
Usage Guidelines:
Please refer to usage guidelines at contact lib-eprints@bbk.ac.uk. or alternatively
Cybersickness: A multisensory integration perspective

Maria Gallagher and Elisa Raffaella Ferrè
Department of Psychology, Royal Holloway University of London, Egham, UK

Corresponding Author:

Elisa Raffaella Ferrè
Department of Psychology
Royal Holloway University of London
Egham, Surrey TW20 0EX, UK
Telephone: +44 (0) 1784 443530
Email: E.Ferre@rhul.ac.uk
Summary

In the past decade, there has been a rapid advance in Virtual Reality (VR) technology. Key to the user’s VR experience are multimodal interactions involving all senses. The human brain must integrate real-time vision, hearing, vestibular and proprioceptive inputs to produce the compelling and captivating feeling of immersion in a VR environment. A serious problem with VR is that users may develop symptoms similar to motion sickness, a malady called cybersickness. At present the underlying cause of cybersickness is not yet fully understood. Cybersickness may be due to a discrepancy between the sensory signals which provide information about the body’s orientation and motion: in many VR applications, optic flow elicits an illusory sensation of motion which tells users that they are moving in a certain direction with certain acceleration. However, since users are not actually moving, their proprioceptive and vestibular organs provide no cues of self-motion. These conflicting signals may lead to sensory discrepancies and eventually cybersickness. Here we review the current literature to develop a conceptual scheme for understanding the neural mechanisms of cybersickness. We discuss an approach to cybersickness based on sensory cue integration, focusing on the dynamic re-weighting of visual and vestibular signals for self-motion.

Keywords

Cybersickness, Virtual Reality, Motion Sickness, Vestibular System, Multisensory Integration
Introduction

Virtual Reality (VR) came to the public’s attention in the late 1980s. However, the real growth in popularity of VR has been observed in more recent years, when the development in technology allowed a user to be immersed in a completely virtual world through the use of 3D real-time computer graphics and advanced display devices. A report by Goldman Sachs in January 2016 predicted that revenue from VR would approach $80 billion by 2025, with 315 million users (Bellini et al. 2016). Pivotal to the user’s VR experience are multimodal interactions involving all senses. The human brain must integrate real-time vision, hearing, vestibular, and proprioceptive inputs to produce a compelling and captivating feeling of immersion and presence in a VR environment akin to real life scenarios (Azmandian et al. 2016; Herbelin et al. 2015). VR has proved to be beneficial in several applications ranging from educational and training platforms, to recreational gaming or media viewing, as well as flight simulators and medical rehabilitation (Alaker et al. 2016; Pelargos et al. 2016; Valmaggia et al. 2016; Viñas-Diz and Sobrido-Prieto 2016).

A troublesome problem with VR is that between 20% and 80% of users exhibit symptoms that parallel symptoms of classical motion sickness (Cobb et al. 1999; Munafo et al. 2017). This so-called cybersickness can be profoundly unsettling, and may compromise well-being and performance in VR training (Cobb et al. 1999; Sharples et al. 2008; Fiore et al. 2013; Llorach et al. 2014; Munafo et al. 2017). Symptoms of cybersickness include discomfort, apathy, nausea, drowsiness, disorientation, eyestrain and fatigue (Stanney et al. 1997; Rebenitsch and Owen 2016). Although its causes are not yet entirely clear, cybersickness occurrence might be dependent on different factors, including the user’s gender (Munafo et al. 2017), age (Arns and Cerney 2005), duration of VR exposure (Liu 2014), and hardware issues,
such as lag (i.e., delays in updating the visual scene following a user’s movement or action) or
flicker (i.e., flashing of the visual scene relating to refresh rates) (Moss et al. 2011).

In the past decade, there has been a rapid advance in VR to increase the simulation
realism by means of integrated multimodal experiences (i.e. 3D audio, haptic and force
feedback, etc.). Significant technical improvements have also occurred, including better
positional tracking, display resolution, and display refresh rate (Harsora et al. 2017). For
example, many Head Mounted Displays (HMDs) (i.e., Oculus Rift CV1 and HTC Vive)
are capable of tracking the position of the head in space via numerous sensors and cameras, have a
refresh rate of 90Hz, and visual resolution of 1080x1200 resolution for each eye. The
improvement in VR technology is therefore self-evident, and even more if compared to earlier
versions of commercial HMDs (i.e., no positional tracking capabilities, a 60Hz refresh rate,
and resolution of 640x800 for each eye) (Harsora et al. 2017). Moreover, the earliest HMDs
had much more limited technical capabilities: 30 frames per second, a 40 degree field of
view, and rather cumbersome head position sensors (Sutherland 1968). Further
improvements are likely in the coming years, such as foveated rendering and adaptive displays
which increase the resolution and refresh rate of the display at selected locations of the VR
scene (Padmanaban et al. 2017; Lee et al. 2017; Hsu et al. 2017), increasing realism and
immersion in VR (see Bastug et al. 2017 for a review). Despite these advances, there are still
some issues in VR and as one leading iconoclast in VR technology - Palmer Luckey - has
indicated, “VR isn’t perfect right now” (Kushner 2016). Cybersickness remains a barrier to VR
use: it has even been argued that the more realistic the VR environment, the more likely it is
that the user experiences cybersickness (Merhi et al. 2007; Davis et al. 2015). Although the
exact reason behind this is still unclear, better resolution of optic flow might potentially
exacerbate visual-vestibular-proprioceptive conflicts (Kennedy et al., 2003; Merhi et al.,
2007; Davis, Nesbitt and Nalivaiko, 2015). Alternatively, one can hypothesise that the
increased feeling of presence (i.e. the feeling of “being there” in a VR environment) due to the availability of detailed simulated environments might exacerbate cybersickness symptoms (Kennedy et al., 2003; Merhi et al. 2007; Davis et al. 2015). In such cases, it is possible that the detailed, realistic environment and increased presence may render the conflict in sensory signals more dramatic, increasing cybersickness. Further research is necessary to explore the exact relationship between increased feeling of presence and cybersickness.

The root causes of cybersickness remain poorly understood, however more general accounts of motion sickness have been adapted as explanations given the similarity between cybersickness and other types of motion sickness. These theories include Reason and Brand’s (1975) Neural Mismatch Theory, based on sensory conflict, and Riccio and Stoffregen's (1991) Postural Instability Theory based on movement control. However, neither theory can fully explain why cybersickness arises, nor can they predict an individual’s likelihood of developing symptoms. Despite these theories highlighting the role of multiple sensory inputs in the cause of cybersickness, more recent advances in knowledge of multisensory integration have only recently applied to this field (Balter et al. 2004; Oman 2012; Jürgens et al. 2016; Weech and Troje 2017).

Here we review the current literature to provide an overview of cybersickness and perceptual after-effects induced by VR, as well as evaluating the current theories of cybersickness. We highlight how dynamic re-weighting of sensory cues can be implicated in cybersickness development and after-effects of VR exposure. This approach may provide insight into cybersickness, facilitating further research and leading to developments which may enhance the VR experience.
The vestibular system and sensory integration for self-motion perception

In everyday life, our perception of self-motion depends on the coherent integration of visual, proprioceptive and vestibular information. When we move in the surrounding environment, the visual system provides retinal-image motion (optic flow) cues, the proprioceptive receptors in the muscles, tendons, and joints sense the relative position of body parts in space and the vestibular system encodes angular and linear acceleration.

Since Gibson in 1950, motion related retinal-images have been considered essential for eliciting sensations of self-motion displacement (Gibson, 1950). However, it is now recognised that extra-visual cues make an equal contribution to self-motion perception. In the absence of visual cues, humans rely on vestibular information to estimate bodily motion (Israël and Berthoz 1989; Israël et al. 1993; Berthoz et al. 1995). The vestibular system is a set of sensory organs located in the inner ear, comprising of three orthogonal semicircular canals (anterior, posterior and horizontal) that sense rotational acceleration of the head in three-dimensional space and around three cardinal axes (yaw, roll, pitch), and two otolith organs (utricle and saccule) that code translational acceleration, including the orientation of the head relative to gravity. Vestibular organs are extremely sensitive to even the slightest changes in rotation and linear movement of the head, providing the feedback necessary for the brain to dictate adjustments that allow the body to maintain balance. Dynamic vestibular inputs from the semicircular canals are associated with low-level visuo-vestibular interactions to control gaze and eye fixation, while gravitational inputs from the otolith organs contribute to path integration and navigation. Thus, vestibular cues are fundamental for perception of self-motion, aiding us in distinguishing self from object motion and providing us with a sense of where we are in space (Green and Angelaki 2010; Greenlee et al. 2016). Thus, it might not be surprising that the vestibular system plays a vital contribution in the development of motion.
sickness, including cybersickness. Accordingly, labyrinthine-defective patients do not experience any motion sickness symptoms (Cheung et al. 1991; Paillard et al. 2013) while blind individuals do, suggesting that visual information, while implicated motion sickness, is not as crucial as vestibular signalling (Greybeil 1970).

Multimodal interactions between visual, somatosensory, proprioceptive and vestibular signals have been described in almost all vestibular relays, including the vestibular nuclei, the thalamus and several areas in the cerebral cortex (Lopez et al. 2012; Zu Eulenburg et al. 2012). Electrophysiological studies have identified a widespread vestibular network in which the core area is the Parieto Insular Vestibular Cortex (PIVC) (Guldin and Grüsser 1998; Chen et al. 2010). This area consists of the posterior insula/retoinsular cortex in the bank of the lateral sulcus (Guldin and Grüsser 1998). The human homologue of the primate PIVC is a distributed set of regions, including retoinsular cortex, temporoparietal junction and somatosensory cortices (Fasold et al. 2002; Lopez and Blanke 2011; Zu Eulenburg et al. 2012). Neuroimaging studies with artificial vestibular stimulation showed activation in large swathes of the cortex, including activations of classically unimodal sensory and motor regions, supporting function integration between signals and inputs from other senses (Ferrè and Haggard 2015).

Multisensory interactions between sensory modalities are fundamental in shaping our perceptual experiences. Sensory signals presented simultaneously in more than one sensory channel tend to be detected more accurately than the same signals presented individually (Stein et al. 1996). Most studies of vestibular-multisensory interactions have focused on multisensory convergence between vestibular and visual signals. Multisensory convergence involves multisensory integration of different signals related to a common external source object or percept (Ernst and Bülthoff 2004). Critically, the neural mechanism underlying multisensory convergence is likely to be a process seeking to reduce perceptual uncertainty about the source
(Knill and Pouget 2004), which often involves optimal combination of cues across modalities (Ernst and Banks 2002).

The predominant theme in recent electrophysiological work has been the convergence between vestibular signals and visual signals for perception of self-motion, spatial orientation, and navigation in the environment. Multisensory neurons coding for visual and vestibular signals have been described in the macaque ventral intraparietal area (VIP, Bremmer et al. 2002), which is considered homologous to human vestibular areas in the posterior parietal cortex (Lopez and Blanke 2011). Visuo-vestibular interactions are often interpreted within the framework of optimal cue combination for multisensory perception of a single underlying quality (Gu et al. 2008; Fetsch et al. 2009). For instance, heading direction is accurately perceived in visuo-vestibular multimodal conditions. Macaques trained to complete a heading discrimination task where cues were provided by an optic flow (vision), or by a motion platform (vestibular), or combined (visuo-vestibular), showed smaller thresholds for detecting head direction under the combined sensory condition than either of the unimodal visual or vestibular conditions (Gu et al. 2008). Importantly, these results were mirrored by the activity of dorsal medial superior temporal (MSTd) neurons: the neurons’ preferred heading direction was similar for both visual and vestibular modalities, suggesting a neural mechanism for perceptual integration. Fetsch et al. (2009) further investigated this integration by dynamically modifying the reliability of the visual cue by reducing optic flow coherence and placing the visual and vestibular cues in conflict. The results matched the predictions of optimal cue integration; as the coherence of the visual cue decreased and became less reliable, the weighting of the vestibular cue increased. Interestingly, the conflict between the visual and vestibular cues did not prevent sensory integration. Similar results were found in human participants. Thus, visual and vestibular signals might be combined optimally when estimating heading direction – and therefore self-motion (however see de Winkel et al. 2010; de Winkel et al. 2013).
for contrary findings). The activity of neurons in MSTd is a likely neural mechanism for this effect (Angelaki et al. 2011).

What is cybersickness?

Cybersickness is an unpleasant sensation, comprising of symptoms of disorientation, drowsiness, eyestrain, and nausea arising from exposure to immersive VR environments. Cybersickness is triggered by visually-induced illusory motion within an immersive VR environment, in which an optic flow provides motion information in the absence of corresponding vestibular signals (Reason and Brand 1975; Hill and Howarth, 2000; Keshavarz et al. 2015; Rebenitsch and Owen 2016). For this reason, cybersickness is slightly different compared to traditional motion sickness syndromes, such as car-sickness, sea-sickness and airsickness, in which the physical movement of the vehicle triggers motion sickness symptoms (Reason and Brand, 1975; Golding 2016). However, given its similarity to motion sickness, cybersickness can be regarded as a type of visually-induced motion sickness.

Many VR applications employ an optic flow pattern which elicits an illusory feeling of self-motion, namely vection\(^1\). In a VR driving simulator, for instance, the simulation provides accurate optic flow patterns of the road, buildings and other parts of the environment, eliciting clear vection sensations. The visual signals tell the user that they are moving in a certain direction with a certain acceleration. However, since the user is not actually moving, the vestibular organs provide no cues for linear or angular acceleration. As visual signals for self-motion are not corroborated by inertial forces transmitted through the vestibular system, a

---

\(^1\) Although vection is a complex perceptual experience (see Palmisano et al. 2015 for a review), for the purposes of this review vection is defined as a sensation of self-motion arising from visual cues in a VR environment.
sensory conflict is likely to occur, and subsequently lead to cybersickness (Keshavarz et al. 2015).

The example above refers to the scenario in which the user does not move their head during VR exposure. However, many VR HMDs are now supplied with positional trackers, which enable the user to physically move in the real world while exploring the VR environment (Harsora et al. 2017). When physically moving in VR, visual cues are supported by vestibular information. This may drastically reduce the conflict between sensory modalities, and may prevent the occurrence of cybersickness. At present, few empirical studies have compared levels of cybersickness between locomotion techniques, and findings are somewhat mixed (Chance et al. 1998; Zanbaka et al. 2004; Peck et al. 2011; Llorach et al. 2014). For example, Llorach et al. (2014) compared navigation in a VR environment via a game controller, in which the user remained stationary and moved via controlling joysticks, and a position estimation system which tracked the users’ actual movements. Levels of cybersickness were much higher when the user did not move and used the game controller compared to when they navigated by physically moving. By contrast, Peck et al. (2011) found no differences in cybersickness symptoms between VR scenarios in which users freely moved, walked in place, or explored the VR environment using a joystick, despite finding better performance in the free movement scenario. Although new developments in positional trackers also allow users to explore the VR environment through head movements while sitting, vection appears much stronger when users move their heads, and possibly contributing to cybersickness (Ash et al. 2011). In addition, it is not possible to exclude that incorrect updating of the visual scene during active head motion might elicit cybersickness (Ash and Palmisano 2012). For example, cybersickness increases when the visual VR scene moves in the same direction of the physical head movement (Palmisano et al. 2017). Similar conflicts between visual and vestibular signals are implicated in car-, sea-, and space-sickness (Lackner 2014).
Traditionally, the presence of cybersickness has been explored via subjective self-reports. The Simulator Sickness Questionnaire (SSQ; Kennedy et al. 1993) is the most frequently used. This questionnaire breaks down motion sickness symptoms into three main categories: disorientation (D), including symptoms such as dizziness, vertigo and difficulty focusing, oculomotor (O), including eyestrain, headache, and blurred vision, and nausea (N), including stomach awareness, increased salivation, as well as nausea itself (Table 1). Cybersickness is characterised by severe and frequent disorientation symptoms, followed by nausea symptoms, and least oculomotor symptoms (a so-called D>N>O profile; Stanney et al. 1997; Rebenitsch and Owen 2016). This symptom profile further distinguishes cybersickness from other types of motion sickness. For example, simulator sickness has an O>N>D profile, sea-sickness an N>O>D profile, and space sickness an N>D>O profile (Stanney et al. 1997; Rebenitsch and Owen, 2016). Moreover, symptoms of cybersickness are reportedly much more severe than simulator sickness and other motion sickness symptoms (Kennedy et al. 2003).

*** PLEASE INSERT TABLE 1 HERE ***

Along with self-reported symptoms of cybersickness, a range of physiological changes have also been described, including increases in tachygastric power, heart rate and eyeblinks, decreases in bradygastric power, and changes in skin temperature and EEG power bands (Kim et al. 2005; Nalivaiko et al. 2015) (Table 1). Kim et al. (2001) identified increases in eyeblinks, skin conductance response, and heart rate, as well as decreases in the amplitude of photoplethysmogram associated with cybersickness. In addition, a selective modulation of the EEG gamma band activity was noted (Kim et al. 2001). Critically, physiological changes can predict cybersickness severity. For instance, bradygastric power, breathing rate, pulse amplitude and blinking rate can predict total or subscale scores of the SSQ (Dennison et al.
Although physiological changes could represent an objective measure of cybersickness, they are not widely employed at present. Exploration of these changes may prove useful in determining the likelihood of cybersickness severity in individual users, therefore allowing intervention before cybersickness develops. An overview of cybersickness symptoms and physiological changes can be seen in Table 1.

The severity of cybersickness symptoms seems to be proportional to the duration of VR exposure: increasing exposure time within a single VR session is likely to increase cybersickness symptoms (Stanney et al. 2002; Moss et al. 2011). Liu (2014) found that participants completing a VR task had more severe symptoms as the duration increased from 5 to 15 minutes. Interestingly, a 20-minute session produced less severe symptoms than the 15-minute session, which might be due to adaptation to the VR environment (see below).

It is also possible that cybersickness symptoms do not recede immediately after cessation of VR, but rather linger on for some time following exposure. Stanney and Kennedy (1998) found cybersickness symptoms present at least one hour post-exposure. In accordance with the general profile of cybersickness, disorientation symptoms were most severe, followed by nausea and oculomotor symptoms. Startlingly, disorientation symptoms were almost 150 times higher immediately following VR exposure, and remained 95 times higher than the pre-exposure level one hour following exposure.

Although the profile of symptoms in cybersickness is relatively consistent, several factors influence whether an individual will develop the syndrome during VR exposure. Not surprisingly, people who have a history of motion sickness susceptibility are more likely to experience cybersickness, and are less likely to enjoy using the technology (Nichols 2000; Rebenitsch and Owen 2014). Age and gender also seem to be important: Arns and Cerney (2005) found that symptom severity and incidence increased with age, and a number of studies report that females are more susceptible to cybersickness than males (Stanney et al. 1999;
Stanney et al. 2003; Flanagan et al. 2005; Kim et al. 2008; Chen et al. 2015). The reason for this gender difference is somewhat unclear, and might be driven by the influence of the menstrual cycle (Clemes and Howarth 2005; however, Golding et al. 2005 argue that the influence of the menstrual cycle is too small to fully account for the gender difference), differences in postural stability between men and women (Koslucher et al. 2016), or a larger field of view in women (LaViola 2000).

**Current interpretations of cybersickness**

While an understanding of cybersickness and its influences is growing, uncovering precise neural mechanisms behind cybersickness is less straightforward. A specific framework for cybersickness has not been widely employed at present. Instead, general theories of motion sickness have been adopted to explain cybersickness (Table 2).

*** PLEASE INSERT TABLE 2 HERE ***

Reason and Brand (1975) suggested that a discrepancy between sensory modalities is the root cause of motion sickness syndromes. Two main forms of sensory conflict have been identified in motion sickness: intersensory conflicts between visual and vestibular signals, and intrasensory conflicts between the semicircular canals and otoliths within the vestibular system (Reason 1978). Thus, the vestibular system seems to be critical in causing motion sickness, as supported by evidence in peripheral vestibular patients who do not experience any form of motion sickness (Cheung et al. 1991; Paillard et al. 2013).

According to Reason and Brand’s (1975) Neural Mismatch Theory, a copy of a self-generated movement is paired with the resulting sensory inputs to form a predicted pattern of
sensory cues, i.e. an engram (Reason, 1978). Then, a comparator module matches the actual sensory inputs with the stored engrams. If the input and engram do not match, a discrepancy arises and a mismatch signal is generated, triggering motion sickness. The strength of this mismatch signal is dependent on how many sensory modalities are in conflict, the extent of the discrepancy and the amount of previous exposure to the conflicting stimuli. Accordingly, the strength of the mismatch signal corresponds to the latency and severity of motion sickness symptoms. When VR users are immersed in applications where they perceive self-motion through vection, visual signals suggesting movement conflict with vestibular inputs signalling the user is stationary. A mismatch signal is then generated if no matching engram is found, triggering cybersickness (Reason and Brand 1975; Reason 1978).

Reason (1978) proposed a further classification of sensory conflicts which have the potential to trigger cybersickness. First, information signalled by visual and vestibular systems is contradictory. For example, this may be the case when HMDs are improperly calibrated, showing VR movements (visual cues) that are not properly aligned with the user’s head movements (vestibular cues). Second, visual information is not corroborated by expected signals from the vestibular organs. This sensory conflict is unsurprisingly common in cybersickness, when vection is not supported by vestibular information. Finally, vestibular information is not corroborated by visual signals, as experienced in the use of HMDs without head-tracking in which changes in head position may not be verified by changes in the VR scene.

Although Reason and Brand’s (1975) theory is widely accepted, it cannot fully account for motion sickness, and therefore cybersickness, onset and development. First, this framework lacks a clear physiological basis which would explain the importance of mismatch signals in facilitating sickness (Oman 1988). Second, Reason and Brand’s (1975) theory cannot account for individual differences in motion sickness (Warwick-Evans et al. 1995; Davis et al. 2014).
For example, it is unclear why females should be more susceptible to motion sickness than males (Stanney et al. 1999; Stanney et al. 2003; Flanagan et al. 2005; Kim et al. 2008; Chen et al. 2015). Finally, the theory is unable to explain why some sensory cues are more likely to cause sickness than others. According to Neural Mismatch Theory, any sensory conflict triggering a mismatch signal should cause sickness, so it is unclear why particular stimuli are more nauseogenic than others. For example, scene oscillations within a VR environment are more likely to cause cybersickness than scenes with no oscillation (So and Lo, 1999; Lo and So, 2001). In particular, oscillations of around 0.2Hz in real motion sickness are highly nauseogenic, with oscillation along the fore-aft axis the most likely to cause sickness (Kennedy et al. 2010).

To address some of these open questions, Oman (1988) proposed that a desired body state prompts muscle activity and postural changes to reach that state. These changes provide signals which, along with external noise, are detected by different sensory modalities. An internal model based on all sensory modalities is formed, which is compared with actual sensory signals, providing a difference vector. Accordingly, greater sensory conflicts lead to a larger vector, which may reflect severe sickness.

Building upon the theory of Oman (1988), Bles et al. (1998) and Bos et al. (2008) proposed a more nuanced description of sensory conflict based on perception of the subjective vertical. The subjective vertical is formed from integrated sensory information from vision, proprioception, and the vestibular organs and is necessary for successful interactions with the external world (Barra et al. 2010). The visual and vestibular senses construct a model of the expected subjective vertical, as well as sensing the actual subjective vertical. The comparison between the sensed and expected verticals leads to a difference vector, prompting motion sickness. Motion sickness may therefore arise when there is an unexpected change in the subjective vertical, causing a conflict between the sensed and expected verticals. In the case of
cybersickness, the VR environment may contain aspects where the visual and vestibular vertical are at odds, however further research is necessary to unpack aspects of this theory of motion sickness.

While the underlying basis for sensory conflict-based models remains debated, empirical evidence for the involvement of sensory conflicts in motion sickness induced by physical movement (i.e., sea-sickness, car-sickness) highlights their contribution to the development of sickness symptoms (Kato and Kitazaki 2008; Wang and Lewis 2016; Wada et al. 2016; Wada and Yoshida 2016). The specific evidence for visuo-vestibular conflicts in cybersickness is growing, suggesting that the strength of the sensory conflict between visual and vestibular cues can lead to increased sickness (Bonato et al. 2009; Nishiike et al. 2013).

For example, Akiduki et al. (2003) induced a visuo-vestibular conflict while participants were immersed in VR. Participants were instructed to follow a virtual ball around the room, allowing for a range of movements. The visuo-vestibular conflict was induced by doubling the range of movement of the VR environment background relative to the participants’ head movements. Cybersickness symptoms were significantly greater during and immediately after VR exposure. In addition, more complex patterns of visual motion are also related to cybersickness. Keshavarz and Hecht (2011) found that rotation across two or three axes induced increased levels of sickness than a single axis of rotation. Thus, greater mismatches between visual and vestibular modalities might trigger symptoms of cybersickness, as purported by Reason and Brand (1975) and Oman (1988).

According to the above theories, sensory conflicts are the cause of motion sickness, however it is unclear how and why such conflicts would cause symptoms such as nausea. One well-cited hypothesis is that of Poison Theory (Treisman 1977). According to this hypothesis, sensory mismatches are part of an early warning system when an animal has ingested toxins. Nausea is therefore an adaptive, evolved response to sensory conflict aimed at ridding the
animal of dangerous toxins (Treisman 1977). While this hypothesis is a plausible explanation for nausea symptoms of motion sickness, it does not account for other symptoms, such as oculomotor or disorientation symptoms, and many authors argue that it is not a compelling explanation for motion sickness (LaViola 2000; Oman 2012; Davis et al. 2015).

Riccio and Stoffregen (1991) proposed that motion sickness is a result of prolonged postural instability: people are likely to suffer motion sickness when experiencing novel situations for which they have not yet learned strategies to stabilise their posture (Stoffregen et al. 2000; Villard et al. 2008). For example, Stoffregen and Smart (1998) found increases in postural sway preceding symptoms of visually induced motion sickness when participants were exposed to low-amplitude optical flow in an immersive environment. Moreover, Smart et al. (2002) found that pitch velocity and vertical variability could predict which participants would become sick when exposed to optic flow stimulation. However, the causal relation between postural instability and cybersickness is not yet clear. For instance, Dennison and D’Zmura (2017) found that postural sway was similar both before and during VR exposure, and cybersickness increased both when participants were seated (and therefore unlikely to have an unstable posture) and when they were standing (and thus subject to greater postural demands and the potential for instability). Similarly, Warwick-Evans et al. (1995) found that motion sickness was equally present when participants viewed a video while standing and while restrained in a chair. Finally, Akiduki et al. (2003) demonstrated that postural instability (in particular, body sway) was only significantly different post-exposure to VR, pointing to instability as a consequence, rather than a cause, of cybersickness.

Is it possible to prevent cybersickness?
Techniques for preventing cybersickness include adaptation to VR through repeated exposure (Barrett 2004; Keshavarz 2013), designing VR environments to include stable visual references of the horizon or perceptual vertical (Han et al. 2011), developing applications based on physical locomotion (Llorach et al. 2014), and providing concurrent vestibular signals by means of galvanic vestibular stimulation (Cevette et al. 2012).

Adaptation to VR is arguably a more readily available technique to prevent cybersickness, as it requires the user to repeatedly engage with VR, rather than modification of VR applications or use of sensory substitution equipment (Keshavarz 2013; Golding 2016).

Several studies have shown reduced cybersickness following adaptation to VR: participants exposed several times to the same VR scenario showed decreased cybersickness symptoms (Regan 1995). Similarly, Hill and Howarth (2000) asked participants to complete five sessions across five days in which they played a racing game for 20 minutes via a HMD. Some participants also passively viewed the scene while the experimenter played the game, increasing their exposure to VR. Participants experienced at least a mild degree of malaise during the first session, however by the end of the fifth session seven out of 11 participants who played and watched the game reported no cybersickness symptoms, suggesting faster adaptation to VR. These findings are in accordance with the mentioned theories of motion sickness. For example, Reason and Brand’s (1975) Neural Mismatch theory predicts that as participants are further exposed to conflicting sensory stimuli, their neural store creates a new engram, and in subsequent exposures a mismatch signal is not generated to trigger cybersickness. Similarly, an internal model of expected sensory signals within the VR environment may be updated as participants are further exposed to the conflicting stimuli, as predicted by Oman (1988) and Bles et al. (1998). By contrast, Riccio and Stoffregen’s (1991) postural instability hypothesis predicts that individuals adapt to motion sickness stimuli when they learn new strategies to control their posture in the provocative environment. While adaptation to a cybersickness-
inducing environment is possible, it is yet unclear which of these motion sickness theories best describes the process by which adaptation might occur.

Although the beneficial effects of VR adaptation on cybersickness symptoms are promising (Howarth and Hodder 2008; Moss et al. 2011), several drawbacks are apparent. First, repeated exposures are necessary for adaptation to be effective, which implies significant commitment from VR users. Second, it is not yet clear how durable the benefits of adaptation may be, limiting its utility. Finally, an inverse relation exists between reduction of cybersickness through adaptation and the development of VR after-effects. These include altered visual perceptions and balance problems (Stanney and Kennedy 1998; Stanney et al. 1999; Di Girolamo and Pic 2001; Harm et al. 2008). Thus, the more habituated an individual becomes to the virtual world, the more likely they are maladapted to the real world on VR cessation, incurring a range of after-effects (Wright 2014).

Sensory conflict appears the most likely explanation of cybersickness. Thus, a reduction of these conflicts might prevent cybersickness. In VR the conflicting information between vestibular and visual cues prevents the user from being able to accurately assess self-motion. The presentation of ‘rest frames’ has been proposed as a method for reducing cybersickness (LaViola 2000; Han et al. 2011). Rest frames are an explicit frame of reference for spatial information concerning stationary objects, providing information on which to base self-motion priors. For instance, cybersickness scores were reduced when users were exposed to a VR rollercoaster scenario with a superimposed grid compared to a standard VR scene (Chang et al. 2013), as Bles et al.’s (1998) subjective vertical conflict hypothesis would predict: by providing a clear frame of reference for the visual vertical in VR, conflicts between predicted and experienced subjective verticals are minimised.

Recent VR scenarios allow users to physically move in the VR environment, reducing the conflict between visual and vestibular systems (Llorach et al. 2014). Despite the possibility
of cybersickness reduction through physical locomotion, limitations of physical space, particularly for home users of VR, may mean that users prefer to remain stationary and navigate the virtual environment by other means (for example, controllers) (Williams et al. 2007; Riecke et al. 2010). In addition, errors in position tracking and lag, while significantly improved in more recent HMDs, may also contribute to cybersickness development (Fiore et al. 2013; Kinsella et al. 2016; Palmisano et al. 2017).

Sensory conflicts can also be reduced by matching visual cues with artificial vestibular signals. Cevette et al. (2012) applied artificial vestibular stimulation (Galvanic Vestibular Stimulation) while participants used a flight simulator. Galvanic Vestibular Stimulation induced illusory sensations of self-motion which were purported to match the visual signals experienced by the participants. Since the conflict between visual and vestibular signals was reduced, a significant reduction in sickness symptoms was found. Similarly, Galvanic Vestibular Stimulation applied during turns in a driving simulator was suggested to reduce scores in motion sickness questionnaires and improve performance (Reed-Jones et al. 2007). In addition, Galvez-Garcia et al. (2015) found that applying Galvanic Cutaneous Stimulation either continuously or intermittently while participants used a driving simulator reduced sickness scores relative to a condition with no stimulation. These results suggest that the use of artificial stimulation may be a potential method for preventing cybersickness in VR.

VR after-effects: A re-adaptation to the real world

After-effects can arise following exposure to a variety of different sensory stimuli. One interesting and well-studied after-effect following exposure to passive motion, such as on a sea voyage, is so-called mal de debarquement. This syndrome induces illusory feelings of self-motion, such as bobbing or swaying lasting for days or even years (Van Ombergen et al. 2016).
Although less severe, after-effects can frequently develop in the hours and days following a prolonged VR experience (Kellog et al. 1980; Gower and Fowlkes 1989). In one of the most bizarre cases, a pilot had his view of the world invert 180 degrees while driving a car hours after having been in a VR flight simulator (Kennedy et al. 1987). As a result of these after-effects, many air force bases have mandatory polices which stipulate that pilots cannot fly an aircraft up to 24 hours after exposure to a VR simulator. Also, many VR entertainment centres require that users do not drive for several minutes after exposure. It is important to note that some VR applications are designed specifically to carry over sensorimotor or behavioural changes following exposure to the stimulus, for example for rehabilitation or training purposes (Cameirao et al. 2011; Verschure 2011; Cameirao et al. 2012; Alaker et al. 2016; Pelargos et al. 2016; Valmaggia et al. 2016; Viñas-Diz and Sobrido-Prieto 2016). However, for the purposes of this review we focus only on maladaptive after-effects.

After-effects might be induced by adaptation to conflicting sensory stimuli. One of the most well-known examples is adaptation to prism lenses (Redding et al. 2005). On first wearing the lenses, which displace the visual field, participants make frequent errors in pointing and grasping. However, participants quickly adapt to the lenses and their accuracy increases. On removal of the glasses, however, participants begin to make errors and a period of re-adaptation is necessary before performance returns to normal (Clower et al. 1996). Adaptation to VR may follow a similar pattern, however a thorough exploration of after-effects of VR exposure has not yet been conducted.

Harm et al. (2008) found that proprioceptive coordination between the eyes, head and hands was worse following 20 minutes of VR exposure. Critically, the performance was not only immediately worse, but approached recovery only by 6 hours post-exposure. Similarly, participants showed increased pointing errors immediately after 30 minutes of VR exposure (Stanney et al. 1999).
A multisensory integration perspective for cybersickness

The evidence reviewed above suggests a pervasive influence of multisensory interaction in VR experience. In this section, we outline a conceptual scheme of how these could underlie cybersickness. Sensory inputs constantly reach the human brain. However, these signals need to be integrated to provide successful descriptions of the environment. Vestibular inputs make an essential contribution in this process, assessing whether visual signals are consistent or not with the movement and position of our head in space. Critically, when self-motion signals provided by the vestibular system cannot be aligned with those from visual cues, multisensory conflict occurs, potentially triggering cybersickness.

Under normal conditions, the visual and vestibular systems interact to provide information about self-motion. Optimal multisensory integration involves higher weighting of more reliable signals (Ernst and Banks, 2002). Thus, sensory cues are weighted according to their reliability, such that vestibular cues are given higher weighting when visual cues are unreliable and vice versa. A growing body of research has shown that visuo-vestibular integration is near-optimal for self-motion, with an over weighting of the vestibular cue relative to “true” optimality. Several results suggest that a process based on dynamic sensory reweighting may be important to explain cybersickness and adaptation to virtual environments (Butler et al. 2010; de Winkel et al. 2010; Fetsch et al. 2010; Angelaki et al. 2011). Three results are particularly relevant here. First, the contribution of optic flow to
perceived self-motion typically emerges only after several seconds of exposure to visual cues when inertial cues are not present, implying that a dynamic process of sensory re-weighting is necessary to resolve sensory conflicts before the perception of self-motion emerges (Young et al. 1973). Second, when vestibular and visual signals are conflicting, the optimal combination of cues cannot occur without a re-weighting of the original cues. In the real world, the vestibular cue is overweighted relative to optimality (Kaliuzhna et al. 2016, but see de Winkel et al. 2010). This implies that the sensory conflict experienced in virtual reality must be resolved by substantial down-weighting of vestibular cues and up-weighting of visual cues. Third, the results on visual or vestibular dominance in such conflicting conditions appear to vary across studies, suggesting that the human brain dynamically chooses which sensory cues are relevant for a particular situation (Young et al. 1973; Zacharias and Young 1981; Probst et al. 1985). However, taken together these results suggest that dynamic reweighting may be implicated in cybersickness.

Cybersickness may be due to the consequences of a conflict between the sensory signals which provide information about the body’s orientation and motion. Visual signals tell users that they are moving in the environment. Since users are not actually moving, their vestibular architecture provides no corresponding cues of linear and angular acceleration. In view of this, when the perception of visual self-motion is not supported by inertial forces transmitted through the vestibular organs, a visuo-vestibular conflict is likely to occur, leading to cybersickness. Alteration of the weight of vestibular cues provides a means of resolving the visuo-vestibular conflict, reducing symptoms of cybersickness.

Multisensory theories suggest that multiple sensory signals need to be combined, and that the nervous system faces a key challenge in selecting the correct weighting for each signal in the combination. As in other cases of sensory conflict, resolution occurs by weighting each individual sensory signal according to its importance. In VR, the brain tends to habituate to
extract self-motion information from visual cues in a visuo-vestibular conflicting environment.

Since vestibular information is usually highly reliable in determining the body’s position and motion in space (Prsa et al. 2012; Kaliuzhna et al. 2016), the vestibular signals must be down-weighted to avoid the occurrence of sensory conflicts. We suggest that a dynamic re-weighting function is an important element of cybersickness which has not yet been extensively researched. In VR, the dynamic re-weighting function increases the weight of visual signals about motion, and decreases the weight of vestibular information responsible for self-motion. As a result, the visuo-vestibular conflict is decreased and therefore no longer perceived, reducing cybersickness. Cybersickness symptoms typically develop within the first minutes of VR exposure (Stanney and Kennedy 1998; Davis et al. 2015). Thus, the re-weighting function must rapidly respond to the visuo-vestibular conflict to prevent the occurrence of symptoms. If the re-weighting function is slow to respond to the conflict, cybersickness may ensue. Interestingly, decreasing the reliability of vestibular cues by applying artificial noisy Galvanic Vestibular Stimulation has been shown to modulate vection perception (Weech and Troje 2017). This might be due to rapid re-weighting of visual and vestibular information. Critically, this approach can be implemented as a method to reduce cybersickness, as proposed by Weech and Troje (2017).

On return to the real world the individual is likely to move and explore the environment, causing a flow of both vestibular and visual signals about self-motion. After exposure to VR, the brain tends to habituate to extract self-motion information from visual cues in a vestibular-conflicting VR environment. As for traditional sensory adaptation phenomena, a form of negative correlation between a current percept and the adapted stimuli may take place (Barlow

---

2 However, in some circumstances vestibular information can be misleading. For example, the semi-circular canals adapt to constant rotation and report no motion, while constant linear accelerations may be perceived as static tilts as in the somatogravic illusion – see Snyder, 1999, for further information.
and Hill 1963). An error correction process needs to occur in order to de-correlate the current percept from the adapted stimuli. Thus, a further re-weighting of both sensory signals is necessary for the optimal integration of sensory input. A period of re-adaptation must occur, whereby the vestibular cue must be up-weighted and visual cue down-weighted. During this period of re-adaptation, VR after-effects may occur over time until the vestibular cue is weighted to its usual state. The time-course of these after-effects is not yet fully known and may range from a period of minutes to hours or days (Kellog et al. 1980; Gower and Fowlkes 1989; Harm et al. 2008).

Traditionally, cybersickness has been explained by prior knowledge of predicted sensory consequences of self-motion (Reason and Brand 1975). Our conceptual scheme suggests a dynamic on-line re-weighting function of sensory cues for self-motion which can explain cybersickness, adaptation, and after-effects of VR. Reciprocal inhibitory vestibular-visual interactions support this hypothesis. Accordingly, PET studies using artificial vestibular stimulation demonstrated not only an activation of the PIVC but also a decrease in rCBF of the visual cortex (Wenzel et al. 1996; Brandt et al. 1998; Deutschländer et al. 2002). Similarly, Bense et al. (2001) showed deactivation of the occipital visual cortex induced by vestibular stimulation, and deactivation of the vestibular areas during optic flow.

**Suggestions for future research**

The conceptual scheme proposed here makes clear testable predictions about multisensory interactions in VR exposure and cybersickness. These could be investigated in lab-based cognitive experiments or in more applied VR scenarios. First, if the vestibular system plays a fundamental role in cybersickness, one might predict that synchronised passive movements or artificial vestibular stimulation may reduce the conflict between visual and
vestibular cues, preventing sickness and forgoing the need for sensory re-weighting function in order to adapt to the VR environment. Indeed, recent reports (Reed-Jones et al. 2007; Cevette et al. 2012; Galvez-Garcia et al. 2015) have demonstrated that both galvanic vestibular stimulation and galvanic cutaneous stimulation can reduce sickness in simulators.

Second, if vestibular cues are down-weighted during VR exposure, one might expect that physiological vestibular functioning is altered during or immediately after VR. Although this aspect has not yet been investigated, one can imagine a testing battery investigating both vestibular sensitivity and physiological functioning in order to identify whether the vestibular organs adapt to VR.

Finally, individual variability in the re-weighting function may correlate with cybersickness susceptibility. Those whose weighting function rapidly changes reliance on the vestibular cue under VR conditions are potentially less likely to experience cybersickness, as the cue conflict is rapidly resolved. By contrast, those with a higher reliance on the vestibular cue under normal conditions may be more susceptible to cybersickness as the magnitude of the cue conflict is greater. Extensive research on these predictions has yet to be conducted, however we note that Balter et al. (2004) found no difference on sensory re-weighting abilities between participants susceptible and non-susceptible to car-sickness. Participants susceptible and non-susceptible to car-sickness were repeatedly administered with galvanic vestibular stimulation while their body sway was measured. Although it was predicted that participants who were less susceptible to car-sickness would habituate to the vestibular stimulation (and therefore show reduced body sway) faster than participants who suffered from car-sickness, both susceptible and non-susceptible participants showed similar habituation gains to the vestibular stimulation. However, it is important to highlight that these findings do not refer to conflicting stimuli; it is therefore possible that susceptible and non-susceptible individuals may show differences in re-weighting when exposed to visual–vestibular conflicting signals.
Conclusion

While significant advances have been made in understanding cybersickness, there is broad scope for further advancement in this field. The symptom profile of cybersickness has been clearly delineated, as well as identification of factors which influence its development. However, explanations for why cybersickness occurs based on sensory conflict theory are tentative, and there is no identified predictive mechanism. In addition, a conspicuous gap in our knowledge concerns the after-effects of virtual reality exposure. A few studies have identified cybersickness symptoms, proprioceptive disruptions, postural instability and oculomotor symptoms as potential after-effects, however their time-course is unknown, despite the time-course of cybersickness itself being well-defined (Kennedy et al. 2000). The current sensory conflict models also cannot fully account for why these after-effects arise.

Here we argued that an approach based on multisensory integration could provide a predictive explanation for cybersickness, adaptation to VR and its after-effects. Under normal circumstances, visual and vestibular cues are optimally integrated for self-motion perception. However, in VR these cues conflict with one another, prompting cybersickness, and must be re-weighted with higher reliance on the visual cue to allow integration. This dynamic re-weighting function has the potential to explain adaptation and after-effects of VR, as well as individual differences in cybersickness susceptibility. It is hoped that this approach could further our knowledge of cybersickness, as well as lead to clearer avenues for prevention of cybersickness symptoms.
Acknowledgments

This work was supported by The British Academy award “Cybersickness: a perceptual information-processing approach” [grant number SG162313]. M.G. is further supported by a ESRC-DTC studentship.
References


Table 1. Symptoms and Physiological Changes in Cybersickness.

Symptoms of cybersickness falling under each category of Kennedy et al.’s (1993) Simulator Sickness Questionnaire, and reported physiological changes caused by cybersickness.

<table>
<thead>
<tr>
<th>Symptoms of cybersickness (according to the Simulator Sickness Questionnaire Categories)</th>
<th>Physiological changes in cybersickness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nausea</td>
<td>Oculomotor</td>
</tr>
<tr>
<td>Discomfort</td>
<td>Discomfort</td>
</tr>
<tr>
<td>Increased</td>
<td>Fatigue</td>
</tr>
<tr>
<td>Salivation</td>
<td>Headache</td>
</tr>
<tr>
<td>Sweating</td>
<td>Eyestrain</td>
</tr>
<tr>
<td>Nausea</td>
<td>Difficulty</td>
</tr>
<tr>
<td>Difficulty</td>
<td>Focusing</td>
</tr>
<tr>
<td>Concentrating</td>
<td>Difficulty</td>
</tr>
<tr>
<td>Stomach Awareness</td>
<td>Concentrating</td>
</tr>
<tr>
<td>Burping</td>
<td>Blurred Vision</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 2. Theories of Cybersickness

A brief overview of motion sickness theories commonly applied to cybersickness.

<table>
<thead>
<tr>
<th>Theory</th>
<th>Authors</th>
<th>Key aspects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensory conflict theories</td>
<td>Reason &amp; Brand 1975</td>
<td>Sensory signals which do not match stored sensory signals generate a mismatch signal, triggering motion sickness</td>
</tr>
<tr>
<td></td>
<td>Oman 1988</td>
<td>Sensory conflicts increase a mismatch vector, triggering motion sickness</td>
</tr>
<tr>
<td></td>
<td>Bles et al. 1998</td>
<td>Conflicts between the sensed and predicted gravitational verticals trigger motion sickness</td>
</tr>
<tr>
<td>Other theories</td>
<td>Riccio &amp; Stoffregen 1991</td>
<td>Postural instability causes motion sickness</td>
</tr>
</tbody>
</table>