RESEARCH ARTICLE

Oculo-Vestibular Recoupling Using Galvanic Vestibular Stimulation to Mitigate Simulator Sickness

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Introduction: Despite improvement in the computational capabilities of visual displays in flight simulators, intersensory visual-vestibular conflict remains the leading cause of simulator sickness (SS). By using galvanic vestibular stimulation (GVS), the vestibular system can be synchronized with a moving visual field in order to lessen the mismatch of sensory inputs thought to result in SS. ***Methods:*** A multisite electrode array was used to deliver combinations of GVS in 21 normal subjects. Optimal electrode combinations were identified and used to establish GVS dose-response predictions for the perception of roll, pitch, and yaw. Based on these data, an algorithm was then implemented in flight simulator hardware in order to synchronize visual and GVS-induced vestibular sensations (oculo-vestibular-recoupled or OVR simulation). Subjects were then randomly exposed to flight simulation either with or without OVR simulation. A self-report SS checklist was administered to all subjects after each session. An overall SS score was calculated for each category of symptoms for both groups. ***Results:*** The analysis of GVS stimulation data yielded six unique combinations of electrode positions inducing motion perceptions in the three rotational axes. This provided the algorithm used for OVR simulation. The overall SS scores for gastrointestinal, central, and peripheral categories were 17%, 22.4%, and 20% for the Control group and 6.3%, 20%, and 8% for the OVR group, respectively. ***Conclusions:*** When virtual head signals produced by GVS are synchronized to the speed and direction of a moving visual field, manifestations of induced SS in a cockpit flight simulator are significantly reduced.

Keywords: electrical stimulation, flight simulation, cybersickness, motion sickness.

THE ABILITY TO maintain spatial orientation and balance is the result of an elaborate synchronization of neural inputs from the vestibular, visual, and proprioceptive systems. Several studies indicate that self-motion signals from the vestibular system are sent to the same brainstem nuclei that are stimulated by visually induced self-motion cues (24). Visual and vestibular self-motion systems, however, differ in response latencies to sudden stimuli (27). For moderately intense inertial stimuli (velocity changes), vestibular responses occur with latencies less than 1 s. By contrast, self-motion perception occurs with latencies on the order of seconds after scene motion onset (27).

When there is a mismatch among these signals or when input patterns from different senses do not correspond to stored expected sensory patterns, spatial disorientation may occur. The two primary conflicts occur between the visual and vestibular senses (i.e., intersensory conflict) and within the vestibular sense between the semicircular canals and otoliths (i.e., intrasensory conflict). Secondary conflict, however, may come from proprioceptive inputs that fail to synchronize with other sensory cues, particularly visual and peripheral proprioceptors connected to the vestibular system through vestibulospinal pathways (9). Sensory conflicts remain one of the most persistent issues facing advanced flight simulation development (25).

Flight simulators have been shown to improve training effectiveness with considerably lower cost and risk than actual flight training. The capability to use simulation in training brings advantages in acquisition of skill sets, development of competencies, the reduction of errors in real environments, and decreased costs. The simulation environment, however, imposes limitations in matching real world sensory experiences. These limitations can manifest in the form of simulator-induced motion sickness, also known as simulator sickness (SS), recognition of which has increased in recent decades (26).

SS is a variant of motion sickness resulting from exposure to simulated environments such as flight simulators, driving simulators, and other virtual, immersive environments. Whereas motion sickness refers to the adverse consequences of exposure to environments that physically put an individual in motion, SS is mainly the result of technological limitations in simulating dynamic environments that create a conflict in the body's self-motion perception sensors (15). Because of the wide

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variety of these symptoms, Kennedy and Fowlkes (14) describe SS as a polysymptomatic phenomenon mainly represented by nausea, oculomotor disorders, and disorientation. SS has also been described as "polygenic" since several factors have been identified, including age, gender, simulator features such as lag and field of view, and factors associated with the task performed such as duration and degree of control (18).

The theory of sensory conflict (also known as the sensory rearrangement or neural mismatch theory) remains the most widely accepted construct of SS. First proposed by Claremont (2), this theory holds that sickness occurs when the pattern of inputs from different senses and within a single sensory modality do not correspond to the stored patterns of such inputs, based on past experience, as a result of both cognitive and perceptual discrepancy. A second theory of SS (also known as ecological theory) proposed by Stoffregen and Riccio (23) states that sickness occurs in situations in which the individual does not possess or has not yet learned strategies for maintaining functionally effective postural control. The latter theory does not explain why experienced aviators are more susceptible to SS than novices. According to the sensory conflict theory, this might be the result of a conflict between the simulator motion environment and learned expectations of actual aircraft motion patterns (13). More recently, Ebenholtz et al. (7) hypothesized that motion sickness is to be understood not as a response to vestibular stimulation as such, but rather as a result of eye movements controlled by the vestibular nuclei. In their theory, the reflexive eye movements, such as those driven by the vestibular system, and perhaps also corrective saccades controlled by error-correcting feedback loops, provide eye muscle afference that ultimately stimulates vagal activity. The latter may represent the first stage in the motion sickness inducing process.

When SS symptoms develop, the value of the training experience and data derived during the experience can be compromised and, in the most extreme cases, result in negative transfer-of-training (13). Moreover, since symptoms may persist or recur spontaneously up to one day after exposure, various training centers routinely ground pilots for 6 to 24 h after simulator time (19,25). These factors can lower the acceptance and overall utility of simulator-enhanced learning.

Preventative pharmacological agents commonly used for motion sickness have been proven ineffective in consistent prevention of SS and are commonly associated with significant medication side effects after the simulated sessions, including drowsiness and fatigue. For these reasons most aeromedical centers do not recommend their routine use by aircrews (19). Simulator design, therefore, plays a significant role in decreasing the incidence of SS. However, even with technological advances, imperfections including optical deficiencies, image scale factor magnifications, system time delays, limited field-of-view displays, and head tracker inaccuracies still remain unsolved limitations which contribute to SS (6).

For all the above reasons, the mitigation of SS at its genesis, by reducing or eliminating the mismatch between sensory cue inputs expected by the users, holds the best promise to improve the overall utility of simulation-based training. The aim of this study was to evaluate whether galvanic vestibular stimulation (GVS) could be used to minimize the intersensory conflicts that are thought to provoke SS. Our hypothesis was that we might be able to reduce SS symptoms by delivering a GVS to induce vestibular sensations in register with vection sensations provoked by the visual display of the simulator. The two objectives of the study were: 1) to create a GVS dose-response model correlating the level of external electrical stimulation at multiple electrode positions with the resulting perception of movement in the three orthogonal rotational axes; and 2) to integrate GVS model data in the flight simulator program in order to combine visual and vestibular stimulation using an oculo-vestibular recoupled (OVR) simulator. Finally, a randomized study was performed during flight simulator sessions, with and without the OVR simulator, in order to investigate whether this technology has a role in mitigating SS.

METHODS

Subjects

There were 21 subjects, 15 men and 6 women, of median age 23.6 yr (range, 19-31; SD, 3.8) who were enrolled in this randomized trial approved by the Mayo Clinic Institutional Review Board to evaluate the effectiveness of GVS to mitigate SS in standard visual flight simulation. Only subjects between 18 and 55 yr of age with no history of motion sickness or vestibular disorders were recruited. A negative urine pregnancy test was required for female participants. Informed consent was obtained from all subjects prior to enrollment.

Equipment and Procedures

The Avatar framework: The first objective of the study was to correlate electrode position, direction, and magnitude of current, and motion perception within the three rotational axes (roll, pitch, and yaw). Four active 20-mm diameter disposable silver/silver chloride gel electrodes (Viasys-Care Fusion, San Diego, CA) were placed on the upper mastoid process (electrode # 1 on the left, # 3 on the right), forehead (electrode # 2), and nape of the neck (electrode # 4). A fifth electrode was affixed to the lower nape of the neck as a ground electrode (**Fig. 1**). The impedance between electrodes was measured pre- and post-testing to ensure optimal recordings (less than 4 kΩ). If the impedance was found to be higher in pretesting, the skin was prepped again until acceptable levels were obtained.

Fig. 1 shows the electrode positions in a lateral view of the head, where the simulation of motion in roll, pitch, and yaw was created by directional stimulation between electrode pairs as described in **Table I**. GVS was applied using a battery operated galvanic vestibular stimulator capable of 4-channel stimulation (Good

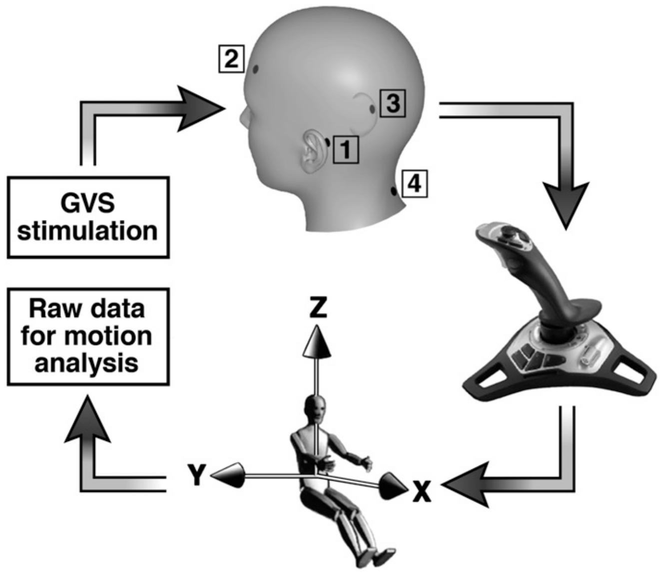

Fig. 1. Avatar framework with 3D avatar in the rest position with assigned x, y, z coordinate system in relation to roll, pitch, and yaw, respectively. The motion perception induced in the subject by GVS dose is shown, through the joystick, by the human figure rotation along one of the corresponding axes that generates raw data in the form of 3D rotational angles. The numbers around the head at the top of the figure show the positions of the electrodes.

Vibrations Engineering Ltd., Toronto, Ontario, Canada), with each electrode channel able to produce current between 2.5 and −2.5 mA in increments of 0.0195 mA. All five electrode pairs were tested with stimulation current levels at −2.5, −2.0, −1.5, 1.5, 2.0, and 2.5 mA for 20 s in a randomized protocol.

Using a 3° of freedom wireless joystick (Logitech® Freedom™ 2.4), each subject was trained to display motions by manipulating the joystick sideways left/right (roll), forward/backward (pitch), and twist counterclockwise/clockwise (yaw), respectively. During GVS experiments, perceived motion inputs were recorded by a custom software program named Avatar (developed by Infoscitex Corporation, Waltham, MA), which also displayed a corresponding animated 3D human on a computer screen visible to the test administrator during testing. Fig. 1 shows the Avatar framework with the 3D Avatar in the starting position with the assigned x, y, z coordinate system in relation to roll, pitch, and yaw, respectively. By monitoring Avatar movements on the screen during testing, the administrator could visualize in real time how the subject perceived sensations of motion resulting from GVS.

TABLE I. MOTION SIMULATION VIA DIRECTIONAL STIMULATION.

No.	Direction of Current (Electrode No.)	Motion Perception, Major Axis
1	1 to 3	Yaw Right
2	3 to 1	Yaw Left
3	1 to 4 or 4 to 3	Roll Left
4	4 to 1 or 3 to 4	Roll Right
5	3 to 2 or 1 to 2	Pitch Forward
6	2 to 3 or 2 to 1	Pitch Backward

The OVR simulator: The second objective of the study was to develop and integrate the GVS model into a specially designed simulator program in order to combine visual and vestibular stimulation. For this task OVR simulation control software was written by Infoscitex Corporation. To induce GVS sensation in real time, GVS was wirelessly interfaced to the helicopter flight simulator hardware (Rotor Wing Hardware, Flight Link Inc. Aviation Training Devices, Chico, CA) via an RS-232 serial Bluetooth connection.

The flight simulator controls included a joystick for pitch and roll, a throttle for speed, and foot pedals for yaw. The joystick transmitted a stream of ascii characters to the computer via Bluetooth. These were interpreted by our custom software and translated into desired electrical stimuli and visual field changes. A second Bluetooth interface was used to transmit the desired electrical stimuli to the vestibular stimulation hardware and the visual field output was hard wired to an Epson MovieMate 72 projector. The total delay from joystick to stimulation was less than 100 ms. This delay lies in the range of the physiologic latency of the visual system, that is, 30-120 ms (10,12). Therefore, the net difference in latency between the vestibular stimulus and visual processing is minimized.

Visual input was projected onto a cylindrical screen in front of the subject spanning 180°. After positioning the electrodes, each subject was seated in the flight simulator chair. Subjects were trained to move the controls in all axes and for the first 5 min, the visual field responded as expected to control inputs. After this initial 5-min period, for the remainder of the test (15 min), input from the throttle was automatically modified by the control software to superimpose a 4-Hz sinusoid with amplitude range 0.5 to 5°. In this way, after the acclimation period a constant rotation at $30° \cdot s^{-1}$, ranging from $30*0.5 = 15° \cdot s^{-1}$ to $30*5 = 150° \cdot s^{-1}$ was introduced to cause all subjects some level of simulator sickness.

Before the simulator session, the subjects were randomly assigned to two parallel arms: 1) sham-GVS (control group), and 2) actual-GVS (OVR group). Each subject was tested in only one arm. In the experimental modality, a maximum stimulation of 2.5 mA per electrode pair was synchronously administered along with the visual stimulation throughout the test. In the control modality a small amount of positive current was applied bilaterally to the mastoid electrodes (1 mA constant throughout the test) to achieve a mild cutaneous sensation of GVS (tingling), but without any resulting motion perception. Current outputs were verified by amp meter for all subjects in all runs. Overall, each session lasted approximately 30 min (10 min of experimental setup and 20 min of actual simulation).

Statistical Analysis

The output Avatar data were stored as log files, allowing investigators to visualize and quantify direction and magnitude of pitch, roll, and yaw induced by GVS for each of the electrode pairs. A modified standardized simulator sickness questionnaire (SSQ) was administered to all subjects immediately after completion of the session (16). The SSQ included a self-report checklist of eight symptoms, with subjects rating symptoms on a 10-point Likert-type scale, with higher numbers indicating greater symptom intensity. The responses were then further categorized in gastrointestinal, central, and peripheral categories following the multiple-dimensions motion sickness assessment classification (11).

An overall SS score was calculated for each category across all subjects using a cumulative sum of the points of all category-specific symptoms rated by all subjects using the Likert-type scale. This entire cumulative sum was then scaled by the total number of symptoms in the category, total number of subjects, and maximum value on the Likert-type scale. The percentage of this scaled ratio was considered as the overall SS score of the category. Mathematically, we can express this as follows:

$$\text{Overall SS Score for Category C} = \frac{S}{t * n * m} \times 100\%$$

where S = sum of points from all symptoms in the category (C) for all subjects; t = total number of symptoms in the category (C); n = total number of subjects; and m = maximum value on the scale (e.g., in 10-point Likert scale; m = 10). We performed inferential statistical analysis tests to show significant difference between the Control group and the OVR group, which includes the nonparametric Kruskal-Wallis test and a one-way ANOVA. In addition, we subsequently evaluated our sample size using Cohen's effect size criteria (4).

RESULTS

The GVS dose response was determined by analyzing the stored log files which recorded the angular displacement for all test conditions. From this analysis, six unique combinations of GVS electrode positions could be categorized (Table I). The majority of subjects reported distinct sensations of movement in response to a 2.5-mA GVS pulse. Overall, during the 10 conditions tested, 17/21 (81%) subjects reported pitch, 19/21 (90%) reported yaw, and 17/21 (81%) reported roll motion perceptions. Additionally, it was possible to visualize and quantify direction and magnitude of pitch, roll, and yaw induced by GVS in each electrode pair.

Fig. 2 shows a typical data plot obtained from the dose response tests. Each curve represents the perceived rotations between electrodes 1 and 3 for each axis (x, y, z) over a stimulation period of 20 s. For this pair of electrodes (1 to 3), there was minimal perceived rotation about the x axis, but substantial excursion for the y and z axes. In all cases, the perceived rotation R was zero for no stimulation S.

The relationship between the perceived rotations about the three axes (R_x, R_y, and R_z) and the stimulation was obtained by performing a linear curve fit through these data plots. These plots yielded nine equations of the type $R_{\{x,y,z\}} = W_i\ S_{\{13,321,143\}}$, where the weights W were the slopes of the curves. With the assumption that

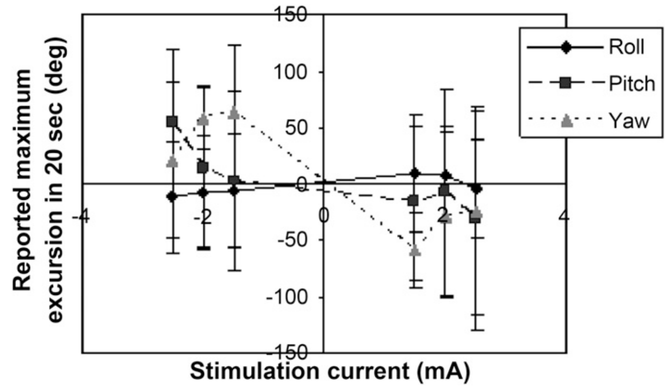

Fig. 2. Relationship between stimulation current in one direction and perceived rotations about each axis. The data shown is for rotations between electrodes 1 and 3.

the responses R were linear to the stimulations S, we formulated the following three sets of stimulation equations:

$$R_x = -0.27S_{13} - 0.07S_{321} + 0.16S_{143},$$

$$R_y = -5.18S_{13} + 4.60S_{321} - 4.76S_{143} \quad R = f(S), \text{ and}$$

$$R_z = -13.4S_{13} + 5.21S_{321} - 9.06S_{143}.$$

Note that in these equations, the values R are for perceived rotations in degrees per second and the values S are stimulations in mA. The key step in the development of the OVR simulator was to establish relationships that showed how the stimulations S were a function of the desired rotations R. Mathematically, this means solving the above equation R = f(S) for S, such that S = f'(R). The capability to invert the equations provides the necessary stimulation pattern to evoke perceived rotations in yaw, pitch, and roll. The inversion of this relationship provided the following equations:

$$S_{13} = -1.92R_x + 0.02R_y - 0.04R_z,$$

$$S_{321} = 1.91R_x + 0.51R_y - 0.24R_z \quad S = f'(R), \text{ and}$$

$$S_{143} = 3.93R_x + 0.27R_y - 0.18R_z.$$

The three equations that show the required stimulations S as a function of the desired rotations R are precisely the inputs needed in order to drive vestibular stimulation with a flight simulator. Since stimulating the vestibular system in the three directions yielded different combinations of roll, pitch, and yaw, we isolated each perceived rotation by using a combination of stimulations.

There were 21 subjects (11 for the Control group, 10 for the OVR group) who completed the simulator session.

A total of 21 SSQs were included in the analysis. In 20 of these, at least 1 (9%) or more symptoms (86%) of SS were reported. Only one (5%) did not report any symptoms. **Table II** shows the SS symptoms and scores reported for each group, where *N* is the number of events reported by the subjects for each symptom.

The number of total events reported was 43 for the Control group and 36 for the OVR group. Nonparametric statistical tests were used given the small sample size ($N = 11$ for the Control group vs. $N = 10$ for the OVR group). The sum of all symptoms' scores in the Gastrointestinal category for the OVR group were minimally lower than the Control group (19 vs. 56, $P = 0.04$, using Kruskal-Wallis one-way ANOVA). The overall normalized SS score reported was 160 for the Control group and 95.5 for the OVR group. The average observed scores for each symptom comparing Control and OVR groups are shown in **Fig. 3**. The mean score and SD values (error bars) obtained for each symptom showed a relevant decrease of severity of most symptoms for the OVR group.

The mean score of the symptom "Nausea" for the OVR group was less than the Control group (1.3 vs. 3.7) with a statistically significant difference ($P = 0.03$) using one-way ANOVA. Also, according to Cohen's conventional effect size criteria (4), the effect size value was $d = 0.84$, representing a large statistical and clinical difference between the OVR and Control groups for the symptom "Nausea." The mean score of the symptom "Dizziness" for the OVR group was less than the Control group (1.9 vs. 3.7), with a minimal statistically significant difference ($P = 0.06$ using one-way ANOVA); however, effect size value was $d = 0.46$, representing a medium statistical and clinical difference between the two groups.

An overall SS score was calculated for each category for both groups. The overall SS scores for the gastrointestinal, central, and peripheral categories were 17%, 22.4%, and 20% for the Control group and 6.3%, 20%, and 8% for the OVR group, respectively. The comparative analysis for the overall SS score between the Control and OVR groups showed a preponderance of symptom intensity in all categories for the Control group, with a statistical significance (**Fig. 4**). The normalized scores of the symptoms in the Peripheral category for the OVR group were significantly lower than the Control group (7 vs. 17 for Sweat, and 9 vs. 23 for Warmth; on analyzing together $P = 0.06$ using one-way ANOVA).

DISCUSSION

Spatial orientation is the result of complex neural activity requiring kinematic information from the head, including orientation, velocity, and acceleration (linear and angular) relative to the global coordinate frame of the external space. The vestibular system sends constant rotational (via semicircular canals) and translational (via the otoliths) information regarding the orientation of the head relative to the outside world to higher processing centers. This information converges with visual and proprioceptive signals at the brainstem vestibular nuclei, and from there ascends to multisensory neurons of the parietoinsular vestibular cortex to form the perception of spatial orientation of the head (5). According to the most widely accepted theory of the sensory conflict, spatial disorientation and SS might result from conflicts between the sensory input patterns involved in the detection of self-motion and actual motion in space (2). Nevertheless, further studies need to be done before the underlying mechanism of motion sickness is fully understood.

Despite sophisticated novel visual displays and improved computational capabilities of flight simulators, inter-sensory visual-vestibular conflict remains the leading cause of SS, with an incidence rate of 68% following simulator exposure (13). This is especially relevant in simulators that do not utilize a motion component. Therefore, the development of a fixed-base flight simulation model able to synchronously stimulate the sensory systems involved in movement perception may have a promising role in mitigating SS. In the OVR simulator described here, the simulation of motion in roll, pitch, and yaw was created by directional galvanic stimulation between electrode pairs.

GVS has been proven to act on the primary irregular vestibular afferents by modulating the hyperpolarization of the neuroepithelia of the cristae and maculae, bypassing the transduction mechanism of the hair cells, and stimulating the axons directly (17). Anodal and cathodal currents selectively decrease and increase

TABLE II. SS SYMPTOMS AND SCORES REPORTED FOR EACH GROUP.

		Control (*N* = 11)		OVR (*N* = 10)	
Category	**Symptoms**	***N***	**Normalized Score**	***N***	**Normalized Score**
Gastrointestinal	Nausea	8	38	3	13
	Salivation	5	10	3	6
	Vomit	1	4	0	0
Central	Headache	4	14	7	13
	Dizziness	9	37	8	19.5
	Drowsiness	6	17	8	28
Peripheral	Sweating	4	17	3	7
	Warmth	6	23	4	9
TOTAL		**43**	**160**	**36**	**95.5**

N = Number of times the symptom was reported.

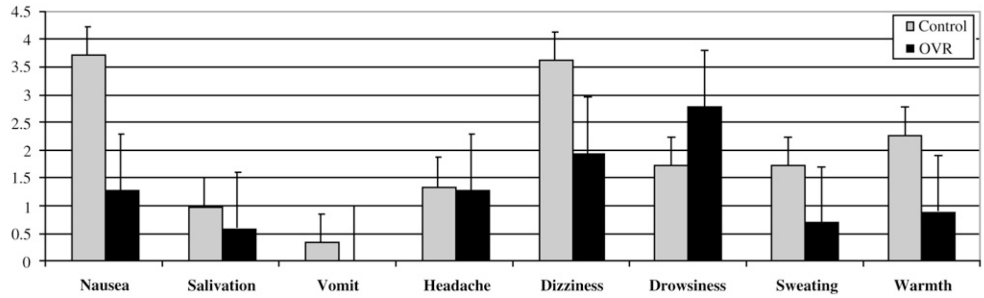

Fig. 3. SS mean score and SD (error bars) for each symptom for both groups.

the firing rate of the afferents that cause standing subjects to sway toward the anodal stimulus and/or away from the cathodal stimulus (3). In the bilateral bipolar configuration proposed by Fitzpatrick (8), cathodal stimulation of the horizontal canal results in a yaw perception toward the ipsilateral or cathodal side and in an ipsilateral ear-down roll for the vertical (anterior and posterior) canals. Anterior and posterior canals also result in nose-down and nose-up pitch, respectively, in relation to their 45° alignment to the skull sagittal axis. Because these opposing signals are equal in size and opposite in direction, and affecting all the three canals, however, the patterns of afferent discharge evoked by GVS result in both yaw and roll components, the latter being larger because of the vector addition from both anterior and posterior vertical canals. GVS has also been shown to independently modulate the firing rates of the otolith system as shown by short latency reflex and small transient sway measurement in the control of balance (1).

In addition to conventional mastoid GVS electrode placement, a differential effect in rotational perception results from electrodes placed at additional positions around the head (M. Cevette; unpublished data; 2007). These preliminary data served as the basis for establishing a dose-dependent motion perceptual response from normal subjects for each of the three axes of rotation. Using the Avatar framework enabled categorization of six combinations of electrode arrays during GVS to selectively evoke the rotational perceptions dominated by roll, pitch, or yaw.

Although the effect of GVS in modulating the vestibular system is widely recognized, its use during flight training programs remains quite limited. Malcik (20) was first to implement the GVS in the flight simulator programs in order to evoke flight illusions during task performances. He concluded that GVS may reduce the tendency of aviators to underestimate disorienting sensations and promote the adaptations during discrepancy between motion perceptions and instrument readings (20).

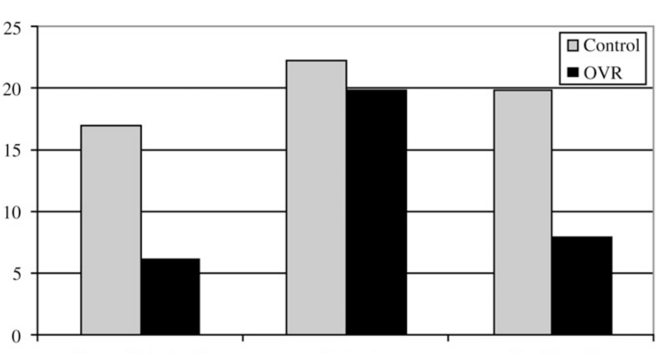

Fig. 4. Overall SS scores between categories for both groups.

GVS has also been used to replicate postflight sensorimotor disturbances commonly experienced by astronauts after returning to Earth's gravity. Moore et al. (21) showed the efficacy of random GVS in replicating postflight imbalance, ataxic gait, reduced visual acuity, and impaired manual control. Based on these results GVS has been proposed as a potential tool to familiarize astronauts with the microgravity effects on the vestibular system (21).

The approach of the current experiments was not to randomly disrupt the vestibular system, but to generate GVS stimuli in controlled patterns synchronized with a moving visual field to mitigate sensory conflict. Despite an established role in evoking motion perceptions, the effect of GVS on vestibuloautonomic symptoms such as nausea and vomiting has not been adequately reported. Park et al. (22) first reported that cathodal stimulation (1-3 mA) of the mastoid process, ipsilateral to cold water irrigation during caloric stimulation, restored normal patterns of gastric motility and abolished caloric stimulation-induced nystagmus. Nausea, vomiting, and dizziness were also ameliorated by GVS (22).

The role of GVS on mitigation of vestibuloautonomic symptoms during flight simulator training programs, however, has never been reported. In the present study implementation of GVS in a visual flight simulator has been shown to mitigate all SS categories, with the most notable results for symptoms of nausea, vomiting, and dizziness. Drowsiness during the session was not anticipated to be greater in the OVR group. This finding suggests the relative strength of the GVS in activating vestibular pathways. The result could represent an involuntary reflex to inhibit SS similar to the adaptive mechanism seen in motion sick infants who involuntarily become drowsy. Based on these results, the following is proposed: implementation of GVS in fixed-base flight simulator training by using the OVR stimulation paradigm in order to overcome the intersensory conflict resulting from the vestibulo-visual mismatch and the resulting symptoms that negatively impact performance and training quality.

OVR simulation has multiple potential applications, including: 1) safe and cost effective testing of vestibular responses in candidate pilots; and 2) improvement of pilot and astronaut training methodologies. For example, the simulation of rotor failure in modern flight simulators does not adequately provide the powerful corresponding vestibular sensation of rotation in the direction of the turn. OVR simulation, however, can induce roll, pitch, and yaw to match the visual input used for simulation. In this way, one may provide a more realistic simulation and better prepare pilots for this type of flight scenario. The OVR model may translate to other simulation scenarios in which head position is in conflict with a moving virtual reality environment that may provoke cybersickness. Given the influence of GVS on both the semicircular canal and otolithic systems, the present technique also has the potential to mitigate seasickness as well as other otolithic related dysfunctions.

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