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REVIEW ARTICLE

The application of subjective visual vertical in balance system disorders

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Abstract

Background and Aim: According to the guidelines approved by international scientific associations, the assessment of the vestibular system and its rehabilitation is carried out by audiologists. The subjective visual vertical (SVV) test, while simply done, is an appropriate test for assessment of otolithic system. This review study has tried to introduce the application of SVV test in different disorder of the vestibular system based on the clinical research in various resources.

Recent Findings: We searched the subject of SVV in various databases from 1900 up to 2017. We used keywords of subjective visual vertical and SVV. 110 papers were found and 78 of them were selected. The application of SVV in balance system diseases was reviewed based on these original articles and three related text books.

Conclusion: The SVV test in the Pusher syndrome, migraine and the chronic phase of Meniere's disease did not statistically differentiate between two groups of normal and abnormal people. However, the SVV deviation in individuals with pusher syndrome was higher than normal subjects. In Parkinson's disease, vestibular neuritis, vestibular schwannoma, Pisa syndrome, benign paroxysmal positional vertigo, aging, hydrocephalus, chronic neck pains, acute phase of Meniere's disease and multiple sclerosis, the SVV test was able to separate normal subjects from abnormal subjects. The method and procedure for performing the SVV test will have a significant impact on the test result. Therefore, it is necessary to standardize the method of performing the SVV test for each particular disease.

Keywords: Vestibular function tests; visual perception; subjective visual vertical; vestibular labyrinth; vestibular diseases

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Introduction

Since linear accelerations are received by the otolithic organs of the labyrinth, these organs play a key role in the perception of gravity, especially verticality [1]. As a result, we can evaluate otolith organs by evaluating the subjective visual vertical or gravitational vertical. The subjective visual vertical (SVV) test has been designed for estimation of mental perception of verticality [2]. This test is an easy, low-cost, simple and reliable test [3]. Curthoys et al., showed a correlation between the torsional eye position and the SVV deviation level.

Therefore, the findings indicate that the SVV deviation is not due to disruption in the mental perception of verticality; rather it can be attributed to ocular torsion [4]. This shows that visual system abnormalities such as rotation around the optic axis of the eye or oblique astigmatism alone can disrupt one's perception of verticality because the first step in setting the SVV lines is the correct formation of the image on the retina [5]. In addition to the otolith organs and the visual system, the proprioceptive sense of the body plays a significant role in one's perception of verticality. Pressure receptors, and receptors in plantar surface of foot and in tendons and muscles provide information to the central nervous system (CNS) about the dynamic and static status of the individual, which is used in one's perception of verticality [6]. To maintain balance, it is necessary to create integrity in the vestibular, visual, and proprioceptive inputs. These inputs converge in the posterior insular vestibular cortex (PIVC) [7,8]. Therefore, all of these three inputs are effective in our perception of verticality, and there is a mutual relationship between these inputs; that is, disruption in one input causes the other inputs to play a more important role in maintaining the balance [9]. Given the role of the multisensory (otolithic, visual and proprioceptive) system in perception of verticality, and a range of disorders that disrupt the functioning of these systems, and also considering the convenience and low-cost of the SVV test, investigating this test in balance system disorders can play a significant role in the interpretation and clinical application of this test. The purpose of this paper was to review the application of the SVV test in balance system disorders to increase the importance and necessity of performing this test in audiology clinics.

Having searched in the sources published regarding the SVV test from 1900 to 2017, we reviewed 110 relevant articles with keywords SVV, Subjective visual vertical, and other related terms from Google Scholar, Proquest, Pubmed, and Sciencedirect and selected 78 research papers among them. We have used those research papers and three related books in this review paper.

Before discussing the application of the SVV test in balance system disorders, we need to provide the reader with information on how to perform the test and about the parameters affecting this test. In the next sections of this paper, we will review the SVV test in two general groups of peripheral and central lesions, multiple sclerosis, vestibular schwannoma, Pusher syndrome, hydrocephalus**,** vestibular neuritis, Parkinson's disease, Pisa syndrome, Meniere's disease, benign paroxysmal positional vertigo (BPPV), migraine, and chronic neck pain, and the impact of aging on the SVV test.

SVV test procedure and parameters influencing it

In the SVV test, a person sits in a dark room, free from any visual symptoms and in front of light bars, which he/she is supposed to set these lines based on his/her perception of verticality (gravitational vertical). When this line is being set, there should be no visual cues in the test environment [2]. Normal people perform this task well, so that they set the lines with 1 to 2 degrees deviation of the vertical plane [10,11]. Today, it is also possible to perform a SVV test with computer systems, in which the individual sets the light bars using the mouse or the keys on the computer keyboard [12]. Fig. 1 shows an example of performing the SVV test in an environment free of any visual cues.

We can also use the visual rotary background when performing the SVV test. In SVV, the visual stimuli that rotate around the straight line cause a greater SVV deviation in the direction of the rotation of visual stimuli, which can be due to the central visuo-vestibular interaction [13,14]. However, the steady and large deviation of the SVV lines is seen when a person is lying to one side at an angle of 80 to 90 degrees and setting the SVV light bars vertically. In this situation, normal people set the light bars with angles of 10 to 30 degrees deviation of the vertical plane and in the direction they are lying, which is known as the "Aubert" or A-effect [15]. A-effect seems to be more related to the contrast between the somatosensory sense and the vestibular system [16], so that through the vestibular system, a person tends to set the SVV vertically, but based on the somatosensory sense, he/she tends to set the SVV along the direction of body tilt [17]. A study conducted by Bronstein et al., investigated the effect of body tilt and moving background in the SVV test [18]. In this study, eight patients with bilateral vestibular loss were asked to set the SVV light bars with and without a moving background. They were also asked to set the SVV light bars while sitting straight or lying on the right side. It was observed that in cases where subjects were sitting up straight with a static background, there was no difference between the patients with bilateral vestibular loss and the normal subjects. In cases where the background was dynamic or animated, a larger deviation was observed in the SVV lines of the patients with bilateral vestibular loss, which can be due to the lack of the otolith input and involvement of the visual system. Further deviations in the SVV lines were observed when the patients with bilateral vestibular loss were lying to one side, so that the mean magnitude of this deviation was 40 degrees to the right of the vertical axis (A-effect). The findings indicate that in these individuals, the increased A-effect is due to the effect of lying down to one side and has a somatosensory origin. In order to confirm the effect of the somatosensory sense on SVV, we can mention Bronstein's study in which a woman with a history of right thalamic ischemic stroke was suffering from an abdominal hemiaesthesia on the left side. This person set the SVV lines within the normal range when sitting straight. Moreover, when this person was lying to the right and set the SVV lines, the A-effect was within the normal range (18 degrees of deviation), but when he/she was lying on the left arm, the mean SVV deviation and the variance of settings were greater [1].

SVV test in general classification based on peripheral and central lesions

Patients with peripheral unilateral vestibular loss set the SVV luminous lines with more deviation to the lesion side [19], which is probably due to ocular tilt in the eye of the lesion side, so that if the lesion is in the acute phase, the deviation of lines will be from 8 to 10 degrees, and the rate of deviation of SVV lines will gradually decrease over time in a few weeks and months as a result of vestibular compensation, [3,4]. The recovery period of the light bar deviations in the SVV test is within the normal range in some people, while this recovery period is longer in some others. There are also reports saying that the light bar deviations of the SVV test in people with complete unilateral vestibular loss may even last for more than two years [20]. Halmagyi et al. examined a patient with unilateral vestibular acute dysfunction. They reported that if a person suffers from an acute peripheral vestibular dysfunction of left labyrinth, then they would see leftward ocular counter rolling of their eyes, leftward head tilting, and skew eye deviation (the right eye's moving upward and the left eye's moving downward), which is referred to as ocular tilt reaction [21]. One of the main limitations of this test is its low sensitivity in identifying the patients with chronic peripheral vestibular disorder [3]. In addition, the deviation of light bars in the SVV test can also be seen in central lesions [22]. Vestibular central lesions, especially brainstem lesions, cause more and longer deviations in the SVV [23]. Unlike peripheral lesions, in central lesions, the patient can set the light bars tilted without ocular tilt [24]. Vestibular nuclear lesions cause ipsilateral deviation in the SVV, because skew deviation can be seen in these lesions so that the lesion-side eye moves downward and the opposite eye moves upward (skewed eye deviation with the lower eye ipsilesional). However, higher lesions of the brainstem cause contralateral deviation in the SVV lines, because the lesion-side eye moves upward and opposite eye moves downward in this lesion (skewed eye deviation with the upper eye ipsilesional) [12]. The hemisphere lesions can also cause SVV line deviations. Although hemisphere lesions cause more head tilt, the deviations of SVV lines in these lesions are less than those of the peripheral otolith organ lesions. The reason for the deviation of SVV lines in hemisphere lesions can be attributed to the involvement of meso-diencephalic subcortical areas, which causes ocular torsion [13]. Because the ocular and proprioceptive systems are also involved in determining the extent of SVV deviations, we can say that the reason for the deviation of the SVV lines in cortical lesions is disruption in the central multisensory integration, rather than the lesion of the central vestibular pathways [24].

Multiple sclerosis

Multiple sclerosis (MS) is a chronic and progressive demyelinating disease with spontaneous nystagmus and dizziness symptoms usually taking less than an hour [25]. The standard test for assessing the movement of the eyes in MS disease involves assessing the range of the eyes' movement and the gaze-evoked nystagmus [26]. However, we can get more information on brainstem and cerebellum function in these patients by looking at the dynamic movements of the eyes [27]. In fact, we can obtain useful information on the brainstem and cerebellum function of MS patients by focusing on the dynamic aspects of eye movements [28], because a network of neurons in the brainstem and cerebellum is constantly monitoring the neuronal activity of the eyes [29]. Therefore, the use of saccade test (in order to assess velocity and accuracy of eye movements) and vestibulo-ocular reflex in these patients can provide useful information on brainstem and cerebellar involvement. Serra et al., conducted a study on 50 patients with MS using saccade, head impulse (HIT) and SVV tests. The results indicated that SVV deviations were higher in 36% of the patients (which had shown poor results in saccade and head impulse tests) than in normal subjects [27]. Therefore, the interpretation of the results of the SVV test with the saccade and HIT can provide useful information on brainstem lesions in MS patients.

Vestibular schwannoma

Vestibular schwannoma is a benign tumor that originates from the superior and inferior vestibular nerves [30]. Patients with vestibular schwannoma have a wide range of auditory and vestibular disorders including delay or absence of V wave in the auditory brainstem response, reduction or absence of caloric response and vestibular evoked myogenic potential (VEMP) [31]. For this reason, Ushio et al., investigated the relationship between the results of the SVV test and the caloric and VEMP tests in patients with vestibular schwannoma [20]. In their study, 77.5% of the patients with vestibular schwannoma adjusted the SVV lines towards lesion side with more deviations than did the normal subjects. Among the patients with vestibular schwannoma, those who had obtained abnormal results in the SVV test showed more abnormalities in the caloric and VEMP tests than those who had obtained normal results in these tests, because the asymmetry of the utricle on both sides and its neural pathways (superior vestibular nerve) was examined in the SVV test [32]. In addition, the horizontal semicircular canal (SCC) and its neural pathway (superior vestibular nerve) is examined in the caloric test, and saccule and its neural pathway (inferior vestibular nerve) is examined in the VEMP test [33]. Therefore, if the caloric and VEMP tests had abnormal results, it would be more likely to have abnormalities in the SVV test. As a result, it can be said that there is more vestibular nerve dysfunction in patients with vestibular schwannoma who have shown abnormal results in the SVV test.

Pusher syndrome

Pusher syndrome is caused by damage to the right or left brain hemispheres. In this syndrome, patients actively bend themselves away from the damaged hemisphere, causing them to lose their postural control. Studies on patients with Pusher syndrome show that the perception of the body position changes to gravity in these patients, as these patients think that their body is erect, while their bodies are actually bent toward the damaged hemisphere. In contrast, patients with Pusher syndrome do not show any impairment in the processing of visual and vestibular inputs [34]. Johannsen et al. reported that 15 patients with severe Pusher syndrome set the light bar in the SVV test with a ±3.2 degree of deviation (3.2 ± 4.8), while those without brain damage set this light bar with a deviation of 1.2 degree [35]. The study suggests that, although the light bar deviations of people with severe Pusher syndrome were higher than those of healthy subjects, the difference between these two groups was not statistically significant. This finding was inconsistent with the SVV test results obtained from people with peripheral and central damage of vestibular system (without Pusher syndrome). For example, in the unilateral damage of otolith organ, the SVV deviates approximately 10 degrees towards the damaged part [35].

Hydrocephalus

Normal pressure hydrocephalus (NPH) is a kind of neurological abnormality that occurs due to excess fluid accumulation in the brain [36]. The most common symptom in NPH patients is gait impairment. These patients take slow, short and loose walks with less flexion in the knee joint. Not only their walking, but also most motor activities are slow in these patients and disequilibrium is one of the most important signs of the disease [37,38]. NPH patients with closed eyes are relatively better in postural function than those with open eyes, which indicates a malfunction in the integration of their visual and postural afferents [39,40]. In the study conducted by Wikkelso et al., the SVV test was performed on two groups of hydrocephalus (the first group with aqueduct stenosis and the second one was NPH) and it was found that those with hydrocephalus obtained abnormal results in the SVV test [40]. Disturbance in the integration of the multisensory central system can be attributed to the abnormal results in the SVV test.

Vestibular neuritis

Vestibular neuritis is the second most common cause of peripheral vestibular vertigo. The cause of this disease is the infection caused by herpes simplex virus type 1 [41]. Vestibular neuritis is accompanied by acute and unilateral reduction of peripheral vestibular system function. In this disease, the auditory system has normal function and only the vestibular system is involved. Symptoms of this disease include spontaneous nystagmus with a fast phase toward the healthy ear, reduction or removal of caloric responses in the affected ear, and the patient's false perception of verticality, so that in the acute phase of the disease, patients tend to set the light bars towards the affected ear [42]. Vibert et al. evaluated the correlation between the caloric test and the SVV test in 55 patients with unilateral performance reduction of peripheral vestibular. Their results indicated that there was no correlation between abnormal results in the SVV test and unilateral weakness of caloric test [43]. Moreover, Böhmer and Rickenmann also studied the association between the caloric test and the SVV test in 14 patients with vestibular neuritis and showed that there was a correlation between the abnormal results in the SVV test and the patients' severity of disequilibrium, but there was no correlation between abnormal results in the SVV test and unilateral weakness of caloric test, so that the results of the SVV test returned to the normal range in all patients 24 months after the onset of the symptoms as a result of the central compensation mechanisms. However, 71% of the patients still showed a unilateral weakness in the caloric test [44]. We will review the recovery process in the tilted light bar during the central compensation period in the following paragraph.

The functional magnetic resonance imaging (fMRI) shows that vestibular information is often processed by the parietal cortex of the non-dominant hemisphere and PIVC on the same side [42,8]. The parietal cortex of the non-dominant hemisphere contributes to the assessment of the position of the body in space relative to other objects [45]. It should be noted that the parietal cortex of the non-dominant hemisphere lies in the right hemisphere in left-handed people, while it lies in the left hemisphere in right- handed people. PIVC is also involved in the processing of multisensory inputs [46] and seems to be directly involved in the evaluation of SVV and the stroke of this region triggers disequilibrium and tilt of the light bar in the SVV test. After the stroke, it takes 6-12 months for the results of the SVV test to improve [42].

The PIVC is often activated in a coordinated manner with the vestibular stimulated side; therefore, through the stimulation of the right-hand vestibular in the left-handed people, the parietal cortex of the right (non-dominant) hemisphere and the right PIVC will be involved in processing the information, but note that in the vestibular neuritis, the PIVC of the opposite side will be activated by removing the neural activity of the involved side. Therefore, in left-handed subjects with right-sided vestibular neuritis, the parietal cortex of the right (non-dominant) hemisphere and the left-hand PIVC will be involved in the processing of information. Thus, the role of corpus callosum will be more prominent in improving the results of the SVV. As a result, the recovery speed in the SVV results seems to depend on the damaged part and left-handedness or right-handedness (body laterality) of the patient. It should be noted that improvement in the tilted light bar in the SVV test occurs faster in left-handed subjects than in right-handed people, since left-handed people have a wider corpus callosum than the right-handed [42]. A study carried out by Toupet et al., investigated the relationship between SVV recovery and body laterality in vestibular neuritis and showed that the recovery speed in the left-handed subjects was independent of the damaged side due to more neurological involvement of corpus callosum, whereas the recovery period in the right-handed subjects was much slower in the right-sided neuritis than in the left-sided vestibular neuritis [42].

Parkinson's disease

Clinical diagnosis of Parkinson's disease is based on akinesia or bradykinesia along with one or more of the main symptoms such as tremor, rigidity, or postural instability [47]. It is now widely known that Parkinson's disease not only affects motor characteristics but also affects non-motor characteristics. These non-motors characteristics may include visual sense, olfactory sense, cognition, autonomic system, and affective function [48]. In addition, the increased visual dependency has been reported in these subjects [49]. Studies that have evaluated vertical perception using the SVV emphasize that the abnormal alignment of the luminous line with verticality in this test may be related to the severity of the disease or indicate the increased visual dependency in this disease [50]. Another study showed that visual impairment in these subjects may have a role in patients' disability in Parkinson's disease by affecting their cognition and locomotion [51]. A study by Pereira et al. showed that Parkinson's patients have a disturbance in perception of verticality and have defects in processing of graviceptive pathways. The disturbed perception of verticality in these patients is associated with instability and severity of the disease. Patients with severe instability showed a higher mean of deviations in the SVV test [52]. They also found that perceptual errors correlate with muscle tremor and rigidity, but they do not correlate with bradykinesia, so basal ganglia disorder can cause perception disorders [52]. Another study investigated the SVV test in two cases of tilted and untilted frame in Parkinson's and healthy subjects. Its results indicated that there is no statistical difference between these two groups of people when the tilted frame was used, but with untilted frame, the two groups will differ in terms of the degree of SVV deviation. However, this difference is not significant enough to be considered in the clinical field [53].

Pisa syndrome

Long-term administration of antipsychotics sometimes causes a persistent dystonia of the trunk, known as Pisa syndrome. In this syndrome, the longitudinal axis of the body bends to the lateral side [54]. Pisa syndrome is often associated with long-term treatments with antipsychotics. However, there are reports about the lower incidence of this disease as a result of taking cholinesterase inhibitors [55] and antiemetics, neurodegenerative disorders, and without any idiopathic cause [54]. Parkinson's disease may also be associated with Pisa syndrome. Parkinson's disease changes the perception and integrity of somatosensory information (visual dependency and body position analysis in space) [56]. Scocco et al. studied patients with

Pisa syndrome in order to determine whether this tilt of body is related to SVV deviation or not, and if it is associated with it, is this light bar deviation in the SVV test due to the tilted body or due to an impairment in spatial perception? This study showed that people with both Parkinson's disease and Pisa syndrome show deviation in SVV in comparison with the normal group in SVV, but this deviation is not due to the body's tilt, and is due to a spatial perception defect [56].

Meniere's disease

Among otolith organs, utricle role is more pronounced than saccule in SVV results [21,57,58]. Therefore, an SVV test to examine the unilateral function of utricle would be a good test [59,60]. Because Meniere disease is often unilateral and utricle might be involved in this disease, it is expected that in the SVV test could be affected in the acute phase of this disease [61]. Accordingly, Kumagami et al. studied 22 patients with unilateral Meniere; all of them had spontaneous nystagmus and disequilibrium. In this study, SVV test was performed before, during and after the acute phase of Meniere disease. Of the 22 patients with the Meniere disease, 14 patients who were in the acute phase of Meniere's disease showed abnormal results in the SVV test while 13 patients out of these 14 patients had adjusted the SVV lines to the damaged side. A few weeks after the acute phase of the disease, the adjustment of SVV lines returned to normal values in 12 patients out of 14 patients. Although both horizontal SCC and utricle are innervated by the superior vestibular nerve, this study indicated no correlation between the abnormal results of the SVV test and the unilateral weakness in the caloric test. It can thus be said that the function of horizontal SCC and utricle may be differently affected by Meniere disease [62].

Benign paroxysmal positional vertigo

The SVV test in patients with BPPV also undergoes changes that can be due to the direct stimulation of vertical SCCs and the skewed eye deviation and ocular torsion, or can be due to the decrease in the density and weight of the macula following the movement of the otoconia into the SCCs [63,64]. Therefore, SVV will be an appropriate test for the evaluation of the function of the otolith organ in case the skewed eye deviation and ocular torsion is due to damage to the otolith and its neural pathways [64]. Bending toward the damaged side in BPPV is a natural occurrence, which can be due to the decreased otoconia, the reduced density and macular mass, and consequently the reduced receptor function. Considering the significant changes in SVV following therapeutic maneuvers, there is significant prediction which may be related to the return of otoconia to utricle [63].

Migraine

Migrainous patients are more prone to dizziness and motion sickness than non-migrainous patients [65,66]. In motion sickness, the patient experiences nausea in case of motion, especially when the motion is performed with a vehicle (car, ship, etc.). The cause of motion sickness seems to be the weight difference of the otoconias in the utricle on both sides [67]. Since the SVV test is affected by the asymmetric operation of the utricle in two sides, this led Crevits et al. to investigate whether migraineurs also have a problem with their perception of verticality or not. In this study, SVV was performed on 47 patients with moderate to severe migraine and 96 normal subjects. The results showed that the deviation of SVV lines was not statistically significant in two groups [68]. However, Asai et al. examined the SVV test on 16 normal subjects and 17 migraineurs. They reported that the deviation of SVV lines in migraine subjects (1.5 ± 1.2) was significantly higher than normal subjects (0.6 ± 0.4). However, SVV deviations in both groups were within the normal range [69].

Chronic neck pain

Chronic neck pain (CNP) has several symptoms including a reduction in postural control [70]. Neck pain is typically caused by damage to the neck's zygapophyseal joints. Since these similar structures may affect the perception of spatial orientation, it may cause vertigo, dizziness, or disturbances associated with proprioceptive receptors stimulation [71]. Neck joints and segmental muscles have nerve endings of several pain receptors. These nociceptive receptors of pain have a detrimental effect on the regular function of sensory receptors [72]. Such disturbances may create difficulty for one's perception of verticality and cause some disturbed feelings of spatial orientation in patients with neck pain [71]. Studies performed on CNP patients have shown that these patients have larger errors in the SVV test than normal people [71,73]. A study conducted by Docherty et al. with the aim of comparing the errors of CNP patients in the SVV test with those of normal people showed that the range of the patients' errors was similar to that of the healthy subjects for the two tests in the absence of a frame and in the presence of an untitled frame, but significant differences were observed in these two groups in the presence of a tilted frame [74]. It seems that there is an increased dependence on visual signals in the group of CNP patients, which is consistent with the findings of previous studies on patients with Parkinson's disease or unilateral and bilateral vestibular loss [18,75]. The neck proprioceptive receptors have been shown to contribute to the perception of SVV [70,76]. The current observations may be interpreted in such a way that if the neck pain is associated with disturbance in the input of the neck proprioceptive receptors, it brings about a change in the balance and interaction between sensory systems which the brain normally uses in order to determine the SVV, and it increases the patient's reliance on the visual input [74]. A study was conducted to test the hypothesis that if the neck pain is accompanied by disturbance of the unilateral neck proprioceptive receptors and is compensated by increased reliance on the visual input, then asymmetry will be created in the SVV results when the frame is tilted ipsilaterally or contralaterally. However, the results did not support this hypothesis, since there was no significant difference between the errors with the ipsilateral tilt of the frame and those with the contralateral tilt.

Aging

The involvement of various sensory systems in postural stabilization varies with age [77]. Additionally, anatomical [78] and physiological changes associated with age have been shown in the vestibular system [79]. In a study carried out on astronauts, it has been shown that the relative involvement of the visual input increases significantly in comparison with the vestibular and proprioceptive inputs [80], which indicate the role of plasticity in the involvement of different senses in determining the SVV. Similarly, the reduction of vestibular inputs due to age-related disorder leads to the increased dependency on the visual system in SVV [81]. Kobayashi et al. conducted a study in order to evaluate the age-related plasticity in the participation of the visual system to determine static and dynamic SVV (with background rotation) in different age groups and concluded that, unlike the static SVV which was similar in the case of all of the subjects with different age groups, dynamic SVV showed a gradual increase in SVV deviation variations with an increase in age, and this increased deviation in background rotation was the same on both sides, which indicates symmetry effect [81]. Therefore, in order to explain these results with an increase in age, the increase in the relative participation of visual input is assumed. Moreover, an increase in the proprioceptive input of neck with an increase in age was also reported. These results were obtained through the measurement of SVV by neck vibration stimulation, which was associated with a gradual weakening of the vestibular nerve fibers as a result of aging [80]. In general, it can be indicated that the function of the Vestibular system decreases with an increase in age, but disequilibrium is not easily affected by aging, as alternative processing systems such as the visual and proprioceptive systems get involved [81].

Conclusion

Due to its ease of use, the SVV test can be used to monitor progressive diseases such as MS, because it is assumed in this test that the light bar deviation is a sign of the malfunction of the brainstem. The findings indicate that the SVV test has lost its value in the chronic phase of the disease, but diseases that are in the acute phase (Meniere's disease and vestibular neuritis) can be identified by this test. It was found that the SVV test has lost its clinical application in the Pusher syndrome. This claim can be substantiated by performing the SVV test on individuals with a head rotation of 15, 30 and 60 degrees in the roll axis, where results are obtained within the normal range, but SVV deviations are significant in central and peripheral vestibular diseases. People with hydrocephalus showed abnormal results in the SVV test due to their poor performance in integrating visual and postural afferents. Visual accuracy decreases in the case of patients with Parkinson's disease, but visual dependency increases in them due to gait impairment. Studies have shown that the SVV test should not be performed inside a tilted frame in the case of these subjects. However, in the case of patients with chronic neck pain, evidence suggests that a tilted frame can be used to differentiate them from normal people. Additionally, if the SVV test is performed in elderly people in a dynamic visual environment, the test's capability will increase. The SVV deviation is not due to the body tilt in the case of the Pisa syndrome; rather it is due to a defect in spatial perception. It should be noted that in the BPPV disease, the light bar in the SVV test deviates to the damage side due to the decreased otoconia, the reduced density, macula spatial weight, and consequently the reduced receptor function. In vestibular Schwanoma patients, the abnormal results of the SVV test indicate more severe damage to the vestibular nerve. The results also indicate that the SVV test has lost its sensitivity in the case of migraineurs. As a result, it can be said that the methods of performing the SVV test significantly affects the test's result. Therefore, it is necessary to modify this test for any particular disease.

Conflict of Interest

The authors declare that they have no conflict of interest.

REFERENCES

1. Bronstein AM. The interaction of otolith and proprioceptive information in the perception of verticality. The effects of labyrinthine and CNS disease. Ann N Y Acad Sci. 1999;871:324-33.
2. Iranfar S, Karimi LJ, Shaabani M, Sharifian Alborzi M, Akbarzadeh Baghban A. Subjective visual vertical test: the effect of preset angle and visual background. J Rehab Med. 2013;2(2):47-54. doi: 10.22037/r.m.v2i2.5582
3. Pagarkar W, Bamiou DE, Ridout D, Luxon LM. Subjective visual vertical and horizontal: effect of the present angle. Arch Otolaryngol Head Neck Surg. 2008;134(4):394-401. doi: 10.1001/archotol.134.4.394
4. Curthoys IS, Dai MJ, Halmagyi GM. Human ocular torsional position before and after unilateral vestibular neurectomy. Exp Brain Res. 1991;85(1):218-25.
5. Van Nechel C, Toupet M, Bodson I. The subjective visual vertical. Adv Otorhinolaryngol. 2001;58:77-87.
6. Stoffregen TA, Riccio GE. An ecological theory of orientation and the vestibular system. Psychol Rev. 1988; 95(1):3-14.
7. Bronstein AM. Evaluation of balance. In: Gleeson M, editor. Scott-Brown's otorhinolaryngology: head and neck surgery, volume 3. 7th ed. London: Hodder Arnold; 2008. p. 3706-47.
8. Lopez C, Blanke O. The thalamocortical vestibular system in animals and humans. Brain Res Rev. 2011;67(1-2):119-46. doi: 10.1016/j.brainresrev.2010.12.002
9. Gans RE, Wende Yellin M. Assessment of vestibular function. In: Roeser RJ, Valente M, Hosford-Dunn H, editors. Audiology diagnosis. 2nd ed. New York: Thieme; 2007. p. 540-63.
10. Neal E. Visual localization of the vertical. Am J Psychol. 1926;37(2):287-91. doi: 10.2307/1413700
11. Howard IP. Human visual orientation. John Wiley & Sons; 1982.
12. Bronstein AM. Tests of otolith function and vestibular perception. In: Jacobson GP, Shepard NT, editors. Balance function assessment and management. 2nd ed. San Diego: Plural publishing; 2016. p. 581-94.
13. Dichgans J, Held R, Young LR, Brandt T. Moving visual scenes influence the apparent direction of gravity. Science. 1972;178(4066):1217-9.
14. Dichgans J, Brandt T, Held R. The role of vision in gravitational orientation. Fortschr Zool. 1975;23(1):255-63.
15. Mittelstaedt H. The information processing structure of the subjective vertical. A cybernetic bridge between its psychophysics and its neurobiology. In: Marko H, Hauske G, Struppler A, editors. Processing structures for perception and action. Weinheim: VCH-Verlagsgesellschaft; 1988. p. 217-63.
16. Yardley L. Contribution of somatosensory information to perception of the visual vertical with body tilt and rotating visual field. Percept Psychophys. 1990;48(2): 131-4.
17. Mittelstaedt H. Somatic versus vestibular gravity reception in man. Ann N Y Acad Sci. 1992;656:124-39.
18. Bronstein AM, Yardley L, Moore AP, Cleeves L. Visually and posturally mediated tilt illusion in Parkinson's disease and in labyrinthine defective subjects. Neurology. 1996;47(3):651-6.
19. Friedmann G. The judgment of the visual vertical and horizontal with peripheral and central vestibular lesions. Brain. 1970;93(2):313-28.

20. Ushio M, Iwasaki S, Chihara Y, Murofushi T. Abnormal deviation of subjective visual horizontal in patients with vestibular schwannoma. Ann Otol Rhinol Laryngol. 2008;117(9):641-4. doi: 10.1177/000348940811700902
21. Halmagyi GM, Gresty MA, Gibson WP. Ocular tilt reaction with peripheral vestibular lesion. Ann Neurol. 1979; 6(1):80-3. doi: 10.1002/ana.410060122
22. Dieterich M, Brandt T. Wallenberg's syndrome: lateropulsion, cyclorotation, and subjective visual vertical in thirty-six patients. Ann Neurol. 1992;31(4):399-408. doi: 10.1002/ana.410310409
23. Dieterich M, Brandt T. Ocular torsion and perceived vertical in oculomotor, trochlear and abducens nerve palsies. Brain. 1993;116 (Pt 5):1095-104.
24. Brandt T, Dieterich M, Danek A. Vestibular cortex lesions affect the perception of verticality. Ann Neurol. 1994;35(4):403-12. doi: 10.1002/ana.410350406
25. Hale T, Trahan H, Parent-Buck T. Evaluation of the patient with dizziness and balance disorders. In: Katz J, Chasin M, English K, Hood LJ, Tillery KL, editors. Handbook of clinical audiology. 7th ed. New York: Wolters Kluwer Health. 2014. p. 399-424.
26. Kurtzke JF. On the evaluation of disability in multiple sclerosis. Neurology. 1961;11(8):686-94.
27. Serra A, Derwenskus J, Downey DL, Leigh RJ. Role of eye movement examination and subjective visual vertical in clinical evaluation of multiple sclerosis. J Neurol. 2003;250(5):569-75. doi: 10.1007/s00415-003-1038-8
28. Downey DL, Stahl JS, Bhidayasiri R, Derwenskus J, Adams NL, Ruff RL, et al. Saccadic and vestibular abnormalities in multiple sclerosis: sensitive clinical signs of brainstem and cerebellar involvement. Ann N Y Acad Sci. 2002;956:438-40.
29. Arnold DB, Robinson DA. The oculomotor integrator: testing of a neural network model. Exp Brain Res. 1997; 113(1):57-74.
30. Ushio M, Iwasaki S, Murofushi T, Sugasawa K, Chihara Y, Fujimoto C, et al. The diagnostic value of vestibular-evoked myogenic potential in patients with vestibular schwannoma. Clin Neurophysiol. 2009;120(6):1149-53. doi: 10.1016/j.clinph.2009.01.017
31. Murofushi T, Curthoys IS, Gilchrist DP. Response of guinea pig vestibular nucleus neurons to clicks. Exp Brain Res. 1996;111(1):149-52.
32. Brandt T, Dieterich M. Pathological eye-head coordination in roll: tonic ocular tilt reaction in mesencephalic and medullary lesions. Brain. 1987;110 (Pt 3):649-66.
33. Sando I, Black FO, Hemenway WG. Spatial distribution of vestibular nerve in internal auditory canal. Ann Otol Rhinol Laryngol. 1972;81(3):305-14. doi: 10.1177/000348947208100301
34. Karnath HO, Broetz D. Understanding and treating "pusher syndrome". Physical therapy. Phys Ther. 2003; 83(12):1119-25.
35. Johannsen L, Fruhmann Berger M, Karnath HO. Subjective visual vertical (SVV) determined in a representative sample of 15 patients with pusher syndrome. J Neurol. 2006;253(10):1367-9. doi: 10.1007/s00415-006-0216-x
36. Bradley WG Jr, Whittemore AR, Kortman KE, Watanabe AS, Homyak M, Teresi LM, et al. Marked cerebrospinal fluid void: indicator of successful shunt in patients with suspected normal-pressure hydrocephalus. Radiology. 1991;178(2):459-66.
37. Blomsterwall E, Bilting M, Stephensen H, Wikkelsö C. Gait abnormality is not the only motor disturbance in normal pressure hydrocephalus. Scand J Rehabil Med. 1995;27(4):205-9.
38. Krauss JK, Regel JP, Droste DW, Orszagh M, Borremans JJ, Vach W. Movement disorders in adult hydrocephalus. Mov Disord. 1997;12(1):53-60.
39. Blomsterwall E, Svantesson U, Carlsson U, Tullberg M, Wikkelsö C. Postural disturbance in patients with normal pressure hydrocephalus. Acta Neurol Scand. 2000; 102(5):284-91.
40. Wikkelsö C, Blomsterwall E, Frisén L. Subjective visual vertical and Romberg's test correlations in hydrocephalus. J Neurol. 2003;250(6):741-5. doi: 10.1007/s00415-003-1076-2
41. Strupp M, Zingler VC, Arbusow V, Niklas D, Maag KP, Dieterich M, et al. Methylprednisolone, valacyclovir, or the combination for vestibular neuritis. N Engl J Med. 2004;351(4):354-61. doi: 10.1056/NEJMoa033280
42. Toupet M, Van Nechel C, Bozorg Grayeli A. Influence of body laterality on recovery from subjective visual vertical tilt after vestibular neuritis. Audiol Neurootol. 2014;19(4):248-55. doi: 10.1159/000360266
43. Vibert D, Häusler R, Safran AB. Subjective visual vertical in peripheral unilateral vestibular diseases. J Vestib Res. 1999;9(2):145-52.
44. Böhmer A, Rickenmann J. The subjective visual vertical as a clinical parameter of vestibular function in peripheral vestibular diseases. J Vestib Res. 1995;5(1):35-45.
45. Borel L, Lopez C, Péruch P, Lacour M. Vestibular syndrome: a change in internal spatial representation. Neurophysiol Clin. 2008;38(6):375-89. doi: 10.1016/j.neucli.2008.09.002
46. Dieterich M, Bense S, Lutz S, Drzezga A, Stephan T, Bartenstein P. Dominance for vestibular cortical function in the non-dominant hemisphere. Cereb Cortex. 2003;13(9):994-1007.
47. Hughes AJ, Daniel SE, Kilford L, Lees AJ. Accuracy of clinical diagnosis of idiopathic Parkinson's disease: a clinico-pathological study of 100 cases. J Neurol Neurosurg Psychiatry. 1992;55(3):181-4.
48. Chaudhuri KR, Yates L, Martinez-Martin P. The non-motor symptom complex of Parkinson's disease: a comprehensive assessment is essential. Curr Neurol Neurosci Rep. 2005;5(4):275-83.
49. Azulay JP, Mesure S, Amblard B, Pouget J. Increased visual dependence in Parkinson's disease. Percept Mot Skills. 2002;95(3 Pt 2):1106-14. doi: 10.2466/pms.2002.95.3f.1106
50. Danta G, Hilton RC. Judgment of the visual vertical and horizontal in patients with Parkinsonism. Neurology. 1975;25(1):43-7.
51. Uc EY, Rizzo M, Anderson SW, Qian S, Rodnitzky RL, Dawson JD. Visual dysfunction in Parkinson disease without dementia. Neurology. 2005;65(12):1907-13. doi: 10.1212/01.wnl.0000191565.11065.11
52. Pereira CB, Kanashiro AK, Maia FM, Barbosa ER. Correlation of impaired subjective visual vertical and postural instability in Parkinson's disease. J Neurol Sci. 2014;346(1-2):60-5. doi: 10.1016/j.jns.2014.07.057
53. Khattab A, Docherty S, Bagust J, Willington R, Thomas P, Amar K. Subjective visual vertical perception and sense of smell in Parkinson disease. J Rehabil Res Dev.

2012;49(6):961-9.
54. Suzuki T, Matsuzaka H. Drug-induced Pisa syndrome (pleurothotonus): epidemiology and management. CNS Drugs. 2002;16(3):165-74.
55. Cossu G, Melis M, Melis G, Maccioni E, Putzu V, Catte O, et al. Reversible Pisa syndrome (pleurothotonus) due to the cholinesterase inhibitor galantamine: case report. Mov Disord. 2004;19(10):1243-4. doi: 10.1002/mds.20164
56. Scocco DH, Wagner JN, Racosta J, Chade A, Gershanik OS. Subjective visual vertical in Pisa syndrome. Parkinsonism Relat Disord. 2014;20(8):878-83. doi: 10.1016/j.parkreldis.2014.04.030
57. Brandt T, Dieterich M. Skew deviation with ocular torsion: a vestibular brainstem sign of topographic diagnostic value. Ann Neurol. 1993;33(5):528-34. doi: 10.1002/ana.410330518
58. Clarke AH, Schönfeld U, Helling K. Unilateral examination of utricle and saccule function. J Vestib Res. 2003;13(4-6):215-25.
59. Fetter M. Assessing vestibular function: which tests, when? J Neurol. 2000;247(5):335-42.
60. Helling K, Schönfeld U, Clarke AH. Treatment of Ménière's disease by low-dosage intratympanic gentamicin application: effect on otolith function. Laryngoscope. 2007;117(12):2244-50. doi: 10.1097/MLG.0b013e3181453a3c
61. Hosseini Fard A, Yazdani N, Rezazadeh N, Sharifian Alborzi M, Akbarzadeh Bagheban A. Subjective visual vertical in patients with unilateral definite Meniere's diseases. Aud Vest Res. 2017;26(2):105-11.
62. Kumagami H, Sainoo Y, Fujiyama D, Baba A, Oku R, Takasaki K, et al. Subjective visual vertical in acute attacks of Ménière's disease. Otol Neurotol. 2009;30(2): 206-9. doi: 10.1097/MAO.0b013e3181925010
63. Faralli M, Manzari L, Panichi R, Botti F, Ricci G, Longari F, et al. Subjective visual vertical before and after treatment of a BPPV episode. Auris Nasus Larynx. 2011;38(3):307-11. doi: 10.1016/j.anl.2010.10.005
64. Jáuregui-Renaud K, Faldon ME, Gresty MA, Bronstein AM. Horizontal ocular vergence and the three-dimensional response to whole-body roll motion. Exp Brain Res. 2001;136(1):79-92.
65. Drummond PD. Triggers of motion sickness in migraine sufferers. Headache. 2005;45(6):653-6. doi: 10.1111/j.1526-4610.2005.05132.x
66. Drummond PD. Effect of tryptophan depletion on symptoms of motion sickness in migraineurs. Neurology. 2005;65(4):620-2. doi: 10.1212/01.wnl.0000172339.15577.a6
67. Scherer H, Helling K, Clarke AH, Hausmann S. Motion sickness and otolith asymmetry. Biol Sci Space. 2001; 15(4):401-4.
68. Crevits L, Vanacker L, Verraes A. Patients with migraine correctly estimate the visual verticality. Clin Neurol Neurosurg. 2012;114(4):313-5. doi: 10.1016/j.clineuro.2011.10.025
69. Asai M, Aoki M, Hayashi H, Yamada N, Mizuta K, Ito Y. Subclinical deviation of the subjective visual vertical in patients affected by a primary headache. Acta Otolaryngol. 2009;129(1):30-5. doi: 10.1080/00016480802032785
70. Karlberg M, Persson L, Magnusson M. Reduced postural control in patients with chronic cervicobrachial pain syndrome. Gait Posture. 1995;3(4):241-9. doi: 10.1016/0966-6362(96)82854-2
71. Grod JP, Diakow PR. Effect of neck pain on verticality perception: a cohort study. Arch Phys Med Rehabil. 2002;83(3):412-5.
72. Bogduk N. Cervical causes of headache and dizziness. In: Grieve GP, editor. Modern manual therapy of the vertebral column. Edinburgh: Churchill Livingstone; 1986. p. 289-302.
73. Bagust J, Rix GD, Hurst HC. Use of a computer rod and frame (CRAF) test to assess errors in the perception of visual vertical in a clinical setting—a pilot study. Clin Chiropr. 2005;8(3):134-9. doi: 10.1016/j.clch.2005.07.001
74. Docherty S, Schärer R, Bagust J, Humphreys BK. Perception of subjective visual vertical and horizontal in patients with chronic neck pain: a cross-sectional observational study. Man Ther. 2012;17(2):133-8. doi: 10.1016/j.math.2011.11.002
75. Hafström A, Fransson PA, Karlberg M, Magnusson M. Idiosyncratic compensation of the subjective visual horizontal and vertical in 60 patients after unilateral vestibular deafferentation. Acta Otolaryngol. 2004;124(2): 165-71.
76. Karlberg M, Aw ST, Halmagyi GM, Black RA. Vibration-induced shift of the subjective visual horizontal: a sign of unilateral vestibular deficit. Arch Otolaryngol Head Neck Surg. 2002;128(1):21-7.
77. Strupp M, Arbusow V, Borges Pereira C, Dieterich M, Brandt T. Subjective straight-ahead during neck muscle vibration: effects of ageing. Neuroreport. 1999;10(15): 3191-4.
78. Rasmussen AT. Studies of the viiith cranial nerve of man. Laryngoscope. 1940;50(1):67-83. doi: 10.1288/00005537-194001000-00008
79. Manchester D, Woollacott M, Zederbauer-Hylton N, Marin O. Visual, vestibular and somatosensory contributions to balance control in the older adult. J Gerontol. 1989;44(4):M118-27.
80. Rosenhall U. Degenerative patterns in the aging human vestibular neuro-epithelia. Acta Otolaryngol. 1973;76 (2):208-20.
81. Kobayashi H, Hayashi Y, Higashino K, Saito A, Kunihiro T, Kanzaki J, et al. Dynamic and static subjective visual vertical with aging. Auris Nasus Larynx. 2002;29(4):325-8.