Coupling Effects of Vection and Compensatory Head Sway on Simulator Sickness and Gender Difference

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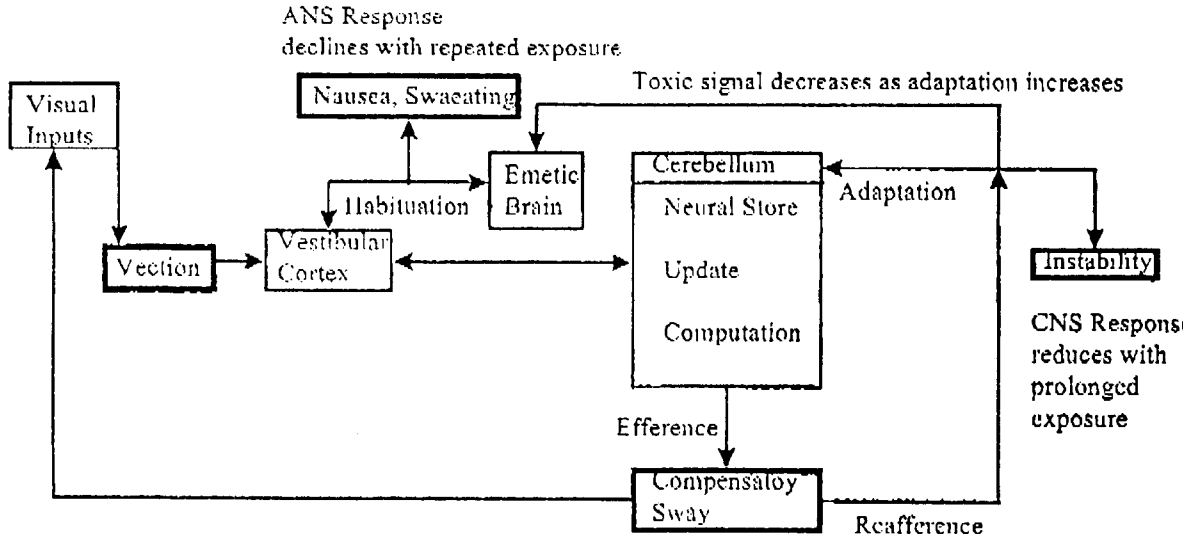
Abstract. A global model of simulator sickness is outlined that suggests the sequence of events leading to the development of simulator aftereffects. The model attempts to link coupling effects of illusory self-motion (vection) and compensatory head sway to the origin of simulator sickness. A pilot study was conducted in support of a research program that will investigate the proposed model. Seven males and four females participated in a 5-min session in a fixed-base automobile simulator. Due to restricted sample size, descriptive statistics are presented for measures of simulator sickness, lateral sway velocity (Y-velocity), driving performance, control inputs, and vection ratings. Although potential trends are discussed, no statistical conclusions can be drawn. Measurement issues for the next phase of research include increasing the sensitivity of vection ratings, and examination of the timecourse for development of compensatory sway.

1. *Introduction*

Motion sickness can occur in the absence of imposed inertial motion, when self motion is suggested by only visual stimulation [1]. Although symptoms are similar to those of motion sickness, this visually-induced motion sickness (VIMS) occurs without any vestibular stimulation. VIMS occurs in fixed-base simulators and is referred to as "Simulator Sickness." Concern for potentially negative training effects, as well as user's safety following simulator exposure, has stimulated much attention to the topic.

Research at the University of Central Florida (UCF) is addressing the etiology of simulator sickness in a fixed-base driving simulator. Our working model (Figure 1), based on the premise that simulator sickness is a form of VIMS, induced by viewing dynamic visual scenes, is as follows: Driver-generated control inputs dictate the frequency and magnitude of visual kinematics. The optical flow patterns conveyed by visual kinematics can produce illusory sensations of self-motion (vection), postural instability, and simulator sickness, although the specific relationship between these outcomes has not been empirically established. We believe that vection is a stimulus for compensatory sway in visually-based simulators, as observers attempt to maintain postural upright with respect to perceived forces of gravity implied by uniform motion of the visual field. In a fixed-base (i.e., non-moving) simulator, compensatory adjustments are not opposed by true gravito-inertial force, as would occur in the real world, when rounding a corner, for instance. From infancy, our learning experiences dictate expectations regarding the relationship between motion conditions and required strategies for maintaining postural control. When these anticipated relationships are not met, postural instability may result. According to a recent theory of motion sickness [2], postural instability is the source of motion-induced malaise, including VIMS. With respect to the proposed model, this paper presents: (1) supporting logic; (2) methodology and dependent measures from a pilot study to support research that will test the model; (3) preliminary (decriptive) data as a function of gender; and (4) lessons learned.

Figure 1. A modeling of simulator sickness



Among the most severe manifestations of VIMS are cases where users experience a strong sensation of vection [1,3]. The vection phenomenon is based primarily on the motion detection capabilities of the peripheral retina and relies upon multi-contrat objects moving at uniform velocity [1]. In a study of vection and simulator sickness, Hettinger et al., [4] concluded that vection is a necessary precondition for VIMS.

There is a strong neural linkage between the visual induction of vection and the vestibular apparatus: Specifically, the optokinetically-induced perception of self-motion is neurophysiologically-based upon visual-vestibular convergence in sub-cortical and cortical pathways and centers [5]. The primary function of the vestibular system is the transduction of linear and angular acceleration and to provide information about the orientation and movement of the head relative to forces of gravity. This information is used for subcortical control of posture and motor activity including maintaining the head in an upright position. The neural link between visual and vestibular afferents has consequences for postural control: Postural imbalance may result from misperception of the postural vertical, induced by a visual stimulus.

Helmholtz [6] was probably were the first to observe an influence of linear vection on postural balance. Lestienne, Soechting, and Berthoz [7] demonstrated that when subjects felt the sensation of forward motion through divergent horizontal motion of vertical stripes, the subjects showed a considerable tendency to fall backward. Dichgans and Brandt [1] report that the compelling sensation of body movement can affect postural balance in a vection drum and concluded that the postural imbalance may be the reaction to a misperception of postural vertical, induced by the moving visual stimulus.

Observers make compensatory postural adjustments in opposition to changes in perceived gravito-inertial force [2]. For example, when cornering a vehicle, all occupants (including the dog) lean into the turn to counteract the change in forces impinging upon the vehicle. Such postural control strategies are learned, in response to gravito-inertial force, but also corresponding information specified by the optic array. In a fixed-base simulator, changes in the optical array imply conditions that would normally be accompanied by changes in the gravito-inertial force vector in the real world, however, such forces are not present. Thus, when participants make compensatory postural adjustments, there is no gravito-inertial resistance. The lack of opposition disrupts the perception/action cycle, which

can lead to postural instability. Riccio and Stoffregen [2] propose that postural instability is the cause of motion sickness, although they do not concur that vection is a necessary precursor to postural sway or sickness.

The subjective nature of vection presents a challenging measurement issue [8]. We believe that measurement of compensatory sway DURING exposure may provide a reliable, objective measure of susceptibility to vection and a predictor of both simulator sickness and postural instability following exposure. Our pilot study, described below, was conducted to establish experimental protocol, ascertain the range and variance of dependent measures, and identify remaining measurement issues.

2. *Method*

Eleven subjects (four females and seven males) between the ages of 19 and 28 years old drove a fixed-base driver training simulator located in the Interactive Driving Simulator Lab at UCF. The simulator presents a visual roadway environment projected on a 7' (v) x 10' (h) flat screen, and the subject responds with appropriate actions involving the steering wheel, accelerator, and brake to control the vehicle's position and heading. Subjects were instructed to maintain position within the center of the driving lane at a speed of 30 miles per hour while performing a 5-min driving course that included 40 turns (20 left and 20 right turns with straight-aways inbetween). The driving course was specifically designed to induce both circular and linear vection.

Dependent Measures

A. Vection. Prior to simulator exposure, the concept of vection was described as the illusion of self-motion, and examples of vection experiences in everyday life (e.g., in cars, movie theaters and amusement park rides) were provided. Upon exiting the simulator, subjects were asked if they experienced vection at any time during their simulator exposure. Vection was treated as a dichotomous variable (i.e., yes or no).

B. Postural Stability. An automated postural stability measure was used to record lateral sway velocity before, during, and after simulator exposure. A video camera mounted on a tripod was used to record a high-contrast 3" by 3" target reticle (attached to a headband) worn on the back of the subject's head. The image of the target reticle was then processed using software developed to permit frame by frame analysis of changes in reticle displacement in the lateral (Y) axis [9]. The psychometric stability and reliability of the automated measure was validated by Kennedy and Stanney [9]. Two 30 sec trials of static standing stability (heel-to-toe, arms folded) were videotaped before and after simulator exposure. Y velocity scores for the two trials were averaged to provide a single index for each of the pre and post-exposure measures and pre/post difference scores were computed for the aggravated means, and is referred to herein as "postural instability." Seated postural sway was recorded continuously during the 5-min simulated driving task and is referred to herein as "compensatory sway."

C. Simulator Sickness. The sixteen-symptom checklist of the Simulator Sickness Questionnaire was administed pre- and post-exposure to assess sickness (see [10], for checklist and scoring procedures).

D. Driving Performance. Control inputs (number of braking inputs and number of steering wheel reversals greater than 2 degrees and 5 degrees, respectively) and performance (yaw deviation from the front center of the car to the yellow centerline of the roadway) were logged by the host computer.

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3. *Results*

One male subject requested nonparticipation following two and a half minutes of exposure due to dizziness and nausea; his data are included in the data analysis. Nine of the eleven subjects (six males and three females) reported vection experiences. Eight out of nine subjects who reported vection also reported sickness, whereas one of the two subjects who reported no vection reported sickness.

Table 1 displays the means and standard deviations (STDev) for total severity of simulator sickness (TSS), compensatory sway during simulator exposure (ComSway), postural instability following simulator exposure (pre-post difference scores [Instability]), driving performance (mean deviation from centerline [MeanDev]) and driver control inputs (number of braking inputs [#Brakes] and number of steering wheel reversals greater than 2 degrees [SWR>2] and 5 degrees[SWR>5], respectively) by gender.

Due to the small sample size, no statistical conclusions can be drawn from our pilot data, and it is not possible to make comparisons for the dependent measures in terms of "Sick" vs. "Not sick". However, there appear to be some potential trends as a function of gender that warrant discussion. As can be seen in Figure 2, males tended to report more simulator sickness symptoms, of greater severity, than did females, although there appears to be greater variability of symptoms among females. Y velocity scores for compensatory sway during simulator exposure tended to be higher for males, with twice the variability amongst subjects. It also appears that males exhibited greater post-exposure postural instability than did females, again, with much greater variability amongst scores for the males. The driving performance data (Figure 3) suggest that females performed the task better than males in terms of deviation from the centerline, and again, the males tended to exhibit greater performance variability. In terms of control inputs, it appears that females tended to make more inputs than males.

Table 1. Means and Standard Deviations for Dependent Measures as a Function of Gender

Gender	TSS	ComSway	Instability	MeanDev	#Brakes	SWR>2	SWR>5
FEMALES							
Mean	55.17	6.85	0.73	4.86	24.00	232.75	199.75
STDev	35.27	2.27	1.50	1.51	7.87	18.75	41.92
MALES							
Mean	66.78	8.95	3.12	7.89	16.00	205.86	169.00
STDev	22.08	4.56	4.34	3.71	8.68	53.68	35.93

Figure 2. Total Sickness Score (TSS), Compensatory Sway, & Post-Instability

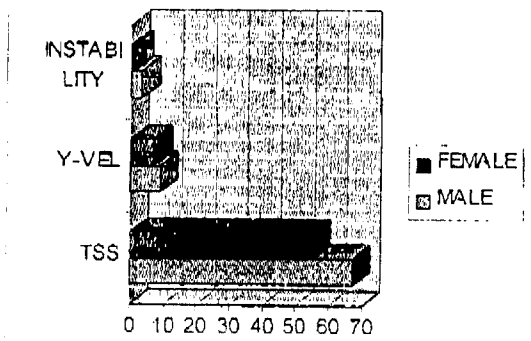
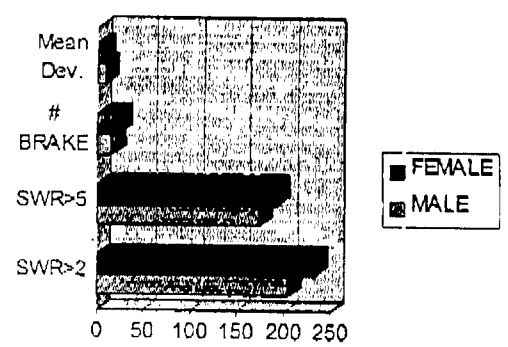


Figure 3. Indicators of Driving Performance and Control Inputs



4. *Conclusions and Discussion*

The dichotomous nature of our vection measure (i.e., yes or no) did not yield the sensitivity required to relate perceptual differences between individuals with magnitude of compensatory sway and simulator sickness. Thus, scaling issues must be addressed that will yield a sufficient range of vection scores. The fact that ten of eleven subjects reported moderate to severe symptoms of simulator sickness suggests a ceiling effect, and the stimulus intensity must be reduced (via reduced exposure duration, number of turns, or both) to permit meaningful comparisons between "sick" vs. "not-sick" in future experiments.

The tendency for males to exhibit greater compensatory sway during simulator exposure as well as greater post-exposure instability than females, is consistent with males' tendency to report greater sickness. With increased sample size, these data may support our hypothesis that compensatory sway during simulator exposure is related to post-exposure instability and reports of sickness. Athough the data suggest that females tended to make greater control inputs, it looks as though males exhibited more erratic driving performance (in terms of deviation from the centerline), which could have resulted in males' tendency toward greater compensatory sway and sickness, which again, may lend support to our hypotheses. Driving speed may influence the dependent measures and will be examined in future studies.

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