

7. Dezsényi B, Sárközi L, Kaiser L, Tárkányi K, Nikolova R, Belics Z. Gynecological and obstetrical aspects of Enterobius vermicularis infection. Acta Microbiol Immunol Hung. 2018; 19: 1–7.
8. Palmas C, Gabriele F, Conchedda M, Bortoletti G, Ecce AR. Causality or coincidence: may the slow disappearance of helminths be responsible for the imbalances in immune control mechanisms? J Helminthol. 2017;77(2):147–53. PubMed PMID: 12756068
9. Patsantara GG, Piperaki ET, Tzoumaka-Bakoula C, Kanariou MG. Immune responses in children infected with the pinworm Enterobius vermicularis in central Greece. J Helminthol. 2016; 90(3): 337–41.
10. Rainova I, Harizanov R, Kaftandjiev I, Tsvetkova N, Mikov O, Kaneva E. Human Parasitic Diseases in Bulgaria in Between 2013–2014. Balkan Med J. 2018; 35(1): 61–67.
11. Sica A, Erreni M, Allavena P, Porta C. Macrophage polarization in pathology. Cell Mol Life Sci. 2015; 72(21):4111–26. doi: 10.1007/s00018-015-1995-y.
12. Sklyarova VO. Epidemiological features of parasitary invasis in women of reproductive age with disorders of reproductive health. Wiad Lek. 2018; 71(3 pt 2): 674–77.
13. Turgeon ML. Immunology & serology in laboratory medicine 6<sup>th</sup> ed., Elsevier Inc, 2017: 592.

Стаття надійшла 28.10.2019 р.

DOI 10.26724/2079-8334-2020-4-74-97-102

UDC 616.281- 008.55-07

**N.S. Mishchanchuk, O.M. Borysenko, S.B. Bezshapochnyi<sup>1</sup>, S.V. Kuzmenko**  
**SI “O.S. Kolomiychenko Institute of Otolaryngology, NAMS of Ukraine”, Kyiv**  
<sup>1</sup>**Ukrainian Medical Stomatological Academy, Poltava**

### FEATURES OF VESTIBULAR DYSFUNCTION IN PATIENTS WITH HEREDITARY AND ACQUIRED MOTION SICKNESS

e-mail: nsmisch@i.ua, nsmishch@gmail.com

It was established that vestibular dysfunction in patients with hereditary and acquired motion sickness plays one of the leading roles in traffic I-loads. Features of vestibular dysfunction in hereditary motion sickness are characteristic for bilateral symmetrical irritative mixed vestibular syndrome of the Ist and the IInd degree in the stage of subcompensation with hearing acuity within the norm symmetrically on both sides. In acquired motion sickness, the features of vestibular dysfunction are manifested by following variability: unilateral irritative peripheral or mixed vestibular syndrome of the IInd degree in the stage of subcompensation or decompensation, as well as asymmetric irritative bilateral mixed syndrome of the IInd degree in the stage of subcompensation with different forms and degrees of hearing loss. It was investigated that unilaterality and asymmetry of irritative vestibular syndromes at the acquired motion sickness is one of signs of basic disease development. Certain differences in vestibular dysfunction increase the efficiency of diagnosis, as well as differential diagnosis of hereditary and acquired motion sickness.

**Key words:** hereditary and acquired motion sickness, experimental nystagmus reaction, peripheral and mixed vestibular syndromes.

### Н.С. Міщанчук, О.М. Борисенко, С.Б. Безшапочний, С.В. Кузьменко

### ОСОБЛИВОСТІ ВЕСТИБУЛЯРНОЇ ДИСФУНКЦІЇ У ПАЦІЄНТІВ ПРИ СПАДКОВИХ ТА НАБУТИХ ХВОРОБАХ РУХУ

Досліджені особливості вестибулярної дисфункції при спадкових хворобах руху, які проявляються двобічним симетричним іритативним змішаним вестибулярним синдромом I та II ступеня в стадії субкомпенсації з гостротою слуху у межах норми симетрично з обох боків. При набутих хворобах її особливості проявляється варіабільністю: однобічним іритативним периферичним або змішаним вестибулярним синдромом II ступеня у стадії субкомпенсації чи декомпенсації, а також асиметричним іритативним двобічним змішаним синдромом II ст. у стадії субкомпенсації з різними формами та ступенями зниження слуху. Визначено, що однобічність та асиметричність іритативних вестибулярних синдромів при набутих хворобах руху є однією із ознак розвитку основного захворювання. Визначені відмінності вестибулярної дисфункції підвищують ефективність діагностики, а також диференційної діагностики спадкових та набутих хвороб руху.

**Ключові слова:** спадкові, набуті хвороби руху, експериментальна ністагмова реакція, периферичний та змішаний вестибулярні синдроми.

*The work is a fragment of the research project “To substantiate the feasibility of observation tactics in patients with stage I-III vestibular schwannoma, depending on the state of auditory and vestibular functions and the immune response of the body”, state registration No. 0119U100495.*

Motion sickness occurs with increasing the angular and linear acceleration or deceleration movements during various transport loads, including high-speed and long-term ones. Symptoms of motion sickness have been known since the ancient times, and there are hereditary (congenital) and acquired motion sickness among them.

The physiological mechanisms underlying the onset of motion sickness have been studied for centuries by numerous researchers, but they have not yet been definitively identified and are often contradictory [2, 4 - 8, 12].

A phenotypic relationship of motion sickness with comorbidities that have common genetic factors, in particular, with migraine and individual diseases of the digestive system were studied by some authors [8, 9].

Hypotheses of the neural and vestibular discrepancies, evolutionary and genetic hypotheses, hypotheses of the sensory conflicts and an activation of the sensory systems are among the known hypotheses of their occurrence mechanisms [5, 8].

The main cause of motion sickness at any age is a sensory conflict during different transport loads according to the proponents of the most common hypothesis of sensory conflicts [9, 10]. According to this hypothesis, under the action of acceleration and deceleration movements there is an acute vestibular dysfunction with inconsistency of the afferent sensory systems (visual, vestibular, proprioceptive) with the participation of hormonal, neurohumoral, biochemical and other factors which have not yet been fully studied.

Most researchers tend to think that motion sickness is the result of vestibular hyperstimulation. However, some authors argue that the parameters of vestibular function on both sides are without the deviations from a norm in hereditary motion sickness. It is known that bilateral vestibular areflexia with postural disorders does not cause motion sickness under any traffic loads. But such people do not achieve the high results in motor sports. They have difficulty in spatial orientation, in performing particularly precise movements, even with persistent training [10].

Sensory conflict does not create certain conditions for stimulation, but on the contrary, promotes the disadaptations. A number of authors considers that the vestibular system has a leading role in the occurrence of motion sickness on the basis of researches [2].

Some researchers believe that motion sickness can be caused by the visual stimulation, without including vestibular stimuli. No differences were showed in the occurrence of motion sickness in examined patients with significantly reduced visual acuity [5, 9].

Sensory conflicts can occur between signals from the otoliths and the semicircular canals of the ear labyrinth with ascending impulses from the muscles connected to the mechanoreceptors, as well as to the eye movements. This is achieved by the active entry of different multilevel systems into the brain, including psycho-neurological ones [9].

Currently, an increase in motion sickness to 30.0% of cases, i.e. in every 3-4 people of working age, is recorded in adults of civilized countries of the world. The increasing prevalence of these diseases dictates the urgent need to improve the diagnosis and differential diagnosis of hereditary and acquired motion sickness, the development of optimal therapeutic and prophylactic, rehabilitation and corrective measures of motion sickness to provide the management of complex modern technologies by people who must be resistant to stimuli acting in unusual current conditions for them.

Because motion sickness, both hereditary and acquired ones, can be the cause of various catastrophes and tragic situations, therefore, the study of the VD features in hereditary and acquired motion sickness is one of the urgent and important tasks.

**The purpose** of the work was to study the features of vestibular dysfunction to increase the efficiency of diagnosis and differential diagnosis of hereditary and acquired motion sickness.

**Materials and methods.** There were examined and analyzed 109 patients (39 men and 70 women) of socially active age from 18 to 40 years. They asked for medical help for hereditary or acquired motion sickness when using the land vehicles.

The patients were divided into 2 main groups. The group 1 included 80 patients with hereditary motion sickness, the group 2 consisted of 29 patients with acquired motion sickness. The control group consisted of 30 healthy individuals (19 kinesio-resistant men and 11 kinesio-resistant women of the appropriate age). No motion sickness was noted in a history of these patients and their relatives.

The complaints in the dynamics, anamnestic, ENT examination were studied. Audiometry, vestibulometry, the necessary additional examinations were performed on the 3th-4th days after stopping an attack of motion sickness.

Audiometry, determining the air and bone conduction thresholds of sound perception in the frequency range of 0,250-8,0 kHz, was performed by the audiometers MA-31 (Germany) and "Uteri" (Denmark).

Vestibulometry was based on the V.G. Bazarov's scheme [1] with registration of postural balance (static equilibrium – by cephalography and kinetic equilibrium by the Fukuda stepping test to determining the rotation of the body around its own axis).

Spontaneous, positional nystagmus and experimental nystagmus responses caused by classical functional stimulations (caloric and rotational) were recorded by electronystagmography (ENG) method on the computer neurocomplex of SPE "DX-systems" (Ukraine).

The main quantitative and qualitative indicators of the experimental nystagmus reactions were determined, taking into account the duration of the pathological vestibulo-sensory reaction, the degree of manifestation of vestibulo-vegetative reflexes according to K.L. Hilov [2]. The caloric stimulation was performed only with the entire eardrum (tempatic membrane).

The nature of vestibular dysfunction was determined by I.B. Soldatov et al. [3], and the degree of its manifestation – by V.G. Bazarov [1].

If necessary, the additional tests were prescribed as following: bacteriological, virological, immunological, impedancemetry, registration of short-latency auditory evoked potentials (SAEPs), computed tomography (CT) of the temporal bones, magnetic resonance imaging (MRI) of the brain, electrocardiography (ECG), electroencephalography (EEG).

**Results of the study and their discussion.** The visible pathology was not revealed at ENT examination in 80 patients of the group 1. It was noted that 62 patients of this group (78.0%) developed motion sickness in the family through the maternal line when using vehicles. Motion sickness was observed in the remaining 18 patients (22.0%) relatives through the paternal line.

No hearing impairments were noted in patients of this group before, during and after occurrence of motion sickness. According to tonal and speech audiometry, their hearing was within the norm, symmetrically on both sides.

The balance disturbances were fixed, spontaneous and positional nystagmus was not revealed at background ENG in all 80 patients of the group 1 by vestibulometry. Bilateral symmetrical hyperreflexia with dysrhythmia of experimental nystagmus reaction was registered by ENG. Extended symmetrical bilateral duration of the vestibulo-sensory reaction and manifestation of degree 1 vestibulo-vegetative reflexes according to K.L. Hilov were noted in 27 patients of this group, and degree 2 – in the remaining 53 female patients. These data together indicate the presence of degree I and II irritative bilateral symmetrical mixed vestibular syndrome in the stage of subcompensation in persons of the group 1 with hereditary motion sickness.

It should be emphasized that the indicators of postural balance were within normal limits in persons of the control group. Neither spontaneous nor positional nystagmus was recorded with background ENG. The experimental nystagmus reaction was rhythmically symmetrical on both sides, without pathological vestibulo-vegetative reflexes and prolonged dizziness – as a manifestation of pathological vestibular-sensory reaction in functional simulations by ENG.

The presence of irritative bilateral mixed vestibular syndrome in hereditary diseases in patients of group I is apparently due to genetic hypersensitivity to transport loads and the evolutionary inability of the human body to adapt to unprecedented high-speed and long-term transport loads due to technical progress of the last century.

It should be noted that 38 women of the group 1 with hereditary motion sickness had frequent migraine attacks (with an acute sense of smell, photophobia and discomfort on sound signals and speech). The vasomotor symptoms as vasomotor rhinitis or gastric disorders with nausea and significant salivation were fixed in a history of the remaining 15 women of this group which was consistent with the following authors [5, 12].

Progressive VD, which was the first sign of body discomfort, when going by land transport, developed in 16 people out of 29 patients of group 2 with acquired motion sickness, prompting the medical attention. No changes in the eardrum were noted in all 16 patients using otoscopy.

No spontaneous and positional nystagmus was detected in 10 out of these 16 patients using vestibulometry. Balance dysfunction was recorded, and unilateral hyperreflexia of the dysrhythmic experimental nystagmus reaction at functional loads was determined with ENG, which indicated unilateral irritative mixed vestibular syndrome of the 2nd degree in the stage of subcompensation.

They fixed the appearance of periodic and then constant noise in one of the ears, and the difference in hearing acuity when using a mobile phone, which was not given due importance.

Using audiometry, unilateral sensorineural hearing loss (USNHL) of the 1st degree was revealed in two out of 10 patients, and USNHL of the 2<sup>nd</sup> degree – in 8 patients. Stage I vestibular schwannoma was observed by contrast enhanced MRI in 6 out of 10 patients and stage II vestibular schwannoma – in 4 patients. It is necessary to emphasize the similarity of VD complaints at transport travels in 6 more patients of the group 2. Meningioma and cholesterol granuloma were obtained by a contrast MRI in 4 and 2 patients of them, respectively. Unilateral sensorineural hearing loss of the IIIrd degree was registered in 2 out of 4 patients with a meningioma, and USNHL of the IVth degree with impaired Speech Intelligibility Test – in 2 more patients. Single-sided complete hearing loss was recorded in 2 patients with cholesterol granuloma. The

irritative unilateral peripheral syndrome of the IIInd degree was determined in the subcompensation stage in these 6 patients.

The first VD manifestations in acquired motion sickness during transport loads occurred in parallel with discomfort feeling in one of the ears (pain and suppuration or congestion in the ear, noise and hearing loss) in the remaining 13 patients of the group 2.

Unilateral exacerbation of chronic suppurative otitis media (CSOM) with a perforated tympanic membrane in 5 out of these 13 patients was revealed by otoscopy. Unilateral chronic suppurative otitis with cholesteatoma and destruction of the lateral wall of the ear labyrinth was recorded by MRI and CT in 3 out of those 5 patients. Mixed single sided deafness was detected in them by audiometry.

Spontaneous nystagmus, balance disturbances, unilateral hyperreflexia with dysrhythmia of experimental nystagmus reaction from ear pathology were revealed with vestibulometry in these 3 patients. Unilaterally prolonged pathological vestibulo-sensory reactions and manifestations of vestibulo-vegetative reflexes of the IIInd degree were recorded in them. Together they indicate unilateral vestibular mixed syndrome of the IIInd degree in the stage of decompensation.

An imbalances, unilateral hyperreflexia of rhythmic experimental nystagmus reaction with pathological vestibulo-vegetative reflexes, which are within the unilateral irritative peripheral vestibular syndrome in the 2nd stage of subcompensation with preservation of the vectorial laws were registered in 2 patients with unilateral exacerbation of chronic suppurative otitis media from the abovementioned 5 patients.

Using otoscopy the intact eardrum was revealed in the remaining 7 out of 13 patients of the group 2. Unilateral conductive (sound-conductive) hearing loss was defined by audiometry in these 7 patients. Unilateral otosclerosis was established in one girl aged 19 years old out of these 7 patients, unilateral secretory otitis media with type B tympanogram – in 6 patients.

Unilateral irritative vestibular peripheral syndrome of the IIInd degree in the stage of subcompensation was fixed by vestibulometry in all 7 patients. VD complaints were noted in 1 young person aged 18 years old out of these 13 patients taking the bus daily.

The intact tympanic membrane was revealed on both sides using the otoscopic examination. According to SAEPs data bilateral almost symmetrical SNHL of the IIInd degree with changes in the brainstem structures of the brain was registered by audiometry.

Asymmetric bilateral hyperreflexia of experimental nystagmus reaction with signs of its reversion and vestibulo-vegetative reflexes of the IIInd degree, and prolonged vestibulo-sensory reactions, indicating an asymmetric bilateral mixed vestibular syndrome of the IIInd degree in the stage of subcompensation was fixed by vestibulometry. MRI of the brain revealed a lowering of the cerebellum below the large occipital foramen (Arnold -Chiari syndrome) when a birth trauma was in the anamnesis.

It should be emphasized that the parents of both young girls (one with otosclerosis and the other with Arnold-Chiari syndrome) considered them inattentive due to frequent questioning, because their ears did not hurt. When the girls began to develop a vestibular symptom complex of motion sickness during transport trips, they sought medical help. звернулись за медичною допомогою.

The measures of postural balance were analyzed in patients of 2 main groups and persons of the control group, which are given in the table.1

Table 1

**Mean postural balance measures in 2 main groups and control group**

Groups	Postural balance measures (balance functions)	
	Static balance	Kinetic balance
	P cephalography, in conventional units	The Fukuda stepping test (the rotation of the body around its own axis, in degrees)
Main 1	3.5±0.1*	40.5±0.2*
Main 2	4.6±0.2*,**	52.5±0.3*,**
Control	2.3±0.1	26.5±0.1

Note: \* - the difference between the measures of static and kinetic balances in patients of two groups is significant at  $p<0.05$  in comparison with the corresponding measures of the control group; \*\* - the difference in balance is significant between patients of groups 1 and 2 at  $p<0.05$ .

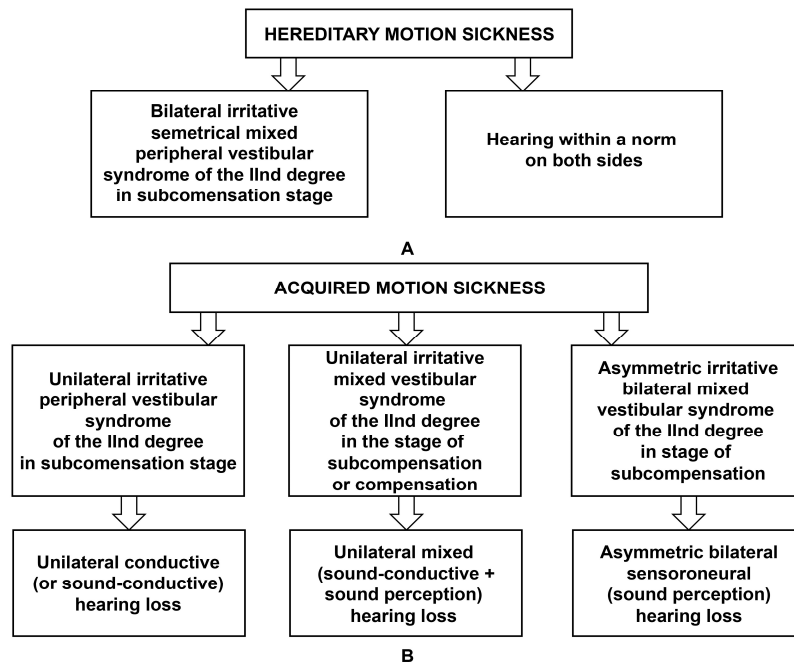


Fig. 1. The state of vestibular and auditory functions in patients with motion sickness (A – hereditary motion sickness, B – acquired motion sickness).

According to fig. 1, unilateral irritative peripheral and mixed vestibular syndromes, as well as asymmetric bilateral mixed syndrome of irritative nature with different hearing impairments is characteristic for motion sickness in patients of the group 2. This feature of the vestibular dysfunction is one of the signs of the basic disease, in particular, in our cases, the manifestation of tumors in the temporal

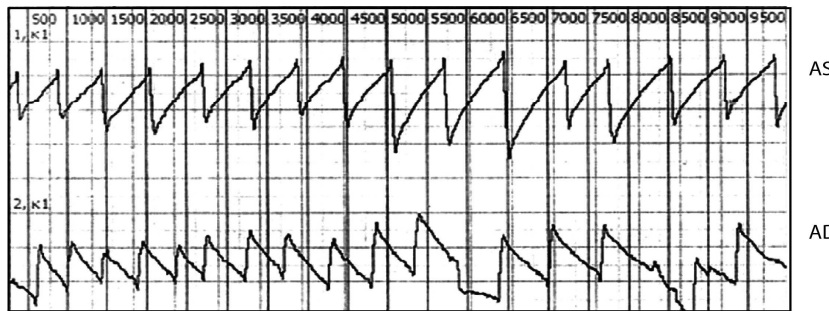


Fig. 2. ENG of patient P., 19 years old with unilateral irritative peripheral syndrome of the IInd degree, left – with otosclerosis.

The results of audiometry of patient P., 19 years old with conductive hearing loss on the left in unilateral otosclerosis are shown in Fig. 3.

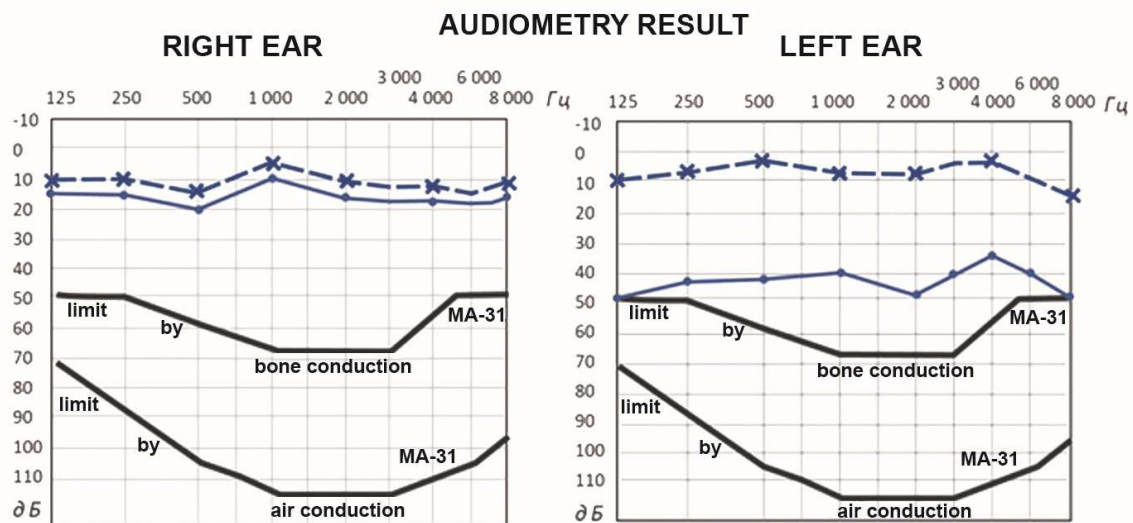


Fig. 3. Audiogram of patient P., 19 years old with conductive hearing loss in tympanic form of otosclerosis – in the left ear, and normal hearing – in the right ear.

Noteworthy is the fact that the measures (indices) of balance functions in the group 2 are almost 2 times higher than in the control group and significantly higher than in the group 1 (at  $p < 0.05$ ). This fact obviously indicates that the imbalance is supported by the basic diseases.

The state of vestibular and auditory functions in patients of two groups with hereditary and acquired motion sickness is shown in fig. 1.

Characteristic bilateral mixed vestibular syndrome of the Ist and IInd degrees with normal hearing acuity was observed in all persons with hereditary motion sickness, as can be seen from fig. 1.

bone, purulent and non-purulent ear pathology, complications after birth trauma.

ENG data demonstrated in fig. 2 and audiometry data of group 2 patient with acquired motion sickness (fig. 3), which manifested itself in otosclerosis are an illustration of obtained results.

These examinations indicate that the vestibular dysfunction plays one of the leading roles in patients with hereditary and acquired motion sickness. These results are consistent with data from other researchers [2, 5, 6, 11, 12].

### Conclusion

Features of the vestibular dysfunction in hereditary motion sickness are manifested by irritative bilateral symmetrical mixed vestibular syndrome of the I<sup>st</sup> and II<sup>nd</sup> degrees in the stage of subcompensation in normal hearing acuity, and in acquired motion sickness is manifested by variability such as irritative unilateral peripheral syndrome of the II<sup>nd</sup> degree in the stage of subcompensation, irritative unilateral mixed syndrome of the II<sup>nd</sup> degree in the stages of subcompensation and decompensation, asymmetric mixed bilateral syndrome of the II<sup>nd</sup> degree in the stage of subcompensation with hearing loss of different forms and degrees associated with the basic diseases.

One-sidedness of irritative peripheral and mixed syndromes, as well as asymmetry of bilateral mixed vestibular syndromes in acquired motion sickness is one of the signs of the basic disease development.

The effectiveness of diagnosis and differential diagnosis in hereditary and acquired motion sickness are increased by certain differences in the vestibular dysfunction. These differences should be used in the expert assessment of motion sickness to develop the effective treatment and preventive measures.

Further developments of pathogenetic mechanisms of motion sickness, effective rehabilitation and correction measures of the vestibular dysfunction to increase the kinesio resistance, rational professional selection, preservation of professional suitability, working capacity in the hectic pace of life today and in the future, which is of paramount not only medical but also social and economic importance, and in the future, which is of paramount not only medical but also social and economic importance, are promising.

### References

1. Bazarov VG. Klinicheskaya vestibulometriya. Kiev, Zdorovya. 1988. 200 s. [in Russian].
2. Gilov KL. Funktsiya organa ravnovesiya i bolezni peredvizheniya. Leningrad, Meditsina, Leningrad. otd-nie, 1969. 280 s [in Russian].
3. Soldatov IB, Sushcheva GP, Hrappo NS. Vestibulyarnaya disfunktsiya. Moskva, Meditsina. 1980. 228 p. [in Russian].
4. Brasses C, Cianetti F. Motion sickness. Part I: development of a model for predicting motion sickness incidence. International Journal of Human Factors Modelling and Simulation (IJHFMS). 2011; 2(3):163-87. doi: 10.1504/IJHFMS.2011.044492.
5. Brasses C, Cianetti F, Elia A. Motion sickness. Part II: experimental verification on the railways of a model for predicting motion sickness incidence. International Journal of Human Factors Modelling and Simulation (IJHFMS). 2016; 2(3):188-203. doi.org/10.1504/ijhfms.2011.044494.
6. Cholin AA, Cholina EI. Kinetosis – motion sickness syndrome: treatment and prevention. Medical Council. 2011;11-12: 45-50.
7. Dennison M, D'Zmura M. Effects of unexpected visual motion on postural sway and motion sickness. J Applied ergonomics, 2018 Sept; 71: 9-16. doi: 1016/apergo. 2018.03.015.
8. Hromatka BS, Tung YJ, Kieferb AK, Chuong BD, Hinds DA, Eriksson N. Genetic variants associated with motion sickness point to roles for inner ear development, neurological processes and glucose homeostasis. Hum Mol Genet. 2015 May 1; 24(9):2700-8. doi: 10.1093/hmg/ddv028.
9. Kuitunen T, Leino T, Parkkova K. Motion sickness: at sea and in the air. Duodecim; Laaketieteellinen Aikakauskirja. 2011;127(13):1378-80.
10. Khox G. Genetic factors in vestibular function and motion sickness. J. Otol. Rinol. 2015; 4:3. doi: 10.4172/2324-8785.1000225.
11. Krueger WWO. Controlling motion sickness and spatial disorientation and enhancing vestibular rehabilitation with a user –worn see-through display. The Laryngoscope. 2010; 121 (S2),S17-S35. doi:10.1002/lary.21373.
12. Thornton WE, Bonato F. Space motion sickness and motion sickness: symptoms and etiology. J. Aviation, Space, and Environmental Medicine. 2013;84(7):716-21. doi:10.3357/asem.3449.2013.

Стаття надійшла 22.12.2019 р.