


The University of Kansas Health System Outpatient Clinical Concussion Comprehensive Protocol: An Interdisciplinary Approach

Health Services Insights
Volume 15: 1–18
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DOI: 10.1177/11786329221114759



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ABSTRACT

OBJECTIVE: The concussion team at The University of Kansas Health System outpatient rehabilitation spine center is comprised of experienced multi-disciplinary experts including physical therapists and a speech language pathologist. The team set forth with a purpose of creating and organizing an internal physical therapy clinical recommendation protocol for initial evaluations and subsequent treatments for the concussed patient. The aim of this paper is to share these recommendation protocols with other therapy teams and provide a foundational layout for treating the patient with post-concussion symptoms in an outpatient physical therapy clinical setting.

STUDY DESIGN: Clinical recommendation protocol provides guidance for patients ages 10+ from initial evaluation through discharge with emphasis on evidence-based research in the areas of: oculomotor, cervical, vestibular, post-concussion migraine influence, mood disorders (such as anxiety and depression), exertion, and cognitive communicative dysfunction.

RESULTS: Finding a written, comprehensive clinical resource protocol for post-concussion outpatient evaluation(s) and treatment strategies can be difficult. This document serves as a resource for other outpatient concussion rehabilitation clinics, providing rationale, and objective measurement tools, for assessing and treating concussion patients. To the authors' knowledge, no other research has produced a practical, efficient evaluation tool to be utilized at bed side, condensing evidence-based research into an easy-to-use form.

CONCLUSION: The University of Kansas Health System outpatient concussion rehabilitation center developed clinical recommendation protocols for concussion care. The intent was to standardize assessment and treatment for concussion patients and to share these objective measurement tools and procedures, focused on a team approach of concussion providers, as a clinical outline for both the novice and seasoned clinician specializing in the field of concussion work in an outpatient rehabilitation setting.

KEYWORDS: Concussion, mTBI

RECEIVED: December 30, 2021. **ACCEPTED:** June 30, 2022.

TYPE: Original Research

FUNDING: The author(s) received no financial support for the research, authorship, and/or publication of this article.

DECLARATION OF CONFLICTING INTERESTS: The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Background and Purpose

The concussion team at The University of Kansas Health System (TUKHS) outpatient rehabilitation spine center is comprised of experienced multidisciplinary therapists including specialized physical therapists and a speech language pathologist. This team treats adult, geriatric, and pediatric populations above the age of 10, as well as patients who sustained concussions from a variety of mechanisms, and with varying timelines of injury. In order to standardize and streamline concussion care, the team set forth with a purpose of creating and organizing an internal physical therapy clinical recommendation protocol(s) for initial evaluations and subsequent treatments (Figures 1–4) for the concussed patient. The aim of this paper is to share that information and provide guidance from initial evaluation through discharge,

with emphasis on evidence-based research in the areas of: oculomotor, cervical, vestibular, post-concussion migraine influence, mood disorders (such as anxiety and depression), exertion, and cognitive communicative dysfunction.¹ This paper offers foundational concepts of concussion care, while providing a clinical outline for both the novice and seasoned clinician specializing in the field of concussion work in an outpatient rehabilitation setting.

Introduction

A concussion is a type of traumatic brain injury (TBI) caused by a bump, blow, jolt to the head, or hit to the body that causes the brain to move rapidly back and forth. This sudden motion can cause the brain to bounce or twist within the skull, creating chemical changes in the brain and possibly damage to brain



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DATE: _____ PATIENT: _____

PMH: Cervical Surgeries: _____ Ocular dysfunction: _____

Anxiety/Depression: _____ Migraines: _____
 Glasses: Y N Bifocal: Y N Prisms: Y N
 Prior orthostatic BP: _____ Prior BPPV: _____
 ADHD: Y N Learning Difficulty: Y N No. of prior Concussions: _____
 Hearing Loss: Y N Imaging: MRI _____
 Tinnitus: Y N CT scan _____ X-ray _____
 Meclizine: Y N Audiology Screen: _____

Sleep Aids: Y N _____ Anti-Depressant(s): Y N _____
 Stimulants: Y N _____ Other Medications: _____

History/ Mechanism of Injury: _____ Date of incident: _____
 Current Concussion Sxs: Dizziness Y N Headache Y N Nausea Y N Foggy Y N
 Other symptoms: _____

Oculomotor/Vestibular Exam:

Saccadic Dysfunction: Y N Horizontal Vertical Diagonal
 ADEM on follow-up: Horizontal sec: _____ Vertical sec: _____ Ratio: _____

Smooth Pursuit Dysfunction: Y N Horizontal Vertical

Convergence: Blurry _____ cm. NPC (near point): _____ cm. (from forehead position)

Accommodation: R _____ cm. L _____ cm. (from cheek position)

Optokinetic Issues: Y N Horizontal Vertical

VOR dysfunction: Y N Horizontal Vertical

VOR cancellation: Y N Horizontal Vertical

Head Thrust: Y N Side: R L (*peripheral test)

Static Visual Acuity: # line _____ ETDRS chart- 4 feet away from wall

DVA (dynamic visual acuity): Horizontal: _____ line(s) change
 Vertical: _____ lines(s) change (* 2 lines or less = normal)

Vision Motion Sensitivity (VMS): Y _____ N _____

Cover/Uncover Test:
 Look at COVERED eye: R or L exophoria esophoria hypophoria hyperphoria
 Look at the UNCOVERED eye: R or L exotropia esotropia hypotropia hypertropia

Randot testing for Stereopsis: _____ deg. of arc (30 degrees of arc-normal)

Dix-Hallpike: R side: upbeat downbeat torsion normal _____ canal/cupulolithiasis
 L side: upbeat downbeat torsion normal _____ canal/cupulolithiasis

Supine Roll Test: R side: geotropic ageotropic
 L side: geotropic ageotropic

Cervical Exam:

Cervical flexion: _____ deg. Extension: _____ deg.
 R SB: _____ deg. L SB: _____ deg.
 R rotation: _____ deg. L rotation: _____ deg.

Vertebral artery dysfunction: Y N R L _____

(Figure 1 Continued)

Alar Ligament dysfunction: Y N R L _____
 Sharp Purser (transverse ligament): Y N R L _____

Deep Neck Flexor Endurance Test: _____ sec.

Cervical Flexion- Rotation Test (CFRT): R Rotation _____ deg L Rotation _____ deg

Smooth Pursuit Neck Torsion (SPNT): R _____ L _____

Head Neck Differentiation Test: Vestibular - Y N Cervical - Y N

Joint Position Error Test: _____

Balance Exam/Vestibular:

mCTSIB (modified clinical test of sensory integration of balance)

Eyes open: _____ sec Eyes closed: _____ sec
 Foam EO: _____ sec Foam EC: _____ sec

Sharpened Romberg: Eyes open: _____ sec. Eyes closed: _____ sec.

Single Leg Stance: R _____ sec. L _____ sec.

FGA: _____/30 Device: Y N Cane Walker Other: _____

Cobalt/ Bertec: _____

Outcome Measures:

DHI: _____ HIT-6: _____ NDI: _____ MIDAS: _____ HADAS: _____

Exertion Test: Stationary Bike _____**Driving Protocol:** BITS/ BIONESS: Reaction Time: _____**Figure 1.** University of Kansas Health Systems outpatient concussion – evaluation.**Objective Measurements**

1) Oculomotor/Vestibular: Detailed descriptions for testing/training are included. Various web based video recordings, not affiliated with our clinic, may be available for further visual assist/learning but are beyond the scope of this paper.

A. Saccades: test at 18 inches from the patient's face; patient looks between two fixed targets: center to 30 degrees to the right, center to 30 degrees to the left. This test is repeated with vertical targets: center to 30 degrees up and center to 30 degrees down. Repeat 10 times each. Include diagonal pattern on both sides to assess saccadic dysfunction in multiplane level. ***Hypometric or hypermetric saccades, decreased ability to increase velocity and/or increase symptoms are indicative of a positive test. Hypermetric saccades are usually indicative of cerebellar involvement.***

B. Smooth Pursuits: test at 18 inches from patient's face, the therapist moves finger / pen from center to 30 degrees right and 30 degrees left at a speed of 60 degrees per second. Repeat 10 times vertical- may use "H" pattern with testing- and repeat 10 times horizontally. ***Saccadic intrusion and/or increased symptoms are indicative of a positive test.***

C. Convergence : Use pen tip or tongue depressor with a 12 pt. font letter. Make sure the patient is wearing corrective lenses, if needed. Starting at arm's length, the therapist brings the target towards his/her nose until he/she sees the target double. Therapist should observe adduction of the two eyes as the object moves closer with the inability of the two eyes to stay adducted when the near point convergence is reached. Patient will report diplopia. Therapist measures the distance between the object and the bridge of the nose. The convergence norms are between 6 to 10 cm. ***A convergence of more than 10 cm is indicative of a convergence insufficiency.***

D. Accommodation: Accommodation is tested in both eyes, with one eye covered during the test with an eye occluder. A tongue depressor with a column of 8 letters, 5 pt. font size, is moved from the patient's cheek outward. The distance from the first clearly seen target by the patient to the cheek is measured in inches and converted to diopters. Normative data exists for age groups. The patient's results are analyzed by comparing values with age norms to determine if an accommodation impairment is present. Measurements above two diopter levels of age norms indicate

there is an impairment in visual acuity and clarity is an issue. Diopter levels below the age norms indicate the patient has excellent accommodation for their age. Diopters are a unit of measurement in the optical field; +1 diopter will focus on approximately 1 meter of visual field. ***A different measurement between the 2 eyes or an increase of more than 2 diopters compared to age norms can be indicative of accommodative insufficiency.***

E. Optokinetic: Patient is sitting looking at a ribbon with a repeating vertical pattern at eye level. While the patient focuses on the ribbon, the examining therapist quickly moves the ribbon horizontally to the right for 5 repetitions. The test is then repeated horizontally to the left, vertically up and vertically down. ***A lack or decrease in nystagmus is indicative of a positive test.***

F. VOR: To test VOR in the yaw plane, bring the patient's head to 30 degrees of flexion. Rotate passively the patient's head at 120 BPM pace for 10 repetitions. Patient should keep their focus on the therapist's nose. Repeat the test in the pitch plane. ***Inability to stabilize the eyes on the nose or increase symptoms is indicative of a positive test.***

G. VOR cancellation: To test VOR cancellation in the yaw plane, bring the patient's head to 30 degrees of flexion. Ask the patient to keep fixation of their eyes on the therapist's nose, while the therapist moves in sync with the patient's rotation at a pace of 60 bpm for 10 repetitions. Repeat this test in the pitch plane. ***Observation of saccadic intrusion or increased symptoms is indicative of a positive test.***

H. Head Thrust/ Head Impulse Test: This test can be very helpful with differential diagnosis to determine chronic peripheral vestibular disorder, as well as determining the side of a hypofunctioning labyrinth. It assesses the angular vestibulo-ocular reflex, specifically testing the horizontal semicircular canal and superior vestibular nerve function. At TUKHS, the patient is seated and is asked to fixate the eyes on a target, i.e. the clinician's nose. The examiner rotates the head rapidly, to each side, looking for a corrective or compensatory saccade. Individuals with normal vestibular function should not have a corrective saccade.

I. Dynamic Visual Acuity (DVA) non-instrumented: Using an ETDRS (Early Treatment of Diabetic Retinopathy Study) eye chart, the patient stands at the correct distance from the wall/chart (TUKHS clinic= 4 feet). Ask the patient to read the eye chart until the lowest line recognizable or until they are unable to identify all the letters on a given line. Bring the patient's head into 30 degrees of flexion and rotate the patient's head in the yaw plane at 120 BPM. While maintaining the rotation, ask the patient to read the eye chart and document how many line(s) above the baseline were observed. Repeat this test in the pitch plane. ***Greater than 2 lines deficit is indicative of a positive test.*** Instrumented testing can also be performed on the BERTEC BVA device.

J. Visual Motion Sensitivity (VMS): In standing with the feet shoulder width apart, the patient turns their head, eyes, and trunk 80 degrees to the right and left at 50 BPM – perform for 5 repetitions. ***Increased symptoms are an indication of a positive test.***

K. Benign Paroxysmal Positional Vertigo: If therapist suspect that dizziness is caused by BPPV,

the Dix-Hallpike and the Supine Roll Test are performed.

L. Ocular Alignment Testing

1. Tropias – Using an eye occluder, the examining therapist perform the single cover test. The first eye is covered for approximately 1-2 seconds. As this eye is covered, the uncovered eye is observed for any shift in fixation. The eye occluder is removed and any refixation movements are noted under binocular conditions. If the unoccluded eye shifts in, or medially, in a temporal to nasal fashion when the opposite eye is occluded – this indicates that there is an exotropia. If the unoccluded eye shifts out, or laterally, in a nasal to temporal fashion when the opposite eye is occluded – this indicates that there is an esotropia. If the unoccluded eye shifts down when the opposite eye is occluded – this indicates that there is a hypertropia present. If the unoccluded eye shifts up when the opposite eye is occluded – this indicates that there is a hypotropia condition. Reference: [eyerounds.org @ https://webeye.ophth.uiowa.edu](https://webeye.ophth.uiowa.edu)

2. Phorias – The cover-uncover test is performed to determine if there is a phoria present, which is a latent strabismus or misalignment that is only present when binocular fusion is suspended or interrupted. This test is performed in the same manner as the single cover test except that attention is turned to the eye that has been covered, as the eye occluder is removed during testing. If the uncovered eye does not show a fixation shift as the eye occluder is placed, but as the eye occluder is removed, the covered eye shows a refixation movement once binocular conditions are restored is called a phoria. If there is an outward, or lateral, refixation in the nasal to temporal direction – this represents an eso deviation. If there is an inward, or medial, refixation in a temporal to nasal fashion – this represents an exo deviation. If there is a vertical refixation movement – this represents either a hyper or hypo deviation. Maddox Rod testing can also be performed. Reference: [eyerounds.org @ https://webeye.ophth.uiowa.edu](https://webeye.ophth.uiowa.edu)

3. Randot Stereo Test: Test is aimed for both adult/ pediatric population in testing for stereognosis, the ability to binocularly discern a difference in the distance from the observer of a static object(s). Binocularly arranged random dot patterns ensure accuracy as the patient will need to identify a form without the help of using solely monocular vision. Adults are asked to identify six geometric shapes during testing, while an animal identification section is present for the pediatric population. The test helps test depth perception, as well as stereo vision. Adult testing ranges from 400 to 20 deg. of arc, while pediatric testing involves 400 to 100 deg. of arc. The patient is asked to wear the darkened, specialized glasses that are included in the kit. The suppression check assists in determining if visual dominance is present, or if suppression of one eye is present. Per Stereo Optical (copyright) testing, 40 deg of arc is baseline in testing. Testing results higher than 40 deg of arc warrant an evaluation by a neuro-optometrist. Reference: www.precision-vision.com

2) Cervical : Screen for unilateral headaches, whiplash injury and/or presence of cervical pain or stiffness

A. Cervical AROM: flexion, extension, right rotation, left rotation, right side bending and left side bending.

B. Vertebral Artery: Patient is in sitting position with chin resting in the palm of their hands and elbows on knees (cervical extension); patients asked to turn the head to the right and count backward from 10 to 0. This test is repeated to the left. The therapist is assessing for signs of 5 D's: drop attack, diplopia, dysphagia, dizziness, dysarthria, and 3 N's: Nausea, Nystagmus, Numbness. Any signs represent a positive test that warrants a call to the referring physician for further evaluation.

C. Alar ligament stability: The rotation stress test is regarded as primarily stressing the contralateral alar ligament in accordance with the bio-mechanical description of Dvorak et al. The test is described for both sitting and supine positions. The axis is stabilized around its laminae and spinous process using a lumbrical grip. The cranium is grasped with a wide hand span and rotated, the occiput taking the atlas segment with it, to the end of available range. No lateral flexion is permitted. Some rotation will occur during the test, but the extent of rotation within the bounds of normal is subject to some variation. Estimates of the range of normal rotation vary between 20 and 40 degrees. As with the side-bending test, the test is repeated in 3 positions in the sagittal plane, with laxity in all 3 positions necessary to establish a positive test finding. However, more recently Osmotherly, Rivett and Rowe suggested that the range of craniocervical rotation during rotation stress testing of intact alar ligaments should typically be 21 degrees or less. Reference: Physiopedia@www.physio-pedia.com

D. Sharp Purser (transverse ligament stability): The patient is seated. The examiner places the palm of one hand on the patient's forehead, and the index finger or thumb of the other hand on the tip of the spinous process of the axis (C2). The patient is asked to slowly flex the head performing a slight cervical nod, at the same time the examiner presses posteriorly on the patient's forehead. A sliding motion of the head in relation to the axis indicates atlantoaxial instability [1] A positive result may also be accompanied by a reduction in symptoms [3], a "clunk" sensation, or patient reports of a "click" or "clunk" felt in the roof of their mouth. It is thought that this technique reduces atlantoaxial subluxation caused by forward flexion of an unstable cervical spine. Reference: Physiopedia@www.physio-pedia.com

E. Deep neck flexor endurance test: To assess the endurance of the deep neck flexors (Rectus Capitis Anterior, Rectus Capitis Lateralis, Longus Capitis, Longus Colli - "Muscle specificity in tests of cervical flexor muscle performance"). Test Position: supine, patient in hook lying position. Patient performs chin tuck and lifts head off the table 1 inch. The examiner looks for substitution of the platysma or SCM muscle.

Normal Values: Men: 38.9 seconds, Women: 29.4 seconds ("The Deep Neck Flexor Endurance Test: normative data scores in healthy adults").
Importance of the Test: Those with neck pain were found to have significantly decreased deep neck flexor endurance, average of 21.4 seconds ("Reliability of a measurement of neck flexor muscle endurance"). They tend to over-utilize other muscles (platysma, hyoid muscles, and especially the sternocleidomastoid) for postural maintenance, which leads to the commonly seen position of forward head postures-a position we commonly see in those who use computers frequently or engage in sedentary activity on a regular basis. Include assessment of a patient's posture and segmental mobility in the cervical spine. Reference: <https://www.thestudentphysicaltherapist.com>

F. The Cervical Flexion-Rotation Test (CFRT), in contrast to other forms of manual examination, is an easily applied clinical test purportedly biased to assess dysfunction at the C1-C2 motion segment. The C1-C2 motion segment accounts for 50% of the rotation in the cervical spine. In this test procedure, the cervical spine is fully flexed to isolate movement to C1-C2, which has a unique ability to rotate in flexion. Normal range of rotation motion in end range flexion has been shown to be 44° to each side. In contrast, subjects suffering from headache with C1-C2 dysfunction have an average of 17° less rotation. Patient is relaxed in supine. Examiner fully flexes the cervical spine with the occiput resting against the examiner's abdomen. The patient's head is then rotated to the left and the right. If a firm resistance is encountered, pain provoked, and range is limited before the expected end range, then the test is considered positive, with a presumptive diagnosis of limited rotation of C1 on C2. Reference: Physiopedia@www.physio-pedia.com

G. Cervicogenic Dizziness/Cervical kinesthetic awareness

1. Smooth Pursuits Neck Torsion Test: This test involves comparison of smooth pursuit performance in a neutral head position with performance in a neck torsion position, 45° degrees left and 45° right of neutral. To achieved neck torsion, ask the patient to position their body at 45 degrees from neutral and position their head in a neutral position. Saccadic intrusion is indicative of a positive test and the cervical spine would be the origin of dizziness.
2. Head Neck Differentiation: The patient is seated in a swivel chair. In the first part of the test, the patient is "swiveled" in the chair for 10- 20 seconds. In the second part of the test, the therapist gently but firmly "holds" the patient's head in neutral, while asking the patient to rotate their body side to side (another colleague may assist with the motion). Resting time between part 1 and part 2 is necessary. Symptoms of dizziness with part one is vestibular in nature; part two suggests cervicogenic.
3. Joint Position Error test: The patient sits in front of a target at a distance of 50 cm with a laser positioned on their forehead. The starting point of the laser is in the center of the bullseye. The patient performs head motions with the eyes closed and tries to reproduce his neutral head position. This is repeated 3 times for each head movement: R rotation, L rotation, Head up, head down. Positive test is indicated when less than 2 trials out of 3 is not within the established target (at TUKHS= within the white circle).

3) Balance/ Vestibular:

- A. mCTSIB
- B. Sharpened Romberg
- C. Single Leg Stance
- D. FGA
- E. Bertec/ Cobalt

4) Exertion: University of Kansas Health System- Stationary bike protocol

5) Reaction Time and Divided Attention on the Bioness: follow set protocol

Figure 2. University of Kansas Health Systems outpatient concussion – special tests.

Treatment Strategies

1) Oculomotor:

- A. Saccades - A to A cards – eye only movements and progress to eye head movements; www.eyecanlearn.com - Bug jump, numbers saccades; 4 panel saccades, Hart chart, Dynavision; Marsden ball
- B. Smooth pursuits – tracking card, www.eyecanlearn.com; pong/super breakout, ball toss, Marsden ball – pendulum; apps – focus builder; mirror for busy background, tennis ball toss; laser light tracking
- C. Convergence – Brock Beads, convergence fusion pictures, pencil push with and without jumps, barrel convergence cards
- D. Accommodation – binocular/monocular poems, Hart Chart, Brock Beads with far target
- E. Optokinetic – ribbon, optokinetic video, you tube train videos
- F. VOR – gaze stabilization x 1, gaze stabilization x 2 – progress 120-150 BPM as appropriate; plain to busy backgrounds; reading an eye chart
- G. VOR cancellation – 60 BPM, sitting/ standing positions, progression to busy background
- H. DVA- gaze stabilization
- I. Motion sensitivity – you tube videos (London at night, drive at night); disco ball, mirror

2) Cervical:

- A. Stretching-stretching tight musculature in cervical flexion, extension, side-bending, rotation, AROM, AAROM, PROM with towel
- B. Strengthening – isometrics, TheraBand- resistive in multiple planes, upper cervical flexion strengthening – sitting, supine; upper cervical extension–in prone and all 4s; ball exercises- on wall
- C. Joint Position exercise - patient sits in front of bullseye and repeats testing for re-training. Exercise is repeated 3 times for each head motion – Right rotation, Left rotation, upwards motion from neutral; downwards from neutral.
- D. Manual therapy- joint mobilizations, traction: manual/ mechanical, deep trigger point massage, occipital release, mid-cervical manipulation

3) Balance:

- A. vestibular spinal reflex (VSR) exercises - progression firm to compliant surface; comfortable stance, Romberg, semi-tandem, tandem; modify surface to close cell to open cell, rocker board, vestibular/vision exercises; progress to ambulation forward and backward.

Figure 3. University of Kansas Health Systems outpatient concussion – treatment.

Therapist's name:

Date:

Visit Number:

Patient's Name:

Date of Birth:

Date of Injury:

Mechanism of Injury:

Total number of concussions:

Maximum Heart Rate: $(220 - \text{age} \times 80\%) =$

Do not perform the test if systolic BP is greater than 160 or diastolic BP is greater than 100

Stop the test if:

1. Pt cannot keep up with the pace of 60 RPM
2. Pt is exhausted and cannot continue
3. HR greater than age predicted heart rate
4. BP greater than 200/115
5. RPE/BORG 17/20
6. Symptoms increase 3 points or more; if symptoms increase 1 point- check all vitals
7. Pt completes 20 minutes/resistance level 21

Minutes	Resistance	HR	RPE/BORG (6-20)	Symptoms (0-10)	BP (every 5 minutes)
Rest (5 mins)	N/A		N/A		
0-1	1				
1-2	2				
2-3	3				
3-4	4				
4-5	5				BP:
5-6	6				
6-7	7				
7-8	8				
8-9	9				
9-10	10				BP:
10-11	11				

11-12	12				
12-13	13				
13-14	14				
14-15	15				BP:
15-16	16				
16-17	17				
17-18	18				
18-19	19				
19-20	20				BP:
Cool Down (2 minutes)					BP:
Immediately after stopping	N/A	N/A	N/A		N/A
Rest (5 minutes)	N/A		N/A		BP:

Reason for terminating the test: cannot keep up with pace, exhaustion, HR, BP, RPE/BORG, symptoms, 20 minutes/level 2: _____

Treatment: stationary bike at an intensity of 80% of the threshold HR achieved during the test once per day for 5-6 days per week for 20 minutes; patients should be advised to terminate the exercises at the first sign of symptom exacerbation.

Treatment Progression: test can be repeated every 2 -3 weeks to establish a new target HR or increase HR 5 BPM every two weeks; progress to treadmill and elliptical as able.

Figure 4. University of Kansas Health Systems outpatient concussion – exertion protocol.

cells. Approximately 3 million patients each year seek medical assistance for a TBI in the US, with a large number classified as a mild TBI (mTBI), also referred to as a concussion.¹ The terms concussion and mild traumatic brain injury are often used interchangeably; however, they are not entirely synonymous, as concussions are not always mild.

Concussion rehabilitation is an ever-developing and changing field as more evidence-based research arises.² The depth and complexity of a concussion requires involvement of multiple medical disciplines,²⁻⁶ all working as an interdisciplinary team to treat the whole person. Ongoing and consistent communication between providers across a spectrum of specialties including neurology, rehabilitation services, neuro-optometry, neuro-ophthalmology, neuropsychiatry, and neuropsychology, is essential, especially as an individual may present with a multitude of symptoms.²⁻⁶ Within the last decade, many articles have been published regarding therapy for the concussed patient, including: vestibular retraining, vision rehabilitation, cervical management, exertional training, and cognitive integration.¹ It

can be a daunting task for the therapist or team to determine the best tests, objective outcomes measurements, and optimal treatment strategies, especially as patients often have pre-existing conditions, and varying individual presentations. The aim of this paper is to provide a thorough, but easily outlined, clinical protocol to guide evaluations (Figures 1, 2, and 4) and treatment (Figure 3) of concussion patients in the 7 largest contributing categories: oculomotor, cervical, vestibular, post-concussion migraine, mood disorders (including anxiety and depression), exertion, and cognitive communicative deficits.¹

By utilizing a “team approach” to concussion rehabilitation care, assessment and treatment will provide a more well-rounded provider perspective and will offer more efficient and effective care for both the patient and care team. Obtaining a comprehensive past medical history intake and subjective portion of the evaluation (Figure 1) is crucial, in not only determining areas of dysfunction, but to capitalize on understanding potential deficits and identifying opportunities for referring concussed patients for speech therapy, neuropsychology, neuro-optometry,

and audiology screenings, as needed.^{1,7-9} These therapies and treatments can address specific areas of deficits or concern. Speech therapy can provide insight on memory, attention, and cognition/communicative dysfunction that affects pragmatic and social skills, such as reading and writing.¹⁰ Neuro-optometry and ophthalmology can provide visual assistance when traditional clinical vision retraining strategies are unsuccessful.^{6,7} A referral to the neuropsychology is advantageous when the individual is limited in therapy recovery due to pre-existing or current concussion mood disorders, such as anxiety and depression.⁹ Audiology referrals can be beneficial if continued balance, hearing loss, or tinnitus symptoms linger.

Clinical Intake and Initial Therapy Evaluation: Concussion Injury and Medical History

At the initial therapy assessment (Figure 1), the therapist should thoroughly review the patient's comprehensive medical history. Our clinical recommendation guidelines encompass pediatric patients above the age of 10, adults, and geriatric patient populations. Examples of pertinent history include: cervical surgeries and musculoskeletal history as it relates to balance dysfunction; history of mood disorders, including anxiety and depression; use of visual aids, such as corrective lenses, prisms, or bifocals; prior orthostatic hypotension issues, specifically any history of difficulties with rapid movement change; diagnosed learning difficulties, such as attention deficit hyperactivity disorder (ADHD); and ocular issues, such as amblyopia, childhood strabismus or Lasix correction.^{1,2,4} In addition, subjective examination should include questioning on personal or family history of migraines, as well as motion sensitivity. A thorough review of the concussion injury, specifically the mechanism of injury, is important for symptoms relating to benign paroxysmal positional vertigo (BPPV). BPPV can be a secondary complication to the injury and the patient may require modified treatment strategies.^{1,2,8} Proper screening can ensure that the patient did not acquire vertigo as a result of a traumatic mTBI incident.

Pharmacology

In addition to medical history, a detailed review of the patient's current pharmacology status is critical.² Medication(s) can modify a patient's normal response, including inhibition or activation of responses, and, therefore, should be reviewed during the initial visit (Figure 1). One example of a pertinent medication(s) includes meclizine. Meclizine is an antihistamine that reduces the amount of inflammation and histamine in the body.¹¹ Meclizine acts as a vestibular suppressant and is used to treat and prevent nausea, vomiting, and dizziness caused by motion sickness.¹² Meclizine can pass into the central nervous system and can diminish the excitability of the labyrinth and peripheral vestibular systems.¹¹ The most common side effect of the drug is drowsiness. Meclizine takes approximately 1 hour to stabilize within the body and show effects, especially for treatment of motion sensitivity, and can

last for 12 to 24 hours.¹¹ Studies have indicated that meclizine timing can reach peak effect at 9 hours post ingestion, so careful subjective questioning on duration of dosage is vital.¹³ If meclizine is active in the body, the therapist needs to ensure that the initial evaluation is outside this window to provide a thorough and accurate screen for BPPV testing. Ideally, the patient would be contacted before the initial evaluation regarding withholding meclizine for 1 to 2 days prior to the visit. If the patient arrives having taken the medication within a 24-hour window, detailed findings are recorded and repeat testing is included in follow-up visit(s), without active meclizine present, to ensure the validity of the results.

Post-concussion patients can sustain a multitude of varying symptoms and may require a prolonged recovery. Many patients may experience subsequent sleep impairments due to the concussion.^{2,14-16} Sleep aids, both over-the-counter and prescription, such as melatonin, anti-depressants, and non-benzodiazepine hypnotics may be beneficial to provide sleep.² There are currently no FDA approved/standard pharmacologic medications for mTBI and individualized care is essential.^{2,17} A specific antidepressant, amitriptyline, has shown some success in treating post-traumatic headaches and the sedative effects from the medication can be beneficial for those struggling with sleep impairments.^{2,18} Cognitive and neuropsychiatric symptoms are very common following a mild traumatic brain injury and the most studied pharmacological aid(s) in the literature are stimulants (eg, methylphenidate). Methylphenidate has been known to treat both patients with traumatic brain injury, as well as clinical attention deficit disorders.¹⁷ The usage of methylphenidate has shown benefit in treating attention and processing speed deficits.^{2,18} The prevalence of disorders of mood following a TBI can range between 20% and 50% and may remain even when the neurologic function has returned.¹⁹ Selective serotonin reuptake inhibitors (SSRI) block the reuptake of serotonin in the presynaptic cells, thus leading to increased activity of serotonin in the synaptic cleft.¹⁹ SSRIs are often provided as an initial treatment regimen for a multitude of neurocognitive and neuropsychiatric disorders.¹⁷ Serotonin affects mood, alertness, emotion regulation and working memory and provides a potential long-term pharmacological intervention.¹⁹ However, literature on antidepressant use for cognition has shown no benefit on cognitive recovery and may actually worsen cognitive symptoms.¹⁹ Beta-blockers, calcium channel blockers, and gabapentin are all potential medical interventions utilized to assist with headache management.¹⁸ Identifying and utilizing key medications across the spectrum can assist the patient with coping skills with everyday activities. Education regarding expectations of medication management, used in conjunction with pacing strategies, is reviewed by the neurologist.

Mood disorders

A prior or existing personal and/or family history of mood disorders or attention deficit and hyperactivity disorder (ADHD)

may increase the risk of prolonged concussion symptoms. Dosage and medication scheduling of ADHD medications can also play a role in the success of therapy intervention.^{2,20,21} These medications may have interactions with the over-the-counter medications and supplements as well. Reviewing current anti-depressant and anti-anxiety medications with the patient would be insightful with mood management during therapy intervention (Figure 1). Communication amongst the team can involve discussion of the degree that mood disorders and anxiety will play a role into recovery and assist in handling any new or exacerbated symptoms that may affect the rehabilitation potential.^{1,2,9,15}

At the TUKHS outpatient rehabilitation center, team focus on open communication with vision specialists, physical therapists, speech language pathologists, neuropsychologists, and the referring physician is commonplace. Mood disorder documentation is placed in a precaution section of the electronic medical record note. A monthly in-person or zoom conference is held between the neurologist, speech pathologist, and physical therapist(s) involved with individual patient care to openly discuss patient progress. Patients exhibiting issues with mood and anxiety history in physical therapy intervention will be discussed with all members of the interdisciplinary team in order to be cognizant of any possible limitations for that patient in achieving a full progression.^{9,15} The neurologist will make the referral to involve neuropsychology as soon as warranted. Speech language pathologists and physical therapists also meet ad hoc to discuss individualized patient treatment plans to incorporate strategies for mood disorders, cognitive communicative deficits, and visual-spatial-perceptual activity delays in overall progression of care.

Clinical Assessment

The concussion therapists at TUKHS rehabilitation spine center compiled assessment and treatment protocols as part of developing their clinical concussion recommendation protocols. These clinical protocols are shared in Figures 2 to 4 and are discussed in the paragraphs below. Figure 2 focuses on specific testing of concussion patients. Figure 3 lists patient treatment strategies to assist the clinician in patient care. Figure 4 outlines the TUKHS concussion exertional protocol.

Oculomotor

Vision dysfunction and poor eye teaming can develop following an mTBI.^{1,2,6,7,12,22-27} An eye exam (Figure 1) is paramount as literature suggests that visual impairments delay mTBI recovery.^{1,7,23} For example, in an article by Master et al,²³ 69% of children have at least one vision abnormality after an mTBI; 51% present with accommodative insufficiency; 49% have some level of convergence insufficiency, and 29% possess saccadic deficits. In addition to the visual deficits noted by Master et al another contributing factor in children is visually induced dizziness, which requires specific training

in exposure to optokinetic stimuli.²⁸ Further, Tannen et al⁷ reported that 30% of the post-concussion subjects screened in their neuro-optometry practice, ages ranged from 14 to 44 years, have an esophoria.

The Vestibular/Ocular Motor Screening (VOMS) tool is used to screen for vestibular-ocular symptoms in post-concussion patients.^{1,6,12,22,24} At TUKHS, an incorporation of the VOMS (Figure 1) for horizontal and vertical saccades are used, with an expansion of testing to include: diagonal testing; smooth pursuits in yaw and pitch planes; near point convergence; vestibular-ocular reflex (VOR) cancellation testing; visual motion sensitivity; and active VOR testing with the subject focusing on a stationary object in both the horizontal and vertical planes. The VOMS is included into each patient's initial evaluation to assist the therapist in determining the root cause of exacerbated symptoms. In addition, assessments can be performed for accommodative testing of visual clarity, screening for phorias and tropias, stereopsis tests, dynamic visual acuity (DVA), and VOR cancellation tests. Concussed patients are assessed on initial evaluation and subsequent visits using the visual analog scale (VAS), which asks the patients to rank the severity of the following symptoms on a scale from 0 to 10: dizziness, headache, fogginess, and nausea.

Saccades and smooth pursuit. Saccades are the ability of an individual to move the eyes quickly between 2 targets. For example, reading a book or looking at varying objects in the visual environment reflect saccadic movement. Research has indicated that many concussion patients that do not receive early treatment with oculomotor integration will struggle with reading and functional skills long after the concussion injury has resolved.⁶ Functional impairments such as computer work, reading comprehension, word recognition, and generalized reading capabilities may be misattributed to a cognitive deficit instead of an oculomotor dysfunction.⁶ A useful clinical tool (Figure 1) for the assessment of horizontal and vertical saccades is the developmental eye movement test for adults (ADEM) and for pediatric patients (DEM).²⁶ While the ADEM and DEM have not been tested and validated as concussion assessments,²⁶ the results of the testing, including reaction times, can assist the clinician in determining the fluidity of saccadic eye movement.

Smooth pursuit is the ability of the patient's eyes to follow a moving target. Smooth pursuit is vital for overall eye quickness and adaptation with varying object movement. For example, watching a butterfly or vehicle in motion reflects smooth pursuit eye movement. Visual delays and dizziness can present with both saccadic and smooth pursuit delays in concussion (Figure 1). In conjunction with the saccadic testing, a smooth pursuit dysfunction indicates a central vestibular injury.

Convergence. Convergence is the simultaneous inward movement of both eyes toward each other when trying to maintain binocular vision when viewing an object. For example, focusing

on a specific word in a sentence while reading. One area of growing research in both the sports-related and generalized post-concussion population is convergence insufficiency.^{1,2,6,7,22-26} Research suggests that normalized convergence contributes to reading ability, depth perception, and visual awareness.^{2,25} Ciuffreda et al²⁹ described how post-concussion patients that underwent vision-based training reported a reduction in symptoms and improved reading ability by 90%. Thiagarajan et al³⁰ reported improvements with targeted vision rehabilitation in the areas of convergence, accommodation, and saccadic re-training. Convergence training involves holding a tongue depressor with size 12 font lettering in front of the patient at a distance to the end of a ruler (Figures 1 and 2). A ruler is placed on the forehead, between the eyebrows, to assist with measurement in centimeters (cm). If convergence delays are present, the patient will exhibit poor eye movement adaptation (diplopia) or 1 eye will drift away from symmetrical binocular viewing, predominantly in an abduction position.²⁴ This is clinically known as near point convergence (NPC), and research has shown benefits of treating NPC with specific, targeted vision therapy in concussion rehabilitation.²² Two studies have reported that the meaningful detectable change (MDC) for near point convergence is 4cm improvement from the initial evaluation distance.^{12,24} A pilot study indicates that convergence may be normalized following 6 weeks of combined office and home-based vision training.²⁵ Convergence insufficiency that is latent to recover may require recommendations to a neuro-optometrist or ophthalmologist, especially with complex diplopia and simultaneous ocular health disorders.^{2,6}

Accommodation. Accommodation is the process by which the eye changes optical power to maintain a clear image, or focus, on an object at varying distances. Accommodation testing (Figure 2) allows the clinical specialist to determine the patient's clarity with letters at varying distances (Figure 1). Master et al²³ completed a study on 100 adolescents that sustained a concussion. Of the participants, 51% had an accommodative disorder, and 22% of the children had both accommodative and convergence deficits. Ciuffreda et al²⁹ demonstrated that accommodation, vergence, strabismus, and cranial nerve palsy can occur after an mTBI, and 90% of the patients improved to near baseline with reports of elevated reading ability. Optimal training occurs in patients below age 40, as presbyopia occurs naturally with the natural aging process.²⁹ At TUKHS outpatient rehabilitation center, beneficial treatment strategies include using a HART chart, binocular and monocular poems, and brock beads for eye integration (Figure 3).

Optokinetic reflex. Motion sensitivity is a common symptom seen in the concussion population. Patients often describe issues with the inability to be a passenger in a moving vehicle or experience newfound difficulty in large, busy environments.² Subjective questioning may indicate that they may have experienced motion sensitivity prior to sustaining their

concussion injury, sometimes even from early childhood. Addressing motion sensitivity is not only essential for daily living and transportation, but also useful for symptom management, especially, as there is an established link between migraines and motion sensitivity.³¹ The traditional testing for motion sensitivity has been a kinetic drum. At TUKHS, optokinetic (OKN) testing (Figures 1 and 2) can be performed with a uniformly stripped ribbon. An informational handout on obtaining an OKN mobile app is included in Figure 3 for technological ease and efficiency in completing the home exercise program (Figure 3).

VOR and VOR cancellation. The VOR is responsible for keeping a stationary object image on the fovea, with head motion in both the yaw and pitch planes.² Ocular muscles assist in keeping the visual image clear²⁴ by initiating ocular movements in proportion, but in opposite direction, of the head motion.² During clinical testing (Figures 1 and 2) it is essential to maintain neck flexion at 30 degrees from neutral head position to facilitate the horizontal (lateral) canals (Figure 2). Gaze stabilization is initiated after the cervical dysfunction is resolved, if applicable. Differential diagnosis between cervical or vestibular issues is required to determine the root cause of headache, dizziness, and other concussion symptoms.³² Neck-related pathology may mimic vestibular symptoms clinically.³² A multitude of symptoms may arise if either the vestibular or ocular system lags, as it should be a 1:1 ratio. If the VOR is impaired,² visual blurring, dizziness, poor visual focus, and oscillopsia may occur during head motion. An abnormal VOR cancellation test (Figure 2) should suggest that underlying central vestibular pathology is present; positive findings include saccadic intrusions or eye tracking delays with testing.

Head impulse/head thrust test. The head impulse test (HIT), or head thrust test, is a vestibular test (Figures 1 and 2) used to help identify an impaired VOR in patients with vertigo, particularly those with acute peripheral vestibulopathy.^{4,33} With testing, a positive finding is a "catch-up" saccade to locate the target at the end of the movement. The HIT is useful in evaluating patients with acute, spontaneous dizziness and is usually negative for central vestibular lesions.³³ Head thrust testing (Figure 2) can be included in the concussion exam to determine if there has been pre-existing dysfunction.

Dynamic visual acuity. Dynamic visual acuity (DVA) is an objective test for the VOR system in both yaw and pitch planes. The measurements are recorded on the evaluation (Figure 1) and objective findings greater than a 2-line deficit indicate a positive test. The Early Treatment of Diabetic Retinopathy Study (ETDRS) eye chart is utilized in testing (Figure 2). Positive findings include elevated symptoms, such as headache, dizziness, nausea, fogginess, and positive testing indicates a peripheral vestibular abnormality.

Vision motion sensitivity. Inhibiting vestibular-induced motor input allows testing of vision in absence of certain external stimuli. This VMS testing assesses the patient's visual motion sensitivity. VMS can present as a sense of disorientation, disequilibrium, dizziness, and postural imbalance when the vision and visual system are in conflict.² Visual imbalance stems from an inability to maintain balance with an overwhelming visual input, replicating issues the patient experiences with busy crowds and grocery stores.² VMS testing is included in the VOMS screening, providing the clinician with a quick assessment for central pathology (Figures 1 and 2).

Phoria/tropia/stereopsis. Advanced visual screening (Figures 1 and 2) involves testing for phorias and tropias, the 2 primary types of ocular deviations. Tropia is when a misalignment occurs when the patient is using binocular vision. Phorias, also a misalignment, can result when the binocular visual field is broken, typically with the occlusion of 1 eye. Traditional clinical reasoning suggests that many mTBI patients will manifest eye coordination dysfunction, an exophoria, in addition to convergence insufficiency.²³ In a retrospective study of neuro-optometric private practice patients, Tannen et al⁷ found that nearly 30% of patients with a diagnosis of concussion exhibited esophoria. These findings reveal that esophoria is more prevalent in concussion patients than originally believed. Stereopsis is the ability of both eyes to see an object clearly and create a perception of depth. Dysfunction in stereopsis results in limited depth perception, which can result in issues with driving, pouring a drink, stair negotiation, and multiple daily activities. In research by Leshno et al³⁴ stereoacuity was measured by using a Randot Stereo Test. In a retrospective chart review of patients ranging in ages from 6 to 17 years old, normal stereoacuity was defined as equal or less than 40 arcsec; subnormal was 50 to 400 arcsec, and >400 arcsec was listed as poor. Furthermore, the study found a correlation between convergence insufficiency and below normal ranges in stereopsis.

Cervical

One of the most important aspects of concussion is the involvement of the cervical region, either in direct or indirect injury, due to mechanisms of injury such as car crashes and sports related activities.^{4,35-38} Cervical pain can occur due to the disruption of upper cervical dorsal root afferents from cervical proprioceptors through the vestibular nucleus.^{4,39} It has been suggested that this disruption results in poor functioning of head and neck orientation in space.^{4,39} Some of the most common cervical-related symptoms are headaches, dizziness, and neck pain.^{5,38-40} Often in cases of concussion, those involving whiplash associated disorders (WAD), a patient will present with cervical pain that requires in-depth differential diagnostic testing.¹ Cervicogenic dizziness^{1,4,5,39} is a relatively common post-concussive symptom that may have underlying cervical

etiology. Joint position sense testing (Figures 1 and 2), or cervical relocation test, is the ability to relocate the natural head alignment without the assistance of vision, to help ascertain the overall cervical proprioception.^{5,41,42} In addition, Treleven³⁹ discovered that individuals with chronic WAD and dizziness had greater errors with joint position sense than those not complaining of dizziness. Results suggest that the symptoms may be due to a greater abnormal cervical afferent input system. Cervicogenic dizziness can result from many sources, including peripheral and central vestibular pathology, vestibular migraine involvement, autonomic dysfunction changes, post whiplash injury, inflammatory, degenerative, or mechanical dysfunctions of the cervical spine.^{1,4} The multitude of findings may suggest focusing on neck motion, the flexion-rotation test,⁴³ palpation of upper cervical vertebrae and segmental assessment to determine if the neck is contributing to persistent post-concussion symptoms.³⁸

Reiley et al⁴ detected a correlation between cervical pain, stiffness, and a lack of sensorimotor proprioception with regards to head righting. Kennedy et al³⁸ reported that the study's participants were 7.5 weeks post-concussion and 90% were considered to have a neck pathology contributing to their current symptoms. About 85% subjectively mentioned having frequent neck pain, with moderate-to-severe pain from the occipital to C4 segmental regions. It is proposed that upper cervical dysfunction propagates post-concussion headaches due to cervical immobility.³⁸ In a study by Marshall et al⁴⁰ a connection between the C2 and C3 dorsal root ganglia and the nociceptive afferents of the trigeminal sensory nucleus suggests a referral to the upper cranium and forehead, imitating a headache. Due to these findings, the physical therapist needs to perform an initial evaluation of active neck range of motion in all planes to assess cervical mobility and address areas of stiffness.⁴ In conjunction with cervical range of motion, studies have found that weakness in the anterior neck flexors, specifically the rectus capitus anterior, rectus capitus lateralis, longus capitus, and longus colli, result in neck pain due to the absence of muscle strength and endurance.³⁷ Various research has shown that the longus colli and capitus weakness is common with WAD or cervicogenic headaches.³⁷ To compensate, patients tend to over-utilize other muscles, such as the platysma, hyoid, and, most commonly, the sternocleidomastoid, to maintain erect postural alignment. Today, the frequent use of electronics and computer-based occupations tends to lead to forward head-round shoulder posture.^{37,44}

In addition to flexion stability, research shows that the lack of superficial and global extensor muscle control also contributes to chronic neck pain.³⁷ The extensor group, specifically the splenius capitis, semispinalis capitis, semispinalis cervicis, and multifidus, play a profound role in cervical stabilization and control. In the study by Parazza et al³⁷ there was a significant level of higher extensor endurance than that of the deep flexor muscles.

To improve overall immobility in the cervical spine, traditional upper cervical joint mobilizations and manual therapy with exercise have shown strong evidence for management of cervicogenic headache.^{38,45} Within the TUKHS interdisciplinary concussion rehabilitation team, cervical care in addition to vestibular management, is provided in order to manage neck-related symptoms. As with any trauma, clearance of any cervical injury or fractures must be considered. Imaging via X-ray or MRI would help detect bone abnormalities or soft tissue injury. The importance of checking arterial blood flow, specifically if any issues involving the vertebral artery and stabilizing ligaments are present, is key. The vertebral artery passes through the foramen of the first 6⁶ vertebral foramen bilaterally and gives rise to the basilar artery posteriorly. A defect or dysfunction with impingement may cause hemorrhaging or ischemic attacks in the brain tissue. The alar ligament stability is important as it originates on the dens of the second cervical vertebrae, the axis, and inserts on the medial side of the occiput. The function of the alar ligament is to provide stability from the skull to the axis region, stabilizing lateral side to side movements with head motion and limiting cervical rotation. The transverse ligament is a major protector and stabilizer of the upper cervical region, as it attaches the ring of the atlas to the odontoid process. This broad ligament attaches along the basilar aspect of the occiput and inserts along the posterior surface of the axis. Specialized ligamentous and arterial examinations are commonplace at TUKHS concussion rehabilitation clinic for comprehensive screenings, to ensure that cervical stability is maintained.

Vestibular

The vestibular system is the primary mechanism involved in balance, postural control, and visual spatial orientation.^{2,6} Kontos et al⁶ describes a wide variety of patient symptoms with vestibular pathology: dizziness, nausea, vertigo, visual delays, disequilibrium, and difficulty with busy environments and motion sensitivity. Given the location of the vestibular system in relation to the head and cervical region, translated forces associated with a concussion can directly impact the vestibular system and brain centers that regulate balance. The vestibular system can be divided into 2 components: peripheral and central. The peripheral component consists of semicircular canals, otoliths, vestibular ganglia, vestibular apparatus, and the vestibular nerve. In the central component, the vestibular nuclei, cerebellum, autonomic nervous system, thalamus, and cerebral cortex all play a role.³² In the VOR reflex described earlier, the peripheral vestibular system needs to match the visual system input by a 1:1 ratio or the patient may complain of unsteadiness and dizziness due to the sensory mismatch. The integration of vestibular, vision, and somatosensory systems is paramount for balance.²⁴ The vestibulo-spinal reflex (VSR) assists with postural stability when sensory input is initiated in the vestibular apparatus, semicircular canals, and otoliths of the inner ear.^{2,6} Endolymph

movement in the semicircular canals causes stimulation of the vestibular nerve in the vestibular nucleus and activates cranial nerve VIII excitation in the brainstem region. Motor and sensory input from the semicircular canals and sensory otoliths assist in static and dynamic balance by providing motor input to skeletal muscles below the cervical region through the medial and lateral vestibulospinal tracts.³² When the patient moves their head to one side, the involved side has elevated extensor activity, while flexor activity is recruited on the opposite side limb(s) to maintain balance. In the sports population, vestibular dysfunction can be as common as 50% to 80% of athletes experiencing dizziness.⁶ In addition, vestibular and ocular dysfunction may lead to visual motion sensitivity.⁶

One of the most clinically used outpatient outcome measurements to determine level of vestibular input regarding balance is the modified clinical testing of sensory integration on balance (mCTSIB). Through the various conditions (Figure 1), the clinician can test the vestibular, vision, and underlying somatosensory components to aid the clinician in distinguishing the root impairment(s) of balance dysfunction. Among the most common for the SRC (sports related concussion) population is the Balance Error Scoring System (BESS).^{6,46} In the acute phase of SRC, BESS has a sensitivity of 34% and specificity 91% to 96% with regards to the differential diagnosis of concussion in athletes.¹ However, the BESS test is intended as an acute measure, and is less sensitive to deficits after the third day of injury.¹ At TUKHS, computerized posturography is performed using a flat, force plate platform with the Bertec Portable Essential balance system,⁴⁷ incorporating a version of the mCTSIB through COBALT testing specifically designed for concussed athletes under the age of 25.

At TUKHS, the Functional Gait Assessment (FGA) is utilized to provide a balance/gait performance outcome measurement (Figure 1). The FGA is a 10-item test based on the Dynamic Gait Index (DGI), with 3 additional sections added: gait with narrow base of support, gait with eyes closed, and ambulation backwards. The maximum score is 30, and higher scores indicate better performance.^{48,49} Literature had determined that the minimal detectable change for the FGA outcome measurement is 6 points in patients with vestibular and balance dysfunction.⁴⁸ Alsalaheen et al discovered a ceiling effect in the vestibular rehabilitation outcome measures, Activities Balance Confidence Scale (ABC) and FGA, in the adolescent population. Due to the ceiling effects, other measures such as Timed Up and Go (TUG), 5 Times Sit to Stand Test (FTSTS), and gait speed are recommended over the FGA for evaluation and testing recovery after concussion in the adolescent population.⁵⁰ In addition, further studies by Alsalaheen et al found that most vestibular outcome improvements did not depend on age. The FTSTS was the only outcome that was drastically different in evaluation testing between the 2 groups, with children having the quicker performance times. In a retrospective chart review of 114 patients, vestibular rehabilitation equally benefited both adolescents and adults.⁴⁹

After a concussion, different vestibular rehabilitation techniques may be used based on individual symptoms and impairments present. Commonly seen impairments with a concussion are BPPV, VOR delays, VMS, especially busy environments, balance impairments, and exercise-induced dizziness.^{2,6} Testing and treatment through canalith repositioning maneuvers could assist if the patient demonstrates BPPV. Impairment of the VOR can be improved with specific gaze-stabilization training.

Gaze stabilization refers to the ability to hold the eyes on a fixed target while the head is in motion. Gaze stabilization training requires the patient to maintain visual focus, while moving his or head in a variety of positions to facilitate recovery. The VOR is the primary system to maintain the eye position during head motion, keeping the eyes stable by generating ocular movements precisely in proportion, but in opposite direction, from the head motion. *Habituation training* is utilized for motion sensitivity. The patient undergoes repeated exposure to visually stimulating environments to “habituate” or normalize patient responses to motion.

Substitution training is to promote alternative strategies for the impaired vestibular function. *Balance training* is utilized to integrate somatosensory, visual, and vestibular systems to assist with overall balance and posture control. Higher level balance training incorporates divided attention training and dynamic balance training to reduce dizziness and improve balance after concussive events. Several studies have shown that patients following an mTBI have greater difficulty maintaining balance under conditions of divided attention.²

Post-traumatic migraines

Post-traumatic headaches (PTH) are quite common, occurring in near 70% of concussion cases.^{1,51} A majority of PTH patients share common symptoms of migraine or tension-type headaches.^{52,53} PTH is defined by The International Classification of Headache Disorders as a headache that will develop within the 7 days following trauma or injury.⁵³ PTH usually resolves within 3 months post injury, but some individuals have lingering chronic headaches.⁵³ These headaches can arise from a variety of sources: medication overuse, cervical dysfunction, exertional, and/or autonomic response, mood disorders, muscular tension and from migraine components.¹ The clinician needs to have a working knowledge of the patient’s personal or familial history of prior migraines, with or without visual auras. Motion sickness is more prominent in patients with migraine history by 30% to 70%.⁵⁴ Research has indicated that children with migraines have 45% elevated chance for motion sickness, compared to other control groups.⁵⁴ In addition, concussed patients often experience photosensitivity to light and phono-sensitivity to loud noises in busy environments.⁵⁵ It is critical to determine if underlying issues in the domains of motion sensitivity, photosensitivity, or phono sensitivity have arisen with

prior migraine history.¹ Subjectively, the patient needs to report any photo- or phono-sensitivity, visual auras, unilateral headaches with throbbing quality, nausea/vomiting, or dizziness^{2,51} and provide a detailed familial medical history of migraine involvement. Migraine headaches are the most common type of post-traumatic headache detected in both SRC and non-sport populations.¹ While research indicates that a family history of migraines may be helpful for a clinical diagnosis, there are no biological markers associated with diagnosis to date. While post-traumatic migraines are not clearly understood, studies indicate that post-traumatic migraines are not solely an extension of pre-existing migraines, but more likely the interaction of concussion-induced cortical hyperexcitability and genetic predisposition.¹

Migraine attacks can often be moderate or severe in intensity, typically unilateral in presentation, and can last from 4 to 72 hours if left untreated.^{1,51} In addition, a variation of migraines, vestibular migraine, has been shown to be a source of routine dizziness, most prevalent in the younger population. Post-traumatic vestibular migraines involved subjective complaints of dizziness following concussion.¹ In cases of vestibular migraines, vertigo and/or dizziness symptoms are caused directly by the migraine, lasting from seconds to days.⁵⁴ Menses, stress, lack of sleep, dehydration, and diet may trigger attacks, although they are not listed as diagnostic criteria due to lack of validated research.⁵³ The pathophysiology of migraines includes the trigeminovascular pain pathways that can radiate pain along the jawline and can result in headaches and other symptoms.¹ Migraines are regarded as neurovascular disorders, and it is hypothesized that the persistent inflammation from the brain injury sensitizes the trigeminal pain neurons.⁵¹ Migraine prophylaxis has been shown to offer some benefit to preventing or shortening vestibular migraine attacks, especially with the use of triptans.^{52,53} In a study by Lauritsen and Marmura,⁵⁶ vestibular migraine acute attacks can benefit from the use of triptans, and prevention medications that have been shown to be helpful include propranolol, venlafaxine, topiramate, and amitriptyline. In addition, tricyclic and SSRI antidepressants, gabapentin, valproic acid, and beta blockers have been suggested to also use as preventive medications in this mTBI population. Triptans (eg, Imitrex, Maxalt) are often utilized as abortive medications for post-traumatic migraines.²

Diagnosing post-traumatic migraine involves utilizing the criteria outlined by the International Headache Society.⁵⁷ The clinician may find it beneficial to utilize the Migraine Disability Assessment Test (MIDAS) or the Head Impact Test (HIT-6) questionnaire (Figure 1) to determine the severity of pain and impact of the migraine on the overall health of the individual.¹ To address migraine symptoms, the TUKHS clinicians provide ocular exercises based on any dysfunction seen on evaluation, as well as cervical manual therapy and stabilization to ease upper neck and shoulder pain often seen with this patient population.

Mood disorders

From a clinical standpoint, it has been well documented that a prior history of mood and emotional disturbances affect outcomes in therapy.^{1,9} Following a concussion, the metabolic and catastrophic mental changes can often affect an individual in having heightened anxiety, panic attacks, and depression.¹ Studies indicate that up to 40% of post-concussion syndrome (PCS) patients suffer from fatigue, sleep disturbances and mood alterations within 1 to 3 months following their mTBI.¹⁵ Patients will often feel irritable with the inability to cope with prior work and household duties and experience more sadness and emotional episodes than prior to the injury. In a study by Chaput et al,¹⁵ patients suffering from sleep complaints were between 6 and 9× more likely to also report feeling depressed at various time periods, and were approximately 12× more likely to report concomitant irritability at 10 days and 4.8× at 6 weeks post-injury. A predisposing history of anxiety or depression and mood changes can elevate an already sensitive state.^{14,40} Issues with sleep dysfunction (either too little or too much) can rapidly escalate the patient's overall frustration or irritability to concentrate, think clearly, and decrease overall cognitive ability.⁵⁸ At TUKHS, the patients' direct and open communication with the physician is highly encouraged to ensure medical management and support is provided. The hospital anxiety and depression scale (HADAS) outcome measurement can be administered (Figure 1) to determine if outside psychological support is warranted.⁵⁹ At TUKHS, access to testing through neuropsychology is available based on provider recommendations and specific patient needs. Psychotherapy should be offered as an alternative or collaborative treatment in conjunction with pharmacology treatment. Studies show that a large portion of patients with TBI prefer psychotherapy to pharmacological interventions,⁶⁰ especially cognitive-behavioral therapy.

Exertion

Studies have found that following a mTBI, there is a decrease in cerebral autoregulation and diffuse reduction in cerebral artery flow.^{1,61-68} Many changes with cerebral vasoreactivity can occur as a result to direct or indirect forces to the head.^{2,64} Patients may notice chronic issues, such as dizziness, headache, and exercise intolerance, especially with exertion and taxing of the cardiovascular system.^{1,65} Research has suggested evaluation on the exertional responses through perceived exertion (BORG) testing, HR, BP, and VAS values regarding symptom magnification in athletes with regard to treadmill testing.^{3,65,69} Using the treadmill as the standard testing protocol tool can prove difficult as provocation of the cervical component of a concussion patient's injury can evoke similar symptoms.⁶⁵ The Buffalo Concussion Treadmill Test (BCTT) and Buffalo Concussion Bike Test (BCBT) research by Leddy and Willer,^{61,62,65-67} have established successful methods that can

assist in the assessment of exertional levels in athletes to assist in guiding both the medical team and athlete in return to play. Similar to our outpatient exertion protocol (Figures 1 and 4) on the stationary bike, the patient pedals at 60 revolutions per minute (RPM) but the resistance is increased every 2 minutes, versus every minute in our clinical protocol.⁶⁵ Vitals such as heart rate, symptom ratings of headache and dizziness, and RPE/BORG are recorded. At TUKHS, BP is checked every 5 minutes, and the test is stopped when: RPE/BORG reaches 17 or more, symptom increase with either headache or dizziness by 3 points or more, the patient cannot maintain a 60 RPM speed or cannot continue due to fatigue. The TUKHS exertion testing has similar parameters to the BCBT.^{65,67,69} After testing, there is a 2-minute cooldown at the first stage resistance at 30 RPM (BCBT); TUKHS protocol continues at 60 RPM speed with resistance decreased to initial testing resistance.⁶⁶ At TUKHS, all mTBI exertional testing is performed on a stationary bike due to the relative stability of the head and torso during testing. In addition, the bike protocol does not stimulate the vestibular and cervical input compared to the treadmill test, and, therefore, the patient is less likely to fall while seated.⁶⁶ Data from the exertional testing assists the clinician and physician in determining if gradual increases in return to normalized gym or workout activity is warranted. All patients that are prescribed aerobic exercise are measured at 80% to 90% (BCBT) or 80% (TUKHS) of the maximum heart rate achieved at symptom exacerbation at initial visit, for daily exercise, 5 to 6 days a week, for 20 minutes.^{3,61,62,66-68} Patients are instructed to stop the home exercise sessions if symptoms increase by more than 2 points of the pre-exercise baseline on a 10-point VAS scale or at 20 minutes, whichever occurs first.^{61,62} Patients are re-assessed every 2 to 3 weeks to establish a new target HR until symptoms are no longer intensified by aerobic training.^{62,67} Athletes often respond quicker, and can increase by 10 BPM every 1 to 2 weeks, whereby the non-athletic population usually responds better to 5 BPM increments every 2 weeks.^{65,67} Resolution of post-concussion exertional delays will be when the participant can exercise at a peak range of 85% to 90% (BCBT) of their age-predicted maximum HR for 20 minutes without any symptom reproduction.⁶⁷

Cognition

A partnership with speech therapy is vital in assisting with the cognitive delays often experienced by concussed patients.^{1,59} Studies suggest that speech intervention should be implemented several months after date of injury to allow time for cognitive recovery and assist with memory impairments.^{1,59} In a study by Alsalaheen et al⁴⁸ on determining effects of cognitive impairment on balance in post-concussion adolescents, 65% of participants with a median of 46 days post injury showed at least 1 cognitive deficit in the areas of visual and verbal memory, reaction time, and processing speed. Visual and

verbal memory skills demonstrated a stronger correlation in gait and balance outcomes compared to reaction time. In a cross-sectional study⁵⁹ including 86 patients, poor memory at 67% and concentration at 88% were some of the mostly commonly reported symptoms of PCS.

Depending upon the location of injury, the frontal cortex, which specifically drives core executive function, may be impaired. Frontal and temporal injury are the 2 most common lobes affected in a concussion injury, and both can impact the short-term memory and attention to task.⁷⁰ A patient's working memory can impact long-term learning outcomes and is essential for comprehension, learning, and reasoning. With frontal cortex impact the patient may exhibit slow behavior changes or personality changes.⁷⁰ The parietal cortex is vital in spatial-perceptual skills for tasks and frontal-parietal damage impairs executive function. In the subjective evaluation, careful observation of the patient appearing dazed or stunned is an indication that further cognitive testing needs performed. A patient will commonly exhibit confusion about the events of the accident, both prior to and after the injury, or even on the day of clinical testing. Delays in articulation and word formation may also be seen. Through strategies and home exercise recommendations, speech therapy can be instrumental in both working memory and attention to task.¹ Initially, the speech concussion specialist may focus on one specific task for a continuous amount of time without distraction before building toward higher levels. Effective speech training should incorporate cognitive training while simultaneously walking around the hallway (Figure 3), busy cafeteria or other stimulating environments to simulate real-life activities in the home and community. Selective attention is the ability of the patient to maintain focus or select only one task while filtering out other distractions. Elevated training would involve the patient switching the focus back and forth between tasks with different cognitive demands, thus providing alternating attention. Lastly, with decreased symptom provocation and improvement, divided attention should be stressed to provide the patient with the tools to return to reacting to different demands simultaneously.

Speech outcome measures for the outpatient population may include but are not limited to: Montreal Cognitive Assessment (MoCA) screening, Wechsler Memory Scale III (Auditory), Working and Verbal Memory, Attention Processing Test (Attention), Cognitive Linguistic Quick Test, RIPA-2/RIPA G-2, Woodcock-Johnson, Cognitive subtest, and Assessment of Language-Related Functional Activities (ALFA) (Functional ADL's).

Conclusion

Patients presenting for assessment and treatment will have sustained their concussion injuries due to assorted mechanisms of injury and will be from various timelines from injury. These concussion patients will also exhibit many different symptoms. Due to the heterogeneity of each patients' injuries and cases,

outpatient physical therapy clinicians should assess and manage each patient on an individual basis, utilizing uniquely applied therapy strategies to address each patient's concerns and impairments. While patient histories and injuries may be varied, treatment strategies and therapies can take a systematic approach. Implementing a standardized team assessment and treatment method and utilizing treatment protocols provide consistent care. Development of the mTBI clinical recommendation protocols by the TUKHS concussion rehabilitation therapy team has proven beneficial to the TUKHS processes. Recognizing the potential utility by others of these foundational comprehensive tools, the TUKHS team wanted to share this information with other concussion providers.

A thorough and comprehensive screening should consist of these key areas: oculomotor, cervical, vestibular, post-concussion migraine influence, mood disorders, exertion, and cognitive dysfunction.¹ An interdisciplinary concussion team can provide the patient with a well-rounded approach to treating cognitive, emotional, and physical aspects of post-concussion recovery. At the TUKHS, referrals and consultations with neuro-optometrists are employed when traditional physical therapy interventions for concussion-related convergence insufficiency, depth perception dysfunction, and visual delays remain. Cervical care is shared both by the concussion physical therapist specialists and orthopedic physical therapist specialists in the clinic. Incorporation of a partnership with the neuropsychology team may also prove beneficial to address the effects of existing or new onset of anxiety-depression with regards to progress in rehabilitation. Additionally, incorporating a neurologist or a medical care team member specializing in post-concussion migraine would be instrumental in providing comprehensive care. With a detailed assessment and a comprehensive team approach, the success of the rehabilitation of the concussed population will be effective and efficient for both the patient and clinical team alike.

Management Strategies

To assist the clinical direction of evaluation and treatment:

Figure 1 – Concussion evaluation/protocol.

Figure 2 – Outpatient concussion – special tests.

Figure 3 – Treatment strategies.

Figure 4 – Exertion protocol.

Author Contributions

The first draft was completed by first author, Maria Davenport, and then subsequent drafts and edits were made by all co-authors. All authors contributed to the content, text, figures, and references, as well as the writing and editing of the manuscript. All authors agreed with the text and conclusions of the article, and reviewed and approved the final version.

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