Nuancing the relationship between motion sickness and postural stability

Jelte E. Bos

TNO Human Factors, P.O. Box 23, 3769 ZG Soesterberg, The Netherlands

ARTICLE INFO

Article history:
Available online 1 October 2010

Keywords:
Posture
Instability
Motion
Sickness

ABSTRACT

The most cited theory on motion sickness is the conflict theory by Reason and Brand (1975) [1], stating that motion sickness occurs due to a conflict between the senses and stored patterns of motion. In addition, there seems to be evidence for another theory stating that postural instability is a necessary and sufficient condition preceding motion sickness (Riccio and Stoffregen, 1991) [2]. A number of additional observations reviewed in this paper, however, are nuancing the relationship at issue, thus devaluating the latter theory. Moreover, a central mechanism as assumed before (Bos and Bles, 2002 [20]), driving both our posture and motion sickness symptoms, may explain why and when postural instability is correlated with sickness, and when it is not.

© 2010 Elsevier B.V. All rights reserved.

1. Introduction

Of all theories on the aetiology of motion sickness, the most cited one is given by Reason and Brand [1], a statement quite often used by itself as well. Their theory states 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 (pp. 274–275). Although successful in many respects, Riccio and Stoffregen [2], going back as far as Gibson [3], posed that sensory conflict, however, is hypothetical rather than a fact itself. This led them to propose their postural instability theory, stating that animals become sick in situations in which they do not possess (or have not yet learned) strategies that are effective for the maintenance of postural stability (p. 195), and that postural instability precedes the symptoms of motion sickness, and that it is necessary to produce symptoms (p. 205). Re-phrased, they also stated explicitly that an animal will become sick if and only if it picks up information about prolonged postural instability (p. 219). Many studies have indeed reported (positive) correlations between postural instabilities (or ataxia) and motion sickness, e.g., [4–10], and postural stability indeed precedes motion sickness in several cases, e.g., [11–14]. Furthermore, restrained stance, e.g., [15] or sitting [13] has been shown to give less sickness than free stance, and balance training has been shown to be effective in rehabilitation for motion sickness [16]. Kennedy and Stanney [17], however, commented on both theories, and reached the conclusion that motion sickness and postural instability may both be second order effects under control of a common center, but, as they stated themselves, they take no position regarding either theory (p. 27). In this paper I will go a step further elaborating on these theories, to reach the conclusion that both postural instability and motion sickness do result from a shared mechanism explaining certain correlations, and implying that postural instability is not a necessary nor a sufficient condition for motion sickness. Although this elaboration does show an overlap with previous publications [18–21], it takes a different point of view, i.e., on the control of body motion.

2. A theoretical framework

Standing, walking, and running erect on two legs is an art requiring a control system with adequate sensors, ample redundancy, and clever adaptive integration of sensory signals. Failing may result in falling. Such a control system should basically perform three tasks: (1) realising body motion in accordance with one’s own will (in the sense of a servo system), (2) to keep balance despite external perturbations (like being pushed by others or by wind, in a homeostatic sense), and (3) to continuously optimise performance despite neural delays and sensor imperfections and characteristics varying over time (e.g., by ageing).

A simple servo system as sketched in Fig. 1, however, will not be able to comply with these three constraints. In a classical servo system, a controller (C) is driven by the difference between the desired state (sd) and the sensory (S) output (su), generating motor commands (m) driving bodily muscles (B) to realise the actual state of the body (u). Here the state of the body includes both motion and attitude parameters. External perturbations (ext) can thus also be cancelled. However, in order to work well, the sensors should be perfect, i.e., transducing exactly and continuously the variable(s) of interest, and the system should be free of delays.
Biological sensors, however, although often close to optimal performance, are not perfect. Even the slightest drift may accumulate to a large error which has to be dealt with by means of an additional mechanism. The semicircular canals, for example, do not respond to constant velocity angular motion due to hydrodynamical properties, which the CNS has been shown to compensate for by means of what is called the velocity storage mechanism [22].

Due to the neurochemical transduction in the retina and complex processing by the visual cortex at the other side of the brain, visual information may be available too late when running through a complex environment. Neural delays may cause the same in general, 100 ms or more being no exception. Moreover, inertial and gravitational accelerations as sensed by the otoliths and other graviceptors are indistinguishable according to Einstein’s equivalence principle [23], a fact if not reckoned by our CNS would imply a continuous struggle to move our bodies down to Earth to counteract the acceleration associated with gravity.

In 1964 Luenberger [24] proposed a way of dealing with such imperfections. Restated in terms of bodily motion control, his idea was that an optimal prediction of the actual state of the body (\( \mathbf{u} \)) in Fig. 2 can be made based on a copy of the motor commands and a set of interlinked copies of all essential transfer functions involved (indicated by primes in Fig. 2). In the perception literature the copy of the motor commands has also been termed an “efference copy” [25] or “corollary discharge” [26]. The copies of the transfer functions involved are acquired most likely during childhood and continuously updated during the further span of life. These copies all together are also referred to as an internal model or neural store, see also [1,27]. If external perturbations (\( \mathbf{ext} \)) are then present, the difference between the sensed and predicted afferents (\( \mathbf{c} = \mathbf{u} - \mathbf{u}' \)) is fed back to the internal model possibly attenuated or amplified by a variable gain (K) to finally make \( \mathbf{u} = \mathbf{u}' \), i.e., to get an optimal estimate of the actual state of the body. The advantages of including the internal model are that the ensuing control mechanism does indeed comply with the three boundary conditions mentioned above, including the compensation for neural delays and sensor imperfections, just because these are part of the internal model as they are in the primary sensory path.

In the model sketched in Fig. 2, actual posture will be determined by the actual state of the body, i.e., by \( \mathbf{u} \), and even without further external perturbations, \( \mathbf{u} \) will rarely be stable. Any system, including the CNS, will show random fluctuations or noise. The output \( \mathbf{u} \) will therefore also show some variability, which will induce a variability in \( \mathbf{u} \) via comparison with \( \mathbf{u}_0 \), which will also be largely affected by the feedback gain K. The other way round, K is assumed to be determined by the amount of noise in the sensory output \( \mathbf{u} \). If this uncertainty is high, K should be small so as to attenuate the amount of noise, however resulting in a sluggish control. If the uncertainty is small, K may be large so as to result in a fast and accurate control of body motion. The process of determining the optimal gain depending on the amount of noise present is in control theory referred to as Kalman filtering. The idea, furthermore, is that the error signal \( \mathbf{c} \) is also used as a signal for the CNS to update the internal model whence it lasts long enough. This accordingly accounts for variations in sensory dynamics over time due to, e.g., disease or ageing, thus explaining the observed phenomenon of “vestibular compensation”, see, e.g. [28].

The most interesting corollary at issue here comes in by the assumption posed by Oman in 1982 [27]. He proposed that the neural correlate of the conflict \( \mathbf{c} \) is the actual cause of several of the pre-nausea symptoms such as dizziness, head aches and is relayed neurally and/or hormonally to the gastro-intestinal system to finally result in nausea and emesis (MS-block in Fig. 2). This gastro-intestinal pathway also explains that postural instability will generally precede sickness, just because it adds an additional delay after the proposed mismatch. Oman’s system analytical corollary can thus be considered an explication of Reason and Brand’s conflict theory [1]. Based on a number of flaws regarding the rather generic conflict theory, Bles et al. [18] explicated this concept further by stating 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 (pp. 481–482). An essential part of this explication comes from the fact that our CNS should make a distinction between inertial and gravitational accelerations as sensed specifically by the otoliths. Because gravity is constant in an Earth fixed frame of reference, the CNS can make this distinction by low pass filtering of otolith afferents, conform [29], yielding the subjective vertical as referred to by Bles et al. [18]. A detail here concerns the fact that translation and attitude perception have been shown to be complementary [20,30]. Any theory explaining motion sickness by tilt therefore inherently explains it by translation or motion; see also [31]. In a series of previous papers we have extensively elaborated on this issue causing several forms of motion sickness [19,20,32], including visual–vestibular interactions resulting in visually induced motion sickness or cybersickness [21]. This concerned: angular and linear motion (when induced visually resulting in circular and linear vector), rod-and-frame effects, (pseudo) Coriolis and Purkinje effects, habituation, the idiotropic vector, and display time delays. The vast amount of phenomena all fitting within this framework therefore make it a likely candidate for describing and explaining the basic functions performed by our CNS regarding both postural stability and motion sickness. As a consequence it also makes sense that both postural stability and motion sickness are correlated, as has indeed been found in many studies referred to in the introduction, and it even seems making sense assuming that postural instability is a necessary and sufficient condition to produce motion sickness as postulated by Riccio and Stoffregen [2]. To this this adds the observation stated before by Bos and Bles [20], that it has never been reported that people do get sick while asleep as on, e.g., a moving ship. Moreover, because of the relatively slow gastro-intestinal processes at issue, it also makes sense that if postural instability and sickness are at issue, the former precedes the latter.

Most arguments and assumptions mentioned by Riccio and Stoffregen [2,33] leading them to state that postural instability is a necessary and sufficient condition to get sick, even more so add

![Fig. 1. Classical servo system (see Section 2 for further explanation).](image1)

![Fig. 2. Observer system (see Section 2 for further explanation).](image2)
to the validity of taking posture as the starting point for modelling motion sickness as outlined here. As a result, however, it can be explained that there are both conditions as observed in which: (1) humans (and animal) are unstable while not getting sick, and (2) motion sickness may occur when they are stable. Irrespective of the peculiarity of these conditions, they do nuance the firm statement on necessity and sufficiency.

3. Nuances

3.1. Incompatible data

Some of these observations have already been explicitly mentioned in the literature. The most obvious are those on negative correlations between postural instability and motion sickness, e.g., [34,35]. When using a number of combinations of posture (standing free and rigidly supported or laying down) and sensory conflict (natural images recorded and played back at different speeds), Warwick-Evans et al. [36] were able to make people sick, but did not find any effect of the factors studied, thus also not of posture. They therefore concluded that the sensory conflict theory provides the best explanation of the cause of motion sickness, and that their data did not support the claim that reducing postural instability reduces motion sickness. Interestingly these authors also mentioned that film speeds deviating from real not only increased sensory conflict but also became so novel that it became unrealistic, a phenomenon later referred to as “quarantining” by Golding et al. [37]. Furthermore, Flanagan et al. [38] explicitly tested the effects of sensory conflict, eye movements and postural stability on motion sickness severity. In a 2 x 2 x 2 design they did find a main effect with interactions of sensory conflict only. Thus, their lack of an effect of postural instability also contradicts the postural instability hypothesis.

3.2. Eyes

The fact that Flanagan et al. [38] took eye movements into account by itself favours the validity of the framework sketched in Fig. 2 and its consequences. Eye movements, including velocity storage, have been explained by an equal mechanism as shown above, e.g., [39,40], and some of its characteristics have been shown to be related with motion sickness, e.g., [41,42]. Although it has not been reported (yet) that, e.g., paraplegic patients without any postural control do suffer from motion sickness (which would further nuance the postural stability theory), the analogy has been reported that blind people do suffer from motion sickness [43].

3.3. (Re-)adaptation

Yet another point of concern has been mentioned by Kennedy and Stanney [17], referring to Gower et al. [44], who studied the effect of postural stability and sickness with repeated simulator trial exposures. Interestingly, they found a clear decrease of sickness symptoms over time but an increase of postural instability, contradicting the postural instability theory. Kennedy and Stanney [17] explained this lucidly by noting that both phenomena naturally follow from the adaptation process to the new virtual environment (VE) with the result of less sickness during and more postural instability due to re-adaptation after the VE exposure.

3.4. Organs of balance

To this comes the fact that people without functioning organs of balance do not get sick from motion, an issue assumed unresolved by Riccio and Stoffregen [2]. This has yet been observed already in totally deaf people in the 19th century aboard ships [45,46], and has been confirmed many times thereafter under different kinds of stimulation [1,47,48]. Interestingly, labyrinthine defective patients also do not suffer from visual motion, even in the absence of physical self-motion [49–51]. Moreover, labyrinthine defective subjects generally do show more postural instability than healthy subjects do [52,53]. When considering the model sketched in Fig. 2, these observations can be explained by taking into account the low pass filtering of otolith afferents mentioned above. Phase lags inherent to low pass filtering are then assumed to cause variations in the conflict thus leading to motion sickness. However, when otolith afferents are absent, as in labyrinthine defective patients, these filters are deprived from input thus minimising the gravity conflict, making these patients both “immune” to motion sickness and instable. Moreover, a calculation showed to predict a peak in sickness incidence induced by vertical motion at about 0.2 Hz, as has indeed been observed [19,20,54,55], adding to the validity of the subjective vertical mismatch theory and the model summarised here. Irrespective of this explanation, however, it can again be concluded that there are conditions in which postural stability and sickness are not correlated, thus also casting doubt on a causal relationship.

Instead of complete labyrinthine deficiency, malfunctioning organs of balance show rather different consequences. Ménière patients, for example, suffer from motion illusions, often accompanied by nausea and vomiting. Taxonomically, this could be categorised as autogenic or endogenous motion sickness because it originates from within the vestibular system, opposed to car sickness or cybersickness which therefore may be categorised as exogenous motion sickness. In this case the internal model will not be able to adapt to the variable condition of the hydrops, e.g., [56], thus leading to a conflict and hence to sickness, even when the system would be out of control of postural stability. The latter may be exemplified by the fact that Ménière patients also suffer from sickness at night when laying in bed. Hence, there are also conditions in which motion sickness is present without postural instability.

3.5. Psychological effects

Although anecdotal, the effect of distraction seems to have a positive effect on reducing sickness. This is known from survival stories at sea in life rafts and boats, and parents with susceptible children on the back seat of the car may confirm this. In both cases the simple act of singing together may already help, and the effect of performing a task per se can do the same [57,58]. Although not proven (yet), it seems unlikely that these distractions will first reduce postural instability, which, in turn will reduce the sickness. Whether the internal model feedback gain K will be affected by these psychological issues comparable to reducing K when the uncertainty about sensory afferents increases, or that another mechanism is at issue, remains, however, speculative.

3.6. Time

It has in addition been suggested that prolonged instability is required to get sick [2]. However, angular Coriolis effects are notorious for their brusque nature, possibly causing sickness right after the first head movement made, and this again fits well within the framework sketched here and elaborated by Bles [32]. In addition, it is known that astronauts may instantaneously get sick when floating into another compartment after leaving a duct, when the orientation of the new environment is opposite to what they expected (due to, e.g., a rotation inside the duct). Both examples seem to deny the necessity of prolonged instability, even when stability would be defined as being controlled by the hands
rather than the feet as required in space [2]. It does, however, add to the validity of taking “expectations” explicitly into account, as has been explicated above.

3.7. Expectations

Taking expectations into account, lastly, has been excluded as a possibility to explain motion sickness by Riccio and Stoffregen [2]. They argued that any theory based on such expectations is not scientifically falsifiable, just because these cannot be measured. However, I would argue that if we are aware of “expectations” in general, there must be neuronal correlates for that. With respect to sensory conflict in particular, or even more so with respect to an expected sense of verticality as assumed essential for the genesis of motion sickness here, such correlates must eventually be measurable by means of electrophysiological or whatever kind of imaging techniques. Hence, apart from the reasoning that the framework sketched here is a plausible mechanism applied by our CNS, there is also no reason to deny the existence of measurable neuronal correlates for any of the signals sketched in Fig. 2, thus making the theory scientifically falsifiable.

4. Conclusions

The above-mentioned list of examples and considerations may not and is not intended to be complete with respect to the relationship between postural instability and motion sickness. Although the latter are correlated in a large range of conditions, this paper indicates a number of critical points nuancing the relationship at issue and devaluing the suggested condition of necessity and sufficiency by Riccio and Stoffregen [2,33]. Moreover, both the conditions showing a correlation and the exceptions can be explained by assuming an observer or internal model calculating an optimal prediction of the state of the body, including the postural instability and the sickness. This observer mechanism thus substantiates Kennedy and Stanney’s [17] assumption that motion sickness and postural instability both are second order effects under control of a common centre.

Acknowledgement

This paper has been written as part of the Project V937 Improved Performance at Motion, carried out for the Dutch Ministry of Defence.

References


