

## The relationship between postural stability and virtual environment adaptation

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Received 1 October 2007; received in revised form 12 February 2008; accepted 18 February 2008

### Abstract

Currently little is known about how adaptive responses to virtual environments are different between individuals who experience sickness related symptoms and those who do not. It is believed that sensory interactions between visually perceived self-motion and static inertial cues from vestibular and/or proprioceptive sensory systems contribute to the development of adaptation symptoms. The aim of this study was to evaluate the relationship between adaptation symptoms and postural stability in a virtual environment (VE) driving simulator. In addition, the role of sensory interaction was assessed using direct electrical stimulation techniques of the vestibular and cutaneous sensory systems. Posture performance was measured using centre of pressure measures of single leg stance tests during eyes open and eyes closed conditions. Correlation analysis of postural measures and symptom scores were conducted, as well as analysis of variance of posture performance between SICK and WELL individuals. Results indicate that posture stability is negatively correlated to symptom reporting. WELL individuals displayed the greatest decrease in postural stability during eyes open single leg stance following VE simulation. Application of a secondary sensory stimulation (vestibular or cutaneous) resulted in increased visual dependency for postural control following simulation. Combined, these results suggest that sensory interactions drive postural changes that are observed following VE simulation and are related to how visual information is used to control posture.

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**Keywords:** Virtual environment; Posture control; Stability; Simulator adaptation; Vision

Interactions between visual, vestibular and proprioceptive sensory information caused by sensory conflict can be manifested in alterations to postural stability [1]. Considerable effort has been directed towards developing posture as an objective measure of virtual environment-induced sickness symptoms [7,15]. Virtual environment technology is becoming more main stream in human research and training where typical responses and performance to particular situations (i.e. driving behaviour in driving simulation studies) are being assessed [13]. However, responses may not be natural if the participant is experiencing disorientation or nausea. Thus, these issues have great impact on the validity of VE use in the study of human behaviours.

Typical measures of adaptation symptoms are based on self-reporting questionnaires and are thus limited by their subjective nature to provide any quantitative indication of physiological derangement [6,10,15,20,21]. In fact, there is evidence to suggest that posture may play an important role in the development of disorientation and nauseous symptoms [19]. Clearly, a greater understanding of the relationship between symptoms and postural instability and sensory adaptation mechanisms to VE is needed; this knowledge will facilitate the development of objective measures to circumvent adverse reactions experienced in these environments. The overall goals of the current work were to: (1) determine the relationship between self-reported symptoms of VE adaptation as measured by a standardized motion sickness susceptibility questionnaire and measures of postural control, and (2) to assess the effects of sensory stimuli on postural instability as it relates to central adaptation of sensory systems to virtual environments.

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To date the relationship between post-exposure postural instability and sickness related symptoms is controversial [6,10]. A number of methods have been used to quantify postural instability; one of the most commonly used has been time to stance during single leg or tandem stance (i.e. sharpened Romberg). However, the sensitivity of these measures to changes in postural control is limited due to high inter- and intra-subject variability and ceiling effects. Surprisingly, little research in this area has been conducted using centre of pressure (COP) displacement. Use of a COP measurement technique to quantify postural control changes that occur following VE simulator exposure may provide a more sensitive and meaningful interpretation of the changes that occur to posture and its relation to adaptation symptoms. We hypothesized that postural instability, as measured by the specific COP measures of sway displacement and velocity, would be correlated with increased symptoms as measured by the simulator sickness questionnaire [14], potentially leading to the use of postural control as an objective (physiological) index for the effects of virtual environments.

Further, it was important to not only define the relationship between symptoms and posture but to also explore the adaptive response itself. Symptoms may result from a conflict of visual motion perception and the actual static global body position as perceived by vestibular and proprioception systems. By comparing sensory contributions to posture control following typical VE exposure (visual stimuli only) with that of a manipulated VE exposure (visual plus secondary stimuli), we hypothesized that we would be able to assess whether sensory interactions do indeed influence VE-induced postural effects and symptoms. Two techniques were used to investigate this further. Galvanic vestibular stimulation (GVS) involves application of an electrical current via electrodes placed bilaterally over the mastoid process to directly stimulate the eighth cranial nerve afferent. The resulting stimulation, which provides a vestibular perception of lateral acceleration of approximately  $2^\circ/s^2$  [9], provides dynamic inertial cues similar to those that may be experienced during curved driving in a VE driving simulator. The second technique involved the same electrodes used for GVS, but positioned approximately 3–4 cm below the mastoid process, on the cutaneous skin over the sternocleidomastoid muscle. This technique, termed galvanic cutaneous stimulation (GCS), was used to stimulate cutaneous stretch receptors in the neck providing some perception of head movement during driving.

Thirty participants volunteered for the study (12 male and 18 female; aged 18–22 years). Participants were initially screened for their involvement in the study using a general health questionnaire and the motion sickness susceptibility questionnaire (MSSQ [11]). Following the initial screening, participants were randomly assigned to one of three experimental groups. These groups included a CONTROL group who drove in the simulator without any intervention, a GVS group who received electrical stimulation of the vestibular nerve while driving, and a GCS group who received electrical stimulation cutaneously on the neck while driving. Details of the GVS and GCS stimulation follow. Ethics approval was obtained from the University of Guelph Human Research Ethics Board prior to the commencement of experimental testing.

A Drive Safety DS-600c fixed base driving simulator was used for virtual environment immersion. Image generation computers projected the virtual environment through LCD display systems onto six seven-foot projection screens that provided a  $300^\circ$  wrap-around virtual environment ( $250^\circ$  in front and  $50^\circ$  in the rear). The virtual environment simulated driving through a rural landscape on a sunny day. In each drive, participants drove 16 gradual curves (8 left and 8 right), and 16 intersection turns (8 left and 8 right); turn type and direction was randomly distributed. Vestibular or cutaneous stimulation was applied using an A395 Linear Stimulus Isolator capable of producing a current output of 10 mA. Current output was adjusted to each participant's threshold to the stimulus, assessed prior to the start of the driving trials, at a range of 0.6–1.25 mA [3]. Post-immersion symptoms of simulator adaptation were assessed using the Simulator Sickness Questionnaire (SSQ [14]), where participants rate each symptom (e.g. fullness of head, stomach awareness, eye strain) on a scale of 0–3. Total score and subscale scores were calculated as described by Kennedy et al. [14]. The current study used a total SSQ score of 15 to classify whether participants were SICK or WELL. As a score of 15 represents the 75th percentile score of individuals experiencing VR flight simulation [14], this study used scores above this percentile to represent individuals that experience moderate to severe reactions following VE immersion.

Each participant drove a twenty minute rural route (farm land environment) twice in the driving simulator (15 min break in between). Participants in the GVS and GCS group drove one of these two drives with the stimulation applied during gradual and sharp curves. Participants were asked to drive at a constant speed of 90 km/h throughout the drive (monitored every 5 min) but they could adjust their speed at curves and intersections if it felt more natural for them to do so. The order of stimulation (whether received on first or second drive) was counterbalanced across participants. After each drive, participants exited the vehicle and returned to the waiting area, where they immediately performed static posturographic tests and then wrote their responses to the SSQ. Posture tests involved a single leg stance position held for 30 s on each foot, first with eyes open (EO) and then with eyes closed (EC). Raw, three-dimensional forces (in the vertical, anteroposterior and medialateral axes) and moment data about the yaw, pitch and roll axes were digitally sampled at 150 Hz using the AMTI AccuGait Portable Force Platform (Watertown, MA, USA).

Centre of pressure was calculated from the force and moment data obtained from the force platform:

$$COP_x = (F_x \times d) - \frac{M_y}{F_z}, \quad (1)$$

$$COP_y = -(F_y \times d) + \frac{M_x}{F_z}, \quad (2)$$

where  $x$  is the displacement in the anterior–posterior plane and  $y$  is the displacement in the medial-lateral plane and  $d$  is the vertical distance between the force platform surface and the transducers. From this data, the resultant distance (RD) time

Table 1  
Correlation coefficients of simulator sickness scores with postural sway area (mm) as measured by mean displacement of the resultant centre of pressure vector

	Mean (S.D.)	Total	Nausea	Oculomotor	Disorientation
Mean (S.D.)		29.7 (35.6)	20.7 (29.5)	25.0 (24.7)	34.5 (48.6)
Pre-eyes open	8.32 (2.2)	0.057	−0.051	0.069	0.110
Pre-eyes closed	21.7 (6.9)	−0.266	−0.123	−0.251	−0.322*
Post-eyes open	9.35 (2.0)	−0.476**	−0.393*	−0.437**	−0.502**
Post-eyes closed	22.5 (9.22)	−0.121	−0.090	−0.141	−0.131

S.D.: standard deviation.

\* Correlation significant at the 0.05 level.

\*\* Correlation significant at the 0.01 level.

series was calculated as per the methods of Preto et al. [20]:

$$RD_{[n]} = [AP_{[n]}^2 + ML_{[n]}^2]^{1/2}, \quad n = 1, \dots, N. \quad (3)$$

RD is the resultant vector distance of COP<sub>x</sub> and COP<sub>y</sub> displacement and reflects the overall COP movement. An RD analysis is preferred compared to a separate analysis of the x and y components separately when foot placement is not constrained to the orientation of the force plate axis system. Not restricting foot placement results in a more natural stance and therefore facilitates measurement of a natural postural control strategy [18].

Postural stability was evaluated using a measure sway area, the mean value of the RD. This measure quantifies COP displacement from the central point of the stabilogram and represents a measure of sway stability [12,18]:

$$\text{Mean RD} = \frac{1}{N} \sum RD_{[n]} \quad (4)$$

Postural control activity was assessed using the average velocity, the total length of the RD over the first 20 s of the balance trial. The total length of the RD was calculated by summing the instantaneous RD time series distances [18]. This value was then divided by the trial duration (20 s).

In addition, to assess visual contributions to postural control, a modified Romberg's quotient was applied to mean RD and average velocity measures [16]:

$$\left[ \left( \frac{\text{EC score} - \text{EO score}}{\text{EC score} + \text{EO score}} \right) \times 100 \right] \quad (5)$$

For statistical analysis, a single tailed non-parametric (Spearman's rho) correlation was used to determine the relationship between postural stability measures and SSQ total and sub-scale scores. A single tailed analysis was used as the hypothesis was directional; postural instability would increase in proportion to SSQ score.

To determine whether exposure to a virtual driving simulator increased postural instability, a 2 × 2 mixed factorial analysis of variance was conducted with a within subjects factor of DRIVE (PRE versus POST) and a between subjects factor of GROUP (SICK versus WELL). Separate analyses were conducted for each postural measure: Mean RD and Path Velocity in each condition: eyes open, eyes closed. Post hoc Bonferroni corrected pair-wise comparisons were used for significant main effects.

To address the research question of whether a sensory conflict facilitated adaptive postural control post-VE exposure, it was important to characterize the effects that GVS or GCS stimulation had on the visual contributions to postural control. Romberg Quotient scores were transformed into a normalized measure "Effect of Stimulation" determined as [STIM − (POST−PRE)]. A univariate analysis of variance was then conducted on the transformed Romberg Quotient scores with the independent variable of STIM (CONTROL, GVS or GCS) and a covariate of GROUP (SICK versus WELL). Tukey post hoc comparisons were used to evaluate significant effects.

Post-VE postural instability was observed to increase with decreased simulator adaptation symptoms. Scores on the simulator sickness questionnaire were statistically greater in individuals who were classified as SICK. A significant negative correlation was observed between all SSQ scale scores (total, nausea, oculomotor and disorientation) and POST eyes open resultant distance mean (Table 1). These results indicate that POST VE postural sway area in fact decreased with increased severity of sickness symptoms. In addition, a significant negative correlation was also observed between PRE eyes open path velocity and SSQ Total score ( $r = -0.344$ ,  $p < 0.05$ ) and Nausea sub-score ( $r = -0.425$ ,  $p < 0.001$ ). This suggests that individuals who express a natural tendency for greater postural control activity are less likely to report severe sickness symptoms. No significant correlations were found for eyes closed postural measures, possibly due to greater inter-subject variability in this measure.

Of the 30 participants studied, 16 were classified as becoming SICK following exposure to the VE driving simulator and 13 were classified as WELL (Table 2). The SICK group reported significantly greater scores for the Total SSQ score ( $p < 0.001$ ) and Nausea ( $p < 0.001$ ), Oculomotor ( $p < 0.001$ ), and Disorientation ( $p < 0.001$ ) sub-scales.

A significant interaction between DRIVE × GROUP ( $F_{(1,27)} = 4.662$ ;  $p = 0.040$ ;  $\eta^2 = 0.147$ ) was observed for sway

Table 2  
Mean, standard deviation (in parentheses) and range (italics) of SSQ scores reported by participants that were classified as WELL and SICK

Group	Total	Nausea	Oculomotor	Disorientation
WELL	6.6 (5.5) <i>0–18.7</i>	3.49 (5.8) <i>0–19.1</i>	7.83 (6.8) <i>0–22.7</i>	5.1 (8.5) <i>0–27.8</i>
SICK	43.7 (35) <i>13.09–160.8</i>	31.3 (31.4) <i>0–133.6</i>	36.24 (22.4) <i>7.6–98.5</i>	50.5 (50.8) <i>7.0–222.7</i>

area (Mean RD) for eyes open single leg stance. WELL individuals significantly increased postural sway following VE simulation ( $p=0.007$ ) while SICK individuals did not significantly alter sway area POST drive ( $p=0.495$ ). Of note, a trend for a decreased Romberg quotient POST drive was observed. WELL individuals decreased their Romberg quotients POST drive by approximately 8%. SICK individuals decreased their Romberg quotients by approximately 5%.

A significant main effect of DRIVE was also observed for Path velocity for eyes open ( $F_{(1,27)}=24.586$ ,  $p<0.001$ ;  $\eta^2=0.477$ ). Path Velocity decreased from PRE immersion (mean = 4.6 cm/s; standard error 0.2 cm/s) to POST immersion (mean =  $3.8 \pm 0.2$  cm/s).

Vestibular and proprioception stimulation were observed to significantly affect visual contributions to postural control post-VE, as measured by the Romberg Quotient (Fig. 1). For illustrative purposes SICK and WELL groups are displayed separately. As can be observed in Fig. 1, stimulation had a greater effect on the SICK individual's postural control. A significant group effect was observed for POST-STIM sway velocity Romberg quotient ( $F_{(2,25)}=3.752$ ;  $p=0.038$ ;  $\eta^2=0.231$ ). Pair-wise comparison indicated GVS and GCS groups had a significantly greater Romberg quotient in comparison to CONTROL (Fig. 1). A trend was observed in which the Romberg quotient for eyes closed sway velocity was increased in GVS ( $6.2 \pm 15.96$ ) and GCS ( $1.7 \pm 4.78$ ) stimulation in comparison to CONTROL ( $-4.7 \pm 8.92$ ). Together these results suggest that individuals who received a secondary sensory stimulation maintained or possibly increased visual dependence for postural control post-VE immersion. No significant effect of stimulation was observed for sway area.

The overall goal of this study was to determine the relationship between postural stability and virtual environment adaptation symptoms. It has been commonly hypothesized that sickness symptoms and postural instability may proportionately increase with one another, and that increased postural instability can be used as an index of SSQ severity over and above

subjective reports [21]. However, studies have failed to find a significant correlation between post-VE immersion instability and SSQ scores. In these studies, postural measures used may not have been as sensitive as COP displacement [15] or may not have had a sufficiently strong provocative stimulus to confirm the relationship [7]. The post-exposure SSQ scores obtained from the current study (Table 2) were significantly greater than those of Cobb and Nichols [6] who reported SSQ sub-scores of nausea (11.69), oculomotor (16.49) and disorientation (17.05), and therefore may have produced greater manifestations of simulator adaptation symptoms due to a stronger VE stimulus.

An important consideration in VE adaptation is the sensory interactions that are believed to occur which ultimately drive adaptive responses to virtual environments. The Postural Instability Theory of virtual environment-induced sickness was proposed based on the relationship between visual, vestibular and proprioception systems and how these systems are cognitively integrated for spatial representation of the body and ultimately motor strategies [19]. In a virtual environment, corrective body movements for postural control are made in response to *simulated* visual motion stimuli, and are not correlated to corrections that should be made based on the actual gravitational position of the body in space. Thus, postural adjustments made in response to visual stimuli are inappropriate and instability results. It is this continuous mismatch between ongoing postural adjustments and the actual environmental state of the body that results in sickness related symptoms. Consequently, one must learn to decouple visually referenced postural control strategies from gravitational or environmental postural cues in order to regain stability and prevent motion sickness [19]. In other words, a recalibration of the relationship between sensory information for the control of corrective postural actions must occur [15]. Thus, attenuation of postural control strategies post-VE exposure may provide a measure of sensory adaptation following VE exposure that could ultimately increase our understanding of how virtual environments affect humans [15,19].

Adaptation to sensory conflict is achieved through the reweighting of sensory information in a hierarchical modification of the relative importance of each sensory system [4,8]. Virtual environments, and in particular driving simulators, create a substantial visual conflict between vestibular and proprioceptive information as the participant is seated in a static vehicle while 'driving' in a moving VE where visual information is dynamic and changes depending on the pressure placed on the gas/brake pedal (this action changes how fast one 'travels' in this environment). In this situation, vision would be interpreted as the conflicting sensory system (the other two systems do not detect any changes within the VE). The altered relationship between sensory systems would be maintained immediately after exiting the simulator, until re-adaptation to the natural environment occurred. Thus, a decrease in the visual contribution to postural control immediately following post-simulator immersion would be expected.

Indeed, significant increases in postural sway were observed in the eyes open stance condition in WELL individuals. In addition, a decrease in the Romberg quotient was observed reflecting a decrease in visual contributions to postural sway con-

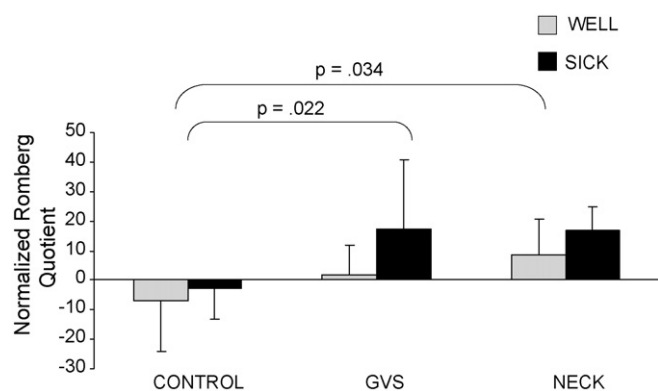


Fig. 1. Comparison of the effects of vestibular (GVS) and cutaneous (GCS) stimulation on the normalized Romberg Quotient [effect of stimulation = STIM - (POST-PRE)]. A significant increase in the visual contribution to postural control was observed as a result of GVS and GCS stimulation for all participants. WELL and SICK individuals are represented separately in the figure to illustrate that in fact SICK individuals were more greatly affected than WELL by the stimulation although not significantly so.



rol (Fig. 1: CONTROL). These observations support the above hypothesis, that the weighting of visual information for postural control is reduced as an adaptive response to the VE driving simulator. As a result, a greater postural sway during eyes open single leg stance is observed POST simulation in comparison to PRE simulation. Further, the fact that an increase in postural sway and a decreased Romberg quotient was not observed for SICK individuals implies that the above response reflects a successful adaptation to the VE. No significant differences or correlations were found in the eyes closed conditions. The variability of this measure however was considerable. It is likely that single leg stance with eyes closed presented a difficult task for all individuals and therefore was not reliable in determining differences between SICK and WELL groups.

A further interpretation of the observed increase in postural sway area is that WELL individuals were not displaying a decrease in stability per se, but rather that they used increased sway as an explorative strategy. This adaptive learning process would involve releasing the body's segments, thereby increasing sway during stance, to determine the limits with which neuromuscular control can maintain the goal of equilibrium. Infants use exploration to develop perception–action relationships for the development of postural control strategies [17]. Thus, although increasing sway area may be interpreted as a risky strategy placing a person at their limits of equilibrium it could also be considered a response which increases the robust nature of the control system. Adoption of this strategy after virtual environment immersion may facilitate the reinstatement of perception–action relationships of the natural world. It therefore may be an advantageous strategy that is representative of individuals who are successful in sensory adaptation responses.

If the natural reaction to adaptation following exposure to a VE driving simulator is to reduce the weight of visual information for spatial orientation, visual contributions to posture control should remain predominant with the application of vestibular and cutaneous stimulation (which would reduce the visual conflict). In fact, the effects of GVS and GCS stimulation were manifested in the efferent control of posture as measured by sway velocity, while postural stability itself was not significantly altered by the application of an additional sensory stimulus. As illustrated in Fig. 1 (CONTROL), and from the findings discussed above, the natural reaction to the visual conflict presented by the VE driving simulator results in a decreased weighting of visual information in postural control activity. However, when a secondary sensory stimulus was given during the simulation (GVS or GCS), visual contributions to postural control actually increased, suggesting that application of an additional sensory perception of motion reduces conflict and attenuates sensory recalibration. Similar modulations of visually induced postural changes have been observed in studies that have applied simultaneous proprioceptive stimulation [2,5]. These studies report that the secondary stimulus intensifies the effect of visual perturbations. The observation that GVS in fact had the greatest effect suggests that head velocity cues may be more influential in motion simulation.

It is interesting that the most pronounced results for GVS and GCS stimulation were observed in individuals who were SICK.

Perhaps this is an indicator that individuals more susceptible to virtual environment adaptation symptoms are more sensitive to contributions from vestibular and proprioceptive information. Unfortunately, it is unclear whether forcing sensory recalibration is advantageous to those who are more susceptible to VE sickness. Definitive conclusions as to the mechanisms that occurred to produce these results cannot be made with the present data set. However, current results do point to a direction for future research which will explore the etiology of simulator based sickness, possibly with a greater understanding for the role the sensory control of posture and its relation to the development of sickness symptoms.

## Acknowledgements

Financial support for this project was provided by the Ontario Graduate Scholarship (RRJ) and the Canadian Foundation for Innovation (LAV and LMT), Ontario Innovation Trust (LAV and LMT), and Auto21 Network Centres of Excellence (LMT). We would like to thank Dr. Leah Bent for her expertise and advice on the GVS technique and use of her equipment. We would also like to thank research assistants Lauren Meegan and Ryan Toxopeus and Drive Lab technician David Wilson for their help in testing participants.

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