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# Management of Migrainous Vertigo Patients: Review Article

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**ABSTRACT**

Vestibular migraine (VM) is one of the most common causes of recurrent vertigo, affecting approximately 1% of the general population and up to 10% of patients seen in dizziness clinics. It is characterized by episodic vertigo associated with migraine features such as headache, photophobia, phonophobia, or visual aura. The exact pathophysiology remains unclear, but it likely involves central mechanisms linking vestibular pathways and migraine-related neuronal excitability, particularly in the brainstem and cerebellum. Diagnosis is primarily clinical, based on the Bárány Society and International Headache Society criteria, which require at least five episodes of vestibular symptoms lasting 5 minutes to 72 hours, with a current or past history of migraine and migraine features during at least half of the episodes. Vestibular migraine is more common in women and typically presents in middle adulthood, although onset may occur at any age. The management of vestibular migraine involves a multidisciplinary approach targeting lifestyle modification, trigger identification, acute attack management, and long-term prophylaxis...

**Keywords:** *Vestibular migraine; vertigo; migraine-associated vertigo; vestibular rehabilitation; prophylaxis; calcium channel blocker; beta-blocker; triptans..*

## 1. INTRODUCTION

Vestibular migraine treatment is challenging, with no widespread consensus. The current treatment options are extrapolated from the non-pharmacological and pharmacological management of migraine headaches. They consist of halting an acute attack (abortive medications) or preventing future attacks (non-pharmacological treatments and prophylactic medications).

### Non-pharmacological treatments

Non-pharmacological treatments focus on managing migraine triggers and include changes in diet, sleep hygiene, stress management, and avoiding intense stimulation. In addition, supplements such as magnesium, riboflavin and CoQ10 were found to be helpful in preventing migraine attacks **(1)**.

Also, vestibular rehabilitation, which involves activities aimed to enhance spatial perception and body coordination (i.e., dancing and ping-pong), have been utilized **(2)**.

### Life style modification:

Evidence-based lifestyle modifications for standard migraine include minimizing stress, identification and avoidance of triggers (e.g., heat, loud sounds, strong odors, bright lights), proper sleep hygiene and having a regular sleep schedule, dietary change (e.g., avoiding hunger, dehydration, caffeine, alcohol), creating a headache diary to identify specific food triggers, regular physical activity, and maintaining a healthy body habits **(3)**.

### Pharmacologic treatments

Pharmacologic treatments have been used both to address acute VM episodes and for preventive treatment, with the exact choice of treatment depending on patient comorbidities and the medication's side effect profile. Abortive therapies include antiemetics like ondansetron, antihistamines such as meclizine, and antidopaminergics such as metoclopramide to treat nausea caused by vestibular dysfunction. In addition,

triptans (almotriptan, sumatriptan, and zolmitriptan) have been found to be effective **(4)**.

Prophylactic medications include beta blockers such as propranolol, antiepileptic drugs such as topiramate and lamotrigine, tricyclic antidepressants such as amitriptyline, selective serotonin reuptake inhibitors (SSRIs) such as venlafaxine and paroxetine, serotonin and norepinephrine reuptake inhibitors (SNRIs), benzodiazepines and calcium channel blockers such as verapamil **(5)**.

CGRP monoclonal antibodies (galcanezumab) and serotonin receptor 5-HT1F agonists (lasmiditan) are novel therapies more recently approved for migraine prevention and treatment and are currently being studied regarding their ability to treat VM **(2)**.

**Complementary therapy**

Complementary therapy for migraine such as the herbal supplement feverfew (Tanacetum parthenium) as well as the natural supplements magnesium, riboflavin, and coenzyme q10 may be used for vestibular migraine, as all of these have some clinical trial evidence as preventives for migraines. It should be noted that in 2015 the **American Academy of Neurology (AAN)** retracted their 2012 complementary therapy guidelines regarding safety concerns surrounding the supplement petasites (butterbur) **(6)**.

**Traditional preventive therapies**

Common classes of medications that have been used to treat migraine for years include antiepileptics such as topiramate and valproic acid, tricyclic antidepressants such as amitriptyline and nortriptyline, serotonin norepinephrine reuptake inhibitors (SNRIs) such as venlafaxine and duloxetine, beta blockers such as propranolol and metoprolol, and calcium channel blockers such as verapamil.

there is some evidence to suggest that medications such as acetazolamide and lamotrigine may be more effective for the vestibular symptoms than the headache symptoms in migraine patients. The choice of agent to initiate may be based on side effect profile versus its utility in treating coexisting conditions **(7)**.

**Table 1.** Vestibular migraine preventive pharmacologic options: **(8)**

| Medication Class | Examples | Considerations | Diseases Medication may also Treat |
|---|---|---|---|
| **Supplements** | Feverfew, magnesium riboflavin, coenzyme q10 | - Minimal side effects | N/A |
| **Antiepileptics** | Topiramate, valproic acid, lamotrigine, gabapentin | - Side effects variable but may include cognitive dysfunction, paresthesias, drowsiness, tremor, nausea, skin rash<br>- Lamotrigine may be more effective for vestibular symptoms of migraine | Gabapentin: PCS |
| **Tricyclic antidepressants** | Amitriptyline, nortriptyline | - Side effects may include anticholinergic effects, drowsiness, confusion, hyponatremia, serotonin syndrome | PCS |
| **Serotonin norepinephrine reuptake inhibitors (SNRIs)** | Venlafaxine, duloxetine | -Side effects may include sexual dysfunction, bleeding, hypertension, hyponatremia, serotonin syndrome<br>-Must titrate up and down slowly | PPPD |
| **Beta blockers** | Propranolol, metoprolol | -Side effects may include fatigue, bradycardia, bronchospasm | N/A |
| **Calcium channel blockers** | Verapamil | -Side effects may include bradyarrhythmias, headache, constipation | MD |
| **Calcitonin gene-related peptide (CGRP)** | Erenumab, galcanezumab, fremanezumab | -Side effects may include injection site reactions, constipation, hypertension<br>-Subcutaneous injections every 28 days | N/A |

| | | | |
|---|---|---|---|
| **antagonists, subcutaneous** | | | |
| **CGRP antagonist, intravenous** | Eptinezumab | -Side effects may include anaphylaxis, angioedema<br>-Intravenously administered every 3 months | N/A |
| **CGRP receptor antagonist, oral** | Rimegepant, atogepant | -Side effects may include gastrointestinal, hypersensitivity reactions<br>-Rimegepant administered every 2 days and may be used as abortive | N/A |
| **Onabotulinum-toxinA** | Onabotulinum-toxinA | -Side effects may include sore neck or discomfort<br>-Administered every 3 months | N/A |

**Beta-Blockers**

Beta blockers block sympathetic/adrenergic-induced vasoconstriction their beneficial effects in migraine headache prophylaxis are unclear. Studies show stabilization of the CNS with restoration of habituation to light stimuli at the occipital cortex. Exact mechanisms and sites of activity are unknown. They can be quick acting in migraine as evidenced by reports of timolol eye drop effectively aborting migraine headache. Long used in migraine headache prophylaxis, and used to treat VM there is one RCT documenting efficacy in VM **(9)**.

**Calcium Channel Blockers**

Voltage-dependent Ca2+ channels are integral membrane proteins that permit extracellular Ca2+ to enter cells down their electrical and concentration gradients and have a universal role in stimulus-response coupling in excitable cells. Calcium channel blockers reduce channel opening in response to membrane depolarization in neurons thereby stabilizing neural tissues and can also decrease vasoconstriction by decreasing calcium influx into muscle cells. There is ample evidence supporting the use of Ca2+ blocking agents in migraine headache prophylaxis **(10)**.

**Anticonvulsants**

The mechanisms of these drugs in migraine are not well understood. It is thought that anticonvulsants act on voltage- and receptor-gated sodium ion channels promoting stabilization of neuronal membranes and preventing repetitive firing. They are thereby able to block excitation leading to cortical spreading depression that may be a central precipitator in migraine. These agents have variable GABA enhancing effects as well **(11)**.

**Tricyclic Antidepressants (TCA)**

TCA Long used in migraine headache these have many potentially beneficial effects. They are medium potency sodium and calcium channel blockers so stabilize membranes and can prevent cortical depression, are anticholinergic so suppress vestibular function and dysautonomia associated with increased parasympathetic outflow common in migraine, and are serotonin and norepinephrine reuptake inhibitors. Varying populations of serotonin receptors are found in the trigeminal nucleus, vestibular nuclei, inner ear and in serotonergic pathways from the dorsal raphe nucleus to the vestibular nuclei **(12)**.

**Novel preventive therapies**

Erenumab was the first calcitonin gene-related peptide (CGRP) antagonist to appear on the market in 2018, reducing weekly migraine days by greater than 50% **(13).**

Later that year, two other CGRP antagonists galcanezumab and fremanezumab were FDA approved, demonstrating similar efficacy. All three are given as subcutaneous injections every 28 days and are considered well-tolerated since most patients report either no or mild side effects, the most common of which are injection site reactions, constipation, and hypertension **(14)**.

In 2020, the IV infusion CGRP antagonist eptinezumab and the oral disintegrating tablet CGRP receptor antagonist rimegepant were also approved for migraine prevention. Eptinezumab has the advantage of only being given every three months, however it has the risk of hypersensitivity reactions such as anaphylaxis and angioedema **(2)**.

Rimegepant is also well tolerated, however gastrointestinal side effects and hypersensitivity reactions are possible **(15)**.

Finally, the oral CGRP receptor antagonist atogepant was approved for preventive treatment of migraine in 2021 as an alternative option to the injections. Possible side effects include constipation and nausea **(16).**

All these treatments have undergone intense scrutiny for the treatment of migraine, however there is a paucity of data on their effectiveness for VM, as no randomized controlled trials of these therapies exist. However emerging evidence suggests that these CGRP antagonists may be beneficial for VM in the same way they are effective for migraine headache **(1)**.

Onabotulinumtoxin A could be used as treatment for prevention of chronic migraine. Onabotulinumtoxin A for migraine involves injecting 31 sites in 7 key areas of the head and neck. Since its approval by the FDA in 2010, this has emerged as an efficacious systemic drug-sparing option for migraine patients. Side effects include sore neck or discomfort. Emerging evidence suggests it is likely to be an effective treatment option for VM **(17)**.

**Abortive therapies**

Triptans have shown clear benefit in aborting migraine headache, their effectiveness for VM was deemed inconclusive. Currently, the default medications to relieve symptoms of vertigo in an acute attack are limited to anti-emetics such as meclizine, dimenhydrinate, benzodiazepines, and metoclopramide, all of which may be sedating **(18)**.

**Abortive Neuromodulation Treatments(devices)**

However, among the newer approaches to abortive therapy in vestibular migraine, non-invasive vagus nerve stimulation (nVNS) has shown some promise. It does not require a surgical procedure for implantation and it does not entail the patient commitment to an embedded device. Patients must have a prescription to obtain a nVNS device, and they can usually watch instructional videos to learn how to self-administer the treatment.

**Cefaly** device external Trigeminal Nerve Stimulation (eTNS). 19 patients reported improvement in vertigo severity with 20 minutes stimulation. 61% improvement in vertigo and 77%improvement in headache **(19)**.

Non-invasive vagus nerve stimulation of 18 patients reported 47% reduction in vertigo severity and 63% reduction in headache **(20).**

**Vestibular rehabilitation**

Vestibular Rehabilitation (VR) has been used to treat dizziness due to vestibular dysfunction. VR is a treatment method used to treat dizziness and balance dysfunction, based on central mechanisms of neuroplasticity, and accelerates vestibular compensation **(21).**

VR is mechanistically based on the plasticity and functional compensation of the central nervous system and the vestibular system. By means of compensation of the cortex, brainstem, and cerebellar pathway, the stability of eye movement and posture is attained. The cerebral cortex possesses tremendous plasticity. Expanding cortical function or re-organizing the conduction pathway can maintain the vestibular function **(22).**

Components of vestibular rehabilitation include “desensitizing” the vestibular system by provoking symptoms, learning to coordinate eye and head movements, developing balance, and walking skills, and learning to handle disturbing situations. VR components: 1) Compensation/habituation; 2) Adaptation (an adaptation of VOR, gaze stabilization), 3) Sensory substitution (substitution of other strategies for lost function), 4) Motor learning to change movement behavior includes postural control exercises, fall prevention, relaxation exercises, reconditioning exercises, and functional retraining **(23)**.

**1. Adaptation:** It is the results of the tissue remodeling due to the up-regulated expression of corresponding genes and proteins. In the literature on vestibular disorders, adaptation referred to long-term changes in the neuronal response to head movements, with the goal of reducing symptoms and normalizing gaze and postural stability. Synaptic inhibition or membrane hyperpolarization of medial vestibular nucleus neurons elicit a persisting elevation in intrinsic excitability through a process known as ‘firing rate potentiation,’ which might be utilized in vivo to mediate behavioral plasticity **(24).**

Adaptation represents the regulation of the neuronal activities mainly of the VOR which permits to stabilize the visual target onto the fovea. The adaptation increases the gain of the VOR that reduced and oscillopsia that occurred after vestibular loss. VM patients had a greater VOR time constant. In these patients, the enhanced suppression of the VOR in patients suffering from dizziness/vertigo was likely to be a cerebellar adaptation to suppress the hypersensitive vestibular system **(25).**

**Substitution:** Substitution is a process by which a sensory system compensates for the deficiency of another sensory system. Such substitution takes place via vision and proprioception to replace or enhance the lack of vestibular inputs, via the central compensation, i.e. sensory integration, to improve the postural and gaze stability. VR exercises must challenge balance mechanisms holistically and target motor, sensory (vision/vestibular/somatosensory), and cognitive aspects, it must be goal – orientated and context-specific **(26).**

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 **(27).**

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 **(28).**

**Habituation:** Habituation exercise is used to treat symptoms of dizziness that is produced because of self-motion or produced because of visual stimuli. The goal of habituation exercise is to reduce the dizziness through repeated exposure to specific movements or visual stimuli that provokes patients' dizziness. These exercises are designed to mildly, or at the most, moderately provoke the patients' symptoms of dizziness. Over time, with good compliance and perseverance, the dizziness intensity can reduce due to the brain learning to ignore the abnormal signal **(29).**

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 **(30).**

The leading VR principles are desensitizing the vestibular system, increasing vestibulo-ocular and vestibulo-spinal reflex gains, and creating new alternative senses against imbalance triggered by position change. Improvement in all these mechanisms results in progressive improvement in dizziness and vertigo **(31).**

The VR program was implemented as three sessions per week for 18 sessions, and the session duration was approximately one and a half hours. The program was completed in 1.5 months.

The VR program applied to the patients consisted of VR treatment protocol included strengthening and stretching exercises, gaze stability exercises, habituation exercises, exercises to promote vestibular compensation, balance and gait training, exercises to enhance the use of specific sensory inputs for balance control. Exercise groups are: a) Head movement exercises; b) Gaze stabilization or VOR adaptation exercises; c) Standing and walking balance exercises; d) Posture control exercises: on trampoline; e) Hand-eye coordination exercises with the ball; f) Coordination exercises with a pilates ball.

In migraine, patients are more sensitive to vestibular stimuli because their sensitivity to external stimuli increases, so their perception of vestibular dysfunction symptoms may be increased. **Vitkovic et al.** think that patients with VM perceive their symptoms more seriously than patients with vestibular disease, although they have a similar peripheral vestibular function, physical performance, and symptom duration (**32)**.

In migraine, perception and behavioral thresholds change due to altered central excitability. The cortical hyperexcitability theory has also been supported by electrophysiological and magnetic stimulation studies. As a result, patients with migraine have a stronger reaction to intense, repetitive, or prolonged stimulation. This altered sensory modulation may be due to increased neural excitability or decreased neural inhibition at the synaptic or intrinsic neuronal level. VR helps create new balance patterns and reduce dizziness complaints by correcting vestibular functions and compensating for defects. Therefore, the auxiliary role of VR in the optimal treatment of VM is accepted **(33).**

**Vestibular rehabilitation program**

The exercises were individualized according to the symptoms and capability of each patient.

**Adaptation exercises**

Adaptation exercises in vestibular rehabilitation.

(a) Eye saccade exercise: The patient places two objects at eye level, and with the head fixed, the patient quickly focuses on the two immovable objects using only eye movement.

(b) Smooth pursuit exercise: With the head fixed, the patient uses only eye movement to slowly follow the object moving up, down, left, and right.

(c) Vestibulo-ocular reflex (VOR) 1 exercise: Keeping the object fixed in front at arm's length, the patient looks at the object by moving the head up, down, left, and right.

(d) VOR 2 exercise: The patient performs the VOR 1 exercise while rotating the head in the opposite direction to the object.

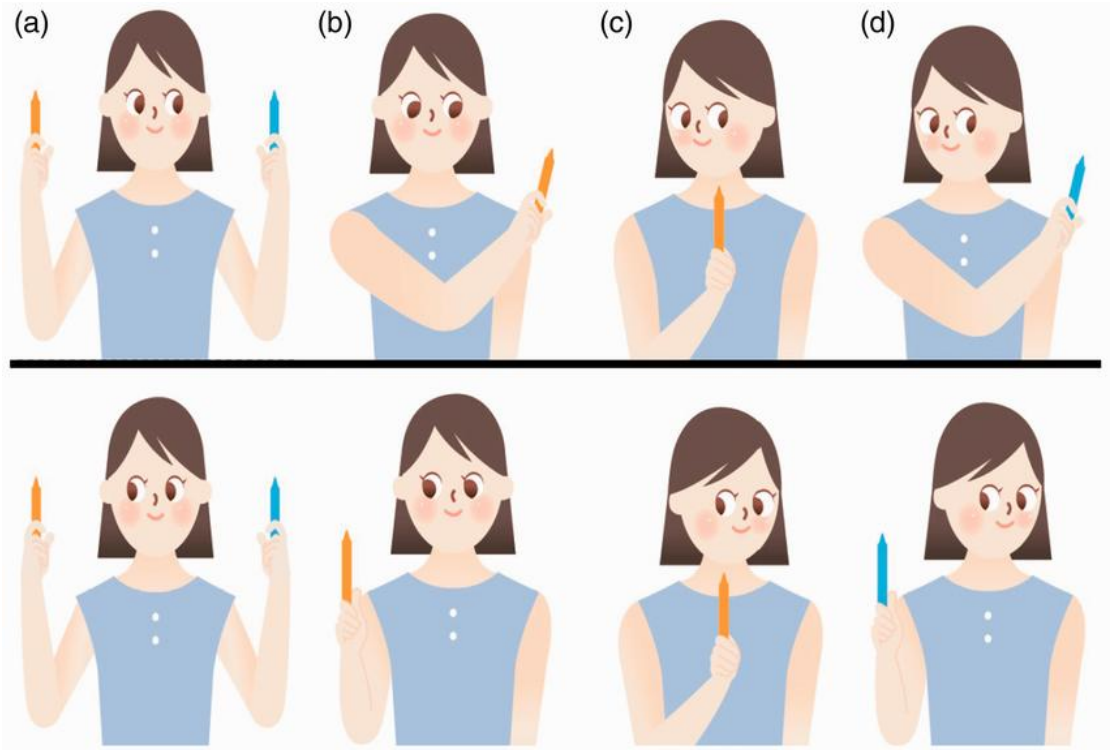


**Figure (1):** Adaptation exercises in vestibular rehabilitation **(34)**

**Substitution and balance exercises**

Substitution exercises are designed to foster the development of alternative strategies as substitutes for lost vestibular function. Other eye movement systems (saccade, central preprogramming, and slow pursuit exercise) are used to protect the participant from perceiving blurred retinal images during head movements. In these exercises, the patients are instructed to follow two different nonmoving objects with eye movements and rapidly alternate their foci from one object to another. The substitution exercises could be modified to become more difficult as the participant improved **(35).**

In balance exercises, the participant tries to restore their standing balance and proceeded to perform standing dynamic balance exercises. For standing balance, participants stand with their eyes open or closed while on a progressively narrowing base of support (feet close together, semi-tandem, tandem stance, or single leg stance). Under these conditions, the participants are also told to turn their heads toward the right and left **(35).**

**Standing dynamic balance exercises**

The participants stand or move without walking. The participants are told to do different moves such as marching in place, stepping forward or backward, stepping to the side, stepping up or down, or turning around.

**Habituation exercises**

These exercises, which cause mild-to-moderate difficulty in daily life, are given to the participants. These exercises involve movements and positions sufficient to cause mild-to-moderate symptoms during the participant's daily activities **(35).**

**Ambulation exercises**

The participants are instructed to walk forward (eyes open or closed), backward, sideways, along a line (narrowing the base of support), and around cones depending on their functional level. Special attention is given to how participants handled walking with head movements (e.g. voluntary head movements, on verbal command, passing balls from hand to hand, turning around). The participants are also instructed to gradually increase their walking speed and walking distance in order to enhance endurance **(35).**

The hospital exercise program consists of one session per week for a period of 8 weeks. Each session lasted approximately 30–45 min, including all exercises, and is conducted in the rehabilitation unit. All participants are followed up once a week by the physical medicine and rehabilitation specialist, who reviews the exercises and makes changes as needed. In addition to the exercises performed at the hospital, all participants are given customized exercises for performing a home exercise program. Home exercises were designed to take approximately 15–20 min to be performed twice a day. During the training at the hospital, a physician and a physical therapist monitors compliance. The home exercises are monitored by having the participant record the session.

In a study by **Goto et al., (36)** aimed to compare the effects of vestibular rehabilitation on headache and other outcomes relating to dizziness, and the psychological factors in patients with VM patients, patients with dizziness and tension-type headache and patients without headache. The vestibular rehabilitation program was as follow:

Patients were hospitalized for 5 days in groups of 8–10 individuals; the groups were then taught how to perform the 30-min vestibular rehabilitation program by themselves. The program comprised repeated training of the vestibulo-ocular (VOR) and vestibulo-spinal reflexes (VSR).

**The VOR training included seven exercises**:

(1) quick horizontal eye movement

(2) quick vertical eye movement

(3) eye tracking horizontal direction

(4) eye tracking vertical direction

(5) horizontal head shaking with gazing fixed target

(6) vertical head shaking with gazing fixed target

(7) oblique head tilting with gazing fixed target.

Each eye or head movement was repeated 20 times.

The VSR training consisted of eight static and five dynamic exercises.

**The eight static exercises were:**

(1) standing up and sitting down with eyes open, three times

(2) standing up and sitting down with eyes closed, three times

(3) standing with eyes closed and feet apart for 20 s

(4) standing with eyes closed and feet closed (narrow stance) for 20 s

(5) standing with tandem gait with right foot in front for 20 s

(6) standing with tandem gait with left foot in front for 20 s

(7) one leg stand; on the right foot for 20 s

(8) one leg stand; on the left foot for 20 s.

**The five dynamic programs were:**

(1) 180° turn to the left, three times

(2) 180° turn to the right, three times

(3) walking with tandem gait for 10 m

(4) walking with horizontal head shaking for 10 m

(5) walking with vertical head shaking for 10 m.

During education, patients performed these exercises three times a day under the supervision of a physician. After 5 days, all patients had learned how to perform the exercises. The patients were then instructed to continue performing the vestibular rehabilitation program three times a day after discharge **(36).**

Vestibular rehabilitation (VR) restores homeostasis in the vestibular system, and these adaptation mechanisms allow the treatment of symptoms and a stable posture in the long term. A Cochrane Database Systematic Review published in 2015 concluded that there is moderate to strong evidence supporting VR in managing patients with vestibular hypofunction for reducing symptoms and improving function **(37).**

VR improves balance skills and increases self-confidence, increasing patient's activity and quality of life. **Wrisley et al. (39)** retrospectively evaluated 30 patients with vestibular impairment with or without migraine headache who treated with VR. They applied general strengthening and stretching exercises, canalith repositioning technique, exercises using different sensory inputs to increase balance control to these patients. They reported that both groups of patients benefited clinically from VR. Recovery occurred in 88% of patients with migraine and 89% of patients with non-migraine. **Vitkovic et al.** evaluated the effect of VR in dizzy patients with and without migraine. In both subjective and objective measurements, VM patients benefited the same as the other patient group **(32).**

To evaluate the effect of VR, the results of pretreatment and post-treatment Dizziness Handicap Inventory (DHI) scores (a

standard questionnaire that quantitatively evaluates the degree of handicap in patients' daily lives with vestibular disorders) **(38)**

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