Case Report

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Case report: Manual therapies promote resolution of persistent post-concussion symptoms in a 24-year-old athlete

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Abstract

This case report illustrates the treatment outcomes of a collegiate athlete presenting with an 18-month history of post-concussion syndrome who received a series of mixed manual therapies in isolation of other therapy. Persistent symptoms were self-reported as debilitating, contributing to self-removal from participation in school, work, and leisure activities. Patient and parent interviews captured the history of multiple concussions and other sports-related injuries. Neurological screening and activities of daily living were baseline measured. Post-Concussion Symptom Checklist and Headache Impact Test-6™ were utilized to track symptom severity. Treatments applied included craniosacral therapy, manual lymphatic drainage, and glymphatic techniques. Eleven treatment sessions were administered over 3 months. Results indicated restoration of oxygen saturation, normalized pupil reactivity, and satisfactory sleep. Post-concussion syndrome symptom severity was reduced by 87% as reflected by accumulative Post-Concussion Symptom Checklist scores. Relief from chronic headaches was achieved, reflected by Headache Impact Test-6 scores. Restoration of mood and quality of life were reported. A 6-month follow-up revealed symptoms remained abated with full re-engagement of daily activities. The author hypothesized that post-concussion syndrome symptoms were related to compression of craniosacral system structures and lymphatic fluid stagnation that contributed to head pressure pain, severe sleep deprivation, and multiple neurological and psychological symptoms. Positive outcomes over a relatively short period of time without adverse effects suggest these therapies may offer viable options for the treatment of post-concussion syndrome.

Keywords

Post-concussion symptoms (PCS), traumatic brain injury (TBI), craniosacral therapy, manual lymphatic drainage, glymphatic system, craniosacral system compression, interstitium

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Introduction

Serious awareness over the past decade of the short- and long-term effects of traumatic head trauma leading to concussion has prompted rigorous diagnostic and clinical management endeavors.[1,2] Sources report the number of sports-related concussions receiving medical attention in the United States ranged between 1.6 million and 3.8 million annually.[3,4] Persistent symptoms following concussions may not reflect a single pathophysiological entity and likely describes individualized patterns of non-specific, post-traumatic symptoms that may be linked to coexisting and/or confounding issues.[1,5] Subtypes of post-concussion syndrome (PCS) are now recognized by physiological, cervicogenic, and neuropsychological manifestations.[2,4,6–9]

A fluid model: glymphatic and meningeal lymphatic systems

The existence of a cerebral fluid-exchange system (different than blood vessels) has been long debated but new discoveries have intensified research into fluid dynamics within the brain parenchyma.[10] Glial cells have been shown to create a

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possible architectural system among the microvasculature which appears to match blood flow with neuronal firing.[11] Pathways formed by glial cells and endothelial walls of penetrating blood vessels have been suggested to serve as a physiological cleansing mechanism called the "glymphatic system."[10,12,13] These pathways appear to form perivascular channels by astroglia end-feet where monitoring and active clearing of the neuronal terrain may occur.[14–16] Furthermore, it is theorized that this cellular transportation network also serves to distribute nutritional compounds and neuromodulators.[15] Brain-wide perivascular pathways, believed to facilitate cerebrospinal fluid (CSF) flow through the parenchyma and the movement of interstitial solutes, have been captured by contrast-enhanced magnetic resonance imaging (MRI) scan.[13] The theoretical function then of this proposed glymphatic system serves to support perfusion of CSF and interstitial fluids as a means of pseudo-lymphatic function of the nervous system, elucidating a possible mechanism of how the brain cleans itself through elimination of soluble proteins and metabolites.[10,13,17–19]

However, there are also proponents who argue a glymphatic system as proposed needs to be re-evaluated, accounting for more specifics to cerebrovascular fluid transport.[20] Regardless, there is within the brain a mechanism that moves fluids through a cleansing process, shown to be active during restorative sleep and rather inactive during wakefulness.[15]

Lymphatic system discovery

In addition, a recent identification of lymphatic projections into meningeal membranes within the cranial vault suggests a functional proximity and/or connection between the brain pseudo-lymphatic and the whole-body lymphatic network.[21,22] Furthermore, lymphatic vessels were discovered lining the dura sinuses of the meninges and in turn have been shown to carry both fluid and immune cells from the CSF, connecting to deep cervical lymph nodes and into the peripheral venous return system.[17,21] Magnetic resonance images provide supporting evidence of glymphatic drainage from human brain to cervical lymph nodes.[23] Other emerging data from both animal models and human studies support this theory that the brain fluid-exchange and body lymphatics system share an intimacy and work together for homeostasis, though we are only at the beginning of understanding how this works.[12,22–24]

Structural impact and pathology of fluid transport systems

Chronic symptoms associated with PCS have been theorized to be a result of "gliopathy," a dysregulation of glial function and drainage in the central and peripheral nervous system.[25] Murine models of glial and glymphatic system function following head injury has begun to shed light on the effect of brain trauma upon astrocytes and the effect upon intracranial edema and resolution.[26,27] Impaired functioning of the meningeal lymphatic vessels has been theorized to accelerate the accumulation of toxic amyloid beta protein in the brain parenchyma.[28]

Upledger illuminated and confirmed the existence of the function and physiology of the craniosacral system (meninges housing the brain, spinal cord, and cerebral spinal fluids).[29,30] Bodywork professionals are guided by the premise that structural compressions such as osseous and/or fascial restrictions can impede fluid movement and exchange, leading to an array of symptomology.[30–34] It is understood that vertebral bone compression into spinal cord space will create various neurological symptoms theorized, in part, by the obstruction of cerebral spinal fluid.[35,36] One animal model has demonstrated that blocking the normal physiologic cerebral spinal fluid drainage sites in the cribriform plate increases resting intracranial pressure.[37]

Manual therapies for the central nervous system

Craniosacral therapy

Manual therapies such as craniosacral therapy (CST) emerged from the science that explored the physiological motion of the central nervous system.[30,38,39] The core intent of CST is the theoretic interactions with connective tissue at osseous sites and, using a sustained low-force stretch, elicit a softening or relaxation response of soft tissues. In turn, a positive effect upon fluid exchange and the body's self-correcting actions can occur.[30] Studies have illuminated the natural motion of cranial bones, organ movement, and fluid exchange, depicting and measuring the dynamic nature of the physiology of the craniosacral system.[33,40,41] Based on observations of one animal study on the structural effects of applying CST, no discernible length difference was found at cranial bone sutures. However, this study concluded that perhaps a different biological basis for such treatment could be at play.[42] In a now classic study, changes in the length of the intracranial falx cerebri membrane were demonstrated with the application of CST on an embalmed cadaver skull. Recorded relative change in tissue length ranged between .28 and 1.44 mm at different sites of the membrane with different CST techniques applied. This study offers some validation that sustained, gentle, and external forces have a measurable effect upon the intracranial membrane system.[43]

Manual lymphatic drainage

Various methods of manual lymphatic drainage (LD) exist, but a contemporary version aligns with discoveries of anatomical pathways and lymph tissue mapping, the depths of lymph flow throughout the body, and using specific rhythms in fluid evacuation.[44] Hand pressure used in the Chikly method is extremely light, matching the tissue dynamics

unique to each patient, and only enough to stimulate the movement of fluid to enhance flow through specific lymph pathways. This is theorized to activate contractions of the functional unit of lymph vessels, called lymphangions, which assist fluid exchange without increasing blood filtration or lymph node collapse from too great of manually imposed pressure. There exists extensive innervation from the autonomic nervous system of these contractile units of the lymphatic system and the conceptualization of the movement of lymphatic fluids.[45,46] LD is believed to influence the whole-body system through gentle manual evacuation, creating a system-wide siphoning effect to enhance fluid exchange in multiple systems.[45]

The use of manual LD by qualified professionals is a treatment option for lymphedema, sports injuries, and fibromyalgia.[44,47–49] Manual LD was first suggested as a specific treatment of post-contusion and PCS for its anti-edematous effects.[50] The evolution and addition of glymphatic treatment methods have only recently emerged and are based on the practical application of the combination of methods such as CST, LD, and non-invasive intention of therapeutic touch.[16,45]

Existing studies on manual therapies for concussions and brain injuries

In clinical application, CST has been shown to have positive effect for a number of chronic syndromes that parallel PCS subtypes, but the body of data is limited to observational designs and low to moderate quality of randomized controlled methods.[51,52] The credibility of a sham control protocol for future study of CST was reported.[53] A recent study of former pro-football players with PCS showed statistically greater improvements in range of motion, pain, sleep, and cognitive function after a series of combined manual therapies.[54] A pilot study of 10 active military members had a reduction of symptoms of post-traumatic stress and head injury through the application of mixed light touch manual therapies.[55] A single blind case series measured clinical outcomes of soldiers with combat-related head injuries receiving CST and other manipulation techniques. Results showed statistically greater improvements in objective and subjective measurements pre- and post-treatment.[56] Case studies report positive outcomes of varied uses of manual therapies for the treatment of PCS.[57,58] One case of an athlete with PCS compared brain scans before and after 15 isolated CST treatments using quantitative electroencephalogram comparing symptom reduction to changes in neurological activity.[59]

Headaches

Headaches are a common symptom of concussion and PCS.[7,60] CST and other manual methods have been shown to have positive effect in the treatment of headaches, migraines, and trigeminal neuralgia.[61–64]

Sleep

Sleep has a critical function in ensuring metabolic homeostasis and clearance of metabolites and is but one important way for the nervous system to heal from trauma and injury.[13,18,65] The glymphatic system functions mainly during sleep and is largely disengaged during wakefulness. Sleep debt and a glymphatic system disruption have been proposed as a mediator of brain trauma, chronic traumatic encephalopathy, and other neurological disorders.[15,66,67]

Concussion symptom assessments

Subjective data —from patient

Self-reporting symptom scales are vital for assisting in the medical management of PCS. Several tracking tools hold substantial overlap with one another in capturing various pains and neurological infirmaries, functional impairment, and quality of life.[68–70] Although only some have been empirically studied, clinical users need to be aware that these scales have evolved rather than being developed scientifically.[71] Nonetheless, patient reports of subjective levels of pain, sensory, and psychological symptoms are crucial to aid in the clinical search for effective treatments.

Post-Concussion Symptom Checklist (PCSC)

The PCSC was utilized to track outcomes and re-administered periodically throughout the treatment process with care given to avoid coaching the patient on symptom manifestations.[70,72] Symptoms were also tracked through open-ended self-reporting by the patient at the onset of each session if he wished to share them.

Headache Impact Test™ (HIT-6)

The HIT-6 was designed to screen and monitor patients' severity of headache pain and the impact on function and quality of life.[73] Utilizing six items with a severity rating scale, the HIT-6 has been shown to be a reliable and valid tool for discriminating the effect of headache on daily living.[74,75] A possible point range of 36–78 reflects the severity of impact that headaches are interfering with daily quality of life and function. The HIT-6 was chosen for this particular patient to track headache symptoms which was also a chief complaint, second only to sleep disturbances.

Measurement of sleep

Sleep function was tracked through a sleep diary and the patient's perception of restfulness and daily energy.

Subjective data —from the clinician

Neurological screenings occurred each session per standard of care, including pupil and oculomotor status, balance

Persistent PCS Symptoms Self-Reported on Initial Intake
1. Headaches (HA) – though improved in frequency and intensity. Used to have 3-4 HA's per day at pain level 8-10. On admission to this clinic he reported 3-4 HA's per week at pain level 5-6.
2. Occasional shooting head pains; intermittent; no known reason or cause
3. Sinus trouble
4. Loss of memory (short term and immediate recall)
5. Mood challenges – depression, anxiety, helplessness, hopelessness, agitation, irritability by events, intolerant of social gatherings, constant nervousness (not present prior to injury)
6. Dizziness – used to be constant; now waxes and wanes in intensity
7. Loss of balance; still can't walk a straight line
8. Tinnitus – constant
9. Extreme hypersensitivity to light and sound; frequent overload from routine activities
10. Grating "noise" in neck
11. Tightness in neck and shoulders
12. Shortness of breath
13. Major sleep problems. Maybe gets 2-3 hours of sleep at the most, randomly. Cat naps all day and night. Exhausted from lack of sleep.
14. All symptoms worsen with increased physical activity.
15. Reported reaching a level of hopelessness and frustration after the lack of progress was verbally noted by one of his other therapists. He reported this exacerbated his and his family's stress levels and he expressed the potential of self-harm.

Figure 1. Post-concussion symptoms (18 month duration) reported on intake questionnaire by patient and in personal and parent interviews.

reactions, and general cognitive assessments conducted through observations of behavior. Palpation findings from full body assessment of musculoskeletal, fascia and fluid fields were conducted at the beginning of each treatment session. Inquiry about mood and stress were made at the beginning of each session.

Objective data

Quantifiable neurological soft sign and biomarkers not captured on the PCSC included fixed and dilated pupils, oxygen saturation, and frequency of urination and thirst.

Case report

The subject, a 24-year-old male, presented with an 18-month history of persistent neurological and behavioral symptoms reportedly related to one sports-induced concussion. Onset was in October 2017, following a head injury while playing in a collegiate-level sport. He reported being screened by an athletic trainer and having a follow-up medical assessment. No hospitalization was required. Prolonged rest was prescribed and adhered to for several months. An eventual referral to a local academic-medicine concussion clinic was made several months post-injury due to escalation of unresolved symptoms where psychological services addressing anxiety and depression and medications for pain and concentration were prescribed. Medications reported on patient intake included Prozac, Xanax, and Adderall. He reported his ability to focus on job tasks improved by the Adderall but the medications generally worsened "brain fog" and memory. He also participated in a physical therapy program for vestibular rehabilitation. Symptoms persisted to the point of full debilitation by sleep deprivation, increasing dizziness, sensory overload, and brain fatigue. Suicidal ideation prompted his parents to seek other recommendations and services.

In the interview with the patient and one of his parents, it was discovered that he sustained a total of six sports-related concussions dating back several years. History also included several orthopedic injuries long since healed (fractures to a foot and nose, and several bone contusions). The patient affirmed that a whiplash occurred with the most recent concussion and had originally sought chiropractic care for neck pain post-injury.

Since symptoms began to intensify, he was forced to take a medical leave from attending school and working a job that required much traveling. He stated he could perform job duties, enjoyed them, and felt capable of the job. However, the sensory intolerances created significant anxiety and began to reduce his confidence in his job performance. His tolerance and engagement in favorite activities were at 10% of normal participation level. He frequently isolated himself to cope with the sensory overload which added to a sense of helplessness.

Based upon client report and clinical observations, functional problems were noted on intake (see Figure 1). Initial clinical findings were also noted (see Figure 2).

Treatment methods employed included the following: (incorporated into each session as the need arose).

Manual LD techniques (Chikly).[45]

CST techniques (Upledger).[30]

Glial and Glymphatic system techniques (Wanveer).[16]

Initial clinical findings obtained through neurological screening and body palpation	
Visual	Fixed and dilated pupils; unreactive to light test Dizziness elicited with ocular motor end-range tracking
Autonomics	Initial oxygen saturation: 91% Shallow breaths (rest), ratchety lung expansion, tense body tone 18-month duration of significant sleep deprivation
Interstitium & Lymphatics	Palpation of neck, head & spine revealed 5/5 edema Lateral lymph chains of neck and posterior skull surface presented as swollen; assessed with light touch palpation Engorged occipital lymph nodes Stagnant lymph channels entire length of vertebral column and at neurovascular bundles along spinal nerve roots
Musculoskeletal	Displaced rib cage alignment with torsion towards left. Lacking full expansion of rib cage & tidal volume with forced inhalation on command. Middle ribs (bilaterally) immobile. Compressed frontal and parietal bones of cranial vault. Laterally displaced sphenoid and compression of spheno-basilar junction. Pain to touch at bilateral edges of atlas (C1 vertebra). Suspected atlas compromise. Sagittal suture ridge override palpated & possibly caused by the compressed frontal and parietal bones.

Figure 2. Initial clinical findings.

Goals for series of therapy encounters

- Reduce and eliminate stagnant and congested edema of spine, neck, and head.
- Soft tissue mobilization and fascial release methods to increase general soft tissue flexibility and subtleness through body to reduce sympathetic tone (through CST).
- Facilitate exchange of fluid through all body fluid systems; reduce interstitial congestion to promote healing and self-correction of nervous system (through LD).
- Neurological rehabilitation strategies to reduce and adapt to sensory challenges; environmental structuring. (These methods were not required due to the speed in which positive changes were experienced and observed with manual therapies.)
- Achieve freedom from pain and neurological dysfunction, restore/raise quality of life.
- Community and work re-entry, adaptations, and modifications assistance where needed.
- Patient and family education on the concepts of manual therapies.
- Client stated just one personal goal: "I just want to be able to sleep."

Treatment process

Treatment was conducted in isolation of any other (new) therapy over the course of three months. The patient participated in eleven, 1-h sessions. Scheduling sessions were recommended at 2–3 times per week initially. Following the first session, he requested daily treatment but was advised to allow his system to acclimate and integrate treatment effects. The first four sessions were scheduled 3 days apart. After the fourth session, scheduling was left to his discretion. Six more appointments were scheduled with 1 week spacing, and the eleventh session was as a follow-up at the patient's discretion 5 weeks later. Each session was an improvisational process of applying the various manual therapies and techniques, based upon whole-body assessment of primary and secondary structural and fluid findings.

Results and treatment outcomes

Quantitative data were collected through self-reporting of severity of symptoms using the PCSC, the HIT-6, and clinical observations. Sessions began most often with an invitation for him to report whatever symptom(s) felt most pressing. Quality of life measurements were self-reporting of sleep function, brain fog, ability to "get through the day," ability to tolerate sensory aspects of daily events, and endurance for mental and physical activities. All were recorded in the daily notes. A total of five recordings of the PCSC and three recordings of the HIT-6 were captured over the course of the treatment series (see Figure 3). Based upon both verbal responses from the patient as well as observation, some particular treatment techniques demonstrated a direct correlation to a reduction of a specific symptom (see Figure 4).

Post-Concussion Symptom Checklist Outcomes		
Point Score Range: 0-126 (higher numbers indicated greater severity of symptoms)		
Sessions of completion	**Score [Range]***	**Description of Severity Range**
Initial intake	81 – 92	Borderline Moderate – Severe
Before 4th session	63 – 68	Moderate
Before 6th session	41 – 47	Borderline Mild – Moderate
After 8th session	24 – 32	Mild
After 11th session	12 – 14	None – Mild (activity related)
*Score range influenced by duration of daily activities/events, sleep, and overload		
Headache Impact Test–6 ™ Outcomes		
Point Score Range: 36 – 78 (indicating severity that headaches impact daily quality of life)		
Initial intake	65	25% relief from initial HA level
Before 6th session	58.5	70% relief from initial HA level
After 11th session	44	90% relief from initial HA level

Figure 3. Self-reported changes of persistent concussion symptoms related to treatment intervention; measured through symptom tracking methods.

Specific techniques	Reported relief and observed changes
Cranial Base Release	Full body relaxation response, less head pressure, sensation of fluids draining out of head
Frontal bone lift	Less light sensitivity; reduced dizziness with tracking
Parietal bone lift; maxilla/palatine release	Less sound sensitivity
Sphenoid decompression	Normalization of fixed-dilated pupils
Glymphatic and Lymphatic Drainage	Full resolution of edema; sensation of feeling fluid drain inside head and down spine

Figure 4. Based upon patient's verbal responses and direct observation in treatment, several techniques had a direct correlation to a reduction of specific symptoms.

Sleep was restored following the first treatment as he reported sleeping 12 h each night between session one and two. Sleep continued to remain at normalized levels throughout the treatment process.

Sessions were not based upon following specific protocol sequence. A narrative summary of this improvisational and experiential treatment process is reflected in a summary of the daily notes (see Figure 5).

Six month follow-up

A 6-month follow-up (via telephone conversation) revealed sustained abatement of PCS symptoms and the patient resumed educational pursuits and gainful employment. His parents recommended that he continue to receive maintenance therapy but he voiced concerned over his mounting financial debt. He denied that symptoms were interfering with daily activities and quality of life. The patient's perspective about these therapies was highly positive.

Discussion

This article offers a hypothesis that persistent symptoms of PCS could be, in part, a result of compromised glymphatic and lymphatic pathway flow stemming from restrictions of movement and balanced position of osseous and soft tissue structures. The theoretical constructs of CST suggest when fascial membranes such as the dura and other meningeal layers and/or cranial plates and vertebral column may be in a compressive state, leading to neurological and/or behavioral symptoms.[30,32,76] The theoretical constructs of LD is that gentle, manual evacuation of lymphatic channels following anatomical mapping enhances a whole-body fluid exchange and removal of cellular wastes between interconnecting fluid systems.[45,49]

Only through whole-body palpation assessment was the first clinical discoveries made in this patient. Asymmetrical rib cage alignment, with sub-optimal breath expansion and lowered oxygen saturation, was subjectively assessed and CST techniques were immediately employed. The second discovery was persistent edema through anterior and lateral

Key:

Pt = Patient
HA = Headache
FL = Frontal bone Lift
TD = Temporal bone Decompression
MFR = Myofascial Release
SpC = Spinal cord
Sx = Symptoms
Tx = treatment, therapy
LD = Lymphatic Drainage (manual)
PL = Parietal bone Lift
CBR = Cranial Base Release
O2 Sat = Oxygen Saturation
PCSC = Post-Concussion Symptom Checklist

SESSION #1 Date: 02/16/2019

S: Full interview. Parent present. Extremely flat affect, listless body posture. Sx for 18 mos.

O: Fixed-dilated pupils. Palpation discovery: Edema-head, neck, along SpC; 5/5; not draining. LD techniques applied; immediate fluid exchange. Pt reported feeling fluid flowing–pressure inside head easing. Torsion thru entire rib cage–intercostal attachments. Ribs displaced anteriorly and torqued left. Soft tissue release techniques applied to all ribs/intercostal/spaces/articulations. O2 Sat: 91% prior to treatment; rose to 97% after rib tx. Pt reported great deal of release 'within head' following rib tx. Occipital lymph nodes engorged/swollen/thick. LD applied. Frontal bone compressed & tipped towards left, compressing left eye orbit. Parietal compression causing bony ridge at Right temporal suture. Pt. remembered to report increased frequency of urination – more than typical.

PCSC score: 81-92 Borderline Moderate to Severe Range **HIT-6 score: 65 of 36-78 range**

A: Pt reported immediate relieve of head pressure. Expressed joy and renewed hope of getting better

P: Treat 2-3 x's per week until sleep/structural physiology of CNS is restored. (Pt. requested daily tx)

SESSION #2 Date: 02/19/2019

S: Pt w/ brighter facial affect, smiling. Reported sleeping 12 straight hours both days after treatment. "Headache yesterday (pain scale 6 of 10); no HA today. Neck sore. Breathing felt very different. Long hx "light-headedness"…now gone. Re: meds: "I'm on 2 kinds of anti-anxiety and ADHD meds since last summer. They make me feel really groggy. The ADHD meds did help me get through the work day when I was working, but not helping light sensitivity or irritability."

O: All previous tx'd sites evaluated for further needs. Edema returned to base of neck/spine but now 2/5. Sites responded quickly with LD. Minimal intercostal treatment. FL and PL after CBR and thoracic diaphragm releases applied. Atlas palpations elicited repeated tenderness. Dural tissue lengthening and mobilization to facilitate improved CSF and lymphatic fluid flow. Initial O2 Sat: 97% ---rose and maintained at 99% during tx process.

A: Tolerating treatment well. Eager to return, expressed desire for daily treatment again.

P: Con't with tx plan

SESSION #3 Date: 02/21/2019

S: Improvements reported in all areas; describing now only 0-30% occurrence rate of sensitivity to light & sound; irritability; sleep disturbance; pounding pressure in head; dizziness. HA (3-4 of 10) now only every other day. LD being most helpful at reducing HA and head pressure.

O: Tx same areas. Rib cage now symmetrical on frame, no longer structurally distorted. Full lung expansion achieved & maintained. All ribs mobile. O2 Sat remains steady: 98-99%. Pupils still fixed and dilated. Tx sphenoid lesions directly today. Inferior strain left side bending distortion of sphenoid--tx. Pain intensified at atlas with sphenoid tx. Immediately following tx of all sphenoid physiological directions of movement, both eye pupils constricted within normal reactive range with light testing.

A: Recommend atlas chiropractor to evaluate state of atlas alignment. Local resource given. Optic pathway compressed by sphenoid but immediate positive change to pupils with tx

P: Con't with tx plan

SESSION #4 Date: 02/26/2019

S: First time patient drove to appointment (40 minutes). "Feeling really good. Can now walk backwards-and close eyes without escalating dizziness or loss of balance. This tx made that happen; no change w/ other tx. Better brain endurance; doing more activities without sx getting worse. Hoping to return to part-time work next week but worried about stress that job entails. Mild body aches and pains encountered this week. Increased neck pain with activity. Felt exhausted after attending basketball game as spectator. Sound of the buzzer hurt head. Stated spontaneously 'I do believe that I am getting better'."

Figure 5. (Continued)

O: Pupils fixed & dilated; reports persistent light sensitivity. Tolerated 30 mins computer work before eyes became over-stressed. Improved sleep with greater ease falling asleep. Reported a few days of waking up with headache, but cleared as day progressed. Pillow options discussed with goal for using one with adequate cervical support. Repeated atlas chiropractic recommendation. Tx sphenoid as inferior strain and side bending re-emerged. Tx for sutural flexibility and physiological balance. Minimal edema at base of head and cervicals; none down spine. Tx sites with LD, CBR brings immediate relief to head pressure. Discussed recollection and specifics of prior concussions including direction of forces that struck head. Treated soft tissue and osseous - fascial fields in all areas. Frontal and parietal bones now symmetrical in cranial vault - holding between tx. Temporal decompress needed today. Reported less 'sound pain' following this technique. End range ocular tracking did not set off dizziness.
A: Pt continues to do well with treatment process of multiple concussions.
PCSC score – 63-68 Moderate range of severity
P: Con't with tx plan.
SESSION #5 Date: 03/05/2019
S: Pt reports, "Sleeping alright. Eyes went blurry again this week despite prior visual clearing. Neck feels tighter. Light sensitivity ramped up. Wants to discontinue Adderall due to feeling miserable. This tx --the only thing helping me."
O: Ten-step protocol (CST) completed for entire CNS system of fascia and fluid pathways. Glymphatic drainage techniques applied at dura attachments to cranial bones and neck soft tissue. Bilateral orbits treated for fascial and myofascial attachments of ocular muscles. Eyes back to being dilated, but resumed normal size following sphenoid decompress and tx of inferior strain pattern. Excessive heat released from tissues during tx to bridge of nose, ethmoid, and cribriform plate. (Old injuries recalled – broken nose multiple times)
A: Pt reports sensation of "brain shaking" throughout session. No pain. "Sensations of relief/ease/calm."
P: Con't with tx plan.
SESSION #6 Date: 03/08/2019
S: Pt indicates desire for daily tx, stating, "I feel so much better." Depression is lifting. Less anxiety. Feel very hopeful /confident in recovery."
O: Eval of torso: ribs remain symmetrical, mobile, and flexible; WNL. Breath and lung/rib expansion WNL. O2 Sat remains 97-99%. MFR for maintenance of soft tissue physiology. FL, PL, temporal decompression techniques applied. Sphenoid decompression and balancing sutural mobility. No evidence of edema in lymphatic or venous system. No need for LD today, though glymphatic pumping technique applied following cranial vault tx to facilitate exchange of CNS fluids. Sphenoid decompression tx consistently restores pupils to normal size when dilated.
A: Clear and measurable evidence of improvement and effective clinical outcomes
PCSC score 41-47. Borderline mild-moderate range of severity. **HIT-6 score: 58.5 of 36-78 range**
P: Con't with tx plan. Weekly tx frequency -- is adequate.
SESSION #7 Date: 03/14/2019
Pt called clinic enroute to scheduled appointment having sudden onset of vertigo. Quite upset by this incident. Had to seek help to get home. Appointment rescheduled.
SESSION #8 Date: 03/19/2019
S: Patient under stress this week due to fight with parent over frustrations with longevity of situation. "My vision went 'wonky' when I got upset." Quit job due to their inability to accommodate environmental needs. Looking into finishing two online classes. Looking for easier job as well. Still having trouble falling asleep but routinely sleeping 8-10 hours each night once asleep. HA pain described as "not so bad." Slight lingering nausea today. Sound and light sensitivity ramped though lessening.
O: Intraoral / maxillary suture tx today. Maxilla fully compressed posteriorly and laterally right, completely immobile. Posterior compression had adverse effect upon occiput. Led to tissues indicating need for fascial tx over exterior surface of skull. Entire scalp fascia, lymph, blood vessels, etc. gently lengthened in sustained stretch against resistance to loosen adhesions around skull circumference.
A: Pt exclaimed delight in relief of head, eyes, and ears with today's session and effects of tx.
PCSC score 24-32 Mild severity for all symptoms. Scores obtained AFTER treatment concluded
P: Resume weekly treatments as schedules allow.

Figure 5. (Continued)

SESSION #9 Date: 03/21/2019
S: "Activity level raised to 50% of normal without sx; the other times sx worsen with increased activity but not as bad. Concentration and focus still impacted but also much less. Sx always nearly disappear for several days following each of our sessions but seem to ramp up toward end of week."
O: Con't to tx full-body fascia, interstitium, lymphatic pathway system as indicated by assessment. Vagal nerve pathways tx following multiple tracts from brain stem to innervation sites, including visceral organs. Vagal projections to digestive sphincters directly tx with report of immediate relaxation and anxiety elimination effect. Sphenoid decompression and intra-oral work at maxillary/vomer/spheno-basilar suture sites and attachments. Bilat palatines tx directly for sutural release. Ethmoid and cribriform plate tx. FL, PL,TD completed. Sustained PL brought immediate reduction in "brain pressure and foggy feeling." Glymphatic pumping technique immediately following for exchange of fluids.
A: Regular, weekly sessions are still required to assist and maintain structural and fascial changes in tissues of CNS. Vagal projections treatment was an additional bonus to positive effect on parasympathetic state.
P: Con't with weekly sessions.
SESSION #10 Date: 04/01/2019
S: "I could tell we skipped a week (due to schedule conflict) as some sx increased. But no light sensitivity (which is typically first symptom exacerbated when exhausted or stressed)."
O: Authentic & candid sharing of emotional state. Anxiety over current medical situation & living arrangements. Assurance given about expressing feelings. Lacking motivation, but currently working with relatives to "exercise brain" with some work activities. States a need to test sensory tolerances. Level of insights & ease with self-disclosure was impressive & believable. Education provided on difference between reactive, adjustment, other mood issues. He continues working with a counselor. Therapist offered consult w/ counselor at Pt's discretion. Full body assessment/tx of tissues revealing remnant restrictions, adhesions, immobility, tension. Internal tension to full length of tentorial membrane expressed. Extra time given to treat more thoroughly and completely. FL, PL, TD techniques. Intra oral structures and TMJ / mandibular attachment sites treated with full MFR. Facial and trigeminal nerve outlets and tracks treated.
A: Cranial nerve treatment directly revealed to Pt that headaches are related to nerve pathways. Therapeutic presence and holding space to share/express emotional struggles related to injuries. Suggested sharing frustrations with other trusted individuals. Pt. also has not contacted atlas chiropractor as he reports he doesn't feel he needs it at this point (neck pain resolved)
P: Continue weekly treatment to maximize full recovery of post-concussion sx by normalizing physiology of entire CNS. Support psycho-emotional healing process.
SESSION #11 Date: 05/10/2019
S: First session in five weeks due to schedule conflicts and Pt's spontaneous family vacation. Traveled for a week to celebrate completion of on-line college classes and his medical recovery. Played golf in bright sun; eyes felt strained but no exhaustion or overwhelming feeling. Entered casinos for short periods without negative effect. Tolerated both air flights. Slight headache during trip but tolerable. Vacation experience described with an animated, full-range affect, calm, upbeat demeanor.
O: Session included full body re-assessment. No edema present throughout entire system. Cranial base tightness, but easily released. Dura lengthening easily obtained. FL, PL, and TD released fully, rather easily. Remnants of inferior strain at sphenoid. Tx palatines, with immediate positive effect on spheno-basilar physiology. Zygoma release reduced structural pressure on bilateral temporals. Pupils WNL and reactive. No sound sensitivity reported. Nausea fully resolved. No dizziness with end-range eye tracking
A: Maxillary sutural releases in previous sessions elicited a breakthrough in improvements/reduction of symptom severity. Suture mobility has held. Normal physiology rhythm has been restored. Future assessments required to ensure maintenance of same.
PCSC score 12-14 Symptoms range from None to Mild range **HIT-6 score: 44 of 36-78 range**
P: Reduce appointment frequency to monthly….. or as-needed if sx increase.

Figure 5. Treatment Progression Depicted through Daily Notes. Abbreviated daily notes to reflect the full therapeutic process of applying CST and LD to treat symptoms of concussion. See KEY for term definitions.

cervical chains, over the expanse of scalp lymphatics, and engorged in bilateral occipital nodes at the cranial base. It is a reasonable assumption that edema may have occurred at the time of injury but after 18 months, edema and interstitial fluid stagnation would be expected to have resolved. That was not the case for this athlete. Palpation for (chronic) edema is a skill set learned through specific LD training methods.

Sleep was the first of the chronic symptoms to demonstrate an effect from treatment, after just one, 1-h treatment

session. It could be suggested that fluid stagnation in and around the central nervous system contributed to the adverse head pressure pain with impact on the lack of restorative sleep (as surmised by the patient's insights). It could be suggested that quality of breathing function may also have had a negative effect upon sleep. It is apparent through the stream of daily notes that the first treatment in and around the thoracic body and rib cage (consisting of fascia mobilization following CST methods) produced immediate elevation in oxygen saturation. It was also immediately apparent that LD through the central spinal lymph node confluences and pathways produced an immediate response of head pressure relief. Sleep resolved to satisfactory levels for the patient and remained so for the remainder of the treatment series, and at the 6-month follow-up.

Other neurological symptoms resolved at different, but steady rates, as reflected on the periodic scoring of the PCSC. To avoid or minimize effects of coaching or influencing the patient on symptom reporting, freely expressed symptom declarations were documented prior to the administration of repeated PCSC scoring. It is with a high rate of certainty that the PCSC yielded an accurate expression of the patient's self-reported experiences in symptom status through the treatment process.

Therapeutic touch and emotional support are aspects of most forms of manual therapies. It is possible that any placebo or autonomic calming effect of therapeutic touch contributed to the reduction of symptoms. However, resolution of certain chronic symptoms did correlate with the application of only specific treatment techniques (see Figure 4). Mood, cognitive function, and emotional well-being improved and the patient attributed this to reduction and elimination of head pain, brain fatigue, and sensory sensitivities. Quality of life improved and he returned to his occupational tasks of completing college and finding a less stressful, yet meaningful, job.

The literature supports interventions for PCS which include psychological, cervical, and vestibular rehabilitation.[5] The differential methods of CST and LD as distinct manual therapies have yet to be fully explored as options for PCS, though emerging studies indicate CST being used clinically with functional PCS subtypes. One contraindication for CST is not to apply during acute stages of brain injury when increased intracranial pressure is present.[30] Contraindications for LD have been cited and include acute infection or inflammatory disease process, thrombosis or phlebitis, acute hemorrhage, active malignant ailments, and acute heart problems as LD increases cardiac load.[44,45]

Future study could possibly isolate the impact CST and LD modalities have upon sleep in PCS. Research of the fluid exchange models of the nervous system could explore possible correlations to persistent symptoms of PCS. Furthermore, efficacy studies into therapies such as CST and LD might be aided by advancements of proving and measuring the exchange of fluids where glymphatic and lymphatic structures merge.

Conclusions

This case report reflects one collegiate athlete's unique constellation of PCS and the self-reported reduction of persistent symptoms through the experiential process of CST and LD intervention. There is little published evidence of the efficacy of CST and/or LD on the symptoms of PCS, though both have been utilized in clinics internationally for over 3 decades. Rest allowance and other therapy interventions had been previously trialed for 18 months, though no concussion symptom tracking was completed or available from previous records for comparative purposes. Previous pharmacologic treatment and vestibular rehabilitation for this patient were self-reported to have reduced symptoms of attention deficits and balance in climbing stairs, respectively. Other psychology and physical medicine endeavors had been trialed over the year and a half, and per his declaration, "Did not help heal me. Medications only helped me get through the work day; they did not take away the symptoms."

The author, an occupational therapist with 20 years of experience applying CST and LD in clinical practice, admits to a strong inherent bias. The strength of this case study, however, is that treatment was provided in isolation of any intervention other than his established medication usage. It could be said that placebo effect had been controlled for where specific treatment techniques clearly resolved some specific symptoms, but not all. Other symptoms resolved over time and may reflect effects of the restoration of sleep and elevation of mood. Though generalization of the findings in this case cannot be applied to the greater population of patients with concussions and brain injuries, this case does suggest that CST and LD are valuable treatment options for PCS and worthy of future study.

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References

1. Harmon KG, Clugston JR, Dec K, et al. American Medical Society for sports medicine position statement on concussion in sport. *Br J Sports Med* 2019; 53(4): 213–225.
2. Kenzie ES, Parks EL, Bigler ED, et al. Concussion as a multiscale complex system: an interdisciplinary synthesis of current knowledge. *Front Neurol* 2017; 8: 513.
3. Kamins J and Giza CC. Concussion—mild traumatic brain injury: recoverable injury with potential for serious sequelae. *Neurosurg Clin* 2016; 27(4): 441–452.
4. Gagnon I and Ptito A (eds) *Sports concussions: a complete guide to recovery and management*. Boca Raton, NJ: CRC Press, 2017.
5. McCrory P, Meeuwisse W, Dvorak J, et al. Consensus statement on concussion in sport—the 5th international conference on concussion in sport held in Berlin, October 2016. *Br J Sports Med* 2017; 51(11): 838–847.
6. Kontos AP, Deitrick JM and Reynolds E. Mental health implications and consequences following sport-related concussion. *Br J Sports Med* 2016; 50(3): 139–140.
7. Kontos AP, Sufrinko A, Sandel N, et al. Sport-related concussion clinical profiles: clinical characteristics, targeted treatments, and preliminary evidence. *Curr Sports Med Rep* 2019; 18(3): 82–92.
8. Lundblad M. A conceptual model for physical therapists treating athletes with protracted recovery following a concussion. *Int J Sports Phys Ther* 2017; 12(2): 286–296.
9. McCrea M, Guskiewicz K, Randolph C, et al. Incidence, clinical course, and predictors of prolonged recovery time following sport-related concussion in high school and college athletes. *J Int Neuropsychol Soc* 2013; 19(1): 22–33.
10. Brinker T, Stopa E, Morrison J, et al. A new look at cerebrospinal fluid circulation. *Fluids Barriers CNS* 2014; 11: 10–16.
11. Nedergaard M, Ransom B and Goldman SA. New roles for astrocytes: redefining the functional architecture of the brain. *Trends Neurosci* 2003; 26(10): 523–530.
12. Matsumae M, Sato O, Hirayama A, et al. Research into the physiology of cerebrospinal fluid reaches a new horizon: intimate exchange between cerebrospinal fluid and interstitial fluid may contribute to maintenance of homeostasis in the central nervous system. *Neurologia Medico-chirurgica* 2016; 56(7): 416–441.
13. Iliff JJ, Lee H, Yu M, et al. Brain-wide pathway for waste clearance captured by contrast-enhanced MRI. *J Clin Invest* 2013; 123(3): 1299–1309.
14. Plog BA and Nedergaard M. The glymphatic system in central nervous system health and disease: past, present, and future. *Ann Rev Pathol* 2018; 13: 379–394.
15. Jessen NA, Munk AS, Lundgaard I, et al. The glymphatic system: a beginner's guide. *Neurochem Res* 2015; 40(12): 2583–2599.
16. Wanveer T. *Brain stars: glia illuminating craniosacral therapy*. Ponte Vedra Beach, FL: Upledger Productions, 2015.
17. Louveau A, Plog BA, Antila S, et al. Understanding the functions and relationships of the glymphatic system and meningeal lymphatics. *J Clin Invest* 2017; 127(9): 3210–3219.
18. Iliff JJ, Wang M, Liao Y, et al. A paravascular pathway facilitates CSF flow through the brain parenchyma and the clearance of interstitial solutes, including amyloid β. *Sci Trans Med* 2012; 4(147): ra111.
19. Yang L, Kress BT, Weber HJ, et al. Evaluating glymphatic pathway function utilizing clinically relevant intrathecal infusion of CSF tracer. *J Trans Med* 2013; 11(1): 107.
20. Abbott NJ, Pizzo ME, Preston JE, et al. The role of brain barriers in fluid movement in the CNS: is there a 'glymphatic' system? *Acta Neuropathol* 2018; 135(3): 387–407.
21. Louveau A, Smirnov I, Keyes TJ, et al. Structural and functional features of central nervous system lymphatic vessels. *Nature* 2015; 523(7560): 337–341.
22. Shaw G. New study suggests brain is connected to the lymphatic system: what the discovery could mean for neurology. *Neurol Today* 2015; 15(13): 1–9.
23. Eide PK, Vatnehol SA, Emblem KE, et al. Magnetic resonance imaging provides evidence of glymphatic drainage from human brain to cervical lymph nodes. *Sci Report* 2018; 8(1): 71949.
24. Aspelund A, Antila S, Proulx ST, et al. A dural lymphatic vascular system that drains brain interstitial fluid and macromolecules. *J Experim Med* 2015; 212(7): 991–999.
25. Ji RR, Berta T and Nedergaard M. Glia and pain: is chronic pain a gliopathy? *Pain* 2013; 154(Suppl. 1): S10–S28.
26. Plog BA, Dashnaw ML, Hitomi E, et al. Biomarkers of traumatic injury are transported from brain to blood via the glymphatic system. *J Neurosci* 2015; 35(2): 518–526.
27. Perez-Polo JR, Rea HC, Johnson KM, et al. Inflammatory consequences in a rodent model of mild traumatic brain injury. *J Neurotrauma* 2013; 30(9): 727–740.
28. DaMesquita S, Fu Z and Kipnis J. The meningeal lymphatic system: a new player in neurophysiology. *Neuron* 2018; 100(2): 375–388.
29. Breman GM and Kratz SV. Scientific evidence supporting craniosacral therapy. In: Breman GM and Kratz SV (eds) *A touch better: two therapists' journey and the lessons they learned from Dr. John E. Upledger about craniosacral therapy*. Palm Beach Gardens, FL: UI Publications, 2019, pp. 199–231.
30. Upledger JE and Vredevoogd JD. *Craniosacral therapy*. Seattle, WA: Eastland Press, 1983, pp. 77–87.
31. Moonen G, Satkunendrarajah K, Wilcox JT, et al. A new acute impact-compression lumbar spinal cord injury model in the rodent. *J Neurotrauma* 2016; 33(3): 278–289.
32. Whedon JM and Glassey D. Cerebrospinal fluid stasis and its clinical significance. *Altern Ther Health Med* 2009; 15(3): 54–60.
33. Moskalenko YE, Frymann V, Weinstein GB, et al. Slow rhythmic oscillations within the human cranium: phenomenology, origin, and informational significance. *Human Physiol* 2001; 27(2): 171–178.
34. Greenman PE and McPartland JM. Cranial findings and iatrogenesis from craniosacral manipulation in patients with traumatic brain syndrome. *J Am Osteopath Assoc* 1995; 95(3): 182–8191.
35. Ropper AE and Ropper AH. Acute spinal cord compression. *New Engl J Med* 2017; 376(14): 1358–1369.
36. Saadoun S, Werndle MC, Lopez de Heredia L, et al. The dura causes spinal cord compression after spinal cord injury. *Br J Neurosurg* 2016; 30(5): 582–584.

37. Mollanji R, Bozanovic-Sosic R, Zakharov A, et al. Blocking cerebrospinal fluid absorption through the cribriform plate increases resting intracranial pressure. *Am J Physiol Regul Integr Comp Physiol* 2002; 282(6): R1593–R1599.
38. Davis CM (ed.) *Complementary therapies in rehabilitation: evidence for efficacy in therapy, prevention, and wellness*. Thorofare, NJ: SLACK Incorporated, 2009.
39. Chaitow L. *Cranial manipulation: theory and practice: osseous and soft tissue approaches*. New York: Elsevier, 2005.
40. Oleski SL, Smith GH and Crow WT. Radiographic evidence of cranial bone mobility. *Cranio* 2002; 20(1): 34–38.
41. Greitz D, Franck A and Nordell B. On the pulsatile nature of intracranial and spinal CSF-circulation demonstrated by MR imaging. *Acta Radiol* 1993; 34(4): 321–328.
42. Downey PA, Barbano T, Kapur-Wadhwa R, et al. Craniosacral therapy: the effects of cranial manipulation on intracranial pressure and cranial bone movement. *J Orthop Sports Phys Ther* 2006; 36(11): 845–853.
43. Kostopoulos DC and Keramidas G. Changes in elongation of falx cerebri during craniosacral therapy techniques applied on the skull of an embalmed cadaver. *Cranio* 1992; 10(1): 9–12.
44. Chikly BJ. Manual techniques addressing the lymphatic system: origins and development. *J Am Osteopath Assoc* 2005; 105(10): 457–464.
45. Chikly B. *Silent waves: theory and practice of lymph drainage therapy: an osteopathic lymphatic technique*. Scottsdale, AZ: IHH Publications, 2004.
46. Rahbar E and Moore JE Jr. A model of a radially expanding and contracting lymphangion. *J Biomech* 2011; 44(6): 1001–1007.
47. Crisóstomo RS and Armada-da-Silva PA. Manual lymphatic drainage in the treatment of chronic venous disease. *Clin Phys Therapy* 2017; 143: 901.
48. Majewski-Schrage T and Snyder K. The effectiveness of manual lymphatic drainage in patients with orthopedic injuries. *J Sport Rehabil* 2016; 25(1): 91–97.
49. Vairo GL, Miller SJ, McBrier NM, et al. Systematic review of efficacy for manual lymphatic drainage techniques in sports medicine and rehabilitation: an evidence-based practice approach. *J Man Manip Ther* 2009; 17(3): e80–e89.
50. Trettin H. Craniocerebral trauma caused by sports. *Z Lymphol* 1993; 17(2): 36–40.
51. Ernst E. Craniosacral therapy: a systematic review of the clinical evidence. *Focus Alternat Complement Therap* 2012; 17(4): 197–201.
52. Jäkel A and von Hauenschild P. A systematic review to evaluate the clinical benefits of craniosacral therapy. *Complement Ther Med* 2012; 20(6): 456–465.
53. Haller H, Ostermann T, Lauche R, et al. Credibility of a comparative sham control intervention for craniosacral therapy in patients with chronic neck pain. *Complement Ther Med* 2014; 22(6): 1053–1059.
54. Wetzler G, Roland M, Fryer-Dietz S, et al. Craniosacral therapy and visceral manipulation: a new treatment intervention for concussion recovery. *Medical Acupuncture* 2017; 29(4): 239–248.
55. Davis L, Hanson B and Gilliam S. Pilot study of the effects of mixed light touch manual therapies on active duty soldiers with chronic post-traumatic stress disorder and injury to the head. *J Bodyw Mov Ther* 2016; 20(1): 42–51.
56. Shitikov T, Shayrin O and Danilko L. A role of complementary medicine in rehabilitation of military traumatic brain injuries. *Pain Med* 2018; 3(2/1): 8.
57. Guernsey DT 3rd, Leder A and Yao S. Resolution of concussion symptoms after osteopathic manipulative treatment: a case report. *J Am Osteopath Assoc* 2016; 116(3): e13–e17.
58. Haller H, Cramer H, Werner M, et al. Treating the sequelae of postoperative meningioma and traumatic brain injury: a case of implementation of craniosacral therapy in integrative inpatient care. *J Altern Complement Med* 2015; 21(2): 110–112.
59. Rice LL. Upledger institute case study CranioSacral therapy —traumatic brain injuries, 2017, https://www.iahe.com/docs/articles/craniosacral-therapy——traumatic-brain-injuries–tbi-.pdf
60. Makdissi M, Schneider KJ, Feddermann-Demont N, et al. Approach to investigation and treatment of persistent symptoms of sport-related concussion: a systematic review. *Br J Sports Med* 2017; 51(12): 958–968.
61. Seffinger MA and Tang MY. Manual craniosacral therapy may reduce symptoms of migraine headache. *J Am Osteopathic Assoc* 2017; 117(1): 59.
62. Rao K and Khatri S. Effectiveness of craniosacral therapy in cervicogenic headache. *MOJ Yoga Physical Ther* 2017; 2(4): 00031.
63. Kratz SV. Manual therapies reduce pain associated with trigeminal neuralgia. *J Pain* 2016; 1(1): 5.
64. Facó SG, Farias R, de Souza NS, et al. Manual therapy in the treatment of primary headaches. *Revista Pesquisa Em Fisioterapia* 2016; 6(3): 341–352.
65. Eugene AR and Masiak J. The neuroprotective aspects of sleep. *Medtube Sci* 2015; 3(1): 35–40.
66. Sullan MJ, Asken BM, Jaffee MS, et al. Glymphatic system disruption as a mediator of brain trauma and chronic traumatic encephalopathy. *Neurosci Biobehav Rev* 2018; 84: 316–324.
67. Xie L, Kang H, Xu Q, et al. Sleep drives metabolite clearance from the adult brain. *Science* 2013; 342(6156): 373–377.
68. McLeod TC and Leach C. Psychometric properties of self-report concussion scales and checklists. *J Athl Train* 2012; 47(2): 221–223.
69. Randolph C, Millis S, Barr WB, et al. Concussion symptom inventory: an empirically derived scale for monitoring resolution of symptoms following sport-related concussion. *Arch Clin Neuropsychol* 2009; 24(3): 219–229.
70. Lovell MR, Iverson GL, Collins MW, et al. Measurement of symptoms following sports-related concussion: reliability and normative data for post-concussion scale. *Appl Neuropsychol* 2006; 13(3): 166–174.
71. Alla S, Sullivan SJ, Hale L, et al. Self-report scales/checklists for the measurement of concussion symptoms: a systematic review. *Br J Sports Med* 2009; 43(Suppl. 1): i3–i12.
72. Post-Concussion Symptom Checklist, 2017, https://www.education.ne.gov/wp-content/uploads/2017/07/Post-Concussion_Symptom_Checklist.pdf
73. Kosinski M, Bayliss MS, Bjorner JB, et al. A six-item short-form survey for measuring headache impact: the HIT-6™. *Qual Life Res* 2003; 12(8): 963–974.
74. Yang M, Rendas-Baum R, Varon SF, et al. Validation of the headache impact test (HIT-6™) across episodic & chronic migraine. *Cephalalgia* 2011; 31(3): 357–367.
75. Kawata AK, Coeytaux RR, Devellis RF, et al. Psychometric properties of the HIT-6 among patients in a headache-specialty practice. *Headache* 2005; 45(6): 638–643.
76. Frymann VM. A study of the rhythmic motions of the living cranium. *J Am Osteopath Assoc* 1971; 70(9): 928–945.