Jen Tooher

Module 4 Assignment

 **Unilateral Vestibular Hypofunction**

**Epidemiology**:

More than 15 million people develop dizziness in the United States each year, with an annual incidence of 5.5%.1 The prevalence of dizziness is dependent on age, sex, and description of dizziness, ranging from as little as 1% up to 35%.6 Patients with dizziness typically characterize their symptoms as feelings of vertigo, spinning, tilting, unsteadiness, imbalance, swaying, disequilibrium, or lightheadedness.2 More specifically, dizziness classified as vertigo has a prevalence of 6.7%, which increases with age and is higher in women. Dizziness is the most common reason that individuals over 75 years old visit the physician. Less than 30% of individuals with complaints of dizziness have resolution of symptoms after 2 weeks.1

Three categories exist to classify different mechanisms for dizziness: age-related changes, environmental or lifestyle changes, and pathologic changes. Age-related changes can provoke dizziness in older adults because of decreased visual acuity, diminished proprioception thresholds, fewer vestibular neurons in Scarpa’s ganglion, and slower motor responses in older adults. Environmental and lifestyle changes include changes due to medication side effects and poor vision correction. Finally, pathologic causes of dizziness can come from cardiovascular insufficiency and lightheadedness, CNS conditions such as TBI or MS, metabolic or endocrine disorders like diabetes and hypoglycemia, anxiety and depression, and peripheral vestibular system dysfunction.2 Most forms of dizziness are associated with decreased quality of life, especially dizziness due to vestibular dysfunction.1

Peripheral vestibular disorders include benign paroxysmal positional vertigo, unilateral vestibular hypofunction (UVH) and bilateral vestibular hypofunction. Approximately 10% of the general population will have problems with the peripheral vestibular system at some point in their life.1,3 Unilateral vestibular hypofunction typically occurs as the result of vestibular neuritis or neuronitis, Meniere’s disease, vestibular schwannoma, vascular lesions affecting the vestibular nerve, and vestibular labyrinthitis.2 The most frequent cause of UVH is vestibular neuronitis, accounting for 5% of all dizziness and occurs more commonly in individuals with herpes simplex virus.1,9 1,710 cases of vestibular neuritis, 500 cases of Meniere’s disease, and 11.5 cases of vestibular schwannoma occur per million people each year.9 People with UVH experience symptoms of vertigo, retinal slip, spontaneous nystagmus, postural instability, gaze instability, and oscillopsia with head movements.1

**APTA Guide Patterns:**

The *APTA Guide to Physical Therapist Practice* has identified several practice patterns for individuals with unilateral vestibular hypofunction, including:

* Pattern 4B: Impaired Posture
* Pattern 5A: Primary Prevention/Risk Reduction for Loss of Balance and Falling
* Pattern 5F: Impaired Peripheral Nerve Integrity and Muscle Performance Associated with Peripheral Nerve Injury10

**Pathology/Pathophysiology:**

The vestibular system has both peripheral and central components, so vestibular pathology can be of either peripheral or central origin.1 UVH is a peripheral vestibular dysfunction that occurs as a result of decreased nerve receptor input.6 The peripheral system includes the vestibular labyrinth, which is made up of three semicircular canals (SCC) and two otolith organs: the saccule and utricle. The vestibular labyrinth and the vestibular portion of cranial nerve VIII are housed in each inner ear.2 Each SCC is aligned to have a contralateral coplanar pair in the opposite ear to provide the brain with bilateral excitation and inhibition inputs during head movements.6 Anywhere from 15,000 to 25,000 vestibular nerve fibers exist to make this system function properly.1

The nerves in the peripheral vestibular system send regular and irregular afferent inputs to the inner ear, with a healthy resting firing rate around 70-100 spikes per second.6 Regular afferents project to the extraocular muscles and provide input to the VOR regarding slow, small-amplitude head movements, whereas irregular afferents provide input during fast, large amplitude movements. These inputs are integrated in the central vestibular system in order to sense and respond to head motion and stabilize images on the fovea of the retina.1 The vestibular system also plays an important role in postural control.2

The functions of the vestibular system are executed via extremely precise vestibulocular, vestibulo-cervical, and vestibulospinal reflexes.7 The VOR reflex allows the eyes to remain fixated on an object by contracting the eye muscles in the opposite direction of the head movement.1 The vestibulo-cervical reflex is activated when semicircular canals sense head and neck movement, sending signals to the medial vestibular nuclei and spinal cord to reflexively activate neck muscles, head position, and forelimb extensors.7 The vestibulospinal reflex occurs when the lateral vestibular nucleus is activated by input from otolith organs, reflexively exciting proximal extensors and assisting in the maintenance of upright posture.6 With UVH, these reflexes may not function properly as a result of nerve damage, causing individuals to complain of dizziness and imbalance.7

Unilateral vestibular hypofunction (UVH) involves damage to the superior vestibular nerve more often than the inferior vestibular nerve.1 Unilateral damage that occurs as a result of inflammation, infection, vascular injury, or tumor affects the push-pull mechanism that is essential for proper vestibular function. The push-pull mechanism compares inputs between the right and left coplanar semicircular canals to detect head movement. When the brain recognizes differences in firing rates, it interprets movement towards to side with excitation.6 With unilateral damage, the neuronal activity to the ipsilateral vestibular nuclei is diminished, which creates an imbalance between the two vestibular nuclei and tricks the brain into thinking that the head is moving to the contralateral side. As a result, the VOR tries to compensate for the head movement and provokes spontaneous nystagmus.8 Individuals with UVH commonly present with severe vertigo, nystagmus and often vomiting, especially in the first few days. The quick beat during nystagmus moves away from the lesion, or towards the intact side.9 Symptoms, most notably spontaneous nystagmus, often resolve within days to weeks, but individuals are left with residual visual complaints and balance deficits.8 Central compensation with vestibular rehabilitation can occur relatively quickly.4 Individuals with unilateral vestibular hypofunction are at an increased risk of falls due to their poor spatial orientation and feelings of unsteadiness.5

**Effects on Systems:**

1. *Vestibular*: Since the pathology directly affects the vestibular nerves, UVH affects the tonic vestibular activity, causing an intense sensation of head rotation and provoking dizziness and vertigo.1-3,6-9
2. *Visual*: Because the vestibular system is so closely tied to the visual system by way of the vestibulo-ocular reflex, visual symptoms are very common with UVH. The VOR reflex is very fast and occurs as a 3-neuron arc. The primary vestibular afferent from the SCC synapses on the ipsilateral medial and ventrolateral vestibular nuclei in the brainstem. Then secondary afferents are innervated from the ipsilateral labyrinth, allowing axons to decussate and synapse on the contralateral abducens nucleus (promoting contralateral eye abduction) and ascend ipsilaterally to the oculomotor nucleus (promoting ipsilateral eye adduction.) When the signal from the ipsilateral vestibular nucleus is diminished from UVH pathology, increased excitation on the contralateral side and inhibition on the ipsilesional side occurs, and the eyes will move towards the contralateral side. This causes spontaneous nystagmus, decreased gaze stability, and poor dynamic visual acuity.1,6,8-9,11
3. *Autonomic*: Nausea and vomiting are very common within the first few days of vestibular pathology. The mismatch between the vestibular and eye signals can provoke the symptoms of nausea and vomiting associated with UVH, and it takes the brain a few days to adapt and compensate before nausea and vomiting subside.6,8-9
4. *Neuromuscular*: Balance deficits are common in individuals with vestibular dysfunction because optimal balance requires the integration of visual, somatosensory, and vestibular input. With UVH, there is decreased or incorrect input from the vestibular system, which causes postural imbalance, gait deficits, and mild ataxia.2,5,9

**Impairments:**

UVH results in various impairments that can affect an individual from completing the activities necessary for everyday life. These impairments include:1-3,5-6,8-9,11

1. Severe Dizziness and Vertigo
2. Dysequilibrium
3. Spontaneous Nystagmus
4. Poor gaze stability
5. Nausea/Vomiting
6. Possible hearing loss, tinnitus, fullness in ears (Meniere’s, labyrinthitis)
7. Impaired visual acuity
8. Positive head thrust
9. Oscillopsia
10. Impaired static balance
11. Impaired dynamic balance
12. Impaired gait
13. Impaired spatial awareness
14. Poor subjective visual vertical and horizontal

**Activity/Participation Limitations:**

Individuals with UVH will likely have to take off work for one to two weeks after initial onset because the symptoms are most severe while the body is trying to fight off the infection and use the central nervous system to compensate.8 Spontaneous nystagmus typically resolves within 3 to 7 days in common daylight.9 Recovery typically takes approximately 3 weeks, although there may be residual motion sensitivity.6 Head motion sensitivity is greatest at large amplitudes and high velocities, which may make athletic activities difficult to perform.8 Falls risk is most severe during the acute phase and then levels off and approaches normal after recovery.9 Some occupations that require high levels of vestibular and visual input, such as pilots, might not be realistic after UVH.8 Some mild problems with thinking have been shown to exist in individuals with vestibular lesions. Decreased quality of life is associated with dizziness, so resolution of symptoms is important to improve activity, participation, and quality of life.6

**Environmental/Personal Factors:**

Anxiety and depression have been associated with decreased balance confidence as well as decreased ability to participate in activities at discharge.Prior level of function and age don’t seem to affect recovery. One study found that comorbidities don’t significantly alter patient outcomes,12 however individuals with diabetes who have diabetic neuropathy and impaired sensation might have a more difficult time recovering than individuals with intact sensation.13 Individuals with joint problems may also have decreased recovery because of difficulty performing exercises secondary to pain.14 Patients with poor subjective complaint of activity limitations, slow gait speed, or who are at a high falls risk at discharge have also been shown to have poorer recovery.12 Patients with progressive vestibular disease or fluctuating, nonstable vestibular lesions are not ideal candidates for vestibular rehabilitation.13 Also, individuals with biphasic head-shaking nystagmus typically have more severe vestibular hypofunction than individuals with monophasic head-shaking nystagmus.22

**Intervention:**

Vestibular rehabilitation (VR) is, not surprisingly, the most common intervention for individuals with unilateral vestibular hypofunction.15 Since research is still somewhat limited regarding vestibular rehabilitation interventions for UVH, I’m going to focus this section on habituation, adaptation, and/or substitution exercises that can be done as a part of vestibular rehabilitation. Gait and balance exercises are a component of vestibular rehabilitation programs as well.

Vestibular rehabilitation for UVH focuses on using central compensation so the body adapts to the dizziness that was provoked from asymmetric tonic firing rates due to unilateral infection, trauma, or vascular changes.6,13,15 A primary goal during vestibular rehabilitation is to stop exercises before exacerbation of symptoms occurs (i.e.- stopping exercises once a patient reports 3/5 dizziness.)13 VOR exercises should be done in the yaw plane, because asymmetries will be most transparent in this plane compared with either pitch or roll.21 Maximal recovery should occur in approximately 6-8 weeks, and home exercise programs need to be incorporated into physical therapy treatment to maximize positive effects.14 Exercises should include low-level versions of the activities that reproduce a patient’s symptoms of dizziness.6

The initial vestibular exercises, developed by Cawthorne and Cooksey, focused on head movements, head and eye coordination, total body movements, and balance tasks. Varying speeds, positions, environment, and visual input caused neuroplasticity of the vestibular system to occur, and patient symptoms of dizziness improved significantly.23,24 These exercises involved challenging patients in ways that promoted habituation, a reduced adverse response with repeated movements. Adaptation exercises are designed expose individuals to retinal slip because it causes long-term changes of the residual vestibular system to improve its’ response and prevent blurring of images when an object moves off the fovea.6 And substitution exercises allow the body to identify and develop alternative strategies to maintain visual fixation and prevent disequilibrium.15

A recent review by Herdman found moderate to strong evidence suggests that vestibular rehabilitation is both effective and tolerable for individuals with UVH. Types of vestibular rehabilitation discussed in this review included adaptation, habituation, and substitution. The most common combination of vestibular rehabilitation in the literature includes habituation, gaze stabilization, and balance training.26 Evidence suggests that vestibular rehabilitation can be useful in acute and chronic vestibular dysfunction,15 although identifying and treating UVH as soon as possible may promote greater improvements.26 Not only does vestibular rehabilitation improve dizziness, VOR, gait, and balance, but improvements have also been shown in quality of life and activities of daily living. Moderate evidence suggests that improvements are maintained months after rehabilitation, promoting the value of VR for individuals with UVH.15

One study had patients perform gaze stability exercises including VORx1, VORx2, and balance retraining exercises while standing and walking. Treatment was catered to individual needs based on their available sensory information (visual, somatosensory, and vestibular.) The participants in this study who had UVH significantly increased their gait speed and decreased their double limb support time. Furthermore, the patients who walked with less vertical displacement had increased stability, suggesting that walking and head rotation vestibular exercises may promote compensatory strategies and improve postural stability.14

Very few studies have examined which type of vestibular rehabilitation exercise is most effective. However, one small study found similar improvements between individuals treated with habituation exercises compared with individuals treated with adaptation and substitution exercises for UVH. Because both exercises were similarly beneficial, head movements with exercises might be an essential component to improving symptoms and visual acuity in individuals with UVH because it allows for simultaneous habituation and gaze stabilization/adaptation.17 Further, studies suggest that habituation should be performed for 30 seconds or until mild to moderate symptoms disappear, whichever comes first.6

In another study, adaptation and ambulation exercises together effectively improved subjective reports of disequilibrium and objective balance tests including the Romberg eyes closed and anterior-posterior sway on force platform in individuals after acute acoustic neuroma resection compared with controls.20 This study, along with others, suggest the value of VR in acute post-operative individuals with UVH.26

A newer study addressed the effects of gaze stabilization training for individuals with UVH. The study used virtual training 30-40 minutes, 2 days a week for 6 weeks via the Ninento Wii to integrate gaze stability while using street backgrounds and interactive games. A corresponding HEP included standing VORx1 and VORx2. Patients reported decreased dizziness, improved balance, and increased walking speed,5 concluding that gaze stability is an effective intervention for individuals with UVH. The study did not have a control group, so the value of these exercises in comparison to gaze stabilization and habituation or just habituation is unknown. Virtual reality exercises might be especially beneficial for individuals with UVH who want to return to high-level function, although larger studies need to better examine the value and feasibility of this technology.

VOR pathways might be more adaptable to change during unilateral incremental active VOR adaptation. A study found that 15 minutes of unilateral VOR adaptation training improved VOR gain 22% during active head rotations and 11% during passive head rotations, whereas the contralateral side only increased by 8% during active rotations. Adaptation is thought to occur from modifiable regular afferents during the tonic component of the vestibulo-ocular reflex since UVH likely damages the irregular afferents responsible for the quick, highly modifiable VOR. 25

Finally, there have been a couple new studies examining if force platforms have additional benefits for vestibular compensation and symptom-relief in individuals with UVH.15 Force platforms are thought to provide additional information to encourage compensation via biofeedback, and preliminary studies suggest that improvements in stability and performance occur after vestibular rehabilitation and force platform balance training.16-18 However, another study did not find significant differences between the vestibular rehabilitation with and without force platform biofeedback.19 Thus, more research needs to be done to substantiate the value of using force platforms as a part of VR for people with UVH.

In conclusion, substantial evidence points to the effectiveness of vestibular rehabilitation for individuals with unilateral vestibular hypofunction.1-2,5-6,8-9,11-26 Dizziness, visual acuity, falls risk, and walking ability are the primary impairments and activity limitations that occur with UVH.6 Adaptation via gaze stabilization exercises, compensation via habituation exercises, substitution exercises, and balance retraining all seem to play a central role in creating long-term changes to the vestibular system in order to positively affect these impairments and functional limitations.26 Although there are studies that aim to address the physiologic mechanisms by which adaptation, habituation, and substitution occur, the research does not clearly delineate the value of using one of type of intervention over the others. And despite the evidence promoting the value of vestibular rehabilitation for individuals with UVH, it is still not effective in all patients.15 Thus, individualizing vestibular rehabilitation interventions based on each patient presentation, visual and vestibular symptoms, and confounding factors will provide the optimal intervention for patient improvement after unilateral vestibular hypofunction.

**Outcome Measures:**

Listed below are assessment tools and outcome measures that may be used during rehabilitation for an individual who presents with UVH.5-6,9,27-33

1. Dynamic Visual Acuity (DVA)- Dynamic Visual Acuity measures visual acuity with horizontal motion of the head. A greater than 3-line difference suggests vestibular hypofunction, including unilateral vestibular hypofunction.
2. Head Thrust test- the Head Thrust or Head Impulse Test assesses semicircular canal function. A positive test is when there is a corrective saccade made to reposition the eyes when the therapist rotates the head toward to side with the hypofunction.
3. Head-shaking Induced Nystagmus (HSN)- this test helps to identify UVH specifically. A positive test is identified when nystagmus is present.1
4. Visual Analog Scale (VAS)- the VAS can be used as a subjective rating scale of many different subjective complaints, most commonly pain. However, it can effectively measure dizziness or vertigo.
5. Dynamic Gait Index (DGI)- The Dynamic Gait Index assesses falls risk and can be effective to assess falls risk in individuals with vestibular dysfunction. A score of less than 19 suggests that a patient is at a high falls risk.
6. Five Times Sit to Stand (FTSTS)- The MCID for individuals with vestibular disorders is 2.3 seconds or more.
7. Dizziness Handicap Index (DHI)- The DHI is a validated 25-item questionnaire that quantitatively measures how an individual believes his or her dizziness affects function and impact on daily activities.
8. Activities-Specific Balance Confidence Scale (ABC)- this is a 16-item subjective questionnaire that asks patients to rate their confidence performing different activities that require balance.
9. Functional Disability Scale- this scale was developed to objectively assess a patient’s response to physical therapy.
10. Motion Sensitivity Quotient- this assessment measures an individual’s subjective report of their dizziness.
11. Sensory Organization Test (SOT)- identifies abnormalities by controlling visual, vestibular, and somatosensory input.
12. Single Legged Stance- has patient stand on one leg for thirty seconds to assess if balance impairments exist in an individual with vestibular disorders.
13. Romberg- has a patient stand and balance with eyes closed and feet together for 30 seconds. Assesses balance impairments.
14. Tinetti Fall Risk Performance Scale- measures balance and gait abilities and identifies risk of falling.
15. Hospital Anxiety and Depression Scale (HADS)–attempts to capture psychological effects of anxiety and depression in individuals with vestibular dysfunction.

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