STUDY OF SIMULATOR SICKNESS AND THE VOR WITH RESPECT TO VR

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Abstract:

This work investigates the cause and consequences of simulator sickness induced by exposure to virtual reality (VR). The cause of simulator sickness has been attributed to conflicts in the vestibulo-ocular reflex (VOR). Retinal images are stabilized by the VOR, which generates smooth eye movements that are equal in amplitude and opposite in direction to head turns (Lisberger, Pavelko, 1988). Another concern raised by simulator sickness is whether it is an indication that the VOR is being altered by prolonged exposure to VR. Simulator sickness in itself is not an indication that there are more severe consequences of exposure to VR. Some researchers argue that brain damage occurs when new neural pathways emerge in the brain to compensate for response errors in VR systems. Brain damage according to theories regarding growth of neural pathways can occur only when there is motor learning, when the VOR adapts to the artificial environment of VR and its limitations. Learning occurs only when there is head movement and simultaneous retinal image motion (Lisberger, 1988). In VR, there is minimal eye movement, since the field of view is limited to what is immediately in front of the eyes of the user. Though the VOR has to maintain the steady position of the eye as the head turns, it is the assertion of this paper that since it is a regular VOR function, it does not require much learning to perform the same function in a VR environment. Furthermore, experiments show there is less learning when the eye is fixed on a point turning with the head than there is when there is separate eye and head movement. Based on these premises it is postulated that minimum learning occurs. Simulator sickness can occur where there is a null signal to the vestibular apparatus while there is visual stimulation, if a user's head remains stationary while she is immersed in VR, she may experience discomfort without the potential threat of corrupting her VOR. Thus in VR systems where there is minimal eye and head movement, there should be a minimum incidence of simulator sickness, because the vestibular apparatus and the ocular muscles receive little stimulation. Our experiments corroborate this theory. There are many factors, such as age, that contribute to the risk and rate of adaptation and then re-adaptation once the user returns to reality. This study examines the research available on simulator sickness and the VOR, presents the results of the experiments done here, and discusses the potential solutions and the future of VR.

Introduction:

Virtual Reality has already penetrated the commercial market, and has been implemented in fields ranging from games to "cybertherapy." As VR reaches the mass market, it is inevitable that consumers will begin to report conditions of discomfort to the manufacturers. It is the responsibility of the companies making VR systems to provide a safe product for the consumers, legal and ethical concerns will therefore emerge with the market penetration by these products.

This report provides a thorough examination of simulator sickness and the VOR through investigation and experiment. The causes and symptoms are presented and analyzed, and the results of experiments done here and elsewhere are presented in order to put the related theories into perspective. Finally, the potential solutions and improvements to VR are discussed.

Causes of Simulator Sickness:

The examination of the causes of simulator sickness is divided into two parts, the limitations of the present technology in VR and the responses of the human body. Though highly sophisticated VR systems have already been developed, there continues to exist a discrepancy between what is rendered to look realistic and reality itself. Simulator sickness occurs when the VOR cannot compensate for the discrepancy.

Visual-proprioceptive conflicts that cause simulator sickness are: conflicts between the visually represented hand or limb and the felt position of the hand or limb of the user, lags in updating body position (view of visual world dragging behind movement of the head), and jittering or oscillation of the representation of the body parts (Biocca, 1992). Distorted graphics, image flicker, and off-axis viewing also contribute to the problem (Kennedy, Fowlkes, 1990), as well as poor calibration of the system. The larger field of view of VR is yet another contributing factor. Among the most severe manifestations of simulator sickness are cases where the user experiences a strong sensation of self-motion, called vection (Hettinger, Riccio, 1992, Dichgans, Brandt, 1978). Other contributing factors are the weight of the head mounted display (HMD) which alters the users center of balance (DiZio, Lackner, 1992), continuously varying rearrangement of the visual field which disrupts visuomotor adaptation, and perceptual adaptation disrupted by delayed feedback. One consequence of this is that the frequent user of the VR system develops certain movements to lessen these effects, which may not be appropriate for the same experience in reality. Sensory conflict theory states that the visual and vestibular senses are receiving conflicting inputs. It is the primary theory used to explain why simulator sickness occurs, and will be thoroughly examined once all the contributing factors have been presented (Parker, Harm, 1992, DiZio, Lackner, 1992).

The susceptibility of the user to simulator sickness as well as the adaptation of the VOR depends not only on the VR system but also on the biology of the individual. Simulator sickness shares symptoms with motion sickness, about which extensive research has been done. Susceptibility to motion sickness has been divided into age groups and sex. Because motion sickness depends directly on the condition of the VOR, it is reasonable to generalize susceptibility to the symptoms of simulator sickness to those of motion sickness. The sensitivity of the VOR depends on the plasticity of the brain which decreases with age. In the experiments performed here each relevant age group was tested in order to assert the validity of this claim. Research shows that under the age of two no motion sickness is experienced, between the ages 2 through 12 there is an increase in susceptibility, between the ages 12 through 21 there is a sharp decline, and over 21

there is a gradual decline of susceptibility. Women tend to be more sensitive to it, and it has been suggested that this is true especially during menstruation (Schwab,1954), and therefore there might even be a hormonal link. Psychologically, it has been suggested that people who experience anxiety, neuroticism, arousal, or introversion are also more likely to become victims of the symptoms of motion sickness (Reason, Brandt, 1975). Field independent individuals who rely more on vestibular cues are more sensitive to the conflict between visual and vestibular information (Barret, Thornton, 1968, Biocca, 1994). Perceptional style, whether the user is field dependent or field independent, offers one of the clearest psychological distinctions between those who will become sick and those who will not experience any upsetting sensations (Witkin, 1949).

Often the causes of simulator sickness can be identified without the certainty of why it was induced. For example, it is unclear whether optical flow induces sickness by increasing the sensation of vection, by altering postural control strategies, or having some other effect on the individual (Hettinger, Riccio, 1992). Perhaps then it is most important to centralize the topic on the vestibular apparatus, without which there cannot exist any manifestation of simulator sickness, and investigate if eliminating the absence of input to the vestibular system will prevent discomfort. Furthermore, the flocculus, which is part of the vestibulo-cerebellum, is necessary for motor learning (Lisberger, 1988), so the neural discharges must be investigated around this point.

Symptoms:

Symptoms of simulator sickness are experienced both during immersion in VR and immediately after the user removes the head mounted display (HMD) providing the VR experience.

Symptoms of simulator sickness are nausea, drowsiness, general discomfort, apathy, headache, stomach awareness, disorientation, fatigue, incapacitation, postural instability (ataxia), and emesis. After effects range from illusory sensations of climbing and turning, perceived inversions of visual field and disrupted motor control (Kennedy, Lane, Lilienthal, Berbaum, Hettinger, 1992).

Discussion:

This discussion first addresses the sensory conflict theory with respect to simulator sickness, as well as the experiments that have been done elsewhere and what their results indicate with respect to this theory. Then the concerns regarding VOR and motor learning are examined.

Simulator Sickness:

It is believed that the conflicting signals from the VOR induce simulator sickness. Sensory conflict theory states that since the visual input implies motion to the VR user while the vestibular system is getting a null signal (l-G), the resulting conflicting information to the brain induces simulator sickness. The term null is misleading, it does not mean there is an absence of information. The signal contains as much information as any other, but rather is an indication that there has been no acceleration of the head or body. When the vestibular system sends a null signal, it is relaying the information that neither linear or rotational acceleration has occurred (Sharkey, 1995).

Experiments were conducted at the NASA-Ames Research Center to examine the validity of the theory. In the MONOMO (Motion / no Motion) study, pilots were put in fixed base and motion base simulators and assigned to perform sawtooth and s-turn flight tasks to determine whether or not a motion based simulator would reduce simulator sickness by reducing the intersensory conflict. The tasks that were chosen had previously made 100% of the pilots sick in the fixed base flight simulator. Pilots continued the flight for 60 minutes or until they reported that they could not continue, in which case they were assigned a 7, the scores being between 1 and 7 to indicate increasing severity of sickness. Contrary to what was expected, the results indicated that just as many pilots experienced sickness in the motion base as in the fixed base simulator. (McCauley, Sharkey, 1992) The simulators have envelopes that define their translational and rotational motion, velocity, and acceleration, which have limits where the motion must be terminated. (Sharkey, 1995). Although it is possible that the simulator was unable to provide completely accurate stimulation to the vestibular apparatus and therefore provided false cues, it is argued that these were under the sub-threshold level and therefore would have no effect on intersensory cue conflict. Furthermore, a power analysis was performed prior to the study, which indicated that there was a better than 80% chance that a 1-point difference in the self reports made by the pilots would be detected. There are also more subtle arguments as to why the experiment did not corroborate sensory conflict theory, which deal with defining the limits of cue conflict. If, for example, in five degrees of freedom the motion base corresponds accurately to the visual stimulus the pilot is experiencing but deviates in the sixth degree of freedom, how much does this contribute to sensory conflict. Another consideration is whether or not more information would be revealed if the difference in acceleration signaled by the visual system and the acceleration provided by the motion base were measured in each degree of freedom at each instant. The problem that emerges with this kind of test is the effectiveness of the visual cues. Intersensory conflict would be greater for more detailed visual scenes with the same accelerations as the sparsely decorated scenes.

Thus far what can be deduced is that there are discrepancies in both determining the causes of simulator sickness as well as the experiments conducted to verify them. Part of the reason for this is that there doesn't exist a standardized system of measurement which would regulate the information gathered about simulator sickness and establish a point of comparison. Assuming that the results of the MONOMO tests demonstrate the questionable basis of conflict sensory theory rather than being a result of discrepancies concerning the motion simulator, the question which must be answered in relation to the

experiments is whether or not motion sickness would be induced if conducted in real helicopters with pilots repeating the same maneuvers in flight.

Another test was conducted with the same flight maneuvers, this time implemented by the pilots in the US. army's Crew Station Research and Development Facility (CSRDF) and an AH-1 helicopter. The CSRDF is a fixed base simulator. The test called Simulator Induced Alteration of Head Movements (SIAHM) showed that while the same tests performed in the MONOMO study caused simulator sickness in the CSRDF, motion sickness was not induced in the AH-1 helicopter (Hennessey, Sharkey, Matsumoto, 1992).

In another experiment done at the University of Edinburgh, tests were done with a bi-ocular (vs. binocular) display, Virtuality's Visette 2000, such that each eye was presented with the identical image rather than one image being shared by both eyes. The results indicated no significant visual problems among the participants for immersion periods up to 30 minutes (Rushton, Mon-Williams, Wann, 1994).

Conclusions about these experiments will not be drawn until the results of tests done for this study are presented.

VOR and Motor Learning:

Studies have been done in neurobiology about the plasticity of the brain and its ability to adapt and develop new neural pathways to compensate for certain areas which have been damaged. The fear with VR is that similar neural paths will grow to compensate for the corruption of the VOR. This consequence has been labeled as brain damage but is actually a function of motor learning. The plasticity of the brain, and therefore its ability to learn new motor skills, decreases with age, therefore children with extensive exposure to VR systems would be most effected by it. The concern is that if the neural paths do develop, then although the user will adapt back to the conditions in reality, the paths will continue to exist, and so the VR user might suffer something along the lines of flashbacks if she has had prolonged exposure to VR.

Motor learning occurs whenever there is simultaneous head movement and image motion. Furthermore, the flocculus, which is part of the vestibulo-cerebellum, is necessary for learning (Lisberger, 1988). It is possible that if an individual does not move her eyes with respect to her head but does move them with respect to the VR scene she sees, then learning will be significantly reduced. Though the VOR is required for the eyes to remain steadily fixed on a point that is moving at the same rate as the individual, it is a task that is required of a normal VOR and does not corrupt it. There is therefore a minimum amount of motor learning happening in VR systems where the field of view is constrained to what is immediately in front of the user. In the experiments outlined below there was a minimum amount of eye movement required, though head turns were required for navigation. It suggests that even if the participants had been immersed in VR multiple times, there would have been little threat of motor learning, because their eyes were generally required to look directly ahead into the scene presented in the glasses.

Another issue is that of latency. The relationship between the latency viewed in the glasses due to slow response time, and the latency of VOR pathways that are modified (Lisberger, Pavelko, 1988) is not clear. It is proposed then, that if the latency in the viewed glasses is kept below that of the modified pathways, that learning might not occur.

Experiment:

Seven subjects in different age groups were tested in this experiment. The participants were asked to play the video game DESCENT for 35+ minutes without intermission unless they became too tired to continue. Virtual I/O's HMD and tracker was used to perform the experiments. Virtual I/O's device has a resolution of 320X200, and a field of view of 30 degrees in each eye. The participants were told to use head turns to choose the direction in which they wanted to go, and then to use the joystick to propel themselves forward or backward and to fire guns and missiles. They were told to adjust the volume and choose the level they wished to play for themselves. This information is provided in the table below. None of the subjects had any prior exposure to VR, though some did have experience playing video games. Each participant was asked to rate how they felt between the values of 0 and 5, to which a description was assigned. Furthermore, they were asked to elaborate on their condition using words to emphasize the extent or lack of their discomfort. They were questioned first with the HMD off while they turned their heads back and forth and then up and down. Once they began playing, the participants were questioned after 5 minutes of exposure, 20 minutes, and then once again after 35 minutes immediately after they removed the HMD. Finally, they were asked to report on their condition two hours after they had finished playing the game.

				Rating:	Rating:	Rating:	Rating:
Age	Sex	Level	Volume	VR off	5 min	20 min	35 min
9.5	F	Rookie	Medium	0	1	1	1
16	M	Rookie/ Ace	Semi-soft	0	1	1	1
18	F	Rookie	Medium	0	1	1	1
21	F	Rookie	Medium	0	2	2	3
28	M	Rookie	Medium	0	1	1	1
29	M	Rookie/ Ace	Semi-soft	0	0	0	1
40	M	Rookie	Medium	0	2	2	2

The ratings correspond to the following descriptions:

0: *completely fine, feels nothing.*
1: *fine but feels some effects of immersion in VR.*
2: *experiencing very slight dizziness or discomfort.*

3: *clearly experiencing a degree of discomfort, but not to an extent where it interferes with participation in the game.*
4: *experiencing significant discomfort and/or dizziness, performance in game is effected and participant is distracted by the discomfort.*
5: *severe discomfort, can't continue.*

Virtual I/O's i-glass headset is consumer oriented. The tracking was poor, there was considerable lag between the time the user turned his/her head and the response from the tracker. Often the response was inaccurate, not what the user expected, and sometimes the participant would turn upside down in the game without being able to flip back to being right side up. The game was rendered in 3-D. The table above shows that though it was a small test group, the results were largely uniform. In the case of the female reporting dizziness after removing the HMD, she had had prior unrelated sensations of spinning and sudden disorientation. Most of the participants however reported that they felt almost completely fine both during and after playing the video game. Comments ranged from feeling slight fatigue in the eyes such as the feeling they might get after staring at a computer screen too long, to most often claiming to feel unaffected by the experience in any significant way. Considering the limitations of the tracker and headset, and the lack of exposure among the subjects, the results are surprising in their consistency and the overall low marks on the discomfort scale outlined above.

Conclusions About Experiment:

There was minimal eye movement required from the participants, since their field of view was constrained to the image projected in the glasses, and since DESCENT generally propels the user directly in the direction chosen. Although the users were required to turn their heads and use a swivel chair for maximum freedom and range (the 28 year old participant chose to stand), these movements provide significantly less stimulation to the vestibular apparatus than motion simulators. Applying sensory conflict theory leads to the conclusion that there weren't significant signal inputs that could provide conflicting information. The visual input provided the most stimulation, but its unclear whether this is enough to induce simulator sickness. The i-glasses are such that the user can establish a point of reference by looking down and seeing parts of the real world, though none of the participants did this during the experiment. The significance of having a point of reference, such as peripheral vision, by which the user can maintain her orientation is yet to be determined.

Conclusions:

At this stage many of the conclusions are theoretical because many questions have yet to be answered and many theories verified thoroughly through experiment. Part of the problem is that the research that has been done has been isolated to specific areas of study. Those doing research about motor learning were not concerned with VR, whereas those examining simulator sickness were not necessarily studying the effects on the VOR. The areas of research do not share a standardized and precise set of definitions which would

clarify the parameters of the experiments that have been done with respect to each other. Furthermore, since there is so much that has yet to be verified, there can be a considerable amount of speculation about the reasons for the experiment results. Below you will find the main points introduced in this paper, and a summary of the evidence that supports their potential validity.

There were no incidents of simulator sickness in the experiments done here using the consumer oriented Virtual I/O i-glasses headset. The research corroborates the sensory conflict theory if it is defined to be the conflict between ocular and vestibular input, which were *both* minimal or nearly null in our experiments, the head movements required to manipulate the tracker were familiar to the users and natural. The question of the effect on the senses of moving images persists, because there have been no known reports of simulator sickness by those playing video games on standard computer monitors.

What has been labeled as brain damage is what would more appropriately be attributed to motor learning. It is not clear whether eye as well as head movement has to occur for motor learning, as is suggested by the relationship to VOR, which refers to the ocular muscles being used. If both eye and head movement are required, rather than just visual stimulation and head movement, then VR systems that require minimal eye motion will be the least harmful as far as motor learning is concerned. It should be noted that once something has been learned, it cannot be unlearned once the new neural pathways have developed, even if they are not used they continue to exist, like abandoned roads. Assuming that motor learning occurs with just image and head motion, perhaps learning can be eliminated if the latency of the projected image is kept under the latency attributed to causing modified pathways to grow.

A recommendation, if infact motor learning does occur as specified above, is to stimulate, or saturate the vestibular apparatus. This idea is presented as an exercise to reveal more issues and questions regarding VOR and the brain. It is the vestibulo-cerebellum which is involved with VOR (Takemori, Seki, Aiba, 1987). The vestibular apparatus can be stimulated by methods other than motion. The caloric test, for example, has been developed to test for nystagmus, which involves irrigating the ear with water at different temperatures. The eyes of a healthy individual would rapidly move back and forth laterally in response to the stimulation. The neural connection between the eyes and the inner ear have also been demonstrated through electrical stimulation, as well as by inputting certain frequencies to the ear, which causes ocular response that cannot be controlled by the individual. The eyes controlled by the ocular muscles roll up or rapidly move according to the stimulation provided. It has also been demonstrated by experiment (Clark, Graybiel, 1966) that visual-vestibular intersensory effects can be bi-directional in cases where the inputs are minimal or on the same order. More significantly, when the input to one sense dominates the input to another sense, the dominant input will determine the interpretation of the brain. There is also a phenomenon known as Tullio's disorder, where certain individuals experience severe motion sickness when they are exposed to very loud sounds (>90 dB) at frequencies between 400-500 Hz (Brandt, Kramer). Tullio's

phenomenon is mentioned in order to demonstrate that in certain cases sound can stimulate the vestibular system as well or as severely as motion can.

Stimulating the vestibular apparatus audially or electrically could lead to a greater understanding of its role in the VOR and motor learning. The apparatus might be manipulated in agreement with sensory conflict theory by trying to reduce the difference between the signals, which would require meticulous manipulation of the input to cancel the activity imposed upon the vestibular apparatus from the visual input, or more simply, by overwhelming it with information stronger than that which is being obtained visually. The first modes of human body resonance in the z-axis occur at approximately 5 Hz and 12 Hz. The natural frequency of the human body is experienced during walking (Rao, Ashley, 1960's). Perhaps providing some sort of audial stimulation to the vestibular apparatus through the ear at these frequencies, since it is a direct input rather than one coming from the visual input which must be interpreted by the brain, might be strong enough to significantly reduce the occurrence of simulator sickness. The input could provide a point of reference for the vestibular apparatus the way peripheral vision would provide visual orientation. It may be argued that such an approach does not deal with the problem of simulator sickness directly, it is an answer which does not appreciate the complexity of the question presented by the event of simulator induced sickness. As a response to this, one must consider that the causes of simulator sickness have yet to be defined and proven. Some investigators have argued in fact that the causes are too complex, that there are too many contributing factors, to be able to isolate a dominant reason and a then find a corresponding solution. Furthermore, though technology will be continually improved upon, and the VR environments will be enhanced to provide more accurate manifestations of reality, there will always exist the distinction between what is virtual and what is real, a border which should not be ambivalent. It will be some time before a virtual environment can compensate for input to all the senses so that the experience becomes realistic. For example, the effects of the lack of stimulation to the somatosensors in VR have yet to be explored.

Finally, suggesting such an inquiry into the problem broadens the range of speculation about the issues that need to be resolved involving simulator sickness. Assuming that the brain will develop compensatory neural pathways to overcome the conflict of VOR, which induces simulator sickness, it has yet to be determined whether stimulating the vestibular system with sound, perhaps by saturating the senses, will eliminate the chance of neural pathways developing by virtue of having one sense overwhelm the other and therefore occupy the activity of the brain which is concerned with orientation. At this stage it is necessary to clarify the questions rather than resolve them, and so what must initially be resolved is whether new neural pathways actually develop, or if the changes to the VOR are temporary. It is a secondary issue to resolve the physical discomfort of VR to the user, because though it may discourage people from exposing themselves to it, generally with increased exposure the symptoms diminish, implying the new compensatory paths have developed, which present the more threatening questions to the future of VR.

It is inevitable that once VR systems enter the commercial market, sudden mass exposure will considerably increase complaints about simulator sickness. For most the discomfort will diminish with each use, and our experiments suggest that even upon initial exposure the fascination with VR overwhelms the potential to be discouraged by discomfort from using it.

Suggestions For Experiments:

This study indicates that there are many experiments which still need to be developed and executed if there are to be any firm conclusions about simulator sickness and the corruption of the VOR. Below a few suggestions are outlined.

Subjects should be fitted with HMDs which present them with a single image in the middle of the screen. They should fixate on the point while they are either turned or told to turn their respective heads back and forth. Data concerning neural firing involved in motor learning should be collected using a PET scan or other appropriate device. From this it can be ascertained whether learning occurs. Subjects should then be asked to chose direction in a given virtual scene by turning their heads. They should only be able to chose a direction in order to view something, such as the contents of the room. Since VR systems respond to head turns rather than eye motion to track movements, the subjects will most likely move their eyes very little. Another step may be added where they are instructed to do so. Data about neural firing should again be collected throughout the experiment. Finally, in the last stage, the subject should be able to both chose a direction and travel in it virtually, and the PET scan should be compared to the previous results.

Subjects should also be asked to play video games while wearing the HMD but to use only a joystick to choose orientation and direction, they should not move their heads or bodies.

The above experiments should be repeated with HMDs of varying resolution, weight, and the amount of the real world the subjects can see around the eye pieces. These steps will help determine the effectiveness of having a point of reference outside the virtual images being projected, as well as the effect they have on the subject if she isn't required to move to orient herself virtually.

Another suggestion is to stimulate or saturate the vestibular apparatus, distract it either aurally or by some other method, and have the subjects, perhaps rhesus monkeys, as were used in the motor learning studies, fitted with VR HMDs and the necessary PET scans to determine the events in the flocculus. This should clarify whether distracting part of the VOR will prevent learning, if it is not sending relevant information to the brain.

Final Remarks:

Results of the experiments done here indicate that the participants exposed to VR either do not experience extensive simulator sickness, or are not discouraged by mild symptoms from using VR. Furthermore, with repeated use, experiments have shown that many of

the symptoms of simulator sickness diminish. The change is attributed to the adaptation of the VOR, which raises more serious questions about the effect of VR on the brain. Whether there are more serious consequences resulting from exposure to VR must be resolved before VR systems permeate the mass market.

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