The Journal of International Advanced Otology

J Int Adv Otol 2024; 20(1): 76–80 • DOI: 10.5152/iao.2024.231181

Review

Frailty of the Vestibular System in the Super-Aging Society

Tadashi Kitahara[1], **Tomoyuki Shiozaki**[1], **Hiroshi Hosoi**[2]

[1]Department of Otolaryngology-Head and Neck Surgery, Nara Medical University, Kashihara, Japan
[2]Medicine Based Town Research Center, Nara Medical University, Kashihara, Japan

ORCID iDs of the authors: T.K. 0000-0001-9598-6287, T.S. 0000-0003-1619-5087, H.H. 0009-0006-0001-8325.

Cite this article as: Kitahara T, Shiozaki T, Hosoi H. Frailty of the vestibular system in the super-aging society. *J Int Adv Otol.* 2024;20(1):76-80.

Vestibular frailty and presbyvestibulopathy, including benign paroxysmal positional vertigo (BPPV), can cause dizziness among elderly patients. Vestibular frailty and presbyvestibulopathy may contribute to the onset of the vicious circle of falling–bone fracture–prolonged bedridden status–senile dementia. Treatment interventions for vestibular frailty and presbyvestibulopathy should be based on vestibular rehabilitation rather than vestibular implantation or regeneration. In acute BPPV, the otolith repositioning maneuver can be used to return otolithic debris to the utricle. At the chronic remission stage, there are nutritional guidelines for improving bone density in otolith organs and rehabilitation guidelines for activating otolith organs to prevent exfoliation. Moreover, sleeping in the head-up position can prevent free-floating debris from entering the semicircular canal. Throughout their old age, the psychiatric care/support is also indispensable to keep their initiative against vestibular frailty.

KEYWORDS: Aging society, benign paroxysmal positional vertigo, peripheral vestibular loss, presbyvestibulopathy, vestibular frailty, vestibular rehabilitation

INRODUCTION

Vestibular Frailty and Presbyvestibulopathy

In the peripheral vestibular system, vestibular otolith end organs sense linear/gravitational acceleration, while the semicircular canals sense rotational acceleration. Acute damage to these organs results in rotatory vertigo or a floating sensation, followed by autonomic symptoms such as nausea and vomiting. Therefore, rehydration and acute-phase symptomatic treatment should be administered, especially to elderly patients with dizziness. According to the disease statistics for vertigo/dizziness at the Vertigo/Dizziness Center at Nara Medical University (Figure 1), most peripheral vestibulopathies in elderly patients are derived from presbyvestibulopathy, including benign paroxysmal positional vertigo (BPPV).[1,2] Accordingly, it is important to address concerns regarding vestibular frailty, including age-related disorders of other organs related to body balancing, in the upcoming super-aging society.

The Bárány Society established the diagnostic criteria for presbyvestibulopathy (Table 1), which were published in the Journal of Vestibular Research in 2019.[3] Subsequently, the Japan Society for Equilibrium Research (JSER) established its criteria and published them in 2021. These diagnostic criteria for presbyvestibulopathy are based on otology/neurotology examination results and are crucial for the implementation of vestibular implantation[4] and vestibular regenerative medicine.[5] Vestibular frailty can be classified as central or peripheral presbyvestibulopathy, which can be further classified into presbycanalopathy and presbyotolithopathy (Figure 2).

Pathology of Vestibular Frailty

Presbycanalopathy has a slow progression involving a gradual decline in the number of hair cells in the semicircular canal. Human autopsy studies have reported a 40% reduction in the number of sensory hair cells in the semicircular canals in patients aged ≥70 years compared with healthy adults.[6] Specifically, the number of type I sensory hair cells markedly decreases with age. An imbalance between bilateral neural activity in the vestibular system lacks clinical manifestations if age-related vestibular compensatory regulation is achieved. However, delayed compensation or decompensation significantly reduces the patient's quality of life (QOL)

Corresponding author: Tadashi Kitahara, e-mail: tkitahara@naramed-u.ac.jp
Received: April 20, 2023 • **Revision requested**: September 19, 2023 • **Last revision received:** September 25, 2023 • **Accepted:** October 1, 2023 • **Publication Date:** January 31, 2024

Available online at www.advancedotology.org

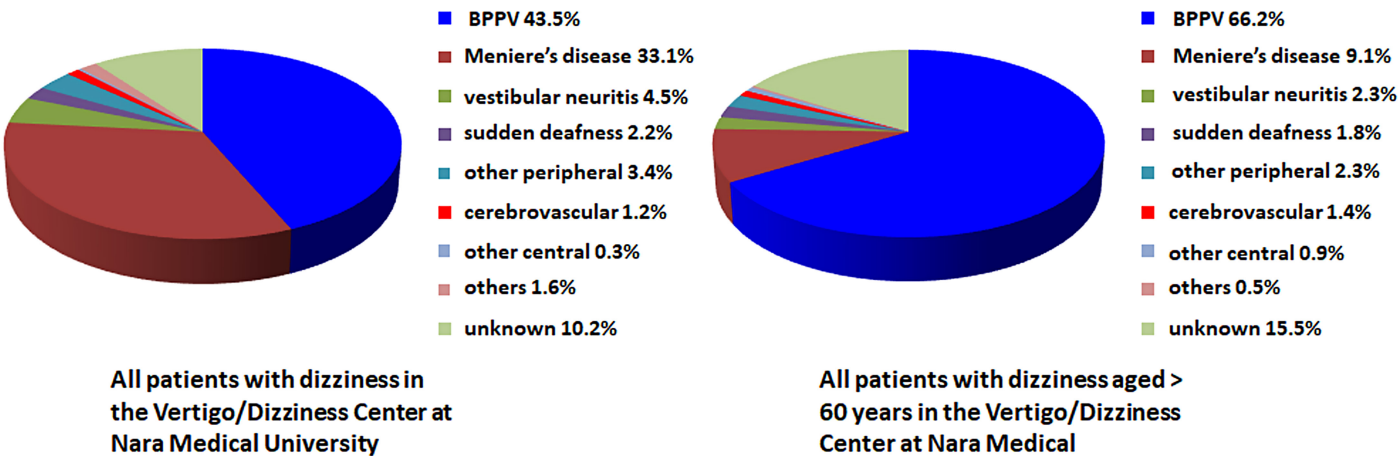

Figure 1. Disease statistics for patients visiting a vertigo/dizziness center at Nara Medical University from May 2014 to April 2018. The circle chart on the left represents the disease statistics for all patients with dizziness in the Vertigo/Dizziness Center at Nara Medical University. The circle chart on the right represents the disease statistics for patients with dizziness aged >60 years in the Vertigo/Dizziness Center at Nara Medical University. Among patients with benign paroxysmal positional vertigo (BPPV) and unknown causes (unknown), including presbyvestibulopathy (PVP), the proportion of PVP reaches up to 50% and 80% on the left and right, respectively.

due to persistent motion-evoked rotatory sensations. Accordingly, presbycanalopathy is exclusively considered a chronic condition.

Presbyotolithopathy is characterized by a gradual decrease in the number of hair cells in the otolith, resulting in a floating sensation due to otolithic deterioration. Human studies have reported a 25% reduction in the number of sensory hair cells in the otolith in patients aged ≥70 years compared with healthy adults.[6] Moreover, a volumetric decrease and morphological changes in otoconia due to aging have been reported.[7] Similar to semicircular canals, successful vestibular compensation does not cause clinical problems; however, decompensation significantly reduces the QOL due to a persistent motion-evoked floating sensation. This condition can be classified as chronic presbyotolithopathy. However, acute presbyotolithopathy may occur due to BPPV, which occurs due to increased otolith exfoliation and migration of the exfoliated otoliths into the semicircular canals. The resulting acute imbalance increases the risk of falls and fractures among elderly patients. Acute presbyotolithopathy can be cured through spontaneous regression of the otolith debris in the semicircular canal or by returning this debris through the otolith repositioning maneuver. Frequent recurrence and/or chronicity of these symptoms significantly reduce the QOL.

Table 1. Diagnostic Criteria for Presbyvestibulopathy

Each of the criteria A through D has to be fulfilled.[3]
A. Chronic vestibular syndrome (at least 3 months duration) with at least 2 of the following symptoms:
1. Postural imbalance or unsteadiness
2. Gait disturbance
3. Chronic dizziness
4. Recurrent falls
B. Mild bilateral peripheral vestibular hypofunction documented by at least 1 of the following:
1. VOR gain measured by vHIT between 0.6 and 0.8 bilaterally
2. VOR gain between 0.1 and 0.3 upon sinusoidal stimulation on a rotatory chair (0.1 Hz, Vmax = 50-60°/s)
3. Reduced caloric response (sum of bithermal maximum peak SPV on each side between 6 and 25°/s)
C. Age ≥60 years
D. Not better accounted for by another disease or disorder

SPV, slow-phase velocity; vHIT, video head impulse test; Vmax, maximum velocity; VOR, vestibulo-ocular reflex.

The number of primary vestibular afferents decreases by 20% between the ages of 30 and 60 years; moreover, the number of vestibular nucleus neurons other than those in the superior nucleus significantly decreases with age.[6]

Vestibular Frailty and Healthy Life Expectancy

Given the upcoming super-aging society, it is important to establish interventions for vestibular frailty and presbyvestibulopathy, which may represent the onset of the vicious circle of falling–bone fracture–prolonged bedridden status–senile dementia, to improve healthy life expectancy and reduce medical costs (Figure 2). A recent review indicated that approximately 40% of the risk factors for dementia were modifiable, including physical inactivity, social isolation due to physical inactivity, and depression due to social isolation, which accounted for 2%, 4%, and 4% of the cases, respectively.[8] Accordingly, prevention of vestibular frailty and presbyvestibulopathy may indirectly reduce the risk of dementia by approximately 10%.

Interventions against vestibular frailty and presbyvestibulopathy should consider the prevention of the age-related decrease in the number of cells in the peripheral vestibular apparatus, cell degeneration, and functional deterioration, as well as deterioration of the central vestibular system and an individual's ability to interact with visual and somatosensory information. The aforementioned age-related deterioration is influenced by both environmental and genetic factors. Environmental factors can be primarily addressed by improving lifestyle habits. For example, it is recommended to correct unbalanced eating habits that lead to arteriosclerosis and smoking habits that cause vasospasms. Moreover, sufficient fluid, calcium, and vitamin D intake is recommended to maintain vestibular end organs, while physical training is recommended to enhance the interaction of the inner ear with visual, somatosensory, and central vestibular

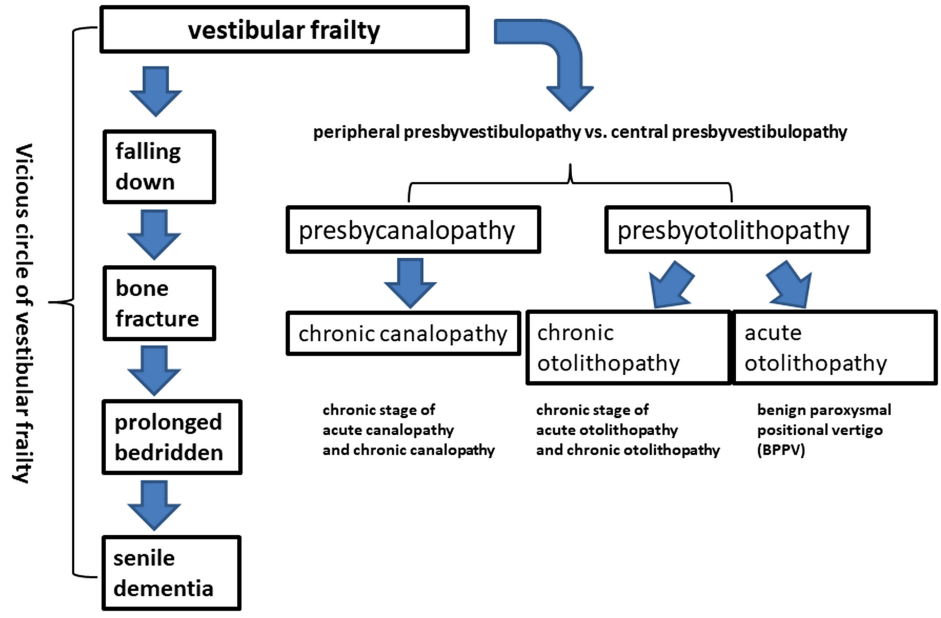

Figure 2. Vicious circle of vestibular frailty in the super-aging society. Vestibular frailty could result in the onset of the following vicious circle: falling, bone fracture, prolonged bedridden status, and senile dementia. Accordingly, vestibular frailty should be managed to improve healthy life expectancy and suppress health-care costs in the upcoming super-aging society.

information. These mitigations environmental factors may also modify genetic factors to minimize cell damage and functional deterioration due to genetic fragility before the establishment of vestibular regenerative medicine.

Treatment of Vestibular Frailty

The treatment of elderly patients with vertigo/dizziness is determined based on the classification of vestibular frailty. In peripheral and central presbyvestibulopathy, there remains no cure for

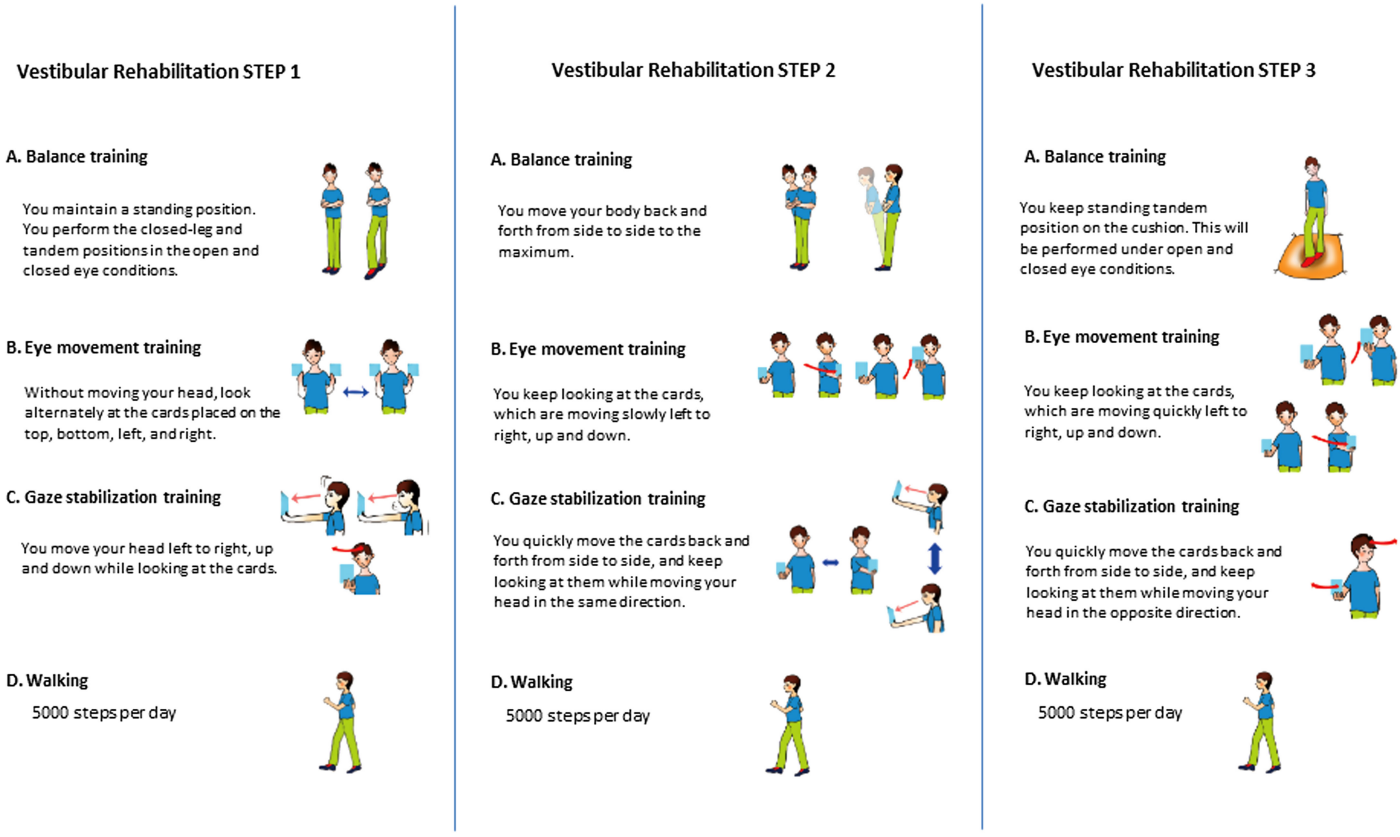

Figure 3. MAHOROBA vestibular rehabilitation. We have established a step-by-step vestibular rehabilitation program termed as MAHOROBA for treating patients with dizziness. MAHOROBA is a word derived from ancient texts in the eighth century of prefecture, Japan, which means a peaceful and wonderful place.[11]

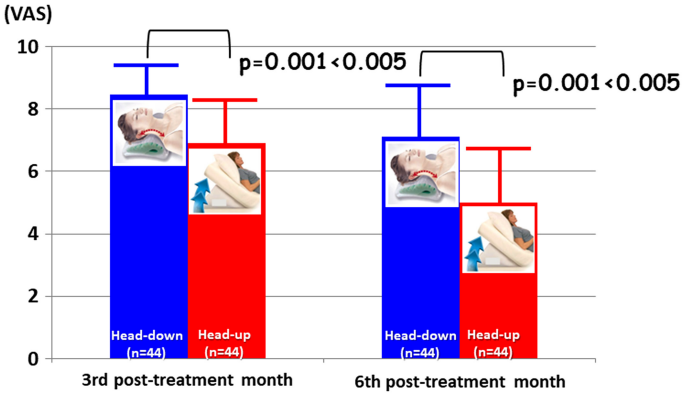

Figure 4. Effects of the head-down vs. head-up sleep position on vertiginous sensation in patients with benign paroxysmal positional vertigo (BPPV). A paired *t*-test confirmed that the visual analog scale (VAS) scores for vertiginous sensation in the head-up sleep group were significantly lower than those in the head-down sleep group at both the third and sixth postintervention months.[17]

aging-related cell damage or functional decline. Therefore, addressing the aforementioned environmental factors is the only prevention intervention for age-related decline. In case this is unsuccessful, the subsequent intervention is vestibular rehabilitation, which enhances the cooperation of the damaged inner ear with the visual, somatosensory, and central vestibular organs.

Peripheral presbyvestibulopathy can be classified as presbycanalopathy or presbytolithopathy. Chronic canal/otolithic disorders and superior/inferior vestibular neuropathy are characterized by persistent rotatory/floating sensations during head and body movement. Various vestibular rehabilitation interventions for these symptoms have been reported.[9-11] However, a recent review found no significant among-intervention differences in the treatment outcomes.[12] Figure 3 illustrates the MAHOROBA vestibular rehabilitation, which involves simple and rational incorporation of visual and somatosensory information according to the patient's condition.[11]

As mentioned earlier, acute imbalance increases the risk of falls among elderly patients. Aging is unlikely to cause acute damage to the semicircular canal; instead, it is likely to be caused by an inner ear disease unrelated to age. Contrastingly, acute otolithic disorders due to aging are common, including BPPV caused by the detachment of utricle otoconia and their migration into semicircular canals. The diagnosis and treatment guidelines for BPPV were established by the Bárány Society in 2015,[13] with the JSER subsequently publishing its guidelines in 2017.[14] The semicircular canal into which utricle debris have entered can be identified based on the type and direction of the observed nystagmus. These detached debris in the semicircular canal can be returned to the utricle through the otolith repositioning maneuver (Epley and Lempert). In case this intervention is unsuccessful, symptomatic treatment using intravenous drips and/or oral medications can be performed for several weeks until the utricle debris in the semicircular canal is naturally metabolized and completely removed.[15]

The recurrence and chronicity of BPPV symptoms are exacerbated with aging, especially among women aged above 60 years.[1,2] Most patients with intractable BPPV cannot receive adequate treatment given the lack of medications for preventing or removing floating debris. There are 2 simple treatment approaches for such refractory cases, namely (1) prevention of otolith exfoliation and (2) prevention of migration of the exfoliated otoliths into the semicircular canal. Otolith exfoliation can be prevented by correcting unbalanced eating habits that lead to arteriosclerosis and smoking habits that cause vasospasm; moreover, fluid, calcium, and vitamin D intake should be promoted to improve blood flow and function of otolith organs.[16] Additionally, there are rehabilitation guidelines for promoting the activation of otolith organs. Finally, there are sleep guidelines that recommend avoiding the stereotyped right or left head position and reducing the burden of gravity on the otolith organs during sleep. Regarding the prevention of migration of the exfoliated otoliths into the semicircular canal, maintaining a sufficiently high head position during sleep reduces the migration of detached otolithic debris to the semicircular canal, which promotes complete recovery.[17,18] A randomized controlled trial demonstrated that this sleep position can improve subjective dizziness symptoms (Figure 4); however, it is important to ensure this sleep position does not cause neck and/or lower back pain.

CONCLUSION

1. Vestibular frailty and presbyvestibulopathy, including BPPV, can cause dizziness among elderly patients (Figure 1).
2. Vestibular frailty and presbyvestibulopathy may contribute to the onset of the vicious circle of falling–bone fracture–prolonged bedridden status–senile dementia (Figure 2).
3. Treatment interventions for vestibular frailty and presbyvestibulopathy should be based on vestibular rehabilitation rather than vestibular implantation or regeneration (Figure 3).
4. In acute BPPV, the otolith repositioning maneuver can be used to return otolithic debris to the utricle. At the chronic remission stage, there are nutritional guidelines for improving bone density in otolith organs and rehabilitation guidelines for activating otolith organs to prevent exfoliation. Moreover, sleeping in the head-up position can prevent free-floating debris from entering the semicircular canal (Figure 4).
5. Throughout their old age, the psychiatric care/support is also indispensable to keep their initiative against vestibular frailty.

Peer-review: Externally peer-reviewed.

Acknowledgments: We wish to thank Dr. Masashi Choubi, a registered statistician (Certificate number: 622017), for helpful advice on statistical analyses. We also thank Editage (https://www.editage.com/) for editing a draft of the manuscript.

Author Contributions: Concept – T.K.; Design – T.K.; Supervision – H.H.; Resources – T.K.; Materials – T.S.; Data Collection and/or Processing – T.S.; Analysis and/or Interpretation – T.S.; Literature Search – T.S. Writing – T.K.; Critical Review – H.H.

Declaration of Interests: The authors have no conflict of interest to declare.

Funding: This study was supported in part by a JSPS KAKENHI grant (2023-2025), AMED (Grant number: 18dk0310092h000a), and a Health and Labour Sciences Research Grant for Research on Rare and Intractable Diseases (H29-Nanchito (Nan)-Ippan-031) from the Ministry of Health, Labour, and Welfare of Japan.

REFERENCES

1. Sakagami M, Kitahara T, Ito T, Nishimura T, Wada Y, Yamanaka T. Disease statistics and abnormal clinical exam ratios for patients visiting a

vertigo/dizziness center at Nara Medical University. *Equilib Res*. 2018;77(3):136-142. [CrossRef]
2. Wada Y, Yamanaka T, Kitahara T, Kurata J. Effect of head roll-tilt on the subjective visual vertical in healthy participants: towards better clinical measurement of gravity perception. *Laryngoscope Investig Otolaryngol*. 2020;5(5):941-949. [CrossRef]
3. Agrawal Y, Van de Berg R, Wuyts F, et al. Presbyvestibulopathy: diagnostic criteria consensus document of the classification committee of the Bárány Society. *J Vestib Res*. 2019;29(4):161-170. [CrossRef]
4. Chow MR, Ayiotis AI, Schoo DP, et al. Posture, gait, quality of life, and hearing with a vestibular implant. *N Engl J Med*. 2021;384(6):521-532. [CrossRef]
5. Sakagami M, Ouji Y, Kawai N, Yoshikawa M. Differentiation of embryonic stem cells into inner ear vestibular hair cells using vestibular cell derived-conditioned medium. *Biochem Biophys Rep*. 2019;19:e100649.
6. Rauch SD, Velazquez-Villaseñor L, Dimitri PS, Merchant SN. Decreasing hair cell counts in aging humans. *Ann N Y Acad Sci*. 2001;942:220-227. [CrossRef]
7. Walther LE, Westhofen M. Presbyvertigo-aging of otoconia and vestibular sensory cells. *J Vestib Res*. 2007;17(2-3):89-92. [CrossRef]
8. Livingston G, Huntley J, Sommerlad A, et al. Dementia prevention, intervention, and care: 2020 report of the Lancet Commission. *Lancet*. 2020;396(10248):413-446. [CrossRef]
9. Cawthorne T. Vestibular injuries. *Proc R Soc Med*. 1946;39(5):270-273. [CrossRef]
10. Fujino A, Tokumasu K, Yosio S, Naganuma H, Yoneda S, Nakamura K. Vestibular training for benign paroxysmal positional vertigo. Its efficacy in comparison with antivertigo drugs. *Arch Otolaryngol Head Neck Surg*. 1994;120(5):497-504. [CrossRef]
11. Shiozaki T, Ito T, Wada Y, Kitahara T. Effects of vestibular rehabilitation on physical activity and subjective dizziness in patients with chronic peripheral vestibular disorders: A six-month randomized trial. *Front Neurol*. 2021;12:e656157.
12. Hillier S, McDonnell M. Is vestibular rehabilitation effective in improving dizziness and function after unilateral peripheral vestibular hypofunction? An abridged version of a Cochrane Review. *Eur J Phys Rehabil Med*. 2016;52(4):541-556.
13. von Brevern M, Bertholon P, Brandt T, et al. Benign paroxysmal positional vertigo: diagnostic criteria. *J Vestib Res*. 2015;25(3-4):105-117. [CrossRef]
14. Imai T, Takeda N, Ikezono T, et al. Classification, diagnostic criteria and management of benign paroxysmal positional vertigo. *Auris Nasus Larynx*. 2017;44(1):1-6. [CrossRef]
15. Imai T, Ito M, Takeda N, et al. Natural course of the remission of vertigo in patients with benign paroxysmal positional vertigo. *Neurology*. 2005;64(5):920-921. [CrossRef]
16. Jeong SH, Kim JS, Kim HJ, et al. Prevention of benign paroxysmal positional vertigo with vitamin D supplementation: A randomized trial. *Neurology*. 2020;95(9):e1117-e1125. [CrossRef]
17. Horinaka A, Kitahara T, Shiozaki T, et al. Head-up sleep may cure patients with intractable benign paroxysmal positional vertigo: a six-month randomized trial. *Laryngoscope Investig Otolaryngol*. 2019;4(3):353-358. [CrossRef]
18. Kitahara T, Horinaka A, Shiozaki T, et al. Combination of head-up sleep and vertical recognition training may cure intractable motion-evoked dizziness with unknown origin. *Acta Otolaryngol*. 2020;140(6):467-472. [CrossRef]