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REVIEW ARTICLE

MODELLING MOTION SICKNESS

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Introduction: Motion sickness is an undesirable phenomenon and continues to be an unresolved problem. Therefore, research is conducted aiming to understand the etiology of this disease better, as well as to anticipate its symptoms. This research is increasingly supported by numerical calculations, for the needs of which models of severity of motion sickness symptoms are developed. The aim of this paper is to review and characterize the models of severity of motion sickness symptoms available in the literature, as well as examples of the use of these models in research.

Methods: Systematic review.

Results: The first part describes the four most commonly used models of severity of motion sickness symptoms. A graphic representation of models and mathematical relationships were presented, based on which severity of the disease is determined. Finally, several examples of the use of these models in research are listed.

Conclusions: Taking into account the limitations of using certain models, the most prospective model for predicting severity of motion sickness symptoms was presented. The specific advantages of this model were described, as well as the conditions under which the study using this model should be conducted, in order to ensure reliable results.

Keywords: motion sickness, mathematical model, sensory conflict, subjective vertical

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INTRODUCTION

Motion sickness, also known as kinetosis or travel sickness, is a term used to describe discomfort caused by actual or apparent movement. Usually, a person does not feel the symptoms associated with this disease while traveling independently. This disease can occur when traveling by such means of transport as e.g. a car, aircraft, ship, as well as during exposition in e.g. flight simulators [2,8]. For this reason, the following terms are interchangeably used to describe this disease: car sickness, air sickness, seasickness and simulator sickness. Due to the fact that motion sickness is an undesirable phenomenon and continues to be an unresolved problem, research is conducted in order to understand its etiology better and to predict its occurrence [17,21,24,26–28,31,57,59]. This research is also carried out in the area of computer simulations for the needs of which models of severity of motion sickness symptoms are developed [3,8,9,13,22,35,37,46,47,50,54,58].

Numerical studies on severity of motion sickness symptoms are conducted in two directions. In the first direction, attempts are made to understand and model basic physiological mechanisms responsible for the development of disease symptoms [2,4,10,48]. The second direction of action focuses on the development of models, which enable to predict the occurrence of disease symptoms in different movement environments [32,37,45]. Two approaches are used in the modeling of motion sickness: empirical and theoretical. In the empirical approach, the model is based on the results obtained from the measurement of motion sickness symptoms experienced by people exposed to various types of movement. Models that belong to the second, theoretical, approach, aim to explain the causes of motion sickness. The empirical approach, although it has not thus far yielded the expected results, is the most popular and most widely used in the research on this phenomenon. This approach is limited to finding interactions between movement and motion sickness, assuming that this is the main cause of the disease. The theoretical approach currently employs two theories: "Sensory Conflict theory" (SC) and "Subjective Vertical Conflict theory" (SVC). SC theory is based on the work of Reason and Brand [50,51], expanded by Oman [47,49], who developed the final form of the mathematical description of this theory. Its essence is that all situations that cause motion sickness are determined by the state of the so-called sensory rearrangement [51]. In this state there is a sensory conflict not only between signals from the organ of vision, vestibular organ and other motion sensitive receptors. This conflict also applies to signals expected by the central nervous system (CNS), based on prior experience. According to the author, the difference in these signals is the cause of motion sickness. The SC theory in motion sickness modeling was also described by Benson [2]. His model of motion sickness is presented in fig. 1.

An essential component of this model (fig. 1) is a comparator that compares the sensed motions with motions that are expected by the CNS.

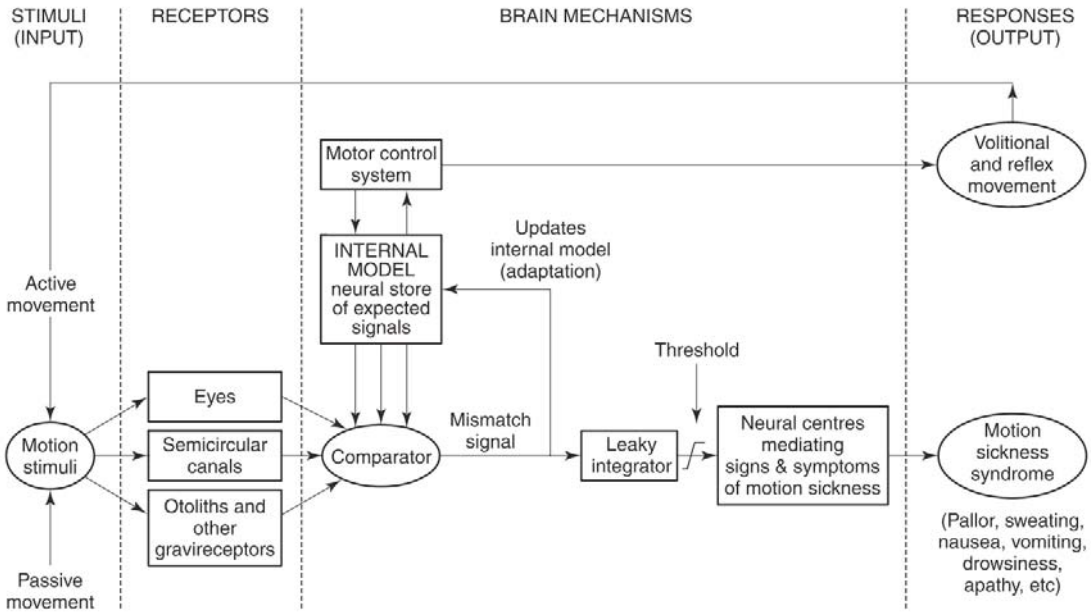

Fig. 1. The model of motion sickness according to the sensory conflict theory [2].

If these signals do not match, the comparator sends the so-called mismatch signal. This signal is used by the CNS to correct movement and position of the body. However, if the mismatch signal has a high value and is present over a longer period of time (e.g. in a boat moving up and down the waves), one of two reactions may occur [2]:

- adaptation to existing motions - internal model (CNS model), based on the mismatch signal, adjusts its motion expectations,
- motion sickness symptoms - cumulative sensory conflict.

The SVC theory, used in the second theoretical approach, was described by Bose and Bles [8]. It is a redefinition of the theory of sensory rearrangement [51]. The authors of this theory assumed that all situations that provoke the onset of motion sickness are characterized by the state in which the sensed vertical is contrary to the subjective vertical (SV) expected on the basis of previous experience [9]. Determination of SV takes place in the CNS on the basis of integrated signals from the organ of vision, vestibular organ and proprioceptors. The internal representation of the sensed vertical (SV), determined in the CNS, is a simplification of the classical theory of sensory rearrangement.

So far, scientists have made many attempts to develop models of severity of motion sickness symptoms [9,13,20,37,47,50,58]. These models describe mainly the mechanisms involved in the development of motion sickness, and also enable to determine the severity of its symptoms.

The aim this paper is to review and characterize the models of severity of motion sickness symptoms available in the literature, as well as examples of the use of these models in research.

As a result of a review of the available literature on motion sickness modeling, four of the most commonly used models of the severity of its symptoms are presented below.

Model Human Factors Research

As a result of numerous experiments conducted in Human Factors Research (HFR) Inc. California, USA [37,45], a model was developed that predicted the Motion Sickness Incidence (MSI) depending on the value, frequency and duration of vertical acceleration [37]. The authors of these studies have found that the results of measurements of the number and severity of motion sickness symptoms during its onset are variable and unique, while the symptom of vomiting is an observable marker of behavior. For this reason, the authors of this study have introduced the MSI index into the HFR model, which identifies the percentage of people who experienced vomiting under the influence of stimulating motion to the onset of the disease. HFR model [37] is described by the standardized normal distribution function for two variables: vertical harmonic oscillation with an amplitude z_a in the form of root mean square (RMS) and variable z'_t describing the vertical frequency f during exposure t. This model is described by the following equation:

$$MSI = 100 \cdot \Phi(z_a) \cdot \Phi(z'_t) \quad (1)$$

where:

$\Phi(z)$- is a normal distribution function of the variable z, determined by the relation:

$$\Phi(z) = \frac{1}{2\pi} \int_{-\infty}^{z} e^{-\frac{1}{2}x^2} dx \quad (2)$$

variables z_a and z'_t are defined as follows:

$$z_a = 2,128 \log_{10}(a) - 9,277 \log_{10}(f) - 5,809 \log_{10}^2(f) - 1,851$$
$$z'_t = 1,13\, z_a + 1,989 \log_{10}(t) - 2,904 \quad (3)$$

in which:

a - root mean square of vertical acceleration component [g],

f - frequency of vertical acceleration component [Hz],

t - time of exposition to acceleration [min].

A graphic, three-dimensional representation of the HFR model is presented in fig. 2. It shows that with the increase of the root mean square (RMS) of the amplitude of acceleration, the level of motion sickness symptoms increases.

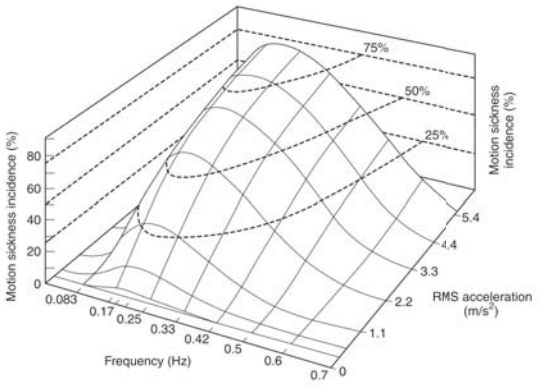

Fig. 2. Incidence of motion sickness as a function of frequency and acceleration, evoked by two-hour exposure to vertical (z-body axis) sinusoidal oscillation [2].

The highest saturation value of the disease symptoms occurs for the stimulus in the form of acceleration with the frequency of 0.16 Hz. In fig. 2, sections (25, 50 and 75%) were marked with horizontal plane, which correspond to the percentage of severity of disease symptoms. This model as-

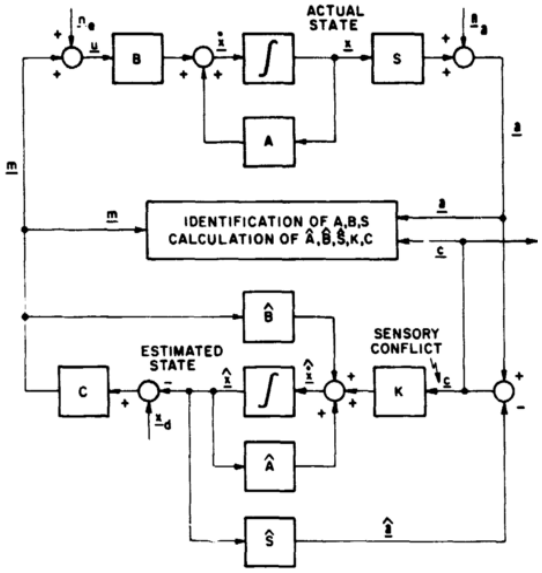

Fig. 3. Model for sensory conflict and movement control based on observer theory [47].

sumes that the maximum achievable level of disease symptoms amounts to 85%.

The disadvantage of using the HFR model is its rather complicated form and the fact that it is limited to vertical accelerations. For this reason, this model has been replaced by the concept of motion sickness dose as described by the Lawther and Griffin's model [32]. The model of motion sickness dose is currently the international standard used to predict the effect of vertical accelerations on the onset of motion sickness symptoms [16,56].

Oman's model

Oman [47,49] proposed a heuristic model of the dynamics of sensory conflict and locomotive motion assessment. This model made it possible to estimate subjective discomfort of a person, in the form of two motion sickness symptoms. The first symptom was the moment of occurrence of subjective complaints, while the second symptom was discomfort described by nonlinear function. The author has developed the model according to the SC concept [50], basing its structure on the optimal estimator model from the work of Borah et al. [6,7]. Fig. 3 presents the first of the two parts of Oman's model. This part is a model of sensory conflict and motion control, based on the state observer theory. Oman has distinguished sensor-level processing (upper part of the scheme) and processing in the CNS (the lower part, called the internal model). He determined the sensory conflict vector c (denotation $\underline{c}$ on fig. 3) from the difference of the vectors of the sensed state $\underline{a}$ and expected state $\underline{\hat{a}}$. These vectors represent information from all available sensors (among others, semicircular canals, otolith organs, organ of vision, proprioceptors). The increase in the value of the sensory conflict vector c indicates the increase of motion sickness symptoms. Fig. 4 presents the second part of Oman's model, which is a model of motion sickness. The input signal of this model is weighed sensory conflict h(*t*) It is processed through two parallel paths with fast and slow dynamics. Signals from both paths are added and then filtered with a fixed threshold. Thus the function R(t) is determined, which describes the severity of human discomfort.

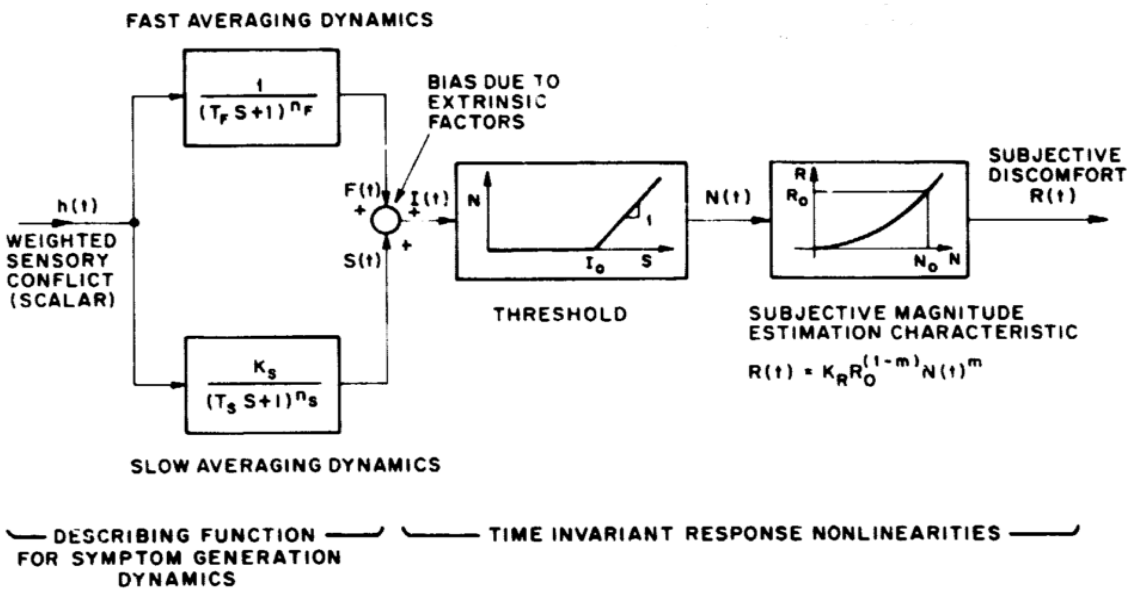

Fig. 4. Oman's model of subjective discomfort - motion sickness [47].

The susceptibility to motion sickness determined in this way depends not only on the degree of sensory conflict but also on the adopted amplitude of the signal processed in two paths, time constants and the threshold of nausea [49].

Oman has combined the sensory conflict model (fig. 3) with the motion sickness model (fig. 4), determining an intermediate signal h(*t*) - weighted sensory conflict. This signal is determined by the dependence:

$$\mathbf{h}(t) = \mathbf{c}^{\mathbf{T}}\mathbf{T}\mathbf{c} \tag{4}$$

where:
c – is the sensory conflict vector determined from the difference of the vector of the sensed state and the expected state vector (estimated in the CNS),
T – symmetrical matrix, whose coefficients describe the individual's sensitivity to the sensory conflict signal.

Oman estimated the orientation of the vertical by using optimum control technology. For this purpose, he assumed that the CNS uses a similar strategy in position and motion estimation. Thus, based on prior experience, the CNS estimates the orientation of the vertical using the responses of physical sensors (otolith organs) and the signals from the organ of vision. Oman's model [49] has some limitations. The model is linear and describes processing in the CNS using the state observer, although some sensory information is probably estimated in a nonlinear manner. In addition, this model does not take into account the process of adaptation to stimuli that cause motion sickness. A detailed description of the model can be found in the paper [47].

Bos and Bles's model

Bos and Bles's model [8,10,11] is an extension of Oman's model of motion sickness [47]. The main assumption underlying the theoretical construction of this model is the redefinition of the theory of sensory rearrangement [38]. The authors have assumed that motion sickness is primarily triggered in situations where the sensed vertical is not consistent (does not coincide) with the expected vertical determined in the CNS, based on prior experience. Bos and Bles formulated the SVC theory in this way. The researchers, when expanding Oman's model of motion sickness [47], included in its structure additional modules for determining the sensed vertical (response from the physical sensor of the otolith organ) and the expected vertical (response from the CNS). Fig. 5 Presents part of Oman's model of motion sickness [47] (thin lines) along with the modules added by Bos and Bles [4] (thick line).

The desirable state of the human body in fig. 5 is represented by the vector x_d. This vector is defined as follows. A person (matrix C) generates motor commands m that affect the model of body dynamics (matrix B). In this way he determines his position x . This signal together with external actuations n_e (e.g. from aircraft movement), is detected by human sense organs (matrix S). They are receptors of vision, vestibular organ and proprioceptors. In this way a signal is generated *a,* representing the sensed state of the body. In the blocks $\hat{\mathbf{B}}$ and $\hat{\mathbf{S}}$ the state of matrix B (motor activities) and matrix S (models of physical receptors) are recorded respectively. These are blocks representing processing at the CNS level. Similarly as in Oman's model [47], sensory conflict c is determined from the difference of the sensed state of the body *a* and expected state â, which is the output signal of the internal model (OUN model). Bos and Bles, using the fact that gravitational acceleration is constant, and the translational acceleration of the human body is usually short-lived, as suggested by Mayne [36], separated from gravitational-inertia acceleration *f=g-a* [42] sensed v_{sens} and expected

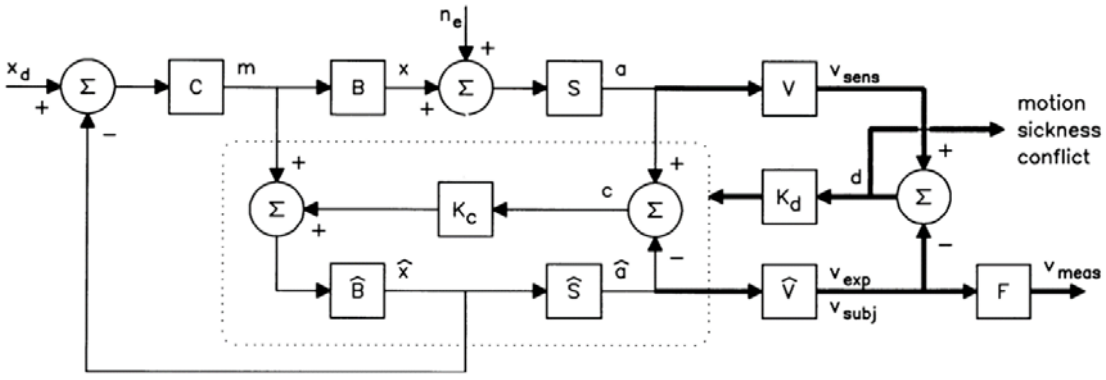

Fig. 5. Oman's model of motion sickness [47] (thin lines) extended by modules for determining the SVC (thick lines) [4].

v_{exp} vertical. For this purpose they used a low-pass (LP) filter described in the Laplace operator:

$$\mathbf{v}=\frac{1}{\tau s+1}\,\mathbf{f} \tag{5}$$

where: *f* - represents the response of the otolith organ to gravitational-inertia acceleration, τ - filter time constant, *s* - Laplace operator.

Equation (5) was used in blocks and $\hat{\mathbf{V}}$ (fig. 5), thus determining respectively sensed vertical v_{sens} and expected vertical. From the difference of vectors v_{sens} and v_{exp} a sensory conflict vector is created *d*, which is used in the next calculation step to update the vector v_{exp}. This updated vector v_{exp} is called a subjective vertical vector $v_{sub\ j}$ (fig. 5). In order to obtain the correct results, the sensed and expected vertical is determined in the Earth-fixed coordinate system. Then, vectors v_{sens} and v_{exp} determined by the dependence (5) are subject to a reverse transformation U^{-1}. As a result, vectors described in the head-fixed coordinate system are obtained. The principle presented above was presented in fig. 6 and described in detail in the paper [11].

Bos and Bles [9] indicate that the correct result of calculating the subjective vertical can be obtained using the following dependence:

$$\frac{d\mathbf{v}}{dt}=\frac{\mathbf{f}-\mathbf{v}}{\tau}-\omega\times\mathbf{v} \tag{6}$$

where:

ω - is the vector of angle velocity of the head, while

$\frac{dv}{dt}$ - derivative of the subjective vertical with respect to time in the inertial system.

Fig. 7 presents a complete Bos and Bles's model of motion sickness, which was developed according to the SVC theory for vertical motion.

The left side of this model (fig. 7), from which the sensory conflict vector is determined *d*, is the same as the extended structure of the Oman's model marked in fig. 5 with thick line. The sensory conflict vector is determined by the authors as follows (fig. 6 and 7):

$$\mathbf{d}=\mathbf{v}_{sens}-\hat{\mathbf{v}}_{exp} \tag{7}$$

In order to determine the MSI index, the sensory conflict vector *d* is nonlinearly transformed into a normalized parameter h and transformed using a second order function (fig. 7). A function

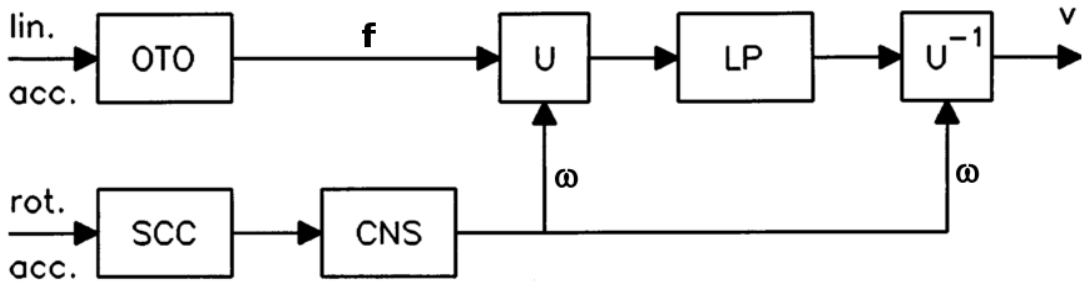

Fig. 6. Conflict model [9]. OTO - otolith organ, SCC - semicircular canal, CNS - central nervous system.

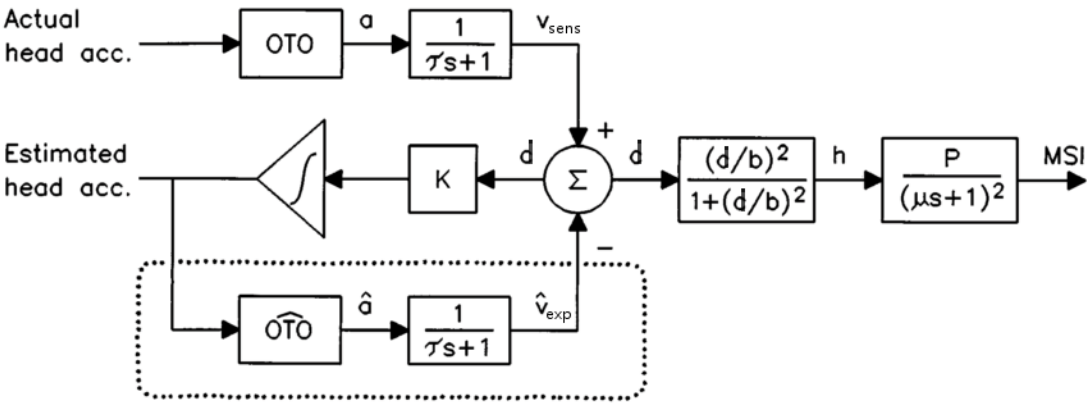

Fig. 7. Model of motion sickness based on the SVC for vertical motion [8].

that assumes a logarithmic or exponential form is used to describe the sensory conflict from mild to strong. For this purpose, the Hill function was used as follows [8]:

$$h=\frac{(\mathbf{d}/b)^n}{1+(\mathbf{d}/b)^n} \quad (8)$$

where:
d - sensory conflict vector, determined from equation (7),
b - parameter whose value is selected in the procedure of fitting the model quantitatively to the experimental data,
n - defines the inclination of the function h. The value of this parameter is in the range of n = 1 to n = ∞. For n = 2 and small sensory conflicts, the function h is exponentially increasing, while for larger conflicts it takes the form of a logarithmic function.

The second order filter was used to determine the severity of motion sickness symptoms, which reaches its maximum asymptotically and returns to zero after the sensory conflict has subsided. Its form is roughly described by the transmittance [8]:

$$MSI=\frac{P}{(\mu s+1)^2}h \quad (9)$$

where:
P - determines the maximum percentage of people who have had signs of motion sickness,
μ – time constant,
s - Laplace operator.

According to the SVC theory, only one conflict of signals is necessary, obtained from the difference of the response of the physical organs (otolith organs) and the SV - signal expected in the CNS, in order to determine the severity of motion sickness symptoms. As a result, stimuli do not need to be classified into different types of conflict, as is the case with the sensory regrouping theory. Although there are some examples of sensory conflicts that can cause motion sickness, according to Bles et al. [3] and Bos and Bles [8] , the internal representation of gravitational acceleration in the CNS is the basis of the SVC theory.

Lawther and Griffin's model

Lawther and Griffin [32,33] developed a motion sickness model, described in British Standard BS 6841 [16]. It is an international standard for predicting vomiting (VI) and illness rating (IR) in adults who have no adaptation to the stimuli that trigger these symptoms. The standard BS 6841 [16] determines the limits of "severe discomfort" for exposure to narrowband vertical acceleration of the body, in the frequency range of 0.1 to 0.63 Hz. In addition, this standard defines frequency filters used to determine the frequencies that cause a person to experience discomfort. The Lawther and Griffin's model is based on observations by McCauley et al. [37], as well as previous data from the work of Alexander et al. [1]. Lawther and Griffin divided the problem of motion sickness prediction into two parts. In the first part they used weighted vertical acceleration filtering, and in the second part root mean square of the response time. The developed model contains cumulative measure of motion sickness, which is determined by the Motion Sickness Dose Value (MSDV). In mathematical terms this model has the following form:

$$MSDV_Z=\sqrt{\int_0^T a_v^2(t)\,dt}=a_{RMS,\,v}\cdot\sqrt{T} \quad (10)$$

where: *T* - time of exposition (between 20 min and 6 hours), a_v - vertical acceleration filtered for a given frequency [45], $a_{RMS,\,v}$ - effective acceleration, defined as:

$$a_{RMS,\,v}=\left[\frac{1}{N}\sum_{n=0}^{N-1}a_v(n)^2\right]^{\frac{1}{2}} \quad (11)$$

for which: $a_v(n)$ - acceleration value from the nth sample after taking into account the weight depending on the direction of acceleration, N - number of data samples.

This model is best suited for the prediction of motion sickness, to which the dominant stimuli are vertical accelerations. A dependency was introduced to predict the percentage of people who may vomit VI=K_m**MSDV*$_z$ [%].

For parameter K_m=0.333 and *MSDV*$_z$ determined from equation (10), vomiting index reaches VI < 70%. This result concerns adults who did not have adaptation to the stimuli that trigger these symptoms [23]. In the case of prediction of disease symptoms, the illness rating (IR) is used, described by the dependency IR=1/50**MSDV*$_z$ [29].

The use of models in research of severity of motion sickness symptoms

Few models of motion sickness make it possible to predict to what extent adults will be agitated to nausea or vomiting under the influence of movement. Among them are the models described in this article. A significant group are models that can indicate only whether a given situation can trigger motion sickness and why, without determining its severity. Currently, more attention is being paid to the development of models of se-

verity of motion sickness symptoms. The research carried out in this respect mainly concerns taking into account other receptors in the model (e.g. vision, proprioceptors) and the effect of all components of linear acceleration and angular velocity in the induction of motion sickness. Particular attention is paid to the organ of vision, which is known to have a major impact on the severity of motion sickness. The presence of visual indicators such as references to horizontal line may sometimes be necessary to reduce the symptoms of this disease (e.g. nausea) [53]. Below are some examples related to this research.

The limitations of the HFR model, including among others the use of vertical linear acceleration and omitting the presence of visual information, were eliminated by Matsangas [34]. The author expanded the use of the HFR model by validating it, using data from the work of McCauley et al. [37].

Griffin [29,30] used the British model of motion sickness BS 6841 [16] to describe the level of well-being of a person, as an opposition to the disease. In the study, the author used a variety of motion stimuli, including vertical movement with frequencies from the range of 0.1 to 0.5 Hz. In the case of sinusoidal movements lasting up to 2 hours, the Griffin model was compatible with the HFR model, although in rare cases the results were different by up to 25% [8].

Förstberg [23] conducted studies on human response to various motion stimuli occurring in high-speed tilting trains. The author additionally included an assessment of the possibility of occurring of motion sickness symptoms to the assessment of the comfort of movement and ability to work. For this purpose he used the Lawther and Griffin's model [32,33], in which he developed his own weighting band-pass filter with the range of 0.08 to 0.35 Hz. As a result of the comparison of the model's responses with the results of the experimental studies, the author proposed supplementing $MSDV_z$ with a component describing distribution (leakage) of accumulated nausea.

Braccesi et al. [12–15] developed models of motion sickness based on both SC theory and SVC theory. The first UNIPG model was based on vestibular stimuli, which included the interaction of all three components of linear acceleration. The second model UNIPGSeMo also included the presence of the organ of vision. Such an extension of the model enabled to determine the inter-sensory conflict, which was not yet used in models that predicted motion sickness.

Bles [3] conducted a study on the effect of Coriolis and Pseudo-Corliolis on the severity of motion sickness symptoms. In this study, the author showed that the subjective vertical (SV) is a marker that can successfully be used to determine the severity of symptoms of this disease.

Elias et al. [22] developed a model of sensory conflict and motion sickness that they used in the study of the effect of artificially generated gravity. In this study, the authors used a motion sickness model developed by Oman [47], which they modified according to the study conditions. The modification involved the introduction of a quantitative sensory conflict model, by which input data for motion sickness model was determined. In this case, the sensory conflict was determined based on the dynamics of the head movements during spinning, and also on the basis of the developed transmittance, binding the angular acceleration with the response of the semicircular canals to this acceleration. In addition, the authors expanded the structure of the model with the adaptation parameter, which they defined in the process of classical validation of the model (comparison of the signal estimated by the model with the results of experimental studies).

Wada et al. [58] developed a mathematical model of the severity of motion sickness symptoms, integrating knowledge of vestibular system neurophysiology. The authors expanded Bos and Bles's model [8] up to six degrees of freedom, additionally taking into account the change in angular position of the head. This model was used in the studies on the effects of head tilt while driving on a bend on the onset of motion sickness symptoms.

Most of the models described above ignore the interaction between the semicircular canals and the otolith organ. This concerns the problem of determining gravitational acceleration from gravitational-inertia acceleration. This problem in most works was solved by applying the dependence (6) [25,36], while other researchers [5,40,43] use the internal model of the CNS for that purpose. Another limitation of the presented models is that they still omit in their structure the mechanism of habitation and restitution of the vestibular organ and the process of adaptation to the stimuli causing the motion sickness. Apart from the models of motion sickness described above, there is a large number of models that have been developed in accordance with the SC theory and are mainly used in estimating the perception of human spatial orientation [11,18,19,25,36,39,41,44,52,55,60]. These models can ensure the determination of human perception of spatial position and movement,

as well as SV. Therefore, they are often the core of the current models of the severity of motion sickness symptoms [13–15].

CONCLUSIONS

In the articles cited above and in the experiments described in them, there are models that meet the highest number of usability criteria, which determines their use in research. The most prospective model in the prediction of motion sickness symptoms seems to be the model developed by Bos and Bles [8,10,11]. This model is an extension of Oman's model [47], which includes the mathematical implementation of the SV conflict theory. Additional modules are used to determine to sensed and expected vertical, as well as the conflict vector between them. This model has several advantages, which include:

- it can be applied to most movement profiles, characteristic e.g. for car, air, maritime sickness, etc. [4].
- limited to only one conflict (subjective sense of the vertical),
- no need to use additional indicators in different movement profiles (situations), to explain why a person under some conditions is sick and under others is not,
- unlike Oman's model [47], as well as Lawther and Griffin's model, [32,33] there is no need for special filtering of the input signal or isolation of the stimulus in the form of pure vertical motion,
- confirmation in numerous frequency validations of 0.2 Hz [37] as the most provocative to the onset of motion sickness symptoms,
- continual development, conducted among others by the authors, as well as Braccessi et al. [13–15] and Wada et al. [58],
- ability to expand its structure in a simple way with additional receptors, e.g. visual [4,11] .

AUTHORS' DECLARATION:

Study Design: Rafał Lewkowicz; **Data Collection:** Rafał Lewkowicz; **Manuscript Preparation:** Rafał Lewkowicz; **Funds Collection:** Rafał Lewkowicz. The Author declares that there is no conflict of interest.

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