Vibro-motor Reprocessing Therapy towards Managing Motion Sickness Reduction: Evidence from EEG

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Abstract- This study examines the neural activities of participants undergoing vibro-motor reprocessing therapy (VRT) while experiencing motion sickness. We evaluated the efficacy of vibro-motor reprocessing therapy, a novel therapeutic technique based on eye movement desensitization and reprocessing (EMDR), in reducing motion sickness. Based on visually induced motion sickness in two sets of performed sessions, eight participants were exposed to VRT stimulation in a VRT/non-VRT setting. Simultaneously, brain activity changes were recorded using electroencephalography (EEG) at baseline and during stimulus exposure, and comparisons made across the VRT/non-VRT conditions. A significant reduction in the alpha (8-12 Hz) spectral power was observed in the frontal and occipital locations, consistent across all participants. Furthermore, significant reductions were also found in the frontal and occipital delta (0.5-4 Hz) and theta (4-8 Hz) spectral power frequency bands between non-VRT and VRT conditions (p < 0.05). Our results offer novel insights for a potential nonpharmacological treatment and attenuation of motion sickness. Furthermore, symptoms can be observed, and alleviated, in real-time using the reported techniques.

Clinical Relevance— Instead of using drugs to treat motion sickness, patients could safely use this VRT technique.

I. INTRODUCTION

The malaise of motion sickness can have negative consequences for healthy travellers and be severely negative for some. Motion sickness is a common and persistent physiological phenomenon, but may become more prevalent with the advent of autonomous vehicles. Studies have shown that passengers report increased sickness when engaged in non-driving related tasks [1]. A recent international study [2], postulates that two thirds of car passengers are affected by the problem. Even mild symptoms of motion sickness can decrease cognitive multitasking performance [3].

Normally, the vestibular, proprioceptive and visual systems communicate nonconflicting signals of (physical, visual or virtual) motion detection to the brain. The problem arises when these systems conflict each other in what is classically known as the sensory conflict or neural mismatch theory, proposed by [4][5]. For example, a passenger reads a static book, meanwhile the vestibular system detects dynamic movement. In this case the visual input from the eyes receives and transmits conflicting signals to the brain, and thus motion sickness is onset.

Motion sickness has a polysymptomatic onset that usually manifests itself as a feeling of nausea which may elevate to vomiting. Measures to counteract motion sickness can be categorised as pharmacological or habituation/behavioural. Popular are the over-the-counter antiemetic compounds such as those comprising of antihistamines or anticholinergics, which can effectively prevent motion sickness. However, these drugs typically work by suppressing normal vestibular sensations or brain cortical processes, and thus commonly induce drowsiness.

Motion sickness changes brain cortical activity. Therefore, brain activity dynamics during onset should be recordable by electroencephalography (EEG). Numerous previous studies have explored which specific brain areas become activated during the experience of motion sickness using EEG. Increased mean power in delta (0.5-4 Hz) and theta (4-8 Hz) activity was reported from the temporo-frontal activity by [6] where participants underwent cross-coupled angular stimulation to provoke symptoms of motion sickness. A recent study found that participants' alpha (8-12 Hz) power from the occipital midline, parietal, and left and right motor brain areas, increased with increasing levels of motion sickness [7]. EEG power from delta at (F3, T3) and beta (12-20 Hz) at (F3, P3) increased and decreased respectively [8].

An observation worth noting is that conclusions made regarding the resultant EEG power changes from previous literature with respect to motion sickness lack consistency. The cause for this inconsistency might be due to fundamental variability in motion sickness susceptibility in individuals. Equally possibly, this might be due to the varying mechanisms used to induce motion sickness.

Vibro-motor reprocessing therapy (VRT, coined by the present authors in [9]) is a variant of a psychotherapeutic technique known as eye movement desensitization and reprocessing (EMDR). EMDR is well known for its efficacy in treating post-traumatic stress disorder (PTSD) [10]. However, treatment procedural protocols between VRT and EMDR are different with EMDR requiring lengthy repeated sessions [11]. Whereas VRT can be administered over relatively short sessions (see section "Experimental Setup and Protocol"). Effectively, EMDR works from the basis of lateralized eye movements, and VRT stems its effects through alternating palm vibro-stimulations.

This study aimed at determining whether VRT stimulation had any effect on the development of motion sickness. Objectively, the goal was to examine VRT as a potential treatment for motion sickness by studying the underpinning brain activities that are pronounced during motion sickness. We performed two sets of subjective and objective measurements in the form of Motion Sickness Assessment

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Questionnaire (MSAQ) [12] and EEG respectively. To the best of our knowledge, this is the first study to investigate VRT effects on EEG dynamics in the power spectra with the idea of alleviating motion sickness.

II. MATERIALS AND METHODS

A. Participants

The study participants were a group of eight healthy individuals (4 females, 4 males) with mean age of 29.88 and standard deviation of 12.8 years. Participants were invited only if they fulfilled the inclusion criteria. Participant cohort had perfect or corrected vision. This study received ethical approval from the University of Kent Faculty Research Ethics Advisory Group for Human Participants (ref: 0082021). All study methods adhered to the standards set by the Declaration of Helsinki. Prior to participation, participants provided written informed consent and were eligible to withdraw their participation without reprisal.

B. Experimental Setup and Protocol

A 10-minute visual stimulus developed as a compilation of four short videos was used to induce motion sickness. The complete visual stimulus was processed on a personal computer using Adobe Premiere Pro (San Jose, USA). Then, presented through a Galaxy S8 smartphone snapped into Samsung Gear Virtual Reality (Samsung Electronics Co., Ltd., Suwon, South Korea). The stimulus composed of shaky spinning-like scenes to provoke a continuous shaking and dizzy sensation on a spot to participants. In order to generate VRT stimulation, we used a Tactile Unit connected to a Boka 9 EMDR device (EMDR Equipment Europe, UK) calibrated to deliver a frequency of 0.9 Hz, Fig. 1.

The experimental protocol used in our study is shown in Fig. 2. A cross-over design experiment was conducted through two separate randomly alternated sessions identified as non-VRT and VRT per participant, performed on different days. The distinct difference in the two sessions was exposure to VRT stimulation. Both sessions had a 5-minute baseline (eyes open) task, pre MSAQ task, a 10-minute visual stimulus presentation task and a post MSAQ task.



Figure 1. The VRT stimulation unit.



Figure 2. The VRT experimental protocol.

The MSAQ uses a nine-point scale (1 = `not at all' and 9 = `severely') to assess the severity of motion sickness across 16 symptoms as a percentage: (sum of points across all items/144) x 100. The stimulus presentation task had a 10-minute duration limit; however, participants could signal for the experiment to be stopped if they begin to experience moderate nausea, to avoid progression to severe discomfort. This task highly depends on how susceptible a participant is to motion sickness. Participants seated comfortably for all study-related activities. During VRT sessions, participants rested their hands comfortably on their thighs, palms up. Then, the Tactile Unit was placed on the palms of participant to allow for a comfortable grip. EEG measurements were recorded before and during visual stimulus exposure for both sessions.

C. Data Processing and Analysis

EEG data acquisition was performed using an 8-channel BioSemi ActiveTwo system (BioSemi B. V., Amsterdam, Netherlands) at a sampling rate of 256 Hz. EEG activity was recorded at 7 scalp locations from the Frontal (F3, F4), Central (C3, C4), Parietal (Pz) and Occipital (O1, O2) sites. Additionally, common mode sense (CMS) and driven right leg (DRL) electrodes were used as reference and ground respectively. All electrodes were placed in accordance with the international 10-20 system.

The obtained BioSemi .bdf files were imported into the open-source EEGLAB [13] toolbox for conversion to MATLAB (The MathWorks, Inc., Natick, MA, USA) compatible format. EEG signal processing and analysis was performed using custom-built scripts developed in MATLAB. First, we excluded the start and end 30s windows of the baseline, and condition EEG time series to avoid task transition edges. A bandpass elliptic infinite impulse response (IIR) filter was applied at 0.5-40 Hz. In order to identify and remove eye blinks, saccades, temporal muscles and other nonbrain artifacts from the EEG data, we applied independent component analysis (ICA). ICA EEG data decomposition was performed using EEGLAB's runica function using the "extended" version; an implementation of the extended logistic infomax ICA algorithm [14]. Then, after ICA blind source decomposition, the neuronal sources obtained were back-projected to the EEG time series.

Artifact-free EEG time series of 240s baseline and 480s condition were used for further analysis. Brain regions of interest were the frontal and occipital sites. Bandpass elliptic IIR filters at the delta, theta and alpha frequency bands were applied to baseline and condition segments of these sites for examination. Baseline and condition signal segments were divided into epochs of 2 s with 50% overlap for estimating the power spectral density (PSD) using the Pwelch method. The computed power spectrum for delta, theta and alpha were baseline-normalised using condition-averaged baseline. The bandwidth-averaged spectral power percent change was computed using

$$\Delta P = 100 \, \left(\frac{P_{condition} - P_{baseline}}{P_{baseline}}\right) \tag{1}$$

where *P* is bandwidth-averaged spectral power, the subscript "condition" represents the VRT and non-VRT condition types; the subscript "baseline" represents the condition-averaged baseline before onset of the stimulus. Equation (1) represents a baseline-normalised computation for a particular frequency band i.e., delta; therefore, similar computations were performed for all other frequency bands of interest. MATLAB was used to perform all subsequent statistical analyses in which sample data was subjected to a paired *t*-test and effect sizes computed based on Cohen's d (d = 0.2, 0.5, 0.8 are considered; small, medium, large [15]).

III. RESULTS

All participants engaged in study-related activities without vomiting at the end of stimuli exposure. Participants were symptomatic i.e., reporting a score of at least 20% on the MSAQ. Our findings on the subjective measurements have already been published [9] and will not be repeated here.

Figs. 3 and 4 plot time-frequency spectrograms from a single participant during both (a) non-VRT and (b) VRT conditions at channel O1. Fig. 3 plots the spectrogram over the complete EEG time course during both conditions. Whereas Fig. 4 zooms into a 120s segment toward the end of the stimuli exposure for both conditions. We anticipate and believe that this is where the level of sickness would be greatest, since a feeling of nausea generally evolves gradually over time (with some variability between participants). In fact, onset for this particular subject is indicated by the burst of alpha power just after the 160s time marker in Fig. 3a.

The bright yellow in the spectrograms indicates higher power spectral components which are plotted over the vertical axis at each instance in time. We can thus see how the power spectrum evolves over time. This tells us the frequencies that predominate and at what point in time those frequency components start and stop. So we can see the alpha frequencies which are being suppressed when VRT reduces the experienced sickness level compared to the non-VRT plots. This occipital (O1) alpha power suppression was statistically significant (t(7) = 4.24, p = 0.0019, d = 1.5) and O2 (t(7) =2.73, p = 0.0146, d = 1.0) between non-VRT and VRT conditions. Statistical differences were also found at O1 and O2 in the delta and theta frequency bands (all p < 0.05, d >0.8); except for theta at O2 where (p = 0.0361, d = 0.7). Our findings from the occipital region were consistent with a recent study by [16] where spectral power from the analysed frequency bands increased in line with the experience of motion sickness symptoms.

Fig. 5 shows grand-averaged spectral power comparisons across participants at F3, statistically significant for delta (t(7) = 4.24, p = 0.0061, d = 1.2), theta (t(7) = 2.97, p = 0.0105, d = 1.0) and alpha (t(7) = 3.66, p = 0.0041, d = 1.3) between non-VRT and VRT conditions. Fig. 6 shows delta power changes for all participants at O1. Increased frontal spectral power in delta and theta was reported by [6] as motion sickness symptoms increased. Relative F3 delta power was found to be positively correlated with increased sickness [8].



Figure 3. EEG alpha (O1) time-frequency spectrogram of single participant for non-VRT and VRT conditions.



Figure 4. EEG alpha (O1) time-frequency spectrogram of single participant for non-VRT and VRT conditions near end of stimuli presentation.



Figure 5. Power comparisons at (F3) for VRT and non-VRT conditions for all participants, displayed as mean \pm standard error.



Figure 6. Mean delta percent changes in power at O1 during VRT and non-VRT conditions, displayed as mean \pm standard error.

IV. DISCUSSION

VRT was well tolerated by all participants with some reporting feeling no form of discomfort in contrast to no intervention. We have long known that the frontal lobe of the brain is associated with emotion [17]. This may help shed some light on why such significant difference in frontal brain activity was found in our study; we believe participants' emotional state becomes increasingly negative as their feeling of sickness (or level of autonomic arousal) increases, and they feel the urge to want to signal a stop to the exposure of the nauseagenic stimuli. Participants may be experiencing some 'friction' in their central nervous system. Researchers in [18] have reported an increase in theta activity where participants are tired but strive to remain vigilant.

Furthermore, Fig. 3 shows that the spectral power remains activated/visible over the time course of stimulus exposure for a participant under non-VRT (Fig. 3a) in contrast to VRT. This shows that the participant suffered severe sickness in the non-VRT session compared to the VRT session. We can also see that there are times in the VRT session time-frequency plot where we see occasional small bursts of power; these may be attributed to moments in the stimulus that are triggering severe sickness. These moments are then effectively alleviated by VRT exposure. This suggests that VRT might elicit positive effects once motion sickness symptoms have already started.

Hence the important possibility that, during conditions of increasingly severe motion sickness, VRT stimulation could potentially be applied in real-time to alleviate symptoms.

V. CONCLUSION

This study has demonstrated that VRT stimulation reduces motion sickness indicators obtained from EEG analysis. We previously showed that VRT alleviates subjective indicators [9]; therefore, we now combine behavioural and EEG evidence for VRT as a potential nonpharmacologic treatment for motion sickness. Notable features of VRT are noninvasiveness (with no known side effects), portability and ease of use.

Future research will examine VRT using a larger sample of

participants, other physiological measurements, and real-time intervention. Further understanding VRT mechanisms is important, as is exploring its potential to manage other sources of nausea, plus conditions such as stress and anxiety.

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