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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.

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

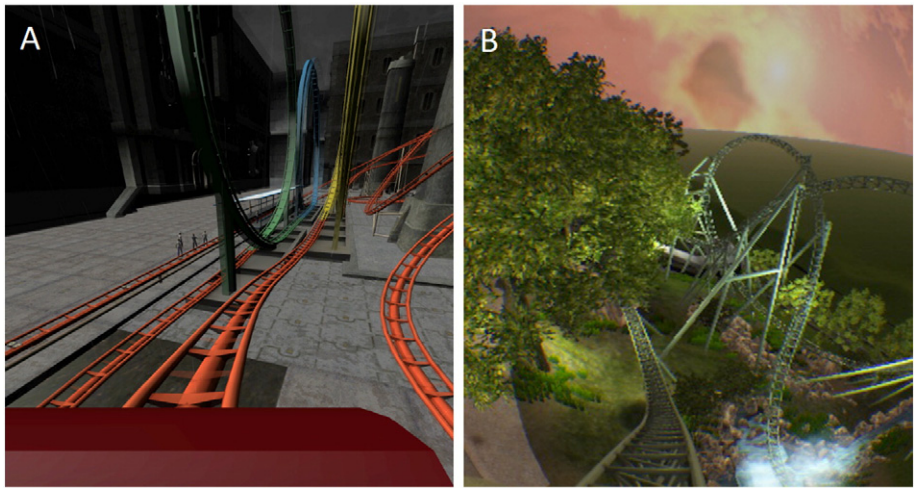

Fig. 1. Example frames from the Parrot (A) and Helix (B) simulations.

determine relationships between nausea score and changes in reaction time, and between MSSQ score and nausea rating.

3. Results

3.1. Immersion in the virtual reality exposure provoked nausea

All subjects in both groups reported vection, and the majority experienced nausea to various degree, with substantial differences between Parrot and Helix riders. Nausea score at the end of the ride was significantly higher for the Helix version vs. Parrot (6.1 ± 0.4 and 3.7 ± 0.8, respectively; $p = 0.006$; Fig. 2). A significantly higher number of Helix riders terminated their ride due to nausea (67% vs. 17% for Helix and Parrot, respectively; $X^2 = 6.2$, $p = 0.04$).

3.2. MSSQ predicts nausea occurrence during VR exposure

The mean group scores obtained from the Motion Sickness Susceptibility Questionnaire (MSSQ) were 47 ± 12 and 29 ± 4 for Parrot and Helix groups, respectively, without between-group difference ($p = 0.08$). MSSQ scores had significant positive correlation with the nausea rating for the Parrot simulation ($r = 0.66$, $p = 0.01$, Fig. 3A).

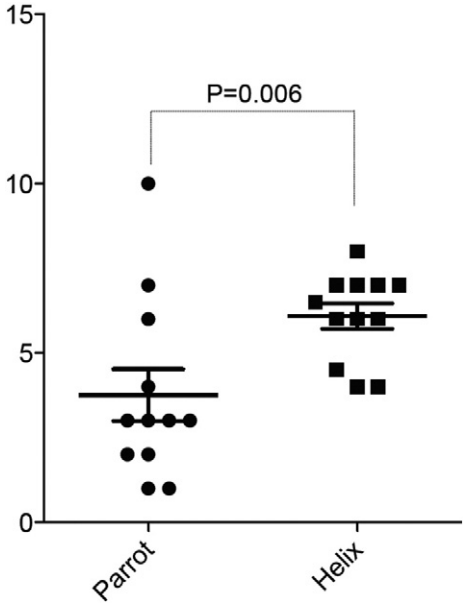

Fig. 2. Individual nausea scores in participants experiencing virtual ride on Parrot and Helix roller coaster simulators. Bars represent mean ± S.E.M.

While there was no such correlation for the Helix group ($p = 0.1$), we found a significant negative correlation between MSSQ scores and the duration of the ride, if the five subjects who completed the ride were excluded from the analysis ($r = -0.68$, $p = 0.03$, Fig. 3B).

3.3. Immersion in the virtual reality causes prolongation of simple reaction time

Pre-test, mean reaction time was 426 ± 5 and 420 ± 5 ms for Parrot and Helix groups, respectively, with no between-group differences. Two-way ANOVA revealed significant effect of time ($F = 10$, $p = 0.0045$), without effect of the ride type ($F = 0.65$, $p = 0.43$). In most participants reaction time increased by 20–50 ms, and in one (with the highest nausea rating of 10) — by 189 ms; the mean group values increased from 426 ± 5 to 456 ± 18 ms for the Parrot simulation and from 421 ± 5 to 443 ± 6 ms for the Helix simulation (Fig. 4).

In the Parrot group, where nausea rating in most participants was <5, there was no correlation between increase in reaction time and nausea rating if one outliner was excluded (Fig. 5A). In contrast, in Helix group where nausea score was higher, increase in reaction time was directly proportional to nausea rating as demonstrated by correlation analysis ($r = 0.55$, $p = 0.03$; Fig. 5B). There was also a significant correlation for the combined data set ($r = 0.5$, $p = 0.006$).

3.4. Immersion in the virtual reality affects heart rate

Prior to VR exposure, mean values for HR were 86 ± 5 and 82 ± 4 bpm for the Parrot and Helix riders, respectively, without any difference between the groups. Relationships between changes in HR and nausea rating are shown in Fig. 6A. While overall changes were quite variable, certain regularities are obvious: in all but one subjects whose nausea rating was >5, heart rate increased; on the other hand, in 6/12 subjects with mild to moderate nausea (score < 5) HR virtually did not change, in four it decreased and in two – increased. These differences in HR changes between mildly and strongly nauseated subjects was confirmed statistically, being −0.9 ± 3 and 13 ± 3 bpm, respectively ($p < 0.005$, Fig. 6B).

3.5. Immersion in the virtual reality affects skin temperature

In the results reported below, we pooled together data from both groups as the question we addressed here was how nausea affects skin temperature.

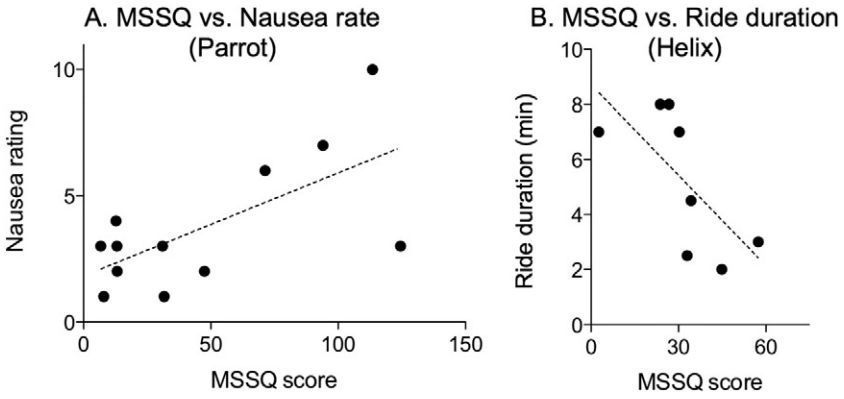

Fig. 3. Relations between motion sickness susceptibility questionnaire (MSSQ) score and nausea rating for Parrot (A) and between MSSQ score and ride duration for Helix (B).

In 26 tested subjects, values of finger temperature determined before the onset of simulated ride had bimodal distribution, with low-T group (n = 8) having values of 23–29 °C, and high-T group (n = 16) having values of 32–36 °C (Fig. 7A).

In 6 of 16 subjects from the high-T group and in 1 of 8 subject from the low-T group, finger temperature remained relatively stable during and after provocative visual stimulation (Fig. 7B, E). Nausea rating in these subjects ranged from 1 to 6, and two of them requested to terminate the test due to discomfort. Nausea rating for each subject is also shown in Fig. 7; these numbers correspond to either a time point when participants requested to terminate the ride, or to the time point of predetermined termination. In 8 of 16 subjects from the high-temperature group and in 3 of 8 subjects from low-temperature group, finger temperature changes were biphasic, with initial cooling followed by warming (Fig. 7C, F). The cooling phase started within one minute from the onset of the virtual ride and lasted for about 5 min. The magnitude of temperature fall was 1–2 °C. The magnitude of subsequent warming was group-dependent: in all but one high-temperature subjects, finger temperature returned to baseline or slightly exceeded it (Fig. 5C). In low-temperature subjects, the temperature rise was more pronounced (about 4 °C above baseline). Nausea rating in these participants ranged from 1 to 7; three of them requested to terminate the virtual ride. In 2 of 16 subjects from the high- temperature group and in 4 of 8 subjects from low- temperature group, visual provocation was associated with substantial (2–8 °C) monophasic increases in finger temperature (Fig. 7D, G). Nausea rating in these subjects ranged from 3 to 8; three of them requested to terminate the virtual ride. Lastly, in 1 of 16 subjects from high-temperature group, the provocation was associated with a large fall in finger temperature (Fig. 7D); he had the highest nausea rating of 10, and terminated the ride. Paired t-test revealed that finger temperature rise was highly significant in the low-T group (from 26.8 ± 0.6 to 30.2 ± 1.0 °C; p = 0.0028) even if the subject with no change (panel E) was included. Overall, it may be concluded that if initially fingers were cold, the most common reaction was substantial warming; this response is preceded by short cooling in those whose finger temperature is at the upper boarder of the "cold" zone. On the other hand, if fingers were warm, there was either no response, or biphasic cooling/rewarming (in majority), or monophasic warming. There was no correlation between the magnitude of changes in the finger temperature and nausea score at the end of simulated ride.

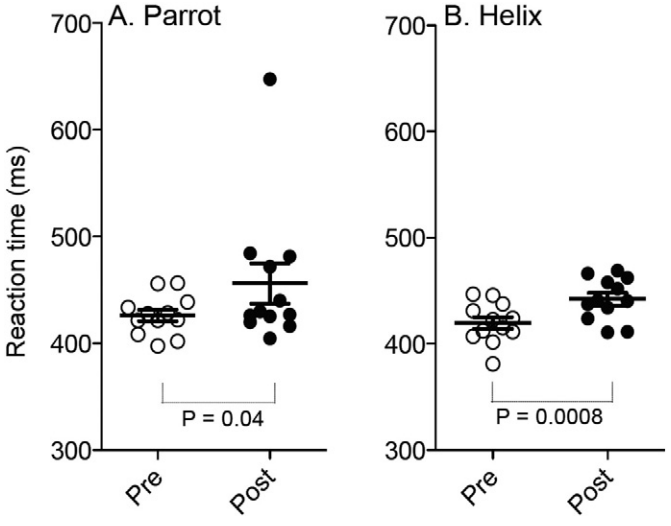

Fig. 4. Changes in the simple reaction time provoked by Parrot (A) and Helix (B) roller coaster simulations. Points are individual values; bars represent mean ± S.E.M.

The values of the forearm temperature determined before the onset of simulated ride were distributed monomodally, ranging from 28.5 to 33.3 °C (mean 31.4 ± 0.2 °C; Fig. 8A).

In the majority of subjects (15/26), there were no changes in the forearm temperature during the ride; four of those 15 subjects terminated the ride early, with nausea scores > 5. Eleven other non-responders completed the ride with nausea rating ranging from 1 to 6 (Fig. 8B). In seven participants, forearm temperature was reduced by 0.5–1.5 °C during the ride or immediately after its termination (Fig. 8C); one of these cases (bottom blue trace with nausea rating 5) could be rather classified as non-responder as the trend to a fall in temperature started before the ride started. Finally, three participants had small increases in the forearm temperature shortly after ride termination (Fig. 8D), and in one case, ride simulation temporarily slowed cooling of the forearm skin (black trace with nausea rating 10, Fig. 8D).

Table 1 shows directions of temperature changes in the two measured sites in each subject, together with their nausea ratings. As can be seen, there was no correlation in the direction of temperature changes between finger and forearm regions. There was also no correlation between absolute values of finger and forearm temperature determined just before the onset of provocative visual stimulation (r = 0.18, p > 0.05).

4. Discussion

Our major and novel finding is that cybersickness is associated with significant changes in the finger skin temperature; this confirms our hypothesis that similar to provocative motion, provocative visual stimuli also affect thermoregulatory cutaneous vascular bed. This finding is of major interest as it is in line with our recently presented concept that disturbances in thermoregulation are a key element of nausea in general and of motion sickness-related nausea in particular [28]. Furthermore, the finding is in full accord with previous reports of reduction of cold-induced finger vasoconstriction in nauseated humans [30,31]. and of dilatation in the tail (thermoregulatory) vascular bed in rodents subjected to provocative motion [11,29].

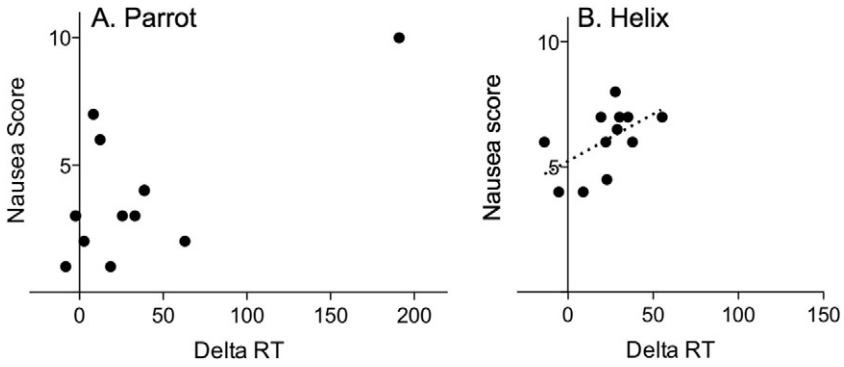

Fig. 5. Correlation between nausea rating at the end of ride and changes in the simple reaction time (Delta RT) for the Parrot (A) and Helix (B) groups. Points are individual values; bars represent mean ± S.E.M.

Distribution of finger temperature values prior to the test was clearly bimodal in our subjects. Similar bimodal distribution was found in a larger sample (>300) of healthy volunteers, with low peak located between 22 and 27 °C and high peak between 30 and 36 °C [5]. When the measures were performed several hours later, it appeared that several subjects from the "high" group moved to the "low" group and vice versa, and authors suggested that in humans finger blood flow tends to follow a "all or none" pattern rather than gradual changes [5]. While we were unable to identify any other studies where finger temperature of finger blood flow were assessed for relatively long (several hours) periods, blood flow in thermoregulatory vascular beds in animal (ear pinna artery in rabbits and tail artery in rats) is controlled in exactly this way: it remains either predominantly high or predominantly low for extended periods of time [27,45]. Mechanistically this could be explained by the likelihood that arterio-venous anastomoses abundantly present in all three vascular beds and involved in heat dissipation are either mainly opened or mainly closed. Several studies employed the difference between forearm and finger temperature as an index of vasoconstriction in the fingers (e.g. [37]). This approach however only suitable for the situations when the forearm skin temperature is constant; this was not the case in our study, and we thus based our analysis on absolute values of skin temperature.

Effects of visual provocation on finger temperature were variable in our subjects, and below we present a possible explanation for this variability. We suggest that immersion in VR can trigger two independent responses in finger vessels: i) initial vasoconstriction, a well-known component of the defence response [2], associated with arousing effects of the simulated ride; and ii) vasodilation related to cybersickness. The fact that finger cooling, if present, occurred shortly after the onset of the ride while the timing of finger warming was quite variable indirectly support our idea. We further suggest that the final integral response depends firstly on which of the two components dominates, and secondly, on the initial state of the finger vasculature. It is quite obvious that finger cooling could not occur if microvessels are already constricted, and, likewise, finger warming could not occur if they are already dilated. Indeed, in all but one subjects with low finger temperature, VR immersion caused substantial finger warming; in three of these subject whose finger temperature was in the upper-low zone, warming was preceded by cooling (Fig. 4F). On the other hand, finger warming was seen in only two subjects from the high-T group, and their initial values were at the bottom of the high zone. In contrast, the majority of the high-T group responded by initial cooling (vasoconstriction) followed by a warming (vasodilation). Contrarily to our expectations, we did not find correlation between the finger skin warming and nausea ratings; we suppose that this could be explained by the aforementioned "all or none"-like mode of operation of cutaneous arterio-venous anastomoses.

Another potential cause of highly variable temperature responses was potential contribution of sweating that accompanies motion sickness. While lack of sweating data is a limitation of our study, several arguments allow one to suggest that vasomotor responses were substantial or even dominant mechanism of changes in skin

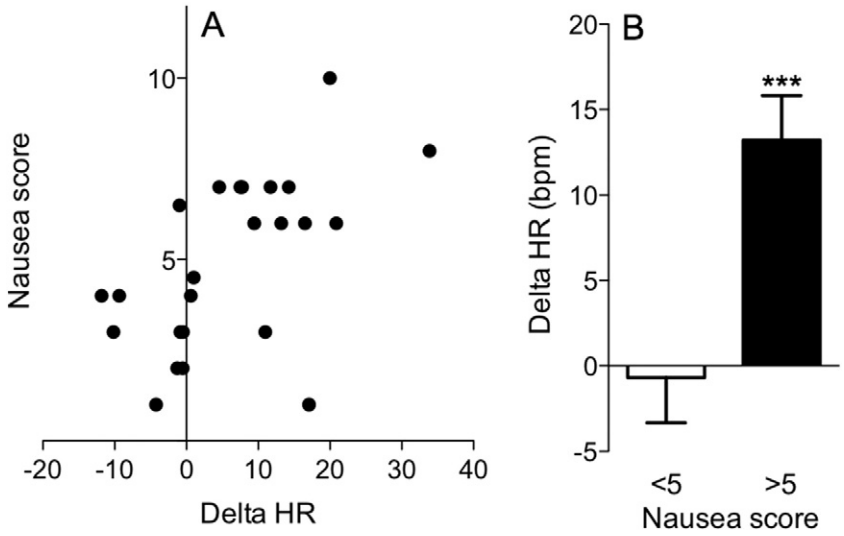

Fig. 6. Changes in heart rate during simulated ride on a rollercoaster. A — relations between delta HR and nausea score at the end of the ride (individual values); B — difference in HR changes between subjects with nausea scores of <5 vs. those with scores of >5 (mean S.E.M). *** — $p < 0.005$.

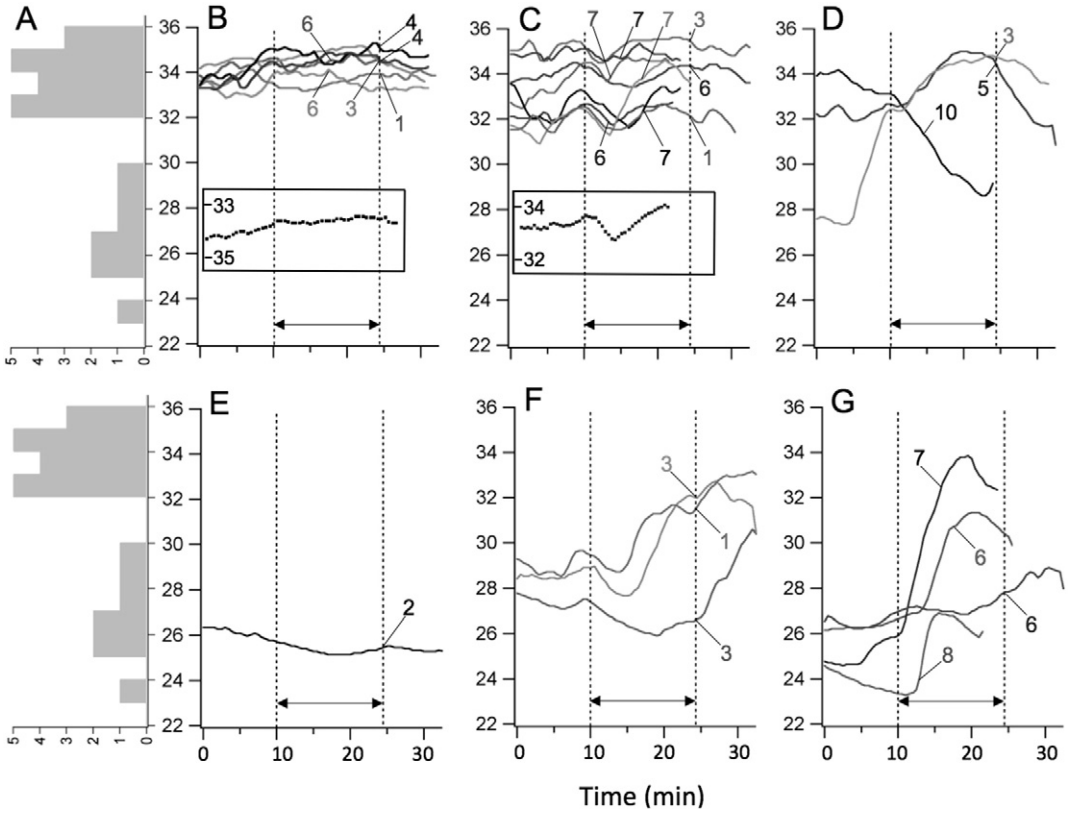

Fig. 7. Changes in the finger temperature (vertical axes) during simulated ride on a rollercoaster. Duplicated panel A shows bimodal distribution of finger temperature just before the ride. Panels B–D show individual records from subjects whose initial finger temperature was in the higher range; panels E–G — from subjects whose temperature was in the lower range. Dotted lines in the insets of panel B and C are the average of the raw traces shown there; they are shorter because averaging could be performed only for the length of the shortest trace. The first dashed line lines indicate the start virtual ride; the second dashed line indicates the end of the ride in those subject who tolerated its whole duration. Colour-matched numbers indicate the level on nausea and the moment on the data trace when the ride was terminated. Responses were classified as no effect (B & E), biphasic (cooling/warming; C & F) and monophasic (warming; D & G), with the exception of one monophasic cooling. Y-axis — skin temperature.

temperature in our experiments. Firstly, it has been previously demonstrated that skin conductance (an index of sweating) in the palmar area of the fingers is not affected by motion sickness (see Figs. 2 and 3 in [12]), and also that motion sickness is associated with increases in cutaneous blood flow in the forearm [6]. While both cited studies employed provocative motion for inducing motion sickness, we believe that their results could be quite safely extrapolated to visually-induced effects. If so, finger changes reported here might have been largely determined by the peripheral circulation. If sweating occurred in the forearms, it might have attenuated the skin warming responses, and potentially be responsible for the cooling responses in this region.

Several human and animal studies have demonstrated that motion-induced motion sickness affects thermoregulation, by triggering coordinated response aiming to reduce body temperature (see Introduction and [28] for more details). Confirming our major hypothesis, we demonstrate here that, similar to provocative motion, provocative visual stimuli also affect thermoregulatory vascular bed that could lead to an increase of heat loss. This finding is in accord with the previously reported increase in skin conductance level (a measure of sweating rate) during cybersickness [21]. Potential physiological mechanisms involved in this response are discussed in details by Nalivaiko et al. [28]; in brief, it has been proposed that any disruption of fine coordination between

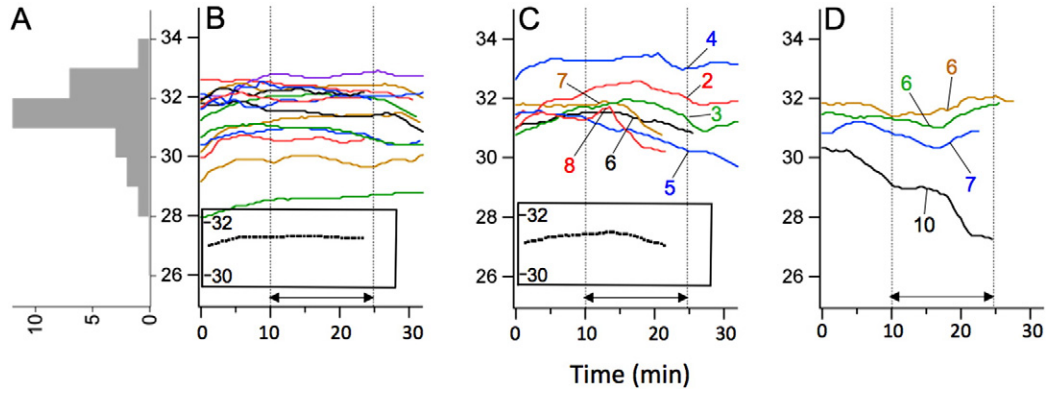

Fig. 8. Changes in the forearm temperature (vertical axes)during simulated ride on a rollercoaster. Panel A shows monomodal distribution of forearm temperature just before the ride. Dotted lines in the insets of panel B and C are the average of the raw traces shown there; they are shorter because averaging could be performed only for the length of the shortest trace The first dashed line lines indicate the start virtual ride; the second dashed line indicates the end of the ride in those subject who tolerated its whole duration. Colour-matched numbers indicate maximal nausea rating and the moment when the ride was terminated. Responses were classified as no effect (B), cooling (C) and warming (D). Y-axis — skin temperature.

Table 1
Lack of correlation between changes in finger and forearm temperature during provocative visual stimulation.

Subject no.	Finger T	Forearm T	Nausea score
1	–	–	1
2	–	Down	2
3	Up	–	2
4	–	–	3
5	–	Up	6
6	Up	–	3
7	–	Down	4
8	Up	–	3
9	Up	–	7
10	Down	Up	10
11	Up	–	1
12	Down/up	Down	3
13	–	Down	6
14	Down/up	–	7
15	Down/up	–	7
16	–	–	4
17	Up	Down	8
18	Down/up	–	7
19	Up	Down	5
20	–	–	4
21	Up	Up	6
22	Down/up	–	7
23	Down/up	–	6
24	Down/up	Down	7
25	Up	Up	7
26	Down/up	–	1

visual, vestibular and proprioceptive inputs is interpreted in the brain as a sign of intoxication, and this in turn triggers defensive hypothermia. This explains why totally different sensory stimuli, such as visual imagery or vestibular activation could lead to similar effects. Close association between motion sickness and thermoregulation provides the basis for the search of brain pathways responsible for motion sickness (and possibly the sensation of nausea in general). Indeed, sensory pathways for both visual and vestibular inputs are well determined, as are the neural circuitry for temperature control. Providing the major postulate of the "sensory conflict" ("sensory mismatch") theory of motion sickness [34], the search should be focused on the brain areas that receive inputs from both visual and vestibular afferents, and project to the areas that integrate thermoregulatory control.

To the best of our knowledge, so far there were no studies addressing potential effects of cybersickness on cognitive performance. Data on cognitive and psychomotor effects of vestibular and/or visual provocative stimulation does exist, but reported results are rather controversial. Overall, it appears that such provocations adversely affect performance in some relatively complex cognitive tasks such as Letter Cancellation Test, Digit Symbol Substitution Test [38] or a Manikin Test, a measure of spatial transformation of mental images [16], but not others (Pattern Comparison [19], Grammatical Reasoning [42] or choice reaction time [16]). This controversy is probably due to the differences in experimental protocols; it may also be that given provocation selectively affects only some particular aspects of cognition. It is also quite possible that cognitive consequences of motion sickness, even if present during provocation, quickly dissipate during the quite lengthy time required for cognitive or psychometric tests. For this reason, we limited the number of trials in our Simple Reaction Time test to 40, so that it could be completed within about 3 min. Our major finding here is that cybersickness causes small (20–50 ms) but significant prolongation in reaction time. This possibly happens because immersion into the provocative virtual environment causes spatial disorientation due to a conflict between visual and vestibular stimuli, so that a part of attentional resources are diverted to deal with this conflict — a "posture first" principle [1,16,20,23,25]. If so, one could suggest that in individuals with higher nausea rating (reflecting higher level of sensory conflict that requires more attentional resources for its compensation), prolongation of reaction time would be more pronounced. This is exactly what we have found in our study, especially with higher nausea scores in subjects who experienced the Helix simulated ride. We currently do not know how long reaction time remains affected after termination of exposure, and our initial finding warrants further investigation of this question.

It is of major interest that two VR simulations with quite similar content (ride on a rollercoaster) were so different in their provocative potential. There are several established hardware-related factors that could facilitate cybersickness (angle of view, flicker, display refresh rate, video lag); these could be safely excluded as both Parrot and Helix softwares were run on the same computer, with the same head-mounted display attached to it. It thus appears that there were some elements of visual scene imagery that were responsible for the observed differences. Some of these factors are known: for example, rotation of visual field is most provocative when it is tilted at 20° related to the horizon; the speed of angular rotation also matters [14]. Also, combined virtual pitch and roll is a more provocative visual stimulus than virtual pitch alone [3]. From the sensory mismatch theory it also follows that the provocative effect would be larger in a simulator that presents more visual evidence for linear and angular accelerations. While quantifying such parameters of visual flow is a complex task, a promising approach was suggested by [39] who suggested a new objective metrics that was called "spatial velocity". This index increased in parallel with increasing scene complexity or scene velocity, and both changes resulted in a higher incidence of cybersickness.

We found that nearly all subjects with nausea score > 5 experienced mild to moderate tachycardia. Similar findings were made in several recent studies of visually-induced motion sickness [21,22,33] and it thus appears that increase in HR is a quite consistent physiological response to this type of provocation. The underlying mechanism of this response is a combination of sympathetic activation and vagal withdrawal. It is noteworthy that cardiovascular pattern during cybersickness (tachycardia and cutaneous vasodilatation) is different from the well-known defence response (tachycardia and cutaneous vasoconstriction). It is most likely that the difference is due to the totally different physiological significance of the two responses.

Motion sickness susceptibility questionnaire is based on the past experiences; it is a reliable tool to assess and predict individual sensitivity to provocative motion [13]. There is some evidence that past history of motion sickness correlates with susceptibility to visually-induced motion sickness, and our data with Parrot riders confirms this. A lack of such correlation for Helix riders could be possibly explained by the fact that in this group there were no individuals with MSSQ score > 60 whereas in the Parrot group there were four such persons. Also, due to higher provocative potential of the Helix simulator, there was fewer low (<5) nausea ratings here, and thus MSSQ could not discriminate between high- and low-susceptible individuals as all of them became quite strongly nauseated. By contrast, MSSQ relatively well predicted how fast the Helix participants reached a level of nausea that forced them to interrupt the ride. As the majority of the Parrot riders successfully completed the ride, the "MSSQ rate/ride time" analysis was not applicable to them.

A major limitation of our study became apparent after we performed analysis of our temperature data: it is now obvious that in order to provoke substantial and consistent finger warming/vasodilatation, provocative stimuli must have been presented during the "cold" phase of vasomotor cycle. This however would require long-term (hours) control recording, and this, in turn, dismisses a promising opportunity to use finger temperature as a simple non-invasive index of nausea.

5. Conclusions

Our major finding that cybersickness is associated with changes in cutaneous thermoregulatory vascular tone further support 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. Lastly, identification of visual scene elements responsible for the generation of cybersickness is essential for the development of future VR applications; as our results show, the same content could be "wrapped" into more or less provocative virtual environment.

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