

Motion-Sickness Related Brain Areas and EEG Power Activates

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Abstract. This study investigates electroencephalographic (EEG) correlates of motion sickness in a virtual-reality based driving simulator. The driving simulator comprised an actual automobile mounted on a Stewart motion platform with six degrees of freedom, providing both visual and vestibular stimulations to induce motion-sickness in a manner that is close to that in daily life. EEG data were acquired at a sampling rate of 500 Hz using a 32-channel EEG system. The acquired EEG signals were analyzed using independent component analysis (ICA) and time-frequency analysis to assess EEG correlates of motion sickness. Subject's degree of motion-sickness was simultaneously and continuously reported using an onsite joystick, providing non-stop psychophysical references to the recorded EEG changes. Five Motion-sickness related brain processes with equivalent dipoles located in the left motor, the parietal, the right motor, the occipital and the occipital midline areas were consistently identified across all subjects. These components exhibited distinct spectral suppressions or augmentation in motion sickness. The results of this study could lead to a practical human-machine interface for noninvasive monitoring of motion sickness of drivers or passengers in real-world environments.

Keywords: EEG, ICA, motion-sickness, delta, theta, alpha, time-frequency.

1 Introduction

Motion-sickness is a common experience to everybody, and it has provoked a great deal of attentiveness in plenty of studies. The sensory conflict theory that came about in the 1970's has become the most widely accepted theorem of motion-sickness among scientists [1]. The theory proposed that the conflict between the incoming sensory inputs could induce motion-sickness. Accordingly, new research studies have appeared to tackle the issue of the vestibular function in central nervous system (CNS). In the previous human subject studies, researchers attempt to confirm the brain areas involved in the conflict in multi-modal sensory systems by means of clinical or anatomical methods. Brandt et al. demonstrated that the posterior insula in

human brain was homologous to PIVC in the monkey by evaluating vestibular functions in patients with vestibular cortex lesions [2]. In agreement with previous clinical studies, the cortical activations during caloric [3] and galvanic vestibular stimulation [4] had been studied by functional imaging technologies such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI). To overcome the temporal limitation of the two imaging modalities, some studies have investigated the vestibular information transmission in time domain, monitoring the brain dynamics induced by motion-sickness because of its high temporal resolution and portability. De Waele et al. , for example, applied current pulse stimulation to patients' vestibular nerve to generate vestibular evoked potentials [5]. In the study, thirty active scalp electrodes were used to record evoked potentials. By means of dipole imaging method, five distinct cortical areas were modeled from the recorded scalp signals. However, there is no general consensus on the motion-sickness related brain areas among the previous studies.

The EEG studies related to motion-sickness can be divided into two groups according to the types of stimuli: vestibular and visual. Vestibular stimuli were normally provided to the subjects with rotating chair [6-7], parallel swing [8], and cross-coupled angular stimulation [9] to induce motion-sickness. Theta power increases in the frontal and central areas were reported to be associated with motion-sickness induced by parallel swing [8] and rotating drum [6-7]. Chelen et al. [9] employed cross-coupled angular stimulation to induce motion-sickness symptoms and found increased delta- and theta-band power during sickness but no significant change in alpha power. Visually induced motion-sickness is also commonly studied in previous studies. Visually induced sickness can be provoked with an optokinetic drum rotating around the yaw axis. This situation can cause a compelling sense of self-motion (calledvection). Vestibular cues indicate that the body is stationary, whereas visual cues report the body is moving. Hu et al. investigated MS triggered by the viewing of an optokinetic rotating drum and found a higher net percentage increase in EEG power in the 0.5-4 Hz band at electrode sites C3 and C4 than in the baseline spectra. [10]. This study employees ahe driving simulator comprised an actual automobile mounted on a Stewart motion platform with six degrees of freedom, providing both visual and vestibular stimulations to induce motion-sickness and accompanied EEG dynamics.

2 Materials and Methods

2.1 Experimental Paradigm

Unlike the previous studies, we provided both visual and vestibular stimuli to participant through a compelling VR environment consisting of 360° projection of VR scene and a motion platform with six degree-of-freedom to induce motion-sickness. With such a setup, we expected to create motion-sickness in a manner that is close to that in daily life.. During the experiment, the subjects were asked to sit inside an actual vehicle mounted on a motion platform, with their hands holding a joystick to report their sickness level continuously. The VR scenes simulating driving in a tunnel were programmed to eliminate any possible visual distracter and shorten the depth of visual field such that motion-sickness could be easily induced. A three-section experimental protocol (shown in Fig. 1) was designed to induce motion-sickness.



Fig. 1. Experimental design of motion-sickness experiments

First, the baseline section contained a 10-minute straight road to record the subjects' baseline state. Then, a 40-minute motion-sickness section, which consisted of a long winding road, was presented to the subjects to induce motion-sickness. Finally a 15-minute rest section with a straight-road condition was displayed for the subjects to recover from their sickness. The level of sickness was continuously reported by the subjects using a joystick with continuous scale on its side. The experimental setting successfully induced motion sickness to more than 80% of subjects in this study.

2.2 Subjects

Twenty-four healthy, right-handed volunteers (15 males and 9 females, ages from 21 to 24 year-old with an average of 22.1 year-old) with no history of gastrointestinal, cardiovascular, or vestibular disorders, no drug or alcohol abuse, no current medication, and having normal or corrected-to-normal vision participated in this experiment. Among the 24 participants, four having no motion-sickness at all and one being too sick were excluded from further data analysis. Therefore, the EEG data and subjective MS level from 19 subjects were included in the further data analysis.

2.3 Data Acquisition and Analysis

Thirty two-channel EEG signals were acquired at the sampling rate of 500 Hz using NuAmps (BioLink Ltd., Australia). The acquired multi-channel EEG signals were first down sampled to 250 Hz. A high pass filter with a cut-off frequency at 1 Hz with transition band of 0.2 Hz was used to remove baseline-drifting artifacts. Then, a low-pass filter with cut-off frequency at 50 Hz with transition band of 7 Hz was applied to the signal to remove muscle artifacts and line noise. During the experiment, the sickness level was continuously reported by subject using a joystick with continuous scale, which was synchronized with the EEG signals. The sickness level was ranged from 0 to 5. The continuous sickness level instead of the traditional motion-sickness questionnaire (MSQ) [11] used in this study gave us real-time sickness level ratings without interrupting the experiment for the subjects to fill out the questionnaire. The new rating system provides continuous information of sickness level and also ensures the quality of EEG signals. A moving 100s window was used to smooth the continuous subjective sickness ratings using steps of 1s.

The acquired EEG signals were analyzed using independent component analysis (ICA) and time-frequency analysis to assess the involved brain regions/circuits during motion-sickness. Then, components with similar scalp topographies, dipole locations and power spectra from many subjects were grouped into component clusters [12]. The activations of independent components (ICs) were then correlated to the MS level to investigate the changes before, during and after motion-sickness sessions.

3 Results

3.1 Single-Subject Time-Frequency Response

The EEG dynamics related to the motion-sickness level were investigated by applying time-frequency analysis to the ICs in the five selected ICs clusters. Fig. 2 shows the time-frequency response of three ICs, the occipital, parietal and right motor components, from one of the 19 subjects. Among these ICs, the alpha power of the left occipital component (Fig. 2B) increased as the MS level increased. In addition, the alpha power changes in parietal and motor areas are also partially co-varied with MS level as shown in Fig. 2C and 2D. As a result, these three IC clusters might be most related to motion sickness as revealed by their time-frequency dynamics.

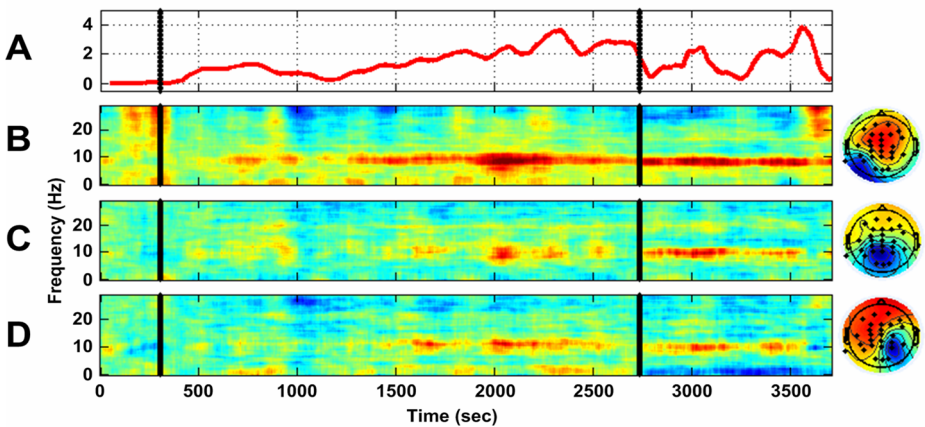


Fig. 2. Single-subject time-frequency results in occipital, parietal and right motor components. The recorded sickness level was shown as red curve in A. Three ICA components were selected as MS-related components in which their time-frequency responses were changed with sickness level. ICA power increases with the severity of motion-sickness were observed in alpha band.

3.2 Cross-Subject EEG Activities Related to MS

Brain signals can be sensitive to any environmental changes. Thus, the EEG signals acquired under different conditions may be confounded by different experimental variables. For example, when the experiment entered the winding-road section, the car began to sway left and right with the VR scene of the curved road, providing both visual and body sensation stimuli to the subjects which might induce large brain responses. As a result, simply comparing the EEG power associated with motion-sick and non-motion sick conditions may fail to dissociate such confounding effects from the main motion-sickness ones. This baseline difference among EEG power spectra associated with the different road conditions must be considered when the MS-related EEG power changes are evaluated. Therefore, the EEG power spectral changes in three periods were initially examined: (1) baseline - the first 3 minutes of the baseline straight road section, (2) low MS level - the first 3 minutes of the curved road section,

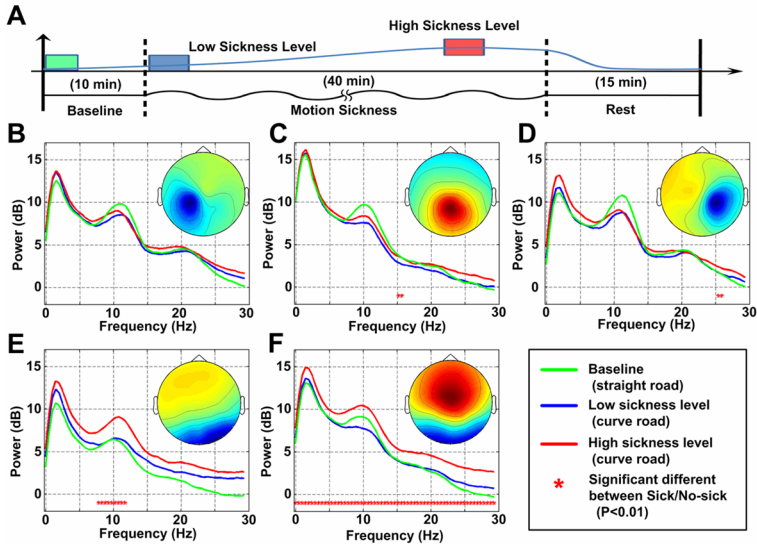


Fig. 3. Statistical comparison of IC power spectra under different conditions. The averaged ICA power spectra of baseline (straight-road / no-sick) were plotted as green lines, the power spectra of low sickness level (curve-road/ no-sick) were shown in blue, and the power spectra of low sickness level (curve-road/ sick) were shown in red. The red stars indicated significant different between sick and no-sick. A paired-sample Wilcoxon signed rank test was applied to the ICA power spectra to verify the significance of the power difference. The significant level was set at $p < 0.01$ in this research.

and (3) high MS level - the first 3 minutes after the highest sickness rating. The power spectra in these three time periods (baseline, low-sickness and high-sickness) were then averaged among subjects in each selected IC cluster. A paired Wilcoxon signed rank test was performed on the averaged ICA power spectra to evaluate the statistical significance of relationship between the power difference and both the road conditions and the motion-sickness level.

Figure 3 compares the mean component power spectra of the IC clusters for different MS levels or under various road conditions. The EEG spectral difference associated with the different road conditions can be assessed by comparing the baseline power spectra (green traces in Figs. 3B-F) and the low-MS level spectra (blue traces). Evidently, the alpha power of the right, left motor and the parietal components were suppressed from the straight-road driving to the winding-road driving as the car swayed from side to side. Additionally, significant alpha power suppression in the occipital midline IC cluster was also observed (Fig. 3F). Comparing the component power spectra under low MS level (blue traces in Fig. 3) and high MS level (red traces) revealed MS-related spectra changes. The red asterisks in Fig. 3 indicate the frequency bins where the component EEG power differed significantly between the maximum and minimum sickness levels under the same curve road condition ($p < 0.01$, Wilcoxon signed rank test). The alpha power of the occipital IC cluster (Fig. 3E) increased significantly with the MS level, whereas the occipital midline component cluster exhibited broadband spectral elevation at high MS.

3.3 Correlation between MS Level and EEG Responses

Figure 4 shows the overall correlations between the component spectra and their corresponding MS levels of the five clusters. The correlation coefficients in the alpha band exceed those in other frequency bands in all five clusters. The maximum correlation coefficient in the alpha band is 0.5 in the occipital midline components, while the correlation coefficients are approximately 0.4 in other IC clusters.

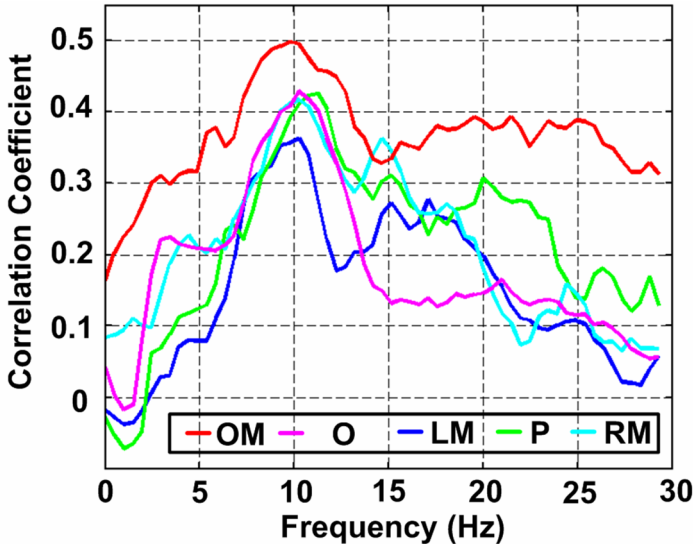


Fig. 4. Correlation between sickness level and the EEG spectra of the five component clusters

4 Discussion

The brain activities related to motion-sickness were studied here by using the technologies of EEG and the dynamic motion platform. ICA was used to separate distinct motion-sickness related EEG processes in the bilateral motor, parietal, occipital and occipital midline regions.

Alpha power suppressions correlated to motion stimuli (Fig.3 B-D) were found in parietal and the two somatosensory areas (the right and left motor areas). The power suppression can be referred to the blocking or desynchronization of central mu rhythms. This suggests that these brain areas might be influenced by vestibular inputs during the experiments. However, the frequency responses in these three brain areas were also affected by severity of motion-sickness. In Fig. 3E-F, we found alpha power increases with sickness-level, especially in the parietal lobe. The results are consistent with a gravity experiment proposed by [13]. They showed that the 10-Hz oscillations in the parieto-occipital and sensorimotor areas increased in the absence of gravity. They also suggested that since parietal lobe is situated at the conjunction between the visual and the sensorimotor cortex and thus it might be involved in integrating the multi-modal sensorimotor inputs.

Correlation analysis (Fig. 4) suggested that the power responses in the occipital midline components were highly correlated with subjective sickness-level, comparing to other brain areas. It suggests that the activations in the occipital midline, followed after sensorimotor integration in parietal and motor areas, might be used as a countermeasure to evaluate self-conscious of motion-sickness.

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