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Desensitization of Motion Sickness by Device-assisted Canalith Repositioning Procedure

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ABSTRACT

Motion sickness (MS) is a common syndrome induced by motion stimulation. Individual susceptibility to MS is key to the development of MS. We propose a hypothesis that displaced otoconia into semicircular canals can increase individual susceptibility to MS and thus Canalith Repositioning Procedure (CRP) can decrease the susceptibility to MS. With a before-after controlled study, we investigated the efficacy of CRP for the desensitization of MS. Seventy-one healthy subjects (23 men and 48 women; mean age of 35.0 years), each having a history of MS and undergoing an initial Off-Vertical Axis Rotation (OVAR) test to induce MS for evaluating the susceptibility to MS, received CRPs one time per week and totally three times in consecutive three weeks. One week after final CRP, MS susceptibility was re-evaluated with OVAR. The severity of OVAR-induced MS was scored with visual analogue scale. Results showed after three CRPs, 64 (90.1%) of 71 subjects showed the reduced susceptibility to MS, with a significant difference in the mean scores of the OVAR-induced MS before and after the CRPs (6.86 ± 1.31 vs. 5.00±1.38, $p < 0.001$). It is indicated that the device-assisted CRP is a novel, effective, and promising desensitization therapy for MS.

Keywords: Canalith Repositioning Procedure; Motion sickness; Off-Vertical Axis Rotation

INTRODUCTION

Motion sickness (MS), also known as carsickness, seasickness, airsickness or space sickness, is a physiological or epidemiological response to motion stimulation, which is characterized by symptoms and signs including pallor, cold sweats, excessive salivation, headaches, dizziness, stomach awareness, nausea, up to recurrent vomiting. MS is a common and troublesome problem in travel by car, boat, aircraft, or other transport.[1] The development and the severity of MS depends on the intensity of motion stimulation and the individual susceptibility to motion

stimulation. There are apparent differences in individual susceptibility to MS. Approximately 70% of astronauts will suffer some degree of MS in their initial space flight.[2]

Many theories about the mechanisms of MS have been proposed over the years and the sensory conflict theory is most widely accepted to explain the development of MS.[2,3] Pharmacological and non-pharmacological countermeasures are used for the prevention and treatment of MS.[3] Drug therapy is a very effective way to control MS, but almost all anti-MS medications have potential side effects, which limit the use of anti-MS drugs.[2,3] Non-pharmacological options include all procedures that reduce conflicting sensory input or accelerate the process of multi-sensory adaptation. For example, most individuals susceptible to MS can be desensitized by adaptation training.[4] However currently available approaches for the prevention and treatment of MS are not yet very satisfactory. It is indicated that there is a prominent increase in MS susceptibility for patients with certain vestibular disorders.[2,5,6] For example, patients with Benign Paroxysmal Positional Vertigo (BPPV) have a higher prevalence of MS compared to healthy individuals, 48.0% vs. 13.4%.[6] It has also been shown that some of patients with BPPV have high susceptibility to MS, but they are no longer susceptible to MS after successfully resolution of BPPV by Canalith Repositioning Procedure (CRP).[7] Thus we propose a hypothesis that displaced otoconia into semicircular canal may contribute to the increase of susceptibility to MS, which also provides the rationale for the development of CRP as a new desensitization therapy for MS. We conducted a before-after control study by comparing MS susceptibility before and after CPR and the short-term efficacy of CRP for the desensitization of MS was evaluated.

METHODS

Subjects: Subjects were healthy volunteers recruited from the undergraduate students of a local college, the staff of our hospital and the residents of local community. The inclusion criteria were as follows: Chinese, both sexes, aged from 18 to 60 years old, having a previous history of MS, no history of otological or vestibular disorders, having normal vestibular and oculomotor function upon otoneurological examination, with no positional vertigo or mystagmus in the Dix-Hallpike test and the roll test for the examination of BPPV.

Motion sickness test: In this study, Off-Vertical Axis Rotation (OVAR) test as an established laboratory stimulus of MS was used to induce MS.[8] OVAR was performed using the Diagnosis and Therapy System for BPPV (the Byrons Medical Science & Technique Inc., Jinan, China). As showed in Figure 1, subject was seated and secured in a computer-controlled rotary chair. The chair tilted 30 degrees off vertical axis and rotated around subject's body longitudinal axis at a velocity of 90 degrees per second without any breaks. OVAR was stopped when subject felt he or she could go no further and the tilted chair was back to upright position. Immediately after OVAR, subject was instructed to evaluate the severity of OVAR-induced MS by scoring on a subjective visual analogue scale between 0 and 10, where 0 represented no symptom and 10 represented maximal symptom.[4] Each subject received OVAR test twice, the first one performed at a week before receiving the initial CRP and the second one at a week after the final CRP. The bearable rotation time of each subject in the first OVAR test was regarded as his or her standard rotation time used in the second OVAR test. None of subjects used any concomitant medication that could affect the central nerve system or the vestibular system within 3 days before OVAR test.

Figure 1: Schematic performance of off-vertical axis rotation test. Subject is seated in a computer-controlled rotary chair. The chair rotates in yaw at a speed of 90 degrees per second while tilted at 30-degree angle to induce motion sickness in the subject.

Interventions: After the first OVAR test, each subject received CRPs, one time per week and totally 3 times in consecutive 3 weeks. CRPs were performed with the same device as used in OVAR test. CRP consisted of four consecutive maneuvers with a 5-minute interval between the maneuvers. Firstly a backward 360-degree rotation maneuver, which was similar to that used for the treatment of posterior canal BPPV[9] or anterior canal BPPV[10], was applied for clearing the possibly displaced otoconia in both the right posterior canal and the left anterior canal (Figure 2), followed by another backward 360-degree rotation maneuver for clearing the possibly displaced otoconia in both the left posterior canal and the right anterior canal (Figure 2). After 5-minute interval, a 360-degree roll maneuver toward the right, which was similar to that used for the treatment of horizontal canal BPPV,[11] was applied for clearing the possibly displaced otoconia in the left horizontal canal (Figure 3), and then a 360-dgree roll maneuver toward the left was performed for clearing the possibly displaced otoconia in the right horizontal canal (Figure 3).

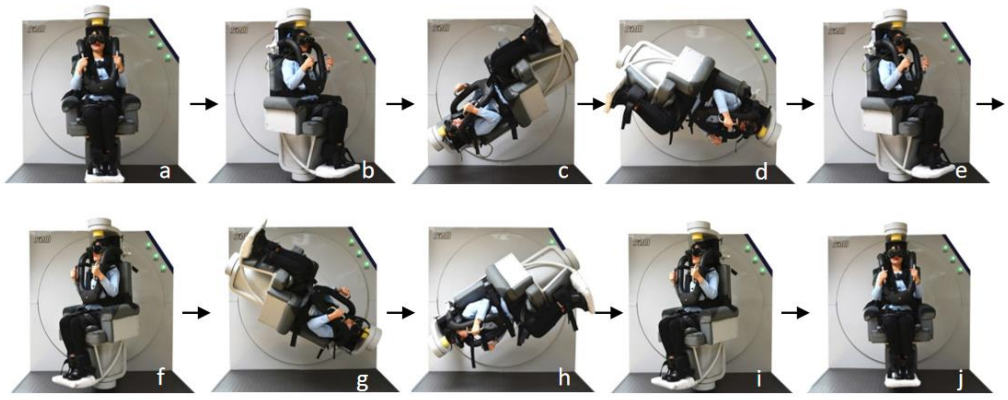

Figure 2: Schematic performance of backward 360-degree rotation maneuver. Subject is seated and secured in a computer-controlled rotary chair (a), and then is slowly turned 45 degrees toward the left (b), thus the right posterior

canal and the left anterior canal are orientated in the plane vertical to rotary axis, followed by backward 360-degree rotating around the anterior-posterior axis of chair, which includes three steps, each step for 120-degree rotation in a velocity of 90 degrees per second, and each position is hold for 30 seconds (c-e), by which the possibly displaced otoconia in the right posterior canal and the left anterior canal could be cleared. With a 5-minute interval, subject is slowly turned 90 degrees toward the right (f), thus the left posterior canal and the right anterior canal are orientated in the plane vertical to rotary axis, followed by another backward 360-degree rotation maneuver (g-i), by which the possibly displaced otoconia in the right posterior canal and the left anterior canal could be cleared, and then back to start position (j).

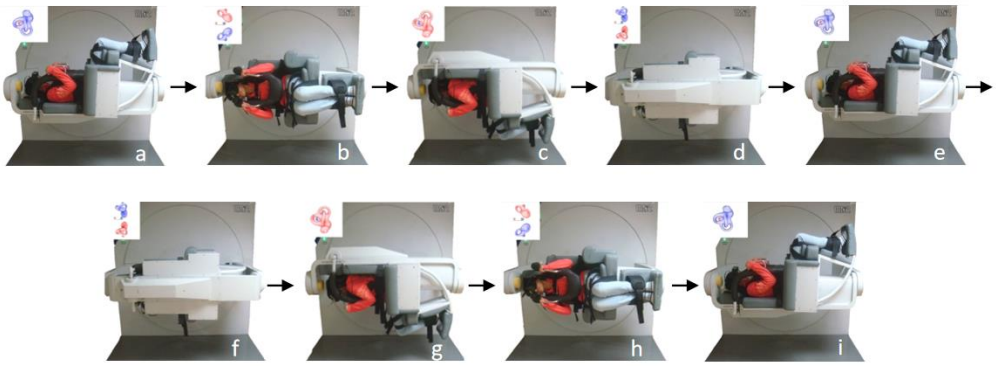

Figure 3: Schematic performance of 360-degree roll maneuver. Subject in computer-controlled rotary chair is slowly changed into supine position from sitting position, with the head bending forward 30 degrees to make horizontal canal be orientated in the plane vertical to rolling axis (a), and then is rolled 360 degrees toward the right in a series of stepwise 90-degree turns with an interval of 30 seconds between the steps (a-e), by which the possibly displaced otoconia in the left horizontal canal could be cleared. With a 5-minute interval, subject is rolled 360 degrees toward the left (e-i), by which the possibly displaced otoconia in the right horizontal canal could be cleared

Main Outcome Measures: To avoid the effect of habituation or adaptation, a one-week interval was given between the first OVAR test, individual CRPs and the second OVAR test. The MS susceptibility of each subject before initial CRP and after final CRP was respectively evaluated with OVAR test. MS scores on OVAR tests were used as main measures for MS susceptibility. Mean MS scores before initial CRP and after final CRP were compared to evaluate the short-term efficacy of CRP for the desensitization of MS.

Statistical Analysis: Quantitative data were expressed as mean and standard deviation (mean ± SD). The significance of difference in mean MS scores before initial CRP and after final CRP was determined by Student *t* test, considered significant as $p < 0.05$. Statistical analysis was performed using with SPSS version 20.

Ethics: This study was conducted in accordance with the the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Informed consent was obtained from all individual subjects included in the study. The Ethics Committee of the Beijing Electric Power Hospital approved the study protocol (JS-2021-a12-01).

RESULTS

A total of 71 healthy subjects were included in the study. There were 23 males and 48 females. Age ranged from 19 to 59 years, with a mean age of 35.0 ± 10.7 years and a median age of 33 years. Rotation time ranged from 24 seconds to 180 seconds, with an average of 69.1 ± 23.7 seconds.

Mean MS score after final CRP were significantly lower than that before initial CRP (5.00±1.38 *vs.* 6.86±1.31, $p < 0.001$), with a mean difference of 1.86 scores. Based on the changes in the mean MS scores before initial CRP and after final CRP, the MS susceptibility after CRP decreased by 27.1% in the subjects.

As showed in Table 1, after final CRP, aside from 7 (9.9%) subjects with no decrease in MS scores, 64 (90.1%) of 71 subjects showed the decrease of MS scores by 1-5 scores; subjects with reduced MS susceptibility by 2 scores accounted for 38.0% of subjects, followed by ones (29.6%) with reduced MS susceptibility by one score.

In addition, CRPs were tolerated well in all subjects, and no obvious adverse effects from the use of CRPs were found during or after CRPs.

Table 1: Frequency distribution of subjects with individual score differences of MS before and after CRPs.

MS score differences before and after CRPs	Number of subjects	%
0	7	9.9
1	21	30
2	27	38
3	8	11
4	7	9.9
5	1	1.4

MS - motion sickness; CPRs - canalith repositioning procedures.

DISCUSSION

The present study denotes that CRP, as a novel desensitization therapy for MS, can effectively reduce individual susceptibility to MS. It was showed that 64 (90.1%) of 71 subjects presented with the decreased MS susceptibility after CRPs.

MS has been recognized for long time.[12] Many theories about the mechanisms of MS have been proposed over the years, each explaining some but not all of the characteristics of MS. The sensory conflict theory is widely accepted as an underlying mechanism of MS.[2] Sensory conflicts can exist either between actual versus expected invariant patterns of vestibular, visual and proprioceptive inputs or between canal and otolith inputs, namely intravestibular or intralabyrinthine conflict.[2,3,13] It is considered that an imbalance in the outputs of the two major organs of the labyrinth, favoring the semicircular canals over the otolith organs, is responsible for most instances of MS.[13] And also sensory conflict can exist between vestibular inputs on two sides. Here we propose a new hypothesis, namely displaced otoconia hypothesis, to explain the occurrence of high MS susceptibility and the underlying mechanism of MS, which also provides the rationale for the development of CRP as a new desensitization therapy for MS.

It has been shown that MS susceptibility increases in patients with vestibular disorders such as vestibular migraine, Menière's disease and BPPV.[2,5,6] And also several studies showed that patients with BPPV had high susceptibility to MS.[7,14] For example, 66.7% of patients with BPPV were susceptible to MS,[14] and patients with BPPV showed the higher prevalence of MS than healthy individuals, 48.0% vs. 13.4%.[6] And also it was showed that the patients with BPPV who had a history of MS were no longer susceptible to MS after successfully resolution of BPPV by CRP.[7]

BPPV is a common vestibular disorder characterized by repeated episodes of positional vertigo produced by changes in head position.[15] Although the etiology and pathophysiology of BPPV have been not fully understood, it is believed that BPPV occurs when otoconia detach from the macula of the utricle and enter the semicircular canal, and the displaced otoconia, free-moving in the semicircular canal (canalolithiasis) or adhering to the cupula of the semicircular canal (capulolithiasis), may provoke vertigo symptom accompanied by nystagmus with head position changes.[12] Based on these theories, CRP designed for removal of the displaced otoconia out from the affected semicircular canal and back into the utricle have been used for the treatment of BPPV, by which BPPV can be relieved successfully.[15,16]

However, not all conditions with existence of displaced otoconia in the semicircular canal, regardless of canalolithiasis or capulolithiasis, certainly cause BPPV with typical symptoms and nystagmus, which depend on the density, volume and number of the displaced otoconia in the canal.[17] For example, there is a subtype of BPPV, also known as subjective BPPV[18] or BPPV without nystagmus,[19] which presents with a history of BPPV with a positioning test positive for vertigo but negative for nystagmus. Subjective BPPV is common, accounting for approximately one-fourth of patients with BPPV.[16,18] However the underlying mechanism of subjective BPPV is not very clear. An explanation is that the presence of a small quantity of loose otoconia in semicircular canal can induce positional vertigo but not be sufficient to provoking nystagmus.[18]

CRP is an effective treatment for BPPV. However residual dizziness is a common condition after successfully resolution of BPPV by CRP, with a prevalence of 31%-61%.[20,21] Causal factors for the residual dizziness are still under debate and a possible cause is the presence of small amounts of residual otoconia in the semicircular canal after successful CRP. These residual otoconia, either free-moving in the semicircular canals or adhering on the cupulae of canals, are very small in quantity so that they are not sufficient to produce typical symptoms and signs aside from mild dizziness.[20]

Furthermore, as showed in previous histological studies, small amounts of displaced otoconia may also be found in the semicircular canal in patients including adults and children who have not historical evidence of BPPV, and the displaced otoconia may occur in each of the semicircular canals, with incidences of 12.7%-34.9%.[22-24] However the presence of the otoconia in the semicircular canals is usually asymptomatic, probably owing to a small quantity not surpassing a critical mass.[17] We think the presence of otoconia in the canals may increase the individual susceptibility to MS, although it is not sufficient to cause BPPV.

Therefore we propose a new "displaced otoconia hypothesis" to explain the increase of individual susceptibility to

MS. Due to some conditions such as head trauma, the otoconia may detach from the utricle macula and enter the semicircular canal, free-moving in the canal (canalolithiasis) or adhering to the cupula of the canal (capulolithiasis). When the displaced otoconia are more enough, they may cause a typical BPPV,[16,18] while the presence of small amounts of otoconia in the canals may be asymptomatic,[22-24] produce subjective BPPV,[16,18,19] or cause residual dizziness occurring after CRP in some patients with BPPV.[20,21] Under these conditions, no matter the presence of otoconia in the canals is symptomatic or asymptomatic, it may increase individual susceptibility to MS due to the occurrence of intravestibular of intervesitular sensory conflicts when exposure to motion stimulation.

Preventive and therapeutic countermeasures for MS include pharmacological and non-pharmacological approaches.[2] However use of anti-MS drugs is limited due to the side effects of medicals. Adaptation is one of effective approaches to prevent MS and most individuals susceptible to MS can be desensitized by adaptation training, with a successful rate of about 85%.[4,25] However, the adaptation training commonly requires for repeated exposures to the motion stimulation, and thus it can be extremely time consuming, lasting many weeks.[2,4] Even successful adaptation to MS is obtained, the retention of adaptation may not last for a long time and trainees can lose their MS adaptation after a gap.[26]

CRP is different from adaptation training in the mechanisms of desensitization for MS and it was developed based on the “displaced otoconia hypothesis” we proposed in the present study. According to this hypothesis, MS susceptibility increases in the individuals with the presence of displaced otoconia in the canals, which produces the intravestibular or intervestibular sensory conflicts in motion environment. CRP may decrease the individual susceptibility to MS by clearing the displaced otoconia from the canals to reduce conflicting sensory inputs.

In some of patients with BPPV, repeated CRPs may be required for the resolution of BPPV.[15,27] A systematic review shows that 32%-90% of patients may obtain successfully resolution of BPPV after the first CRP, with cumulative success rates of 67%-98% after the third CRP.[27] A recent study shows that all patients may obtain successfully resolution of BPPV after three CRPs with one-week interval between CRPs.[28] In present study we designed the CRP protocol with one time per week and totally 3 times in 3 consecutive weeks. It demonstrates that this CRP protocol can effectively decrease the severity of MS in most individual susceptible to MS.

There are several limitations for this study. First, although CRP offered the desensitization of MS in about 90% of subjects, about 10% of individuals showed no reduction in MS susceptibility after three CRPs. Cause for this condition is not clear. It may relate with the residual of otoconia in the canals after CRPs. Similarly not yet all patients with BPPV may be successfully treated with CRP. Treatment failure may occur in up to 33% of patients.[15,27] In addition, the degree of reduced MS susceptibility by CRP was not enough in most subjects and 67.7% of subjects only obtained the reduction of MS susceptibility by one or 2 scores or lesson after three CRPs. It indicates there are other factors contributing to elevated MS susceptibility in these individuals, while the presence of displaced otoconia in the canals is only one of possible causes accounting for MS susceptibility. In order to improve the efficacy of CRP for the desensitization of MS, whether alternative CRP protocols are required? More studies are needed. Second, only short-term efficacy of CRP for the desensitization of MS was evaluated in the current study. Further evaluating for the long-term efficacy of CRP for the desensitization of MS is required. BPPV is a high

recurrent disease.[15] Because there are possibly shared common mechanisms (canalolithiasis or capulolithiasis) between BPPV and elevated MS susceptibility, the individuals with reduced MS susceptibility may again become susceptible to MS due to the re-occurrence of displaced otoconia in the canals. A recent study showed that after successful treatment of BPPV with CRP, regular home CRP decreased the recurrence of BPPV.[28] Perhaps the individuals after desensitization of MS by CRP could be regularly applied with CRP to protect them from re-susceptibility to MS. Third, in the present study, MS susceptibility and the efficacy of CRP for the desensitization of MS were evaluated only based on the OVAR tests. Subsequent observation is required to confirm if the benefits of CRP may be kept in real motion environment.

In conclusion, CRP is a novel and effective desensitization therapy for MS, with a promising efficacy. However, the evaluation of long-term efficacy of the CRP for the desensitization of MS needs further research; whether the severity of MS in the CRP-treated individuals may be reduced when exposure to real motion stimulation still needs to examine; and also further insight into the underlying mechanisms for the desensitization of MS by CRP is required.

AUTHOR CONTRIBUTIONS

All authors contributed to the study conception and design. Material preparation, data collection and analysis were performed by X.S., X.P., L.Z. and E.W. The first draft of the manuscript was written by E.W. and all authors commented on previous versions of the manuscript. All authors reviewed and approved the final manuscript.

REFERENCES

1. Murdin L, Golding J, Bronstein A. Managing motion sickness. BMJ. 2011;343:d7430.
2. Golding JF. Motion sickness. Handb Clin Neurol. 2016;137:371-90.
3. Schmäl, F. Neuronal mechanisms and the treatment of motion sickness. Pharmacology. 2013;91(3-4):229-41.
4. Newman MC, McCarthy GW, Glaser ST, Bonato F, Bubka A. Motion sickness adaptation to Coriolis-inducing head movements in a sustained G flight simulator. Aviat Space Environ Med. 201;84(2):104-9.
5. Murdin L, Chamberlain F, Cheema S, Arshad Q, Gresty MA, Golding JF, Bronstein A. Motion sickness in migraine and vestibular disorders. J Neurol Neurosurg Psychiatry. 2015;86(5):585-7.
6. Strupp M, Brandt T, Huppert D, Grill E. Prevalence of motion sickness in various vestibular disorders: a study on 749 patients. J Neurol. 2018;265(Suppl 1):95-97.
7. Rahko T. The test and treatment methods of benign paroxysmal positional vertigo and an addition to the management of vertigo due to the superior vestibular canal (BPPV-SC). Clin Otolaryngol Allied Sci. 2002;27(5):392-5.
8. Cian C, Ohlmann T, Ceyte H, Gresty MA, Golding JF. Off vertical axis rotation motion sickness and field dependence. Aviat Space Environ Med. 2011;82(10):959-63.
9. Lempert T, Wolsley C, Davies R, Gresty MA, Bronstein AM. Curing benign positional vertigo in a 3D

flight simulator. Lancet. 1996;347(9009):1192.

10. Iglebekk W, Tjell C, Borenstein P. Pain and other symptoms in patients with chronic benign paroxysmal positional vertigo (BPPV). Scand J Pain. 2013;4(4):233-240.
11. Shan X, Peng X, Wang E. Efficacy of computer-controlled and modified roll maneuver for treatment of geotropic lateral canal benign paroxysmal positional vertigo. Otol Neurotol. 2015;36(8):1412-6.
12. Huppert D, Benson J, Brandt T. A historical view of motion sickness - a plague at sea and on land, also with military impact. Front Neurol. 2017;8:114.
13. Previc FH. Intravestibular balance and motion sickness. Aerosp Med Hum Perform. 2018;89(2):130-140.
14. Uneri A. Migraine and benign paroxysmal positional vertigo: an outcome study of 476 patients. Ear Nose Throat J. 2004;83(12):814-5.
15. Bhattacharyya N, Gubbels SP, Schwartz SR, Edlow JA, El-Kashlan H, Fife T, et al. Clinical practice guideline: Benign paroxysmal positional vertigo (update). Otolaryngol Head Neck Surg. 2017;156(3_suppl):S1-S47.
16. Kim JS, Zee DS. Benign paroxysmal positional vertigo. N Engl J Med. 2014;370:1138-1147.
17. Korres S, Balatsouras DG, Kaberos A, Economou C, Kandiloros D, Ferekidis E. Occurrence of semicircular canal involvement in benign paroxysmal positional vertigo. Otol Neurotol. 2002;23(6):926-32.
18. Balatsouras DG, Korres SG. Subjective benign paroxysmal positional vertigo. Otolaryngol Head Neck Surg. 2012;146(1):98-103.
19. Hornibrook, J. Benign paroxysmal positional vertigo without nystgamus. Otolaryngol Head Neck Surg. 2012;146(6):1041;1041-2.
20. Giommetti G, Lapenna R, Panichi R, Mobaraki PD, Longari F, Ricci G, Faralli M. Residual Dizziness after Successful Repositioning Maneuver for Idiopathic Benign Paroxysmal Positional Vertigo: A Review. Audiol Res. 2017;7(1):178.
21. Martellucci S, Pagliuca G, de Vincentiis M, Greco A, De Virgilio A, Nobili Benedetti FM, et al. Features of Residual Dizziness after Canalith Repositioning Procedures for Benign Paroxysmal Positional Vertigo. Otolaryngol Head Neck Surg. 2016;154(4):693-701.
22. Bachor E, Wright CG. Karmody CS. The incidence and distribution of cupular deposits in the pediatric vestibular labyrinth. Laryngoscope. 2002;112(1):147-51.
23. Moriarty B, Rutka J, Hawke M. The incidence and distribution of cupular deposits in the labyrinth. Laryngoscope. 1992;102(1):56-9.
24. Naganuma H, Kohut RI, Ryu JH, Tokumasu K, Okamoto M, Fujino A, et al. Basophilic deposits on the cupula: preliminary findings describing the problems involved in studies regarding the incidence of basophilic deposits on the cupula. Acta Otolaryngol Suppl. 1996;524:9-15.
25. Lucertini M, Verde P, Trivelloni P. Rehabilitation from airsickness in military pilots: long-term treatment effectiveness. Aviat Space Environ Med. 2013;84(11):1196-200.
26. Lucertini M, Lugli V, Casagrande M, Trivelloni P. Effects of airsickness in male and female student

pilots: adaptation rates and 4-year outcome. Aviat Space Environ Med. 2008;79(7):677-84.

27. Reninik H, Wegner I, Stegeman I, Grolman W. Rapid systematic review of repeated application of the Epley maneuver for treating posterior BPPV. Otolaryngol Head Neck Surg. 2014;151(3):399-406.
28. Ismail EI, Morgan AE, Abdeltawwab MM. Home particle repositioning maneuver to prevent the recurrence of posterior canal BPPV. Auris Nasus Larynx. 2018;45(5):980-984.