



Cybersickness provoked by head-mounted display affects cutaneous vascular tone, heart rate and reaction time



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HIGHLIGHTS

- Head-mounted virtual reality display simulating roller coaster elicited nausea
- Nausea was associated with rise in finger temperature in some subjects.
- Nausea score correlated with the prolongation of the reaction time.
- Tachycardia occurred in subjects with high nausea scores.

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ABSTRACT

Evidence from studies of provocative motion indicates that motion sickness is tightly linked to the disturbances of thermoregulation. The major aim of the current study was to determine whether provocative visual stimuli (immersion into the virtual reality simulating rides on a rollercoaster) affect skin temperature that reflects thermoregulatory cutaneous responses, and to test whether such stimuli alter cognitive functions. In 26 healthy young volunteers wearing head-mounted display (Oculus Rift), simulated rides consistently provoked vection and nausea, with a significant difference between the two versions of simulation software (Parrot Coaster and Helix). Basal finger temperature had bimodal distribution, with low-temperature group ($n = 8$) having values of 23–29 °C, and high-temperature group ($n = 18$) having values of 32–36 °C. Effects of cybersickness on finger temperature depended on the basal level of this variable: in subjects from former group it raised by 3–4 °C, while in most subjects from the latter group it either did not change or transiently reduced by 1.5–2 °C. There was no correlation between the magnitude of changes in the finger temperature and nausea score at the end of simulated ride. Provocative visual stimulation caused prolongation of simple reaction time by 20–50 ms; this increase closely correlated with the subjective rating of nausea. Lastly, in subjects who experienced pronounced nausea, heart rate was elevated. We conclude that cybersickness is associated with changes in cutaneous thermoregulatory vascular tone; this further supports the idea of a tight link between motion sickness and thermoregulation. Cybersickness-induced prolongation of reaction time raises obvious concerns regarding the safety of this technology.

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1. Introduction

Cybersickness is a subtype of motion sickness induced by an immersion into virtual reality (VR), the latter being defined as an interactive, immersive, realistic, three-dimensional, computer-simulated world [24]. Main symptoms of cybersickness are generally similar to those of

“classical” motion sickness – dizziness, nausea, cold sweating, disorientation and eye strain [7,24]. VR is not a new concept, many of the ideas associated with virtual environments were described by Sutherland [40] as a part of his Ultimate Display. Sutherland also created what is widely believed to be the first head mounted display in 1968 [41]. Despite the fact that cybersickness has been found to be a common occurrence amongst users of VR devices [7], it was, for a long time, outside the focus of the mainstream research, since such VR devices were expensive and had limited applications. New head-mounted displays, such as the Oculus Rift (Oculus VR, USA), have regenerated interest in immersive

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VR, particularly amongst the gaming community. With the recent acquisition of Oculus VR (an Oculus Rift developer) by Facebook and with the release of other head-mounted VR displays by other companies, there is little doubt that in the near future VR will spread broadly in the consumer market – not only as entertainment but likely with educational, training, communicative and professional applications. This warrants an intensification in studies of cybersickness, both in its pathophysiology and in the means of its prevention.

Neural mechanisms responsible for motion sickness in general and visually-induced motion sickness (and cybersickness) in particular are presently unknown. Their theoretical explanation is based on Reason and Brands “sensory conflict” theory, suggesting that motion sickness arises when “...the motion signals transmitted by the eyes, the vestibular system, and the nonvestibular proprioceptors are at variance with one another, and hence with what is expected on the basis of previous transactions with the spatial environment” [34]. More recently, based on Reason and Brand’s work, Bos and colleagues [4] have developed their “vertical mismatch” theory that provides advanced framework for describing and predicting visually-induced motion sickness.

Mechanistic understanding of cybersickness requires, in the first instance, its quantification. This could of course be done using subjective rating of nausea and other symptoms, but such an approach would not provide much insight into the pathogenesis of cybersickness. While subjective signs of cybersickness were initially reported more than two decades ago [35], very few studies documented objective symptoms that are associated with this condition. Cobb [8] described and quantified postural instability elicited by cybersickness. The most comprehensive study of physiological changes caused by cybersickness was conducted by Kim et al. [21] who found that its severity was correlated with gastric tachyarrhythmia, eye blink rate, heart period, and electroencephalography (EEG) delta- and beta-power. Selection of physiological variables in these and other works were based on empirical evidence provided by numerous earlier works with motion provocation. There is however a growing body of evidence suggesting that disturbances in temperature is a key element in the pathogenesis of motion sickness (see [28] for review), and thus it would be more productive to focus on relevant thermoregulatory indices. It appears that motion sickness triggers coordinated physiological response directed towards reducing body temperature – increase in heat loss via sweating [9,12,15,17,18,26,31,44], reduced thermogenesis [30], altered perception of ambient temperature and preference for cooler environment [32]. Thus it would not be unreasonable to suggest that motion sickness may also facilitate heat loss via vasodilatation in the cutaneous thermoregulatory vascular bed. Indeed, several human studies demonstrated that provocative motion attenuates skin vasoconstriction induced by immersion into cold or cool water [30,31]. Likewise, provocative motion causes sustained and dramatic vasodilation in the rat tail, leading to 2–3 °C fall in the core body temperature [29].

The peculiarity of cutaneous vascular beds involved in thermoregulation in animals (e.g. tail artery bed in rats and ear vascular bed in rabbits) is the presence of an extensive network of arterio-venous anastomoses that, when open, could allow shunting warm blood to the superficial layers of the skin, thus facilitating heat loss [36]. In humans, such anastomoses are also present, mainly in the skin of fingers, toes and face, and this allows one to suggest that if provocative motion causes vasodilation in humans, it would be most noticeable in these regions. Thus, our hypothesis was that similar to provocative motion, provocative visual stimuli would also lead to skin warming due to vasodilatation, and that these changes are more prominent in cutaneous areas with well-developed arterio-venous anastomoses. To test this hypothesis, we measured finger and forearm temperature in subjects experiencing a virtual ride on a roller coaster. We also hypothesized that changes in finger temperature would correlate with subjective rating of nausea, and thus could be used as a physiological marker of cybersickness. Our additional aim was to determine effects of cybersickness on cognitive performance as only very few

publications addressed this issue while it might represent a potential safety hazard.

2. Materials and methods

2.1. Subjects and experimental outline

The study was conducted on 26 young healthy volunteers (mean age 22.5 ± 2.2 years, range 18–30) of both genders (18 males and 8 females). They were randomly assigned to two experimental groups with equal number of males and females (9 and 4, respectively) in each group. Experimental protocol was approved by the University of Newcastle Human Research Ethics Committee. On arrival to the lab (air conditioned room kept at 21–22 °C), subjects remained rested for 10 min, signed informed consent, completed revised motion sickness susceptibility questionnaire, MSSQ [13] and performed a computer-based Deary–Liewald simple reaction time test [10], with 40 trials; in this test, subjects are asked to press a key on a computer keyboard as soon as they see a cross appearing on a computer screen. Following that, participants were fitted with a head-mounted VR display and baseline recording of finger and forearm temperatures and of heart rate were performed for 10 min. During this time the stereoscopic display presented a stationary neutral picture. Subsequently, rollercoaster simulation was activated and lasted for 14 min or until subjects requested to terminate it due to nausea sensation, whichever came first. The subjects were randomly divided into two equal groups, and each group experienced one of the two simulations (see next section). During the ride, nausea was rated by subjects every 2 min on the scale from 0 (no signs) to 10 (ready to vomit). Immediately after the end of the simulated ride, subjects performed a second reaction time test, and recording was terminated 5 min later.

2.2. Provocative visual stimuli

We employed Oculus Rift DK1 head-mounted display (Oculus Rift DK1, Oculus VR, USA), connected to a desktop computer for immersion into the virtual environment. The latter was represented by a simulated ride on a rollercoaster generated by either the ParrotCoaster (ArchiVision, Wierden, Netherlands) or Helix (Psychic Parrot Games, USA) rollercoaster simulation softwares. The two differed in the amount of visual details, such that Helix simulation subjectively looks more realistic compared to the Parrot (Fig. 1).

2.3. Data collection and analysis

Skin temperature was measured on the palmar side of the left index finger and on the dorsal side of the left forearm by means of Thermocron TCS miniature programmable data loggers (OnSolution, Sydney, Australia) that were previously validated for temperature studies in humans [43]. The loggers were attached to the skin by a hypoallergic tape. Sampling rate was 30 s; loggers were programmed and digital data were retrieved by means of USB-reader and eTemperature software (OnSolution, Sydney, Australia). Finger pulse (from the left thumb) was recorded by means of piezoelectric pulse transducer MLT-1010D connected to PowerLab-8 s and a computer running Chart 7.0 (ADInstruments, Sydney, Australia). Sampling rate was 1 kHz; heart rate was computed online from the peaks of pressure pulses.

Statistical significance for the differences in physiological measurements between Parrot and Helix groups was assessed using one-tailed unpaired t-test with Welch’s correction, with $p < 0.05$ indicating significance. Statistical differences in heart rate and reaction time pre- vs. post-exposure to a simulated ride were assessed using one-tailed paired t-test. Chi-square test was used to assess significant differences in the number of subjects who terminated their ride before the predetermined time. Pearson’s correlation with linear regression was used to

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