Vestibular rehabilitation therapy in Patients with Multiple Sclerosis

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Abstract

Background: Dizziness refers to various abnormal sensations relating to perception of the body's relationship to space. Vertigo is the most frequent classification associated with dizziness. It is generally defined as the sensation of motion when still or a perception of distorted motion with normal head movement. In each ear, the vestibular nerve connects directly to the ipsilateral vestibular nuclei (VN), as well as to the contralateral side through several interconnecting neurons. The cerebellum, the reticular formation, the spinal cord, and the cervical junction all provide additional afferent information to the VN. Efferent information is relayed from the VN back to these same areas. Central vestibular disorders (CVDs) are group of disorders that are caused by lesions along the vestibular pathways, which extend from the VN in the medulla oblongata to the ocular motor nuclei and integration centers in the pons and rostral midbrain; and to the vestibulocerebellum, the thalamus, and multisensory vestibular cortex areas in the temporoparietal cortex. Vestibular rehabilitation therapy (VRT) is an exercise-based treatment program designed to promote vestibular adaptive and compensatory mechanisms already existing in the human brain. VRT speeds up the process of improvement. Among patients with CVDs, cerebellar dysfunction improves the least after VRT, while others improve significantly, although most continue to exhibit gait dysfunction and continue to be at risk of falling. In central dizziness, postural instability may improve slowly or not improve with VRT. Furthermore, in some degenerative cases, it may progress even with vigorous rehabilitation treatments. Mechanistically, several areas along the peripheral and central vestibular neural pathways may be involved in the pathogenesis of MS. These regions include the eighth nerve, VN, the oculomotor tracts, the medial longitudinal fasciculus, and the cerebellum. Interventions specifically designed to improve balance in MS should yield better results than interventions to strengthen lower limbs or aerobic exercises

Keywords: Vestibular rehabilitation therapy, central vestibular disorders, Multiple Sclerosis

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Introduction

Dizziness refers to various abnormal sensations relating to perception of the body's relationship to space. It is a nonspecific term used to describe a subjective sensation that patients may find difficult to fully explain. Various broad concepts have been used to describe and classify dizziness. These categories include vertigo, presyncope or syncope, disequilibrium, lightheadedness, and nonspecific dizziness **(1).**

Vertigo is the most frequent classification associated with dizziness. It is generally defined as the sensation of motion when still or a perception of distorted motion with normal head movement. A sensation of spinning is also linked with vertigo. Vertigo is the result of dysfunction of the vestibular system which contributes to various aspects of movement and gait, balance, stance, vision, spatial orientation, and memory. The slightest disturbance of the vestibular system can result in vertigo **(2).**

Presyncope and syncope; Syncope describes the loss of consciousness as a result of temporary disruption to cerebral oxygenation due to interruption of blood flow to the brain. Presyncope, a more common occurrence, is the prodromal symptom of fainting or a near fainting. Common causes of presyncope consist of orthostatic hypotension, cardiac arrhythmias, psychogenic disorders, and vasovagal syncope **(3).**

Lightheadedness is a vague term and can be clarified as "wooziness" or a sense that one might be close to fainting. It is often correlated with the presyncope category because of similarity **(4).**

Disequilibrium is a sense of unsteadiness and loss of balance involving the legs or trunk. A number of different underlying disorders may result in disequilibrium. These include peripheral neuropathy, vestibular disorder, musculoskeletal disorder, gait disorder, and Parkinson's disease **(5).**

Nonspecific dizziness is often difficult for the patient to describe and lacks diagnostic physical exam findings. A variety of etiologies are associated with nonspecific dizziness, such as depression, anxiety, hyperventilation, and adverse reactions of medications **(6).**

Vestibular disorders are traditionally classified by the anatomical site of the lesion into peripheral or central disorders. Lesions at sites such as the labyrinth (semicircular canals and otoliths) and the vestibular nerve, the first-order neurons, are classified as peripheral vestibular disorders. By contrast, conditions involving the VN in the pontomedullary brainstem and the vestibular pathways that project from these nuclei to the vestibulocerebellum (via the cerebellar peduncles), brainstem, thalamus, and cortex are grouped as central vestibular disorders. When applied clinically, this simple anatomical distinction suffers from certain weaknesses. The first weakness is the inaccuracy in diagnosing disorders originating at the transition from the peripheral to the central system, i.e., the root entry zone of the eighth nerve. This area is subserved by the peripheral vestibular neuron traversing from the vestibular ganglion to the vestibular nucleus and is "peripheral" by definition. Thus, lesions of the root entry zone of the eighth nerve, although anatomically peripheral, require a neurological approach to exclude central lesions **(7).**

Central vestibular disorders are clearly defined clinical syndromes of various etiologies. The occurrence of typical ocular motor findings, for example, downbeat or upbeat nystagmus in only cerebellar or brainstem lesions allows their definite localization. On the other hand, central vestibular symptoms can also be a part of a more complex infratentorial clinical syndromes with other symptoms or supranuclear/nuclear/fascicular ocular motor disorders and/or other neurological brainstem deficits (e.g., in Wallenberg's syndrome or midbrain infarction). Central vestibular syndromes are most often caused by: infarction, hemorrhage, tumor, degenerative brain diseases, or multiple sclerosis (MS) **(8).**

Vestibular rehabilitation therapy in central vestibular disorders

Central vestibular disorders (CVDs)

A) Functional anatomy of central vestibular system

The peripheral vestibular system contains five sensory structures: three semicircular canals (the horizontal, also termed lateral; anterior, also termed superior and posterior, also termed inferior canals) and two otolith organs (the utricle and the saccule). After peripheral end-organ excitation, labyrinthine sensory information is transmitted by the eighth cranial nerve through the internal auditory canal, entering the brainstem at the pontomedullary junction to relay in the VN **(9).**

The starting place of the central vestibular pathways is the VN. VN are located at the fourth ventricle and extend in two columns from the pons to the medulla. As the primary recipients of vestibular input, the VN include four major nuclei, the medial, superior, lateral, and inferior which function to process vestibular input before transmission to motor centers**.** All vestibular nerve afferents synapse in the VN and as many as 70% of the afferents continue into the cerebellum **(10).**

In each ear, the vestibular nerve connects directly to the ipsilateral VN, as well as to the contralateral side through several interconnecting neurons. The cerebellum, the reticular formation, the spinal cord, and the

cervical junction all provide additional afferent information to the VN. Efferent information is relayed from the VN back to these same areas **(11).**

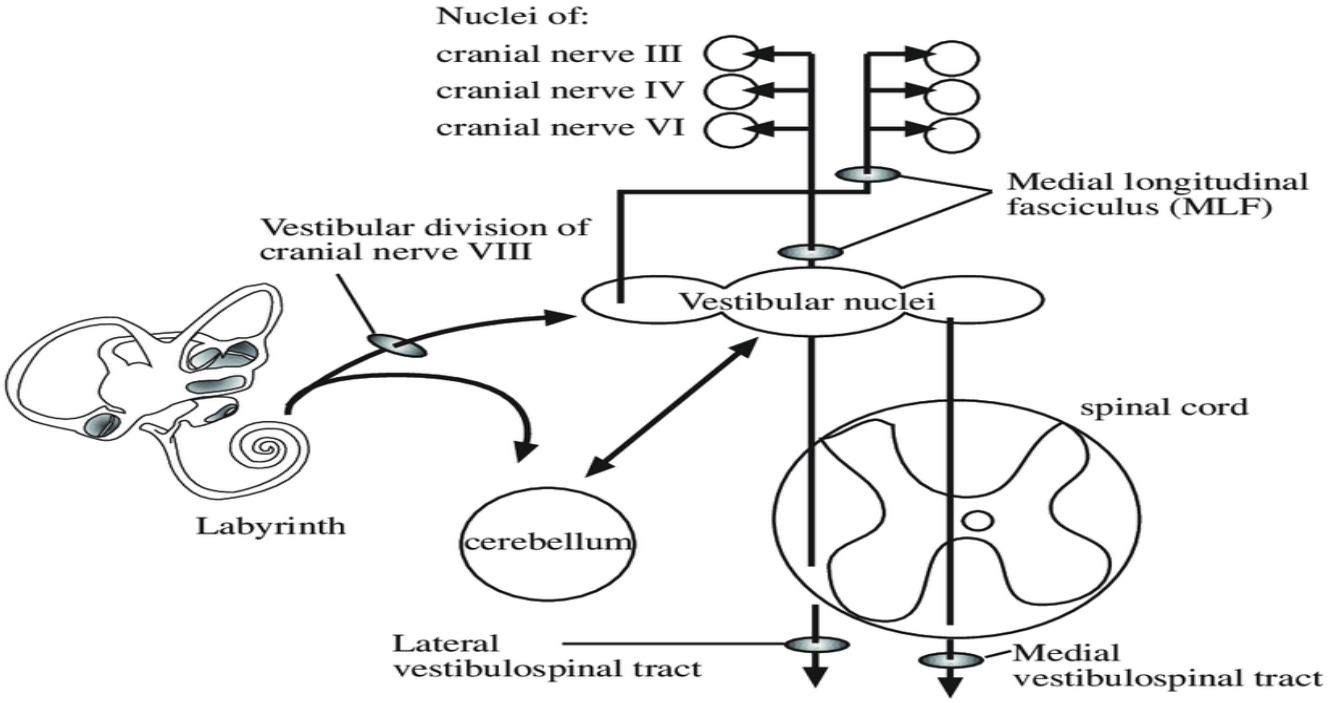

Figure (1): Diagram of central vestibular pathways with multiple interactions. Quoted from: **Harno (12).**

B) Definition of CVDs

CVDs are group of disorders that are caused by lesions along the vestibular pathways, which extend from the VN in the medulla oblongata to the ocular motor nuclei and integration centers in the pons and rostral midbrain; and to the vestibulocerebellum, the thalamus, and multisensory vestibular cortex areas in the temporoparietal cortex. These central disorders are often clearly defined as clinical syndromes of various etiologies, with typical ocular motor, perceptual, and postural manifestations that permit a topographic brainstem diagnosis. Depending on the size of the lesion, central vestibular disorders can occur in isolation or as part of a complex infratentorial syndrome **(13).**

C) Prevalence of CVDs

Vertigo and dizziness belong to the most frequent symptoms in primary care units with an estimated lifetime prevalence of 17–30% In tertiary interdisciplinary outpatient dizziness units, central vestibular disorders comprise about 25% of the established diagnoses **(14)** with vestibular migraine is the most prevalent cause **(15).**

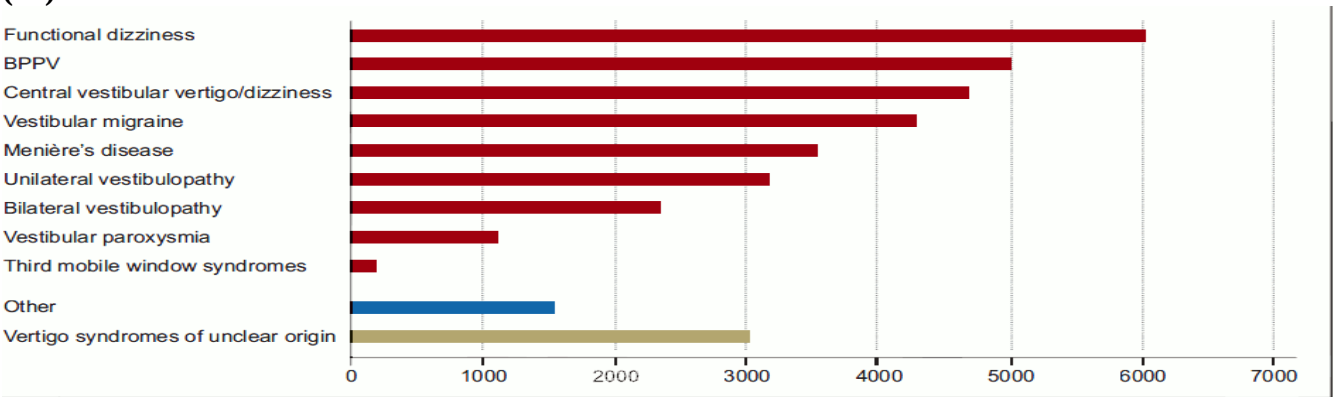

Figure (2): The frequency of various vestibular syndromes among 34 860 patients in a specialized outpatient clinic. Quoted from: **Strupp et al. (16).**

D) Classification of CVDs

Based on the International Classification of Vestibular Disorders (ICVD), vestibular symptoms of central origin are divided into three syndromes, namely acute vestibular syndrome (AVS), episodic vestibular syndrome (EVS), and chronic vestibular syndrome (CVS) **(17).**

- ***Acute central vestibular syndrome:*** e.g; stroke and brainstem or cerebellar ischemia/infarction. Other less common causes are demyelinating lesions, structural lesions, immune-mediated disorders such as cerebellitis or thiamine deficiency. It is an emergency calling for immediate referral to a hospital and, ideally, a stroke unit **(18).**
- ***Episodic central vestibular syndrome:*** It is an intermittent vestibular symptom that lasts from seconds to hours with periods of remission between attacks. The most common causes are vestibular migraine, transient ischemic attacks, and central paroxysmal positional vertigo **(17).**
- ***Chronic central vestibular syndrome:*** It is a vestibular symptom that appears from months to years. The most common causes are posterior fossa tumors, intoxications, cerebellar ataxia, and other neuro-degenerative lesions **(19)**.

E) Assessment of CVDs

Despite the marked progress in laboratory medicine and neuroimaging, detailed history and bedside identification of central vestibular signs are essential for diagnosis of central vestibular disorders **(20).**

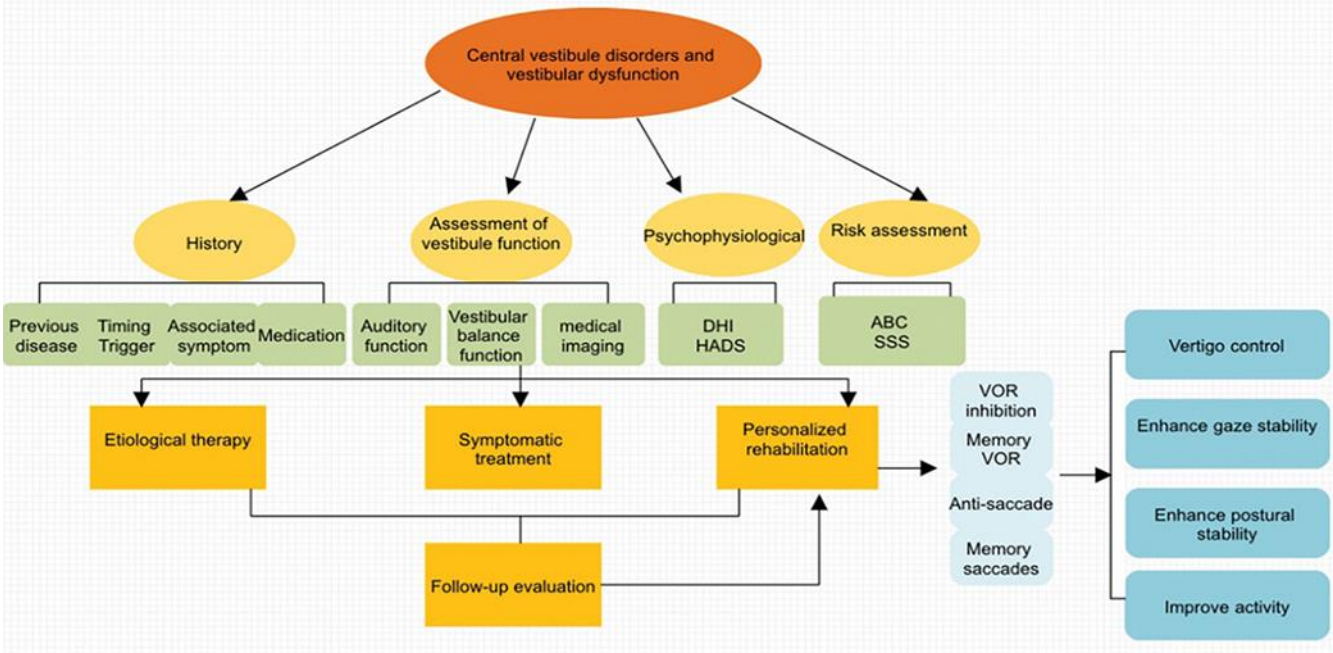

Figure (3): Diagnosis and treatment process of vestibular rehabilitation in central vestibular disorders and vestibular dysfunction. Quoted from: **Zhang et al. (21).**

Abbreviations: VOR=Vestibulo-Ocular Reflex; DHI=Dizziness Handicap Inventory; HADS=Hospital Anxiety Depression Scales; ABC=Activity-specific Balance Confidence Scale; SSS=Somatic Self-rating Scale.

History

Patients with central vertigo are often severely unstable and have difficulty walking without falling. Brainstem involvement with vertigo should be suspected if the patient reports the following neurologic symptoms: staggering or ataxic gait, vomiting, headache, double vision, visual loss, slurred speech, numbness of the face or body, weakness, clumsiness, or incoordination. Gait disturbance also can indicate cerebellar dysfunction **(6).**

	Nausea& Vomiting	Imbalance	Hearing loss	Oscillopsia	Neurological	Compensation
Peripheral	**Severe**	**Mild**	**Common**	**Mild**	**Rare**	**Rapid**
Central	**Moderate**	**Severe**	**Rare**	**Severe**	**Common**	**Slow**

Table (1): Differentiation between peripheral (end-organ and nerve) and central causes of vertigo. Quoted from: **Furman & Whitney. (22).**

Bedside examination

Pure downbeat, upbeat, and torsional nystagmus are well-known features of central vestibular lesions. Direction changing gaze-evoked nystagmus in the horizontal or vertical plane is generally considered a sign of impaired neural integration in the central neural network. Head impulse nystagmus test of skew (HINTS) exam has been shown effective at differentiating an acute stroke (central cause) from a peripheral cause of dizziness, such as vestibular neuritis. In head impulse test, the absence of saccadic eye movement indicates a higher likelihood of central etiology. Spontaneous nystagmus of peripheral origin is typically horizontal with a torsional (rotational) component and it does not change direction with change in gaze. By contrast, spontaneous nystagmus of central origin is often purely horizontal, vertical or torsional and usually changes direction with changes in the position of gaze. The final component in the HINTS assessment is the test of skew. Brainstem pathology is indicated if the patient's covered eye deviates vertically **(1).**

Head-shaking and positional maneuvers may give additional information for diagnosis of central lesions, especially those involving both peripheral and central vestibular structures. Vigorous or perverted downbeat head-shaking nystagmus after horizontal head shaking should be considered central sign. Positional maneuvers can evoke nystagmus or modulate the spontaneous nystagmus in central as well as peripheral vestibular disorders. A central lesion should be suspected in patients with positional nystagmus refractory to repeated canal repositioning maneuvers **(23).**

Vestibular testing

As regards VNG**, Moideen et al., (24)** reported that abnormal oculomotor tests e.g; delayed saccades and saccadic dysmetria, saccadic pursuit, and symmetric or asymmetric low optokinetic gain were noted while evaluating patients with central vestibular lesions.

Radiological and laboratory workup

A complete workup with lab and diagnostic testing may be required for central vertigo to determine the actual cause. Neuroimaging studies including computed tomography and MRI are also needed to evaluate cerebrovascular disease, demyelinating lesions, brain neoplasms, or other conditions. Magnetic resonance angiography may be used if vertebrobasilar insufficiency is suspected **(4).**

F) Treatment options of CVDs

Identifying the cause of the CVDs a patient has is crucial, as this determines the therapeutic approach and the urgency of initiating treatment e.g; the mainstay of treatment for CVDs caused by an ischemic stroke can include emergent intravenous thrombolytic therapy and interventional clot retrieval. Symptomatic treatment for vertigo and nausea also is needed. Finally, there is advancing evidence for the proposed critical role of vestibular rehabilitation in improving balance, dizziness, and quality of life in patients with CVDs **(25).**

Vestibular compensation

a) Recovery of static versus dynamic vestibular deficits

The vestibular compensation includes two different features related to the time-domain: 1) Static symptoms which is fully compensated within a relatively long time constant (three months for the postural and ocular motor deficits and up to one year for the perception of verticality). 2) Dynamic symptoms which recover in months and moreover seems not to recover totally when looking thoroughly at fine vestibular testing, but patients rarely complain of any persisting dizziness **(26).**

b) Mechanisms

The vestibular compensation occurs through three main mechanisms developed by the patients: adaptation, substitution, and habituation **(27).**

1. **Adaptation**: It represents the regulation of the neuronal activities mainly of the VOR. Physiologically, the VOR permits to stabilize the visual target onto the fovea and consists in displacement of the eye with amplitude and speed exactly in the opposite of the head movement. Its gain is equal to one. After a vestibular loss, the gain of VOR is reduced and oscillopsia occurs. The adaptation increases the gain of the VOR to help in stabilization of vision. The fixation may reduce the intensity of the nystagmus and may explain a faster recovery. However, alteration of the VOR persists far after the resolution of the dizziness without complain **(28).**
2. **Substitution:** It means that the lost functions are not restored but replaced by new operating modes using either other sensory cues (sensory substitution) or newly elaborated motor strategies (behavioral substitution) **(29).**
    - Sensory substitution plays a key role since vestibular functions are multisensory determined and need the integration of vestibular, visual, and somatosensory cues, which constitute potential sources of possible sensory reweighting **(30).**
    - Behavioral substitution is based on the distributed property of the CNS to control the vestibular functions, several neuronal networks in the brain being able to reorganize functionally by learning, and to mimic the lost dynamic vestibular functions. A nice illustration is the covert saccade during head impulse test, which is a saccadic substitution of the normally slow phase eye movement, aimed at preventing oscillopsia during head rotation towards the lesion side **(31).**
3. **Habituation:** It aims at reducing progressively the vestibular lesion-induced asymmetry at the peripheral or central levels by repetition of the triggering signals. This mechanism plays the least significant role in the vestibular compensation process **(32).**

All three mechanisms of vestibular compensation have complex interaction in the functional recovery of the patient. Each patient goes through a unique process of vestibular compensation with differential utilization of these mechanisms **(33).**

Vestibular rehabilitation therapy in central vestibular disorders

a) Definition

VRT is an exercise-based treatment program designed to promote vestibular adaptive and compensatory mechanisms already existing in the human brain **(34).**

b) Indications of VRT in CVDs

Patients with stable central vestibular lesions or mixed central and peripheral lesions should not be excluded from treatment, although their prognoses are likely to be more limited than the average patients with a stable peripheral injury. Head injury: Patients with head injuries suffer from significant disability due to vestibular symptoms. Their conditions often include cognitive and central vestibular involvement along with the peripheral component; therefore, VRT techniques are used as a supplement to a comprehensive, multidisciplinary head injury program **(35).**

c) Objectives of VRT

The program is designed to achieve these goals:

I. Decrease dizziness and visual symptoms.

II. Improve balance and walking functions.
III. Reduce symptoms of dizziness.
IV. Increase general activity levels **(36).**

d) Physiological basis for VRT

Vestibular rehabilitation is based on the following principles:

- **Adaptation:** It refers to a long-term improvement in the vestibular systems ability to adapt to head movement, achieved by the movement of an image across the retina. The best stimulus to induce adaptation is one that produces an error signal. Patient is encouraged to continue even if symptoms increase **(37).**
- **Habituation:** It refers to reduction in symptoms and pathological responses produced by repetitive exposure to the provoking stimulus. It is a central process. Movements performed should be quick and through sufficient range to produce mild to moderate symptoms. As habituation occurs, movements performed should be more vigorous with pauses at the end of every movement until dizziness subsides **(38).**
- **Substitution:** These exercises encourage the use of vision and somatosensory cues with vestibular cues to enhance central programming to improve gaze stability and postural stability. Pathology within the vestibular system leads to an alteration in the relative reliance on sensory information. By performing exercises in environments with altered sensory information, vestibular rehabilitation is able to affect a person's use of sensory information or sensory reweighting **(37).**

e) Principles of VRT

I. Vestibular adaptation exercises to enhance gaze stabilization

Gaze instability is due to the decreased gain of the vestibular response to head movements. The best stimulus to increase the gain of the VOR is the error signal induced by retinal slip (**39).**

- ***VOR adaptation exercises:*** One of the most important structures for vertigo of central vestibular origin is the neuronal pathways that mediate the VOR. VOR adaptation exercises are designed mainly to expose patients to retinal slip. The most frequently utilized exercises are VORx1 and VORx2 exercises **(Figure 4) (40).** In the VORx1 exercise, the patient visually fixates on a stationary target and rotates the head sinusoidally in either the yaw or pitch plane. In VORx2, both the head and the target are rotated in equal and opposite directions, e.g., as the head rotates to the left the target is moved by the patient to the right and vice versa in a sinusoidal pattern. Parameters of distance from the target, head velocity, target size, exercise frequency, and duration are usually individualized and progressed over time **(41).**

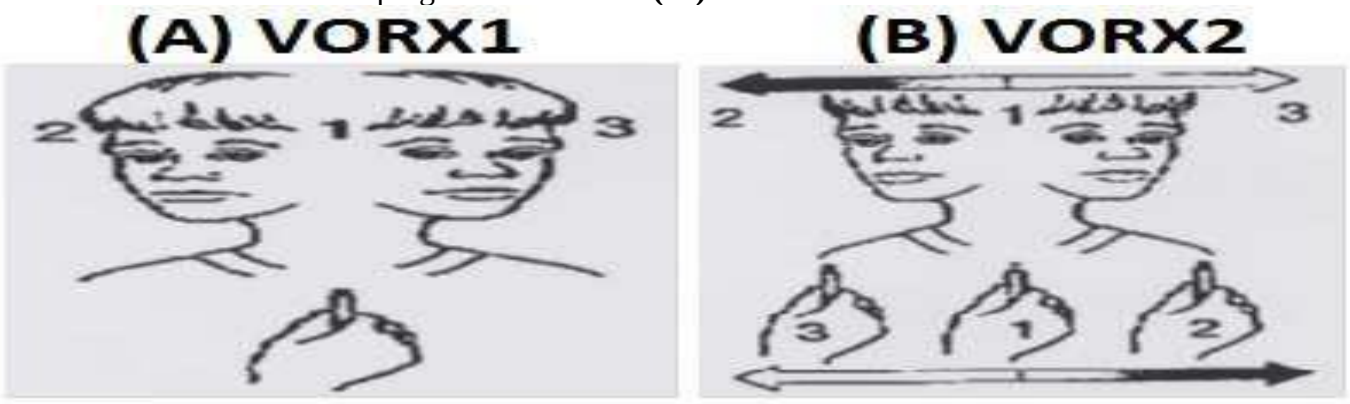

Figure (4): [A] VORx1 in which the visual target is stationary, and the subject moves his head back and forth while trying to maintain visual fixation on the target. **[B]** VORx2 in which the visual target and the head move in the opposite directions while the subject keeps the target in focus. Quoted from: **Tusa & Herdman. (42).**

- ***The VOR suppression*** can be trained by asking the patient to move his or her head and at the same time, tracks a visual target that is going with the head movement. During VOR suppression, the eyeballs are virtually still and does not have abnormal movements. Since the VOR response is canceled by the simultaneously moving visual target, the VOR suppression is also dubbed VOR cancellation (**Figure 5) (43).**

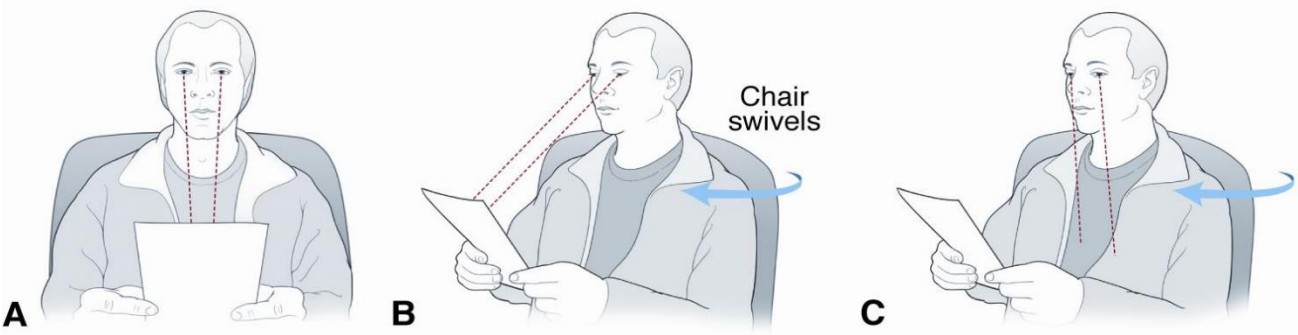

Figure (5): VOR suppression exercise. **A**, the patient is seated in a swivel chair, fixating on the letters of a near card held at arm's length. **B**, If VOR suppression is normal (intact), the eyes maintain fixation on the target as the chair, the patient's head and arms and the card rotate together as a unit. **C**, Conversely, if VOR suppression is impaired, the eyes are dragged off the target during rotation owing to an inability to cancel the VOR. Quoted from:**https://www.aao.org/education/image/vestibular-ocular-reflex-suppression**

VOR suppression is commonly used for the rehabilitation of CVDs because the VOR pathway is a part of the neural reflex of the brainstem and the pathway is controlled by the cerebellar and cortical centers. In addition, CVDs damage the superior center of VOR, and its inhibitory effect is weakened **(21).**

II. Vestibular adaptation exercises to enhance postural stability

Postural stability recovery is slower than gaze stability recovery. The goal of VRT for postural stability is to enhance the use of stable visual and somatosensory information and remained vestibular function. The exercises also help to identify efficient and effective alternative postural strategies. Postural stability exercises include: (1) Standing balance exercises (2) Postural strategy exercises **(44).**

- ***Standing balance exercises***

Static exercises include balancing under conditions of altered visual and somatosensory input such as standing on firm surface, standing on a cushion, standing with the feet heel-to-toe, or standing on one leg. Dynamic conditions challenge high level-balance and include walking with head turns, walking with quick turns to the right or left, or performing a secondary task while walking such as tossing a ball to a partner or performing a cognitive task while walking **(45).**

Figure (6): Standing balance exercises. Quoted from: **https://kutychiropractic.com/auto-accident-chiropractor/strength-and-balance-exercises-prevent-sports-injuries/**

- ***Postural strategy exercises***

Patients with vestibular loss use the ankle strategy but not the hip strategy, even when the hip strategy is required for postural stability, such as when standing on one foot, across a narrow beam, or in a heel-toe stance **(46).** The ankle strategy can be practiced by swaying back and forth and side to side within small ranges, keeping the body straight and not bending at the hips or knees. Small perturbations are used, such as a small pull or push at the hips or shoulders. Patients perform various tasks, such as reaching, lifting, and throwing **(47).**

Hip strategy exercises involve rhythmically shifting the center of gravity by moving the body back and forth in any direction while keeping the feet in place. Weight shifts can be performed from side to side, forward and back (**Figure 7)**, and with hip/trunk rotation. When performing these exercises, the patient must stand in a corner or close to a wall for safety and use a chair for support **(44).**

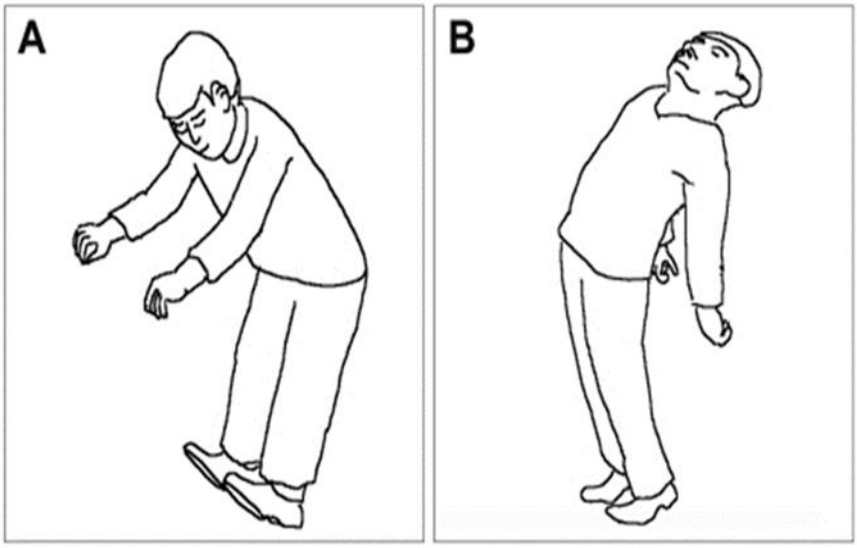

Figure (7): Swaying back and forth. **A:** Bend forward and move the center of your body backward with your toes up. **B:** Bend backward and move the center of your body forward with your heels up. Quoted from: **Han et al. (48).**

III. Vestibular substitution exercises to enhance eye movements

Substitution by other eye-movement systems can effectively cancel the vestibular deficit and protect the patient from perceiving smeared retinal images during head movements **(49).** Vestibular substitution exercises to enhance eye movements involve:

- **Saccade exercise:** The patient holds both his thumbs up to eye level and extend his arms. Without moving his head, the patient quickly alternate his gaze from one thumb to the other and repeat the exercise 5 to 10 times **(44).**
- **Smooth pursuit exercise:** It is a gaze pursuit exercise for a continuously moving object. The patient is asked to keep the head still and moves only the eyes. He extends one arm forward and put his thumb up, slowly moves the arm side to side and follows his thumb with his eyes while keeping the head still and repeat the exercise 5 to 10 times **(44).**
- **Imagery pursuit (VOR memory):** The patient stares at the central target, then closes eyes, slowly turns head to one side, the eyes are glued to the imaginary target in memory, open eyes after five seconds to see if there is a gap between the actual point and the imaginary one and then turn head back to normal position. The session is repeated five times **(21).**
- **The anti-saccade exercise:** The examiner raises the target with both hands and randomly indicates one of the two visual targets. Patient's head stays static and the eyes quickly scan in the opposite direction of the indicated target **(Figure 8)**. The session is repeated five times. It requires suppression

mechanisms to prevent the automatic execution of a visually driven saccade toward the stimulus; hence anti-saccades impose a higher demand on attentional and cognitive resources **(50).**

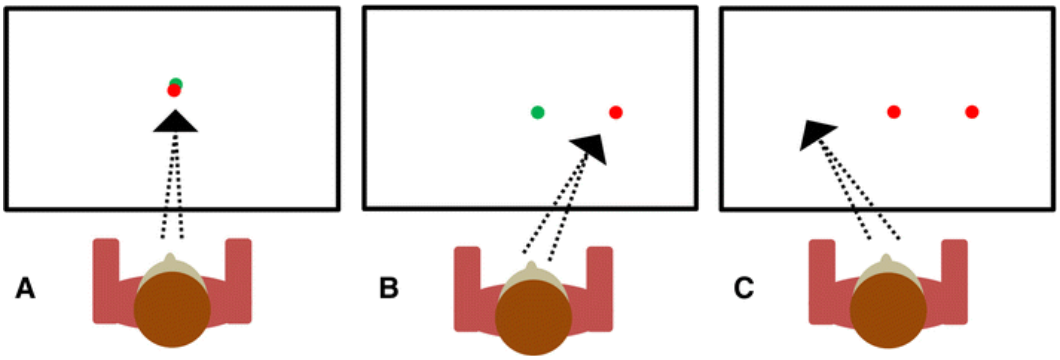

Figure (8): Antisaccade task. Quoted from: **Walton et al. (51).**

- **Memory-guided saccade:** The examiner holds two visual targets with both hands. At a certain angle, the patient stares at the two vision targets on the left and the right. After allowing for adaptation for some time, the patient closes eyes and imagines the eye movement when eyes are open and move eyes to the left and right **(Figure 9)**. The session is repeated five times **(52).**

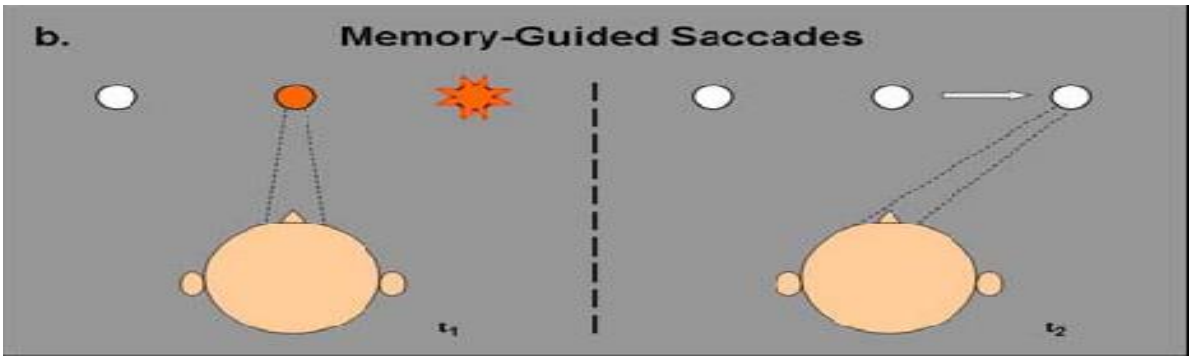

Figure (9): Memory-guided saccades. Quoted from: **Landgraf et al. (53).**

IV. Vestibular substitution exercises to enhance postural stability

- **Exercises to overcome visual dependency**

For patients who are visually dependent, exercises can be devised involving balancing with reduced or distorted visual input but good somatosensory inputs (e.g., in bare feet). These patients should practice maintaining balance during exposure to optokinetic stimuli such as moving curtains with stripes, moving discs with multicolored and differently sized circles or even entire moving rooms. Patients may watch a video showing visually conflicting stimuli while performing head and body movements and while sitting, standing, and walking **(47).**

- **Exercises to overcome somatosensory dependency**

Patients should practice performing tasks while sitting or standing on surfaces with disrupted somatosensory cues such as carpets, compliant foam, and moving surfaces (e.g., a tilt board). An example is catching a ball while standing on a carpet **(47).**

V. Vestibular habituation exercises for improving vertigo

The typical movements that produce the most intense symptoms must be identified at first and then provides the patient with a list of exercises that reproduce these movements **(35).** Habituation is specific to the type, intensity, and direction of the eliciting stimuli. **Figure (10)** illustrates an example for habituation exercises.

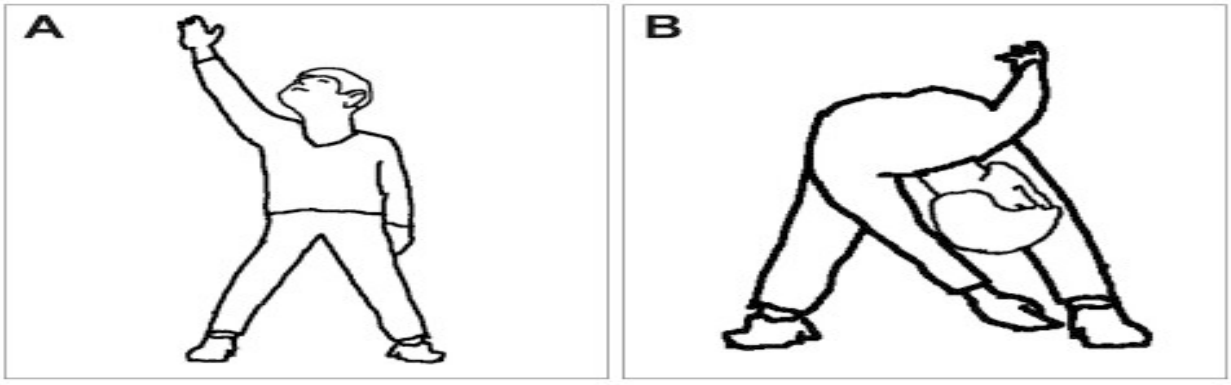

Figure (10): Exercises for improving vertigo. **A:** Stand with one arm elevated over the head, with the eyes looking at the elevated hand. **B:** Bend over and lower the arm diagonally with the eyes continuously looking at the hand until the hand arrives at the opposite foot. Repeat with the other arm. Quoted from: **Han et al. (48).**

VI. Habituation exercises for improving daily living activities

To achieve the final goal of vestibular recovery, the exercise is integrated into normal activities such as walking rather than being performed with the patient sitting or standing quietly. Various games can be introduced. Moreover, a more strenuous program is suggested that may include jogging, walking on a treadmill, doing aerobic exercises, or bicycling. Activities that involve coordinated eye, head and body movements such as golf, bowling, handball, or racquet sports may be appropriate. Swimming should be approached cautiously because of the disorientation experienced by many vestibular patients in the relative weightlessness of the aquatic environment **(54).**

f) Progression of VRT exercises

- ***Follow-up visits:*** Patients are typically seen once every 1-2 weeks and are provided with a specific daily home exercise program. During each visit the therapist addresses the specific problems and goals of the individual patient. As a patient's progress in therapy plateaus, he or she is switched from customized exercises to a maintenance program comprising a wide variety of motion-orientated activities **(55).**
- ***Progressing to the next exercise:*** The ability to perform specific exercises without dizziness means that the condition of the patient has improved and that those particular exercises have become easier. Therefore, more difficult and demanding tasks using varying speeds of movements should be introduced as the patient improves **(44).**

g) Efficacy of VRT in CVDs

Regarding the natural course, central dizziness may not improve or may do so more slowly. Nonetheless, VRT speeds up the process of improvement. Among patients with CVDs, cerebellar dysfunction improves the least after VRT, while others improve significantly, although most continue to exhibit gait dysfunction and continue to be at risk of falling. In central dizziness, postural instability may improve slowly or not improve with VRT. Furthermore, in some degenerative cases, it may progress even with vigorous rehabilitation treatments **(56).**

Treatment lengths vary for persons with CVDs, but generally these patients are more difficult to treat and require more time for optimal rehabilitation outcomes. Despite these limitations, such patients often respond well to VRT. The reason for this may be that patients with CVDs are likely to have unilateral lesions, with preserved function contralaterally. In addition, there may be sufficient redundancy in central vestibular pathways to allow partial recovery of balance function **(22).**

h) Outcome measures of VRT

Subjective (or self-report) and objective (or observed performance) measures are commonly undertaken at the start and the end of rehabilitation sessions.

Table (2): Summary of typical outcome measures. Quoted from: **Corcoran & Jones. (57).**

Self-Report (Subjective) Measures		**Observed Performance (Objective) Measures**	
Dizziness Handicap Inventory	**DHI**	**Functional Gait Assessment**	**FGA**
Vertigo Symptom Scale	**VSS**	**Dynamic Visual Acuity Test**	**DVAT**
Situational Characteristics Questionnaire	**SCQ**		
Vestibular Disorders Activities of Daily Living Scale	**VADL**		
Hospital Anxiety and Depression Scale	**HADS**		

i) Recent trends in VRT approaches

There are additional methods of management that can be combined with VRT to increase its efficacy:

- **Dual task training:** It means simultaneous training of motor or cognitive dual tasks during conventional rehabilitation therapy. It might be a promising approach for improving gait and balance **(58).**
- **Virtual reality:** It is defined as any computer hardware and software system that generates simulations of real or imagined environments with which participants interact using their own movements. It immerses individuals in realistic and visually challenging environments **(59).**
- **Tai Chi:** It is a balance-based exercise guided by the yin-yang theory of traditional Chinese medicine. It combines deep-breath relaxation and slow and gentle movements with awareness. Tai Chi may be effective in improving balance and flexibility and in enhancing muscle strength, which may help to reduce falls in older people **(60).**

Efficacy of vestibular rehabilitation for MS

Mechanistically, several areas along the peripheral and central vestibular neural pathways may be involved in the pathogenesis of MS. These regions include the eighth nerve, the VN, the oculomotor tracts, the medial longitudinal fasciculus, and the cerebellum. Furthermore, a deficit in the integration of these sensory cues along the subcortical and/or cortical areas has also been observed to be linked to impaired balance performance. Impaired balance in MS is also associated with lesions in the brainstem and cerebellum. Irrespective of the location of the lesion, either in the cerebellum or in brainstem, VRT strategies such as anti-saccade or memory-guided saccade may improve balance, dizziness, fatigue symptoms, and health-related quality of life **(21).**

Interventions specifically designed to improve balance in MS should yield better results than interventions to strengthen lower limbs or aerobic exercises. **Hebert et al., (61)** found that fixation exercises, static and dynamic balance exercises, acclimatization, walking in various postures, and walking training that change visual inputs could effectively improve vestibular function in MS patients. Furthermore, **Gandolfi et al. (62)** reported that a sensory integration balance training performed under different sensory conditions may

improve balance more in MS patients. Intensive multidisciplinary rehabilitation is recommended for all MS patients and the treatment should start as early as possible to maximize functional recovery **(63).**

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