Why cybersickness

Jelte E. Bos[1]

[1]TNO Human Factors, P.O. Box 23, 3769 ZG Soesterberg, the Netherlands

[1]Jelte.Bos@tno.nl

Keywords: Visual-vestibular interaction; Motion sickness; Gravity, Subjective vertical; Virtual environments.

Abstract. This paper reviews a framework explaining motion sickness in general and cybersickness in particular. It takes the control of body motion as a starting point. An essential problem concerns the ambiguity between gravity and inertia. Although visual information can be used to make the distinction between these two phenomena, the visual system is yet too slow for accurate control of active body motion. The result of the apparent unsolvable ambiguity is a conflict between sensory and expected signals in a number of cases, such as when viewing certain visual motion unaccompanied by congruent physical motion, the conflict being highly correlated with sickness severity.

Introduction

When moving our body, the central nervous system (CNS) has to control the muscles in the body very accurately. Failing may result in falling. Adequate feedback using accurate sensors is required and both the eyes and the organs of balance play a major role in this respect. However, the visual system is relatively slow and only signals velocity and position. This is, for example, insufficient when avoiding obstacles while running, an almost impossible task for people without functioning organs of balance (e.g. deaf mutes). The vestibular system is fast, but only signals acceleration, including (the orientation of) gravity. This, by itself, is insufficient too, which is obvious when considering the previous example, i.e., we cannot avoid obstacles while running with our eyes closed. The integration of visual and vestibular signals in healthy people seems to be optimal for natural conditions, i.e., for self-propelled motion. When we are exposed to unnatural motion, however, like being moved by a car, ship, or airplane, or when visual motion is unaccompanied by physical self-motion, the solutions exploited by the CNS may appear to be less optimal. As in any feedback system, an error signal is probably used by our CNS to move the output (body) towards a desired state. It is assumed that this error signal correlates with motion sickness in general and cybersickness in particular. In the following sections, this will be explained further. For a proper understanding thereof, some general observations on motion sickness will be highlighted first. Then vestibular and visual perception and their interactions will be considered. The implications regarding visually induced motion sickness will finally be discussed, i.e., the question why we may suffer from cybersickness.

Motion sickness

There are a number of facts about motion sickness in general that are important for a proper understanding of the phenomenon. A trivial one concerns the fact that, by definition, (apparent) motion is a prerequisite. However, not all motion is as

provocative. For example, rotation about an Earth vertical axis without further head movements is by far less provocative than an equal rotation about an Earth horizontal axis (or barbecue spit rotation; Bles, 1998).

Second, probably the most important fact concerns the observation that people without functioning organs of balance do not get sick from motion at all (e.g., Irwin, 1881; Money, 1970), which, interestingly and importantly, also holds for visual motion (Cheung et al., 1991). This implies that functioning organs of balance also are a prerequisite for suffering from cybersickness.

The visual system, third, however, is not necessarily required for motion sickness, as is illustrated by the observation that blind people can get motion sick (Graybiel, 1970).

A fourth important factor in motion sickness is anticipation. Car drivers rarely get sick, while passenger do (Rolnick and Lubow, 1991). As will be explained below, this is due to the amount of information available on self-(generated) motion.

Part of these facts led Reason and Brand (1975) to state 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 the stored patterns derived from recent transactions with the spatial environment". As a consequence it is often erroneously assumed that intersensory conflicts per se may also lead to motion sickness. Rotation about an Earth vertical axis in the light is an example of the opposite. Then there is a conflict between vanishing vestibular signals and lasting visual signals (see below), a condition generally not leading to motion sickness. As a consequence, Bles et al. (1998) therefore further detailed the theory of Reason and Brand (1975) by emphasising the role of expectancy and recognising the important role of the vertical in motion sickness. This was prompted by 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 characterized 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". This theory will be further elaborated in the next sections, finally focussing on visually induced motion sickness. Because of the crucial role played by the organs of balance, this elaboration will start with that of vestibular perception.

Vestibular perception

When sitting still on Earth, we feel a force of the seat pushing to our bottom opposing gravity (Newton's 3rd law). This force would be exactly equal to that caused by an acceleration of the seat upward in microgravity by 9.8 m/s^2 (Newton's 2nd law). Though of different physical natures of gravity and inertia (or motion), these forces, or the associated accelerations, are yet indistinguishable (Einstein's equivalence principle). To state this differently, there are no devices capable of making the distinction between an acceleration due to gravity and due to inertia. Consequently this also holds for the organs of balance as well as all other proprioceptive sensors. If our CNS would not reckon the difference, we might feel being displaced over a distance of about 440 km in only 5 minutes when sitting still on Earth [1]. This, however, is not what we generally experience. Our CNS apparently makes some inference allowing the distinction to be made yet. At the end this may be the most crucial point in the aetiology of motion sickness, why this unravelling will be discussed further below.

When moving on Earth, the otoliths, the linear accelerometers within the inner ears,

[1] Calculated by integrating 9.8 m/s^2 twice over a time interval of 300 s.

respond to the specific force (**f**), i.e., the vector sum of gravitational (**g**) and inertial (**a**) accelerations:

$$\mathbf{f} = \mathbf{a} + \mathbf{g}\,. \tag{1}$$

One way to make the distinction between the two types of acceleration is filtering. This suggestion is based on the notion that gravity is constant whereas accelerations due to self-propelled motion are generally of short duration or oscillating. Thus, low pass filtering of otolith afferents[2] will give an estimate of gravity, and high pass filtering will give an estimate of inertial acceleration. This idea was first posed by Mayne (1974), and was incited by the somatogravic illusion. This illusion comes about when subjects are seated upright at the end of a centrifuge arm, which is abruptly brought to a constant angular velocity. Subjects will then initially feel substantial motion, but this is gradually changed into a static perceived tilt, suggesting that the direction of the specific force is assumed to be that of gravity. The amount of tilt was shown to behave like the output of a low pass filter.

Where Mayne (1974) posed his theory in two dimensions, we have further elaborated on this in three dimensions. When including the fact that gravity is only constant in an Earth fixed frame of reference, it was shown that the head-fixed process of low-pass filtering realised in this Earth fixed frame of reference can be described by the vector differential equation

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

The tilde is used to indicate the estimated gravity vector. The time constant τ is assumed to be between 1 to 5 s (see Bos & Bles, 2002). Here, $\boldsymbol{\omega}$ represents angular velocity that can be obtained from the visual system and the semicircular canals (SCC). The SCC are part of the organs of balance and respond to angular acceleration ($\boldsymbol{\alpha}$) according (in the Laplace domain):

$$\begin{aligned} \boldsymbol{\omega}_{SCC}(s) &\approx \frac{\tau}{\tau s + 1}\boldsymbol{\alpha}(s) \\ &= \frac{\tau s}{\tau s + 1}\boldsymbol{\omega}(s). \end{aligned} \tag{3}$$

with a time constant $\tau \approx 5$s (Raphan et al., 1979). This implies that the SCC function as angular rate sensors above frequencies of about 0.5 Hz.

The importance of Eq. 2 is given by three reasons. First, it shows the imperfection of our estimate of attitude ($\tilde{\mathbf{g}}$) and self-motion (conform Eq. 1: $\tilde{\mathbf{a}} = \mathbf{f} - \tilde{\mathbf{g}}$) as observed in the somatogravic illusion, for example (see Bos and Bles, 2002).

Second, very low frequency motion does not make sick, as motions with frequencies above 1 Hz do not do either. About 0.2 Hz, however, motions are most provocative (O'Hanlon and McCauley, 1974; Golding et al., 2001). Bos and Bles (1998) have previously shown that this can be attributed to the low pass filter essentially making the distinction between gravity and inertia, which time constant determines the mentioned 0.2 Hz peak.

Third, it points to the way our CNS integrates visual and vestibular information, and this will be explained in the next section.

Visual perception

The visual system is rather complex. Two optical devices with variable focus and automatic luminance control project images for stereo vision onto chemo-electrical light sensitive layers with some pre-processing capabilities. Signals are relayed to a complex cortical processing unit, which derives estimates of object and self motion. These signals, in turn, are used to move the eyes focussing on the object of interest again. This process requires profuse neural activity and many synaptic relays, and these may just be too slow for the delicate control

[2] Afference means coming from a sensor.

of self motion. Yet for certain tasks the visual system is indispensable, and there are three phenomena essential to visually induced motion sickness. These phenomena are all related to the vertical.

First, the visual scene generally shows information about what is up and what is down, termed polarity. Trees have got their crown, houses their roof, and people (generally) their head on top.

Second, most visual scenes do have essential structures oriented horizontally (floors, the horizon), and vertically (trees, walls), called frame information. The importance thereof is shown by the famous rod and frame test, described originally by Wertheimer (1912) and Gibson and Mowrer (1938) thereafter. When asked to align a free rotating rod within a fixed but tilted frame with the true vertical, subjects generally move it towards the orientation of the frame.

Third, the visual system is also sensitive to optic flow. Interestingly, it does not respond to linear acceleration, but does respond to velocity (Monen and Brenner, 1994). Moreover, visual neurons project onto the vestibular nuclei, as do neurons from the organs of balance, giving rise to self motion perception (Allum et al., 1976). Here, self motion perception in stationary subjects induced by optic flow is called vection. If this furthermore concerns angular motion, circular vection typically shows low-pass filter behaviour (Dichgans and Brandt, 1978). The time constant thereof seems to be in the same order of magnitude as that of the (high-pass) transfer function of the semicircular canals (see Eq. 3), i.e.,

$$\boldsymbol{\omega}_{vis}(s) \approx \frac{1}{\tau s + 1}\boldsymbol{\omega}(s)\,. \qquad (4)$$

This implies that under normal conditions, visual and vestibular signals do give a veridical sense of angular motion when added directly. If the optic flow is presented rotating about an Earth horizontal axis, the vestibular sensors inhibit a continuous head-over-heel motion sensation. Instead, it is observed that, depending on the angular velocity of the optic flow pattern, people feel tilted over a fixed angle. Interestingly, Bos and Bles (2002) have shown that this can be explained by Eq. 1. Moreover, following the theory by Bles et al. (1998), this perceived tilt of the vertical can thus indeed cause motion sickness too, and this will be explained further by considering the control of body motion more closely.

A control model

Returning to the delicate control of body motion, we might still fall when using all available sensory information directly for comparison with a desired state, as in a simple servo mechanism. Neural delays, the slow response of the visual system, and the imperfect estimate of inertial accelerations and gravity, would lead to instabilities and drift. It may therefore be assumed that the CNS uses an observer strategy instead (see Oman, 1982). A possible implementation thereof is shown in Fig. 1. Then, starting with a desired state of the body ($\mathbf{u}_d$), a control mechanism (C) generates motor commands (**m**) driving the muscles in our body (B). Here, the body may be moved by external motion ($\mathbf{u}_e$) in addition, resulting in the actual state **u**. A copy of the motor commands (the efference [3] copy **m**') is assumed to drive an internal model of the body dynamics (B'). If B' would be exactly equal to B, the prediction **u**' would be the perfect control variable for the primary feedback purpose. The internal model can next be extended with a copy of the sensory system ("som", "vis", "vest", and the CNS integration of these signals as shown in Fig. 1). The final output of the internal model ($\mathbf{u}_s$') should then be equal to the sensory output ($\mathbf{u}_s$). If external motion ($\mathbf{u}_e$) perturbs the actual body state, the difference **c** = $\mathbf{u}_s$'−$\mathbf{u}_s$ can be fed back via a gain (K) to the internal model so as to drive the conflict towards zero again and the output **u**' to **u**. Returning to the main topic of this paper, the conflict **c** is then assumed to be correlated with motion sickness (MS), via a

[3] Efference means driving an actuator.

neuro-vegetative transfer function H. Interestingly, the fact that motion sickness has to do with the control of body motion indeed is exemplified by the fact that babies not moving themselves around never get sick from motion. They only start getting sick when they start learning to walk. Problems increase up to an age of about 20, and decrease again when grown up and the internal model is mature as well (Bos et al., 2007). The reader interested in further details is referred to Bos and Bles (1998, 2002), and to Bos et al. (2007) for a focus on visually induced motion sickness.

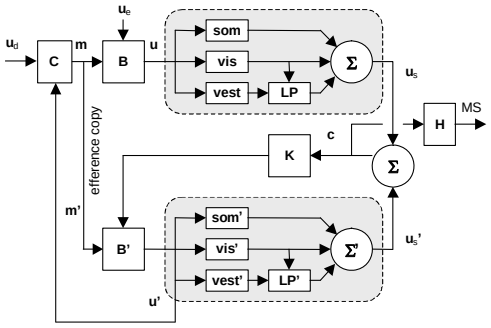

Fig. 1. Observer model integrating visual, vestibular and somatosensory signals (see text for further explanation.

That's possibly why

Fig. 1 essentially summarises why visual motion can be provocative.

First, it follows from this figure that in stationary subjects viewing a stationary scene, the output $\mathbf{u}_s$' is always driven towards the output $\mathbf{u}_s$. No sickness therefore occurs in static conditions, even if these are illusory. Of course this only holds for healthy subjects whose internal models are up to date. Hence, the model brings up that motion is a prerequisite for motion sickness.

Second, and most essentially, it shows that a delay in sensory output is primarily caused by low-pass filtering of vestibular signals, and these signals are relayed to the internal model causing a second delay. The result is that the conflict signal **c** behaves as a second order response, corresponding with the general observations on the frequency dependency of motion sickness as discussed above.

Third, it shows that somatosensory information is used, but these are assumed not to be delayed by the low-pass filter, hence not giving rise to motion sickness. In people without functioning organs of balance the low-pass filter is therefore ineffective. This makes them insensitive to motion sickness, including visually induced motion sickness.

Visual information, fourth, is relayed to the low-pass filter as discussed above, why visual motion may also make people sick. However, these visual signals are used to modulate vestibular signals, why labyrinthine defectives do also not suffer from cybersickness.

Fifth, Fig. 1 shows that once an estimate of motion and gravity have been determined by the senses, these single estimates are relayed to the internal model as an equal input for all its subsystems. Hence, any intersensory conflict between the actual senses does not occur in the internal model. It is therefore not the intersensory conflict per se that causes motion sickness, but it is the indirect effect thereof resulting in a conflict between internal model and sensory signals.

Lastly, the conflict is assumed to not only drive the internal model output towards the sensed output, but it is also assumed to trigger a process of updating the internal model in case of a lasting conflict. This typically happens when learning to walk and growing up. It also seems to be case when we are exposed to novel motion environments which are provocative initially, but to which we habituate when exposed to this longer and/or more frequently.

Though the presented framework does need further elaboration, it is believed to encompass the essentials leading to motion sickness in general, and visually induced motion sickness in particular. In conclusion it is suggested that the cause of cybersickness is the result of a combination of three issues involved in the control of body

motion: 1 - the disambiguation of attitude and motion (or gravity and inertia), 2 - the relatively slow response of the visual system, and 3 - the use of an internal model to optimise the control of body motion. For a more thorough elaboration, including that of sickness induced by optokinetic drums, linear vection, pseudo Coriolis and Purkinje effects and time delays, the reader is referred to Bos et al. (2007).

Acknowledgement

This research has been supported by the GATE project, funded by the Netherlands Organization for Scientific Research (NWO) and the Netherlands ICT Research and Innovation Authority (ICT Regie).

References

Allum J.H., Graf W., Dichgans J. and Schmidt C.L. (1976) Visual-vestibular interactions in the vestibular nuclei of the goldfish. Experimental Brain Research 26 pp. 463-85.

Bles W. (1998) Coriolis effects and motion sickness modelling. Brain Research Bulletin 47 pp. 543-9.

Bles W., Bos J.E., De Graaf B, Groen E. and Wertheim A.H. (1998) Motion sickness: only one provocative conflict? Brain Research Bulletin 47 pp. 481-7.

Bos J.E. and Bles W. (1998) Modelling motion sickness and subjective vertical mismatch detailed for vertical motions. Brain Research Bulletin 47 pp. 537-42.

Bos J.E. and Bles W. (2002) Theoretical considerations on canal-otolith interaction and an observer model. Biological Cybernetics 86 pp. 191-207.

Bos J.E., Bles W. and Groen E.L. (2007) A Theory on visually induced motion sickncess. Displays, accepted.

Bos J.E., Damala D., Lewis C., Ganguly, A. And Turan, O. (2007) Susceptibility to seasickness. Ergonomics 50 pp. 890-901.

Dichgans J. and Brandt T. (1978) Visual-vestibular interaction: Effects on self-motion perception and postural control. In: R. Held, H.W. Leibowitz, H.L. Teuber (eds), Handbook of sensory physiology Vol. VIII: Perception (Springer-Verlag, Berlin, pp. 805-45.

Cheung B.S.K., Howard I.P. and Money K.E. (1991) Visually-induced sickness in normal and bilaterally labyrinthine-defective subjects. Aviation Space and Environmental Medicine 62 pp. 527-31.

Gibson J.J. and Mowrer O.H. (1938) Determinants of the perceived vertical and horizontal. Psychological Reviews 45 pp. 300-23.

Golding J.F., Phil D., Mueller A.G. and Gresty M.A. (2001) A motion sickness maximum around the 0.2 hz frequency range of horizontal translational oscillation. Aviation Space and Environmental Medicine 72 pp. 188-92.

Graybiel A. (1970) Susceptibility to acute motion sickness in blind persons. Aerospace Medicine 41 pp. 650-3.

Irwin J.A. (1881) The pathology of seasickness. The Lancet 2 pp. 907-9.

Monen J. and Brenner E. (1994) Detecting changes in one's own velocity from the optic flow. Perception 23 pp. 681-90.

Money K.E. (1970) Motion sickness. Physiological Reviews 50 pp. 1-39.

O'Hanlon J.F. and McCauley M.E. (1974) Motion sickness incidence as a function of the frequency and acceleration of vertical sinusoidal motion. Aerospace Medicine 45 pp. 366-9.

Oman C.M. (1982) A heuristic mathematical model for the dynamics of sensory conflict and motion sickness. Acta Otolaryngologica Supplement 392.

Raphan T., Matsuo V. and Cohen B. (1979) Velocity storage in the vestibulo-ocular reflex arc (VOR). Experimental Brain Research 35 pp. 229-248.

Reason J.T. and Brand J.J. (1975) Motion sickness. Academic Press, London.

Rolnick A. and Lubow R.E. (1991) Why is the driver rarely motion sick? The role of controllability in motion sickness. Ergonomics 34 pp. 867-79.

Wertheimer M. (1912) Experimentelle Studien ueber das Sehen von Bewegung. [Experimental studies on visual observation of motion] Zeitschrift fuer Psychologie 61 pp. 161-265.