**Sports Related Concussion**

Introduction:

Concussions are characterized as mild brain injuries (mTBI) and the mildest form of diffuse axonal injury (DAI).4 The standard and accepted definition in the literature states concussions are “a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces.”1,2,4 Concussions commonly occur after falls, motor vehicle accidents, unintentional head trauma, assaults, and sports.5 Concussions occur when acceleration-deceleration forces are directed at the head and cause a mechanical “shake” to the brain immediately after impact.4 Rotational acceleration ranging from 4,500 to 12,500 rad/s2 is required to cause a concussion and mild DAI.4 This mechanism of injury results in a range of functional disturbances with minimal structural damage to the brain.4 This level of apparent injury contributes to what the CDC refers to concussions as a “silent epidemic”.4

All sports have some degree of risk related to concussion.7 In the United States, there are about 1.6 to 3.8 million sports and recreational related brain injuries that occur annually.3,5 About 75% of these are classified as mild brain injuries or concussions. The CDC has seen a 62% increase in the incidence of sport related concussion in the 19 year old and younger age group between 2001 and 2009.1 This wide and growing incidence can be attributed to the public’s decreased awareness, athletes underreporting symptoms, difficulty detecting and diagnosing concussion.4,5 It is suggested that about 50% of concussions may not be reported.7 Although the incidence for concussion is relatively high, the good news is that the death rate for these injuries is extremely low at 0.2%.

The highest concussion rates are found in football, soccer, hockey, rugby, and basketball for both the high school and collegiate athletes.5,7 However, collegiate athletes have a higher concussion rate than high school athletes.5 Further, males are at a higher risk of concussion, but females report a higher rate of concussion.3 This is possibly due to lower neck strength and girth, which generates greater head acceleration after impact.5 Females also experience worse concussive symptoms, outcomes, a longer symptom duration and recovery period compared to males.3

The APTA’s Guide to Physical Therapy Practice6 contains three practice patterns that are associated with concussion. Pattern 5A: primary prevention/risk reduction for loss of balance and falling. This pattern includes the side effects that can be associated with concussion such as dizziness, depression, and vestibular dysfunction. The management of concussion is clearly stated in patterns 5C and 5D: impaired motor function and sensory integrity with non-progressive disorders of the central nervous system – congenital origin or acquired in infancy or childhood, and acquired in adolescence or adulthood.6

Pathophysiology and Systems Affected:

Despite concussions being characterized as mTBI, profound biochemical changes occur immediately after a concussion. The rotational forces directed to the head cause microscopic damage by stretching the neuronal and axonal membranes. This activates a complex “neurometabolic cascade” of events. Ionic homeostasis is altered by the influx of ions and the wide release of neurotransmitters. The cell membrane permeability malfunctions due to an influx of calcium that overloads the mitochondria, and leads to oxidative stress. This requires the neurons to work overtime and generate ATP through rapid glycolysis. These events and the body’s attempt to repair itself cause an “energy crisis”, where there is not enough ATP. The imbalance between ATP production and utilization stems from mitochondrial dysfunction and decreased cerebral blood flow.4,7 Studies have seen a 46% increase in glucose within only 30 minutes after injury and can remain for 4 hours. These biochemical changes are primarily responsible for cognitive deficits, impaired synaptic plasticity, and depressed brain metabolism seen after concussion. 4

These metabolic changes are reversible; however, they make the brain more susceptible to another injury.4 The brain remains in a vulnerable state for multiple days following the incident until complete recovery ensues. The exact duration of vulnerability is unknown due to unique, individual characteristics of each concussion. Additional injury can further escalate concussive symptoms, cause severe and irreversible cellular damage to the brain, prolong recovery, and ultimately result in cell death.3 Animal studies have found that two mTBIs occurring in close proximity of each other can cause cumulative cellular damage that is more indicative of a severe brain injury (sTBI) compared to the group with only one mTBI. This event is known as secondary impact syndrome (SIS).4

Concussive signs and symptoms involve changes in physical, cognitive, emotional, and sleep functioning. The most two common signs and symptoms associated with concussion are headache and dizziness. Other physical symptoms include nausea, vomiting, balance deficits, fatigue, visual problems, sensitivity to light and sound, dazed and confused. Cognitive symptoms involve feeling mentally foggy, slowed down, difficulty concentrating, and forgetfulness. Next, feelings of depression, nervousness, irritability, and sadness are common changes in emotional status. Concussion can also cause sleep disturbances that involve more or less sleep than usual, difficulty falling asleep, and general drowsiness.It is important to note that loss of consciousness is not necessary and only occurs in 10% of concussion cases.7

Based on the location of the trauma and the signs and symptoms above, concussion primarily affects the neuromuscular, vestibular, and cognitive systems. Neuromuscular deficits influence gross coordinated movements (i.e. balance, gait, transfers, and mobility), motor control, and motor learning.Impairments of the vestibular system involved prolonged dizziness, BPPV, and also influences balance, coordination, and mobility. Concussion can alter acute consciousness, awareness, and orientation to person, place, and time. It can create educational barriers as communication, learning preferences and needs change. Cognitive impairments also influences personality, emotional and behavioral responses.6

The severity of deficits in these systems is dependent upon age. Young athletes are believed to be more resilient. However, there are critical periods of development where their brains are more susceptible to injury.10 Sustaining a concussion during this prime period impairs current and future brain plasticity.9,10 Prolonged postconcussive symptoms and recovery is also associated with the severity of the injury19, multiple concussions3, inappropriate initial concussion management14, and premature return to play (RTP)7. The presence of dizziness, headache, and nausea at the initial ER visit is strongly associated with continued symptoms at 6 months post-injury.14 Additionally, the exacerbation of symptoms 45-90 days post-injury19, continued dizziness 21 days post-injury7, increased migraines and cognitive symptoms7 are also associated with a long recovery.

Impairments:

The impairments caused by sports related concussion are: 7, 8

* Impaired cognitive function
* Slowed reaction time
* Impaired quantity and quality of sleep
* Head and neck pain
* Impaired balance and postural control
* Vestibular deficits
* Dizziness
* Fatigue
* Emotional dysregulation
* Personality changes

Activity and Participation Limitations:

Studies commonly report that 80-90% of the patient’s concussive symptoms are resolved within 3-10 days of the initial injury.4,7 However, to complicate issues, brain metabolism may remain altered when clinical signs and symptoms are no longer present.4 Therefore, the lack of symptoms does not indicate a full recovery! This can offer a false sense of security and places the patient at a significant risk for another concussion.7 Because of the brain’s vulnerability after the initial injury and the risk for secondary injury, physical and mental activities should be limited. The duration of activity limitation is dependent upon individual healing factors, recurrent concussion, the severity of concussion, age, health, and degree of rest.13

After a concussion, physical and mental rest is highly recommended until symptoms resolve.11 Rest is thought to facilitate recovery from the “neurometabolic crisis” that occurs and acts to protect from further injury.11 This may indicate time away from school, work, sports, reading, television, video games, recreational activities, and exercise.12 Exercise has also been seen to facilitate neuroplasticity, improve cognitive and memory functioning, sleep quality, and self-esteem. However, if exercise is initiated too soon, it can impair neuroplasticity, prolong the “neurometabolic crisis”, and extend the risk of secondary injury.11 To reduce these negative effects, exercise should not begin until initial symptoms subside. It is important to note that if exercise and physical activity is too restricted for athletes then it can led to deconditioning, stress, anxiety, irritability, and depression, which can further complicate concussive symptoms and prolong recovery.11 Therefore, rest and properly timed exercise can initiate brain healing and reduce possible complications after a concussion.

Once symptoms begin to subside then a gradual, submaximal return to activities can begin. This process should not exacerbate or provoke concussive symptoms.11 The Zurich Protocol2 is a recently revised RTP protocol that acts as a guide to progress the individual back to prior level of activities. It consists of 6 steps that are at least 24 hours in length. Generally, the rehabilitation protocol should take about a week if the athlete remains asymptomatic. If symptoms reoccur, the athlete returns to the previous asymptomatic stage for the next 24 hours and restarts the process.2

Stage 1 involves complete physical and cognitive rest.

Stage 2 permits light aerobic exercise such as walking, swimming, or stationary cycling. The intensity should be kept below 70% of the maximal predicted heart rate.

Stage 3 includes sport specific exercise and allows drills; however, no head impact activities are allowed.

Stage 4 begins to progress drills into more complex training drills such as passing. Athletes may start progressive resistance training to improve exercise capacity, coordination, and cognitive load.

Stage 5 aims to restore the athlete’s confidence and functional skills by allowing full contact practice and a normal training schedule.

Stage 6 is the final stage where full participation is allowed in practice and competitions.

Other RTP guidelines suggest the athlete should not immediately return following a concussion or a suspected concussion. Play should not resume if the athlete requires medication to mask concussive symptoms. Additionally, the athlete should be completely symptom free at rest and with exertion, and return to academic workload prior to athletic activities.7

Environmental and Individual Factors that Influence Outcomes:

The diagnosis of concussion is very much dependent on the awareness and knowledge of concussive signs and symptoms and on the self-report of the patient. Research has shown that athletes, coaches, and parents lack the knowledge to make informed decisions relating to concussion identification, management, RTP, and prevention.7,23 This leads to premature RTP that can result in secondary injury, worsening and prolonged symptoms, and a severe brain injury.7 Affected athletes may be suffering in silence and unaware of the treatment options that can enable them to return to their prior level of function. Therefore, organizing education into the preseason period of at risk sports could help increase the awareness of concussive symptoms, RTP protocols, and treatment options for all parties involved.

Intervention:

Concussive symptoms of dizziness and balance deficits may continue to persist beyond the expected 10 day recovery period. These symptoms can extend for weeks, months, and years!11 This knowingly occurs in 10-20% of concussive cases.4,7 However, the percentage of this group receiving subsequent treatment is unknown. When these symptoms do not resolve spontaneously, vestibular rehabilitation may be indicated. The research shows vestibular intervention to be effective in resolving and managing these continued symptoms.

Concussion can cause positional dizziness, migraine associated dizziness, exercise induced dizziness, and spatial disorientation. A detailed examination is required to determine the type of provocation and the source of impairment to apply the appropriate treatment options. Positional vertigo will present with positive nystagmus on the Dix-Hallpike test and can be immediately treated with canalith repositioning maneuvers. Common reports of migraine associated dizziness involve headaches with variable periods of unsteadiness. These individuals will display normal static posture, but will show abnormalities with challenged gait activities. Exertional dizziness occurs during and after exercise where challenged gait activities will produce abnormalities. Additionally, spatial disorientation involves a constant feeling of unsteadiness in all postures, with standing being the worst. These individuals will drift from midline during static standing and gait. Vestibular physical therapy intervention can reduce dizziness, increase balance, and general activity levels through specific exercises. Intervention that includes gaze stabilization, dynamic visual acuity, static and dynamic postural stability, desensitization of head movements, and aerobic conditioning creates the greatest reduction of prolonged concussive symptoms. Gottshall recommends this intervention to be monitored and progressed in the clinic twice a week for 8 weeks and patients should also complete these exercises at home twice daily.21

Gurley et al discusses the importance of early assessment and proper management after concussion. The vestibular system should be evaluated early to differentiate symptoms related to the “neurometabolic crisis” or to vestibular dysfunction. If not identified early, concussive symptoms can become chronic. Prolonged dizziness can be cause by BPPV, gaze instability, impaired integration of sensory systems, and early return to exercise. Balance assessments are integral in evaluating protective and righting reactions to LOB and changing environments. The authors note that concussive patients do not use these reactions correctly and will overcompensate with stepping and hip strategies with a minimal LOB. Therefore, challenging all sensory systems via eyes closed/open, head turns and tilts, various surfaces, rocker board, weight shifting, and walking challenges will help reintegrate the systems. Vestibular physical therapy should begin slow and simple in a quiet environment and progressed in complexity over time. Multiple sessions of limited exercises aims to improve exercise tolerance and gain program adherence.14

Alsalaheen et al performed a study to determine if vestibular rehabilitation was effective in decreasing dizziness, and improving gait and balance in children and adults after a concussion. A retrospective chart review was completed on 114 patients. The average time between concussion and initial evaluation was 61 days. The intervention averaged 4 sessions over 33 days. The most frequent exercises prescribed included: gaze stabilization exercises (VORx1) in sitting and standing, static standing balance on firm and compliant surfaces, and walking with balance challenges such as head turns, tandem walking, and obstacle avoidance. These exercises were performed once a day as a HEP. Significant treatment effects were noted in all self-report (DHI, ABC, and dizziness severity), balance and gait measures (DGI, gait speed, and SOT) by discharge. Interestingly, the children experienced greater improvements in dizziness severity. These results show that vestibular therapy is beneficial and effective to reduce prolonged dizziness, improve gait, and balance after a concussion in both children and adults. Vestibular rehabilitation should be part of the management following concussion in order to prevent these prolonged concussive symptoms.20

It is also helpful to differentiate between cervicogenic dizziness vs. vestibular dizziness. Cervicogenic dizziness commonly results from a whiplash-like injury and can mimic similar symptoms. It will most likely present with neck pain and decreased cervical range of motion. Conversely, athletes with vestibular dysfunction heavily rely on visual input for balance and are unable to effectively use their vestibular system after a concussion. Vestibular rehabilitation therapy can restore normal function by inducing neural plasticity within the existing, healthy neural pathways that allow for adaption and compensation of functional deficits. The three main compensatory systems are habituation, adaptation, and substitution. The goal of habituation is to resolve dizzy symptoms through increased exposure of provoking stimuli and/or head movements. Adaptation exercises stimulate the CNS to modify the loss of vestibular system input by strengthening functional neural output. Common exercises are VORx1 and VORx2. Lastly, substitution utilizes other eye movements such as saccades to compensate for deficits in the VOR in order to achieve gaze stability.22

Outcome Measures:

Using outcome measures to assess function after a suspected concussion is imperative. It is also important to retest the individual before complete RTP because they may deny active symptoms or have no symptoms, but still display balance and cognitive deficits upon testing. There are many outcomes measures that can be completed on the sideline or in the clinic.

The sideline tests include:

* Standardized Assessment of Concussion (SAC) – This is a brief screening tool that measures orientation, immediate memory, concentration, and delayed recall. This test alone is not appropriate to determine RTP and the results should be interpreted with caution.3
* Balance Error Scoring System (BESS) – Measures static postural stability by assessing 6 conditions in Romberg, tandem, and SLS with eyes closed for 20 seconds on firm and compliant surfaces. The score ranges from 0-60 where errors are counted. A low score indicates few errors and better balance.16
* Sensory Organization Testing (SOT) – A 20-minute test that assesses the patient’s balance by testing 6 conditions to identify sources of instability. Each condition should be tested 3 times and averaged together for a score. Each position needs to be maintained for 30 seconds. Points (1-4) are based on the level of sway where 1 is minimal sway and 4 is a fall.16
* Sport Concussion Assessment Tool (SCAT2) – This is both a sideline and clinical assessment tool. It contains a 22-item Postconcussion Symptom Scale that assesses the number and severity of symptoms, the signs of LOC, balance deficits, Glasgow Coma Scale (GCS), Maddock score of orientation, the SAC, coordination tests such as finger to nose, and the modified Balance Error Scoring System (BESS).3 There is a maximum of 100 points.
* Concussion Recognition Tool (CRT) – Identifies concussion through the recognition of visible cues of concussion, a list signs and symptoms, memory, and red flags. The presence of one of more symptom in either category indicates the athlete’s removal from play.17
* Maddock’s Test – Contains 5 simple and adaptable questions that assess orientation and memory. This test is sensitive in distinguishing concussion vs. non-concussion.15 Each question is worth 1 point, with a total of 5 points.

Outcome measures utilized in the clinic are:

* Sensory Organization Testing via NeuroCom – A 15-minute computerized test that utilizes a force plate to measure postural sway and center of pressure under 6 testing conditions. It identifies abnormalities of the somatosensory, vestibular, and visual systems that influence postural sway, integration of sensory systems, ankle and hip strategies, and center of gravity alignment. The higher the scores, the better the balance.16
* Immediate Postconcussion Assessment and Cognitive Testing (ImPACT) – This is a computerized neuropsychological test battery that measures various aspects of cognitive functioning such as attention, working memory, reaction time, and problem solving. This is a valuable tool because it is able to detect neuropsychological deficits in individuals who are asymptomatic.3

Other tests include dizziness measures: 14

* Dizziness Handicap Inventory (DHI) – A 25 item self-assessment that evaluates self-perceived handicap due to dizziness in 3 categories --functional, emotional and physical. The score ranges form 0-100. The higher the score, the greater perceived dizziness.16
* Activities-specific Balance Confidence Scale (ABC) – A 16 item self-report that measures confidence in various ambulatory activities without falling or feeling unsteady. Items are ranked on a 0-100 scale with 0 having no confidence and 100 being very confident.16
* Post-Concussion Symptom Scale (PCSS) – Self-identification of concussive symptoms. The presence of symptoms are ranked on a scale of 0-6, with 0 indicating no symptoms and 6 indicating severe symptoms.18
* Visual Vertigo Scale (VVS) – Rates the intensity of visual vertigo in 9 dizziness provoking situations on a 0-10cm line with 10 indicating severe dizziness.16

The three most commonly used assessments for evaluating concussion are the Sideline Assessment of Concussion (SAC), Sport Concussion Assessment Tool 2 (SCAT2), and the Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT).3 Both the SAC and the SCAT2 are used to assess neurocognitive impairment; however, neither test has been validated to evaluate recovery from a concussion.3

References:

1. Clay MB, Glover KL, Lowe DT. Epidemiology of concussion in sport: A literature review. *J Chiropr Med*. 2013;12(4):230-251. doi: 10.1016/j.jcm.2012.11.005; 10.1016/j.jcm.2012.11.005.
2. McCrory P, Meeuwisse W, Johnston K, et al. Consensus statement on concussion in sport - the 3rd international conference on concussion in sport held in Zurich, November 2008. *PM R*. 2009;1(5):406-420. doi: 10.1016/j.pmrj.2009.03.010; 10.1016/j.pmrj.2009.03.010.
3. Borich MR, Cheung KL, Jones P, et al. Concussion: Current concepts in diagnosis and management. *J Neurol Phys Ther*. 2013;37(3):133-139. doi: 10.1097/NPT.0b013e31829f7460; 10.1097/NPT.0b013e31829f7460.
4. Signoretti S, Lazzarino G, Tavazzi B, Vagnozzi R. The pathophysiology of concussion. *PM R*. 2011;3(10 Suppl 2):S359-68. doi: 10.1016/j.pmrj.2011.07.018; 10.1016/j.pmrj.2011.07.018.
5. Almasi SJ, Wilson JJ. An update on the diagnosis and management of concussion. *WMJ*. 2012;111(1):21-7; quiz 28.
6. Interactive Guide to Physical Therapy Practice. ATPA. Published online February 4, 2010, ISBN: 978-1-887759-87-8.doi:10.2522/ptguide.3.2\_3. Published in: 2003.
7. Harmon KG, Drezner JA, Gammons M, et al. American medical society for sports medicine position statement: Concussion in sport. *Br J Sports Med*. 2013;47(1):15-26. doi: 10.1136/bjsports-2012-091941; 10.1136/bjsports-2012-091941.
8. Schmidt JD, Register-Mihalik JK, Mihalik JP, Kerr ZY, Guskiewicz KM. Identifying impairments after concussion: Normative data versus individualized baselines. *Med Sci Sports Exerc*. 2012;44(9):1621-1628. doi: 10.1249/MSS.0b013e318258a9fb; 10.1249/MSS.0b013e318258a9fb.
9. McKeever CK, Schatz P. Current issues in the identification, assessment, and management of concussions in sports-related injuries. *Appl Neuropsychol*. 2003;10(1):4-11. doi: 10.1207/S15324826AN1001\_2.
10. Giza CC, Hovda DA. The neurometabolic cascade of concussion. *J Athl Train*. 2001;36(3):228-235.
11. Schneider KJ, Iverson GL, Emery CA, McCrory P, Herring SA, Meeuwisse WH. The effects of rest and treatment following sport-related concussion: A systematic review of the literature. *Br J Sports Med*. 2013;47(5):304-307. doi: 10.1136/bjsports-2013-092190; 10.1136/bjsports-2013-092190.
12. Purcell L, Kissick J, Rizos J, Canadian Concussion Collaborative. Concussion. *CMAJ*. 2013;185(11):981. doi: 10.1503/cmaj.120511; 10.1503/cmaj.120511.
13. Concussion and mild TBI. Centers for Disease Control and Prevention Web site. <http://www.cdc.gov.libproxy.lib.unc.edu/Concussion/>. Updated 2013. Accessed March 6, 2014.
14. Gurley JM, Hujsak BD, Kelly JL. Vestibular rehabilitation following mild traumatic brain injury. *NeuroRehabilitation*. 2013;32(3):519-528. doi: 10.3233/NRE-130874; 10.3233/NRE-130874.
15. Concussion diagnosis and management. Sports Concussion South Africa Web site. <http://www.sportsconcussion.co.za.libproxy.lib.unc.edu/about-concussion/diagnosis-management/>. Accessed March 25, 2014.
16. Rehabilitation Institute of Chicago. Rehabilitation measures database. <http://www.rehabmeasures.org/Lists/RehabMeasures/Admin.aspx>. Accessed March 25, 2014.
17. Concussion recognition tool. British Journal of Sports Medicine Web site. <http://bjsm.bmj.com.libproxy.lib.unc.edu/content/47/5/267.full.pdf>. Published 2013. Accessed March 26, 2014.
18. Post concussion symptom scale. <http://www.hawaiiconcussion.com/PDF/Post-Concussion-Symptom-Scale.aspx>. Accessed March 25, 2014.
19. 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. doi: 10.1017/S1355617712000872; 10.1017/S1355617712000872.
20. Alsalaheen BA, Mucha A, Morris LO, et al. Vestibular rehabilitation for dizziness and balance disorders after concussion. *J Neurol Phys Ther*. 2010;34(2):87-93. doi: 10.1097/NPT.0b013e3181dde568; 10.1097/NPT.0b013e3181dde568.
21. Gottshall K. Vestibular rehabilitation after mild traumatic brain injury with vestibular pathology. *NeuroRehabilitation*. 2011;29(2):167-171. doi: 10.3233/NRE-2011-0691; 10.3233/NRE-2011-0691.
22. Aligene K, Lin E. Vestibular and balance treatment of the concussed athlete. *NeuroRehabilitation*. 2013;32(3):543-553. doi: 10.3233/NRE-130876; 10.3233/NRE-130876.
23. King D, Brughelli M, Hume P, Gissane C. Assessment, management and knowledge of sport-related concussion: Systematic review. *Sports Med*. 2014. doi: 10.1007/s40279-013-0134-x.