	Fogginess (0-10)	1: Normal 2: Dysfunctional
Baseline symptoms					
Smooth Pursuit					
Comments					
Saccades - Horizontal					
Comments					
Saccades - Vertical					
Comments					
Convergence					
Measure	1: ___cm	2: ___cm	3: ___cm		
VOR - Horizontal					
Comments					
VOR - Vertical					
Comments					
Visual Motion Sensitivity					
Comments					

Participant code: _____
Date: _____
Time: _____
Manual evaluator code: _____

Manual Evaluation Form

Structure	Not Restricted	Restricted	Comments
Occiput- Right condyle	☐	☐	
Occiput- Left condyle	☐	☐	
C1	☐	☐	
C2	☐	☐	
SBS- Flexion	☐	☐	
SBS- Extension	☐	☐	
SBS- Right torsion	☐	☐	
SBS- Left torsion	☐	☐	
SBS- Right SBR	☐	☐	
SBS- Left SBR	☐	☐	
SBS- High vertical strain	☐	☐	
SBS- Low vertical strain	☐	☐	
SBS- Right lateral strain	☐	☐	
SBS- Left lateral strain	☐	☐	
SBS	☐	☐	
Right temporal	☐	☐	
Left temporal	☐	☐	
Right Parietal	☐	☐	
Left Parietal	☐	☐	
Frontal	☐	☐	

Comments legend:

P: Physiological dysfunction

T: Traumatic dysfunction

I.O.: Intra-osseous dysfunction

ER: External Rotation

IR: Internal Rotation