12

Research Issues in Simulator Sickness:

Proceedings of a Workshop

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Michael E. McCauley, Editor

Committee on Human Factors
Commission on Behavioral and Social Sciences and Education
National Research Council

NATIONAL ACADEMY PRESS
Washington, D.C. 1984

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The Committee on Human Factors in the Commission on Behavioral and Social Sciences and Education is sponsored jointly by the Air Force Office of Scientific Research, the Army Research Institute for the Behavioral and Social Sciences, the Office of Naval Research, the National Aeronautics and Space Administration, and the National Science Foundation.

This work relates to Department of the Navy Contract N00014-81-C-0017 issued by the Office of Naval Research under Contract Authority NR 196-167. However, the content does not necessarily reflect the position or the policy of the government, and no official endorsement should be inferred.

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WORKSHOP ON SIMULATOR SICKNESS

HERSCHEL W. LEIBOWITZ (Chair), Department of Psychology, Pennsylvania State University
ALAN J. BENSON, Royal Air Force Institute of Aviation Medicine, Farnborough, Hants, U.K.
JOHN G. CASALI, Vehicle Simulation Laboratory, Virginia Polytechnic Institute and State University
MALCOLM M. COHEN, Biomedical Research Division, NASA Ames Research Center, Moffet Field, Calif.
SHELDON M. EBENHOLTZ, Department of Psychology, University of Wisconsin
LAWRENCE H. FRANK, Naval Training Equipment Center, Orlando, Fla.
KENT GILLINGHAM, USAF SAM/VNB, Brooks Air Force Base, Tex.
FREDERICK E. GUEDRY, Naval Aerospace Medical Research Laboratory, Pensacola, Fla.
CHARLES W. HUTCHINS, U.S. Naval Postgraduate School, Monterey, Calif.
ROBERT S. KELLOGG, University of Dayton Research Institute, Williams Air Force Base, Ariz.
ROBERT S. KENNEDY, Essex Corporation, Orlando, Fla.
JAMES R. LACKNER, Department of Psychology, Brandeis University
MICHAEL E. McCAULEY, Monterey Technologies, Inc., Carmel, Calif.
GRANT B. McNAUGHTON, Norton Air Force Base, Calif.
KEN E. MONEY, Defense and Civil Institute of Environmental Medicine, Downsview, Ont.
ROBERT POST, Department of Psychology, University of California, Davis

JOHN B. SINACORI, John B. Sinacori Associates, Monterey, Calif.
EDWARD A. STARK, Link Flight/Simulator Division, Singer Company, Binghamton, N.Y.
DANIEL J. WEINTRAUB, Human Performance Center, University of Michigan
ROBERT H. WRIGHT, U.S. Army Research Institute, Fort Rucker, Ala.

ROBERT T. HENNESSY, Workshop Organizer
JEANNE RICHARDS, Administrative Secretary

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STANLEY DEUTSCH, Study Director
JEANNE RICHARDS, Administrative Secretary

CONTENTS

PREFACE xi

INTRODUCTION 1

INCIDENCE AND PREVALENCE 4

SIMULATOR CHARACTERISTICS 8

- Visual Systems, 8
- Lags, 10
- Motion Systems, 12
- Other Motion Cueing Devices, 15
- Comment on Visual and Motion Systems, 15
- Designing a Flight Simulator, 16
- Design Characteristics of Automobile Simulators, 19

THEORIES OF MOTION SICKNESS AND ADAPTATION 22

- Sensory Conflict Theory, 25
- Adaptation Studies, 30
- Oculomotor Adaptive Systems, 33
- Comments on Theories of Motion Sickness, 41

RECOMMENDED COUNTERMEASURES FOR EXISTING SIMULATORS 43

RECOMMENDATIONS FOR RESEARCH 48

Problem Definition, 48
Theory/Model Development, 49
Methodology, 50
Determination of Causes and Processes, 51
Evaluation of Candidate Countermeasures, 53

APPENDIX: POTENTIAL CONTRIBUTORS TO SIMULATOR SICKNESS 55

REFERENCES 59

TABLES AND FIGURES

Table 1	Major Studies of Simulator Sickness	5
Table 2	Types of Sensory Conflict	31
Table 3	Recommended Countermeasures for Current Simulators	44
Figure 1	A Comparison Between MIL-STD 1472C Vomiting Criteria and a Projected Envelope for Lesser Symptomatology	14
Figure 2	Cross-Coupled Semicircular Canal Stimuli	29
Figure 3	Etiology of Motion Sickness	30
Figure 4	Vergence and Accommodation Control Systems	36
Figure 5	Vector Analysis of the Conditions Productive of Increased Gain, Reversed Gain, and Directional Shifts in the Vestibular-Ocular Reflex	38

PREFACE

The Committee on Human Factors was established in October 1980 by the Commission on Behavioral and Social Sciences and Education of the National Research Council in response to a request by the Office of Naval Research, the Air Force Office of Scientific Research, and the Army Research Institute for the Behavioral and Social Sciences. In addition, its sponsors currently include the National Aeronautics and Space Administration and the National Science Foundation. The committee's objectives are to provide new perspectives on theoretical and methodological issues, identify basic research needed to expand and strengthen the scientific basis of human factors, and to attract scientists both within and outside the field to perform the needed research. Its overall goal is to provide a solid foundation of research as a base on which effective human factors practices can build.

Human factors issues arise in every domain in which people interact with the products of a technological society. To perform its role effectively, the committee draws on experts from a wide range of scientific and engineering disciplines, including specialists in the fields of psychology, engineering, biomechanics, cognitive sciences, machine intelligence, computer sciences, sociology, and human factors engineering. Experts in additional disciplines also participate in the working groups, workshops, and symposia organized by the committee. Each of these disciplines contributes to the basic data, theory, and methods needed to improve

the scientific basis of human factors.

Simulator sickness, the experience of symptoms similar to motion sickness, has occurred frequently in civilian and military flight training simulators and, in some cases, has persisted or arisen several hours after a simulator session. The syndrome is of concern to simulator designers and users, including simulator developers, flight instructors, training and operational personnel, aerospace physicians, physiologists, psychologists, and, of course, pilots.

It is important to bear in mind that simulator sickness may be a special case of spatial disorientation, or at least result from the same basic mechanisms. Spatial orientation is a fundamental biological function that incorporates posture, locomotion, and knowledge of one's position in space. As such, it is of continuing interest to a wide spectrum of scientists concerned with its underlying mechanisms, individual differences, pathology, and developmental aspects. Thus, the study of simulator sickness is valuable not only from the point of view of simulators but also in terms of the contribution it may make to the more general phenomenon of spatial orientation.

Recognizing the potential importance of the problem, the Naval Training Equipment Center, the Army Research Institute, and the Air Force School of Aerospace Medicine asked the Committee on Human Factors to identify current information and to recommend research aimed at the development of countermeasures. The committee convened a three-day workshop September 26-28, 1983, at the Naval Postgraduate School in Monterey, California, to: (1) review available information on the nature and severity of the symptoms of simulator sickness, their frequency, and circumstances of occurrence; (2) identify its likely etiology and contributing factors, such as simulator design characteristics and training methods; (3) assess the efficacy of current available countermeasures to the effects of simulator sickness; (4) assess whether its occurrence is an indication of deficiencies in simulators that may adversely affect the transfer of learning, operational performance, or safety; and (5) recommend research and other courses of action necessary to eliminate the problem of simulator sickness.

Nineteen experts in one or more of the following fields participated in the workshop: motion sickness, vestibular dynamics, visual processes, simulator

sickness, and simulator design and use. This report of the proceedings reflects the discussion that took place and the group's recommendations for research. It is based on audio recordings of the workshop, background position papers provided by the participants in advance of the meeting, presentations at the meeting, and comments on a preliminary draft. In a number of sections of the report, attribution is given to indicate that the section reflects the thinking of an individual participant. Most participants made comments on the draft report, which were incorporated into the text. We have attempted to reflect the workshop issues and the thinking of the participants as accurately as possible.

A background paper prepared by Robert S. Kennedy and Lawrence H. Frank, "A Review of Motion Sickness With Special Reference to Simulator Sickness," provided a review of existing information for the deliberations of the workshop participants. The paper, which is available from the Committee on Human Factors, describes the phenomenon of motion sickness and presents various theories concerning its etiology and response characteristics.

It should be noted that the workshop participants support the use of flight simulators for training. Our discussions of simulator sickness do not imply an indictment of simulators. Our intent is rather to strive for progress in the design and application of simulators through understanding. As technology advances, continued advances are also necessary in the human/machine interface.

A related study on simulation has recently been completed by a special working group of the Committee on Human Factors. The working group's report is scheduled for release early in 1985.

In addition to the workshop participants, a number of people contributed in important ways to the success of the study. Michael E. McCauley did a fine job as editor of the proceedings report. Robert T. Hennessy, the committee's study director in 1983, and workshop participants Robert Kennedy and Larry Frank effectively planned and organized the workshop. Gerald S. Malecki, of the Office of Naval Research, Alfred R. Fregly, of the Air Force Office of Scientific Research, and Robert M. Sasmor, of the Army Research Institute for the Behavioral and Social Sciences, provided important assistance in organizing and supporting this effort. Charles W. Hutchins hosted the meeting on behalf of the

Naval Postgraduate School; his hospitality and hard work in preparing the facilities for the workshop are sincerely appreciated. Stanley Deutsch, the committee's study director, made valuable contributions in organizing and drafting the report. Christine L. McShane, editor of the Commission on Behavioral and Social Sciences and Education, was extremely helpful in improving its style, organization, and clarity. Ann G. Polvinale, administrative associate of the Commission, and Jeanne Richards, the committee's administrative secretary, provided extensive secretarial and administrative support.

Herschel W. Leibowitz, Chair
Workshop on Research Issues in
Simulator Sickness

INTRODUCTION

Simulator sickness is a term used to describe the diverse signs or symptoms that have been experienced by flight crews during or after a training session in a flight simulator. The phenomenon has been described as polygenic and polysymptomatic; symptoms include nausea, dizziness, spinning sensations, visual flashbacks, motor dyskinesia, confusion, and drowsiness (Frank et al., 1983). Observable evidence (signs) of simulator sickness include pallor, cold sweating, and emesis.

Motion sickness is a general term for a constellation of symptoms and signs, generally adverse, due to exposure to abrupt, periodic, or unnatural accelerations. Simulator sickness is a special case of motion sickness that may be due to these accelerative forces or may be caused by visual motion cues without actual movement of the subject (Crampton and Young, 1953; Dichgans and Brandt, 1973).

Although some scientists have objected to the term simulator sickness because the constellation of effects associated with it would be described better as a syndrome than a sickness, most of the workshop participants concurred with the use of the term because it is idiomatic, similar to terms for other subsets of motion sickness such as sea sickness, car sickness, and space sickness. (The National Aeronautic and Space Administration has adopted the term space adaptation syndrome--Homick, 1982; Nicogossian and Parker, 1982).

Simulator sickness has been experienced by pilots, copilots, and other crew members in flight simulators as well as by drivers and passengers in automobile simulators. The workshop emphasized simulator sickness

related to flight simulators, but pertinent evidence related to automobile simulators was also discussed. In all documented cases of simulator sickness, a visual display of vehicle dynamics has been involved.

Simulator sickness occurs in both fixed- and motion-base simulators. It occurs during the simulator flight, immediately afterward, and many hours later. The phenomenon was first reported in connection with a helicopter simulator (Havron and Butler, 1957; Miller and Goodson, 1958), but it has occurred in patrol, transport, and fighter/attack aircraft simulators as well. The highest incidence (88 percent) has been reported in an air combat maneuvering (ACM) simulator during fixed-base operations, in which high visual acceleration maneuvers are common (Kellogg et al., 1980).

Experienced aviators and test pilots seem to be more susceptible to simulator sickness than inexperienced trainees. This fact seems surprising, but it is consonant with the most common explanation of the cause of simulator sickness, i.e., intersensory conflict. The sensory conflict hypothesis suggests that experienced aviators have a well-established neural store representing the relationships among manual control actions, visual dynamics, and the orientation and inertial senses subserved by the vestibular/proprioceptive systems. Inexperienced aviators do not have such a well-established neurophysiological "expectancy" for these relationships. To the extent that the simulator violates the sensory expectancies, a conflict exists.

Simulator sickness may be of operational significance because of four kinds of problems:

1. Compromised Training. Symptoms experienced in the simulator may compromise training through distraction and decreased motivation. Behaviors learned in the simulator to avoid symptoms (e.g., not looking out the window, reducing head movements, avoiding aggressive maneuvers) may be inappropriate for flight.
2. Decreased Simulator Use. Because of the unpleasant symptoms and aftereffects, simulator users may be reluctant to return for subsequent training sessions. They also may have reduced confidence in the training they receive from the simulator.
3. Ground Safety. Aftereffects, such as disequilibrium, could be potentially hazardous for users when exiting the simulator or driving home.

4. Flight Safety. No direct evidence exists for a relationship between simulator sickness aftereffects and accident probability. However, from the scientific literature on perceptual adaptation, one could predict that adaptation to a simulator's rearranged perceptual dynamics would be counterproducti e in flight. Indeed, anecdotal reports from the Royal Air Force in the early 1970s indicate that flight instructors claimed increased susceptibility to disorientation in flight hours after a simulator session.

This report covers the topics that were discussed in detail at the three-day workshop. It begins with an account of the major studies of simulator sickness and what we know about its incidence and prevalence. The next section describes the relevant design characteristics of simulators: it begins by describing some of the characteristics of simulators and their operators that may be involved in simulator sickness, including visual systems, lags, motion systems, and other motion cueing devices; it then discusses design problems specific to flight simulators and those specific to automobile simulators. Theories of motion sickness and adaptation are dealt with in the following section, and sensory conflict theory emerges as the most plausible explanation for the phenomenon of motion sickness. The report then makes a number of practical suggestions for avoiding the effects of simulator sickness in the equipment currently in use, although these countermeasures remain to be validated. It ends with a summary of the recommendations for research that surfaced throughout the workshop.

It is important to note that the workshop did not review the relationship of space sickness (or space adaption syndrome) to simulator sickness. This omission was deliberate, largely due to the fact that the relationship between motion sickness in a one-gravity environment and the space adaptation syndrome is poorly understood at this time. In studies by NASA there was great difficulty in predicting susceptibility to space sickness using tests performed in one-gravity environments. Even incidents of motion sickness in provocative one-gravity environments prior to space flight were not useful in predicting incidents of space adaptation syndrome (Nicogossian and Parker, 1982).

INCIDENCE AND PREVALENCE*

The overall incidence of simulator sickness in flight simulators across the armed services is unknown, even though the problem was first reported nearly 30 years ago. There are suggestions, however, that the incidence is increasing. Pilots tend not to talk about such problems as simulator sickness, so we may be underestimating the problem. As new video systems become operational in simulators and in aircraft, information about simulator sickness and motion perception may become even more important.

This section is a brief review of the documented evidence of simulator sickness. For a more complete review, see Kennedy et al. (1983) and Frank et al. (1983). In addition, Puig (1970, 1971) has reviewed the theoretical basis for disorientation and sickness in simulation.

Table 1 summarizes the major studies that have been made of simulator sickness. The studies by Havron and Butler (1957) and Miller and Goodson (1958, 1960), the first published reports of simulator sickness, found a substantial incidence (72 percent) of symptoms among users of the Navy's 2-FH-2 Hover Trainer. An interesting finding from the Miller and Goodson work, as previously noted, is that "the more experienced instructors seemed to be those most susceptible to unpleasant sensations." Other findings of note were that "the more violent maneuvers were found to produce a greater degree of

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*This section was drafted from the workshop presentation by Lawrence H. Frank.

TABLE 1 Major Studies of Simulator Sickness

Report	Device	Incidence (Percent)	Base	Visual Type	Field of View (H x V)
Havron and Butler (1957)	2-FH-2	77	Fixed	PS	260 x 75
Miller and Goodson (1958, 1960)	2-FH-2	60(I) 12(S)	Fixed	PS	260 x 75
Hartman and Hatsell (1976)	SAAC	52	Moving	CIG	296 x 180
Kellogg, Castore, and Coward (1980)	SAAC	88	Fixed[a]	CIG	296 x 180
Money (1980)	CF140 FDS	43	Moving	T/N/CIG	48 x 36(?)[b]
McGuinness, Bouwman, and Forbes (1981)	2E6	27	Fixed	T/PS CAM MOD	360 x 150
Frank (1981)	2F112	10	Fixed	T/PS CAM MOD	360 x 150
Frank (1981)	2F110	48	Moving	T/N/CIG	120 x 36
Crosby and Kennedy (1982)	2F87	50	Moving[c]	T/N/CIG	48 x 36[b]
Frank and Crosby (1982)	2F117	---	Moving	CIG	175 x 50

Notes: PS = point source; T = twilight; N = night; CIG = computer image generation; CAM MOD = target camera model; I = instructors; S = students.

[a]Motion not used in study.
[b]One window.
[c]Motion or lack of motion had no effect.

Source: Kennedy et al. (1983).

'motion sickness.' Instructors have reported that they are more prone to become sick when sitting as a passenger . . . than when they are actually 'flying' the simulator" (Miller and Goodson, 1960:210). Miller and Goodson (1958) also reported the occurrence of delayed effects in an instructor pilot who became "so badly disoriented in the simulator that he was later forced to stop his car, get out, and walk around in order to regain his bearings enough to continue driving" (p. 9).

Sinacori (1967) studied simulation techniques for vertical short takeoff and landing (VSTOL) flight. Using only one test pilot, he was able to report:

> Pilot vertigo was induced as the time duration of a particular flight increased. Vertigo was especially annoying to the pilot during attitude reversals or hovering. The pilot felt he could do better with cockpit motion cues. . . . Pilot vertigo may be caused by the conflict between the sometimes "fair" visual cues acquired during attempted hover and the highly trained kinesthetic sensations which are expected but not felt because the cockpit is fixed. Inadvertent pilot head motions were observed frequently.

Kellogg et al. (1980) studied simulator sickness during fixed-base operations in the Air Force Simulator for Air to Air Combat (SAAC), which has a wide field of view. The 48 pilots surveyed were undergoing an intense exposure--a high acceleration environment (implied visually) during 550 ACM engagements over 5 days, averaging a total of 12 hours of exposure for each pilot. More than 87 percent of the pilots surveyed reported some symptoms of simulator sickness, primarily nausea. Symptoms were most prevalent in the first few days. Pilots reported visual flashbacks, sometimes 8-10 hours after exposure; these included sensations of climbing and turning while watching TV and experiencing an inversion of the visual field while lying down.

In a study for the Canadian Defence and Civil Institute of Environmental Medicine, Money (1980) investigated reports of simulator sickness in the Aurora CP 140 FDS (analogous to the U.S. Navy's P-3 Orion). He found that 44 percent of the pilots reported symptoms, ranging from slight discomfort to mild nausea. The symptoms were usually experienced only in the first one or two simulator exercises. Subsequent exercises were symptom-free, presumably due to habituation.

McGuinness, Bouwman, and Forbes (1981) investigated the incidence of simulator sickness among 66 air crew in the Navy's F-4/F-14 Air Combat Maneuvering Simulator (ACMS), the 2E6, and reported an overall incidence of 27 percent. However, the more experienced air crews, those with more than 1,500 lifetime flight hours, had an incidence of 50 percent, while those with less than 1,500 hours had an incidence of 18 percent. Pilots had a greater incidence than Radar Intercept Officers (RIOs or "backseaters") with incidences of 36 percent and 15 percent, respectively. There were no reports of visual flashbacks in the 2E6. Dizziness was the most frequent symptom, followed by vertigo, disorientation, "leans," and nausea.

Frank (1981) reported that approximately 10 percent of those using the Navy's F-14 simulator, the 2F112, experienced symptoms of simulator sickness and that approximately 48 percent of those sampled in the E-2C simulator, the 2F110, experienced symptoms. In the 2F110, several of the pilots commented that the visual streaming that occurred during turns-while-taxiing was particularly disconcerting.

Crosby and Kennedy (1982) found that flight engineers were having problems in the P-3C simulator, the 2F87. They found that the flight engineer was viewing the independent CRT/CGI displays of the pilot and the copilot from 30 degrees off-axis. Measures of postural equilibrium in walking and standing, which have been used previously as indices of vestibular disruption (Fregly, 1974), indicated significant decrements in 50 percent of the flight engineers after a normal four-hour exposure in the simulator. Occluding the flight engineer's view of the pilot's and copilot's displays eliminated the postural equilibrium problems.

Frank and Crosby (1982) investigated the CH-46E helicopter simulator, the 2F117, while it was in the final stages of development and production. They reported some tendency for symptoms of simulator sickness and suggested that a more rigorous study be conducted after the 2F117 is introduced to the fleet.

From the brief review given above it is clear that a constellation of effects have been found in air crews during or after exposure to flight simulation. These effects include the classic signs and symptoms of motion sickness and phenomena associated with perceptual adaptation. The overall incidence and severity of the problem across a broad spectrum of flight simulators, however, has not been established.

SIMULATOR CHARACTERISTICS

A large number of simulator and operator characteristics are suspected of playing a role in simulator sickness. The workshop participants generated a long list of them, which provided an initial structure for discussion. Limitations of time at the workshop allowed only a few of these characteristics to be discussed in detail. However, as a potential guide to other investigators, the complete list appears as an appendix to this report.

This section presents the discussions that took place on visual systems, time lags, motion systems, and other motion cueing devices. It also incorporates workshop presentations on the design of a particular flight simulator with its attendant problems and problems involved in automobile simulators.

Visual Systems

Several types of visual systems have been used in flight simulators, the major ones being point-light source, model board, and computer-generated imagery (CGI). Cases of simulator sickness have been documented in CGI and point-light source visual systems, but they seem to be less frequent in model board systems.

One of the important variables for simulator sickness is the field of view (FOV) of the visual system. An estimated range of horizontal FOV for flight simulators is 40-360 deg, depending on the purposes of the simulation. For example, the Navy's Night Carrier Landing Trainer (NCLT, 2F103) has a relatively narrow FOV, approximately 40 deg. In contrast, ACM simulators,

such as the Air Force SAAC and the Navy's ACMS, have wide FOVs, in excess of 300 deg.

Research by Leibowitz et al. (1982) has suggested the importance of the ambient visual system in processing dynamic and orientation information. The general finding with respect to retinal location is that information at the peripheral retina is a more powerful determinant of spatial orientation effects than is information at the central retina. This is confounded, however, by subtended angle; things usually get larger in the periphery because one is moving forward. So perhaps it would be more accurate to say that larger things affect orientation more than smaller things.

Thus, a wider FOV would provide more stimulation for the ambient system, resulting in a more compelling visual display of motion. This enhanced sense of visual motion may contribute to more conflict with vestibular inputs, which are relatively impoverished in the simulator.

The SAAC has a mosaic of eight electronic screens that surround the canopy of an F-4 cockpit, yielding a 296 x 180 degree (H x V) FOV. Anecdotal reports suggest that disorientation and symptomatology occur with the full eight-window display, but not with three windows. Also, a compelling illusion of tilt was perceived with the eight-window display when a flight scenario was frozen in a 45-deg angle of bank.

A study by Reason and Diaz (1971), however, found no increase in the incidence of sickness in an automobile simulator as the FOV was increased from 45 to 90 deg. More research is needed on FOV.

Scene detail is another variable that may be important in the genesis of simulator sickness. Greater scene detail provides the human visual system with more information about spatial dynamics, presumably sharpening the perception of motion and generating greater conflict with the vestibular inputs. The effects of scene detail, however, have not been investigated systematically.

Lags in the temporal presentation of the visual display have been suggested as a contributing factor in simulator sickness. This issue is discussed more fully in the section below on lags.

There has been some suggestion that the detailed process of writing a visual display across the screen may be registered by the human visual system, if not perceived consciously. According to one estimate, a typical time period for writing the video image is

approximately 16 msec. In several simulators, e.g., the Advanced Simulator for Pilot Training (ASPT) and SAAC, the video images are written in different directions on adjacent windows. This may create an unusual visual stimulus of simultaneous movement in different directions in adjacent locations. It is possible that these kinds of effects may contribute either to symptoms of simulator sickness or to the visual aftereffects that have been reported. The same problem of multidirectional video writing is likely in the new area-of-interest simulator displays, in which one display is contained within a larger one, their video images being written in different directions.

Some simulators have visual systems with a 2:1 interlace system in which the video imagery is updated by the computer at 30 Hz but the display is updated at 60 Hz. With this type of system it is inevitable that moving targets create double images, which may create illusory movement and other problems, such as a strobing effect. The contribution of these effects to simulator sickness is unknown.

Other features of visual displays have been cited as potential contributors to the problem. Optical distortions were mentioned by Miller and Goodson (1958) as a probable contributing factor in simulator sickess. Poor resolution, flicker, and off-axis viewing also have been implicated (Frank et al., 1983).

As new video displays become operational in aircraft, it will be particularly important to ensure that the visual dynamics provided in the simulator are compatible with those experienced in the aircraft. For example, a helmet-mounted TV display will be included in one of the weapon systems on the Army's new gunship, the 64 Apache. Research on visually coupled systems should include questions of perceptual aftereffects in the simulator and the aircraft.

Eventually, three-dimensional visual displays are likely to be introduced. The study of motion perception, perceptual adaptation and aftereffects will be even more important when three-dimensional displays become operational in simulation.

Lags

Temporal incongruities may exist in the presentation of motion information in the simulator. Time lags may

occur in the visual system (Crane, 1983), the motion base, or both (Ricard et al., 1975; Ricard and Puig, 1977; Semple et al., 1980).

Lags should be defined with reference to the temporal relationships found in the aircraft as well as to the usual description of total time. As an example, suppose that 50 msec elapsed between a pilot's roll input with the stick and the beginning of the aircraft roll. Given the same input in a simulator, realistic estimates of lags might be 250 msec before the visual system begins to respond and 350 msec for the motion base. There are thus several sources of error for the highly tuned neural store of the experienced pilot: a 200 msec lag in the visual, a 300 msec lag in the inertial, and a 100 msec discrepancy between the two. This is a complex problem, because there probably is not a constant optimal lag time. Puig (1970) has pointed out that the optimal lag time is likely to be a function of the intensity of the stimulus (i.e., the level of acceleration).

Moreover, lag is just one index of the fidelity of dynamic information. The accelerative responses of the visual and inertial systems should not only begin at the proper time, but follow the rise time and amplitude characteristics with reasonable fidelity.

Experienced pilots have learned a set of temporal and spatial patterns in the aircraft related to control stick inputs and the resultant visual, vestibular, and proprioceptive feedback of acceleration information. In the simulator, they are confronted with a new set of temporal and spatial patterns, i.e., lags, rise times, washout, etc. This discrepancy is probably the main source of simulator sickness.

We gain more insight into this problem by considering the differing dynamics of the visual and vestibular sensory systems in the perception of motion. Retinal receptors signal position and velocity of a visual target, from which acceleration may be perceptually derived. In contrast, the otoliths (in company with somaesthetic mechanoreceptors) are sensitive to linear acceleration and rate of change of acceleration (jerk) and hence give information about body movement that is phase-advanced on that provided by the visual system. Sensory integration of these gravireceptor signals is required in order to perceive transient linear velocity and displacement. According to Benson (1978), the semicircular canals signal, for

transient angular movements, the angular velocity of the head and provide cues that allow the change in angular position or angular acceleration to be perceived by integration or differentiation of the afferent signal within the central nervous system. The implication of these differing sensory dynamics is that sensory conflict is likely to be the greater if mechanical movement of the simulator (and hence the operator) lags visual movement of the visual display than if the motion system leads the visual.

Why are more experienced pilots more susceptible to sickness? Perhaps only people who are very susceptible to motion sickness are likely to have a problem when they are inexperienced, but as experience is acquired, the less susceptible pilots also may be affected. This information is important to simulator designers, because they can aim their design at the experience level of the users. Highly experienced pilots may not tolerate as much error between visual and motion cues. With inexperienced students, however, high transfer of training may require a less exacting simulation.

A simulator (particularly the motion base) cannot reproduce the acceleration waveforms of the aircraft exactly. What degree of departure from the nominal acceleration waveforms is acceptable? The answer is not entirely known. It depends in part on human visual and vestibular processes as influenced by simulator characteristics and the experience of the user, both in the aircraft and the simulator. This is certainly an area for further research that could have an effect on simulator sickness, the transfer of training, and simulator design guidelines.

Motion Systems

The majority of today's military flight simulators have a motion base. The number of axes of motion varies from one to six, although six is most common. The typical motion base is hydraulically driven and has a maximum angular displacement of 32 deg and a maximum linear (translational) displacement of about 1 m. Because of the displacement limitations, motion systems are driven by command signals using washout algorithms that permit high fidelity of movement initiation, with subsequent diminution of the motion response even though the accelerations associated with the maneuver (and implied

by the visual display) continue. The importance of tuning the washout algorithms is discussed in the section on designing a flight simulator.

The responsiveness of a good motion base provides vestibular and proprioceptive cues to motion for subtle aircraft maneuvers. In very extreme maneuvers, such as those required in air-to-air combat, the motion base has the hopeless task of keeping up with sustained high-gravity turns and rapid roll rates. It has been reported that the Air Force air combat simulator (SAAC) had a motion base that was eventually disengaged because of its ineffectiveness (Seevers and Makinney, 1979). The Navy's 2E6 air combat simulator is also fixed-base (McGuinness et al., 1981). Despite good design characteristics and periodic calibration, the effectiveness of a motion base is likely to be limited in simulators intended for scenarios with high-acceleration maneuvers.

Frank et al. (1983) have emphasized the importance of simulator resonant frequency as a possible contributory factor to simulator sickness. It is known that symptoms are greatest at a frequency resonance of about 0.2 Hz (McCauley and Kennedy, 1976; Money, 1970). Hence, it would appear advisable to avoid this very low frequency range at the trainee's sitting position in building simulators. Frank et al. (1983:7-8) describe a case in point, which appears as Figure 1:

> [The figure] presents a comparison between Military Standard 1472C (MIL-STD 1472C, 1981) vibration protection limits, projected envelopes for lesser symptomatology, and the SAAC frequency spectrum. The two solid lines are from MIL-STD 1472 and represent the 90% protection limits for an 8-hour vibration exposure. The solid U-shaped line, representing the exposure limit for below 1 Hz, is based on a criterion of frank emesis. The solid line, representing the exposure limit for above 1 Hz, is based upon a criterion of fatigue-decreased proficiency. The criteria for these two differ as a result of the large quantity of data generated on the effects of vibration on human performance above 1 Hz. In contrast, human performance data are currently insufficient to reach hard conclusions for exposures to very low frequency vibrations (i.e., below 1 Hz). The limits for below 1 Hz, then, should be viewed as conservative, since it can be

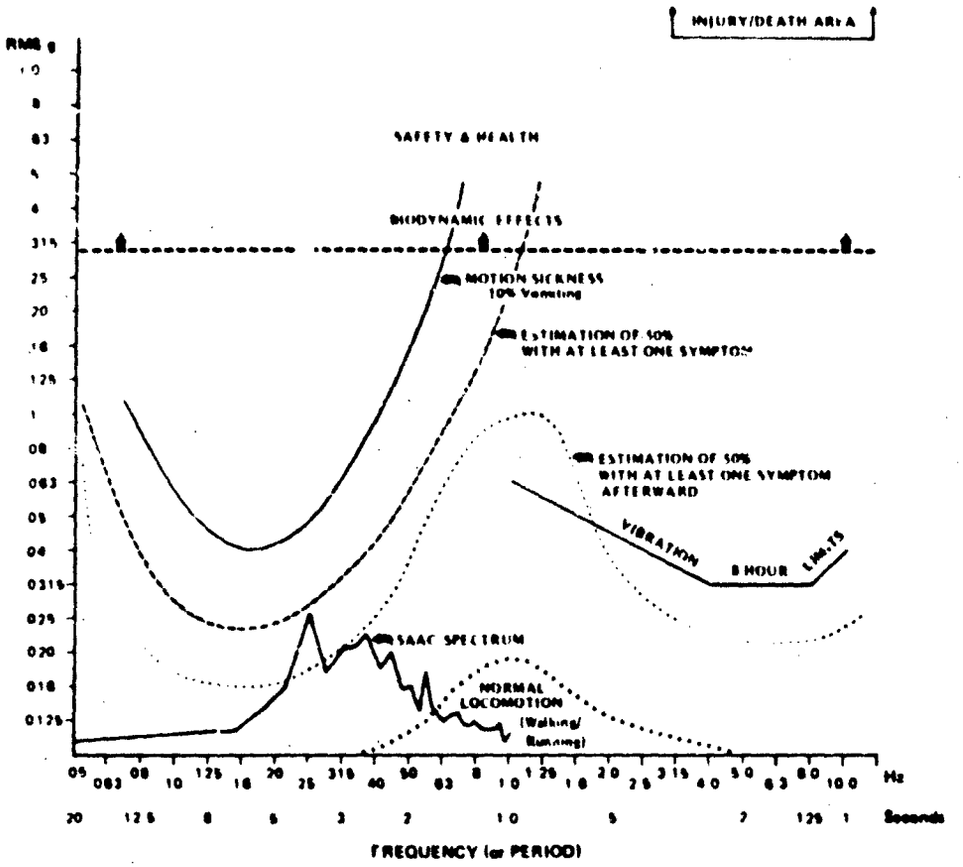

FIGURE 1 A Comparison Between MIL-STD 1472C Vomiting Criteria and a Projected Envelope for Lesser Symptomatology.

Source: Frank et al. (1983).

predicted that decrements in performance can be expected to occur before emesis. Consequently, the heavy-dashed line represents our estimation of where 50% of the population will exhibit at least one symptom of simulator or motion sickness (e.g., pallor). The light-dashed line is our estimation of where at least one post-effect will occur. Note that the tolerance limits for each of these envelopes shift upward, coincident with the spectrum for normal locomotion. Note, also,

that the lowest thresholds correspond to those energy regions that are most associated with motion sickness. . . .

The SAAC spectrum depicted in [the figure] was replotted from Hartman (1976). This mapping reveals quite clearly that the resonant frequency of the SAAC inertial system intersects our estimated tolerance envelopes and, therefore, may be conducive to simulator sickness. Indeed, Hartman and Hatsell reported incidence rates for spatial disorientation, eye strain, tiredness, headache and nausea of 52%, 50%, 38%, 32%, and 14%, respectively.

It is readily apparent from the figure that simulator resonant frequency is of critical saliency, relative to simulator sickness; and that simulators should be designed with these envelopes in mind.

Other Motion Cueing Devices

In addition to a motion base, G-seats, G-suits, helmet loaders, and other devices have been used in flight simulators to provide pseudo-inertial cues to the pilot. The cueing algorithms for these devices require further development to ensure that the proper temporal patterns are achieved relative to the vision and motion base systems.

G-seats may be pneumatic or hydraulic. The pneumatic seats have longer lag times, which must be compensated for if they are to provide useful motion cues. G-seats also change the location of the pilot's head, a feature that combines with voluntary head movement to change the point of regard of the visual display. These variables have not been thoroughly studied with respect to the pilot's perception of motion from the display or the potential discrepancies relative to the aircraft.

Comment on Visual and Motion Systems

In summary, the characteristics of both visual displays and motion systems for simulation present problems, not only with regard to simulator sickness, but in the larger context of the selection and communication of motion information to support learning and optimize transfer of training.

Designing a Flight Simulator*

In 1965 the Northrup Corporation was committed to building a flight simulator to study the problems of aerodynamics and flight control for vertical short take-off and landing aircraft. The concepts for building a simulator then were not much different than when Miller and Goodson first reported simulator sickness in 1958, according to the DeFlores Principle; i.e., the pilot sat in a fixed platform surrounded by a spherical screen with about a 12-ft radius. At the top center was a point-light source that provided a screen luminance of about 1 ft-Lambert. Features on a glass plate below the light were projected onto the screen as shadows. The plate was servo-driven under the light to simulate the progress of the aircraft over the ground.

Field of view is the first factor identified as important in simulator sickness as discussed above. Both size and shape are relevant aspects of FOV. The Northrup simulator had a FOV of about 200 by 60 degrees, which is equivalent to about 28 percent of a complete sphere. Such a large field is quite compelling to the pilot.

The X-14 aircraft at Edwards Air Force Base was used as a validation aircraft for the simulator. It had T-34 wings and tail and a "home-made" fuselage with an open cockpit and two J85 engines. The project team at Northrup tried to design the simulator so that the pilot ratings of flight handling characteristics would be equivalent for the simulator and the X-14. They were also interested in the test pilot's (N = 1) subjective impressions of workload.

One of the first things they looked for was the ability to maintain a hover. There was a good correspondence between the performance of the aircraft and the simulator for the hover. The visual display and the control characteristics seemed quite adequate.

Scene detail became an issue as they attempted to put features on the glass plate for the visual display. The raw number of stimuli that represent the real world, probably an important factor in simulator sickness, is represented as the number of patch boundaries in the luminance distribution. There are metrics for this factor.

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*This section was drafted from the workshop presentation by John B. Sinacori.

When they tried the second maneuver, the lateral-quick-stop, the test pilot complained about stomach awareness and nausea. After about 10-20 minutes of performing lateral-quick-stops, the pilot had to take a break to avoid being sick.

The pilot felt that these effects would go away if he had a motion base. Bolstered by discussions with experts such as Fred Guedry and an introduction to the concept of sensory conflict (discussed below), they acquired a motion base. It had 3 df, pitch, roll, and yaw with +/- 12 deg angular displacement--and no translatory motion. The visual system was capable of about +/- 30 deg, so some type of washout was required. The motion base would initially follow a roll acceleration, for example, and then drift back, even though the aircraft was still at the full roll attitude. So a motion base with washout was considered as a way to reduce the conflict between the visual and vestibular inputs that occur in a fixed-base simulator. Washout is a compromise: it does not produce the full motion environment of flight, but it provides more cues than a fixed-base simulator.

Designing the washout required a bit of trial and error. A 12 deg roll would be followed closely by the motion base, but for larger rolls, the washout would begin to reduce the response. On the basis of the work of Guedry, they knew that the time constant of the semicircular canals was on the order of 10 sec, so they guessed that the critical time constant for the washout would be somewhere between 1 sec and infinity (which is equivalent to no time constant--a 1:1 motion case). They swept through the time constants in the simulator with the test pilot giving his opinions about the simulation characteristics. With an acceleration time constant of 2-3 sec (2-3 deg/sec/sec), the visual and motion systems were quite acceptable to the pilot. With time constants less than 2 sec, he reported nausea and related problems, much as in the fixed-base mode. There was also the tendency for the pilot to overcontrol, both in fixed-base and short time constants. Longer time constants created other problems, because, while the roll was accurate, the linear acceleration was absent in the simulator (but present in the aircraft).

Although the tendency toward sickness was greatly reduced with a time constant of 2-3 sec, some traces remained. This may have been due to visual distortions, as mentioned by Miller and Goodson (1958), who evaluated

a helicopter simulator with a similar type of visual system.

Head movements are another subtle but important factor. Pilots in the simulator tended to roll their heads back toward the upright in a roll maneuver, perhaps in an attempt to maintain a stable visual image. Later it was verified that the pilots did the same thing when flying the helicopter. With the simulator in the fixed-base mode, the pilots again tried to decrease the angle of the horizon, but in this case the head movement was in the opposite direction--with the roll rather than against it.

Overall, approximately 75 percent of the experienced pilots who flew the simulator in the fixed-base mode had problems with sickness. With the moving base, only about 10 percent indicated any symptoms. Anecdotal evidence suggests that these conditions may have the opposite effects on inexperienced pilots. The simulator operator/technician learned to "fly" the simulator in the fixed-base mode. Subsequently, when the motion base was added, he experienced symptoms of sickness. The implication is that his prior adaptation to the sensory conflict conditions in the fixed-base mode made him susceptible to sickness when the visual and vestibular sources of motion information were rearranged by adding the motion base.

A device like this simulator would be a good tool for research on simulator sickness. One of the lessons to be learned from this experience was that, in this particular situation, a washout time constant of 2-3 sec, which is considerably less than the washout time constant of the semicircular canals (about 10 sec), seemed sufficient to reduce sensory conflict to generally tolerable levels. One might guess that a simulator time constant that matches the sensory one would be best, but it may be that as one increases the time constant toward 10 sec, the intravestibular conflict (canals versus the otoliths) are magnified. The best goal may be a perceptual realism rather than a physical realism in the simulation.

The changes in manual control performance that occurred as a result of the motion base were very small. Simulator design must take into account both performance and sickness.

Working on the visual systems will probably help to reduce the incidence of simulator sickness. Using a motion base and manipulating the acceleration time

constant should not be construed as the solution to the simulator sickness problem, although they worked in this particular case, with a certain type of aircraft, certain maneuvers, etc. In cases of more extreme maneuvers, alleviation of simulator sickness by "beefing up" the motion base would be a very impractical approach. A motion base is not a quick fix to the simulator sickness problem.

Tilt is another feature on many motion-base simulators, used to simulate linear acceleration. It is important to be very careful of this technique because the human senses are very good at perceiving tilt for what it is--a change in pitch attitude rather than a translatory acceleration. According to Guedry, the otolith system provides both a position signal, i.e., static tilt relative to gravity, and a transient (phasic) response to change in position relative to gravity. Horizontal linear acceleration, within certain frequency and magnitude limits, are perceived as linear velocity rather than tilt, probably because the transient otolith signal is unaccompanied by a complementary semicircular canal signal.

Design Characteristics of Automobile Simulators*

There are many parallel issues in the design of driving simulators and flight simulators. Due largely to their custom-made nature, high cost, and the large driving population, most driving simulators have been research tools rather than training devices. Several years ago it became apparent that research using a number of different driving simulators was hindered by the subjects' experiences of symptoms akin to motion sickness after even brief exposures (Reason and Diaz, 1971). The incidence of sickness in research driving simulators is difficult to document; however, in several cases symptoms have been quite overt and subjects have voluntarily ended driving trials due to imminent emesis.

The basic concepts of the sensory conflict theory (discussed in a later section) seem to apply to the problem of simulator sickness in driving simulators. Closed-loop delay, over and above normal vehicle

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*This section was drafted from the workshop presentation by John G. Casali.

dynamics delay, seems to be particularly important. Historically, driving simulators have tended to have less computational power for the vehicle dynamics and display systems than flight simulators. And the motion-base systems have in many instances been cruder than those used for flight simulation, although this is changing of late. For these reasons, there have sometimes been closed-loop lags in the system that are perceptible to the drivers, which may contribute to cue expectancy conflicts and also degrade vehicular control.

Automobile simulators also face the same problem of tilt. Certain automobile simulators have used oversize roll and pitch motions to simulate lateral and longitudinal transactions, respectively. Instances of driver discomfort and manual control difficulties have been reported for several of these devices. Apparently the vestibular system is not easily fooled into believing that tilt (angular position) is equivalent to a linear acceleration. A potential conflict may arise when the subject perceives the motion as rotational, when the motion cue that is anticipated in response to control input is primarily translational.

In an attempt to catalogue simulator characteristics that potentially contribute to simulator sickness, it was found that subjects in about 10 of a total of 25 driving simulators had reported motion sickness. Both fixed- and motion-base simulators were represented among those with symptoms.

Casali and Wierwille (1980) performed a complete factorial experimental study of driving simulator sickness using a modified version of the Virginia Tech simulator. This device includes a CGI 50 deg (horizontal) FOV, monochrome display of a two-lane highway, a 4 df motion base (does not include pitch and heave), and a full sound system. The lateral translation cue was replaced with an oversized roll cue, and a 300 msec pure delay with smoothing in the closed-loop dynamics was artificially introduced. The subjects' view of the surrounding room was occluded by a narrow, boxlike cab. All these "degraded" simulator variables had some negative effects, either in increasing vehicle control problems or inducing mild symptoms of sickness. They found no profound cases of simulator sickness, however, from more than 1,000 subjects in various studies.

A test of field dependence was used to match subjects in the study. They found no relationship between this

measure and motion sickness, contrary to previous results reported by Barrett and Thornton (1968), who found that extremely field-independent subjects were more susceptible to sickness than field-dependent subjects in a driving simulator. However, Barrett and Thornton also reported more severe symptoms of sickness in their experimental trials than Casali and Wierwille found in their study, perhaps accounting for the different results of field dependence.

Because of the multivariable nature of driving simulator sickness, it is difficult to design research that will allow enough variables to be included in a factorial design to allow proper assessment of the interactions. This same problem applies to studies of flight simulators.

THEORIES OF MOTION SICKNESS AND ADAPTATION

A theoretical understanding of the physiology and etiology of motion sickness is important in solving the problems associated with simulator sickness. The workshop participants reviewed some of the more prominent theories that have been suggested on the physiological factors involved. This section opens with an overview of motion sickness as an adaptive response to the stimulation of the vestibular system.* It then discusses sensory conflict theory, the most persuasive argument to date. Next it describes studies of adaptation to rearranged sensory inputs, illustrating how people adjust to the conflicting messages from the visual and vestibular systems. It then describes adaptive changes in oculomotor systems. The section ends with some comments on theories of motion sickness and some questions for further research.

The Greek word naus means ship and that is the origin of the words nausea and nautical. It was once thought that the sea brought about sickness by acting on the stomach; it was some time before it was known that motion rather than the sea itself causes sickness. In 1882 William James reported that motion causes sickness by acting on the vestibular apparatus rather than the stomach (James, 1882). The term motion sickness is

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*This overview was drafted from the workshop presentation of Ken E. Money; for a comprehensive review of motion sickness, see Money (1970), Reason and Brand (1975), Benson (1973), and Kennedy and Frank (1985).

usually defined as sickness caused by motion.

Treisman (1977) addressed the evolutionary significance of the emetic response of motion sickness: How could vomiting in response to a dynamic environment contribute to the survival of the species? He suggested that the significance of the emetic response was in the expulsion of ingested toxins from the body. That is, vomiting occurs because the brain interprets the stimulus as if it were a poison.

Treisman's "poison theory" provides the basis for Money's view of motion sickness, that it is the activation, by motion, of the vestibular mechanisms that normally facilitate the emetic response to poisons. This view implies that motion sickness is a response to the stimulation of the vestibular system and that it involves the unnatural activation of a normal physiological mechanism, i.e., vomiting in response to poison.

It is well known that central vestibular units can be driven by stimulating the ambient visual system (Waespe and Henn, 1977, 1978). Therefore, it is not surprising that the vestibular mechanisms can be activated by visual stimulation, as they are in simulator sickness. It is also well known that in the absence of the vestibular system, motion sickness cannot occur. Someone who does not have a vestibular apparatus is absolutely immune to motion sickness and, presumably, to simulator sickness as well, although this has not been demonstrated directly.

The absence of motion sickness in subjects without a vestibular system has been demonstrated many times, in both human and animal subjects. In a classic study of labyrinthine defective subjects (LDs) aboard a ship in the North Atlantic, all of the experimenters and the crew got sick, but the LDs did not (Kennedy et al., 1968). They were terrified and praying, but not sick. This experiment also dealt a blow to the notion that anxiety gives rise to motion sickness; at least it does not in LDs. There is also no reason to think that anxiety plays a major role in simulator sickness.

Money and Cheung (1983) tested the hypothesis that the surgical removal of the vestibular system should leave an animal with a defective response to poisons--which is one of the four physiological functions of the inner ear (i.e., hearing, body balance, control of eye movements, and the emetic response to poisons). The emetic response of dogs to poisons was measured before and after surgical removal of the inner ear. The poisons

used in the study included lobeline, nicotine, apomorphine, and pilocarpine. The results of the study were that the dogs no longer vomited in response to motion and that their emetic response to several poisons was greatly reduced. For several other poisons, the emetic response was not reduced. There are a number of mechanisms by which poisons can induce vomiting, and the vestibular system seems to be active only in some of them.

Money and Cheung concluded that there is a normal vestibular facilitation of the emetic response to certain poisons. This supports the view of motion sickness given above, that stresses the central role of the vestibular system and at the same time indicates that it results from an activation, by motion, of a normal response. Motion sickness is a normal response both for a wide variety of species when confronted with certain conditions of real (or visually mediated) motion. We all use the term motion sickness, but it is not really sickness: it is a normal physiological response. (There are, of course, dozens of mechanisms whereby vomiting can occur, and motion sickness is just one of them).

Treisman's poison theory gives credence to the sensory conflict or sensory mismatch theory of motion sickness (described in detail below), which is a useful way to think about the problem of motion sickness and can provide a meaningful framework for research.

The evolutionary survival value of a physiological mechanism such as that suggested by Treisman is significant. It is accessible by stimulation arising from peculiar motions--motions that exceed the normal dynamic limits of the vestibular system. When exposed to such motion, the vestibular system therefore sends information that is false (or distorted). The brain then recognizes these inputs as false because they are in conflict with other information about motion from vision, from another part of the vestibular system, or from proprioception. The result is motion sickness. It is the false information from the vestibular system that becomes the stimulus for the brain's vomiting center.

Sensory Conflict Theory*

A good starting point in understanding the role of sensory conflict in motion sickness and simulator sickness is to consider sensory inputs from the vestibular, visual, and proprioceptor systems in natural movements in everyday conditions. Children spend years perfecting control of eye, head, and limb movements, first crawling, then walking and running. With maturation and practice, control of motion becomes skillful and hence largely automatic.

In the normal adult, the vestibular system works in close coordination with the visual system to stabilize the eyes relative to selected earth-targets during walking, running, and turning movements, and the three systems work in close harmony in maintaining control of motion during locomotion. Typically, most of this coordination occurs automatically and without conscious awareness--until something goes wrong, e.g., a partial unilateral loss of vestibular function. An afflicted individual will be quite disturbed and acutely aware of the challenge to his or her control of movement. Blurred vision (oscillopsia) will probably result from turning the head and nausea and vomiting from persisting in moving about. However, by moving about with care, in time the disturbing symptoms usually will disappear as he or she adjusts to the new sensory inputs that occur during movement.

In some respects the normal adult moving about on the earth is like a skilled pilot, using skills developed over the years in controlling whole-body motion relative to the earth. The pilot, whose life depends on the skilled control of motion of the aircraft, becomes an integral part of the motion control-loop of the aircraft. With experience the sensory feedback from control actions is used as an important part of the control loop, and, as in any skilled perceptual-motor control performance, the relations between control actions and the unique feedback induced by the forces and torques of flight recede from conscious awareness (i.e., the feedback is used automatically). Only deviations from expected sequences of sensory feedback achieve conscious awareness and are assessed as potential indicators for corrective actions.

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*This section was adapted from the workshop presentation of Frederick E. Guedry.

A quotation from Melvill Jones (1974:874) puts the foregoing in neurophysiological perspective:

> There is good evidence that much of our normal motor control is organized not merely as an ongoing interaction between continuously operating automatic Sherringtonian reflexes, but rather as centrally released preformed packages of programmed neural information. One might well guess that adaptation to new requirements could be relatively easily met by merely reprogramming relevant patterns of the outgoing central neural discharge. However, there is a growing body of research findings which indicates that even cortically released patterns of motor drive are not devoid of early interaction with corresponding sensory mechanisms. For example we now know that such a central discharge of motor drive is not only destined to activate muscles through relatively direct connections with spinal motoneurones, but also, through collateral branches of central fibers, to act directly upon SENSORY neural relay stations in spinal cord pathways. Thus the corticospinal (Pyramidal) motor tract, not only descends to spinal cord networks generating motoneurone activity to drive skeletal muscles, but also sends many collateral branches to synapse directly on second order afferent neurons in the sensory gracile and cuneate nuclei of the dorsal columns of the cord.
>
> A clue to the functional implication of this rather surprising fact is perhaps to be found in somewhat analogous mechanisms operating in the periphery. For example it is now well established that in many circumstances both alpha and gamma motoneurones, innervating the main (extrafusil) and muscle spindle (intrafusil) fibers of a skeletal muscle, can be coactivated at the same time. It has been proposed that when the combined alpha-gamma program operates "according to plan," the muscle spindles contract (or relax) in just such a way as to null out any change in their sensory discharge caused by mechanical shortening of the main muscle. This rather neat arrangement would ensure that if all went well (i.e., according to plan), then the central nervous system (CNS) would not be bothered with unnecessary sensory information. By contrast, if the "intended" response was not achieved, then

needed sensory information would indeed reach the CNS which, in turn, would presumably modify the next motor command. This capability to recode rearranged motion inputs raises several questions.

Thus, the experienced pilot is apt to be disturbed and perhaps made sick by the unexpected sensory feedback in simulators that may differ considerably from the feedback engendered by the same control actions in the aircraft. This statement concerning the reason for the pilot's disturbance is based on what is called the sensory conflict theory of motion sickness.

In many situations that provoke motion sickness, no single component of the motion stimulus is either strong or nauseogenic, but in combination the sensory stimuli induce sickness. In other situations, highly nauseogenic stimuli can be rendered benign by the addition of other motion stimuli, apparently because the added stimuli remove the conflict. These lines of evidence offer strong support for conflict theory. A brief description of two experiments will serve to illustrate these points.

Tilting the head 30 deg laterally toward the left shoulder during sustained constant speed clockwise whole-body rotation about an earth-vertical axis (velocity 1.0 rad/sec) produces a cross-coupled ("coriolis") stimulus to the semicircular canals resulting in a sensation of forward tumble (about 90 deg out of the plane of the head movement). This well-known stimulus is disturbing and nauseogenic to most people, especially when repeated several times, yet no single component of the stimulus is strong or even disturbing. The vertical semicircular canals have received an "angular impulse" equivalent to a velocity change of 0.5 rad/sec. The horizontal canals have received even less stimulation. Thus the canal stimuli are not strong or disturbing. A 30 deg lateral tilt stimulus to the otolith system is also neither disturbing nor nauseogenic. However, in combination these stimuli are nauseogenic because in terms of the conflict theory, the semicircular canals have signaled rotation of the head about one axis while the otolith system (influenced primarily by the gravity vector) has signaled change in head position about another axis. Thus there is an intralabyrinthine conflict, and in addition there is conflict between the intended head movement and the perceived motion of the head and body. The central nervous system has been presented with sensory input calling for compensatory reactions in two

different directions at the same time; the sensation is confusing and the situation is nauseogenic.

If the same head tilt is made during the same rotation velocity of the body but while the initial angular acceleration is in progress, then exactly the same cross-coupled stimulus occurs, but it is not at all disturbing, disorienting, or nauseogenic. The reason is that the angular acceleration vector has been added, and its vectoral resolution with the cross-coupled vector yields a resultant vector that remains aligned with gravity (Guedry and Benson, 1978). In this case the semicircular canals signal rotation about an earth-vertical axis that is in alignment with the direction of gravity signaled by the otolith system, and thus there is no conflict. Almost the same degree of amelioration of effects of cross-coupled semicircular canal stimuli can be produced by horizontal optokinetic aftereffects (Guedry, 1978) (Figure 2), which appear to modulate activity in the vestibular nuclei as though the horizontal semicircular canal had been stimulated (Henn et al., 1974). Observations like these lend credence to the conflict theory and illustrate that the patterning of sensory cues can play a significant role in the nauseogenic characteristics of motion stimuli.

A conceptualization (Benson, 1978; Reason and Brand, 1975) of the sensory conflict theory of motion sickness is shown in Figure 3. Basically, the idea is that when sensory inputs from the eyes, semicircular canals, otolith organs, and other mechanoreceptors are in repeated or continual conflict with regard to the state of motion of the body relative to the earth, then adaptive changes of the central processing of these inputs must occur to yield efficient control of motion in the unusual motion environment. Meanwhile, the mismatched inputs also set off the motion sickness syndrome, perhaps for reasons elaborated earlier. Reason and Brand (1975) propose that most sickness-provoking conflicts are of three types, shown in Table 2, and they cite examples for each type. It is possible that all conditions that induce motion sickness are not subsumed by the sensory conflict theory, but most cases of simulator sickness are probably attributable to sensory conflict because simulators seldom introduce strong accelerative stimuli. One of the more compelling arguments for the role of sensory conflict in simulator sickness is that experienced pilots seem to be more susceptible to simulator sickness than are novices.

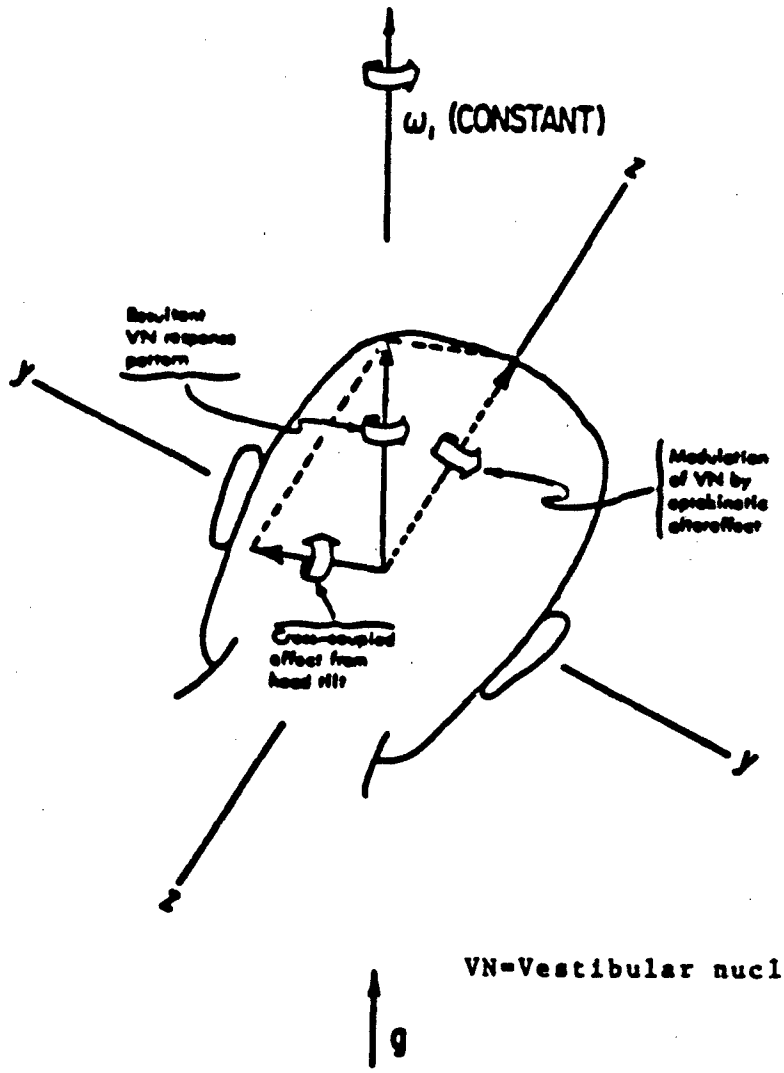

FIGURE 2 Cross-Coupled Semicircular Canal Stimuli

Source: Guedry (1978).

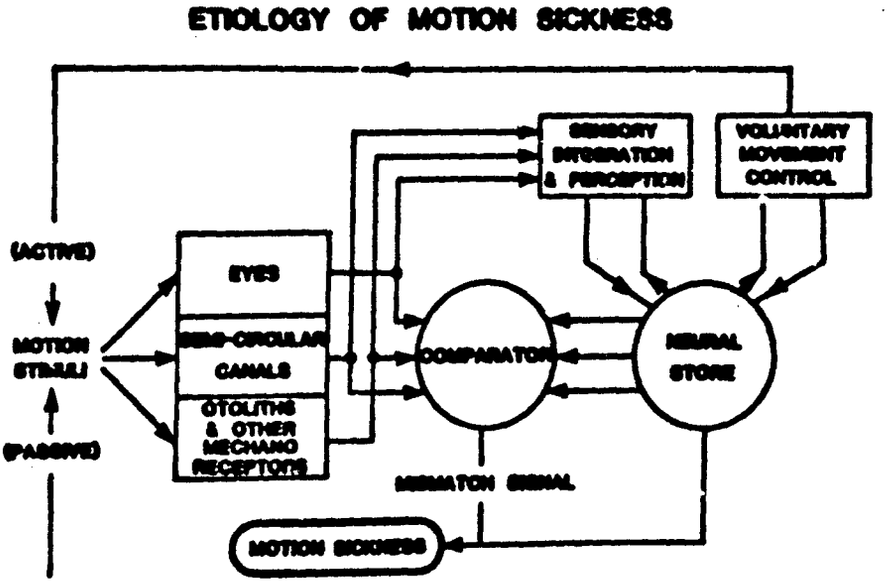

FIGURE 3 Etiology of Motion Sickness

Source: Benson (1978).

Adaptation Studies*

Much of the basis of the sensory conflict theory of motion sickness was formed from studies of adaptation to experimentally contrived rearranged sensory inputs involving motion cues. Two rather extreme experimental situations illustrate the essential points of such studies.

In the first situation, individuals moving about in an enclosed rotating room are subjected to a number of rearranged sensory inputs. Because the room is rotating, any movement along a straight line relative to the floor of the room is a curved path relative to the earth (vision-proprioception conflict). Any tilting movement

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*This section was adapted from the workshop presentation of Frederick E. Guedry.

TABLE 2 Types of Sensory Conflict

Type 1	When A and B simultaneously signal contradictory information
Type 2	When A signals in the absence of an expected B signal
Type 3	When B signals in the absence of an expected A signal

Note: A and B represent members of a pair of normally correlated receptor systems: visual-inertial and canal-otolith.

Adapted from Reason and Brand (1975).

of the head produces semicircular canal responses roughly at right angles to change-in-position signals from the otolith organs, and thus there is the intralabyrinthine conflict described above.

The second situation involves the use of right/left reversal dove prisms. When an individual moves about in the normal force environment, visual feedback is right/left reversed.

Initially both of these situations induce severe problems with control of movement and motion sickess. Within several days, control of motion is improved and sickness subsides. In the course of this adaptation, changes in sensorimotor reflexes and in perceived motion occur. Upon return to a normal environment the recoded sensorimotor system causes problems.

After adjustment to the rotating room, head tilts in a normal static environment induce perceptions of motion and a vestibulo-ocular reflex (VOR) at right angles to the head movement plane and thus 90 deg displaced from a normal response. Control of body movement in the normal environment is severely degraded, and nausea and vomiting are common complaints.

As adjustment to reversing prisms occurs, locomotor performance improves, the gain of VOR produced by passive whole-body oscillation in the dark is reduced and, after a few days, the phase of the eye movements shifts and

eventually achieves almost 180 deg of phase reversal. At night upon retiring, some subjects experience feelings of turning to and fro or unidirectionally.

After removal of the reversing lenses, locomotor disturbances are pronounced and the gain of the VOR to passive oscillatory stimuli in the dark requires several days for recovery, although the phase of the oculomotor response returns fairly quickly (2 hours) toward normalcy. Voluntary head turning produces blurred vision and illusory movement of the world, an effect that may persist for two or three days.

The time course of adaptation seems to vary, not only according to the severity of the sensory conflict but also according to different aspects of responses selected for measurement. For example, in the experiments by Gonshor and Melvill Jones (1976) in which subjects adapted to the left/right reversing prisms, the first evidence of VOR adaptation was gain reduction (revealed by sinusoidal oscillation in darkness); subsequently the VOR returned somewhat, but with a large phase shift to bring the VOR into accord with the reversed vision. After removal of the lenses, oscillation in the dark still revealed a large phase shift in the VOR but the phase shift dissipated in about 2 hours, whereas the gain of the response required several days for readaptation to normal levels. Thus, different components of the response system can have different courses of adaptation and recovery. In the slow rotation room studies at Pensacola (Guedry, 1965; Graybiel et al., 1965), recovery from prolonged rotation also seemed to reveal divergence of response changes. Some subjects, tested 3 weeks after exposure to 12 days of rotation at 10 rpm, reported return of vertiginous sensations and exhibited recovery toward normalcy of VOR responses to head movements during rotation, yet they did not experience motion sickness within a 2-hour exposure. Thus, motion sickness, which is one aspect of the overall response to an unusual motion environment, may have an adaptation and recovery time course that does not necessarily parallel other responses undergoing adaptive change.

These extreme examples of sensory rearrangement in sensory conflict studies are mentioned to make the point that substantial changes in perception of motion, postural control, and sensorimotor reflexes occur when individuals attempt to control body motion in rearranged sensory environments for protracted periods. The

beginnings of these effects can be seen after short exposures. Studies that form the basis of these descriptions suggest that the central nervous system commences rather quickly to recode itself for adjustment of perceptual and motor response to rearranged sensory inputs having to do with the voluntary control of motion.

They also raise several questions for further research. Simulators probably never introduce the extreme sensory rearrangement involved in these experimental studies. But the recoding of responses probably occurs more rapidly with less extreme situations (Collewijn et al., 1981). Unlike the studies just described, simulators do not require the individual to be exposed to the rearrangement situation continuously for extended periods. But how concentrated can simulator exposure be before some recoding occurs such that perceptual and sensorimotor reflexes are changed? The strong simulator aftereffects that have been occasionally reported suggest some reason for concern. How specific are changed sets of responses to the simulated flight control situation? The answer seems to be not very specific, since simulator aftereffects have occurred during normal locomotion, driving, and while lying down. These aftereffects suggest that something more than intellectual learning of procedures has occurred.

Oculomotor Adaptive Systems*

Many conditions that produce adaptive changes in oculomotor systems also produce dysfunction in the form of motion sickness symptoms. Viewing flight simulator visual systems may involve some of the very conditions that produce oculomotor adaptation. The results of laboratory studies of oculomotor plasticity may therefore be relevant to simulator sickness.

These laboratory studies have emphasized different aspects of the same problem. Studies with prisms have been very popular with psychologists, probably because of the traditional interest in perceptual adaptation, while optometric and physiologically oriented scientists have explored oculomotor adaptations. The two domains are, of course, related, and, in most if not all cases,

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*This section was adapted from the workshop presentation of Sheldon M. Ebenholtz.

changes in perception can be deduced directly from the known changes in oculomotor function.

Vertical divergence, for example, can be induced through the use of prisms (one eye elevated more than the other) with concomitant shifts in apparent target elevation, after removal of prisms, that are specific to each eye (Ebenholtz, 1978). Another common condition is induced asymmetric convergence or shift in lateral gaze. Both of these conditions lead to adaptation in gaze direction during the experience (training) followed by a period of readaptation in which the individual is returned to the normal environment. Adaptation in lateral gaze also leads to a change in apparent gaze direction and a shift in the lateral orientation of objects. Still another type of adaptation results from left and right sustained head orientation. For example, maintaining a 30 deg head rotation to one side for about 8-10 minutes may cause the subject to make an error (in the direction of the sustained position) when attempting to orient the head straight ahead and in describing the spatial orientation of objects.

At a theoretical level, these types of adaptations may be thought of as representing a resetting of the set-point or steady-state level of certain reflexes. The "doll reflex," for example, refers to the tendency of the eyes to roll downward and the eyelids to close when the head is rotated backwards (Ebenholtz and Shebilske, 1975). Conscious attempts to counter the doll reflex will, over time, lead to adaptation of the response (Shebilske and Karmichl, 1978), resulting in a change in both eye level and perceptual effects that include a change in the apparent elevation of a visual target. It is as though the gain of the system is modified and a new resting level established. This is an example of an adaptation in an otolith-dependent system, and it is interesting that merely sustained posture in the presence of a visual target is sufficient for adaptation in this oculomotor control system.

In each of these cases it is a negative feedback loop that operates to null error signals and thus maintain normal postural and oculomotor control in the face of perturbations of reflexive origin. On this basis we may be able to understand more clearly the meaning of the conflict theory of motion sickness, in terms of the processing of error signals in feedback loops in the nervous system. For example, one way of thinking about conflict is to consider it a demand for

change, i.e., an error signal in a negative feedback control system acting as a command to an adaptive nervous system to change its parameters.

The study of oculomotor function provides additional excellent examples of adaptive control systems. Thus, the resting level of vergence (phoria) shifts over time, and distance perception changes, as a result of maintaining fixation on a near target (Ebenholtz and Fisher, 1982) or from wearing prisms that induce a change in vergence (Schor, 1979). Another closely related adaptive system is that involved with the control of focus or blur. In this case, the resting level of accommodation is modulated by maintaining focus on a near or far target (Ebenholtz, 1983). Presumably, the longer an individual stays focused on the target, the more likely is the tonus of the ciliary muscle to change, modifying the resting level or dark focus of accommodation (Leibowitz and Owens, 1978). This is a feedforward type of control that complements the more widely understood negative feedback blur-control loop. The vergence and accommodation systems are represented in Figure 4, in which tonus control represents the adaptive feedforward loops, to which we are now referring.

There is a reciprocal relationship between these two types of control loops, and in the case of convergence and accommodation, they differ in their time constants, one (feedback) being on the order of 100-500 ms and the other (feedforward) capable of lasting for hours. This is a situation that could be described as functional symbiosis between a feedback and an adaptive feedforward loop. To the extent that the feedforward loop (tonus control) is successful, it takes the load off the negative feedback (blur or disparity) control loop. But the feedforward system is not "intelligent"; it can only be updated when an error exists in the negative feedback loop--hence the symbiotic nature of the relationship between the two control loops.

This symbiotic relationship between feedforward and feedback loops probably underlies adaptive plasticity. Updating the feedforward systems by changing their set-points allows them to be more effective, which, in turn, allows the error detecting feedback control systems to relax.

In unpublished studies of the resting state of convergence and accommodation, it was found that the aftereffects were functions of exposure duration and the magnitude of the change demanded of the feedforward

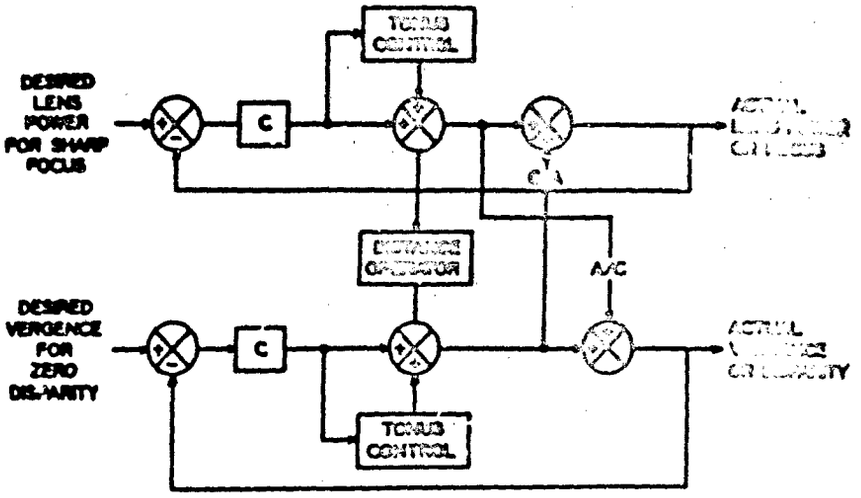


FIGURE 4 Vergence and Accommodation Control Systems

Note: C: controller; A/C: accommodative convergence; C/A: convergence-induced accommodation.

Source: Ebenholtz and Fisher (1982).

loop. These variables may also be critical for the time course (retention) of the aftereffects that occur with visual simulation systems.

Perhaps the single most significant adaptive control system for the simulator sickness problem is the vestibulo-ocular reflex (VOR). This is a compensatory ocular response that maintains the direction of gaze, enabling one to fix on a visual target while moving the head. It has no feedback loop of its own responding primarily to radial accelerative forces, but it does receive the benefits of feedback from other sources, such as the optokinetic, pursuit, and vergence systems (e.g., Post and Leibowitz, 1982). Dove prisms have often been used to reverse the VOR, making it appear, before adaptation, that the world turns with you, but at twice the head velocity. Motion sickness almost invariably occurs with this procedure, both when the prisms are initially worn and again after adaptation, when the prisms are removed and normal viewing is restored.

Adaptation of the direction and gain of the VOR has been reported in several studies using different procedures (Callan and Ebenholtz, 1982; Gauthier and Robinson, 1975; Melvill Jones, 1977; Miles and Fuller, 1974). The symptoms of motion sickness that so commonly occur in studies of adaptation of the VOR that there must be strong clues for understanding simulator sickness. We may be looking at the same problem, stemming from plasticity in oculomotor control systems, that can be induced experimentally or in a simulator.

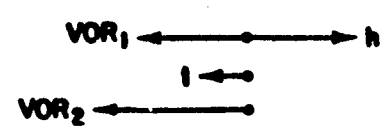
When the VOR is manipulated experimentally, a head movement produces a "slip" in the target image relative to the tracking eye, rather than maintenance of gaze. This slip of the optokinetic stimulus represents the error signal that the oculomotor contol system tries to eliminate, leading to a change in the feedforward signal that controls the direction and velocity of the VOR. This error signal may also be the neurological basis for conflict leading to the symptoms of motion sickness. Figure 5 displays the course of gain changes and directional plasticity in the VOR in vector diagrams representing the circumstances that initiate the adaptive response and those that result after adaptation has occurred. The diagram also permits the prediction of the direction of movement illusions that typically accompany VOR adaptations (Dubois, 1982).

Robinson (1976) has studied the physiological basis for adaptive plasticity in the VOR on the premise that an incoming velocity gradient, an optokinetic stimulus, is an important source of information that updates the VOR. The vestibular cerebellum (the flocculus) may be a region in which incoming visual information could perform the updating function. In support of this, ablation studies with cats have shown that flocculectomy eliminates the ability to adapt the VOR.

Under stroboscopic illumination, exposure to right/left reversing prisms produced some plasticity in the VOR but the gain was greatly reduced over that obtained with steady illumination, according to a study by Melvill Jones and Mandl (1979). Note, however, that nausea, which normally accompanies this type of adaptation, was absent. This supports the view that the velocity of the slip signal is important to the magnitude of the adaptation, since intermittent stimulation lowers the number of discrepant signals per unit time that the system is forced to cope with. This concept leads to the suggestion that we may be able to reduce the motion

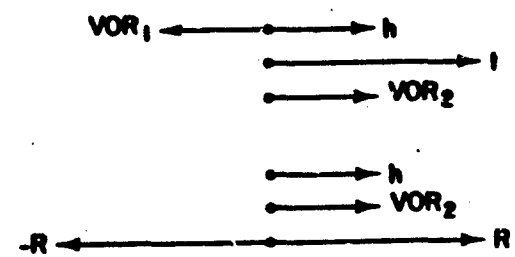
Gaze (R) = vector sum of VOR and head (h) velocities.

t = optokinetic and/or pursuit-target velocity.



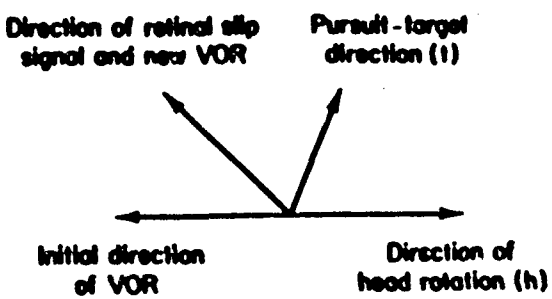
Conditions for training an increased VOR gain (g) typical of magnifying telescope lenses (e.g., Gauthier & Robinson, 1975):

After adaptation with g>1 illusory movement occurs in the anti-gaze direction (-R).



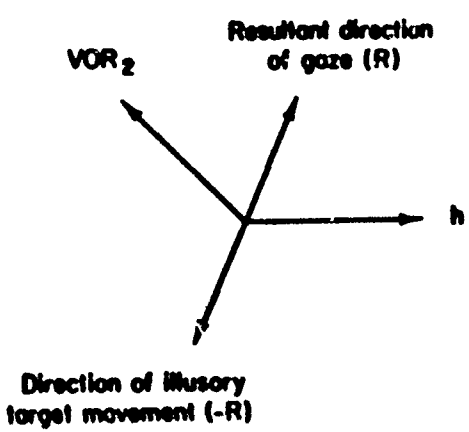
Conditions for training a reversal in the direction of the VOR (e.g., Melvill Jones, 1977).

After adaptation head movement induces illusory movement in a stationary target in direction -R.



Conditions productive of directional plasticity in the VOR (e.g., Collan & Ebenholtz, 1982).

During adaptation the salience of pursuit-target movement diminishes.



After adaptation the direction of illusory movement of a target fixated during head oscillation is opposite the direction of gaze.

FIGURE 5 Vector Analysis of the Conditions Productive of Increased Gain, Reversed Gain, and Directional Shifts of the Vestibulo-Ocular Reflex

Note: Cancellation of the resultant vector by the pursuit systems is a source of visual illusions.

sickness properties of a visual display by reducing the velocity of the input signals. The update rate would only have to be sufficient to support the apparent motion desired in the display.

These adaptations all seem to require an error signal. The plastic changes occur as a negative feedback system responds to this error (slip) signal. Exercising the VOR feedforward loop by itself, however, is not sufficient to permit either initial adaptation or readaptation back to "normal" levels.

The principle that emerges is that the very function of adaptation is to eliminate or reduce the load on the closed-loop feedback system by substituting a properly updated (adapted) feedforward signal. The evidence suggests that recurrent, systematic directional errors in negative feedback loops tend to generate adaptive responses in the associated feedforward loops. These adaptive shifts, in turn, serve to reduce the errors in the associated negative feedback loops. In short, negative feedback loops serve as the updating system for the feedforward loops.

Unresolved and systematic errors in negative feedback loops are not well tolerated by organisms. These signals, in addition to calling forth an adaptive feedforward response, also cause eye strain, dizziness, apparent motion, disorientation, and motion sickness. It follows from this hypothesis that the conflicts specified by the sensory conflict theory may be isomorphic with the conditions that produce sustained errors in negative feedback loops governing postural control.

There is strong neurophysiological evidence for the interaction between visual and vestibular inputs at the vestibular nucleus (Waespe and Henn, 1977; Daunton and Thomsen, 1979). It supports the notion that the visual stimulus modulates the output of the vestibular nucleus. If this output is modulated beyond some level, the symptoms of motion sickness are likely to occur.

Simulator sickness is visual stimulation producing nausea. According to the Treisman (1977) hypothesis and the supporting studies by Money, visual stimulation activates the vestibular mechanisms that normally facilitate the emetic response to poisons. There is probably also an adaptive response leading to a change in the gain, phase, and direction of the VOR and other vestibular-spinal reflexes.

The vestibular system, including the vestibular

nuclei and the vestibular cerebellum, have, as one essential function, the control of muscle tonus. This extends to the extraocular muscles and the control of eye movements, driving the muscles that contribute to head positioning, and those that contribute to the antigravity reflexes. This implies that it is critically important to identify the relationship between the stimuli (visual, vestibular, and proprioceptive) and the reflexes mediated by the vestibular system. If we can specify these stimuli (inputs) in the flight simulator, and the resultant motor responses, we will also have gone a long way toward defining and measuring conflict. If one can measure the linear and angular acceleration vectors that are operating instantaneously, then one can attempt to correlate them with the direction and magnitude of the optokinetic vector at that same time. The comparison of those two vectors could be a measure of conflict and of the likelihood either of ensuing adaptation or simulator sickness. This is at least a testable hypothesis, which, if supported, could lead to the development of effective countermeasures. This is of special importance, since one of the problems with the conflict theory is that it is too general to be tested.

As a final observation, it should be noted that substantial individual differences exist in susceptibility to the various forms of oculomotor adaptations, including VOR plasticity. The relationship between these potential indices and measures of symptoms on exposure to flight simulators remains to be determined.

Comments on Theories of Motion Sickness

By whatever name it is known--neural mismatch, cue conflict, perceptual decorrelation, or sensory incongruity--sensory conflict theory is the most common explanation for motion sickness. It postulates a referencing function in which motion information from vision, the vestibular system, and proprioception may be in conflict with the expected values of these inputs, based on a neural store that reflects past experience (Kennedy and Frank, 1983; Reason, 1978). (For a summary of sensory conflict theory applied to simulator sickness, see Kennedy and Frank, 1983.)

Although sensory conflict theory often seems

explanatory after the fact, it is not sufficiently defined to enable us to make predictions about the magnitude of the conflict in various dynamic situations. Several types of conflict seem to apply: between sensory modalities (i.e., visual-vestibular), within a sensory system (i.e., otolith-canal or ambient-focal); or between expected and experienced patterns of motion stimulation. Furthermore, it has been suggested (Benson, 1978) that phase and gain parameters of various sensory systems may play a role in the magnitude of the conflict. This implies that the conflict cannot be precisely defined based on the physical (distal) stimulus properties, but must also incorporate the characteristics of the sensory transducers, which are subject to variation.

Conflict theory does not explain why sickness fails to occur in certain situations with apparent conflict, and, conversely, it does not clearly predict sickness in certain situations such as vertical linear oscillation in which sickness has been observed (O'Hanlon and McCauley, 1974).

Despite the deficiencies of sensory conflict theory, it is accepted by most researchers as a good working hypothesis for motion sickness, and it is in accord with much of the data. The questions it raises are valuable challenges for improving the state of the theory.

RECOMMENDED COUNTERMEASURES FOR EXISTING SIMULATORS

As we can see from the current state of knowledge, a great deal still remains to be learned about the effects of simulator sickness. At the same time, simulators are an effective training device and the simulators currently in use will be operational for some time. Thus an important immediate goal is to find ways for users to cope with the adverse effects of simulator sickness.

The workshop participants felt that simply recommending directions for basic research on the fundamental mechanisms was not adequate. They discussed a number of hypotheses for ameliorating simulator sickness in current simulators and generated a preliminary list of suggested countermeasures (see Table 3, which augments a list suggested earlier by Frank et al., 1983). Although these countermeasures remain to be validated, this tentative list can be the source of ad hoc solutions to the practical problems.

Freeze. Although freeze can be a valuable instructional feature in a simulator, it can also be very disconcerting if used indiscriminately. Freeze in an off-horizon situation, i.e., 30 degrees wing down, is not good procedure. Anecdotal evidence suggests that when a fixed-base simulator is frozen and the pilot is asked to exit, some pilots will refuse to get out. Others crawl out very carefully, feeling that they may fall, even when they know it is a fixed-base system. If freeze is used judiciously it should eliminate some feelings of discomfort without reducing the training value of the freeze feature.

TABLE 3 Recommended Countermeasures for Current Simulators

Judicious use of freeze and reset
Avoid prolonged and intensive exposure
Remove scene content before entering or exiting
Tune/calibrate visual and inertial lags
Avoid motion at a frequency of 0.2 Hz
Briefing pilots on likelihood of symptoms
Preadaptation and incremental exposure
Post-simulator visual-motor games
Syllabus/scenario solutions (reduce maneuvers):
Turns-while-taxiing
High-acceleration maneuvers
Inversion or steep turns
Reduce scenario turbulence
Check personal and medical status
Avoid large head movements
Autogenic feedback therapy (biofeedback)
Motion-sickness medication

Reset. The reset function usually is used in conjunction with freeze. After the freeze or at the end of some scenario, the instructor may decide to initiate a new scenario by pressing the reset button. The pilot may see many miles of video stream by, perhaps backward, within a second or two. This can be very disconcerting and sometimes disorienting to the pilot. The screen should be blanked or the pilot should close his eyes when the reset function is used.

Exposure Duration. The data generally indicate that exposure duration contributes to sickness. Limiting exposure duration can provide temporary relief from building symptoms. The recommendation to limit exposure duration and intensity is related to the "incremental exposure" suggestion.

Maneuver Intensity. On the basis of experiences in the SAAC and anecdotal information, the number and intensity of flight maneuvers in a simulator session appears to be related to simulator sickness. The high

incidence of symptoms in the SAAC occurred when the pilots were flying 35 air combat maneuvering engagements in daily two-hour sessions.

Visual Scene Termination. At the end of a scenario or a simulation session, the visual scene should be removed before the pilot is asked to leave the simulator. The visual scene should also be off when the pilot enters the simulator.

Visual and Inertial Lags. Periodic checks of the visual and inertial lags are recommended. Even if they met specifications during acceptance testing, they should be checked and calibrated regularly.

Motion Spectrum. Research has indicated that whole-body motion at a frequency of about 0.2 Hz is the most nauseogenic. For motion-based simulators, sickness may be less likely if the simulator motion at that frequency can be reduced.

Briefing. Providing a briefing for pilots who are unfamiliar with the simulator can be helpful by indicating that the experience of symptoms is not an abnormal reaction. Giving them knowledge about the syndrome reduces the anxiety that sometimes accompanies the development of unexpected symptoms.

Preadaptation and Incremental Exposure. Research has shown that tolerance to head movements during rotation can be developed by a series of exposures at an acceleration level low enough to avoid symptoms. This technique of incremental exposure also may be effective for simulator sickness.

Visual-Motor Games. There is some evidence that active body movement facilitates readaptation to a normal inertial environment. Playing a game like ping pong that involves hand-eye coordination and body movement could help speed recovery from simulator aftereffects.

Syllabus Planning. The dose effect in simulator sickness is probably related to the frequency and intensity of flight maneuvers and the duration of the simulated mission. Planning the training syllabus to reduce the dose (especially in the first few days) should reduce the incidence of symptoms.

Scenario Turbulence. In some simulators, turbulence is an instructional variable. Avoiding high turbulence levels is recommended when problems of simulator sickness are anticipated (i.e., with an experienced pilot who has a mild case of flu and has not flown in the simulator for an extended period of time).

Personal Health Status. Hangover, flu shots, etc. can contribute to an individual's susceptibility to simulator sickness, and should be avoided prior to "flying" a simulator. (Chewing tobacco also is not recommended.)

Reduce Head Movement. Large, rapid head movements during angular motion can cause vestibular coriolis effects, and head movements during visually represented angular motion can cause pseudocoriolis effects. Both of the effects are excellent stimuli for motion sickness. Excessive head movement is not recommended in an environment in which there is either actual motion or visually implied motion effects. A motion-base simulator has both.

Autogenic Feedback Therapy. Research at NASA-Ames and the Air Force School of Aviation Medicine has found autogenic feedback therapy (biofeedback) to be effective in the reduction of motion sickness symptoms in provocative laboratory tests.

Medication. Many medications have been applied for motion sickness; some quite successfully. Flight surgeons can recommend medication to reduce symptoms without interfering significantly with performance.

No firm data exist on the effects of simulator sickness or its aftereffects on the probability of disorientation during flight. A pilot suffering from simulator sickness, however, is a likely candidate for disorientation in flight and should exercise caution until the effects have subsided. For most people this is likely to take 1 or 2 hours, but for others the effects are intermittent and have been reported as much as 10 hours after an intense ACM simulator session (Kellogg et al., 1980).

The operational commands should become aware of the problem of simulator sickness. In one case, for two Navy simulators, a rule was established that pilots

could not fly an aircraft for 12 hours after their first exposure to the simulator; that rule has been relaxed over the last year or so, but the problem of simulator sickness remains. A "12 hour no fly" rule may not be warranted in the absence of supporting data, if such a rule plays havoc with scheduling of the simulator or the aircraft. Some countermeasures will clearly be more practical than others in the context of operations and flight training.

RECOMMENDATIONS FOR RESEARCH

Numerous topics for research surfaced in the three days of the workshop. At this stage there are more questions than answers about simulator sickness. In this section we attempt to summarize the many research suggestions that were generated by the participants. Although the limitations of the workshop precluded our giving priorities to the many research suggestions, they are categorized here by objective:

1. Problem Definition
2. Theory/Model Development
3. Methodology
4. Determination of Causes and Processes
5. Validation of Candidate Countermeasures

Problem Definition

1.1 How extensive is the simulator sickness problem? The magnitude and consequences of simulator sickness must be determined in order to make reasonable recommendations about solutions. Field studies of the incidence of the problem are needed. (The Naval Training Equipment Center plans to undertake such a study in 1984.)

1.2 A coordinated survey of all military flight simulators should be conducted using a standardized questionnaire. Comprehensive results should be compiled, listing the precipitating causes and resultant symptoms. To ensure confidentiality, the results of such a questionnaire should be compiled by an agency,

such as NASA, that does not represent a threat to the pilots and air crew who respond.

Theory/Model Development

2.1 The sensory conflict theory presents a good starting point for the study and understanding of simulator sickness. Simulator designers need the type of information that can be communicated in a good model. However, in order to be really helpful, the terms will have to be reduced to mathematical statements of the nature and extent of the conflicts that cause the problem. Such a mathematical representation (e.g., Oman, 1980) will help make the theory testable and will guide the necessary research.

2.2 Sensory conflict theory in its present form does not satisfactorily address the coherence or predictability of the sensory mismatch. It is likely that adaptation occurs only in response to predictable sensory mismatches. We need to know more about both sickness and adaptation as a function of the predictability of the sensory conflict.

2.3 We should be able to quantify the conflict if we have accurate measures of the acceleration imparted to the head and compare that vector with the vector required by the motion pattern implied by the simulator visual system. The difference between these two vectors would represent a good first approximation to the quantification of the sensory conflict. Using reflexive responses such as the vestibulo-ocular reflex (VOR), would make predictions possible about the outcome of visual and vestibular vectors, and the discrepancy between the actual and predicted VOR could be considered an index of the mismatch.

Some workshop participants suggested that the VOR approach may be impractical in a simulator environment, in which the visual display is very complex, pilot head movement is complex, and there are many more variables than in the laboratory.

Other researchers caution that phase relationships of various sensory processes can lead to peculiar effects. Even if the amplitudes of visual and vestibular inputs are equal, sensory mismatch can result from phase differences in the two sensory systems. This makes it difficult to quantify the conflict on the basis of the external dynamics alone.

Quantification of sensory conflict is a major research goal for the study of simulator sickness.

Methodology

3.1 Because the simulator sickness problem involves so many variables (see the appendix for a summary list), consideration should be given to using efficient research methods such as economical multifactor designs (Simon, 1979). We should determine the variables with the greatest potential payoff and investigate them first.

3.2 The reliability of the dependent measures of simulator sickness need to be investigated. With what consistency do the symptoms of simulator sickness occur in individuals? If there is considerable fluctuation in an individual's motion sickness response system, the reliability of the criterion measures may be, say, $r = 0.5$. We need to develop reliable criteria for effective analysis of the problem.

3.3 Physiological indices of motion sickness should be investigated. It was well established, however, that people tend to have idiosyncratic patterns of symptom development. The possibility of developing individual profiles of symptom development should be investigated.

3.4 Individual differences are important and should be accounted for in any comprehensive model of simulator sickness. There are large individual differences in roll vection, for example, and also apparently in simulator sickness, yet we have no data on their correlations. We should seek to predict susceptibility to simulator sickness in individuals on the basis of knowledge of other sensory processes.

3.5 The gain of the VOR may be a good measure of simulator aftereffects and adaptation. The use of the VOR would require the development of techniques to measure pilots' head and eye movements accurately in a simulator; some researchers suggest that it may be impractical to obtain such measures. The feasibility of obtaining accurate measures of the visual scene and the pilots' head and eye movement in a simulation environment should be determined.

3.6 Measurement of the motion in the visual scene is not straightforward, particularly translational movement. What is the proper measure of the visual stimulus? There is the physical motion on the screen and there is the implied (perceived) motion of the pilot.

For example, a lateral-quick-stop maneuver at two different altitudes may involve the same perceived body motion by the pilot but different motion metrics on the screen. Perceived distance may be an important variable that relates displayed motion and perceived body-motion.

Determination of Causes and Processes

4.1 A task analysis is needed to index the tasks being performed in the simulator relative to the incidence of simulator sickness. The visual components of the task may be important as well as pilot workload and flight maneuvers.

4.2 Head movement analysis is needed. An example of a complete record of head and eye movements during a simulator session is needed to determine head/eye coordination and its relationship to the visual display system. Also, patterns of head and eye movements of experienced and inexperienced pilots should be analyzed both in the simulator and in the aircraft for various maneuvers. Experienced pilots may have established patterns of head movements in the aircraft that are inappropriate for particular simulator dynamics.

4.3 The most direct approach to investigating simulator sickness would be to manipulate the visual display variables and the motion-base variables in a simulator. A research simulator that enabled these variables to be changed easily would be needed. Visual system FOV and scene detail, for example, could be studied in combination with various lags and distortions of the motion base, all with a variety of simulated aircraft maneuvers. Such a research simulator could also support study of the efficacy of candidate countermeasures.

4.4 The contribution of other sources of motion information, such as G-seats and helmet-loaders, needs to be studied. How is this information integrated into the pilot's perception of spatial orientation and body dynamics? Do these devices contribute to simulator sickness? Could they contribute to prevention?

4.5 Is there an optimal FOV, large enough to enable positive transfer of training from the simulator to the aircraft, but small enough to reduce the incidence of simulator sickness?

4.6 More information is needed about the phase and gain relationship of the various sensory processes

involved. For example, if the otolith response is phase advanced at a frequency of 0.2 Hz but vision is not, this could be the basis for conflict, at least in some cases.

4.7 More basic research is needed on vection. The effects on vection of leads and lags in the visual and inertial systems should be investigated. For example, is visual motion most provocative in the same frequency domain (less than 0.5 Hz) as real motion?

4.8 Certain oculomotor variables should be investigated for their contribution to simulator sickness, such as distance to the display, convergence, and accommodation. Simulator displays should not inadvertently encourage convergence and accommodation to drift apart.

4.9 To understand the simulator sickness phenomenon, we need to know the underlying neurological mechanisms, from the retina to the vomiting center. The vestibular nuclei and the cerebellum may be of particular importance, particularly their role in motion perception and sensory conflicts.

4.10 We need to know more about how visual motion influences the vestibular system. The work of Waespe and Henn (1977) should be extended.

4.11 Would labyrinthine defectives (LDs) be immune to simulator sickness when confronted by a strong moving visual environment? This information would underscore the essential role of the vestibular system in simulator sickness and support the notion that simulator sickness is a subset of motion sickness.

4.12 The study of adaptation is essential for understanding simulator sickness. Some aftereffects seem to be transitory and intermittent, while others seem to be continuous and dissipating. The time course of adaptation and readaptation with periodic exposures is an important unknown. Similarly, the transfer of adaptation between the simulator and the aircraft is unknown. These phenomena must be understood to support recommendations that could affect flight safety, such as the minimum times between exposure and flight. Much of our understanding of these processes has come from basic research. Perhaps more applied research would be fruitful in the area of adaptation to real and visually implied motion.

Evaluation of Candidate Countermeasures

5.1 Incremental exposure regimens (Graybiel et al., 1969) could be developed if we could establish a range of severity of the conditions that contribute to simulator sickness. Susceptible individuals could be "preadapted" before being exposed to the most severe conditions, such as intensive ACM engagements.

5.2 Active controllers and passive observers may differ in their probability of experiencing sickness (McGuinness et al., 1981) and in their rate of adaptation (Reason and Benson, 1978; Guedry and Benson, 1983). Research on these differences in active and passive roles could contribute to our knowledge of the etiology of simulator sickness and to strategies for reducing it.

5.3 Field of view is suggested as a primary factor in simulator sickness. More information is needed on the relationship between FOV and sickness. If the incidence of sickness rose steeply beyond some value of FOV, there would be support for guidelines for FOV in simulation. A related concept is to increase FOV over repeated exposures as adaptation is developed. These possibilities should be explored in research.

5.4 Simulator exposure probably involves a recalibration of the sensorimotor processes, followed by another period of recalibration upon removal from the simulation. Certain hand-eye coordination games, like ping pong, might facilitate this recalibration process. They also would be useful from a motivational standpoint.

5.5 Medications used for motion sickness might be effective in the prevention of simulator sickness.

5.6 The velocity of the optokinetic stimulus on the simulator display is a very useful variable because it represents the magnitude of the stimulus to adapt. Stroboscopic illumination of such a "slip signal" eliminates the motion sickness symptoms that accompany optokinetically induced nausea (Melvill Jones and Mandl, 1979). This seems to offer reasonable hope that degrading the update rate of moving stimuli in the simulator display might be sufficient to support training while eliminating simulator sickness. Advocating degraded update rates, however, appears to be counter to engineering advancements. Further work is needed to define simulator specifications in terms of the desired perceptual effects rather than engineering advances for physical realism.

APPENDIX
POTENTIAL CONTRIBUTORS TO SIMULATOR SICKNESS

Characteristics of simulators and users that must be considered as potential contributors to simulator sickness:

INDEPENDENT VARIABLES

- Motion and vibration
  - Axes
  - Frequency
  - Acceleration
  - Exposure duration
  - Lags
  - Phase/gain
- Vision
  - Field of view
  - Framing effect
  - Retinal eccentricity
  - Off-axis display
  - Scene features
    - Number
    - Appearance
  - Display type
    - Model board
    - Computer-generated imagery
    - Point source
  - Pilot head movement

- Visual motion
  - Lags
  - Phase/gain
  - Optometric properties
  - Spatial frequency
  - Raster scan
  - Phosphoresis
  - Refresh rate
  - Velocity
  - Temporal frequency
  - Li ear/angular acceleration vectors
  - Spectral density
  - Distortion (temporal and spatial)
  - Collimation
  - Magnification
  - Aliasing

- Simulator features
  - Motion base/fixed base
  - Washout
  - Lags
    - Visual system
    - Motion system
    - Other motion cueing systems
  - Dome versus window
  - Dynamic modeling of aircraft
  - G-seat, helmet-loader, anti-G-suit
  - Maneuvers
    - Frequency
    - Amplitude
    - Duration
    - Turns-while-taxiing

- Simulator use
  - Freeze
  - Reset
  - (Re)initialization
  - Dissolve scenes
  - Warning signals
  - Controller/passenger
  - Task loading
  - Indoctrination
  - Briefing/demonstration
  - Buffet

Individual differences
- Experience/skill level
  - Aircraft
  - Simulator
- Field dependence/independence
- Attitude (set)
- Medical status
- Motion sickness susceptibility

DEPENDENT VARIABLES

Motion sickness symptomatology
- Objective (signs)
- Subjective (symptoms)

Performance

Flashbacks

Postural disequilibrium (ataxia and other balance measures)

Eye movement (and vestibulo-ocular reflex)

OTHER ISSUES

Adaptation
- Time course
- Specificity
- Transfer of adaptation
  - Dual (multiple) adaptation
  - Positive/negative transfer
- Active/passive

Transfer of training
- Positive/negative
- Flight safety

Sensory conflict identification
- Visual-vestibular
- Otolith-canal
- Intravisual (focal-ambient)
- Somatosensory-other (G-seat)
- Multiple sources of conflict
- Spatial/temporal conflict
- Conflict with neural store
- Measurement (magnitude) of conflict
- Significance of conflict

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Unclassified

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

REPORT DOCUMENTATION PAGE	READ INSTRUCTIONS BEFORE COMPLETING FORM
1 REPORT NUMBER	2 GOVT ACCESSION NO. AD-A148543	3 RECIPIENT'S CATALOG NUMBER
4 TITLE (and Subtitle) Research Issues in Simulator Sickness: Proceedings of a Workshop	5 TYPE OF REPORT & PERIOD COVERED Technical Report
	6 PERFORMING ORG. REPORT NUMBER
7 AUTHOR(s) Michael E. McCauley, editor	8 CONTRACT OR GRANT NUMBER(s) N00014-81-C-0017
9 PERFORMING ORGANIZATION NAME AND ADDRESS Committee on Human Factors National Research Council Washington, D. C. 20418	10 PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS Work Unit No. NR196-167
11 CONTROLLING OFFICE NAME AND ADDRESS Engineering Psychology Programs Office of Naval Research Arlington, VA 22217	12 REPORT DATE October 1984
	13 NUMBER OF PAGES
14 MONITORING AGENCY NAME & ADDRESS(if different from Controlling Office)	15 SECURITY CLASS. (of this report) Unclassified
	15a DECLASSIFICATION/DOWNGRADING SCHEDULE

16 DISTRIBUTION STATEMENT (of this Report)

Approved for Public Release: Distribution Unlimited

17 DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)

18 SUPPLEMENTARY NOTES

This workshop was sponsored by the USAF School of Aerospace Medicine, Brooks AFB, Texas, the Army Research Institute Field Unit, Fort Rucker, Alabama, and the U. S. Naval Training Equipment Center, Orlando, Florida.

19 KEY WORDS (Continue on reverse side if necessary and identify by block number)

Simulator
Simulation
Motion Sickness
Sensory Conflict
Simulator Sickness
Human Factors Research
Human Engineering Research
Vestibular
Vision
Committee on Human Factors

20 ABSTRACT (Continue on reverse side if necessary and identify by block number)

Simulator sickness, with symptoms similar to motion sickness, occurs frequently in military and civilian flight trainers. Simulator sickness appears to be independent of whether a fixed base or moving base simulator is used. Methods for amelioration are described as well as recommendations for future research to develop countermeasures.

Originator supplied keywords include:

DD FORM 1 JAN 73 1473 EDITION OF 1 NOV 65 IS OBSOLETE S/N 0102-LF-014-6601

Unclassified

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

END

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