

Vestibular Rehabilitation Using Optokinetic Stimulation

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Introduction

Visual vertigo (VV), whose symptoms are brought on or made worse by an abundance of or disorienting visual stimuli, can occur in people with vestibular dysfunction (e.g., supermarkets). It is thought that people with VV depend too much on visual information for balance (i.e., visually dependent). When certain vestibular rehabilitation exercises and exposure to optokinetic stimuli are coupled, VV can be considerably improved. However, it is challenging to implement these strategies into routine clinical practise where exercises may be performed unsupervised due to the frequency of treatment sessions (twice weekly for 8 weeks) and the equipment utilised (expensive and space-consuming). This focused review's goals are to give an overview of recent findings looking at (a) how people with vestibular deficits respond to a tailored exercise programme that includes exposure to optokinetic stimuli through a "high-tech" visual environment rotator or a "low-tech" DVD with and without supervision, and (b) the mechanism of recovery. Additionally, additional recent advancements in vestibular rehabilitation methods and upcoming research will be presented in relation to optokinetic stimulation [1].

Description

The current accepted standard of therapy for people with peripheral vestibular diseases, regardless of age or length of symptom duration, is the use of exercise in the form of appropriate motions and sensory exposure (i.e., vestibular rehabilitation). With notable improvements in subjective symptoms, dynamic visual acuity, gait, and postural stability, customised vestibular rehabilitation is more beneficial than a general exercise programme (such as the Cawthorne-Cooksey exercise). In between 50 and 80 percent of people who complete a personalised vestibular rehabilitation programme, symptoms and postural stability improve. Complete healing, on the other hand, is less frequent and happens in roughly all cases. It is unclear why some people with peripheral vestibular disorders do not completely recover [2].

In settings involving visual-vestibular conflict or severe visual motion stimulation, people with peripheral vestibular disorders may feel discomfort, postural instability, and symptoms of dizziness, light-headedness, and/or confusion (i.e., supermarket aisles, crowds, watching moving scenes, and driving on highways). Visual vertigo (VV), space and motion discomfort, visual vestibular mismatch, and drivers' disorientation syndrome are all terms used to describe this problem. Studies have demonstrated that people with VV experience verticality perception and postural stability differently from people with vestibular dysfunction who do not have VV. People with VV experience verticality perception and postural stability differently. As a result, VV patients depend too much on visual signals for perception and postural reactions (ie, visually dependent). Particularly in settings generating visual-vestibular conflict, where there is a discrepancy between visual and vestibular data about

movement and orientation, it is thought that this overreliance on visual signals is one of the causes of inadequate vestibular compensation (i.e., 1 sensory system is indicating movement and the other is not) [3].

Desensitizing patients by gradual, regulated exposure to events and motions that cause symptoms is one of the goals of vestibular rehabilitation. Exercises are designed to test a person's capacity to maintain balance when one or more sensory inputs are unavailable or inaccurate, which occurs when the ability to choose the proper sensory input for postural stability is impaired. Exercises that entail inaccurate, conflicting, or non-existent visual input are used with those who have a visual reliance to teach them to rely more on proprioceptive and accessible vestibular cues. VV patients in particular would benefit from rehabilitation programmes that encourage desensitisation and improved tolerance to visual stimuli by exposure to visual motion (i.e., optokinetic stimulation).

In earlier research, postural stability and optokinetic nystagmus significantly improved after 6 weeks of exposure to optokinetic stimulation in people with unilateral or bilateral peripheral vestibular disorders. However, these trials lacked a control group and did not evaluate the type or severity of symptomatology. Additionally, patients were in a stage of illness known as subacute, from which the majority of people will recover either naturally or with traditional treatment. As a result, it was unable to determine if exposure to optokinetic stimulation offered any additional advantages over individually tailored vestibular rehabilitation alone and, if so, which subset of patients would gain the most from this kind of therapy. Basically, it was unclear if visual motion and visual-vestibular conflict stimulation were clinically effective. These problems were examined in a study that contrasted patients' responses between two regimens: one that only included exposure to optokinetic stimulation using whole-body or visual environment rotators, and the other that also included it. The results demonstrated that in patients with persistent peripheral vestibular symptoms, personalised vestibular rehabilitation using optokinetic stimuli was more advantageous for reducing vertigo, postural instability, and particularly VV symptoms.

However, it was challenging to translate this rehabilitation approach from a research setting into routine clinical practise due to the 8-week, twice-weekly therapy sessions and the equipment required (i.e., custom made, expensive, and space consuming). Additionally, no measurements of visual reliance were recorded, and no recovery mechanism could be found. This focused review's goal is to give a summary of recent research on (a) the use of high-tech versus low-tech optokinetic stimulation and the importance of supervision, as well as (b) the mechanism of recovery. The topic of optokinetic stimulation is also covered in relation to upcoming research and other recent advancements in vestibular rehabilitation methods [4].

Mechanism of vestibular recovery

After exposure to optokinetic stimulation, changes in the gain of the vestibulo-ocular reflex have been seen in monkeys, healthy people, and those with chronic peripheral vestibular disorders, among other changes in particular vestibular parameters. In healthy people, exposure to repetitive vestibular or optokinetic stimulation also shortens the time that postrotational vestibular sensation lasts. There have been no studies done to date that compare visual dependency measures before and after rehabilitation, despite the fact that it is thought that improvements in VV symptoms observed in people with peripheral vestibular disorders after exposure to optokinetic stimulation are caused by a decreased reliance on vision for perceptual and postural responses.

A recently concluded study used healthy volunteers to measure visual reliance before and after repeated exposure to optokinetic stimuli in order

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to better explore this issue. 38 participants were divided at random into two groups: the intervention group, which had graded exposure to visual motion stimuli for five days straight, and the control group, which underwent no intervention. After baseline and at the conclusion of the intervention, static and dynamic elements of perceptual preferences for spatial orientation and postural sway measures with eyes open and closed, as well as in the presence of visual motion stimuli, were measured. Measurement of visual dependence before and after repeated exposure to optokinetic stimuli in healthy subjects was the goal of a recently completed study that sought to shed more light on this problem. 38 participants were divided at random into two groups: one received an intervention that involved graded exposure to visual motion stimuli for five days straight, while the other received no intervention at all. At baseline and after the intervention, measurements of perceptual preferences for spatial orientation and postural sway were made with and without the eyes open, as well as in the presence of visual motion cues. Small-field optokinetic stimulation results in deactivation of parietoinsular vestibular cortices and activation of cortical regions involved in processing visual motion and controlling eye movement. Processing of horizontal and vertical optokinetic stimulation also involves neuronal substrates in the cerebellum and brainstem [5].

Conclusion

In order to improve dizziness, postural instability, and especially VV symptoms in those with persistent peripheral vestibular symptoms, customised vestibular rehabilitation integrating optokinetic stimuli is preferable to vestibular rehabilitation without optokinetic stimuli. However, it is challenging to integrate this rehabilitation approach into routine clinical practise due to the usage of cutting-edge equipment and the frequency of therapy sessions. A practical,

affordable, and efficient way to use optokinetic stimulation in vestibular rehabilitation regimens is the visual motion DVD. To increase compliance, postural stability, and psychological state, it is proposed that some kind of supervision is necessary. Improvements following exposure to optokinetic stimuli are thought to be the result of flexible, adaptive changes in the degree of visual dependence at both the perceptual and postural levels. Therefore, it is recommended that exposure to visual motion stimuli be used in the therapy of visual dependence in both people with and without vestibular impairment. Future studies will be necessary to determine the best treatment duration, stimulation, and long-term benefit.

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