

Vestibular and balance treatment of the concussed athlete

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Abstract.

OBJECTIVES: The purpose of this chapter is to provide an update on the clinical management of vestibular and balance dysfunction in a concussed athlete with a focus on diagnosis, initial work-up, and initial and continuing management. Although much is still unknown about the etiology of vestibular and balance dysfunction in a concussed athlete, we briefly review current theories about neural pathophysiology to help link proposed treatment methodologies.

INTRODUCTION: The treatment and management of vestibular and balance dysfunction in concussed athletes requires a multidisciplinary approach and is based on continuous reassessment of the presenting symptoms. The clinical challenge toward managing persistent symptoms of the post-concussive athlete is discerning whether a set of symptoms match diagnostic testing and whether further neurological work up is necessary. Because there are no discrete time boundaries to make such judgment calls, we offer a guide to help with the difficult clinical decisions necessary to treat the post-concussive athlete.

METHODS: Literature search was performed using the following keywords: Vestibular and balance dysfunction, concussion, concussed athlete and treatment, vestibular rehabilitation therapy. Original research studies, literature reviews, and clinical guidelines were reviewed between 1997 and 2012, with the majority of articles dating beyond 2004. Although we acknowledge that post-concussive states lie within a continuum, we decided to divide treatment and management into three stages: time after initial impact, recovery, and prolonged recovery.

RESULTS: In post-concussive athletes, impairments in balance may exist as a result of transmitted force to peripheral and central neural substrates that integrate sensory information and coordinate motor function. Corroborative information, clinical examination, neuropsychological testing, and continual reassessment are means to determine severity of dysfunction and track clinical course and resolution of symptoms. Persistence of symptoms beyond initial impact may require medication trials or adjustments that are tailored to the patient's medical history and/or neurocognitive rehabilitative techniques such as vestibular rehabilitation therapy to prevent progression of neurologic sequelae. Prolonged recovery of more than six months may require neurological consultation.

CONCLUSION: Concussion management and treatment of vestibular and balance impairments in athletes should be assessed in a stepwise manner, from initial impact to resolution of symptoms. If symptoms are prolonged, impaired neuronal mechanisms or irreversible cerebral damage may underlie persistent symptoms and cognitive deficits seen in neurocognitive testing. Management protocols are currently focused on individualized assessment of neurocognitive assessment and comprehensive symptomatic evaluation (Reddy et al., 2008). It is widely accepted that neurocognitive and resolution of concussion-induced symptoms must be resolved prior to returning to sport or play and therefore, the athlete should be reassessed and treated until symptoms resolved.

Keywords: Vestibular and balance dysfunction, concussion, concussed athlete and treatment, vestibular rehabilitation therapy

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1. Introduction

The treatment and management of vestibular and balance dysfunction in concussed athletes requires a multidisciplinary approach and is based on continuous reassessment of the presenting symptoms. The clinical challenge toward managing persistent symptoms of the post-concussive athlete is discerning whether a set of symptoms match diagnostic testing and whether further neurological work up is necessary. Because there are no discrete time boundaries to make such judgment calls, we offer a guide to help with the difficult clinical decisions necessary to treat the post-concussive athlete. Persistence of symptoms after initial impact determines specific management with an overall goal of preventing progression of neurologic dysfunction and sequelae.

2. Concussion, the brain, and balance

Concussion is defined by the constellation of symptoms associated with impact to the head, face, neck or elsewhere in the body such that a mechanical force is transmitted to the brain (McCrory et al., 2009; Sahler et al., 2012). This article is focused on vestibular and balance impairments in athletes sustaining a concussion. It is important to keep in mind, the presence of other symptoms of concussion should be addressed to optimize recovery and to prevent further progression. Neuropsychological deficits as well as other somatic symptoms (eg. headache), impaired sleep-wake cycle, mood symptoms and personality changes are frequently found in athletes with concussion (Reddy et al., 2008; Sarmiento et al., 2010).

Given the location of the vestibular system and its relation to the head and neck, the translated forces associated with a concussion can directly impact the vestibular system and/or supportive brain regions important for balance (Reddy et al., 2008). The vestibular system can be divided into a peripheral component consisting of the semicircular canals, otoliths (utricle and saccule), vestibular ganglia and the vestibular nerve, and a central component consisting of vestibular nuclei, cerebellum, autonomic nervous system, thalamus and cerebral cortex (Guskiewicz, 2001). Because balance is a coordinated process that integrates multimodal sensory information to invoke reflexive and voluntary motor responses, we will address balance systems peripherally to centrally. Moreover, the literature suggests that the majority of post-concussive balance impairments are mediated through dysfunction in the

peripheral vestibular system, particularly the inner ear (Maskell et al., 2006).

Athletes with damage to vestibular or related balance pathways may develop persistent symptoms of balance impairment (Lovell, 2009). Therefore it is critical to thoroughly assess the vestibular system directly via clinical tests and visual assessment, especially in athletes with delayed recovery. The management and treatment may vary depending on the etiology of the vestibular dysfunction (see Table 1). The main supportive neural systems responsible for balance include vestibular integration with voluntary ocular motor coordination, voluntary skeletal motor coordination and proprioception. Higher order sensory information is processed by the vestibular nuclei and coordinates with the ocular motor circuits responsible for the vestibulo-ocular reflex (McCrory et al., 2009). Somatosensory and proprioceptive information is transmitted to the cerebellum via the spino-cerebellar tracts and integrates with the vestibular cochlear system in the inner ear to allow for an integration of body positional information in relation to gravity. Vestibular information is also relayed via the vestibular nuclei to the lateral and medial vestibulo-spinal tracts to elicit voluntary motor control of the lower extremities by way of the vestibular spinal reflex (VSR) and acts to maintain balance, posture, and stability in an environment with gravity. Unfortunately, patients with mixed central and peripheral vestibular dysfunction have slower and incomplete recovery (Brown et al., 2006).

2.1. Overview of cellular pathophysiology of concussion

The principal mechanisms of traumatic brain injury are either direct impact force causing focal damage to the brain (eg. contusion, laceration, or intracranial hemorrhage) or translated forces resulting in diffuse axonal injury or brain swelling (Marshall et al., 2000). Animal models of mild traumatic brain injury (mTBI) have suggested that in cases of concussion or mTBI, it is the acceleration/deceleration that invokes the latter mechanism, insofar as a skull fracture is not involved (Hovda et al., 1999). Although the exact biomechanical events leading to concussion are nearly impossible to ascertain in any one individual, a likely combination of stretching, shearing, compression, or deformation injuries to the neural tissue gives rise to a set of cellular and cerebral blood flow changes that associate with the acute injury and subsequent compensatory repair mechanisms that aim to re-establish cellular homeostasis

Table 1
Vestibular and Related Structural Abnormalities

Type	Clinical Finding	Onset	Treatment	Note
Peripheral				
<i>Vestibular (Inner Ear)</i> Benign Paroxysmal Positional Vertigo (BPPV)	Nystagmus with Dix Hallpike Test	Delayed (weeks to months)	Canalith Repositioning Maneuver	Common with concussion. Good prognosis with treatment; 2-3 times daily, until asymptomatic
Labyrinthine concussion	Vertigo and ataxia worse with initial insult but often progressively improves	Acute, unilateral	Rest, Vestibular suppressants if symptoms persist, Will need to rule out co-existing BPPV, PLF, or Hydrops Surgical repair of ruptured oval or round window.	Usually reversible, resolves in few weeks; If >2 months, may lead to persistent lifelong vestibular symptoms May appear similar to hydrops, but PLF appears acutely, hydrops delayed onset
Perilymphatic Fistula (PLF)	Dizziness with unilateral sensorineuronal hearing loss	Acute		
Post-traumatic meniere syndrome (Hydrops)	Meriere-like symptoms without tinnitus or hearing loss. Fluctuating symptoms lasting minutes to hours.	Delayed (months to years)	Vestibular suppressants; Vestibular rehabilitation	
<i>Head</i> Temporal bone fracture	Severe vertigo and ataxia. Diagnosis confirmed with imaging (CT)	Acute	Vestibular suppressants	Resolution in about 2months
Central				
<i>Neck</i> Cervical vertigo	Neck pain with limited neck range of motion with dizziness and impaired balance	Episodic, last minutes to hours	Analgesics, Cervical soft tissue modalities (massage, soft collar, etc)	Vestibular Rehabilitation indicated for prolonged residual balance impairment
<i>Head</i> Epileptic vertigo	Trauma induced simple or partial complex sensory seizures with associated vertigo, paresthesia, olfactory and gustatory symptoms	Provoked by sensory stimuli	Avoid provocation, Anticonvulsants, Vestibular Rehabilitation	Seizure work-up usually unremarkable.
Migraine associated vertigo	Chronic persistent migraine, days to weeks	Acute	Anti-migraine medications (analgesics, triptans, ergotamines, other)	
Ocular motor abnormalities	Deficits in saccades and impaired upper limb visuomotor function	Acute	Vestibular Rehabilitation	Can be present despite absence of oculomotor or visuomotor deficits on standard clinical examination

Differential diagnosis for persistent vestibular symptoms central and peripheral etiologies of vestibular and related structural impairments that may affect balance. Adapted from (Friedman, 2004). Note: The table above was created by current authors.

(Shaw et al., 2002; Werner et al., 2007). Metabolic changes within the neuron causes amino acid-induced ionic shifts and resulting hyperglycolysis (Bergsneider et al., 1997). Since the cellular energy demand is greater than that supplied by anaerobic metabolism, cellular ATP is depleted and failure of energy-dependent membrane ion pumps gives rise to progressively unregulated membrane depolarization and subsequent release of excitatory neurotransmitters (Werner et al., 2007). Paradoxically, neurovascular changes in the form of vasoconstriction worsen this failure in cellular bioenergetics. Finally, diffuse axonal injury due to initial mechanical forces act concomitantly with cellular and metabolic changes contributing to these disturbances in neurotransmission (Shaw et al., 2002; Willer et al., 2006).

2.2. Neuropsychological impairments

Neuropsychological deficits are frequently found in athletes with concussion (Reddy et al., 2011; Levin et al., 2012; Shrier et al., 2012). To measure deficits in specific domains of cognition, various neuropsychological batteries and instruments have been developed such as the Post-Concussion Assessment and Cognitive Testing (ImpACT) (Echemendia et al., 2012) and the Sideline Concussion Assessment Tool 2 (SCAT2) which includes cognitive and non-cognitive assessment. These tests can be used as a screening tool or as a means of monitoring progression or resolution of sustained impairments. Ideally, these neurocognitive diagnostic tools provide an objective measure of even subtle cognitive dysfunction in a concussed athlete (Lovell, 2009).

The ImpACT is a computerized concussion evaluation system and provides an expedient way of determining gross cognitive deficits. The SCAT2 is more widely accepted in the literature as a comprehensive post-concussive assessment including neuropsychological test. Although this tool has not yet been validated as a whole, individual components have been validated, such as the SAC, the Balance Error Scoring System (BESS), the Maddox Score, Glasgow Coma Scale, and the Concussion Symptom Inventory (Echlin et al., 2010). Regardless of test administered, it is imperative that a neurocognitive assessment be included in the post-concussive period.

2.3. The temporality of concussive symptoms

It is unclear exactly when the onset of symptoms arises after sustaining a concussion. Onset of symptoms

can range between 3–24 hours and may relate to pre-morbid structural and functional vulnerabilities unique to the individual and circumstances of the impact. In the sports concussion literature, symptoms typically resolve within 5–10 days, however, beyond this time frame, persistent symptoms may be related to residual neuronal facilitation abnormalities to peripheral or central vestibular-balance systems (McCrea et al., 2003). It is therefore useful to divide treatment and management into 3 major categories: time after initial impact, recovery, and prolonged recovery.

The first 24 hours after initial impact is most important in the detection of medical emergencies and determination of severity of concussion-related symptoms, such as those affecting balance. Cognitive and physical rest is essential in hastening recovery, however the recovery period may last up to 6 months (McCrory et al., 2009). Reassessment allows for the identification of persistence of vestibular and balance symptoms that may suggest underlying structural damage and treatment is aimed at rehabilitating vestibular and non-vestibular neurological functioning. The prolonged recovery period may last more than 6 months; persistent symptoms may reflect irreversible structural brain damage. Rehabilitative therapy and symptomatic treatment with medication helps to provide benefits to recovery and some functional improvement (Crooks et al., 2007).

3. Initial impact

3.1. Assessment

Initial presentation of symptoms occur within the first 24 hours after impact and the goal of assessment is to determine severity of impairments in order to prevent progression and allow healing. The majority (80–90%) of concussions resolve in a short (5–10 day) period, although the recovery time frame may be longer in children and adolescents (Alsalaheen et al., 2010). During this time period, it is critical to triage and identify trauma to the spinal cord, neck, head or face that may become medical emergencies if left untreated. This is most commonly evaluated on the “side line” during a game or sporting event.

Recognition of concussion during the sideline assessment can be challenging in that the symptoms are usually non-specific or an athlete may not be reporting symptoms due to lack of recognition or intentionally to return to play (McCrea et al., 2003). Most common

symptoms of post-concussive vestibular and balance dysfunction include dizziness (55–78%), impaired balance (43–56%), and blurred vision or diplopia (49%) (Lovell, 2009). When presented early in the post-concussive period, these symptoms are believed to largely reflect neurometabolic disturbances rather than a structural injury (McCrory et al., 2009) and may represent non-specific brain deficits, such as headache and dizziness, or more localizable neurological impairments such as blurry vision or diplopia.

3.2. Goals of treatment

The goal of treatment is to allow healing via complete withdrawal from physical and cognitive activities (ie. rest) for a period of 24 hrs (McCrory et al., 2009). Given that the presence of vestibular and balance symptoms early after concussion represents subtle diffuse neuronal injury, rest allows restoration of brain metabolism and may reduce the risk of further complications, such as, prolonged post-concussive syndrome, second impact syndrome, which is potentially fatal from significant progressive cerebral edema may lead to death (Willer et al., 2006) or long-term neurological deterioration sequela of chronic traumatic encephalopathy (CTE) (Saulle & Greenwald, 2012). The persistence of symptoms beyond 2 weeks may be suggestive of more severe brain injury or compounded injury such as those in athletes who have sustained multiple concussions and may require further neurological evaluation (Reddy et al., 2008).

3.3. Medication

Although there is no good evidence for medications to treat post-concussive vestibular and balance impairments, caution must be made regarding use of medications that alter cognition or mood states to prevent further neurologic and/or psychiatric manifestations. Particular vulnerable populations include adolescents who may be taking stimulant medication for ADHD or adolescents/adults who are on psychiatric medications for anxiety, depression or other psychiatric disorders. Inversely, athletes who newly develop psychiatric symptomatology or neurocognitive deficits as a result of concussion may benefit from antidepressant medication or methylphenidate respectively (Silver et al., 2009). There are no clear guidelines or evidence based studies regarding optimal time to start oral medication. However, some authors recommend abstaining from pharmacological intervention in the first 3 weeks (Reddy et al., 2008). In particular, meclizine should be

avoided due to potential for decline in cognitive functioning (Black et al., 2000). Aspirin should be avoided during this time period due to potential for inducing or exacerbating intracranial hemorrhage (Willer et al., 2006). Headache may be treated with Tylenol and NSAIDs (Crooks et al., 2007). NSAIDs has potential risk of increase bleeding due to known antiplatelet properties, caution must be taken. Non-pharmacological, conservative management is recommended initially, consistent good sleep wake pattern, hydration, avoid triggers and stress (Blume et al., 2011). Athletes should be instructed to avoid alcohol and illicit drugs due to risk for psychiatric and cognitive disturbances (Guskiewicz et al., 2004).

3.4. Return to play

The recommended duration of time required before return to play after an initial suspected concussion is controversial. Recent guidelines recommend that all athletes who demonstrate signs or symptoms of concussion be withheld from participation and seen by a physician before returning to play. The general concussion consensus policies from professional organizations, the NFL and NCAA (Leddy et al., 2012) prohibit athletes suspected of having a concussion from returning to the game on the same day after evaluated and assessed by a medical profession. The general guidelines for high school and non-professional athletes are even more stringent. The high school and non-professional athletes should be restricted from returning to play even if suspected of a possible concussion regardless of severity of injury (Leddy et al., 2012). These athletes suspected of concussion should have a comprehensive sports related concussion assessment by medical professional including detailed history, physical exam and sequential monitoring until symptoms resolve. If any symptoms occur during assessment after strenuous activity, the athlete must wait at least 24 hours prior to another assessment.

3.5. Prevention

A multidisciplinary team, often comprised of the sideline medical team, primary care/referring physician, coach(es), parent/guardian(s), teachers, and athletic trainers, is essential for continued monitoring and reassessment (Sabini et al., 2010). Assessment and reassessment of vestibular and balance dysfunction is important in determining whether a more extensive neurological work up is necessary. This may include brain

imaging and neurocognitive testing to rule out a structural lesion or more severe cerebral dysfunction. Often times, the athlete may not be aware he/she sustained a prior concussion. This may require communication with other members of the multidisciplinary team.

Athlete education about concussion has been shown to help with treatment compliance and adherence to follow up (Blume et al., 2011). A common athlete concern is the amount of time he/she needs to wait before playing. Therefore, rapport building with continued conversations between athlete and physician may allay fears regarding the expected time frame required before returning to play. Lastly, although an athlete may be symptom free from balance or gait dysfunction, cognitive impairments to attention and memory, may still be impaired.

4. Recovery

4.1. Assessment

Even in situations when an athlete sustains an uncomplicated concussion, exact time of recovery varies and the duration of symptoms are unpredictable (McCrea et al., 2003; Peterson et al., 2003). Athletes who are compliant with cognitive and physical rest have more favorable outcomes. However, in an athlete whose symptoms persist for longer than the expected time course, it is critical that the athlete is reassessed and reevaluated by a physician, preferably a concussion specialist, physiatrist or neurologist.

The assessment should include detailed history, such as when the concussion occurred, initial presenting symptoms, any new or existing medications, prior history of concussions, or any past imaging or treatment. Clinical diagnostic tools are used to determine the severity of the symptoms and are most important at this stage because of the need to identify potential structural lesions. Clinical tests such as the Balance Error Scoring System (BESS) test, computerized dynamic posturography (CDP) which includes balance tests, the Sensory Organization Test, and visual tracking technologies (Lovell, 2009) help to uncover latent impairments by provoking and unmasking specific impairments in neural systems that subservise balance. The role of neurocognitive testing in the reassessment stage, allows clinicians to identify patients at risk of persistent symptoms and disability.

Specific pathologies as a result of a concussion may affect the structural components of the vestibular sys-

tem (inner ear), and associated structures head and neck (See Table 1). The possible causes of dizziness, impaired balance or vertigo include benign paroxysmal positional vertigo (BPPV), labyrinthine concussion, perilymphatic fistula (PLF), post-traumatic Meniere Syndrome (hydrops), temporal bone fracture, cervical (cervicogenic) vertigo, epileptic vertigo, migraine associated vertigo and ocular motor abnormalities. BPPV is often associated with prolonged symptomatic vertigo. Diagnostically assessment and treatment are straightforward. BPPV is usually the first related vestibular pathology to rule out ((Friedman et al., 2004). Subsequently, neck related problems may mimic vestibular symptoms clinically. Cervicogenic vertigo or dizziness is described as a non-specific sensation of altered orientation in space, disequilibrium resulting from abnormal afferent activity from the neck. This is often associated with “whiplash” type injury, cervical flexion and extension (Sabini et al., 2010). Patient may appear with balance impairments, postural instability or motion sense abnormalities. A key distinctive feature to differentiate cervicogenic dizziness from vestibular dizziness is the association of neck pain and possible limited neck range of motion (Ernst et al., 2005).

4.2. Goal of treatment

If the symptoms persist despite rest and preventative measures, referral to vestibular therapy is highly recommended. Persons with concussion may show residual and persistent symptoms that may suggest underlying structural damage in the brain. A subset of athletes with mild TBI shows persistent postural instability (Blume et al., 2011). Balance testing is helpful to establish the presence of concussion as well as track recovery (Reddy et al., 2008). Disordered balance as measured by sub-optimal balance scores after concussion may be caused by central deficits within the vestibular system or more peripheral issues such as persistent posttraumatic headache (Guskiewicz, 2001). Athletes with vestibular dysfunction appear to have increased reliance on visual inputs for balance and less able to use vestibular orienting information (Willer et al., 2006).

The primary function of the vestibular system is to maintain the eyes fixed on a stationary target in the presence of head and body movement. In addition maintain balance when other sensory inputs, such as visual or somatosensory, signals are integrated (Guskiewicz, 2001). In cases of post-concussive vestibular dysfunction or central balance system disorders, Vestibular Rehabilitation Therapy (VRT) has been

shown to improve persistent dizziness, gaze instability and balance dysfunction (Alsalaheen et al., 2010). The theoretical therapeutic mechanisms are based on reinforcement of already existing neural systems such that adaptation and compensation (habituation) through neural plasticity in the brain.

Self-reported symptoms (cognitive, mood, sleep and somatic) must be addressed by the vestibular therapist and tailors specific therapies for the individual. Other goals of treatment include improvement of gaze stability and eye-head coordination and to provide a graded exertional program with progression to sports specific activities.

4.3. Modalities of vestibular therapy

Providing symptomatic relief for vestibular symptoms has been the mainstay of treatment, however there is limited evidence to provide clear guidelines. Although cognitive and physical rest in the initial time period following concussion is critical for cerebral functional restoration, pharmacological treatment and/or surgical intervention may improve the clinical course if a central or peripheral vestibular syndrome is diagnosed. In these situations, further clinical assessment is needed to better determine if and when to start pharmacologic treatment or VRT alone or initiate medication in conjunction with VRT (Crooks et al., 2007).

Restoration of concussion related vertigo or impaired balance requires basic understanding of the complex interaction between the vestibular system, visual system, and the somatosensory system with particular focus on proprioception (Hall et al., 2009). The concept of VRT is to help restore function by inducing neural plasticity within existing healthy neural systems in order to allow for adaptation and compensation for functional deficits as a result of concussion. The three main compensatory mechanisms used in VRT (see Table 2) are habituation, adaption and substitution (Crooks et al., 2007; Han et al., 2011) These different mechanisms are achieved by using specific cognitive and sensorimotor exercise protocols to increase sensitivity and restore functional symmetry. This results in an improvement in vestibuloocular control, an increase in the gain of the vestibuloocular reflex (McCrary et al., 2009), better postural strategies, and increased levels of motor control for movement.

4.3.1. Habituation exercises

The goal of these exercises involves repeated exposure to a provoking stimuli or head movements, as a

result the pathologic response to the stimuli is reduced. For example, under the guidance of vestibular specialists, selected movements induces symptoms such dizziness and vertigo. The patient repeats these motions until the symptoms resolve.

4.3.2. Adaptation exercises and vestibular ocular reflex

The goal of these exercises is to assist the central nervous system to adapt to a change or loss in vestibular system input. Adaptation uses functioning vestibular system to adapt to sensory neuronal responses. The focus is on the retinal slip during head movement, verticle or horizontal (McCrary et al., 2009). This helps to reduce symptoms and improve functional neuronal output produces stable gaze and improved balance. Head movement is performed while maintaining focus the target. The target is either moving (VOR 1) or stationary (VOR 2). For example, in VORx1, the patient moves the head to both sides (yaw or pitch) while keeping their eyes fixed on a stationary target. In VORx2, the head and the hand holding the target are moving in opposite directions with the eyes fixated on the target. Progressive adaptation exercise include increasing the velocity of head movement, increasing the velocity of target movement, target placement with distracting visual pattern in the background and focus on maintaining proper posture.

4.3.3. Substitution exercises

The goal of these exercises is to promote alternative strategies, use of other eye movements, for impaired vestibular function. These exercises take advantage of saccade to replace the slow phase of the VOR component. Secondly, enhancing the smooth pursuit eye movement to help maintain gaze stability. Facilitating preprogrammed eye movement using large eye movement on a target before the head turns to the target is another strategy. These exercises are instructed by a vestibular therapy specialist.

4.4. Medication

There is limited evidence in the efficacy of pharmacotherapy successfully treating prolonged post concussive vertigo and balance impairment. During the recovery period, medications are targeted at reducing specific persistent symptoms and help improve daily function (Willer et al., 2006). When considering pharmacological intervention many factors are considered, age of athlete, side effect profile and goal and time

Table 2
Comprehensive Vestibular Rehabilitation Therapy

Type and purpose	Theoretical description	Example
<i>Canalith repositioning maneuver</i> (Curative for BPPV)	Diagnostic and therapeutic maneuvers simple and effective for BPPV	Repetitive head movements
<i>Habituation</i> (For impaired motion sensitivity)	Provocation of stimuli induces symptoms; enhances vestibular compensation; requires repetition; intensity of exercise proportional to severity of symptoms	Head position or movement inducing dizziness or vertigo
<i>Adaptation</i> (For impairments in convergence)	Enhancement of intact vestibular circuits to compensate for loss of function within same system; Use of retinal slip during head movement (verticle or horizontal)	Instructed to move head while maintaining focus on moving (VOR1) or stationary (VOR2) target. Degree of difficulty of exercise increased progressively
<i>Substitution</i> (For major vestibular impairment)	Replacement of deficient vestibular system by enhancement of ocular systems	Exercises that facilitate preprogrammed eye movements to scan field and detect target in order to prompt head and neck movements to override vestibular-ocular reflex
<i>Balance exercises</i> (To enhance supportive balance systems)	Positional Exercises Proprioceptive Neuromuscular Facilitation	<i>Static balance</i> = alternating visual and somatosensory input, with change of support base-> Wide vs Narrow. <i>Dynamic balance</i> = higher level of challenge. Head turning while walking; quick head turn (right or left) while walking; incorporating task while walking-> tossing an object or cognitive task while walking
<i>Aerobic exercise</i> (To strengthen balance via muscle conditioning)	Promotes strengthening of muscle groups to help improve balance reaction time	Progressive walking exercise with increase time and intensity. Advance gradually to sustained aerobic activity.

Summary of general concept of vestibular therapy exercises. Adapted from (Hall et al., 2009; Han et al., 2011). *Note:* The table above was created by current authors.

course of medication. Further details are outlined in pharmacology section.

4.5. Return to play

According to the Third International Conference on Concussion in 2008, there needs to be a gradual return to play. This protocol is a six-step program that increases an athlete's physical demands by progressing from no activity to full return to play. During the program, each of the steps is 24 hours in length, and the athlete must remain asymptomatic during that stage, otherwise they are to return to the previous step.

There are many useful tools for monitoring symptoms and cognition to help aid in the decision on when the athlete is ready to return to full participation. For example, the ImPACT has been shown to be a reliable concussion and post-concussion instrument with 81.9% sensitivity, 89.4% specificity in detecting decline

of cognitive function, especially for processing speed and reaction time (Iverson, 2005; Echemendia et al., 2012). Limitations of the ImPACT, not readily available and best used to compare with asymptomatic baseline. However the test is able to help monitoring progression over time (Ma et al., 2012).

While there is no single clinical test to establish full recovery, best current practice models require that patients are symptom-free at rest, have normal neurocognitive and balance testing, and experience no return of abnormalities when full cognitive and physical activity are introduced (Lovell et al., 2004; McCrory et al., 2009)

4.6. Persistence of symptoms

If symptoms of post-concussive vestibular and balance impairments persist, further evaluation of etiology is important to help develop an individualized vestibular

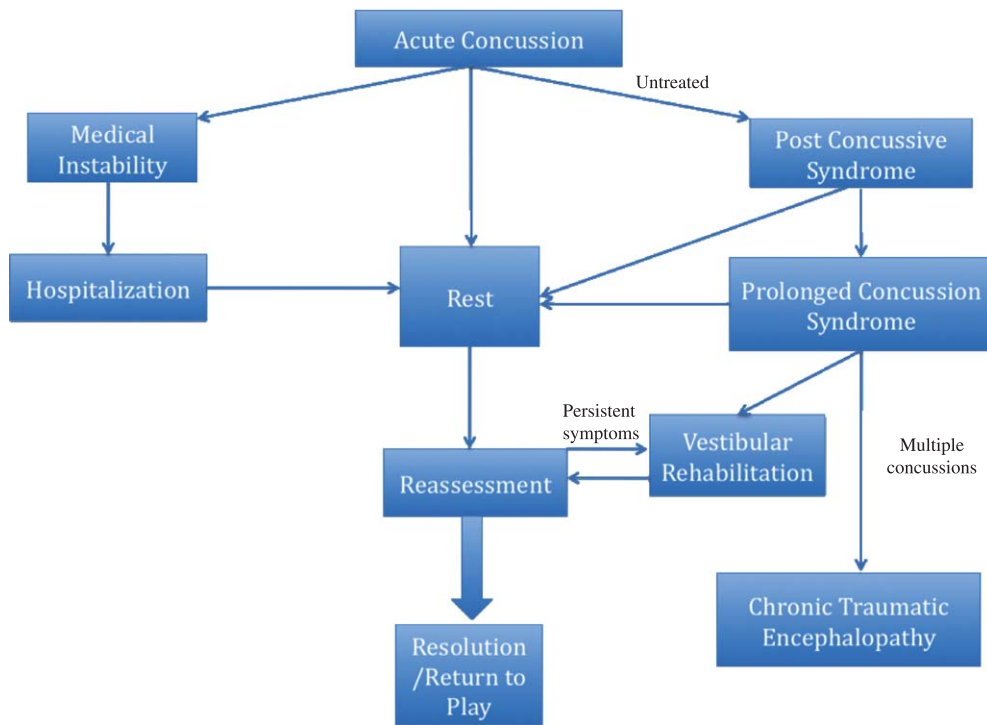


Fig. 1. Overview of Treatment for Vestibular and Balance Impairments in post concussed athletes. *Note:* This figure was created by current authors to capture the overall general algorithm of clinical management.

rehabilitation program. Table 1 shows a list of common etiologies with treatment option.

5. Prolonged recovery

5.1. Assessment

Athletes who continue to experience symptoms beyond 6 months are considered to be at high risk for structural brain damage as well as risk for repeated concussions as a result of the persistence of functional impairments (Ma et al., 2012). Previously undiagnosed neurocognitive deficits or other neurologic and/or psychiatric impairments tend to present after continued reassessments as the athlete, family members, or other people close to the athlete may relay persistence forgetfulness, decreased concentration, or behavioral and personality changes. These symptoms are most alarming because they may indicate the athlete has succumbed to prolonged concussion syndrome.

At this stage, neuropsychological testing is paramount to ascertain which domains of cognitive functioning have been compromised. Head imaging such as CT can show slow hemorrhage or bone struc-

tural deformations missed during prior assessments. Structural MRI may show subtle and non-specific changes such as diffuse axonal injury (Moen et al., 2012), or DTI may show decreased integrity to specific white matter tracts (Yeo et al., 2012). Although functional MRI has been predominantly used in the research setting, this methodology has shown promise in detecting more subtle issues with neural functioning and patterns of dysfunctional connectivity in the brain (Benson et al., 2012). Altogether, these imaging modalities, neurocognitive testing, and the previously mentioned diagnostic testing may be used to track progression of brain damage or progression of recovery.

5.2. Goal of treatment

The rehabilitation program provided graded exposure to head and body movements, anxiety management and coping strategies and education. Concern is that the athlete is at risk of subsequent concussion with a mild or minimal blunt trauma. Studies have shown benefit of vestibular therapy even 6 months to 1 year post-concussion (Alsalaheen et al., 2010).

5.3. Disqualification from play

At this point, a multidisciplinary decision to disqualify the athlete from the season or athletic career must be discussed. There are no clear guidelines. The literature suggests once an athlete sustains a concussion, the athlete is at greater risk of subsequent concussion within even the same season, at about 3fold increase (Guskiewicz et al., 2000). The general consensus is each athlete should be thoroughly evaluated, with appropriate management and treatment of the initial onset of a concussion to help prevent potentially fatal functional limitations and sequela, as mentioned earlier (Guskiewicz et al., 2004). As more research focuses on better understanding of the pathophysiology and earlier diagnosis of potential CTE (Saulle et al., 2012). This will hopefully provide more definitive guideline.

6. Conclusion

Concussion management and treatment of vestibular and balance impairments in athletes should be assessed in a stepwise manner, from initial impact to resolution of symptoms.

Guidelines require concussed athletes asymptomatic at rest and with activity prior to returning to sport. Vestibular and impaired balance postconcussive symptoms will often spontaneously recovery in about 80% of athletes with an uncomplicated concussion. Resolution of symptoms are thought to correlate with resolution of impaired metabolic pathways. If symptoms are prolonged, the athlete should be reassessed to determine if underlying vestibular structural or related structural impairments are present. In addition to possible prolonged or progressively neurometabolic impairment. Clinical reassessment of symptoms are needed until symptoms completely resolve. Management protocols are now focused on individualized assessment of neurocognitive assessment and comprehensive symptomatic evaluation. (Reddy et al., 2008). The onset of symptoms, severity and potential progression of symptoms various among each athlete. Evidence shows early comprehensive vestibular rehabilitation therapy provides improvement of symptoms and recovery time. Education is critical to help the athlete understand the signs and symptoms of vestibular and balance associate concussion, the appropriate measures to prevent progression or worsening of symptom and even delay recovery. Limited medication uses for vestibular symptoms in the first few weeks. However, symptomatic

treatment of headache is recommended with caution. Further studies are needed to create evidence based standardized management for concussed athlete with vestibular and balance impairments.

References

- Alsalaheen, B. A., Mucha, A., Morris, L. O., Whitney, S. L., Furman, J. M., Camiolo-Reddy, C. E., & Sparto, P. J. (2010). Vestibular rehabilitation for dizziness and balance disorders after concussion. *J Neurol Phys Ther*, *34*(2), 87-93.
- Benson, R. R., Gattu, R., Sewick, B., Kou, Z., Zakariah, N., Cavanaugh, J. M., & Haacke, E. M. (2012). Detection of hemorrhagic and axonal pathology in mild traumatic brain injury using advanced MRI: Implications for neurorehabilitation. *NeuroRehabilitation*, *31*(3), 261-279.
- Bergsneider, M., Hovda, D. A., Shalmon, E., Kelly, D. F., Vespa, P. M., Martin, N. A., & Becker, D. P. (1997). Cerebral hyperglycolysis following severe traumatic brain injury in humans: A positron emission tomography study. *J Neurosurg*, *86*(2), 241-251.
- Black, F. O., Angel, C. R., Pesznecker, S. C., & Gianna, C. (2000). Outcome analysis of individualized vestibular rehabilitation protocols. *Am J Otol*, *21*(4), 543-551.
- Blume, H. K., Lucas, S., & Bell, K. R. (2011). Subacute concussion-related symptoms in youth. Review. *Phys Med Rehabil Clin N Am*, *22*(4), 665-681, viii-ix.
- Brown, K. E., Whitney, S. L., Marchetti, G. F., Wrisley, D. M., & Furman, J. M. (2006). Physical therapy for central vestibular dysfunction. *Arch Phys Med Rehabil*, *87*(1), 76-81.
- Crooks, C. Y., Zumsteg, J. M., & Bell, K. R. (2007). Traumatic brain injury: A review of practice management and recent advances. Research Support, N.I.H., Extramural.
- Echemendia, R. J., Bruce, J. M., Bailey, C. M., Sanders, J. F., Arnett, P., & Vargas, G. (2012). The utility of post-concussion neuropsychological data in identifying cognitive change following sports-related MTBI in the absence of baseline data. *Clin Neuropsychol*, *26*(7), 1077-1091.
- Ernst, A., Basta, D., Seidl, R. O., Todt, I., Scherer, H., & Clarke, A. (2005). Management of posttraumatic vertigo. *Otolaryngol Head Neck Surg*, *132*(4), 554-558.
- Friedman, J. M. (2004). Post-traumatic vertigo. Review. *Med Health R I*, *87*(10), 296-300.
- Guskiewicz, K. M., Weaver, N. L., Padua, D. A., & Garrett, W. E. Jr. (2000). Epidemiology of concussion in collegiate and high school football players. *Am J Sports Med*, *28*(5), 643-650.
- Guskiewicz, K. M. (2001). Postural stability assessment following concussion: One piece of the puzzle. *Clin J Sport Med*, *11*(3), 182-189.
- Guskiewicz, K. M., Bruce, S. L., Cantu, R. C., Ferrara, M. S., Kelly, J. P., McCrea, M., & Valovich McLeod, T. C. (2004). National athletic trainers' association position statement: Management of sport-related concussion. *J Athl Train*, *39*(3), 280-297.
- Hall, C. D., & Cox, L. C. (2009). The role of vestibular rehabilitation in the balance disorder patient. *Otolaryngol Clin North Am*, *42*(1), 161-169.
- Han, B. I., Song, H. S., & Kim, J. S. (2011). Vestibular rehabilitation therapy: Review of indications, mechanisms, and key exercises. *J Clin Neurol*, *7*(4), 184-196.

- Hovda, D. A., Prins, M., Becker, D. P., Lee, S., Bergsneider, M., & Martin, N. A. (1999). Neurobiology of concussion. In L. M. Bales J. E., & Maroon J. C. (Eds.), *Sports Related Concussion*. (pp. 12-51). St. Louis, Quality Medical Publishing, Inc.
- Iverson, G. L., Lovell, M. R., & Collins, M. W. (2005). Validity of ImPACT for measuring processing speed following sports-related concussion. *J Clin Exp Neuropsychol*, 27(6), 683-689.
- Leddy, J. J., Sandhu, H., Sodhi, V., Baker, J. G., & Willer, B. (2012). Rehabilitation of concussion and post-concussion syndrome. *Sports Health*, 4(2), 147-154.
- Levin, H., Li, X., McCauley, S. R., Hanten, G., Wilde, E. A., & Swank, P. R. (2012). Neuropsychological outcome of MTBI: A principal component analysis approach. *J Neurotrauma*.
- Lovell, M. (2009). The management of sports-related concussion: Current status and future trends. *Clinics in Sports Medicine*, 28(1), 95-111.
- Lovell, M., Collins, M., & Bradley, J. (2004). Return to play following sports-related concussion. *Clin Sports Med*, 23(3), 421-441, ix.
- Ma, R., Miller, C. D., Hogan, M. V., Diduch, B. K., Carson, E. W., & Miller, M. D. (2012). Sports-related concussion: Assessment and management. *J Bone Joint Surg Am*, 94(17), 1618-1627.
- Marshall, L. F. (2000). Head injury: Recent past, present, and future. *Neurosurgery*, 47(3), 546-561.
- Maskell, F., Chiarelli, P., & Isles, R. (2006). Dizziness after traumatic brain injury: Overview and measurement in the clinical setting. *Brain Inj*, 20(3), 293-305.
- McCrea, M., Guskiewicz, K. M., Marshall, S. W., Barr, W., Randolph, C., Cantu, R. C., & Kelly, J. P. (2003). Acute effects and recovery time following concussion in collegiate football players: The NCAA Concussion Study. Research Support, Non-U.S. Gov't. *JAMA*, 290(19), 2556-2563.
- McCrary, P., Meeuwisse, W., Johnston, K., Dvorak, J., Aubry, M., Molloy, M., & Cantu, R. (2009). Consensus statement on concussion in sport - The 3rd international conference on concussion in sport held in Zurich, November 2008. *PM R*, 1(5), 406-420.
- Moen, K. G., Skandsen, T., Folvik, M., Brezova, V., Kvistad, K. A., Rydland, J., & Vik, A. (2012). A longitudinal MRI study of traumatic axonal injury in patients with moderate and severe traumatic brain injury. *J Neurol Neurosurg Psychiatry*.
- Peterson, C. L., Ferrara, M. S., Mrazik, M., Piland, S., & Elliott, R. (2003). Evaluation of neuropsychological domain scores and postural stability following cerebral concussion in sports. *Clin J Sport Med*, 13(4), 230-237.
- Reddy, C. C. (2011). Postconcussion syndrome: A physiatrist's approach. *PM R*, 3(10 Suppl 2), S396-S405.
- Reddy, C. C., Collins, M. W., & Gioia, G. A. (2008). Adolescent sports concussion. *Phys Med Rehabil Clin N Am*, 19(2), 247-269.
- Sabini, R. C., & Reddy, C. C. (2010). Concussion management and treatment considerations in the adolescent population. *Phys Sportsmed*, 38(1), 139-146.
- Sahler, C. S., & Greenwald, B. D. (2012). Traumatic brain injury in sports: A review. *Rehabil Res Pract*, 2012, 659652.
- Sarmiento, K., Mitchko, J., Klein, C., & Wong, S. (2010). Evaluation of the centers for disease control and prevention's concussion initiative for high school coaches: Heads up: Concussion in high school sports". *J Sch Health*, 80(3), 112-118.
- Saulle, M., & Greenwald, B. D. (2012). Chronic traumatic encephalopathy: A review. *Rehabil Res Pract*, 2012, 816069.
- Shaw, N. A. (2002). The neurophysiology of concussion. *Prog Neurol*, 67(4), 281-344.
- Shrier, I. (2012). Neuropsychological testing and concussions: A reasoned approach. *Clin J Sport Med*, 22(3), 211-213.
- Silver, J. M., McAllister, T. W., & Arciniegas, D. B. (2009). Depression and cognitive complaints following mild traumatic brain injury. *Am J Psychiatry*, 166(6), 653-661.
- Werner, C., & Engelhard, K. (2007). Pathophysiology of traumatic brain injury. *Br J Anaesth*, 99(1), 4-9.
- Willer, B., & Leddy, J. J. (2006). Management of concussion and post-concussion syndrome. *Curr Treat Options Neurol*, 8(5), 415-426.
- Yeo, S. S., Chang, M. C., Kim, S. H., Son, S. M., & Jang, S. H. (2012). Neural connection between injured cingulum and pedunculo-pontine nucleus in a patient with traumatic brain injury. *NeuroRehabilitation*, 31(2), 143-146.

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