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A theory on visually induced motion sickness

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Abstract

This paper deals with visual–vestibular interactions as these occur when viewing images, and may cause motion sickness. Some basic vestibular matters fundamental to motion sickness are highlighted, such as the observation that people without functioning inner ears do not get sick from motion, including visual motion. Furthermore, the subjective vertical mismatch theory is described, and its corollaries regarding visual motion. A theoretical framework including visual–vestibular interactions is presented. This framework provides opportunities for studying, understanding, describing, and predicting visually induced motion sickness.

Keywords: Visual–vestibular interaction; Motion sickness; Gravity; Subjective vertical; Virtual environments

1. Introduction

When humans (and some other species as well) are exposed to motion, they may get sick. This sickness is characterised by ordinary feelings of malaise and typical symptoms like pallor, sweating, nausea, and eventually vomiting. Motion sickness severity and symptoms may differ between people and conditions. Psychological, physiological, sexual, ethnic, behavioural, and many, many other factors contribute to differences in individual sickness susceptibility. There are, however, universal trends in the severity of sickness depending on the motion characteristics, and here we will focus on these trends. The motions may concern physical self-motion, but also motion of the visual scene alone, and combinations thereof. Generally, self-propelled motion in a natural environment does not lead to sickness in healthy people. Typically, only artificial conditions do, like being on a moving platform or in a vehicle (possibly in space). Viewing visual motion may also induce sickness in stationary observers. Depending on the type of motion, one speaks of carsickness, airsickness, seasickness, cinerama sickness, cybersickness, simulator sickness, space sickness, and what more.

Though there are differences in motion characteristics between these different types of sickness, there are some fundamental similarities. A definite finding, known already for over 100 years, concerns the fact that people without functioning organs of balance in the inner ears, so called labyrinthine defectives (LD), never get sick from motion. This was first described by Irwin [32], who observed that a group of deaf mute co-passengers were immune during a sea voyage. James [33] described similar effects in his large record of LD patients. These observations have been confirmed many times thereafter under all different kinds of stimulation [35,46,51]. Interestingly, LD patients do also not suffer from visual motion, even in the absence of physical self-motion [18,19,34]. Another observation concerns the fact that people who do control their motion themselves (like car drivers and pilots) usually do not get sick, whereas passive passengers do [53,54]. Based on such observations, though often explicated at a later date, Reason and Brand [51] postulated the most cited conflict theory on motion sickness, stating that "motion sickness is a self-inflicted maladaptation phenomenon..., which occurs at the onset and cessation of conditions of sensory rearrangement when the pattern of inputs from the vestibular system, other proprioceptors and vision is at variance with

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the stored patterns derived from recent transactions with the spatial environment''. Though very successful in explaining a lot of observations, it also had some flaws, why Bles et al. [6] further detailed this theory focusing on the observation that people only get sick when there is an (apparent) change of gravity with respect to their head. They accordingly stated that ''all situations which provoke motion sickness are characterised by a condition in which the sensed vertical as determined on the basis of integrated information from the eyes, the vestibular system and the non-vestibular proprioceptors is at variance with the expected vertical as predicted on the basis of previous experience''. Visual–vestibular interactions can thus be assumed to be responsible for causing visually induced motion sickness too, and we will further elaborate on these issues in the next sections.

This elaboration does have a profound vestibular and rather hypothetical basis. Nevertheless, in our view, it is *the* basis of all sickness provoked by motion, and thus also by viewing moving images using whatever type of artificial display. Recall that labyrinthine defective subjects do not suffer from visually induced motion sickness either. Furthermore, it gives the possibility to discern different aspects involved in visually induced motion sickness, such as circular and linear vection, rod and frame effects, pseudo Coriolis and Purkinje effects, time delays, foreground/background motion, field of view, image velocity, frame rates, and the use of head mounted displays. These aspects will be included in the next sections.

2. A framework

2.1. Vestibular basis

To account for what may be called ''expectation'', as mentioned in both theories on motion sickness, Oman [48] postulated an internal model. Fig. 1 shows an elaborated version of this concept. Basically, this model describes the control of body motion and attitude. Here, a desired body state ($\mathbf{u}_d$), first enters a preparatory phase (P) thus directing a controller (C) that generates motor commands ($\mathbf{m}$) that subsequently drive the muscles in our body (B) to fulfil the desire.[1] With additional external perturbations ($\mathbf{u}_e$, e.g. by a vehicle, and including the acceleration due to gravity), this results in the actual body state ($\mathbf{u}$). This state $\mathbf{u}$ is sensed by visual, vestibular, and somatic sensors (som, vis, vest) which, together with subsequent central nervous system (CNS) processing and delays as discussed in the following sections, results in afferent signals[2] representing the state of the body ($\mathbf{u}_s$). It is assumed that the brain is expecting certain sensory feedback in response to the self-initiated changes in body state. This expectation is represented in the model by $\mathbf{u}'_s$, which is the output of a so called internal model. This internal model or ''neural store'' comprises a copy of the primary path (B′, som′, vis′, vest′, LP′), and is supposed to be created (or at least updated) by previous experiences. The primary input of this internal model is a copy of the motor commands $\mathbf{m}'$ that is also called an ''efference copy'' or ''corollary discharge'', terms originating from neuro-vestibular research.[3] Here the output $\mathbf{u}'$ should be a better estimate of the body state as compared to the output $\mathbf{u}_s$, and it is this estimate that is compared with the desired state $\mathbf{u}_d$ to generate the error signal ($\mathbf{e}$). It is also assumed that this output $\mathbf{u}'$ drives our psycho-physical responses like the subjective vertical (SV) and motion perception (MP), but also physiological responses like eye movements (EM) [10,12]. Hence, a coarse statement could be that we do not feel with our senses but with our thoughts (be it not in a cognitive way, and yes, sensory signals are at issue). Optimally, the other output of the internal model, $\mathbf{u}'_s$, should be equal to that of the primary path $\mathbf{u}_s$. If, for example, an external perturbation is present, these signals will not be equal. The difference or conflict $\mathbf{c} = \mathbf{u}_s - \mathbf{u}'_s$ may then give rise to an (additional) internal feedback signal, weighted by K, and used by the internal model to drive the difference towards zero. Whether the weighting K should be a mere gain matrix or whether it should also include dynamics, remains to be seen. Nevertheless, Oman [48] suggested that the resulting multi-dimensional conflict $\mathbf{c}$ (including signals coding for linear and angular motion, both sensed by the visual, vestibular, and somatosensory systems) is correlated with motion sickness ($\mathbf{s}$) as postulated by Reason and Brand [51]. Bles et al. [6], on the other hand, assumed that one set of motion parameters is created by all sensory systems and one by the internal model, and that only the difference between the gravity components correlates with motion sickness.

2.2. Gravity

An essential issue in this concept is the (neural) estimation of gravity. The basic problem here is that for the control of body motion, only inertial accelerations should be reckoned, where the acceleration due to gravity should be discarded. Because both accelerations are physically indistinguishable (Einstein's equivalence principle) our CNS is in trouble. If our CNS would not reckon the difference, for example, we would perceive the acceleration due to gravity as an upward motion with an acceleration of $g = 9.8\ \mathrm{m/s^2}$, thus resulting in orbital flight within five minutes ($\iint g\mathrm{d}t = 1/2gt^2 = 441\ \mathrm{km}$, with $t = 300\ \mathrm{s}$, which is about the level of the International Space Station). Obviously this is not the case, and our CNS is capable of distinguishing between inertial and gravitational accelerations.

[1] Note that throughout this paper we refer to scalars by italic low case characters, to vectors by bold characters, and to operators or matrices by capital characters.

[2] Afference means coming from a sensor.

[3] Efference means driving actuators.

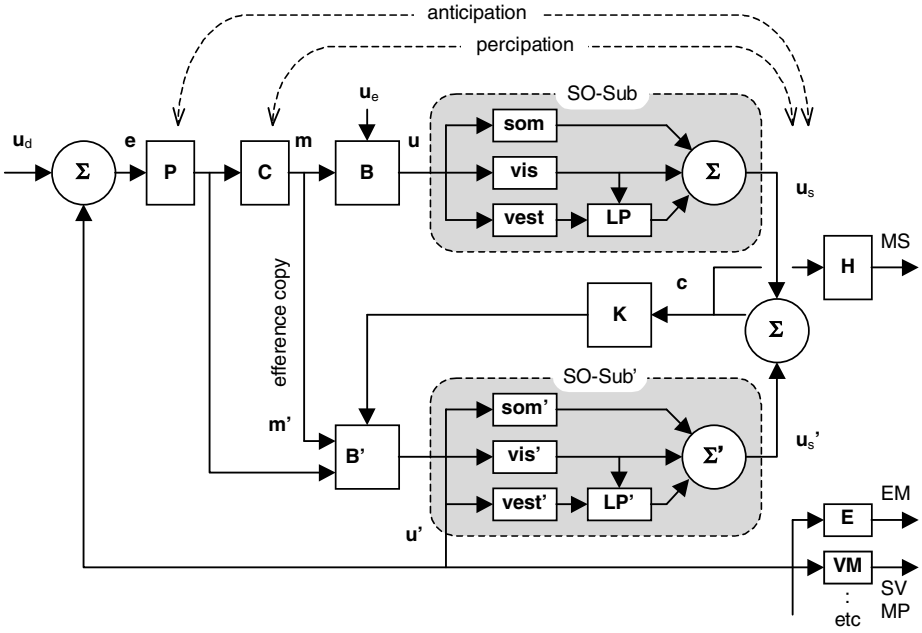

Fig. 1. Global overview of the spatial orientation and motion sickness model. **P** = preparatory phase, **C** = motor commands generating controller, **B** = body, **vis** = visual system, **som** = somatosensory system, **vest** = vestibular system, **LP** = low-pass filter, **H** = transfer function for motion sickness, **E** = eye movement system, **V/M** = verbal/manual system to finally give the SV and motion perception (MP), **K** = internal feedback (or Kalman) gain. Within the internal model, all functions have been indicated with a prime. The model's inputs are the desired body state ($\mathbf{u}_d$) and the external disturbances ($\mathbf{u}_e$). The outputs are motion sickness (MS), eye movements (EM), the subjective vertical (SV), and motion perception variables (MP). Note that summations may as well be complex (weighted) functions. Within this model, the grey areas encompass the two spatial orientation submodels essential for describing motion sickness.

Here, the somatogravic effect may give the answer to how our CNS makes the distinction.

If subjects are exposed to an acceleration step (e.g. a centripetal acceleration in a centrifuge), their sense of verticality will align gradually with the resultant specific force **f**, being the vector addition of inertial (**a**) and gravitational (**g**) accelerations: $\mathbf{f} = \mathbf{a} + \mathbf{g}$, where **a** is the centripetal acceleration here. Because this illusory sense of body tilt builds up logarithmically, e.g. [25], Mayne used a simple first order low-pass filter to describe the separation of the specific force into its inertial and gravitational components [39]. Though a large variability in time constants has been observed, we previously showed that this variability can be explained largely by the angular velocity in case the effect is shown in a centrifuge [10]. Taking this effect into account, the low-pass filter time constant is probably in the range of 1–5 s. Using low-pass filtering makes sense, because gravity, as we know it, is always constant, while accelerations due to self-propelled motions are variable. However, gravity is sensed by the otoliths within our inner ears. These otoliths function as head fixed linear accelerometers [21–23,38,39], whose transfer function is usually approximated by an identity matrix [41]. Thus, to account for head tilt, an estimation should be made of the acceleration components with respect to an Earth fixed frame of reference. To that end, the signals from the semi-circular canals can be used, the angular rate sensors within our inner ears. These canals are known to give a fair estimate of angular velocity ($\boldsymbol{\omega}$), be it that they exhibit high-pass characteristics [50,55] according to Eq. (1) (in Laplace notation), with a time constant $\sigma \approx 5$ s,

$$\boldsymbol{\omega}_{SCC} = \frac{\sigma s}{\sigma s + 1}\boldsymbol{\omega}. \tag{1}$$

Taking these matters together, we previously derived a three-dimensional differential equation approximating the estimation of gravity used by our CNS, according to the following equation

$$\frac{d\mathbf{g}}{dt} = \frac{(\mathbf{f} - \mathbf{g})}{\tau} - \boldsymbol{\omega} \times \mathbf{g}. \tag{2}$$

Here **g** represents the estimation of gravity, **f** the specific force as sensed by the otoliths [10], and $\boldsymbol{\omega} = \boldsymbol{\omega}_{SCC}$ the angular velocity as sensed by the semi-circular canals. In [10] it was also shown that (2) is the mathematical equivalent of describing the process by a low-pass filter, LP, operating in an Earth fixed frame of reference, where a rotation, R, first estimates the otolith afferent components in this Earth fixed reference frame, and an inverse rotation, R^{-1}, rotates the filtered components back into a head fixed frame of reference [7,10], giving

$$\mathbf{g} = R^{-1}(\boldsymbol{\omega}) \cdot LP \cdot R(\boldsymbol{\omega}) \cdot \mathbf{f}, \tag{3}$$

operating from right to left, and the rotation R using angular velocity $\boldsymbol{\omega}$ as signalled by the semi-circular canals according to Eq. (1).

In Fig. 1, Eq. (2) (or (3)) is applied twice, once in the primary path resulting in what may be called the sensed vertical, and once in the internal model giving an expected vertical. Because the time constant τ in Eq. (2) is relatively large, gravity conflicts $\mathbf{c}$ may easily exceed a certain threshold, thus giving rise to motion sickness. In this way, it is also explained why LD patients do not get sick from motion, an observation not accounted for by the original Reason and Brand theory [51], and the model proposed by Oman [48]. In these patients, there is no specific force to be low-pass filtered, thus reducing the lag between expected and sensed verticals. This is also why we assume that somatosensory afferents add to our sense of verticality only after this low-pass filtering has been realised. Note that, in theory, an incorrectly tuned internal model may still give rise to a conflict in these patients, why a total insensitivity not needs to be ruled out. Interestingly, some sickness in LD patients participating in a driving simulation experiment has been reported only recently [4].

This theory thus predicts that in static conditions the expected vertical always tends towards the sensed vertical. When $\boldsymbol{\omega} = 0$ and no inertial acceleration is added, the output of the LP-filter within the internal model will be equal to its input which input then equals the sensed vertical. No sickness therefore occurs in static conditions, even if these are illusory. Hence, also this theory brings up that motion is a prerequisite for motion sickness.

2.3. Seasickness

To account for further neuro-vegetative and gastrointestinal processes, an additional transfer function H is required resulting in a motion sickness incidence or an individual misery measure (MS in Fig. 1). This function H also reckons the fact that most people do not get sick immediately after motion onset, but symptoms generally become evident slowly, where they only reach a limit after several tens of minutes (but there are exceptions).

The concept described in the previous section has been elaborated for passive vertical sinusoidal motions in the absence of vision [9,10]. These conditions are in accordance with those that have been used in a large experimental laboratory study by McCauley cum suis in the 1970s to chart the effect of motion characteristics on observed seasickness severity [40]. The results of both exercises are shown in Fig. 2. Where the peak in incidence between 0.1 and 0.2 Hz was known before, the subjective vertical mismatch theory now gives a fundamental basis for the occurrence of this peak, and the high degree of similarity thus supports this hypothesis.

2.4. Percipation and anticipation

The internal model as part of the presented framework (already accounted for by Oman [48]), also accounts for the fact that drivers do generally not get sick from motion, while passengers do. Drivers do make use of efference copies by means of their internal model, making a prediction of self-motion. When their internal model is properly tuned, the expected and sensed verticals can be equal. In this respect, it seems likely that there is a reflex like control, which we propose to call "percipation", and a more extensive, possibly cognitive control, that normally would be referred to as "anticipation". The inner percipation or instantaneous control is typically realised within a second or so, while the anticipatory control may take longer. A typical example of percipation would be the result of controlling the steering wheel of a car and its gas pedal while making a curve. Anticipation would refer to the result of heading for a distant road curve and "knowing" that, after a while, this curve will be taken. In both conditions, the driver has most information and will thus suffer least from motion sickness. Passengers, however, may also benefit

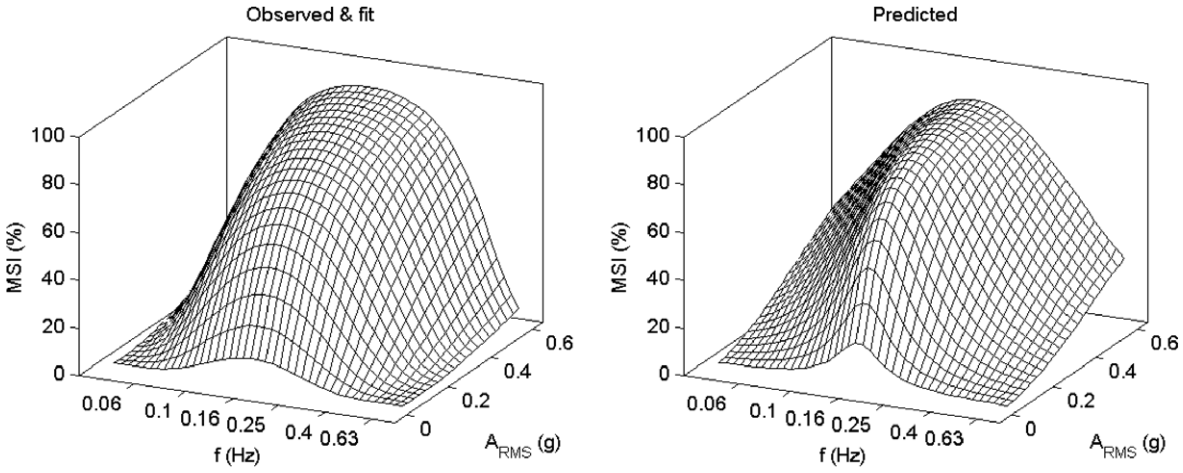

Fig. 2. MSI-descriptions. Left: fit through observed MSI-data of over 500 subjects by McCauley et al. [40] versus frequency (f) and RMS-acceleration amplitude (A_{RMS}). Right: predicted data by Bos and Bles [9].

from the additional (visual) information, explaining why sitting in the front is appreciated most by sufferers from car sickness, and facing backward (mere percipation) is worse than facing forward (allowing anticipation too). We assume that similar effects exist when viewing motion in simulators or when playing a computer game.

2.5. Habituation

Viewers of moving images may feel sick when exposed to a new environment, and feel better when exposed repeatedly. This process of habituation may be realised by the viewer "learning" the dynamics of the simulated environment, as realised by adaptation of his internal model. Only then he will be able to minimise the conflict between his sensed and expected verticals, and thus to minimise his motion sickness. With Reason and Brand [51] and Oman [48] we assume that the conflict itself plays an essential role in this habituation process. When a conflict lasts long enough, this is probably taken as a signal for the CNS to alter its internal model in such a way that the conflict is minimised. In case of disease this seems to work in a similar way. If, for example, sensor dynamics have been changed, this will give rise to a lasting conflict, associated with vertigo and nausea. Updating the parameters of the internal model duplicating the disease can, however, minimise this lasting conflict such that the symptoms disappear again. Habituation is well known to sailors too, and, interestingly, returning to a stable environment may cause a similar process to happen, which is known as "mal de debarquement". Furthermore, the process of internal model updating seems to have happened to us all in childhood. When making our first steps, we are "learning" the transfer functions of our body and sensor dynamics, a process of trial and error resulting in more than average conflicts. This thus explains why babies are immune to motion sickness: they just do not control their own body motion yet. It furthermore explains why children are most susceptible, and the elderly are the least susceptible [14], the latter having the largest neural store. Though never discussed before, it also explains why we do not get sick when asleep: something we can only do when there is no need for the control of body motion. The explanation of habituation may, lastly, also account for the anecdotal observations that young pilots do not get sick when trained in flight simulators, but may get sick once in the air, while older pilots do not get sick in the air (where they have been trained), but some do get sick in simulators.

3. Visually induced motion sickness

3.1. Circular vection

Within the framework sketched in Fig. 1, a dominant lag between sensed and expected afferents due to the low-pass filters LP and LP′, or more specifically, the differential equations (2) or (3), is essential for explaining motion sickness. Then, to explain that visual motion may give rise to motion sickness, this low-pass filtering should be affected by visual motion too. This is straightforward when considering circular vection, i.e., the sense of self-motion induced by circular optic flow, while being stationary oneself. It is known that visual and vestibular afferents both project onto the vestibular nuclei, thus giving rise to a sense of motion [1]. Because vection typically shows low-pass behaviour [20] with a time constant in the same order of magnitude as that of the canals (Eq. (1)) the addition of visual and vestibular afferents gives a fair estimate of true self-rotation [52]. The angular velocity $\boldsymbol{\omega}$ in (2) and (3) may thus be a direct linear addition of vestibular afferents ($\boldsymbol{\omega}_{SCC}$ according to Eq. (1)) and visual afferents coding for optic flow.

A corollary of the above reasoning concerns the sensation of a static self-tilt while viewing an optic flow pattern rotating in roll, i.e., about the naso-occipital axis. This is a paradoxical sensation, because the optic flow itself would result in complete vection, i.e., a sensation of continuous head-over-heels motion. Interestingly, Eq. (2) does have a stable solution, i.e., $d\mathbf{g}/dt = 0$, resulting in a constant angle of perceived self-tilt θ being proportional to the angular velocity of the optic flow pattern as derived by [10] according to the following equation

$$\frac{1}{\tau}(\mathbf{f}-\mathbf{g}) = \boldsymbol{\omega}\times\mathbf{g} \Rightarrow \frac{g\theta}{\tau} \approx \omega g \quad \Rightarrow \quad \theta \approx \omega\tau, \tag{4}$$

where $\mathbf{f} = \mathbf{g}_{veridical}$, $\boldsymbol{\omega}$ is perpendicular to $\mathbf{g}$, and a small angle approximation has been applied.

Applying this finding to our theory on motion sickness, yet another issue apart from the tilt according to Eq. (4) should be reckoned. We hypothesise that within the internal model there is only one variable for each kinematic component, i.e., there is only one $\boldsymbol{\omega}$, one $\mathbf{a}$, one $\mathbf{g}$, etc., and all sensory subsystems thus get the same input. While an artificial condition may cause $\boldsymbol{\omega}$ to differ for the semicircular canals and the visual system in the primary path of Fig. 1 (SO-sub), for example, these are equal in the internal model (SO-sub′ in Fig. 1). Viewing optic flow rotating about an off-vertical axis while being stationary oneself therefore likely leads to a conflict.

Here it is essential that the axis of rotation is not aligned with the Earth vertical. As with physical self-rotation, when the head is centred on an Earth vertical rotation axis, head movements are avoided (see below), and a view on the surroundings is present, self-rotation may make people dizzy, but sickness is exceptional or takes long to develop. It is only for off-vertical axis rotation (like barbecue-spit rotation) that subjects clearly develop sickness within a short interval of time. This may be either due to self-motion or to oblique visual motion [17,47]. Note that when the axis of rotation of the optic flow pattern is Earth vertical, $\boldsymbol{\omega}$ and $\mathbf{g}$ are parallel, why $\boldsymbol{\omega}\times\mathbf{g} = 0$ in Eqs. (2)–(4), and the sensed vertical will thus not tilt, why sickness is unlikely.

We are aware of the fact that optokinetic stimuli rotating about an Earth vertical axis are often used to produce

motion sickness, e.g. [8,17,31,56], and this could oppose our theory [11]. However, there is strong evidence that these motion sickness symptoms are typically much weaker and occur later than with other kinds of stimuli. For example, Bubka and Bonato [17] have shown that with only a 5-deg tilt of the rotation axis, significantly stronger effects come about, thus validating our assumption. Explanations why people may get sick when viewing optokinetic stimuli rotating about an Earth vertical axis include the possibility that they perceive the centre of rotation erroneously or may "think" they are viewing lateral motion. Both effects may result in tilts of the vertical. The perceived angular velocity may furthermore vary (as reported by Bubka and Bonato [17]), thus resulting in subjective tilt variations as well. This is straightforward when the axis of rotation is off-vertical, but also when the rotation axis is vertical these variations may add to tilt perceptions as explained above. A last argument in this respect concerns the natural variability in the internal representation of gravity that may be enhanced under artificial conditions such as viewing optokinetic stimuli. This variability is closely related to uncertainties present in the sensed and expected gravity estimations as discussed in the next section. Any variation in sensed and or expected verticals may then give rise to supra-threshold conflicts and thus to (visually induced) motion sickness. These effects may well explain why a standing observer who is yawing his head back and forth around an Earth gravity axis while looking through inverting goggles gets sick [57].[4] Because it is hardly possible to generate perfect on axis Earth vertical head rotation by oneself, we here assume a rather large conflict between the sensory afferents and internal model afferents where the intended rotation is Earth vertical. Uncertainties, rod and frame effects, and (pseudo) Coriolis and Purkinje effects as described below may further increase the conflict in such conditions.

All together, vection is a major factor explaining visually induced motion sickness characteristics. Effects of foreground/background motion (background motion typically giving more vection than foreground motion) field of view (peripheral motion giving more vection than central motion) and image velocity as described above may thus (at least partially) be understood.

3.2. Uncertainties

The internal model that determines our expectation derives its input from at least two sources: efference copies and sensory signals. Efference copies are used to make a prediction on self-motion. Sensor afferents are used to drive the internal model towards an output equal to that of the sensors, thus minimising the sensory-expectation conflict that is correlated with motion sickness. If the integrated signal coming from the senses is accompanied by significant noise or a high uncertainty due to intersensory differences for example, the weighting of this output is assumed to be low. Consequently conflicts are large, and motion sickness is likely to occur. A typical example would be a subject exposed to a highly artificial environment such as the optokinetic drum. Unusual postures with the neck extended may further increase this uncertainty. On the other hand, if subjects are certain about their being on a stable platform, this likely results in a high gain. Then, however, even the slightest motion of the assumed stable platform may result in a supra-threshold conflict thus giving motion sickness too. This "mental set" may explain why clients of a new mobile library complained about vertigo and nausea when others got on the bus thus causing it to move slightly, as recently reported in a Dutch newspaper. The use of this bus had to be delayed, and the suspension adjusted. The same motion would certainly not have given rise to motion sickness when experienced under normal driving conditions, even without vision.

3.3. Rod and frame effects

Furthermore, we assume that estimates of verticality are attracted by the orientation of a striped pattern, for example, equivalent to the rod and frame effect [24,58]. Hence, when any pattern with clear horizontal and vertical texture is tilted away from true vertical, a subjective vertical mismatch may arise thus making people sick. It is intriguing that this also holds for a static tilted environment, where people move themselves. This is observed, for example, in buildings tilted as the result of an Earth quake [36] or when moving around in a tilted fairground attraction [59].

3.4. (Pseudo) Coriolis and Purkinje effects

All of the above described effects are an issue when viewing any display where the image is moving, and/or the viewer is moving his head. In that respect (pseudo) Coriolis and Purkinje effects should be reckoned too. Coriolis (cross-coupled) effects refer to conditions in which the subject is rotating in the dark about an Earth vertical axis and the head tilts about another axis. Then, an illusory tilt is perceived perpendicular to both these axes, frequently accompanied by strong feelings of nausea with a fast onset, i.e., possibly within several seconds [5,28]. Purkinje effects refer to similar conditions; however, the head is then only tilted after the subject has come to a stand still and the canals signal rotation in the opposite direction [49]. Interestingly, these sensations only occur when the eyes are closed. When subjects do have a view on the Earth fixed environment around them, they will have a veridical

[4] Though inverting lenses as used by Stratton [57] do not necessarily alter the orientation of the visual scene, all visual motion is directed opposite to what is expected and to what the inner ear senses in case of actual head motion. This may yet indirectly affect our sense of verticality thus causing sickness. Moreover Stratton [57] did mention a deviation of the optical axis away from the visual axis causing a steady tilt of the visual environment. This may have caused nausea comparable to the observations by Kitahara and Uno [36] and de Wit [59]. Interestingly, Stratton [57] already refers to a "representation" or "idea" of certain conditions, a concept equal to that of an "internal model".

sensation of continuous self-rotation, and when the head is tilted, the visual information will further suppress the erroneous sensation of tilt. Pseudo Coriolis and pseudo Purkinje effects refer to conditions where the sense of rotation is induced by vision, and similar effects of tilt and nausea occur [16]. Here, again, it is the conflict between an unexpected tilt of the sensed vertical with respect to the expected vertical, the latter only tilting with the head (see Fig. 1) [5]. Because these effects are so strong, even small head movements while being submerged in a visual environment may induce pseudo Coriolis sensations thus explaining part of the visually induced motion sickness symptoms.

3.5. Linear vection

As with circular vection, linear visual motion may also give rise to a sensation of motion. The erroneous sensation of your train leaving, while viewing a train that is really leaving from an adjacent platform, is a well known phenomenon. However, where visual and vestibular subsystems both code for angular velocity, the otoliths code for linear acceleration [21–23,38,39], while the visual system typically is insensitive to linear acceleration [45]. Hence, to integrate visual and vestibular information into one percept of linear motion, either visual velocity has to be differentiated, or vestibular acceleration be integrated. Because we are aware of our self-motion velocity, while the existence of an acceleration percept is uncertain, we favour the integration of vestibular acceleration before adding this to a visual velocity signal. This process requires four essential steps: the estimation of inertial acceleration, acceleration integration, creating vection, and merging vestibular and visual signals.

Contrary to the estimation of gravity out of the otolith afferents, inertial acceleration (**a**) can first be estimated by high-pass filtering. This may be realised by simply considering

$$\mathbf{a} = \mathbf{f} - \mathbf{g} = \mathbf{f} - \frac{1}{\tau s + 1}\mathbf{f} = \frac{\tau s}{\tau s + 1}\mathbf{f}, \tag{5}$$

where we previously have shown that indeed our CNS seems to apply this rule [10], instead of using a separate high-pass filter with a different time constant as that used for the estimation of gravity.

By mere integration of linear acceleration, secondly, perceived velocity would continuously increase due to a constant acceleration like realised by centrifugation. Taking into account the estimated acceleration decrease to zero due to the low-pass filtering, still an acceleration pulse like signal would then result in a velocity offset. It has, however, never been observed that subjects do experience a constant velocity towards a centrifuge axis, for example. A relatively short acceleration pulse (like in a car) should result in a similar velocity offset by mere integration, and this too is not observed (at least with eyes closed). We therefore assume an additional high-pass filter with a time constant $v \approx 5$ s to cancel this residual velocity. Note that the integrator $(1/s)$ and high-pass filter can be merged into one leaky integrator $(v/(vs+1))$.

A simple first order low-pass filter

$$\frac{1}{\mu s + 1}, \tag{6}$$

thirdly, can create vection. In the literature, often a latency to vection onset is described of about 1 s [3]. To account for this latency, a logarithmic increase of perceived velocity conform (6) in combination with a velocity perception threshold may be assumed, with $\mu \approx 2$ s. This may also explain the general observation that latencies decrease with increasing stimulus amplitude.

The visual–vestibular interaction itself, lastly, may be assumed to be realised by a direct linear addition of the processed visual and vestibular signals, for these again exhibit opposite dynamics, low-pass versus high-pass, as was the case with circular vection.

Though this approach has been proven successful in predicting motion perception [26], the effects of linear visual motion regarding motion sickness are still undecided. Because we assume that visual flow information is only added to vestibular signals after the low-pass filtering has been realised, we would, however, predict that the nauseogenic properties of linear visual motion are less than those of circular flow. Nevertheless, especially under highly artificial conditions, differences between the sensed and expected verticals may give rise to sickness, because the internal model will be fed with signals that differ from those that feed the sensory subsystems in the primary path of Fig. 1.

3.6. Time delays

All of the above mentioned arguments hold not only for spatial aspects of visual–vestibular interactions, but also for the temporal aspects, like image processing induced delays in virtual environments. There too, most nausea is observed when head orientation changes with respect to gravity (personal observations). To explain the nauseating effect of time delays, it is essential again to understand that one sense of verticality is determined by our senses, and another by our experience or neural store as included in the internal model (Fig. 1). In case of a delayed visual scene, the sensed vertical will also be delayed particularly because of the dominance of visual cues. Our internal model, again, only ''knows'' synchronous motions, why the sensed and subjective verticals will be at variance with each other and sickness may be the result.

Similar effects may be realised when wearing a head mounted display (HMD). To stabilise our gaze direction in space, normally our eyes rotate opposite to movements of the head by means of the vestibulo-ocular reflex (VOR). If the image we are looking at moves with our head at low frequencies, the VOR is suppressed, however [37]. When high frequency head movements are not

appropriately compensated for in an HMD (which often is the case), there may thus be discrepancies as described above, likely causing sickness too [30].

3.7. Idiotropic information

Besides external visual and vestibular cues determining our sense of verticality, internal cues are of importance too. It has been shown, for example, that there is "a tendency to shift the subjective vertical towards the person's own longitudinal axis" or "idiotropic vector" [42–44]. This also explained the Aubert effect [2], i.e., a general tendency of subjects to underestimate the amount of self-tilt when subjected to physical lateral tilt. In addition, it has been shown that even in space where there is no net force acting on the body, subjects still do have a sense of verticality [60]. This latter too, can be explained by the existence of an idiotropic vector [44]. In that sense, we also assume that it is actually a subjective vertical mismatch causing space motion sickness, instead of merely an otolith–canal conflict as assumed before (e.g. [48,60]). This may further be exemplified by an astronaut's report of getting sick in the International Space Station immediately after floating from one compartment to another. Both compartments had ample visual orientation cues, but the circular tube in between did not. The astronaut had been rotating about his longitudinal axis within this tube, and was totally surprised by the orientation of the other compartment, which differed from what was expected (personal communication).

Because the idiotropic vector does play a major role in our estimation of verticality under certain conditions, it should be taken into account when integrating all possible cues determining our final subjective vertical. We anticipate that it will thus also affect motion sickness, be it that there is no data known to us explicitly validating this assumption yet.

3.8. Subjective vertical synthesis

The assumption posed by Mittelstaed [42] implicitly includes the possibility that the CNS performs a vector addition on the visual, vestibular and idiotropic vectors representative for the vertical, resulting in one sensed vertical. We here extend this idea by assuming that all components are represented by a vector which orientation is given by the specific subsystem, and which length represents the relative strength, i.e., its weighting. Note that, again, this process should be replicated in the internal model, after which the difference between the sensed and expected verticals may make us sick. Interestingly, Groen et al. [27] studied the effects of the individually varied vectors. This data can be used to further quantify the weighing of these vector contributions.

Using a tilting (tumbling) room, Groen et al. [27] manipulated the visual vector, i.e., its orientation with respect to the longitudinal body axis of the subject. Here, the visual vector will be represented by **p**, and is determined on the basis of frame and polarity conform [29]. The idiotropic vector, **i**, is assumed to be parallel with the longitudinal body axis. Subjects could be tilted within this room in addition, thus manipulating the orientation of the gravity vector (**g**) with respect to the subject. All tilts were realised in roll, i.e., in a lateral direction in the frontal plane. The four conditions of interest are represented by the vector plots of Fig. 3, drawn in a head fixed frame of reference.

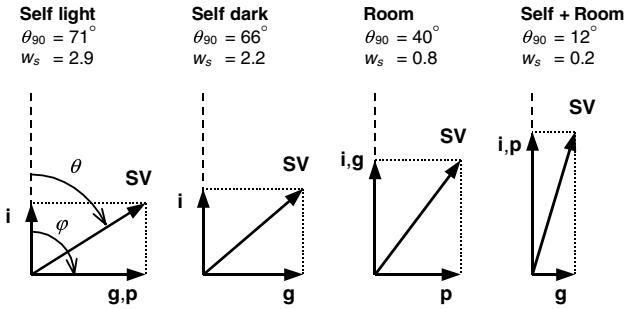

Fig. 3. Vector representation of vestibular (**g**), visual (**p**), and idiotropic (**i**) information in a head fixed frame of reference, inferred from Groen et al. [27]. "Self light" refers to lateral self-tilt with a view on the Earth vertical room. "Self dark" refers to lateral self-tilt without any vision. "Room" refers to lateral tilt of the tumbling room while the subject remains erect. "Self + Room" refers to a combined lateral tilt of both the room and the subject. At maximum tilt ($\varphi = 90°$) the subjective vertical θ_{90} as observed by Groen et al. [27] is given. The weighting coefficient w_s indicates the relative vector magnitude that best predicts the observed SV-settings.

As in the paper by Groen et al. [27], we take the angle θ of the subjective vertical, i.e., the angle between the subject's longitudinal body axis (**i**) and a tactile rod manipulated parallel with the subject's estimate of Earth vertical (or gravity) according to the following equation

$$\theta = \arctan\left(\frac{w_s \sin\varphi}{w_s \cos\varphi + 1}\right), \tag{7}$$

where w_s represents the ratio between the magnitude of the (set of) vectors that tilt about an angle φ with respect to the subject, and those which remain fixed to the head. From (7) it follows that

$$\tan\theta_{90} = w_s, \tag{8}$$

with θ_{90} being the angle θ at $\varphi = 90°$. Taking the data of Groen et al. [27], w_s reach the values as indicated in Fig. 3 in addition.[5]

From these data, its seems that whenever **p** and **i** are present, they are weighted mutually in approximately the same way, while the effect of **g** is variable. More specifically, it seems that the weighting of **g** depends on the angle between **p** and **i**. In case **p** and **i** are parallel, the weighting of **g** is small, if they are at an angle, **g** is weighted more heavily. If we consequently would assume that

[5] A detail concerns the fact that we defined w_s according to Eq. (8) here, being the reciprocal of that used by Groen et al. [27].

$$\mathbf{SV} = |\mathbf{g}| \frac{w_g(\mathbf{p},\mathbf{i})\frac{\mathbf{g}}{|\mathbf{g}|} + w_p\frac{\mathbf{p}}{|\mathbf{p}|} + w_i\frac{\mathbf{i}}{|\mathbf{i}|}}{w_g + w_p + w_i}, \tag{9}$$

with

$$w_g(\mathbf{p},\mathbf{i}) = a + \frac{b\alpha(\mathbf{p},\mathbf{i})}{90^\circ} \quad \text{and} \quad \alpha = \arccos\frac{\mathbf{p}\cdot\mathbf{i}}{|\mathbf{p}||\mathbf{i}|}, \tag{10}$$

where $\mathbf{p}\cdot\mathbf{i}$ represents the dot product of $\mathbf{p}$ and $\mathbf{i}$ and we confine to the interval $-90 \leqslant \alpha \leqslant +90°$. This implies that w_g assumes a certain minimum a when $\mathbf{p}$ and $\mathbf{i}$ are parallel, and a certain maximum $a+b$ when they are perpendicular. When applying Eqs. (9) and (10) to the different conditions from Fig. 3, and calculating the angle θ by which the subjects perceive the SV by

$$\theta = \arctan\frac{SV_y}{SV_z}, \tag{11}$$

we found in a first order approximation that $a = 0.2$, $b = 0.6$, $w_p = 1.0$, and $w_i = 0.4$. This suggests that the visual polarity is the dominant factor contributing the our subjective vertical, followed by either the vestibular or the idiotropic vector, depending on the angle between the visual polarity and the longitudinal body or head axis.

A more than philosophical issue, lastly, concerns the fact that the visual polarity and idiotropic vectors only give us information about the orientation of the gravity vector. To explain motion sickness, it is also essential to consider its magnitude. Because this magnitude is only sensed by the vestibular system, we consequently assigned the SV in (9) the magnitude of the vestibularly estimated gravity vector.

Combining all observations listed above, and building on previous work [13], the SO-submodel of Fig. 1 can thus be redrawn as sketched in Fig. 4. It may be argued, however, that the estimation of linear motion (resulting in **v** at the right in Fig. 4) does not use gravity as estimated by the R, LP, R^{-1} sequence, but an estimation of gravity that is also influenced by visual polarity, like the output **g** at the bottom right of Fig. 4, as demonstrated by [26].

4. Conclusion

In this paper, we discussed the subjective vertical mismatch theory on motion sickness as a further specification of the classical conflict theory by Reason and Brand [51], in the light of visual cues adding to our sense of verticality. All of these effects may well be induced by watching displays of whatever type. It may explain the sickness subjects submerged in flight or driving simulators experience (simulator sickness) as well as the sickness experienced by players of computer games (cybersickness). It may also explain why appropriate visual cues are beneficial when at sea and looking at the horizon, for example, while inappropriate cues are detrimental (like looking to an interior that is moving with the subject) [15]. On the other hand, it is also known from flight and driving simulation, that when a motion platform is tuned incorrectly, people may get sick, why it is sometimes said that "no motion is better than bad motion". The concepts presented here may thus provide a basis to optimise the motion characteristics not only with respect to realism, i.e., the realism of the perception, but to reduce motion sickness symptoms in these circumstances too.

We are aware of the fact that the framework presented here regarding visual–vestibular interactions is immature, and some details are rather hypothetical. Many results have not been validated yet, or are even still hidden. However, with the current state of technology, the visual–vestibular environment can be manipulated in sufficient detail to study all effects necessary to validate or to reject (parts of)

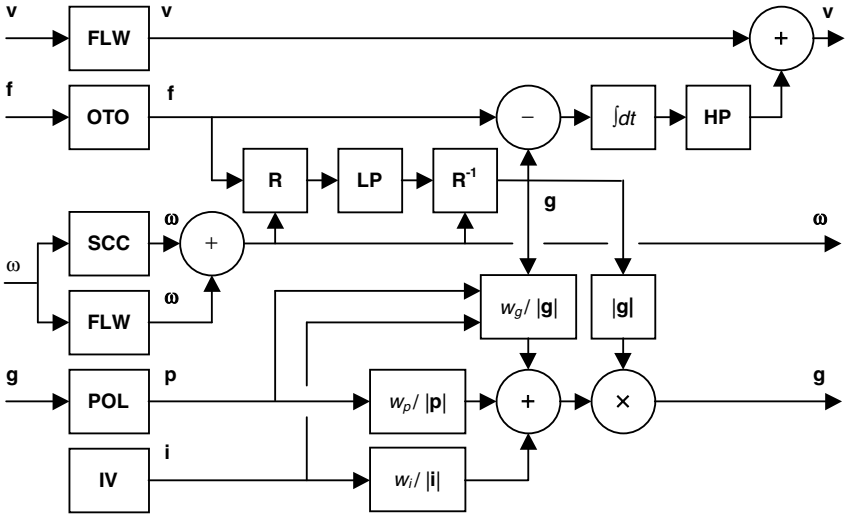

Fig. 4. Visual–vestibular SO-model. The blocks FLW give visual flow (m/s en °/s), OTO the specific force (m/s^2), SCC angular velocity (°/s), POL the orientation of the visual vertical (m/s^2, without referring to its magnitude) en IV gives the idiotropic vector (m/s^2, magnitude irrelevant as well). The factors w give the different weights regarding the different cues, where w_g has been made explicitly dependent upon the mutual orientation of the visual and idiotropic verticals.

our theory. Without going into details any further, each solution has its advantages (i.e., predicting observations properly) and disadvantages (predicting observations incorrectly). Because of the lack of specific data, it thus remains to be seen how the signal flow in a model like presented in Fig. 4 describes the processes realised in our CNS most appropriately.

Nevertheless, the theory presented here gives a consistent framework for understanding, explaining, and predicting visually induced motion sickness characteristics in more detail than possible before. It does put facts about the effect of foreground/background motion, field of view, image velocity, head movements, frame rates and delays, and the use of HMDs in a broader perspective. Because people who design and use visual displays should be concerned about the possibility of making their users sick, they should also be given the appropriate background knowledge to understand and deal with it. This paper may serve that purpose.

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