Vestibular rehabilitation following mild traumatic brain injury

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Abstract
INTRODUCTION: Vertigo, dizziness, and imbalance are a symptom complex that is commonly found following concussion. Early metabolic changes following concussion may lead to worsening of the injury and symptoms in individuals not properly managed from the outset. When symptoms do not recover spontaneously, skilled vestibular rehabilitation can be an effective modality in an attempt to normalize the individual’s vestibular responses.
PURPOSE: The purpose of this review is to appraise the current and accepted methods available to the skilled clinician in quantifying and treating vestibular dysfunction following concussion. Incidence and prognostic indicators will be reviewed along with common barriers to recovery.
SUMMARY: Vestibular Rehabilitation following concussion utilizes similar tools and techniques employed when treating those solely with peripheral pathology. The clinician must not only have a solid understanding of when and why certain exercises are required, but also be willing to accept that less exercise may be indicated in this population. As injury to the system following mild traumatic brain injury can include both peripheral and central structures, the duration of therapy and the time to recovery may be prolonged. Co-morbidities including cognitive and behavioral issues, visual-perceptual dysfunction, metabolic dysfunction, and autonomic dysfunction may hamper the effectiveness of the traditional Vestibular Rehabilitation approach. As successful treatment does not occur in a vacuum, working closely with other disciplines well versed in treating these co-morbid issues will help the individual to obtain optimal recovery.
CONCLUSION: Vestibular Rehabilitation is an effective modality for managing dizziness, vertigo, and imbalance following concussion. Careful consideration of the acuity of the injury, along with effective management of co-morbid conditions will optimize the result.

Keywords: Vestibular rehabilitation, traumatic brain injury, concussion

1. Introduction
Vertigo, dizziness, and imbalance are common symptoms after concussion and mild traumatic brain injury (mTBI) and patients with persistent symptoms can benefit from vestibular rehabilitation (Gurr, 2001; Alsalaheen et al., 2010; Gottshall, 2010). Vestibular rehabilitation involves the use of specific, targeted exercises in order to decrease dizziness and vertigo and improve balance and overall function of the patient. Oftentimes it requires a skilled therapist to prescribe and administer the appropriate exercises to alleviate the patient of their symptoms. It is important to understand the different treatment approaches, as well as the concepts underlying them, in order to implement an effective treatment plan and ultimately improve the function of the patient.
It is important to understand the physiology of mTBI and concussion when rehabilitating these patients. The brain undergoes a series of neurometabolic changes following even mild head trauma and it can take weeks for the brain to regain homeostasis (Giza, 2001). Acutely, trauma to the head causes a sudden increase glucose metabolism as well as a decrease in cerebral blood flow. The result is a mismatch in energy supply and demand. The hypermetabolic state of requiring more energy in order to restore homeostasis lasts for up to 24 hours and is followed by a period of reduced glucose metabolism which can last as long as 1 month (Giza, 2001). Exercise and other stimulation can worsen this mismatch of energy and possibly lead to further damage if performed within this time period (Giza, 2001).

There are two main clinical presentations that we see in the mTBI population: patients that are properly diagnosed and receive the appropriate care following their injury and those that do not. Unfortunately, too often we see patients whose brain injuries were not recognized or managed correctly at the time of their injury and present with chronic symptoms of vertigo, dizziness, and imbalance. Knowing the physiology of TBI, overstimulation too soon after the injury can lead to further damage (Bergsneider, 2000).

2. Patient history

A thorough history of the patient’s injury including the care they received after the insult as well as their pre-morbid health and current subjective symptoms are very important in directing your evaluation, treatment plan, and plan of care. These factors can also assist in making referrals to other health care providers, as a multidisciplinary approach is often necessary for positive outcomes.

There are several factors that can help predict outcomes in this patient population. These factors can be broken down into three categories: pre-injury factors, injury related factors, and post-injury co-existence (Ponsford, 2012).

2.1. Pre-injury factors

2.1.1. Age

There is ongoing debate in the literature regarding age as related to recovery from TBI with studies showing age is both both negative and positive predictors of outcomes post mTBI. Current literature seems to indicate younger age being a negative predictor for recovery post mTBI. It is thought that children and adolescents are more vulnerable to effects of concussion as they can have diffuse and prolonged cerebral swelling after mTBI, which increases their sensitivity to glutamate, which is released acutely at the time of the injury. This increases their risk for secondary injury, second impact syndrome and possibly accounts for delayed recovery in younger athletes (Reddy, 2009).

2.1.2. Gender

Observational studies of humans generally show poorer outcomes for females as compared to males following mTBI, especially during the reproductive years (Bazarian, 2010). This is possibly due to a disruption of the hormones estrogen and progesterone (Bazarian, 2010). Studies involving animal models actually suggest the opposite, that females have better outcomes than males, and the reason for this inconsistency is not clear.

2.1.3. Concussion history

Prior concussion injury has been shown to be a negative predictor for recovery post concussion in high school male athletes (Reddy, 2009; Lau, 2011; Ponsford, 2012). It has been shown that in this population, athletes with 3 or more concussions had more severe on field symptoms of concussion, were more likely to have headaches at baseline, more vulnerable to subsequent injury and were 3 times more likely to sustain an additional injury (Reddy, 2009).

2.1.4. Other pre-existing factors

Patients with history of learning disability, lower education, and pre-injury psychiatric disability all had protracted recovery post mTBI (Lau, 2011; Ponsford, 2012).

2.2. Injury related factors

2.2.1. Amnesia/LOC

Loss of consciousness (LOC) does not predict post concussion syndrome (PCS), but athletes with post-traumatic amnesia had significantly more symptoms, longer duration of symptoms and more impairment on neurocognitive testing (Reddy, 2009).

2.2.2. Dizziness

The presence of dizziness, nausea and headaches in the ER after mTBI is strongly associated with severity of complaints at 6 months post injury (De Kruijff, 2002). It has also been found that dizziness at 2 weeks
post injury was the single predictor of persistent PCS in mTBI (Yang, 2009).

2.2.3. **Behavioral**

Hou et al., found that all or nothing behavior after mTBI was the most important predictor of PCS at 3 months. A negative perception of mTBI at 6 months was a positive predictor of PCS (Hou, 2012). It has also been shown that over exerters post mTBI predicts prolonged PCS (Reddy, 2009). This is likely due to the metabolic changes that the brain undergoes and the negative impact of overstimulation as discussed above.

2.2.4. **Headaches/migraines**

Post-traumatic headaches occur in 86% of post concussion athletes. High school athletes who had headaches 7 days after concussion had a larger number of post-concussive symptoms. Athletes with migraine symptoms (light and noise sensitivity, nausea and vomiting) have increased neurocognitive deficits then athletes with PCS with no post traumatic migraines. Post traumatic migrainous symptoms may have a poorer outcome then post traumatic headaches alone (Reddy 2009).

2.2.5. **Post injury co-existence**

The presence of post injury anxiety, depression, pain, post traumatic stress and litigation may contribute to the symptoms experienced by patients with protracted symptoms after mTBI (Ponsford).

3. **Subjective measures**

There are several self-report measures that can be useful when evaluating patients with vestibular symptoms associated with mTBI. They address a variety of symptoms and the impact these symptoms have on the patient’s daily life. As there is increased pressure from insurance companies for justification for treatment, these scales can give useful information regarding disability and quality of life and can also assist in setting goals that are functionally based. Among the many different self-report measures are the Dizziness Handicap Inventory (DHI), the Activities-specific Balance Confidence Scale (ABC), the Post-Concussion Symptom Scale (PCSS), and the Visual Vertigo Scale (VVS). Additionally, the DHI and the ABC have been shown to record change over time and can be used as an outcome measure (Whitney, 2011).

The DHI is a 25-item questionnaire that measures self-perceived disability due to dizziness and unsteadiness (Jacobson, 1990). The scale is scored from 0 to 100 with a higher score indicating a greater perceived level of disability. A change of 18 or more on the scale has been shown to be clinically significant (Jacobson, 1990).

The ABC is a 16-item questionnaire that asks the patient to rate their confidence in balance with activities of daily living (Powell, 1995). Each item is rated from 0 to 100, with 100 being completely confident that they will not lose their balance or become unsteady with the task. It was originally developed for community dwelling older adults to measure fear of falling but can be useful when assessing patients with mTBI and has been shown to be valid and reliable in patients with vestibular disorders (Whitney, 1999). A score of 67 or less has been shown to indicate an increased risk for falls (Lajoie, 2002).

The PCSS evaluates for symptoms associated with concussion (Lovell, 2006). It is a 22-item questionnaire that requires the patient to rate the severity of 22 different symptoms of concussion on a 0–6 scale. The PCSS can give the practitioner an idea of the severity as well as the breadth of symptoms, including those that are non-vestibular and may be pertinent to their care.

The VVS is a condition specific questionnaire assessing perceived severity of vertigo symptoms during the last month by measuring frequency of dizziness, vertigo, imbalance and related autonomic symptoms (Yardley, 1992). The 15-item scale is broken down into 2 subscales indicating the impact of vertigo and balance and autonomic/anxiety. The total score is 60 and indicates symptom severity with higher score (Yardley, 1992).

4. **Treatment**

4.1. **Acute/sub-acute/chronic considerations**

When addressing the vestibular needs of a patient with mTBI it is important to consider not only the severity of the injury but the degree of acuity. In early mTBI, the main focus of treatment is related to limiting the patient’s activity, restoring normal sleep patterns, and managing acute symptoms pharmacologically (McCrory, 2009). Although animal models have given us general time-frames relating to the altered cerebral metabolism (Giza, 2001; 2011), the clinically accepted standard is to limit activity until the patient no longer
is experiencing symptoms while at rest (Willer, 2006). Although empirically a viable approach, symptoms of dizziness and vertigo from a secondary source may persist long after normal metabolic function of the brain has been restored. Early assessment of the vestibular system may help to differentiate between dizziness that arises from the metabolic insufficiency and symptoms related to vestibular dysfunction. Other contributors to early persistent dizziness may include post-traumatic migraine (Hoffer, 2010; Donaldson, 2010), cervical spine dysfunction (Wrisley, 2000), visual-perceptual dysfunction (Kapoor, 2002), and autonomic dysfunction (Lekdy, 2007). As dizziness following concussion has been shown to be a marker of poorer prognosis and prolonged recovery as noted above (Lau, 2011), careful early assessment and treatment of these areas should be considered.

Since these patients often have multiple impairments across disciplines, it is important to coordinate their care with other practitioners. One important factor is the management of post-traumatic headaches. Vestibular rehabilitation is most effective in patients who have their headaches well controlled (Gottshall, 2005; 2011), so it is important to monitor their headaches and help coordinate their treatment with the physician. Although it is well established that vestibular rehabilitation can have a positive impact on recovery from peripheral lesions (Hillier, 2007; 2011), incomplete compensation, prolonged treatment, and slower progress has been noted for dysfunction in the central vestibular pathways (Shepard, 1995; Furman, 2000). Recent research specific to mTBI has shown that vestibular rehabilitation can help to facilitate recovery (Garr, 2001; Alsalaheen, 2010; Gottshall, 2010), but what constitutes “Vestibular Rehabilitation” is loosely defined and broad. It is encouraging that there has been an exponential increase in the study of this specific population, but currently the depth and breadth of evidence substantiating the benefit of vestibular rehabilitation following mTBI is not yet on par with that seen in peripheral vestibular dysfunction.

4.2. Benign paroxysmal positional vertigo

Benign Paroxysmal Positional Vertigo (BPPV) following traumatic brain injury accounts for 8.5% to 20% of all cases of BPPV (Ahn, 2010). Post-traumatic BPPV has been shown to require more repositioning treatments for full resolution than idiopathic cases of BPPV, but there is no difference in recurrence rates (Ahn, 2010). It is important to clear the patient of BPPV prior to initiating other exercises as it alone can change a patient’s balance and thus skew your evaluation findings (DiGirolamo, 1998; Blatt, 2000; Chang, 2006; Celebiyou, 2009). Specific treatment for BPPV is well established and does not need to be revisited.

4.3. Dynamic gaze stability principles

One of the major treatment areas in vestibular rehabilitation focuses on restoring dynamic gaze stability. The ability to maintain focus while the head is in motion is primarily mediated by the vestibular ocular reflex (VOR), the cervical ocular reflex (COR), and the visual system via smooth pursuit and optokinetic responses (Paige, 1994). Following injury to the vestibular system, impairments in the VOR are readily observed. Patients may complain of dizziness with head motion, vertigo, visual distortion, and oscillopsia (Schubert, 2004). Following mTBI, impairment in the VOR may be due to peripheral injury, as seen in labyrinthine concussion, vestibular nerve transaction, endolymphatic hydrops, or perilymphatic fistula (Fausti, 2006). There may also be impairment of the central vestibular connections in the brain stem or the modulating structures in the cerebellum (Davies, 1995). It has also been proposed that dysfunction in the pre-motor cortex efference copy signals may play a role in dynamic gaze stability deficits during active head movements following mTBI blast trauma (Sherer, 2011).

Restoration of dynamic gaze stability following vestibular injury emphasizes activity that promotes adaptation of an uncompensated VOR. It has been demonstrated that presenting a foveal stimulus while the head is rotated facilitates improvements in the gain of the reflex over time (Viirre, 2002). In the clinical setting, this paradigm is commonly described as “X1” and “X2” viewing (Herdman, 2007).

In “X1” viewing, the patient actively rotates their head through 30 degrees in the yaw and pitch planes while maintaining focus on a discreet stationary object. The patient is instructed to move their head as quickly as possible without visual distortion. Minor errors of retinal slip of the foveated image are thought to drive the adaptation of the VOR gain (Miles, 1981). Once the patient demonstrates adequate performance with a stationary target, they are progressed to the “X2” viewing paradigm. Under this condition, the patient maintains focus on a target that they move opposite their head. This effectively doubles the gain of the response and satisfies three rehabilitation needs: 1. Improving functional performance of the VOR at faster...
head angular acceleration. 2. Providing a coordination task that acts as a cognitive distraction, making the task of gaze stability more automatic, and 3. Following moving objects visually is more functional, as the human eye is behaviorally biased toward moving objects. The need to maintain synchrony with the head and target does not seem to be as important as previously thought. Recent evidence suggests that asynchronous head and target movements facilitate faster adaptation in individuals with peripheral lesions (Schubert, 2008). The effect of asynchronous movement in adaptation of dynamic gaze stability following mTBI has not been reported.

Sometimes, therapists are quick to progress the patient to the "X2" paradigm or use complex visual stimuli when the basic ability of simple stationary target gaze stability has not been satisfied. A major rationale for this pre-mature progression is the fallacy that the patient must be made dizzier in order to improve.

It is our opinion that in treating patients with mTBI, symptom minimization coupled with performance optimization provides a more desirable outcome than gratuitous stimulation. Unlike patients with peripheral vestibular dysfunction, most patients with mTBI usually require very basic targets. High contrast single asymmetrical optotypes of adequate size is generally an accepted starting-point. More impaired patients may require targets with the added benefit of proprioception afforded by using their thumb or fist.

As image quality in adaptation is paramount, a major barrier to recovery in dynamic gaze stability occurs when the visual system is also impaired. Common injuries to the visual system following mTBI include damage to the globe, visual pathways, and nuclei in the brainstem. Occipital lobe injury and damage to cranial nerves III, IV, and VI are also prevalent (Suchoff 1999). Injury to the pre-frontal cortex may not only lead to the common behavioral issues that arise following mTBI, but also can directly impact volitional saccades and pursuit movements. More subtle defects such as convergence and or divergence insufficiency, visual field loss, and fusion disorders may not be grossly obvious and may only be elucidated with more sophisticated screening and testing techniques. The resultant impairment in static gaze stability can generate greater visual distortion with head motion.

In animal studies, Zee et al., (1980) demonstrated the importance of the visual experience in adaptation. Monkeys with vestibular lesions that were denied visual experience via occipital lobectomy did not show adaptation in the impaired VOR. In another testing group, monkeys with an intact visual system were able to adapt the gain of the VOR to pre-lesion levels, but did not maintain this compensation after being exposed to prolonged periods of environmental darkness.

Other studies have demonstrated the effect of optical magnification on VOR adaptation. Differing magnifications required different gains in order to maintain foveal image stability, a process modulated by the floculus of the cerebellum (Lasberger, 1984). Traumas to this region can affect the ability to modulate the VOR gain and impair adaptation (Rambold, 2002). This may be compounded by any refractive correction worn by the patient. This effect is increased when they wear the correction intermittently, or are trying to function in bi-focal or multi-focal lenses.

Attempts at dynamic gaze stability may worsen static gaze stability issues. Although adaptation is expected to take longer than in a purely peripheral dysfunction, failure to adapt over time may be the first indicator that there is an underlying dysfunction with static gaze stability. For example slow progress in the yaw plane of rotation as compared to pitch may also indicate an underlying convergence insufficiency. This is can be attributed to the static angle of convergence seen with head motion in the pitch plane versus the very dynamic convergence angle in the yaw plane. Some other symptoms that may indicate problems with static gaze stability include diplopia, “shadowing” of an image, complaints of eye-strain and fatigue. Functionally, these patients may complain of difficulty with reading, hesitancy and imbalance descending curbs and stairs, and tripping on objects. Underlying visual dysfunctions should be treated by a neuro-optometrist. These patients oftentimes require treatment for these dysfunctions before they are ready for gaze stability exercises.

4.4. Postural control

Another major treatment area for patients with mTBI in vestibular rehabilitation is in restoring postural control. It is well documented that mTBI and concussion can cause static and dynamic balance impairments (Parker, 2005; 2006; 2008; Slobounov, 2006; Sozoff, 2011; Guerts, 1996, Basford, 2003). Normally, postural control is maintained through an integration of information from the visual, vestibular, and somatosensory systems. When treating patients with postural control and balance dysfunction post mTBI one must look closely at the interaction between these 3 systems of balance as the brain weights the afferent information differently depending on the conditions of the environment (Mahboobin, 2005).
For patients post-mTBI, they may have difficulty re-weighting the sensory information with changes in environment (Slobounov, 2006). For example, they may have difficulty maintaining their balance when walking from a relatively calm environment visually into a very crowded or visually stimulating environment. Thorough testing needs to be performed to assess the visual, vestibular, and somatosensory systems individually and how they work together. One cannot underestimate the importance of how task complexity increases demands on the CNS. Several studies have shown patients post-mTBI and concussion have greater difficulty maintaining postural control under conditions of divided attention (Parker, 2005; 2006)

Balance assessments give the therapist a chance to look at the patient’s protective and righting reactions to loss of balance and changes in environment. In our experience, patients post-mTBI can exhibit almost paradoxical righting and balance reactions with both eyes open and eyes closed. This may be due to impairments in their ability to perceive the environment and process the vast amount of information flooding their central nervous system. These patients typically will use inappropriate righting reactions and have increased use of stepping and hip strategies for even the smallest loss of balance.

As with gaze stability exercises, it is important to begin postural control exercises with basic environments before advancing the patient to environments with increased complexity. Incorporating treatment of balance impairments with variations of eyes open/closed, head turns and head tilts, changes in surface (floor vs. foam vs. rocker board), weight shifting and walking with head turns can assist in improving the integration of postural control systems.

A useful test for both assessment and treatment is the mCTSIB in which can give the therapist quick and useful information regarding utilization of vestibular cues, postural responses, and visual dependency (Cohen, 1993). If a pattern of visual dependency is determined, the therapist can then work on having the patient decrease visual reliance through eyes closed activities, increasing the difficulty from static tasks to dynamic. Once the patient has demonstrated ability to adequately use vestibular cues for stability with eyes closed, the therapist can proceed to training the patient with visual/vestibular mismatch i.e. using optokinetic training as referred to previously.

One must not forget to assess the effect of other system impairments on balance. The importance of thorough neurological testing cannot be overlooked as impairments in strength, coordination, sensation, tone and cognition can impair balance and stability.

4.5. Approaches to cervicogenic dizziness

Cervicogenic dizziness has long been considered a diagnosis of exclusion and its existence has been historically questioned (Brandt, 1996). Recent evidence has given more conclusive clinical tests to assist in determining if this is a contributing factor (Tjell, 1998; Treleaven, 2008). Despite the academic debate, it is important to consider cervical dysfunction in the care of patients following traumatic brain injury for a number of reasons. First, in order to provide adequate stimulus for vestibular rehabilitation, the patient must be able to tolerate head and neck motions. Secondly, erroneous cervical proprioceptive input to the medial vestibular nuclei is hypothesized to create the sensory mismatch that results in dizziness (Roy, 2001). In addition, gaze stability during head movements below 1 Hz are not mediated by the vestibular ocular reflex, but by smooth pursuit, saccades, optokinetic responses, and the cervical ocular reflex (Shepard, 1996). The cervical colic reflex is also thought to play a role in postural stability. Based on these factors, careful screening of patients for cervical dysfunction following mTBI should be part of every exam. In order to consider the cervical spine as a potential contributor to a patient’s dizziness, there must be some degree of neck pain. Typically the pain experienced is mild to moderate. Tests for alar and transverse ligamentous instability should also be performed, especially if the patient is presenting with bilateral upper extremity pain and parasthesias provoked by cervical flexion. Gross screening for cervicogenic dizziness can be performed by assessing if the symptoms of dizziness are relieved with manual cervical traction. Body on head movement that provokes symptoms over whole body rotation is thought to isolate cervical stimulation over vestibular stimulation, and may be used as a screening tool as well. This is described by Norre (1987) and van de Calseyde (1976). More recent tests include the Smooth Pursuit Neck Torsion Test (Tjell, 1998; Treleaven, 2008) and the Joint Position Error Test (Revel, 1991; Swait, 2007). Treatment is geared toward addressing the cervical dysfunction. Therapeutic exercises for cervical range of motion, manual therapy for segmental hypomobility can be utilized. Manual, mechanical, and self-cervical traction may help to improve ROM and alleviate symptoms of pain and dizziness. Deep cervical flexor isometric stabilization exercises and joint position error retraining are also
modalities used to rehabilitate the cervical spine. In a randomized controlled trial by Revel et al. (1994), significant reductions in symptoms and improved cervical kinesthesia was demonstrated by patients following a cervical proprioception rehabilitation program. In addition, addressing any postural instability is a critical part of treating cervicogenic dizziness. The accepted standard of care in circumstances where the patient presents with cervical dysfunction and dizziness is to use both techniques for rehabilitation of the cervical spine and an impaired vestibular system (Wrisley, 2000).

4.6. Pacing and graded exertional exercise

In athletes and service members the goal of rehabilitation is to return the individual to the field or active duty. The peak performance required to fulfill these roles requires a high tolerance to exertional activity, a major contraindication in early recovery following mTBI. Although no protocols have been established, the current guidelines recommend restricting return to play until athletes are asymptomatic following maximal exercise (McCrory, 2009). This is entirely premised on the idea that the individual is asymptomatic at rest.

In the non-athlete it has been hypothesized that graded exertional exercise can have beneficial effects when introduced in the sub-acute stage of recovery. Exercise post-injury has been shown to result in up-regulation of Brain Derived Neurotropic Factor (BDNF) (Griesbach, 2004) which is thought to play a role in recovery on a neuronal level. Sub-symptom graded exercise may also help to restore normal cerebral homeostasis (Leddy, 2007) as dysfunction in cerebral blood pressure regulation has been attributed to the symptoms of headache and dizziness experienced on exertion (DeWitt, 2003). In addition, the use of graded exercise is being explored as a possible solution to the autonomic dysfunction seen in some individuals with PCS. Abnormal Heart Rate Variability (HRV) in chronic mTBI is considered an indicator of increased sympathetic activity and decreased parasympathetic tone. Graded exercise training in patient with congestive heart failure (Larsen, 2004) have shown shifts in HRV that indicate a change from sympathetic drive to increased parasympathetic activity at rest. This has only been studied in acute concussion (Gall, 2004) but to date has not been explored in the patient with refractory PCS.

Fatigue can be a common barrier to performance optimization in individuals with PCS. Because of this, patients are often instructed in pacing techniques to try to limit the degree of their fatigue. Despite a lack of strong evidence to support this technique, it is commonly used to manage this symptom. Pacing is also prescribed early in recovery, as patients are educated on avoiding activity levels that provoke or exacerbate symptoms.

4.7. Space and motion sensitivity

Visual Vertigo (VV) is defined as discomfort, postural instability, and symptoms of dizziness, lightheadedness, and/or disorientation in situations involving visual-vestibular conflict or intense visual motion stimulation (supermarkets, crowds, watching movies, and driving) (Pavlou, 2010). Visual motion stimulus has a strong influence of perception of verticality and postural control in patients with VV than individuals with peripheral dysfunction without VV. Pavlou, 2010 Therefore patients with VV tend to be overly reliant on visual cues for balance and verticality. Currently rehab professionals are using optokinetic nystagmus (OKN) training for addressing VV and motion sensitivity in patients with peripheral vestibular dysfunction. These studies have been shown to be effective, though the exact parameters of treatment are still unknown (Pavlou, 2010). Treatment has included fall field and partial field optokinetics, such as a disco ball, virtual reality and use of DVD or computer at home for stimulation. There have been no studies that look at using optokinetic stimulation in central vestibular patients or in patients with mTBI/PCS. Optokinetic stimulation can have a place in vestibular therapy in mTBI, but the therapist should rule out oculomotor dysfunction as cause of symptoms before proceeding with treatment using optokinetics. These patients tend to be very sensitive to visual surround motion and care should be taken not to over stimulate patient. As with all vestibular exercises, monitoring of symptoms before and after optokinetic training is important as these patients can easily be over stimulated and have delayed symptoms response to treatment.

4.8. Other considerations

It would be negligent not to discuss the high degree of emotional and cognitive exertion that accompanies PCS and mTBI. Decreased cognitive efficiency, coupled with the taxing elements of depression and anxiety can profoundly impact the individual’s ability to function. In addition, we have observed a subset of patients with PCS who struggle with cycled periods of hyperactivity and vigilance followed by greater than
normal fatigue levels and hypersomnia. Hyperstimulation may be the result of cognitive-behavioral issues including task perseveration, disinhibition, and poor insight due to impairments in abstract thought. In the PCS brain, it has also been theorized that there is a high degree of neuronal recruitment for even in the simplest of cognitive and physical tasks (Chen, 2004; Jantzen, 2004; Kemp, 1995). This increased energy demand, coupled with a metabolic and autonomic dysfunction can result in severe functional impairment. Although this pattern is not seen in all patients with PCS, it can be a major confounding factor in vestibular rehabilitation. These patients demonstrate difficulty maintaining the consistent cognitive efficiency needed to sustain the desired adaptive changes promoted by the therapeutic intervention. In addition, deficits in attention, sequencing, memory, and insight present global barriers to the learning process that is critical to vestibular adaptation. If the patient is fortunate enough to be receiving neuropsychology counseling, it is important to coordinate learning strategies in order to maximize adaptation and motor learning and avoid overstimulation.

5. Conclusion

Patients with mTBI and concussion commonly have complaints of vertigo, dizziness, and imbalance. Vestibular rehabilitation can decrease dizziness and vertigo and improve static and dynamic balance through implementation of exercises targeting the patients’ specific impairments. Vital to successful treatment of these patients is the coordination of care with other practitioners, particularly for patients with post-traumatic headaches and for patients with underlying visual and ocular motor dysfunctions. We also cannot stress enough the importance of initiating treatment with simple exercises in quite environments and progressing these patients slowly with more complex tasks. We are of the opinion that less is more and routinely prescribe a limited exercise regimen that is performed several times per day as tolerated.

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