Vection in virtual reality modulates vestibular-evoked myogenic potentials

Maria Gallagher | Ross Dowsett | Elisa Raffaella Ferrè

Department of Psychology, Royal Holloway University of London, Egham, UK

1 INTRODUCTION

Any organism moving through its environment receives a constant stream of sensory signals about self-motion: optic flow inputs from vision, proprioceptive information about the position of the body from muscles, joints and tendons, and inputs for acceleration via the vestibular system. This latter seems particularly important for self-motion (Green & Angelaki, 2010). Three orthogonal semi-circular canals detect rotational movements of the head in the three-dimensional space, and two otolith organs (utricle and saccule) sense translational acceleration. Vestibular inputs are integrated...
with signals from other sensory modalities, such as vision, proprioception and touch (Alberts et al., 2016; Angelaki, Gu, & DeAngelis, 2011; Ferré & Haggard, 2015; Greenlee et al., 2016). Multimodal interactions have been described in almost all vestibular relays, including the vestibular nuclei, the thalamus and several areas in the cerebral cortex (Lopez, Blanke, & Mast, 2012; Zu Eulenburg, Caspers, Roski, & Eickhoff, 2012). Such sensory convergence architecture reflects its key role in self-motion, and the redundancy with other modalities, described above.

Under normal circumstances, sensory signals for self-motion are successfully integrated to produce a coherent representation of the organism in the external environment. However, conflicts between sensory modalities may occur when sensory signals carry discrepant information. This seems to be the case in virtual reality (VR). The popularity of VR has increased rapidly in recent years. While significant technological advancements are apparent, a troublesome problem with VR is that between 20% and 80% of users experience unpleasant side effects such as nausea, disorientation, blurred vision and headaches—a malady known as Cybersickness (Munafo, Diedrick, & Stoffregen, 2017; Stanney, Kennedy, & Drexler, 1997). Critically, many VR applications induce an illusory sense of self-motion, namely vection (Palmsano, Allison, Schira, & Barry, 2015). During vection in VR, the user is stationary while feeling a compelling sense of translation or rotation induced by optic flow. Consider for instance a typical VR scenario, in which a VR user is driving a car. The simulation provides accurate optic flow patterns of the road, buildings and other parts of the environment. Thus, the visual signals tell the user that they are moving in a certain direction with certain acceleration. However, since the user is not actually moving, the vestibular organs signal that the user is stationary, causing a sensory conflict which may lead to VR-induced motion sickness.

The underlying mechanisms of Cybersickness are not entirely clear, and several theories have been proposed (Riccio & Stoffregen, 1991; Bles, Bos, De Graaf, Groen, & Wertheim, 1998; see Rebenitsch & Owen, 2016, for a review). However, evidence suggests that Cybersickness, as in more general motion sickness (LaViola, 2000; Reason & Brand, 1975; Rebenitsch & Owen, 2016), may be triggered by visuo-vestibular conflict in VR. Accordingly, the severity of sickness symptoms increases with increased visuo-vestibular conflict (Akiduki et al., 2003; Keshavarz & Hecht, 2011). It might be surprising that recent improvements in VR technology have not reduced Cybersickness: although several improvements in refresh rate, display resolution, position tracking of HMDs have increased realism in VR, no significant reduction of Cybersickness has been observed (Shafer, Carbonara, & Korpi, 2017, 2019). Moreover, VR applications with greater levels of realism have been shown to increase levels of Cybersickness, possibly due to even stronger visuo-vestibular conflicts (Davis, Nesbitt, & Nalivaiko, 2015; Merhi, Faugloire, Flanagan, & Stoffregen, 2007; Stanney, Hale, Nahmens, & Kennedy, 2003). Thus, VR requires the brain to adjust incoming sensory information to extract self-motion information in a vection-only environment.

Senses are usually integrated and weighted according to their reliability, with increased weight placed on more reliable ones (Ernst & Bülthoff, 2004; Knill & Pouget, 2004; Stein, London, Wilkinson, & Price, 1996). As such, when a sensory modality becomes unreliable the weighting placed on it is lowered, and other sensory modalities are given higher weighting (Ernst & Banks, 2002). Electrophysiological evidence supports this optimal integration framework for visuo-vestibular integration for self-motion (Angelaki et al., 2011; DeAngelis & Angelaki, 2012; Fetsch, Pouget, DeAngelis, & Angelaki, 2009; Gu, Angelaki, & DeAngelis, 2008). For example, heading direction is more accurately detected when both visual (vection) and vestibular (acceleration) cues are present (Gu et al., 2008). Moreover, as the coherence of vection cues decreases, reliance on vestibular cues increases (Fetsch et al., 2009). Thus, to deal with visuo-vestibular conflicts, such as in VR, vestibular cues may be substantially down-weighted (see Gallagher & Ferré, 2018, for a review). Accordingly, changing the reliability of vestibular cues by noisy galvanic vestibular stimulation has been shown to reduce visuo-vestibular conflict in VR (Weech & Troje, 2017). Brain regions associated with visuo-vestibular integration are likely to support this process. Vection has been shown to activate MT+, CSv, precuneus and parieto-insular vestibular cortex (PIVC) (Cardin & Smith, 2010; Kovács, Raabe, & Greenlee, 2008; Uesaki & Ashida, 2015; Wall & Smith, 2008). Importantly, activity in PIVC decreases when people experience a sensation of vection, supporting a functional modulation of vestibular activity (Brandt, Bartenstein, Janek, & Dieterich, 1998; Kleinschmidt, 2002; however, see Uesaki & Ashida, 2015, for contrasting findings).

Changes in vestibular functioning may be reflected by altered vestibular experiences occurring during or in the hours and days following VR exposure (Di Girolamo & Pic, 2001; Lampton et al., 1994; Stanney, Kennedy, Drexler, & Harm, 1999). For example, vestibulo-ocular reflex gain decreases following VR exposure (Di Girolamo & Pic, 2001), and coordination between the eyes, head and hands is poorer (Harm, Taylor, Reschke, Somers, & Bloomberg, 2008). Furthermore, in one of the more bizarre cases, a pilot had his view of the world invert 180 degrees while driving a car hours after being trained in a flight simulator (Kennedy et al., 1987). This disturbing VR after-effect may be due to altered reliability of vestibular cues for orientation: on returning to the real world after VR, vestibular inputs are once again present—the user
would possibly move in the environment—and the brain would need to re-weight the vestibular signals which have been attenuated during VR exposure. The vestibular cues are now given a higher weighting than during the VR exposure itself. The mentioned changes in vestibulo-ocular reflex gain are a proxy for this (Di Girolamo & Pic, 2001).

Here, we investigated whether vestibular signals are modulated by a brief exposure to full-field vection in VR. We measured reflex responses to sound-evoked vestibular stimulation (vestibular-evoked myogenic potentials, VEMPs) after immersion in a VR environment eliciting an illusory sensation of linear vection. Importantly, VEMPs are a gold-standard measure for the functioning of the otolith receptors, widely used in clinical settings (Rosengren & Kingma, 2013). Loud sounds stimulate the saccule and generate a characteristic motor response in the sternocleidomastoid muscle, which is functionally involved with neck flexion and head rotation. Thus, VEMPs could be taken as an indicator of wider vestibular—cortical changes elicited by the sensory conflict in VR. Previous research has found changes in VEMPs related to motion sickness susceptibility: motion sickness is positively correlated with both VEMP amplitudes and asymmetry ratios (Fowler, Sweet, & Steffel, 2014; Tal et al., 2013). Similarly, exposure to microgravity, which alters otolith functioning, caused changes in VEMP asymmetry ratios (Clarke & Schönfeld, 2015). Therefore, we predict that similar changes may occur during exposure to VR-induced sensory conflict: exposure to vection in VR would modulate the amplitude, and subsequently asymmetry ratio, of VEMPs induced by sound-evoked vestibular stimulation.

2 | METHODS

2.1 | Ethics

Written informed consent was obtained from participants before commencing the experiment. The experimental protocol was approved by the local ethics committee (Royal Holloway University of London), and the study was conducted in line with the Declaration of Helsinki. The authors declare no conflicts of interest.

2.2 | Participants

Twenty-four healthy participants (17 female, M age = 21.13, SD = 3.90) completed the study. Twenty-one participants were right-handed according to their Edinburgh Handedness Inventory scores. Exclusion criteria were any history of neurological, psychiatric, hearing or vestibular disorders, epilepsy or family history of epilepsy.
2.3 | Visual stimuli

Visual stimuli were presented on an Oculus Rift DK2 head-mounted display (HMD) (Figure 1a). We note that other VR displays, such as CAVEs or surround screens, may cause similar conflicts in visuo-vestibular processing (Kennedy, Drexler, & Kennedy, 2010). Thus, while not under direct consideration in the present study, we believe that the underlying mechanisms in these alternative VR displays may be similar to those when viewing stimuli via a HMD. Participants viewed a full-field pattern of moving white dots on a black background (Figure 1b). In the random motion condition, the dots moved randomly. In the vection condition, the dots formed an expanding flow pattern, causing a sensation of linear acceleration. Each dot was assigned a random scaling factor between 0.01 and 1.5. On each frame, each dot expanded in size by its scaling factor in pixels from a minimum of 1 to a maximum of 9 pixels in diameter. Once the maximum size was reached, the size reset to 1-pixel diameter. The location of the dot on each frame was determined by multiplying its default X and Y coordinates by $\text{ ScalingFactor} = 1.5$. Thus, dots nearer the centre travelled less distance than dots farther from the centre. A white fixation cross was displayed at the centre of the HMD. These parameters were used to investigate the specific effects of vection on vestibular processing. We thus eliminated other features usually present in more complex VR scenarios. Participants viewed the display for 60s before VEMP recording was taken and continued to view the display until the VEMP recording was completed.

2.4 | VEMP recording

VEMPs were measured according to standard procedures (Clarke & Schönfeld, 2015; Colebatch, Halmagyi, & Skuse, 1994; Fowler et al., 2014) using BioMed eVEMP USB software and hardware. Electrodes were placed on the left and right sternocleidomastoid muscles in a bipolar configuration, with ground electrodes on the forehead and sternum or collar bone (Figure 1a). HDA 280 Sennheiser headphones were worn by the participants to deliver the stimuli. VEMPs were elicited via 500 Hz tone burst stimuli of 7-ms duration at 100 dB SPL into the ear ipsilateral to the side of measurement. Muscle contraction was achieved by asking the participant to turn the head to the contralateral side and push the head down towards the floor while lying supine. The visual stimulus remained directly in front of the participant during the head movement, and participants were asked to maintain the correct posture while the VEMP measurements were taken. Measurements were recorded at 2000 Hz sampling frequency when the software detected that muscle tension was between 120 and 400 μV RMS and electrode impedance less than 20kΩ. Given that VEMP amplitudes depend on the activation of the sternocleidomastoid muscle, we used a repeated-measures design whereby each participant completed both vection and random motion conditions on both muscle sides. The first side of measurement was counterbalanced across participants, while the visual condition was randomised within each muscle side. One hundred single trials of 80-ms duration were averaged to give the final VEMP measurement, with amplitudes and latencies provided automatically by the eVEMP software. P1-N1 intervals were calculated by taking the time difference between N1 and P1 latencies. Asymmetry ratios were calculated accordingly, with negative values indicating higher amplitudes on the left muscle side and positive values indicating higher amplitudes on the right muscle side:

$$\text{Asymmetry ratio} = \frac{|P1_{\text{N1Amp}}_L - P1_{\text{N1Amp}}_R|}{P1_{\text{N1Amp}}_L + |P1_{\text{N1Amp}}_R|} \times 100$$

2.5 | Procedure

After completing informed consent procedures, participants were instructed to watch the stimuli on the HMD in a relaxed supine position for one minute before turning the head to the relevant muscle side and completing the VEMP measurement. Participants first completed practice trials on each muscle side without wearing the HMD to ensure that they adopted the correct posture and to verify accurate VEMP recording. Left and right VEMPs were then recorded in both vection and random motion conditions. The first side of measurement was counterbalanced across participants, while initial visual motion type was randomised within each muscle side. Participants were instructed to rest for three minutes in between measurements to allow the muscles to relax. If measurements were not successfully obtained, the trial was repeated. Participants were asked to report whether they experienced any sensations of self-motion while watching the stimuli. Participants also completed a Motion Sickness Susceptibility Questionnaire (MSSQ, Golding, 1998) at the start of the session.

2.6 | Data analysis

Differences between random motion and vection conditions and measurement side were analysed using 2x2 repeated-measures ANOVAs for P1-N1 peak-to-peak amplitudes, P1 and N1 latencies and P1-N1 intervals. Paired t tests with Bonferroni correction were used to follow up any significant main effects or interactions. A paired $t$ test was conducted on asymmetry ratios between random motion and vection conditions.

MSSQ percentile scores were calculated according to Golding (2006). Pearson’s correlations between amplitudes following vection exposure and MSSQ percentile scores were also conducted.
TABLE 1  Means (SDs) for P1-N1 peak-to-peak amplitudes, P1 and N1 latencies, and P1-N1 intervals by muscle side and vection condition

|          | Left        | Right       |
|----------|-------------|-------------|
|          | Vection     | Random      | Vection     | Random      |
| P1-N1 amplitude (µV) | 281.47 (80.59) | 252.42 (63.03) | 255.02 (64.41) | 255.03 (40.75) |
| P1 latency (ms) | 15.80 (4.19) | 14.78 (3.78) | 14.21 (4.71) | 15.56 (4.40) |
| N1 latency (ms) | 27.18 (3.95) | 27.69 (3.77) | 26.03 (4.41) | 27.40 (4.16) |
| P1-N1 interval (ms) | 11.38 (3.20) | 12.90 (4.30) | 11.82 (4.23) | 11.83 (4.43) |

3  | RESULTS

3.1  | Vection reports

As expected, 22 of 24 participants experienced self-motion during the vection condition. 3 of 24 participants reported motion sensations during the random condition. 2 of 24 reported no sensations of self-motion at all. All participants were included in the main analysis.

3.2  | Asymmetry ratio

A significant difference in asymmetry ratio was found between vection and random motion ($t(23) = -2.14, p = 0.04$, Cohen’s $d = 0.42$). Specifically, asymmetry increased following exposure to vection (mean = 4.51, $SD = 14.58$) compared to random (mean = 1.22, $SD = 12.67$) motion, with larger amplitudes on the left (mean = 281.47, $SD = 80.59$) versus right (mean = 255.02, $SD = 64.41$) muscle side.

3.3  | P1-N1 peak-to-peak amplitude

Means and SDs for P1-N1 peak-to-peak amplitudes for each condition can be seen in Table 1. No significant main effects of visual condition ($F_{1,23} = 2.26$, $p = 0.15$, $\eta^2_p = 0.089$) or muscle side ($F_{1,23} = 0.75$, $p = 0.40$, $\eta^2_p = 0.03$) were found on P1-N1 peak-to-peak amplitudes. However, a significant interaction between vection and muscle side was found ($F_{1,23} = 4.42$, $p = 0.047$, $\eta^2_p = 0.16$). Follow-up $t$ tests revealed a significant increase in VEMP amplitude on the left muscle side following exposure to vection ($M = 281.47$, $SD = 80.59$) compared to random motion ($M = 252.42$, $SD = 63.03$) stimuli ($t_{23} = 2.80$, $p = 0.01$, Cohen’s $d = 0.40$) (Figure 1c).

3.4  | P1 and N1 latency

Means and SDs for P1 and N1 latencies and P1-N1 intervals for each condition can be seen in Table 1. No significant main effect of visual condition ($F_{1,23} = 0.58$, $p = 0.81$, $\eta^2_p = 0.003$) or muscle side ($F_{1,23} = 0.25$, $p = 0.62$, $\eta^2_p = 0.011$) was found on P1 latency. No significant interaction was found ($F_{1,23} = 2.07$, $p = 0.16$, $\eta^2_p = 0.083$). Similarly, no significant main effect of visual condition ($F_{1,23} = 1.17$, $p = 0.29$, $\eta^2_p = 0.048$) or muscle side ($F_{1,23} = 1.51$, $p = 0.23$, $\eta^2_p = 0.062$) was found on N1 latency. No significant interaction was found ($F_{1,23} = 0.38$, $p = 0.54$, $\eta^2_p = 0.016$). Finally, no significant main effect of visual condition ($F_{1,23} = 0.72$, $p = 0.41$, $\eta^2_p = 0.03$) or muscle side ($F_{1,23} = 0.17$, $p = 0.69$, $\eta^2_p = 0.007$) was found on P1-N1 intervals. No significant interaction between factors emerged ($F_{1,23} = 1.47$, $p = 0.24$, $\eta^2_p = 0.06$).

3.5  | MSSQ correlation

The average MSSQ percentile score was 41.80%, corresponding to moderate motion sickness susceptibility. Individuals can be classified as having low susceptibility to motion sickness with percentile scores from 0 to 25%, moderate susceptibility from 25 to 75% and high susceptibility with scores above 75% (Golding, 2006). Accordingly, 8 participants in the present study had low susceptibility to motion sickness, 12 had moderate susceptibility, and 4 had high susceptibility. No significant correlations were found between MSSQ percentile scores and VEMP amplitudes after exposure to vection on either the left ($r = -0.07$, $p = 0.76$) or right ($r = 0.18$, $p = 0.39$) muscle side; thus, motion sickness susceptibility does not seem to influence the VR-induced increase in VEMP amplitude.

4  | DISCUSSION

Under normal conditions, the brain optimally combines sensory signals according to their reliability (Ernst & Banks, 2002). When experiencing vection, for example in VR, the visual system signals that the user is moving through the environment (vection); however, vestibular information signals that the body is stationary. This sensory conflict may subsequently lead to symptoms of Cybersickness (Stanney & Kennedy, 1998). The brain thus has to habituate to extract self-motion information from vection in a visuo-vestibular conflicting environment (Akiduki et al., 2003; Keshavarz & Hecht, 2011; Reason & Brand, 1975). To resolve this sensory conflict, vestibular signals for self-motion may be down-weighted, which may in turn affect how the brain processes incoming online...
vestibular information (Gallagher & Ferrè, 2018; Weech & Troje, 2017). As a result, the visuo-vestibular conflict is decreased. Critically, the re-weighting must rapidly occur to counteract the occurrence of the visuo-vestibular sensory conflict. Accordingly, Cybersickness symptoms have been shown to typically develop within the first minutes of VR exposure (Davis, Nesbitt, & Nalivaiko, 2014; Stanney & Kennedy, 1998). After VR exposure, vestibular signals for self-motion are once again present and a further adjustment must occur, whereby the vestibular signals are up-weighted. Here, we have found changes in vestibular processing after exposure to full-field vection in VR, supporting the idea of vestibular re-weighting.

A growing body of literature suggests dynamic re-weighting of visual and vestibular cues during and after VR vection exposure. Vestibular–ocular reflex (VOR) gain is decreased immediately after VR exposure (Di Girolamo & Pic, 2001), and neuroimaging studies report deactivation of vestibular brain regions (i.e. PIVC) during vection (Brandt et al., 1998; Kleinschmidt, 2002). These findings imply a down-weighting of vestibular cues when self-motion is experienced from vision. When vestibular cues become available, an up-weighting may occur, which may be reflected in our finding of increased left VEMP amplitude during vection in VR. Similarly, previous studies have reported an increased reliance on vestibular cues during distance perception in VR when both visual and vestibular cues are available (Harris, Jenkin, & Zikovitz, 1998, 2000; Jaekl, Jenkin, & Harris, 2005), and in postural control (Akizuki et al., 2005) or perception of heading direction (Ter Horst, Koppen, Selen, & Pieter Medendorp, 2015) when visual cues become unreliable. Overall, these findings therefore highlight the dynamic re-weighting of vestibular cues, which may explain adaptation and after-effects of VR exposure.

To our knowledge, no previous research has used VEMPs to investigate the effects of vection exposure on vestibular processing. VEMPs are a gold-standard measure that has been largely used both in clinical settings and research to establish the functionality of vestibular processing (Colebatch et al., 1994; Rosengren & Kingma, 2013). Previous research has demonstrated alterations in VEMPs induced by motion sickness elicited by real motion, such as seasickness (Fowler et al., 2014; Tal et al., 2013). For instance, Fowler et al. (2014) showed a correlation between VEMP amplitude and motion sickness susceptibility, with higher amplitude in individuals with high motion sickness susceptibility. We found changes in VEMP asymmetry ratio, with a substantial increase in VEMP amplitude recorded on the left sternocleidomastoid muscle following just one minute of exposure to vection in VR. Similarly, VEMP asymmetry has been reported to positively correlate with susceptibility to motion sickness (Xie et al., 2012; Neupane, Gururaj, & Sinha, 2018; however, see Buyuklu, Tarhan, & Ozluoglu, 2009, for contradictory findings). While our results showed changes in VEMP asymmetry following exposure to vection in VR, we did not find a correlation between VEMP amplitude and motion sickness susceptibility in our sample. Caution is required in interpreting null results, and we note that motion sickness susceptibility in our sample was low (8 participants) or moderate (12 participants). Thus, we cannot exclude that this might explain the absence of correlation between physiological measures and motion sickness susceptibility. Moreover, future research could consider whether changes in VEMPs correspond to alterations in levels of Cybersickness induced by vection in VR.

In the present study, we found an increase in VEMP asymmetry ratios following one minute of exposure to vection in VR. Interestingly, asymmetries in vestibular reflexes have been reported in other visuo-vestibular discrepant contexts. For example, changes in VEMP asymmetry have been described after exposure to altered gravity environments (Clarke & Schönfeld, 2015). In microgravity, the absence of gravitational cues alters vestibular functioning, which may be similar to the absence of vestibular cues during vection in VR. Accordingly, Clarke and Schönfeld (2015) found greater VEMP asymmetry immediately after individuals returned from a short-term Shuttle mission, with symmetry returning to baseline levels 5–8 days postflight.

The changes in VEMP asymmetry ratio in the present study corresponded to a substantial increase in VEMP amplitude recorded on the left sternocleidomastoid muscle following exposure to VR vection. It is possible that this asymmetry may be related to asymmetries in cortical vestibular, VEMPs and vection processing. Firstly, the vestibular cortical network is distributed asymmetrically, with a preponderance of vestibular cortical regions on the right hemisphere in right-handed individuals (Dieterich et al., 2003). Thus, differences in vestibular cortical processing might have caused an interaction between vestibular responses and vection conditions. Secondly, VEMPs have been demonstrated to elicit differences in hemispherical cortical activity (Schlindwein et al., 2008). Specifically, both left and right VEMPs activated ipsilateral superior, transverse and middle temporal gyri and posterior insula; however, left VEMPs also included a deactivation of bilateral dorsomedial frontal cortex, right postcentral and supramarginal gyrus, and left caudate body and cerebellar tonsil. In addition, right VEMP activations were comparatively stronger than left VEMP activations, potentially reflecting the right hemisphere preponderance previously reported (Dieterich et al., 2003; Schlindwein et al., 2008). Thus, we cannot exclude that these asymmetries in cortical VEMP processing are further enhanced following exposure to vection in VR. Finally, as well as asymmetries in cortical vestibular processing, asymmetric hemispheric effects have been found in relation to vection processing. Kovács et al. (2008), for example, found greater activation in right MT+ during self vs object motion perception, as...
well as greater left precuneus activation. Moreover, several changes relating to visually induced motion sickness (VIMS) have been found, including a decreased correlation between left and right MT+ activity (Miyazaki et al., 2015), reduced connectivity between left and right V1, and increased connectivity between right MT+ and anterior insula and left MT+ and MCC (Toschi et al., 2017). Taken together, differences in cortical activity induced by VEMPs, vestibular functioning, and vection may account for the differential effects of vection on left versus right VEMPs in the present study; however, further verification is necessary.

An extensive account of after-effects of VR exposure has yet not been conducted (Gallagher & Ferrè, 2018). Previous research has found that 20 minutes of exposure to VR has detrimental effects on proprioceptive coordination between eyes, hands and head (Harm et al., 2008), increased pointing errors (Stanney et al., 1999) and decreases in vestibular–ocular reflex gain (Di Girolamo & Pic, 2001). Here, we found increases in VEMP asymmetry and amplitude following just one minute of exposure to vection in VR, suggesting that the effects of VR adaptation may occur within the first moments of VR exposure. As participants in the present study were exposed to VR self-motion over a very brief timescale (less than 2 minutes), further changes in VEMP asymmetry may become apparent after longer exposures to VR as participants habituate to the sensory conflict. Moreover, while the majority of participants in the present study reported that they felt the sensation of vection, we did not include additional measures of vection qualities, such as its intensity. Future research may therefore consider whether such qualities correlate with modulation of the VEMPs. Furthermore, while we investigated vection in VR, it is possible that similar changes may arise from vection induced by other sources, such as projections or computer screens (Keshavarz, Speck, Haycock, & Berti, 2017). Further research could therefore consider any potential differences in VEMPs according to display type.

VR is predicted to be pervasive in our lives: in five years, we will use VR as we are now using smartphones. Although VR is revolutionising our approach to technologies, education and entertainment, there is a widely recognised need to identify whether such technology can affect neural processing and behaviours (Gallagher & Ferrè, 2018). Our results indicate that vestibular processing is rapidly altered during vection in VR. Importantly, this occurs below the user’s conscious perception and might explain the after-effects often reported after VR exposure (Di Girolamo & Pic, 2001; Stanney et al., 1999).

Acknowledgements

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. We would like to thank BioMed, Craig Arnold and Alex Cheyne for their assistance with VEMP recording.

Competing Interests

The authors declare no conflicts of interest regarding this work.

Author Contributions

M.G. and R.D. performed experiments; M.G. analysed data; E.R.F. and M.G. involved in the conception and design of research; E.R.F. and M.G. interpreted results of experiments; E.R.F. and M.G. edited and revised manuscript; all authors approved final version of manuscript.

Data Accessibility

Data are available online as supplementary material.

ORCID

Maria Gallagher https://orcid.org/0000-0002-2933-4579

References

Akizuki, H., Uno, A., Ariki, K., Morioka, S., Ohyama, S., Nishiike, S., … Takeda, N. (2005). Effects of immersion in virtual reality on postural control. Neuroscience Letters, 379, 23–26.

Angelaki, D. E., Gu, Y., & Deangelis, G. C. (2011). Visual and vestibular cue integration for heading perception in extrastriate visual cortex. Journal of Physiology, 589, 825–833.

Bles, W., Bos, J. E., De Graaf, B., Groen, E., & Wertheim, A. H. (1998). Motion sickness: Only one provocative conflict?. Brain Research Bulletin, 47, 481–487.

Brandt, T., Bartenstein, P., Janek, A., & Dieterich, M. (1998). Reciprocal inhibitory visual-vestibular interaction. Visual motion stimulation deactivates the parieto-insular vestibular cortex. Brain, 121, 1749–1758.

Buyl, K., Turhan, E., & Ozluoglu, L. (2009). Vestibular functions in motion sickness susceptible individuals. European Archives of Otorhino-Laryngology, 266, 1365–1371.

Cardin, V., & Smith, A. T. (2010). Sensitivity of human visual and vestibular cortical regions to egomotion-compatible visual stimulation. Cerebral Cortex, 20, 1964–1973.

Clarke, A. H., & Schönfeld, U. (2015). Modification of unilateral otolith responses following spaceflight. Experimental Brain Research, 233, 3613–3624.

Colebatch, J. G., Halmagyi, G. M., & Skuse, N. F. (1994). Myogenic potentials generated by a click-evoked vestibuloocular reflex. Journal of Neurology, Neurosurgery and Psychiatry, 57, 190–197.
Davis, S., Nesbitt, K., & Nalivaiko, E. (2014). A Systematic Review of Cybersickness. *Proc. 2014 Conf. Interact. Entertain. - IE2014*, 1–9.

Davis, S., Nesbitt, K., & Nalivaiko, E. (2015). Comparing the onset of cybersickness using the Oculus Rift and two virtual roller coasters. *11th Australas. Conf. Interact. Entertain. (IE 2015)*, 27–30.

DeAngelis, G. C., & Angelaki, D. E. (2012). Visual – vestibular integration for self motion perception. In M. Murray & M. Wallace (Eds.), *The neural bases of multisensory processes* (pp. 1–21). Boca Raton, FL: CRC Press/Taylor & Francis.

Di Girolamo, S., & Pic, P. (2001). Vestibulo-ocular reflex modification after Davis, S., Nesbitt, K., & Nalivaiko, E. (2015). Comparing the onset of cybersickness using the Oculus Rift and two virtual roller coasters. *11th Australas. Conf. Interact. Entertain. (IE 2015)*, 27–30.

Davis, S., Nesbitt, K., & Nalivaiko, E. (2014). A Systematic Review of Cybersickness. *Proc. 2014 Conf. Interact. Entertain. - IE2014*, 1–9.

DeAngelis, G. C., & Angelaki, D. E. (2012). Visual – vestibular integration for self motion perception. In M. Murray & M. Wallace (Eds.), *The neural bases of multisensory processes* (pp. 1–21). Boca Raton, FL: CRC Press/Taylor & Francis.

Fetsch, C. R., Turner, A. H., DeAngelis, G. C., & Angelaki, D. E. (2012). Dynamic reweighting of visual and vestibular cues during self-motion perception. *Journal of Neuroscience*, 29, 15601–15612.

Fetsch, C. R., Pouget, A., DeAngelis, G. C., & Angelaki, D. E. (2009). Neural correlates of reliability-based cue weighting during multisensory integration. *Nature Neuroscience*, 15, 146–154.

Fetsch, C. R., Turner, A. H., DeAngelis, G. C., & Angelaki, D. E. (2009). Dynamic reweighting of visual and vestibular cues during self-motion perception. *Journal of Neuroscience*, 29, 15601–15612.

Fowler, C. G., Sweet, A., & Steffel, E. (2014). Effects of motion sickness severity on the vestibular-evoked myogenic potentials. *Journal of the American Academy of Audiology*, 14, 724–732.

Gallagher, M., & Ferré, E. R. (2018). Cybersickness : A multisensory integration perspective. *Multisensory Research*, 31, 645–674.

Golding, J. F. (1998). Motion sickness susceptibility questionnaire revisited and its relationship to other forms of sickness. *Brain Research Bulletin*, 47, 507–516.

Golding, J. F. (2006). Predicting individual differences in motion sickness susceptibility by questionnaire. *Personality and Individual Differences*, 41, 237–248.

Green, A. M., & Angelaki, D. E. (2010). Multisensory integration: Resolving sensory ambiguities to build novel representations. *Current Opinion in Neurobiology*, 20, 353–360.

Greenlee, M. W., Frank, S. M., Kaluzhna, M., Blanke, O., Bremmer, F., Churan, J., … Smith, A. T. (2016). Multisensory integration in self motion perception. *Multisensory Research*, 29, 1–32.

Gu, Y., Angelaki, D. E., & DeAngelis, G. C. (2008). Neural correlates of multisensory cue integration in macaque MSTd. *Nature Neuroscience*, 11, 1201–1210.

Harm, D. L., Taylor, L. C., Reschke, M. F., Somers, J. T., & Bloomberg, J. J. (2008). Sensorimotor coordination after-effects of exposure to a virtual environment. *The Visual Computer*, 24, 995–999.

Harris, L., Jenkin, M., & Zikovitz, D. C. (1998). Vestibular cues and virtual environments choosing the magnitude of the vestibular cue. *Proceedings IEEE 1998 Virtual Real. Ann. Int. Symp.* (Cat. No. 98CB36180), 389–399.

Harris, L. R., Jenkin, M., & Zikovitz, D. C. (2000). Visual and non-visual cues in the perception of linear self motion. *Experimental Brain Research*, 135, 12–21.

Jaeck, P. M., Jenkin, M. R., & Harris, L. R. (2005). Perceiving a stable world during active rotational and translational head movements. *Experimental Brain Research*, 163, 389–399.

Kennedy, R. S., Berbaum, K. S., Lilienthal, M. G., Dunlap, W. P., Mulligan, B. E., & Funaro, J. F. (1987). Guidelines for Alleviation of Simulator Sickness Symptomatology, 559–563.

Kennedy, R. S., Drexler, J., & Kennedy, R. C. (2010). Research in visually induced motion sickness. *Applied Ergonomics*, 41, 494–503.

Keshavarz, B., & Hecht, H. (2011). Axis rotation and visually induced motion sickness: The role of combined roll, pitch, and yaw motion. *Aviation, Space, and Environmental Medicine*, 82, 1023–1029.

Keshavarz, B., Speck, M., Haycock, B., & Berti, S. (2017). Effect of different display types on vection and its interaction with motion direction and field dependence. *Iperception*, 8, 1–18.

Kleinschmidt, A. (2002). Neural correlates of visual-motion perception as object- or self-motion. *NeuroImage*, 16, 873–882.

Knill, D. C., & Pouget, A. (2004). The Bayesian brain: The role of uncertainty in neural coding and computation. *Trends in Neurosciences*, 27, 712–719.

Kovács, G., Raabe, M., & Greenlee, M. W. (2008). Neural correlates of visually induced self-motion illusion in depth. *Cerebral Cortex*, 18, 1779–1787.

Lampton, D. R., Kolasinski, E. M., Knerr, B. W., Bliss, J. P., Bailey, J. H., & Witmer, B. G. (1994). Side effects and after-effects of immersion in virtual environments. *Proceedings of the Human Factors and Ergonomics Society Annual Meeting*, 38, 1154–1157.

LaViola, J. J. (2000). A discussion of cybersickness in virtual environments. *ACM SIGCHI Bulletin*, 32, 47–56.

Lopez, C., Blanke, O., & Mast, F. W. (2012). The human vestibular cortex revealed by coordinate-based activation likelihood estimation meta-analysis. *Neuroscience*, 212, 159–179.

Merhi, O., Faugloire, E., Flanagan, M., & Stoffregen, T. A. (2007). Motion sickness, console video games, and head-mounted displays. *Human Factors*, 49, 920–934.

Miyazaki, J., Yamamoto, H., Ichimura, Y., Yamashiro, H., Murase, T., Yamamoto, T., … Higuchi, T. (2015). Inter-hemispheric desynchronization of the human MT+ during visually induced motion sickness. *Experimental Brain Research*, 233, 2421–2431.

Munafo, J., Diedrick, M., & Stoffregen, T. A. (2017). The virtual reality head-mounted display Oculus Rift induces motion sickness and is sexist in its effects. *Experimental Brain Research*, 235, 889–901.

Neupane, A. K., Gururaj, K., & Sinha, S. K. (2018). Higher asymmetry ratio and refixation saccades in individuals with motion sickness. *Journal of the American Academy of Audiology*, 29, 175–186.

Palmisano, S., Allison, R. S., Schira, M. M., & Barry, R. J. (2015). Future challenges for vection research: Definitions, functional significance, measures, and neural bases. *Frontiers Psychology*, 6, 1–15.

Reason, J. T., & Brand, J. J. (1975). *Motion sickness*. New York, NY: Academic Press.

Rebenitsch, L., & Owen, C. (2016). Review on cybersickness in applications and visual displays. *Virtual Reality*, 20, 101–125.

Riccio, G. E., & Stoffregen, T. A. (1991). An ecological theory of motion sickness and postural instability. *Ecological Psychology*, 3, 195–240.

Rosengren, S. M., & Kingma, H. (2013). New perspectives on vestibular evoked myogenic potentials. *Current Opinion in Neurology*, 26, 74–80.

Schlindwein, P., Mueller, M., Bauerum, T., Brandt, T., Stoeter, P., & Dieterich, M. (2008). Cortical representation of saccular vestibular stimulation: VEMPs in MRI. *NeuroImage*, 39, 19–31.

Shafer, D. M., Carbonara, C. P., & Korpi, M. F. (2017). Modern virtual reality technology: Cybersickness, sense of presence, and gender. *Media Psychology Review*, 11(2), 1–13.
Shafer, D. M., Carbonara, C. P., & Korpi, M. F. (2019). Factors affecting enjoyment of virtual reality games: A comparison involving consumer-grade virtual reality technology. *Games Health Journal, 8*, 15–23.

Stanney, K. M., Hale, K. S., Nahmens, I., & Kennedy, R. S. (2003). What to expect from immersive virtual environment exposure: influences of gender, body mass index, and past experience. *Human Factors, 45*, 504–520.

Stanney, K. M., & Kennedy, R. S. (1998). After-effects from virtual environment exposure: How long do they last? *Proceedings of the Human Factors and Ergonomics Society Annual Meeting, 42*, 1476–1480.

Stanney, K. M., Kennedy, R. S., & Drexler, J. M. (1997). Cybersickness is not simulator sickness. *Proc. Hum. Factors Ergon. Soc. 41st Annu. Meet.*, 1138–1142.

Stanney, K. M., Kennedy, R. S., Drexler, J. M., & Harm, D. L. (1999). Motion sickness and proprioceptive after-effects following virtual environment exposure. *Applied Ergonomics, 30*, 27–38.

Stein, B. E., London, N., Wilkinson, L. K., & Price, D. D. (1996). Enhancement of Perceived Visual Intensity by Auditory Stimuli: A Psychophysical Analysis. *Journal of Cognitive Neuroscience, 8*, 497–506.

Tal, D., Hershkovitz, D., Kaminski-Graif, G., Wiener, G., Samuel, O., & Shupak, A. (2013). Vestibular evoked myogenic potentials and habituation to seasickness. *Clinical Neurophysiology, 124*, 2445–2449.

Ter Horst, A. C., Koppen, M., Selen, L. P. J., & Pieter Medendorp, W. (2015). Reliability-based weighting of visual and vestibular cues in displacement estimation. *PLoS ONE, 10*, 10–17.

Toschi, N., Kim, J., Sclocco, R., Duggento, A., Barbieri, R., Kuo, B., & Napadow, V. (2017). Motion sickness increases functional connectivity between visual motion and nausea-associated brain regions. *Autonomic Neuroscience, 202*, 108–113.

Uesaki, M., & Ashida, H. (2015). Optic-flow selective cortical sensory regions associated with self-reported states of vection. *Frontiers Psychology, 6*, 1–9.

Wall, M. B., & Smith, A. T. (2008). The representation of egomotion in the human brain. *Current Biology, 18*, 191–194.

Wecch, S., & Troje, N. F. (2017). Vection latency is reduced by bone-conducted vibration and noisy galvanic vestibular stimulation. *Multisensory Research, 30*, 65–90.

Xie, S.-J., Chen, W., Jia, H.-B., Wang, Z.-J., Yao, Q., & Jiang, Y.-Y. (2012). Ocular vestibular evoked myogenic potentials and motion sickness susceptibility. *Aviation, 83*, 14–18.

Zu Eulenburg, P., Caspers, S., Roski, C., & Eickhoff, S. B. (2012). Meta-analytical definition and functional connectivity of the human vestibular cortex. *NeuroImage, 60*, 162–169.

---

**How to cite this article:** Gallagher M, Dowsett R, Ferrè ER. Vection in virtual reality modulates vestibular-evoked myogenic potentials. *Eur J Neurosci*. 2019;50:3557–3565. [https://doi.org/10.1111/ejn.14499](https://doi.org/10.1111/ejn.14499)