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**Introduction to and Review of
Simulator Sickness Research**

David M. Johnson
U.S. Army Research Institute

April 2005

**U.S. Army Research Institute
for the Behavioral and Social Sciences**

**A Directorate of the Department of the Army
Deputy Chief of Staff, G1**

**ZITA M. SIMUTIS
Director**

Technical review by

Donald R. Lampton, U.S. Army Research Institute
David B. Durbin, ARL HRED AVNC Field Element

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**Introduction to and Review of
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David M. Johnson
U.S. Army Research Institute

Rotary-Wing Aviation Research Unit
William R. Howse, Acting Chief

U.S. Army Research Institute for the Behavioral and Social Sciences
2511 Jefferson Davis Highway, Arlington Virginia 22202-3926

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FOREWORD

The Rotary-Wing Aviation Research Unit (RWARU) of the U.S. Army Research Institute for the Behavioral and Social Sciences (ARI) is located at Fort Rucker, Alabama. Fort Rucker is the home of the U.S. Army Aviation Center (USAAVNC). The ARI Aircrew Performance Team is committed to enhancing aviation training. Recently USAAVNC has embarked on its Flight School XXI training program. Among other innovations, the Flight School XXI program will enhance basic and advanced aviator training with additional simulator-based flight instruction.

However, USAAVNC is aware that some aviators experience discomfort when operating flight simulators. Foreseeing the possibility that increasing the simulator-based augmentation of flight training would result in increasing levels of simulator sickness, the Directorate of Simulation suggested that ARI-RWARU perform an analysis of this phenomenon. This analysis was to include two portions: a review of the relevant research literature and a data-collection exercise on a non-interference basis. This report contains the literature review. The empirical work will be reported in a separate document.

Initial results from this project were briefed to the Commander and representatives of the 1st Battalion/14th Aviation Regiment on 21 November 2001. This resulted in an informal agreement to limit the total number of daily hours any aviator would spend in simulator-based training. Early results of this work were also briefed to the Directorate of Simulation on 23 April 2003.

MICHELLE SAMS
Technical Director

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INTRODUCTION TO AND REVIEW OF SIMULATOR SICKNESS RESEARCH

EXECUTIVE SUMMARY

Research Requirement:

The purpose of this research was to review and explain the literature pertaining to simulator sickness. Special emphasis was given to sickness issues as they relate to simulator-based helicopter training.

Procedure:

A library search was undertaken to uncover the relevant simulator sickness literature. Helicopter studies were emphasized, where appropriate. Since simulator-induced discomfort is a form of motion sickness, key findings from the literature of motion sickness were also pursued. Findings were organized by principle issues, including: terminology, selected history, sickness signs and symptoms, sensory basis for the perception of motion, measurement, incidence of sickness, residual aftereffects, adaptation, susceptibility factors, performance, training, safety, treatment, theory, guidelines for simulator-based flight training, and suggestions for future research. The sensory conflict theory and the postural instability theory were described in detail. These two competing theories are the key explanatory systems both for motion sickness and simulator sickness.

Findings:

Research findings make up the bulk of the text of this review. Examples follow. Motion sickness (MS)—specifically seasickness—has been known and documented since at least the ancient Greeks. Simulator sickness (SS) has been known and documented since the first helicopter flight simulator was procured in the mid-1950s. The most common signs and symptoms of MS are pallor, cold sweating, nausea, and vomiting. Vomiting is rare in SS. The most common signs and symptoms of SS involve visual symptoms, disorientation, and nausea. Only individuals with intact and normally functioning vestibular systems are susceptible to MS and SS. Several measurement techniques exist. The most practical is the Simulator Sickness Questionnaire. The incidence of SS can range from very low to exceedingly high, depending upon the simulator, tasks, conditions, and population. In most individuals the symptoms of SS subside in less than one hour. Residual aftereffects lasting longer than 12 hours are relatively rare. Adaptation is the single most effective solution to the problem of MS and SS. Most individuals adapt within a few sessions, some individuals require considerable exposure to adapt, and 3%-5% of individuals never adapt. Susceptibility to SS varies depending upon many factors, including age, flight experience, and prior history of MS. Performance of cognitive, perceptual, and psychomotor tasks is largely

unaffected by MS and SS. Motivation to perform, however, is strongly affected. The author could find no direct evidence that SS adversely affects training effectiveness. The author could find no direct evidence that SS harms personal safety or flight safety. There are drugs that have proven effective for treating MS and SS—although no drug is 100% effective and all drugs have side effects. The two major theories in this domain make different predictions about the effect of chronological age on SS—which suggests future research. Several useful guidelines were presented to reduce the incidence and severity of SS. One important guideline was that no simulator session should ever exceed two hours in duration.

Utilization of Findings:

Early results of this project were briefed to the 1st Battalion/14th Aviation Regiment as well as to the Directorate of Simulation (DOS) at the U.S. Army Aviation Center (USAAVNC). Final results will be made available to DOS, which suggested this review, in order to support the enhanced simulator-based flight training envisioned as a part of USAAVNC's Flight School XXI program.

INTRODUCTION TO AND REVIEW OF SIMULATOR SICKNESS RESEARCH

CONTENTS

	Page
INTRODUCTION.....	1
Cost Comparisons.....	1
MOTION SICKNESS.....	2
Reviews.....	2
Selected History.....	3
Signs, Symptoms, and Terminology.....	5
Sensation of Motion.....	6
Incidence.....	8
Adaptation.....	10
Susceptibility.....	11
Motion Sickness and Performance.....	13
Treatment.....	13
Theory.....	14
SIMULATOR SICKNESS.....	21
Reviews.....	21
Terminology and Distinctions.....	21
Selected History.....	22
Signs and Symptoms.....	27
Measurement.....	28
Incidence.....	33
Residual Aftereffects.....	34
Adaptation.....	36
Susceptibility.....	38
Simulator Sickness, Performance, and Training.....	44
Treatment.....	47
Theory.....	48
Guidelines for Reducing Simulator Sickness and Risks from Aftereffects.....	50
Suggestions for Future Research.....	51
REFERENCES.....	53

INTRODUCTION TO AND REVIEW OF SIMULATOR SICKNESS RESEARCH

Introduction

There are several advantages of simulation for aviation training (e.g., Kennedy, Berbaum, Lilienthal, Dunlap, Mulligan, & Funaro, 1987; Kennedy, Lilienthal, Berbaum, Baltzley, & McCauley, 1989). Emergency procedures can be trained and practiced in safety. Foul weather does not delay or halt simulator-based training. Further, there are special training options available with simulation that do not exist in live aircraft training. For example, the freeze command can be used to stop the aircraft to provide instruction or prevent a crash. Preprogrammed reset locations can be used to reposition the aircraft for the next task, or a repeat of the prior task, without having to expend valuable training time flying back to an optimal starting point. Simulator-based training can occur in real time, slow motion, or faster-than-real time (Crane & Guckenberger, 2000). Automatic feedback features can be preprogrammed and simulator flights can be recorded for replay and later examination. Not least among the advantages of simulator-based training is cost savings.

Cost Comparisons

Simulators have been shown to provide effective training at relatively low cost since at least the 1970s (e.g., Orlansky & String, 1977). Simulators are 10 to 30 times more available for training than aircraft. They save on fuel. In some cases the cost of the simulator can be regained in savings within the first 18 months. The relative cost per hour of training in a simulator has been estimated as 5% to 20% that of the actual aircraft (Pausch, Crea, & Conway, 1992). According to U.S. Air Force (USAF) calculations (Moorman, 2002), an hour of training in a C-5 aircraft costs taxpayers \$10,000 while an hour in a USAF C-5 simulator is \$500, a ratio of 20:1 in favor of the simulator. *Jane's Defence Weekly* (2002) reported that the hourly cost of a Boeing 747 aircraft versus a Level D flight simulator of that aircraft was about 40:1. *Jane's* went on to report that for military fighter or attack aircraft the relative aircraft to simulator ratios were in the region of 10 or 20 to 1. The U.S. Navy reported that the relative cost ratios for training in a F/A-18 are 18:1 and for training in a SH-60 Blackhawk helicopter are 15:1 (*Jane's Defence Weekly*). Similar figures for training in an M-1 tank versus an M-1 tank simulator, not counting ammunition, were reported as 33:1 (*Jane's Defence Weekly*). Lampton, Kraemer, Kolasinski, and Knerr (1995) reported that the cost per mile of driving an M-1 tank was \$92, while it was \$6 per mile for an M-1 tank driver simulator, a cost ratio of about 15:1.

The U.S. Army Aviation Center (USAAVNC) at Fort Rucker trains more than 1,200 aviators every year (Spires, 2003, March). It costs the Army at least \$1.1 million to train a pilot (Czarnecki, 2004; Department of the Army, 1994, November). The estimated cost per hour of flight training in a live aircraft can vary widely depending upon how much of the training infrastructure and associated materiel costs are included. Spires estimated that the hourly cost of operating the TH-67 training helicopter was

\$125, while the UH-60 Blackhawk was \$2,000 per hour. USAAVNC (2003) calculated costs based on a variety of factors including instructor labor, maintenance labor and parts, installation overhead, fuel, oil, and lubricants. The costs per flight hour for training varied from a low of \$781 for the TH-67 to a high of \$6,095 for the AH-64D Apache. Pate (1988) calculated the costs saved by Army aviation through the use of simulators for a part of aviator training. (Amounts in parentheses represent inflation-adjusted 2005 dollars.) Compared to live aircraft costs, use of the UH-1 flight simulator saved USAAVNC in excess of \$50 million (\$84 million) in cost avoidance annually. Training cost avoidance for the field was in excess of \$41 million (\$69 million) annually for the UH-1. At USAAVNC, use of the CH-47D flight simulator resulted in cost avoidance of more than \$3 million (\$5 million) annually. For the field, training cost avoidance attributable to this simulator was in excess of \$24 million (\$41 million) annually. Training cost avoidance annually attributable to the UH-60 flight simulator was in excess of \$10 million (\$17 million) at USAAVNC and \$121 million (\$201 million) in the field. The USAAVNC and field simulator for the AH-64A Apache is the Combat Mission Simulator (CMS). Annual cost avoidance figures for this device at USAAVNC were more than \$13 million (\$22 million) and more than \$117 million (\$194 million) in the field. Although the CMS allows the training of gunnery skills, these figures did not include ammunition costs. Cost avoidance calculations would be many times higher if ammunition costs were included.

The point of these comparisons is that Army aviation training is expensive; that training with live aircraft is many times more expensive per hour than simulator-based training; and that Army aviation training can save, and has already saved, many millions of dollars per year by using simulators for a part of required training. Simulator-based training to augment live-aircraft training is here to stay. Unfortunately, some aviators experience simulator-induced discomfort, or simulator sickness, when operating a flight simulator. It is simulator-induced discomfort that is the subject of this report.

Motion Sickness

Whenever we relinquish our intended status as self-propelled animals and step aboard some vehicle or device that transports us passively we incur the risk of motion sickness. This wretched and debilitating condition has always been intimately linked with man's technological efforts to improve and extend his natural powers of locomotion. (Reason & Brand, 1975, v)

Simulator sickness is a form of motion sickness. A review of motion sickness (MS) is therefore required to place simulator sickness (SS) in an appropriate context.

Reviews

There are several reviews of MS. The book by Reason and Brand (1975) entitled *Motion Sickness* is arguably the best single source because of its exhaustive

discussion of research results, sensory systems, and theory as they apply to MS. AGARD (1988), Benson (1978, 1988), Kennedy and Frank (1985), and Mooij (1988) also provide useful reviews of research results, sensory systems, and theory. Young (2003) provides a clear and thorough discussion of MS with special emphasis on the anatomical sensory systems that underlie spatial orientation (e.g., the vestibular system). Theoretical discussions of MS can also be found in Reason (1970, 1978), Kennedy and Frank, Benson, and Riccio and Stoffregen (1991).

Selected History

The many different names that have been used to designate this disorder reflect both its long history and the wide range of circumstances that can provoke it. In the beginning there was sea-sickness, then coach-sickness, train-sickness, car-sickness, air-sickness, and, [more recently] space-sickness. (Reason, 1970, p. 386)

Documented accounts of motion sickness date back at least as far as the ancient Greeks. Nausea derives from the Greek word “naus” meaning ship (Benson, 1978; Reason & Brand, 1975). Hippocrates (460—377 B.C.) wrote, “Sailing on the sea proves that motion disorders the body” (Reason & Brand, p. 2). Admiral Lord Nelson (1758-1805) was a chronic sufferer of seasickness during his entire naval career. Both Napoleon Bonaparte (1769-1821) and T. E. Lawrence (1888-1935) learned of the MS that accompanies the swaying gait of riding a camel in their respective Middle East campaigns. MS was used as a legal punishment for delinquent youths in 19th Century Germany. The juvenile delinquent was placed inside a box that was suspended outside town hall and rotated by a policeman until the offender had provided a “disgusting spectacle” to those present (Reason & Brand, p. 4).

Prior to the 1950s, people who wanted to travel across oceans had no choice but to attempt a sea voyage. For inexperienced mariners this frequently resulted in seasickness. A ship’s doctor quickly became an expert on the subject of seasickness. One such physician described this all-too-typical occurrence this way:

After a period of lively chaff, the victim suddenly becomes quiet and subdued... he evinces a strong dislike to the use of tobacco by himself or others; he feels an indefinable something in the epigastrium, is cold and miserable, smiling feebly when spoken to, and apparently wishing to be left alone... While in this state, with lips blue, eyes suffused, complexion varied states of green, and a general disorder of mind, body and apparel, his condition is one of abject misery, and strongly deserving of sympathy. Then it is his plaint that the ship will not sink, whereas earlier on, he may have been afraid that she would and drown all on board. The stage of convalescence is generally marked by an air of blatant superiority over his

less advanced fellows. (Reason & Brand, 1975, p. 73)

A large variety of ostensible seasickness preventatives were sold to customers contemplating an ocean crossing. Reason and Brand (1975) listed all the drugs recommended for seasickness in the British medical journal *Lancet* between 1828 and 1928. Some of these concoctions were amyl nitrite, chloroform, atropine, creosote, opium, quinine, cocaine, strychnine, laudanum, cayenne pepper, nitrous oxide, and tincture of belladonna. Interestingly, hyoscine hydrobromide, a derivative of belladonna, has been shown through controlled experimentation to be one of the most effective medications to prevent MS (Benson, 1978; Reason & Brand) and SS (Regan & Ramsey, 1996).

In 1881, Irwin (Kennedy & Frank, 1985; Reason, 1978; Reason & Brand, 1975) was the first to report on the importance of the vestibular system, and specifically the semicircular canals, in the etiology of MS. Additionally, he noted that being onboard a ship presented the inexperienced vestibular system with an unusual (in the sense of novel) current force environment compared to prior (land) experience. Thus, Irwin not only implicated the vestibular system but also the importance of the unfamiliarity of the motion in MS.

In 1882, William James, a pioneering American psychologist, published his experiments with deaf mutes (Kennedy & Frank, 1985; Reason & Brand, 1975). Frequently the deaf also have impairment of the semicircular canals of the vestibular system. James found that 36 percent of his deaf sample could not be made dizzy no matter how much they were spun in a rotating chair. Further, of those 15 who could not be made dizzy, and who also had experience onboard ships during rough seas, none of them (0%) had ever experienced any symptoms of seasickness. This groundbreaking research established the centrality of the vestibular system to MS. Later researchers repeatedly demonstrated that deaf mutes and others with middle ear damage could not be made seasick under conditions that characteristically produced MS in normal populations (Benson, 1978; Kennedy & Frank; Reason & Brand).

Meanwhile, other research showed that blind people, and sighted people closed into completely darkened cabins or blindfolded, experienced seasickness at normal rates (Kennedy & Frank, 1985; Reason & Brand, 1975). Loss of visual stimulation did not prevent the onset of MS. Virtually all scientists practicing in this field now acknowledge that MS is only possible in people with a normally functioning vestibular system. An intact and normally functioning vestibular system is a necessary condition to experience MS (e.g., Benson, 1978; Biocca, 1992; Ebenholtz, 1992; Kennedy & Frank; Pausch et al., 1992; Reason, 1970, 1978; Reason & Brand; Young, 2003; but see Riccio & Stoffregen, 1991).

The coming of the Second World War, and with it the requirement to transport vast numbers of men overseas, and have them physically fit upon disembarkation, changed the study of MS substantially. Seasickness went from being a common, if ultimately harmless, discomfort of sea travel to a major military problem. The same was

true for carsickness and train-sickness, because large numbers of troops were being transported overland both in Europe and in the United States. The era of airsickness began as well, because for the first time enormous numbers of pilots and navigators were being trained in the air.

An exhaustive listing of all the provocative motion environments will not be attempted. However, historically the disorder has been caused in a wide range of motion conditions. These include: ships, small boats, life rafts, coaches, trains, automobiles, aircraft, spacecraft, carnival rides, swings, tilted rooms, rotating rooms, rotating chairs, camel riding, elephant riding, earthquakes, and newly prescribed eye glasses (e.g., AGARD, 1988; Benson, 1978; Ebenholtz, 1992; Lilienthal, Kennedy, Berbaum, Dunlap, & Mulligan, 1987; Kennedy & Frank, 1985; McCauley, 1984; Reason, 1970; Reason & Brand, 1975). Interestingly, riding horses, bicycles, or motorcycles does not cause the disorder (Kennedy & Frank).

... and the motion of the earth made my stomach sick, like one that was tossed at sea. (Defoe, 1955, p. 60)

Signs, Symptoms, and Terminology

There is no doubt that motion sickness can be a profoundly unpleasant experience. The degree of discomfort, malaise and wretchedness suffered by the victim is really out of all proportion to the banal and comparatively harmless nature of the disorder. Few people have perished as a consequence of motion sickness, but many in its grips have wished for death. In some individuals, motion sickness brings with it an almost pathological state of depression, apathy and listlessness. (Reason & Brand, 1975, p. 73)

The term “sickness” usually means to be afflicted with a disease or malady. The word sickness in MS is, therefore, a misnomer. MS is not a disease or malady. MS is, in fact, a “...normal response of a healthy individual, without organic or functional disorder, when exposed for a sufficient length of time to unfamiliar motion of sufficient severity” (Benson, 1978, p. 469). Contrarily, people who are incapable of experiencing MS are the ones with the pathology, and that pathology is an absent or nonfunctional vestibular system (Benson; Reason & Brand, 1975; Young, 2003). MS is more properly called a syndrome-- a motion maladaptation syndrome-- and is frequently referred to as such in the scientific literature (e.g., Benson, 1978, 1988; Kennedy & Fowlkes, 1992; Kennedy & Frank, 1985; McCauley, 1984; Reason & Brand).

Within the medical community the “signs” of a disease or syndrome are objective conditions that can be observed or measured by a physician. “Symptoms” are subjective conditions that the patient experiences and reports to the physician. In this context “cardinal” means of foremost importance or pivotal. The signs and symptoms associated with MS are many and varied. Virtually all scientific reports of MS provide a

listing of the cardinal signs and symptoms. For a detailed description of signs and symptoms see Benson (1978), Kennedy & Frank (1985), and Reason and Brand (1975).

The cardinal symptom of MS is nausea. The cardinal signs are pallor, sweating, and vomiting (also called emesis). Nausea is the most commonly reported symptom. It begins with stomach awareness, progresses to queasiness, and then vomiting. Vomiting often leads to a temporary improvement in symptoms. Facial pallor and cold sweating are the most commonly reported signs of MS. In fair-complexioned persons facial skin takes on a whitish-greenish tint caused by vasoconstriction of the extremities. Cold sweating is sweating in the absence of an adequate thermal stimulus. That is, the ambient temperature is not warm and, therefore, cold sweating serves no useful thermal-regulatory function. Pallor and cold sweating reliably precede vomiting. Additional signs of MS are increased salivation, sighing, yawning, hyperventilation, burping, flatulence, and locomotor ataxia. In this context "ataxia" means postural instability or postural disequilibrium (e.g., Kennedy, Berbaum, & Lilienthal, 1997; Kolasinski, 1995). Additional symptoms are headache, depression, apathy, drowsiness, somnolence, and mental disorientation.

Sensation of Motion

The anatomical and physiological basis for the sensation of motion is a large, complicated field of study in its own right. Reason and Brand (1975) review this field from the perspective of its relevance to MS (see also Mooij, 1988). Young (2003) reviews this field from the perspective of the sensation of motion in aviation and aviation simulation (see also Hall, 1989).

The visual system and motion. The eyes detect motion as a change in position or as velocity in the periphery of the visual field. Velocity and acceleration are assessed by changes in position over time. The eyes have no ability to detect acceleration directly.

For purposes of perceiving motion there are two visual systems that operate in parallel (Young, 2003). The ambient system is principally concerned with detecting large objects in the periphery and with detection of self-motion (vection) through sensations of visual flow in the peripheral field. Visual flow increases as a function of velocity, scene detail, and reductions in height above terrain. There is greater scene detail closer to the ground, so whether one is flying an aircraft or operating a flight simulator, the lower the height above terrain the greater the visual flow and the greater the sensation of vection. Although under special laboratory conditions vection has been reported with a small field of view (FOV), generally a minimum FOV of 60 degrees horizontal is required to experience the sensation of motion from visual information alone (Mooij, 1988). The focal system is principally used for the discrimination of fine detail in the central, or foveal, visual field. Motion is sensed through such focal cues as size and shape constancy, perspective, and interposition. The focal system is used in an automobile, aircraft, or simulator when the operator examines the onboard

instruments to determine motion, direction, and velocity. An aircraft pilot is forced to use this system when weather conditions make out-the-window viewing unreliable, a condition known as instrumented flight.

The vestibular system. This system is composed of the semicircular canals and the otolith organs. They are about the size of a pea, are located in each inner ear, and are thoroughly encased in the temporal bones. The semicircular canals give information as to angular velocity, and rate of change in angular velocity, of the head. The otolith organs transduce the force of gravity and linear acceleration acting on the head. In ordinary terrestrial activities the vestibular system is primarily responsible for maintaining balance and postural equilibrium. Usually the actions of the vestibular system are entirely transparent. Children born without a functioning vestibular system grow up to lead normal lives, with other senses providing motion information, and remain unaware of their disability unless exposed to rare laboratory tests or odd motion environments. In addition, as stated before, such individuals never experience MS.

In each inner ear there are three semicircular canals (superior, posterior, and horizontal; Young, 2003). These canals are positioned in planes approximately orthogonal to each other. Each is filled with a fluid called endolymph. A widening at the base of each canal is called the ampulla. Inside each ampulla is a gelatinous wedge called the cupula that prevents the endolymph from flowing through the ampulla. At the base of each cupula are cilia projecting from hair cells. Movement of the endolymph inside the canal will cause a slight movement of the cupula that will bend the cilia projecting from the hair cells. This bending will cause the hair cells to fire, sending a pattern of discharges up the afferent nerve fibers that connect the semicircular canals to the brain. Each canal will respond to angular acceleration of the head perpendicular to its plane. For brief head movements the canals can correctly indicate velocity as well as acceleration, but this signal will decay to zero for constant velocity turns. Thus, the semicircular canals are primarily responsible for providing information as to rate of change (acceleration/deceleration) in angular velocity. For a more sophisticated understanding of the semicircular canals, Young provides both more detailed information and several important references (see also Reason & Brand, 1975).

The influence of alcohol on the semicircular canals accounts for the all-too-well-known phenomenon of drinking until the room spins. Normally the cupula floats with near neutral buoyancy in the surrounding fluid. Alcohol alters the density of the cupula and the endolymph. The cupula becomes lighter. When the head is tilted, the cupula rises slightly (bobs up). This mimics a condition where the head is being rotated in the direction of the ear that is down. This is called Positional Alcohol Nystagmus I "... and is accompanied by a consistent, disturbing sensation of rotation, the degree of which depends on both the head orientation relative to gravity and the alcohol level" (Young, 2003, p. 90). There are several other harmful effects of acute alcohol consumption on balance, oculomotor coordination, and the perception of motion should the reader care to investigate (cf., Riccio & Stoffregen, 1991; Young).

The otolith organs indicate the orientation of the head relative to gravity, and are, therefore, crucial in maintaining balance while moving naturally around the Earth. Under ideal conditions, the otolith organs indicate postural vertical to within about two degrees. "However, just as with any other physical accelerometer, they follow Einstein's equivalence principle and cannot distinguish between gravity and the inertial reaction force to any linear acceleration" (Young, 2003, p. 77). The otolith organs function analogously to a carpenter's plumb bob. When stationary and free to hang, a plumb bob accurately indicates the direction of vertical by pointing to the center of the Earth. Accelerate horizontally by racing your automobile or taking off in a jet and the plumb bob no longer points "down" to the center of the earth. It signals apparent vertical, the direction vector determined by the combined gravity-inertial force.

There are two otolithic receptors in each inner ear, one in the utricle and one in the saccule. The function of the saccule receptor is not completely understood, and what is known will not be discussed here. The utricle consists of two parts, the otolithic mass and the macula. The otolith is a flattened blob of jelly covered with dense calcium carbonate crystals. The macula is the base and receptor portion of the organ, on which the otolith sits. Cilia from hair cells in the macula extend up into the otolith. These hair cells respond to movement of the cilia. These cilia move in response to displacement of the heavier otolithic mass as a result of head tilt and/or linear acceleration. There are hair cells that are optimally sensitive to, and discharge maximally for, any orientation of the head. These discharge patterns are sent to the brain via the vestibular nerve. For a more thorough understanding of the otolith organs, Young (2003) provides both more detailed information and additional references (see also Reason & Brand, 1975).

Other proprioceptive senses and motion. Vestibular, kinesthetic, and touch receptors are often classed together as the proprioceptive senses (Hall, 1989; Young, 2003). However classified, other sensory systems play a role in the perception of motion. Kinesthetic receptors in the joints, muscles, and tendons signal limb, head, and body position as well as the force required to maintain a given position in combination with externally applied forces. This provides information on the forces acting on the body (linear accelerative forces). Touch (tactile or somatic) sensors respond to pressure applied to the skin. The direction and magnitude of these pressures provide information about bodily orientation, direction of motion, and acceleration.

Incidence

MS is not limited to those in delicate health, possessing a neurotic disposition, or who are weak and frail. "It is a truly functional disorder of the intact, healthy individual, and occurs in the absence of any pathogenic agency or structural damage to the body" (Reason & Brand, 1975, p. 28). Anyone with a normally functioning vestibular system can become motion sick (Benson, 1978; Reason & Brand; Young, 2003). It is a normal response to an abnormal motion environment. This being the case, the incidence of MS varies depending upon how, and under what conditions, the data are collected. Questionnaire studies typically report incidence rates as high as 90 percent, while laboratory studies report rates as high as 98 percent (Reason & Brand). The best

single source for a listing of incidence rates under a variety of conditions is Reason and Brand. A selection of these incidence rates is presented below.

Commercial transport. Obviously, companies and government agencies in the business of transporting people do not want their customers to experience MS. Steps are taken to provide as smooth and uneventful a ride as possible. So statistics from commercial studies can be expected to be conservative estimates. Seasickness rates in moderate turbulence were 30 percent but as high as 90 percent in severe seas (99% reported in Benson, 1978). Airsickness rates in scheduled airliners were less than one percent. Rates in commercial rail transport were also less than one percent. A majority of people experienced MS symptoms on rides at amusement parks.

Data were aggregated by age for automobiles and buses. Fifty-eight percent (58%) of children below the age of 12 reported having experienced nausea while riding in automobiles and 33 percent reported vomiting. Forty-seven percent (47%) of children between 12 and 20 years of age reported having experienced nausea while riding in automobiles and 14 percent reported vomiting. The reported rates for adults in automobiles were 11 percent nausea and 1 percent vomiting. The reported nausea rates for children below the age of 12 while riding in buses was 57 percent and for vomiting 26 percent. For adults the reported rate of nausea in buses was 13 percent and 1 percent for reported vomiting.

Military transport. World War II provided an opportunity for researchers to assess seasickness rates for large samples of primarily adult men. Eleven percent (11%) to 100 percent of these samples reported experiencing seasickness in large ships depending upon sea state. Fifteen percent (15%) to 60 percent reported vomiting, again depending upon sea state. The rate of seasickness reported for studies of military personnel in life rafts averaged 60 percent.

All airsickness rates based on self-report of trainees during aircrew training must be assumed to be underestimates of the true incidence. Aircrew trainees have good reasons for not believing it is in their best interests to be candid on this subject-- regardless of what the psychologist with the clipboard tells them about their anonymity being assured (see Wright, 1995, for a discussion of this problem). Benson (1978) cites an example. He reported that 4 percent of flight students questioned at their pre-entry medical exams reported a history of MS. However, the same students when questioned in confidence reported a 59 percent history of MS.

Military airsickness data are often aggregated by pilot training and navigator training. The pilot is in control of the aircraft and spends most of his or her time looking out of the cockpit. The navigator is not in control of the aircraft and spends part of his or her time looking at tabular material presented on screens or paper inside the cockpit. Reported airsickness rates for pilot trainees varied between 3 percent and 13 percent. Reported airsickness rates for navigator trainees varied between 8 percent and 65 percent. Five percent (5%) of navigator trainees were disqualified from finishing flight training because of severe MS symptoms (usually noticed by the instructor pilot).

Between 5 percent and 19 percent of bomber crew trainees were disqualified from finishing flight training because of MS, with navigator trainees having the highest washout rates.

Research has shown a small but statistically significant correlation between self-reported susceptibility to motion sickness and completion of introductory flight training (Hutchins & Kennedy, 1965; Kennedy, 1975). Susceptibility was measured prior to flight training using the Motion Sickness Questionnaire. Students who reported a greater past history of MS were more likely to withdraw voluntarily from flight school or be removed for cause.

Airsickness is not limited to trainees. With experience (i.e., adaptation) the incidence of airsickness falls, so that MS is rare in operational flights. However, during missions that expose aircrews to severe turbulence, such as hurricane penetration, 90 percent of experienced aviators reported airsickness symptoms. Among crewmembers inexperienced with hurricane penetration the rates were 100 percent (Benson, 1978).

Low-frequency oscillation. Laboratory research has shown that MS can be reliably produced by exposing individuals to low-frequency, sinusoidal, vertical oscillations between the frequency range 0.08 Hz to 0.4 Hz (Biocca, 1992; Kennedy & Frank, 1985; Mooij, 1988; Reason & Brand, 1975; Stoffregen, Hettinger, Haas, Roe, & Smart, 2000). This fact was clearly established by the end of World War II (Reason & Brand). Not surprisingly, all manner of large ships, small boats, automobiles, buses, aircraft, and amusement park rides have been shown to oscillate within this frequency range depending upon design, operator actions, and environmental conditions.

Adaptation

There are at least two facts about motion sickness that are not in dispute. One is that the vestibular system is clearly implicated in its development. The other is that prolonged exposure to any one type of provocative stimulus leads to a diminution and eventual disappearance of the signs and symptoms in most people... This reduction in symptomatology occurs without any alteration in the properties of the provocative stimulus. Indeed, the absence of such variation is a necessary prerequisite for its occurrence. These changes are therefore clearly rooted in the organism rather than the environment... (Reason & Brand, 1975, p. 135)

It is well established that MS usually disappears with repeated exposures to the sickness-producing environment (Benson, 1978, 1988; Biocca, 1992; Kennedy & Frank, 1985; Reason, 1978; Reason & Brand, 1975; Young, 2003). This reduction in symptoms with experience in the provocative environment is called "adaptation" (sometimes "habituation"). Between 95 and 97 percent of people studied eventually adapt to the novel motion environment (Biocca; Reason & Brand). The remainder never

adapt, regardless of the length of their exposure, and remain chronically motion sick. Laboratory studies show large differences in rates of adaptability among individuals, and also that these adaptability rates are a consistent trait for each individual (Reason & Brand). Most people adapt to a new motion environment fairly quickly, many people require a very long time to adapt, and a small percentage of unfortunates never adapt.

Adaptation to motion sickness is highly specific. Navy sailors adapted to a destroyer get seasick again, and must readapt again, to the different motions of an aircraft carrier or ocean liner (Reason & Brand, 1975). Positive transfer from one conveyance to another occurs when the motions are the same or very similar. In order to adapt to a novel motion environment one must be exposed to it and, sadly, get sick. Active movement within the environment, while making one sicker initially, will adapt one faster than passive movements. Faster adaptation can be achieved with incremental exposures, distributed daily over a series of days, than with abrupt total exposure from the beginning (Reason & Brand).

Ironically, after adaptation to a novel motion environment, a return to the previously customary conditions produces MS symptoms again. This is known as “land sickness” or “mal de débarquement” to passengers returning ashore after a prolonged sea voyage (Reason & Brand, 1975, p. 145). In such cases, passengers will literally experience MS symptoms after disembarking onto land and require a period of adaptation—usually short—before feeling normal again.

This last point is particularly important. People who have lived on land all their lives take an ocean cruise, get seasick, adapt to the new mode of travel, and later when disembarking at port, get land sickness, and must readapt to their former environment. Clearly MS is not caused by the specific environmental conditions of travel, but by the conflict between current conditions and past history. For this reason MS has been described as a phenomenon of maladaptation or “maladaptation sickness” (Reason & Brand, 1975, p. 145).

Susceptibility

As previously mentioned, anyone with a normally functioning vestibular system is susceptible to MS. Like all biological phenomena, however, there is a distribution of susceptibility throughout the human population (Kennedy & Frank, 1985; Reason & Brand, 1975). The literature on MS is in general agreement about which categories of individuals are more susceptible than others.

Gender. Females are reported to be more susceptible to MS than are males (Benson, 1978; Kennedy & Frank, 1985; Reason & Brand, 1975). The female endocrine system has been implicated as (somehow) causal in this case because research has shown that women are more susceptible to MS during the time of menstruation (Benson; Reason & Brand).

Age. It is widely reported that susceptibility to MS varies with age (e.g., Benson, 1978; Biocca, 1992; Kennedy & Frank, 1985; Reason & Brand, 1975; Young, 2003). All the reports of this age effect make the identical claims, and often use the same language, as that of Reason and Brand. So, to repeat Reason and Brand again, they state, "There is overwhelming evidence to show that motion sickness susceptibility fluctuates with age" (Reason & Brand, p. 187). Below age 2 infants are "generally immune" to MS. Susceptibility is at its "highest level" between age 2 and about 12. There is a "highly significant decline" between age 12 and 21. This decline in susceptibility continues "through young adulthood to middle age and beyond." Motion sickness of any kind is "very rare" beyond age 50.

This curvilinear relationship between susceptibility to MS and age was based on self-report questionnaires administered to people of various ages who reported on their experiences with one or more mode(s) of travel. For example, seasickness was self-reported by 5,000 Soldiers on transatlantic troop ships during World War II. The rate of reported seasickness among Soldiers aged 17 to 19 years was 31 percent, while for Soldiers aged 30 to 39 it was 13 percent (Reason & Brand, 1975). Similarly, airsickness rates on commercial airliners in the early 1950s showed this relationship using passenger self-reports (Reason & Brand). It is by no means clear how this self-report method would provide evidence that children below age 2 are immune from motion sickness. However, the authors point out the common experience of new parents that rocking motion, automobile motion, perambulator motion, and train motion do not upset their babies, but put them to sleep.

Reason and Brand (1975) were well aware of the potential pitfalls of using self-report data for establishing susceptibility rates by age (e.g., Reason & Brand, pp. 181-186). For example, respondents may lie, mangle, have a defective recollection of past MS experiences, or attribute bona fide MS events to some other cause. Also self-report does not take into account relative travel experience. Persons who are not particularly susceptible to MS, but who have a large and varied travel experience, will likely report more MS symptoms than those with greater susceptibility but who are little traveled. Nonetheless these authors pointed out the obvious practical advantages of the survey technique and trusted the results collected thereby. They noted that people usually have neither difficulty recalling MS episodes, nor objection to communicating these experiences.

Reason and Brand (1975) offered several possible explanations for the reduction in MS symptoms with age. The most intuitive of these being that older individuals have had more opportunities to experience, and adapt to, more motion environments. Such individuals would therefore experience less MS.

Miscellaneous. The consumption of alcohol increases one's susceptibility to MS (Biocca, 1992; Young, 2003). The more head movements one makes while in motion, the greater the susceptibility to MS (Benson, 1978; Kennedy & Frank, 1985; Reason & Brand, 1975). The more introspective one is, the more attention one pays to one's internal sensations during motion, the greater the susceptibility to MS (Benson; Reason

& Brand). This is closely related to the evidence that people whose personality style is field independent are more susceptible to MS than those who are field dependent (Reason & Brand).

Motion Sickness and Performance

A number of studies reviewed by Reason and Brand (1975) and Kennedy and Frank (1985) reported that MS does not harm performance. A wide variety of psychophysical tasks have been measured, including: perceptual-motor tracking, running through sand and around obstacles, 600-yard dash, dart throwing, speed and accuracy of rifle shooting, code substitution, mirror drawing, strength of grip, standing on one or two feet, walking a straight line, ball tossing, card sorting, opening combination locks, dial setting, arithmetic computation, conceptual reasoning, temporal sequencing, postural equilibrium, optical accommodation and convergence, and mental arithmetic. Only postural equilibrium was reliably worse when sick than baseline. Even so, sick participants had no difficulty walking with adequate visual references present. In other words, even though postural equilibrium was measurably worse than baseline, it would not be expected to make any difference in normal life activities.

How can this be? How can MS, which is universally recognized as a wretched and debilitating condition, have no effect on performance? The answer is motivation. MS does not impair one's capability to perform; it impairs one's proclivity to perform. An individual in the throws of MS is unwilling to do anything except lie in bed. Yet, if this same individual can be induced to perform, he or she will be able to perform at an acceptable level. This fact has also been widely recognized, at least since the Second World War.

During rough weather, seasick personnel lose interest in doing anything except the barest necessities, and an obvious lack of spontaneity can be observed aboard ship even in those men not frankly seasick... These comparisons further support the notion that canal sickness may reduce a subject's motivation to a very low level, but if the subject is willing or able to try, he can usually make good scores... stories of sailors and air gunners incapacitated by motion sickness who apparently recovered or at least responded effectively to an urgent call to action. Clearly, motivation plays a very large part in determining the level of performance in a motion sick individual. (Reason & Brand, 1975, pp. 64, 67)

Treatment

For the vast majority of people the surest treatment for the relief of MS has not changed since the 19th Century. It is simple adaptation. With rare exceptions, travelers will eventually adapt to a novel motion environment (Benson, 1978, 1988; Biocca, 1992; Kennedy & Frank, 1985; Reason, 1970, 1978; Reason & Brand, 1975; Young, 2003).

For those unwilling or unable to risk the rigors of natural adaptation, there are effective drugs.

A number of drugs have been shown to reduce either the incidence of MS or the severity of the symptoms (Benson, 1978; Reason & Brand, 1975). However, no drug can reduce the occurrence of MS for everyone. Further, every drug has side effects. Hyoscine hydrobromide has proven to be an effective treatment for MS (Benson; Reason & Brand; Regan & Ramsey, 1996). It has been available since World War II when English, American, and Canadian forces used it during the crossing of the English Channel on D-Day (Reason & Brand).

Dimenhydrinate (Dramamine) is another effective treatment (Benson, 1978; Reason & Brand, 1975). Dramamine began life as an antihistamine. Its ability to treat MS was discovered by accident in 1949 when a woman was prescribed it for the treatment of hives (Reason & Brand). She had been a lifelong sufferer of carsickness. Upon discovering that she was completely immune to carsickness when taking the drug, she told her physician. Subsequent field trials established the efficacy of Dramamine for preventing several types of MS. Other useful medications besides hyoscine hydrobromide and dimenhydrinate are discussed elsewhere (Benson; Reason & Brand).

Theory

It is particularly important to understand the sensory conflict theory of MS. In addition to MS, this theory has been used to explain the related problem of simulator sickness. For a discussion of several theories of MS that are now of only historical interest, see reviews by Kennedy and Frank (1985) and Reason and Brand (1975).

Sensory conflict theory. The commonly accepted explanation for MS has many different names: sensory conflict theory, sensory rearrangement theory, sensory incongruity theory, sensorimotor conflict theory, perceptual conflict theory, perceptual decorrelation theory, cue conflict theory, neural mismatch theory, mismatch theory, and incongruity theory. Sensory conflict theory is routinely described in research reports, review articles, and theoretical works (e.g., Biocca, 1992; Benson, 1978, 1988; Duh, Parker, Philips, & Furness, 2004; Gower & Fowlkes, 1989a, 1989b; Kennedy & Frank, 1985; Kolasinski, 1995; McCauley, 1984; Mooij, 1988; Pausch et al., 1992; Reason, 1970, 1978; Reason & Brand, 1975; Riccio & Stoffregen, 1991; Stoffregen et al., 2000; Young, 2003). The most thorough description of the theory is by its author (Reason) and, secondarily, the papers by Benson.

Under natural conditions of self-propelled locomotion, all of these sensory components of the basic orienting system transmit correlated information with regard to the position and motion of the body. But in a wide variety of situations, the harmony which normally exists between these receptors can be disrupted so that the inputs from one or more of these functionally related receptors conflicts with the other inputs,

and, as a result, the combined influx is incompatible with stored expectations. (Reason & Brand, 1975, p. 26)

... all situations which provoke motion sickness are characterized by a condition of sensory rearrangement in which the motion signals transmitted by the eyes, the vestibular system and the nonvestibular proprioceptors are at variance not only with one another, but also—and this is the crucial factor—with what is expected on the basis of past experience... (Reason & Brand, 1975, p. 105)

... motion sickness occurs when the sensory information about bodily movement, provided by the eyes, the vestibular apparatus and other receptors stimulated by forces acting on the body, is at variance with the inputs that the central nervous system expects to receive. Essential to the theory is the postulated existence within the central nervous system of a model of afferent and efferent neural activity associated with bodily movement; a model that is derived through daily experience... In normal locomotor activity, disturbances of body movement, such as when one accidentally trips, are typically brief and the mismatch between actual and expected sensory inputs from the body's motion detectors is employed to initiate corrective motor responses. However, when there is a sustained change in the sensory input—as occurs, for example, in atypical motion environments... then the presence of the mismatch between actual and expected sensory inputs indicates to the central nervous system that the internal model is no longer appropriate... The presence of a sustained mismatch signal has two effects: one, it causes a rearrangement of the internal model; and two, it evokes the sequence of neural responses that constitute the motion sickness syndrome. (Benson, 1988, p. 3)

The principal components of the sensory conflict theory are these: (1) sensory inputs from the eyes, vestibular system, proprioceptive system, and somatosensory system register motion in space; (2) a neural store of past patterns of sensory input from prior motion history provides the expected motion baseline; (3) a comparator unit compares the current pattern of sensory inputs with the expected pattern from the neural store; (4) the current pattern of input from the motion sensors during motion is provided in parallel both to the neural store, for updates, and to the comparator unit for comparison; (5) individual differences in thresholds throughout the system account for the wide individual differences in the incidence, severity, and adaptation to MS; (6) a sustained suprathreshold mismatch between the current sensory pattern and the expected sensory pattern will generate a mismatch signal; and (7) this mismatch signal

initiates both adaptation—the modification of the internal neural store or baseline—and the unpleasant signs and symptoms of MS.

Notice how the sensory conflict theory is able to explain the key issues of incidence, adaptation, and susceptibility. Consider seasickness. A Soldier who has never before been onboard an ocean liner embarks on a trans-Atlantic crossing. The motion environment onboard is different from his experience ashore. There is low frequency vertical motion, pitch, and roll. This novel motion is sensed by his vestibular system. Further, especially when below decks, there is a conflict between the motion that his vestibular system is accurately signaling and the lack of motion that his visual system is inaccurately signaling (because both the ship and the passenger are moving in perfect synchrony). So the Soldier is experiencing a novel pattern of sensorimotor cues that do not match his expected (baseline) pattern of motion cues. A mismatch exists and the passenger experiences MS.

However, the mismatch also acts to initiate a modification of the internal store. With experience the sensory pattern aboard ship becomes less novel and more expected. The passenger adapts to the shipboard motion over time, the sensory conflict is reduced, and the symptoms of MS decrease to zero.

Then, upon disembarkation, the former passenger experiences MS again. His prior pattern of land-based sensations have now become novel. He has adapted to the onboard cues and so, once again, a mismatch signal has been generated. He must readapt to a land-based motion environment. Readaptation, however, will not take so long nor be so debilitating because he has a lifetime of land-based motion memories in his neural store available to be reactivated.

In general terms, this is how the sensory conflict theory accounts both for the incidence of MS and the adaptation to it. What about susceptibility? One way for the theory to account for the decreasing susceptibility with age is by arguing that older persons have usually had more opportunity to experience varied motion environments and that these sensorimotor patterns of motion memories have been stored in the neural repository. Reactivation of these traces reduces, and sometimes eliminates, MS in the older traveler.

Reason and Brand (1975) discuss the sensory conflict theory in excruciating detail. The reader who wants a deeper understanding of this theory should begin with them. However, there are two more theoretical concepts that need to be introduced now. They are receptivity and adaptability.

Receptivity and adaptability are terms meant to name stable individual traits that govern the perceived strength (receptivity) and duration (adaptability) of the mismatch signal. Receptivity refers to the initial size of the discrepancy between the current sensory pattern and that pattern expected based on the neural store. Adaptability refers to the rate at which the neural store is updated as a result of experience with the novel sensory pattern. These concepts are important because they help to explain both the

large individual differences in the incidence of motion sickness and the reduced susceptibility with age. Receptivity and adaptability are orthogonal to one another.

Reason and Brand provided evidence from several perceptual psychophysical experiments to support the hypothesis that people differ in a systematic and consistent manner on the continuum of receptivity. Highly receptive people, they argued, experience all sensory phenomena more intensely. This means that the currently discordant sensory patterns caused by exposure to a novel motion environment will differ from stored patterns by a greater amount, will produce a greater subjective mismatch, and therefore more MS. Hence, there are individual differences in the incidence of MS. Assuming this receptivity degrades with age, they argued, this would help account for the reported reduction in MS with age. Human sensory systems do, in fact, decline with age (Kane, Ouslander, & Abrass, 1994).

The term adaptability "... refers to that rate at which an individual typically adjusts to conditions of sensory rearrangement" (Reason & Brand, 1975, p. 200). This means the time taken for the neural store to update its contents to be compatible with the current sensory pattern from the spatial senses. The more rapidly this store is updated, they argued, the shorter the adaptation period and the less MS the traveler must experience. Reason and Brand reviewed laboratory evidence to show that there are consistent individual rates of adaptability across a variety of different motion environments. Most people are fairly quick adaptors, many are very long adaptors, and an unfortunately few never adapt. Large individual differences in the trait of adaptability, they argued, would help account for the wide range of individual differences found in the incidence of MS.

The sensory conflict theory was created to explain the cause of MS. It says nothing, however, about the particular pattern of signs and symptoms of MS. One can ask why the generation of a mismatch signal should initiate such a wretched and debilitating syndrome complete with apathy, depression, pallor, sweating, stomach awareness, nausea, and vomiting. It certainly appears to be a violent overreaction to a simple need for adapting to a new motion environment.

Treisman (1977) argued that the pattern of signs and symptoms might have an evolutionary significance. MS is not limited to human beings. It is widespread throughout the animal kingdom. Many laboratory animals, mammals, and even fish have been made motion sick. In natural locomotion through its environment, an animal will only experience sensory decorrelation when there is a problem with one or more of its sensory systems. Such a problem would likely be caused by the accidental ingestion of neural toxins from a food source. Thus, it was hypothesized that the brain interprets sensory conflict as evidence of having been poisoned. So the mismatch signal initiates an emetic response to expel the poison. The evolutionary hypothesis, then, was that the mismatch signal triggers an emetic response that allows the animal to survive the poison and procreate (and learn what not to eat in future). So MS is part of the human phylogenetic legacy because it had survival value. MS only became a problem recently,

in evolutionary time, when human beings developed artificial modes of travel. There is some evidence in support of this hypothesis (Money & Cheung, 1983).

Although sensory conflict theory is almost universally accepted as the nominal explanation for MS, it is not without weaknesses. Even its proponents have leveled criticisms. For example, Reason and Brand (1975) reported large individual differences in incidence, severity, and duration of MS given constant conditions of motion. In laboratory experiments involving unique and novel motion, the period of time before sickness signs manifested themselves upon individuals could vary from a few minutes to several hours. This is an enormous range, given a constant and unique laboratory motion environment. The theory simply cannot explain this range except by positing wide individual differences in receptivity and adaptability thresholds. However, more pointed criticisms than this one are commonly stated in the literature.

Sensory conflict theory was criticized for being *post hoc* and having little predictive power (Kolasinski, 1995; Kennedy & Frank, 1985; McCauley, 1984; Riccio & Stoffregen, 1991; Stoffregen et al., 2000). This theory usually seems clear and reasonable after the fact, but it does not produce consistent—much less quantifiable—predictions. MS does not occur in environments where conflict would seem to be clearly present, such as in some tilted rooms or fixed-base simulators (Kennedy & Frank). Yet MS does occur in environments where conflict is apparently absent, such as vertical linear oscillations (McCauley), or self-induced MS from dancing, spinning, or vigorous head movements (Riccio & Stoffregen). Further, sensory conflict theory does not explain why the passive passenger in an aircraft, automobile, or simulator usually experiences more MS than the active pilot/driver/operator at the controls (Riccio & Stoffregen). Both passive and active individuals are experiencing the identical physical forces and sensory conflicts. In addition, it is widely reported that oscillations between 0.08—0.4 Hz cause MS. Yet, the normal human unperturbed standing sway oscillates between 0.01—0.4 Hz without people experiencing MS every time they stand still (Kennedy & Frank; Stoffregen, et al.).

Sensory conflict theory explains MS in terms of a mismatch between a current pattern of sensory stimulation and an expectation of a different pattern of sensory stimulation. This conceptualization has caused problems. First, the nature of the conflict is not defined in terms of the physical stimulus but in terms of variable sensory transducers (McCauley, 1984). Second, the nature of the conflict is between a current sensory pattern and an expectation of another. It is impossible to know the subjective expectation against which the sensory pattern is being compared. Sensory expectations are internal, subjective, and in principle not measurable (Riccio & Stoffregen, 1991). Hence, the sensory conflict theory is able, after the fact, to “explain” almost all results. Rapid onset of severe symptoms demonstrates that there was a large conflict between current and expected patterns. No MS demonstrates that there was evidently a slight or nonexistent conflict between current and expected patterns. The critical explanatory constructs are both variable and subjective. For these reasons it has been stated that the sensory conflict theory “...in its present form, it may be

untestable” (Stoffregen et al., 2000, p. 459) because in its present form it is “...not scientifically falsifiable” (Riccio & Stoffregen, p. 207).

Postural instability theory. Though not perfect, sensory conflict theory is the currently accepted explanation for MS. As described by Kuhn (1962), scientists do not abandon a usable and widely held theory just because it has flaws. A better more-comprehensive theory must first be created, disseminated, and defended. The postural instability theory (Riccio & Stoffregen, 1991) is a more recent explanation that is working its way through the domain of MS.

...animals become sick in situations in which they do not possess (or have not yet learned) strategies that are effective for the maintenance of postural stability... motion sickness results from prolonged instability in the control of posture. (Riccio & Stoffregen, 1991, pp. 195-196)

Situations that produce MS are characterized by their unfamiliarity to the participant—whether it is an animal, pilot, or subject in laboratory research. Postural instability (PI) theory states that this unfamiliarity leads to an inability to maintain postural control and this lack of control causes MS until the participant adapts. Riccio and Stoffregen (1991) argued that changes in sensory stimulation (i.e., conflict theory) do not cause MS. It is the control of action (i.e., postural control) that is the proximate cause of MS. This theory is based not on the sensory pattern or expectations, but on response to the motion environment. The locus of the problem of MS is action; the inability to control posture in a provocative motion environment.

This theory requires prolonged postural instability. One can produce the required level of instability in oneself simply by spinning around or vigorously moving one’s head while located in one’s ordinary everyday environment. This is a game children commonly play, and the author can vouch for its effectiveness at bringing forth MS. One can reduce postural instability merely by lying down. The fact that one can reduce both the onset and severity of symptoms of MS merely by lying in a supine position has been known since at least the 19th Century (Benson, 1978; Reason & Brand, 1975).

PI theory makes clear and testable predictions. For example, it states categorically that postural instability precedes the symptoms of MS and that without postural instability there will be no MS. Postural instability is a necessary factor in MS. Further, PI theory states that as the duration of instability increases the number and severity of MS symptoms increases. Riccio and Stoffregen (1991) went so far as to claim that, even when sensory conflict is present, if the participant is passively stabilized no MS will result. Passive stabilization can be achieved, they argue, by thoroughly restraining (strapping down) the participant’s head, body, and limbs or by making the participant neutrally buoyant in water.

As mentioned above, exposing participants to low frequency oscillations is nauseogenic. The range of frequencies that cause MS are within the range of

frequencies for normal bodily sway. According to PI theory, oscillations at this frequency produce MS because they interfere with normal human postural control motions and this instability causes the MS. The concept is one of wave interference, such as is found in physical acoustics. The interaction of the body's natural postural oscillation frequency with an oscillation frequency that is imposed by a ship, aircraft, camel, or laboratory device produces a new complex waveform that interferes with postural stability. It is the postural instability produced by this wave interference effect that causes MS. Stoffregen and Smart (1998) and Stoffregen et al. (2000) provided direct experimental evidence in support of this theory. That is, imposed oscillations produced objectively measurable postural instability, and this instability preceded signs and symptoms of MS.

Postural instability theory and age. "Instability of gait and falls are common among the elderly" (Kane et al., 1994, p. 197). Anywhere from 20 percent (Lyon, 2003, October) to 33 percent (Kane et al., 1994) of the elderly living at home suffer a serious fall each year. A major cause of these falls is age-related changes in neuromuscular function, gait, and postural reflexes. Among these age-related changes are:

...diminished proprioceptive input, slower righting reflexes, diminished strength of muscles important in maintaining posture, and increased postural sway. (Kane et al., 1994, p. 199)

Dizziness and unsteadiness are extremely common complaints among elderly people who fall (as well as those who don't). (Kane et al., 1994, p. 204)

Selected populations, such as those seen in an outpatient geriatric department, report incidences of dizziness as high as 81-90% of patients questioned... (Lyon, 2003, p. 2)

Aviators are not immune from these age-related changes. A study by Olive (as cited in McGuinness, Bouwman, & Forbes, 1981) correlated physical and medical data from 1,000 Naval aviators over a twenty-year period. Results indicated that susceptibility to vertigo and disorientation increased with age.

The maintenance of postural stability involves the interaction of several bodily systems, as discussed earlier, but the contribution of the vestibular system is primary. Age dependent vestibular degeneration is an established fact for human beings as well as other mammals (Lyon, 2003, October).

PI theory states clearly and repeatedly that postural instability is the proximate cause of MS. Postural instability increases with age—markedly so for the elderly. It follows from this that PI theory must predict an increase in MS with age. The incidence and severity of MS among the aged would be predicted to be significantly and substantially worse than that for young adults.

Note that this prediction from PI theory is precisely opposite to what sensory conflict theory predicts, as well as to the available self-report data. Sensory conflict theory predicts very little or no MS with advanced age because of greater motion experience and reduced receptivity. Further, as previously noted, it is widely reported that MS of any kind is “very rare” beyond age 50. This difference in theoretical predictions cannot be settled by reference to the self-report data. These data suffer from serious methodological weaknesses. It appears that further laboratory research is called for to answer this question. This research will probably need to use a cross-sectional methodology for reasons of practical efficiency (Tsang, 2003).

Simulator Sickness

Reviews

With the growth of simulation for aviator training in the 1980s came a concomitant increase in simulator-induced MS, which was labeled SS. This problem was duly noted and became the justification for increased research into the magnitude, correlates, causes, and treatment of SS. The results of this work have been reviewed extensively. Crowley and Gower (1988) offered an introductory review for the experienced aviator. The excellent books edited by McCauley (1984) and AGARD (1988) reviewed key areas of this research. Reviews by Kennedy and colleagues described the earlier research with special emphasis on the large Navy database (Kennedy, Berbaum, Allgood, Lane, Lilienthal, & Baltzley, 1988; Kennedy et al., 1987; Kennedy & Frank, 1985; Lilienthal et al., 1987). With the emergence of virtual environment technologies and helmet-mounted displays in the 1990s, the salience of the problem of SS increased again—and this time not just for aviation training but for consumer entertainment as well. Later reviews (Biocca, 1992; Ebenholtz, 1992; Kennedy & Fowlkes, 1992; Kennedy, Lane, Lilienthal, Berbaum, & Hettinger, 1992; Kolasinski, 1995, 1997; Pausch et al., 1992) expanded on the earlier reviews by including these newer technologies, where research was available, and addressing issues related to virtual reality. The detailed review by Wright (1995) addressed the problem of SS in the training of Army helicopter pilots.

Terminology and Distinctions

SS is a form of MS that does not require true motion—but does require a wide FOV visual display (Biocca, 1992; Mooij, 1988; Young, 2003). Like all varieties of MS, an intact vestibular system is necessary to experience SS (Ebenholtz, 1992). It has been called visually induced motion sickness (Benson, 1978; Reason & Brand, 1975; Pausch et al., 1992) and Cinerama sickness (Benson; Biocca, 1992; Reason & Brand). The term “vection” is used to describe a visually induced sense of self-motion. Vection is “... produced by the nearly uniform motion of a large part of the visual field... When the entire field moves, subjects soon begin to feel that the relative motion is their own” (Young, p. 98). Whether found in a flight simulator, Cinerama theatre, IMAX theatre, or virtual reality simulation, vection causes a MS-like discomfort for a substantial minority of participants. This unpleasant experience is now universally referred to as SS.

Further, these MS-like symptoms are now referred to as SS whether the simulator is a fixed-base model, and has no true motion, or a motion-base one with a (limited) range of movement. In other words, if the discomfort occurs in a simulator of any kind it will be called SS in the literature.

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... 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... (McCauley, 1984, p. 1)

A subtle distinction has been made between true MS and SS. MS is caused by motion. SS is caused by an inability to simulate the motion environment accurately enough (Kennedy et al., 1988; Kolasinski, 1995; Pausch et al., 1992). If a particular flight profile in an aircraft causes discomfort, this is MS. If the same profile is simulated veridically in a simulator, with the same physical forces present, and discomfort is caused, technically this is still MS. If a particular flight profile in the aircraft does not cause discomfort, but when simulated it does, this is SS. SS is discomfort produced in the simulator that does not occur when the same profile is executed in the physical motion environment. However, this is a logical distinction that apparently has no practical significance. As before, if the discomfort occurs in a simulator it will be called SS in the literature.

Selected History

Signs and symptoms of MS have been produced by visual stimulation alone in persons with an intact vestibular system. "This problem has been known to ophthalmologists and optometrists since the 1840s as the disorder termed asthenopia..." (Ebenholtz, 1992, p. 302). Asthenopia remained a little-known optical disorder until 1956 when aviators began operating the first fixed-base (non-motion) helicopter simulator.

Miller and Goodson (1958, 1960). Bell Aircraft Corporation was contracted by the Navy to develop a helicopter simulator for training visual flight skills and hovering. During preliminary demonstrations at Bell, prior to delivery to the Navy, it was found "...that a large number of observers (mostly helicopter pilots) experienced some degree of vertigo during these demonstrations" (Miller & Goodson, 1958, p. 7). The observers commented that their discomfort stemmed from the lack of vestibular cues to motion available from the fixed-base device.

Upon installation at the Naval Air Station, Pensacola, two psychologists (Havron & Butler) conducted an initial training evaluation of the device. During this evaluation

“... a questionnaire revealed that twenty-eight of thirty-six respondents experienced some degree of sickness” (Miller & Goodson, 1958, p. 8). These participants included flight instructors, students, and other personnel experienced both in the simulator and the helicopter. “The more experienced instructors seemed to be the most susceptible to these unpleasant sensations” (Miller & Goodson, p. 8). Sixty percent (60%) of the instructors reported SS symptoms, but only twelve percent (12%) of the students (Miller & Goodson, 1960). This SS usually occurred in the first ten minutes of a training session and frequently lasted for several hours afterward. The incidence and severity of this SS “... became such a serious problem that it was felt that unless it can be remedied in some way the utilization of such simulators as training devices would be limited considerably” (Miller & Goodson, p. 8).

As a part of their evaluation, Miller and Goodson (1958) interviewed several of the instructors from the earlier Havron and Butler study. “One of these men had been 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” (Miller & Goodson, p. 9). Miller and Goodson reported positive transfer of training from simulator to aircraft, albeit with a tiny sample size. Later Miller and Goodson conducted an experiment in an attempt to determine the effect of retinal disparity and convergence on SS in this device. They recruited 10 Navy enlisted men as participants. They were unable to find any effect of their independent variables upon SS and concluded that, due to large individual differences in the report of sickness, a “... great many more than ten subjects” (Miller & Goodson, p. 11) were needed to perform behavioral research on this phenomenon. They discussed problems with the device that caused several optical abnormalities. Specifically, Miller and Goodson (1960) noted visual distortions and conflicts that could have caused the SS, including: blurring of the image, distorted size perspective, and distorted movement parallax. While Miller and Goodson concluded that the discomfort found could have been caused by some combination of conflicts within the visual modality alone, they also reported that an inter-sensory conflict between vision and proprioception existed. Finally, they listed a number of advantages to using a simulator for aircraft training, including: safety, weather independence, training for special missions, and large economic savings. However, the SS problem “...became so serious that it was one of the chief reasons for discontinuing the use of the simulator” (Miller & Goodson, p. 212).

The events described above represent the first published accounts of SS. Several of the issues identified at the dawn of SS research have remained issues throughout the history of the field. To wit:

1. A substantial percentage of the people who operate the simulator experience SS. This is not a trivial event for simulator-based training—especially for helicopter training.
2. The personnel with more experience in the aircraft appear to have an increased susceptibility to SS.

3. Conflicts both inter-sensory (visual/vestibular) and intra-sensory (visual/visual or vestibular/vestibular) are implicated as the cause of SS.
4. The aftereffects of SS can last for hours.
5. Unless remedied in some way, SS will limit simulator-based training.
6. The Miller-Goodson anecdote. “One of these men had been 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.” This anecdote has been repeated frequently throughout the literature as evidence that safety issues are at stake in simulator-based training.
7. Sample size matters. Individual differences in susceptibility to, and reporting of, SS are so large that behavioral research requires large sample sizes.
8. Research shows positive transfer of training from the simulator to the aircraft for many tasks.
9. There are many advantages to simulator-based training besides positive transfer of training, including: safety, independence from (non-flyable) weather, the opportunity to train special missions (mission rehearsal), and large savings in the resources required for flight training.

McGuinness, Bouwman, and Forbes (1981). The Air Combat Maneuvering Simulator (ACMS) was installed at the Naval Air Station, Virginia Beach, in November 1979; it was commissioned in February 1980; and by March of 1980 reports of SS had found their way to the Naval Training Equipment Center for investigation (McGuinness et al.). The ACMS was a wide FOV, fixed-base, fixed-wing aircraft simulator designed to resemble the cockpits of F-4 and F-14 fighters. Questionnaires were administered to 66 aviators during individual, confidential interviews. The aviators were either pilots or radar intercept officers with flight experience ranging from 250 to 4000 hours. Each had four one-hour training sessions in the ACMS over a period of approximately one week.

Twenty-seven percent (27%) of the participants experienced at least one symptom of SS. The rate for participants with greater than 1500 flight hours experience was 47%, while for those with 1500 or fewer hours it was 18%. The ages of participants were not reported, nor were the incidence rates presented by age. The most common symptom reported was dizziness, followed by vertigo, disorientation, and nausea. There were no reports of flashbacks. Of those who reported symptoms of SS, 61% stated that these symptoms persisted between 15 minutes and 6 hours. Of those who reported symptoms, all symptoms subsided completely after a night's rest. Thirty-three percent (33%) of the aviators reported that the reset function (freezing the visual display and returning to a new set of initial conditions) was the most probable cause of SS onset. There was some evidence of adaptation to the simulator over the course of

several sessions. Finally, as a part of their literature review, the authors repeated the Miller-Goodson anecdote.

Several of the findings and explanations reported by McGuinness et al. (1981) have been replicated or cited in many other articles since then. For example:

1. The authors explained the SS found in their study with reference to the sensory conflict theory. They argued that there was an inter-sensory conflict between the vection produced by the wide FOV visual display and the lack of any actual motion (vestibular stimulation) in the fixed-base simulator.
2. They explained the differential rate of SS as a function of flight experience, measured by flight hours, in the same fashion. The relative sensory conflict would have been greater for the more experienced aviators because these aviators had a larger neural store of prior flight experience. Therefore, a larger conflict between the current pattern of sensory inputs and the expected pattern would translate into more SS. However, unlike many later researchers, McGuinness et al. did not ignore age entirely. They cited a report by Olive stating that susceptibility to vertigo and disorientation increased with increasing age of Naval aviators. They also stated:

Physiological body changes resulting from physical aging may also be a contributing factor to this phenomenon, since those with more flight hours naturally tend to fall into older age groups. (McGuinness et al., 1981, p. 25)

3. The SS symptoms reported by the participants, though similar to MS symptoms, were not identical. There were more vision and disorientation symptoms and fewer gastrointestinal symptoms. That is, there was less nausea and no emesis.
4. The symptoms had abated after one night's rest.
5. The freeze/reset function was implicated as causal in producing SS.
6. There was some evidence of adaptation over repeated simulator sessions.

McCauley (1984). McCauley described several potential operational problems that could result from SS. This discussion (McCauley's four points) was quickly adopted and repeated by later authors.

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 counterproductive in flight.

(McCauley, 1984, pp. 2-3)

These issues were discussed as potentially significant operational problems. For those who work in the field of simulator-based flight training, it is not a stretch to imagine that SS can affect safety and training. This possibility was noticed immediately (Miller & Goodson, 1958, 1960). However, note that McCauley explicitly stated that there was "no direct evidence" suggesting simulators are causally implicated in aircraft accidents. McCauley's four points appear frequently in published reports of SS.

Crowley (1987). In August 1984 the AH-1 Cobra Flight Weapons Simulator (FWS) became operational at Hanau U.S. Army Airfield in Germany. Soon thereafter reports of pilots becoming ill were made to Dr. Crowley, a flight surgeon at Hanau. Crowley's study was performed during the spring of 1985. The FWS was a motion-base simulator, employing a terrain board database, and moderately narrow FOV visual displays (48 degrees horizontal gunner station, 96 degrees horizontal pilot station). Anonymous questionnaires were administered to 115 Army Cobra pilots who were training using the FWS simulator at Hanau. One hundred twelve (112) questionnaires were returned (97%).

Forty percent (40%) of the participants reported at least one symptom of SS. Nausea was the most frequent symptom, followed by sweating, and dizziness. Three pilots (3%) reported vomiting. Pilots who reported SS symptoms had significantly more total flight time than those who did not report symptoms. Pilots with greater than 1,000 hours of Cobra flight time were significantly more likely to report SS than pilots with fewer than 1,000 hours. Experience in the FWS was significantly and negatively correlated with reported SS. That is, more simulator time in the FWS was associated with fewer reports of SS symptoms. Crowley (1987) explained these results in terms of the sensory conflict theory. He quoted the Miller-Goodson anecdote. He also discussed McCauley's four points and observed that any negative effects of SS upon training remained to be documented.

Because Crowley believed SS to be a potential hazard to aviation safety, a mandatory grounding policy was instituted at Hanau Army Airfield. The most significant portions of the Hanau policy were:

Aviators flying the AH-1 Flight Weapons Simulator (FWS) are medically restricted from flying duties until the beginning of the next duty day, (normally 0630-0730)... Any aviator forced to stop a simulator period early due to motion sickness is grounded until seen by a flight surgeon and returned to flying duty. (Crowley, 1987, p. 357)

Signs and Symptoms

SS is polysymptomatic (Kennedy & Fowlkes, 1992; Kennedy & Frank, 1985; Kennedy, Lane, Berbaum, & Lilienthal, 1993). Symptoms include nausea, dizziness, spinning sensations, visual flashbacks, motor dyskinesia, confusion, and drowsiness (McCauley, 1984). Observable signs of SS include pallor, cold sweating, and emesis (McCauley, 1984). The standard measurement instrument for SS, the Simulator Sickness Questionnaire (Kennedy, Lane, et al.), lists 16 symptoms: general discomfort, fatigue, headache, eyestrain, difficulty focusing, increased salivation, sweating, nausea, difficulty concentrating, fullness of head, blurred vision, dizzy (eyes open), dizzy (eyes closed), vertigo, stomach awareness, and burping. Reports of visual flashbacks and visual hallucinations have been documented (McCauley, 1984; Wright, 1995; Young, 2003) although they are reported to be exceedingly rare.

The reader will note that the signs and symptoms of SS overlap with those described above for MS. There are several differences, however. The most consistently reported difference is that while major symptoms of MS involve gastrointestinal distress (e.g., burping, stomach awareness, nausea, emesis), for SS there are fewer gastrointestinal symptoms and more visual ones (e.g., eyestrain, difficulty focusing, blurred vision, headache)(e.g., Kennedy et al., 1988; Kennedy & Fowlkes, 1992; Kennedy, Lane, et al., 1993; Kennedy, Lane, et al., 1992; Lilienthal et al., 1987; Uliano, Lambert, Kennedy, & Sheppard, 1986). Vomiting is a common sign of MS. For example, 75 percent of those suffering from seasickness vomit (Kennedy & Fowlkes, 1992). By comparison, vomiting is rare in SS—usually occurring in less than one percent (1%) of the cases (Kennedy & Fowlkes, 1992; Kennedy, Lane, et al., 1993). Finally, in cases of vection-induced SS, such as a fixed-base flight simulator, closing one's eyes will end the perceived motion and dramatically reduce the symptoms (Kennedy, Lane, et al., 1993). Closing one's eyes, however, will have no such effect on MS, as noted above.

Helicopter simulators have been widely reported to produce more SS than fixed-wing simulators (Baltzley, Kennedy, Berbaum, Lilienthal, & Gower, 1989; Kennedy et al., 1988; Kennedy, Lane, et al., 1992; Kennedy et al., 1989; Wright, 1995; Young, 2003). This is probably because helicopters are usually flown closer to the ground. Discomfort level varies inversely with height above terrain (Kennedy & Fowlkes, 1992; Kolasinski, 1995; Wright, 1995). There is a greater perception of visual flow, caused by greater visual detail, at lower height above terrain.

Several reports of original research include a listing of the most common symptoms found in helicopter simulators. Gower and Fowlkes (1989a) reported a study of the Cobra AH-1 FWS. This device incorporated a six-degree of freedom (6-DOF) motion base. (These six dimensions of motion are pitch, roll, yaw, vertical [heave], lateral [sway], and longitudinal [surge]). The most commonly reported symptoms from Gower and Fowlkes were eyestrain (37% of the participants) and fatigue (27%).

Gower, Lilienthal, Kennedy, Fowlkes, and Baltzley (1987) reported on another simulator of an attack helicopter. This was the Combat Mission Simulator for the Apache AH-64A. The CMS is an interactive, full-mission, 6-DOF simulator. The most commonly reported symptoms were fatigue (43% of participants), sweating (30%), and eyestrain (29%). Braithwaite and Braithwaite (1990) reported on a simulator for the British attack helicopter the Lynx. This device included a 6-DOF motion system with a 130 degree (horizontal) by 30 degree (vertical) FOV color projection visual system. The most commonly reported symptoms were disorientation (24% of participants) and difficulty focusing (24%).

Gower and Fowlkes (1989b) studied the SS potential of a simulator for the UH-60 Blackhawk utility helicopter. This device incorporated a 6-DOF motion base plus forward, left, and right out-the-window views from a collimated visual display. The most common symptoms were fatigue (35% of participants) and eyestrain (34%). Silverman and Slaughter (1995) reported on an operational flight trainer for the MH-60G PAVE Hawk helicopter. This was a fixed-base device. It provided a 150 degree (h) by 40 degree (v) out-the-window visual display plus two chin window displays. The most commonly reported symptoms were stomach awareness, dizziness, nausea, fatigue, and sweating in descending order of frequency.

Gower, Fowlkes, and Baltzley (1989) reported on the SS symptoms produced by the full-mission simulator model 2B31 for the CH-47 Chinook cargo helicopter. This was a 6-DOF motion device with a 48 degree (h) by 36 degree (v) forward visual display plus a 22 degree (h) by 30 degree (v) chin window display. The most commonly reported symptoms of SS were fatigue (34% of participants), eyestrain (29%), headache (17%), difficulty focusing (13%), sweating (11%), nausea (9%), and stomach awareness (9%).

Measurement

Several reviews discussed the difficulties with and tools for measuring SS (Casali & Frank, 1988; Hettinger, Nolan, Kennedy, Berbaum, Schnitzius, & Edinger, 1987; Kennedy & Fowlkes, 1992; Kolasinski, 1995). Because SS is polysymptomatic one cannot measure just one dependent variable (Kennedy & Fowlkes). Another measurement difficulty is that there are large individual differences in susceptibility to SS. It is common in this research to find that fully 50 percent of simulator operators experience no symptoms at all (Kennedy & Fowlkes). When effects of SS exist, they are often small, weak effects that disappear quickly upon exiting the simulator. Further, because most participants eventually adapt to the motion environment of a particular

simulator, researchers cannot reuse the same participants (such as in a within-subjects research design). Thus, researchers are forced to employ between-subjects research designs (Kennedy & Fowlkes). When one combines these factors of large individual differences, weak effects, adaptation, and between-subjects designs it invariably leads to the conclusion that research into SS requires large sample sizes. To get samples of this large size, researchers are forced to survey pilots training in simulators at military training centers (Kennedy & Fowlkes). However, these military centers exist to train pilots efficiently and effectively, not to perform research. This means that the level of experimental control exercised by a researcher is usually low. So research studies investigating SS are either vast surveys of nearly all pilots operating a particular simulator at a particular facility at a particular time, or small-scale experiments with rather more experimental control but much smaller sample sizes.

There are a number of possible ways to measure SS (Casali & Frank, 1988; Hettinger et al., 1987). One could employ direct observation of participants during a simulator session and note signs such as facial pallor and sweating. This is seldom done for research measurement (cf., Uliano et al., 1986), but often used by instructors at the simulator site to monitor their students. Another option would be self-report measures, such as the Simulator Sickness Questionnaire, that ask the participant to note the type and severity of symptoms currently being experienced. This method is universally performed in some fashion. A third option would be to instrument the participants and measure physiological conditions such as respiration rate and stomach activity. This method has been used upon occasion. Finally, one can employ tests of postural equilibrium to measure simulator-induced disorientation or ataxia. These tests have been widely employed, but with equivocal results.

Simulator Sickness Questionnaire (SSQ). The SSQ is currently the gold standard for measuring SS. This instrument was developed and validated by Kennedy, Lane, et al. (1993). The SSQ was developed based upon 1,119 pairs of pre-exposure/post-exposure scores from data that were collected and reported earlier (Baltzley et al., 1989; Kennedy et al., 1989). These data were collected from 10 Navy flight simulators representing both fixed-wing and rotary-wing aircraft. The simulators selected were both 6-DOF motion and fixed-base models, and also represented a variety of visual display technologies. The SSQ was developed and validated with data from pilots who reported to simulator training healthy and fit.

The SSQ is a self-report symptom checklist. It includes 16 symptoms that are associated with SS. Participants indicate the level of severity of the 16 symptoms that they are experiencing currently. For each of the 16 symptoms there are four levels of severity (none, slight, moderate, severe). The SSQ provides a Total Severity score as well as scores for three subscales (Nausea, Oculomotor, and Disorientation). The Total Severity score is a composite created from the three subscales. It is the best single measure because it provides an index of the overall symptoms. The three subscales provide diagnostic information about particular symptom categories. The Nausea subscale is made up of symptoms such as increased salivation, sweating, nausea, stomach awareness, and burping. The Oculomotor subscale includes symptoms such

as fatigue, headache, eyestrain, and difficulty focusing. The Disorientation subscale is composed of symptoms such as vertigo, dizzy (eyes open), dizzy (eyes closed), and blurred vision. The three subscales are not orthogonal to one another. There is a general factor common to all of them. Nonetheless, the subscales provide differential information as to symptomatology and are useful for determining the particular pattern of discomfort produced by a given simulator. All scores have as their lowest level a natural zero (no symptoms) and increase with increasing symptoms reported.

An important advantage of the SSQ is that a wide variety of symptoms can be measured quickly and easily with the administration of this one questionnaire. Another important advantage is that it allows quantitative comparisons across simulators, populations, and within the same simulator over time (as a diagnostic to determine if recalibration is needed, for example).

However, Kennedy, Lane, et al., (1993) stated restrictions in the use of the SSQ also. First, the SSQ is not to be used with participants who are in other than their usual state of health and fitness. The instrument was developed and validated based on data from healthy, fit pilots. Any scores obtained from participants who arrived for simulator training ill would be uninterpretable. Second, the authors recommended that the SSQ be administered immediately after a simulator session, but not before one. They did not recommend using pre-post difference scores. This is because the high correlation usually found between pre and post can render the difference scores unreliable. Nonetheless, researchers are so comfortable with the SSQ that they sometimes report pre-post difference scores anyway (e.g., Regan & Ramsey, 1996).

Instrumented physiological measures.

Changes in bodily cardiovascular, gastrointestinal, respiratory, biochemical, and temperature regulation functions often arise with simulator sickness. Several physiological measures have been electronically or electro-optically instrumented and transduced directly from subjects in simulator experiments. (Casali & Frank, 1988, pp. 9-10)

Heart rate, or pulse rate, has been reported to change from baseline levels as a function of simulator exposure (Casali & Frank, 1988). Unfortunately these reported changes are not sensitive, reliable, or always in the same direction. Respiration rate has proven to be a sensitive index of SS (Casali & Frank). However, the direction of the change is not consistent across individuals. As with MS (Reason & Brand, 1975) some individuals increase respiration rate upon simulator exposure, while others decrease rate. Casali and Frank recommend using an absolute difference score. Sweating is a common symptom of SS and this can be measured as an increase in skin conductance or a decrease in skin resistance (Casali & Frank). Facial pallor is also a common symptom of SS. Paleness of the skin can be measured using photo-optical sensors and has been shown to vary as a function of conditions that cause SS (Casali & Frank). Gastric activity can be measured with an electrogastragram. Gastric activity in the form

of tachygastria, a dramatic increase in stomach motility, has been shown to occur along with other symptoms of SS during exposure tovection (Casali & Frank; Hettinger et al., 1987).

Tests of postural equilibrium. Reviews of this methodology can be found in Casali and Frank (1988), Kennedy et al., (1997), and Kolasinski (1995). Postural equilibrium tests (PETs) exist to provide a behavioral measure of ataxia. Ataxia is a potentially dangerous symptom of SS. It is usually defined generically as:

An inability to coordinate voluntary muscular movements that is symptomatic of any of several disorders of the nervous system. (Merriam-Webster, 1971, p. 137)

Marked incoordination in voluntary muscular movements. (English & English, 1958, p. 48)

In the domain of SS research, ataxia is defined as postural instability, postural unsteadiness, or postural disequilibrium (e.g., Kennedy et al., 1997; Kolasinski & Gilson, 1999). It is thought that any disruption of balance and coordination that results from exposure to a simulator may be a safety concern for pilots who need to walk, climb stairs, drive, or fly after a simulator training session. The PETs are used to provide a direct index of postural instability.

Loss of balance and ataxia are common problems noted by trainees and subjects after exiting a dynamic simulator. The simulator presents an altered sensory environment which usually entails considerablevection, and some adaptation to this environment occurs in the operator's visual and vestibular sensory systems. Upon return to the "normal" environment, balance and equilibrium may be disrupted until the person progresses through re-adaptation. Such effects may be measured using pre-post simulator postural equilibrium tests. (Casali & Frank, 1988, p. 14)

There are several PETs that are described in the literature. They all involve some permutation of the following procedures: standing heel to toe with eyes closed and arms folded across the chest or back; or standing on one leg (preferred leg or non-preferred leg) with eyes closed or open and arms folded across the chest; or walking a straight line (on floor or rail) heel to toe with eyes closed or open and arms folded across the chest. The names and acronyms, where available, for several PETs are listed: Sharpened Romberg (SR), Stand on One Leg Eyes Closed (SOLEC), Stand On Preferred Leg Eyes Closed (SOPLEC, SOPL), Stand On Non-preferred Leg Eyes Closed (SONLEC, SONL), walk toe to heel, Walk On Floor Eyes Closed (WOFEC), Walk On Line Eyes Closed (WOLEC), and Walk On Rail Eyes Open (WOREO).

An example of a method for using PETs in research is described below:

Standing on Preferred Leg (SOPL): This test of standing steadiness required pilots to first determine which leg they preferred to stand on. Pilots were asked to stand, fold their arms against their chest, close their eyes, lift their non-preferred leg and lay it about two-thirds of the way up the standing leg's calf. They attempted to remain in that position for 30 s. If they moved their pivot foot, moved their raised foot away from their standing leg, grossly lost their erect body position, the trial ended and the time up to that point (in seconds) was recorded as the score for that trial.

Standing on Non-Preferred Leg (SONL): The procedure for this test was identical to that of the SOPL test except that pilots stood on their non-preferred leg. (Kennedy et al., 1997, p. 15)

The research literature shows mixed results when using PETs to demonstrate an effect of simulator exposure upon postural stability. Some studies have found no statistically significant effect of simulator exposure upon performance of PETs (Gower & Fowlkes, 1989b; Gower et al., 1989; Hamilton, Kantor, & Magee, 1989; Lampton et al., 1995; Uliano et al., 1986). Other studies have found a statistically significant effect for some or all PETs used (Duh, Parker, & Furness, 2001; Gower & Fowlkes, 1989a; Gower et al., 1987; Kennedy, Fowlkes, et al., 1993; Lerman, Sadovsky, Goldberg, Kedem, Peritz, & Pines, 1993; Warner, Serfoss, Baruch, & Hubbard, 1993).

There are several differences among the reports cited above. Nonetheless possible explanations for these equivocal results present themselves. As mentioned above with regard to SS in general, if an effect is highly subject to individual variability then large sample sizes are required. The mean sample size for the five studies listed above that did not report a significant difference was 61. For the six studies that reported positive results the mean sample size was 120. One cause of variability in performance can be differential rates of learning. Hamilton et al. (1989) demonstrated significant learning effects in the performance of four PETs (SR, SOLEC, WOREO, WOLEC). Further, performance on these four PETs continued to improve over the 10 practice sessions they measured. Therefore, when using PETs one must be aware that any improvement in performance occasioned by learning will tend to mask any decrement in performance caused by simulator exposure—if such a decrement exists.

Finally, Kennedy et al. (1997) found a statistically significant correlation between the Disorientation subscale of the SSQ and performance measures taken from two PETs (SOPL, SONL). The higher the Disorientation scores on the SSQ, the poorer the performance on the two PETs. In other words, the subjective self-reports of the pilot participants accurately reflected the behavioral measures taken from them after exiting the simulators. Given the potential measurement problems associated with PETs, the time and effort required in their administration, and the fact that similar results can be acquired more easily and quickly with the SSQ, the use of tests of postural equilibrium should probably be limited to research questions where their specific contribution is necessary.

Incidence

The incidence of SS varies widely across simulators and conditions. A common method of presenting incidence is to list the percentage of participants who reported at least one symptom. In the review by McCauley (1984) incidence was reported to range from 10 to 88 percent. In their review Kennedy and Frank (1985) reported that incidence ranged from 27 to 88 percent. In later reviews Kennedy and colleagues (Kennedy et al., 1987; Kennedy & Fowlkes, 1992) reported that the incidence of SS ranged from 12 to 60 percent in Navy flight simulators. Pausch et al., (1992) reported in their review that it could range from 0 to 90 percent in flight simulators. Wright (1995) limited his review to helicopter flight simulators. He reported that the incidence ranged from a low of 13 percent, when a strict criterion was employed to define SS, to a high of 70 percent, when a lax criterion was used.

It is widely reported that simulators of rotary-wing (RW) aircraft cause participants more SS than simulators of fixed-wing (FW) aircraft. Assuming a constant criterion of at least one reported symptom, there are several studies that report incidence by simulated aircraft type. Kennedy and colleagues (Kennedy et al., 1988; Kennedy et al., 1989) collected data from 1,186 simulator exposures. Their sample included data from 10 flight simulators. These simulators represented both FW and RW aircraft, and included both motion-base and fixed-base models. The incidence rates for FW simulators ranged from 10 to 47 percent. The rates for RW simulators ranged from 26 to 69 percent. Baltzley et al., (1989) collected data from 742 exposures using a self-report questionnaire. Their sample included data from operators of 11 flight simulators (7 FW, 4 RW). All participants had experience training in flight simulators. The incidence rates reported by pilots training in FW simulators ranged from 6 to 62 percent. The rates reported by pilots training in helicopter simulators ranged from 48 to 57 percent. These results have the advantages of large sample sizes, multiple flight simulators, and a constant method of research and analysis performed by the same investigators.

Magee, Kantor, and Sweeney (1988) collected data from a sample of 42 C-130 pilots and flight engineers. The C-130 Hercules is a multi-engine, propeller-driven, FW, cargo aircraft. The C-130 simulator included a 6-DOF motion base and a 120 degree (h) by 40 degree (v) FOV visual display. Participants performed a four-hour simulator session with a short break at the mid-point. Ninety-five percent (95%) of the participants reported at least one symptom of SS upon exiting the simulator.

Crowley (1987) reported an incidence rate of 40 percent for the RW Cobra FWS. Braithwaite and Braithwaite (1990) reported an incidence rate of 60 percent for 183 Lynx helicopter crewmembers that returned self-report questionnaires. Gower et al., (1987) collected data from 127 participants training in the AH-64 CMS. This simulator represents the AH-64A Apache helicopter. An incidence rate of 44 percent was reported. Gower and Fowlkes (1989a) collected data from 74 Army aviators training in the Cobra FWS. Thirty-seven percent (37%) of the participants reported at least one

symptom of SS. All four of the studies described in this paragraph reported results obtained from participants operating 6-DOF motion-base devices that simulated attack helicopters.

Lerman et al. (1993) collected data from 59 armor Soldiers performing tank driver training in a 3-DOF (pitch, roll, yaw) tank simulator. Sixty-eight percent (68%) of this sample reported at least one symptom of SS. Using the SSQ, Lampton et al. (1995) measured SS in an M-1 tank driver simulator mounted on a 6-DOF motion platform. They also measured discomfort in the actual M-1 tank. The authors reported significantly greater symptom scores in the simulator than in the tank. Upon interview, thirty-six percent (36%) of their sample reported experiencing discomfort in the simulator. The authors also reviewed the training records of six armor companies that had experienced the device previously. They found that 25 percent of these training records documented SS among the prior trainees. It is plausible that these incidence rates reported by Lampton and colleagues are conservative estimates. Instructors are not likely to mention SS in a written training document unless it is a significant phenomenon.

SS also exists in virtual reality (VR) simulators. For a review of SS from this perspective see Kolasinski (1995). Regan and Ramsey (1996) reported a 75 percent incidence rate for subjects in the placebo control group of a VR drug experiment. This level of discomfort was produced by a 20-minute immersion in the VR simulator. Kolasinski and Gilson (1999) immersed 40 research participants in a commercially available VR simulator for 20 minutes. Eighty-five percent (85%) of the participants reported at least one symptom of SS. It was because of high sickness rates such as these, produced by relatively short simulator sessions, that the practical future of VR technology became a subject of discussion (e.g., Biocca, 1992; Kolasinski, 1997; Pausch et al., 1992).

It is clear from the literature reviewed above that the incidence of SS varies within a large range. Depending upon the simulator, the conditions of operation, and the criterion definition applied, the rate of SS can vary from low to extremely high.

Residual Aftereffects

The potential for dangerous aftereffects of simulator exposure—including ataxia, loss of balance, flashbacks—has been noted right from the beginning (Miller & Goodson, 1958, 1960). In fact, the careful reader will meet the Miller-Goodson anecdote frequently in the literature—either quoted directly (e.g., Crowley, 1987; McCauley, 1984; McGuinness et al., 1981; Pausch et al., 1992; Wright, 1995) or, more often, referred to obliquely. McCauley's four points—two of which concern safety—are ubiquitous. Virtually every report refers in some way to these points, usually in the introductory section. So researchers have done their part to alert the community of the potential for dangerous aftereffects of simulator-based flight training.

However, it is only prudent to assure the reader that this potential danger has not manifested itself objectively. Many of the same authors reported that there were no documented cases of flight incidents or automobile accidents linked to prior simulator-based training (Crowley, 1987; Kennedy & Frank, 1985; McCauley, 1984; Wright, 1995). The present author has performed a follow-up study on several hundred simulator-trained Apache pilots (Johnson, in preparation). Not one aviator has reported an automobile or motorcycle accident within 12 hours of exiting the simulator.

Baltzley et al. (1989) reported data from a large study involving 742 simulator exposures across 11 Navy and Army simulators. Overall, 45 percent of the participants reported experiencing symptoms of SS upon exiting the simulator. Of these pilots who reported symptoms, 75 percent said that their symptoms disappeared within 1 hour. Six percent (6%) reported that their symptoms dissipated in 1 to 2 hours, 6 percent in 2 to 4 hours, 5 percent in 4 to 6 hours, and 8 percent reported that their symptoms lasted longer than 6 hours. The most common category of aftereffect was nausea (51%), followed by disorientation (28%), and oculomotor (21%).

Braithwaite and Braithwaite (1990) reported that 17 percent of their sample experienced aftereffects. The most frequently stated aftereffects were nausea, which dissipated in 2 hours, and headache, which sometimes lasted as long as 6 hours. Crowley (1987) reported that 11 percent of his sample experienced delayed effects of simulator training. The most commonly reported delayed symptom was a perception of illusory movement. Gower et al. (1987) reported aftereffects following training in the Apache CMS. Over a series of 10 training sessions, preflight minus postflight performance on 3 PETs decreased until session number 4 and then remained stable for the remainder of the simulator periods. This was interpreted as behavioral evidence of increasing simulator-induced disequilibrium over training trials.

McGuinness et al. (1981) reported that 18 members of their sample of 66 aviators (27%) experienced at least one symptom of SS. Of these 18, 11 (61%) stated that their symptoms persisted anywhere from 15 minutes to 6 hours. Silverman and Slaughter (1995) reported results from participants operating a wide FOV, fixed-base MH-60G operational flight trainer for the PAVE Hawk helicopter. Data were collected in conjunction with an operational test and evaluation of the simulator. Sortie lengths were at least 3 hours and included a full range of flight tasks. A total of 13 experienced aviators participated and filled-out self-report questionnaires. Eight (8) of these 13 participants (62%) reported at least one symptom aftereffect. The most commonly reported aftereffects were fatigue, stomach awareness, and vertigo, in that order. Most of these aftereffects came and went within 2 hours of exiting the simulator, although some participants reported symptoms lasting up to "...several hours after the simulator training session" (Silverman & Slaughter, p. 11).

There are some crude conclusions that emerge about the aftereffects of simulator exposure. First, approximately 10 percent of the sample will experience pronounced aftereffects (Kennedy et al., 1988; Kennedy & Fowlkes, 1992). Second, there is a significant positive correlation between the number and severity of symptoms

reported immediately upon leaving the simulator, and the duration and severity of aftereffects (Chappelow, 1988; Silverman & Slaughter, 1995). That is, those who experience the most SS during the simulator session usually experience the most aftereffects later. Third, the aftereffects of simulator exposure usually wear off in an hour or two. The persistence of symptoms longer than 6 hours has been documented repeatedly, but fortunately remains statistically infrequent.

It is understood in the training community that a potential exists for residual aftereffects to be a risk to flight safety. For this reason, guidelines recommending a mandatory grounding policy after training in a flight simulator have appeared both in the research literature and the training environment (Chappelow, 1988; Crowley, 1987; Crowley & Gower, 1988; Kennedy et al., 1988; Kennedy et al., 1987; Kennedy, Lane, et al., 1992; Lilienthal et al., 1987; NTSC, 1988). The minimum recommended period from simulator to aircraft has ranged from 6 to 12 hours and usually includes the admonition to wait until the next day. In cases of severe discomfort, temporary curtailment of other duties for up to 24 hours has been recommended (Kennedy et al., 1988). Currently in the U.S. Army, the policy on how much time must elapse from the end of a simulator training session to flight duty is a matter of unit standard operating procedure and is set by the unit commander (J. Riley, personal communication, April 26, 2004). At USAAVNC there is currently no regulation that restricts simulator-to-aircraft time—except for the Longbow Crew Trainer where the required delay is 1 hour (J. Riley, personal communication, April 26, 2004).

Allowing a night's sleep before recommencing flying duties should reduce residual risks to negligible proportions.
(Chappelow, 1988, p. 10)

During initial simulator training sessions or after a long period of not using the simulator, avoid scheduling simulator and aircraft flights on the same day.
(NTSC, 1988, p. 8)

Adaptation

The concept of adaptation in the literature of SS is identical to that in the literature of MS. Several reviewers have discussed adaptation to a novel simulated motion environment (Biocca, 1992; Crowley & Gower, 1988; Kennedy & Fowlkes, 1992; Kennedy & Frank, 1985; Kolasinski, 1995; Wright, 1995). The theoretical approach used to explain the fact that most participants adapt to the simulator after approximately six sessions (Biocca; Wright) is the sensory conflict theory.

Crowley (1987) found that there was a statistically significant inverse relationship between the prior number of hours spent training in the Cobra simulator and the amount of SS reported. The more prior exposure to the simulator, the less SS experienced currently. This was interpreted as evidence of adaptation. Gower and Fowlkes (1989a) reported the same inverse relationship with a different sample of Cobra pilots and

another FWS. Gower et al. (1989) reported a significant negative correlation between prior history of hours spent training in the CH-47 flight simulator and SS for a sample of experienced CH-47 pilots. Gower et al. (1987) investigated the effects of exposure to the AH-64A CMS on discomfort levels for 127 Apache aviators. Over the course of 10 training sessions, they found that self-reported SS symptoms decreased with increasing sessions in the CMS. They also reported an inverse relationship between the amount of simulator exposure during the prior 3 months and SS. Finally, they noted a significant negative correlation between the amount of recent CMS exposure and disequilibrium as measured by a PET. These results were interpreted as evidence of adaptation to the CMS.

Silverman and Slaughter (1995) reported evidence of adaptation to a MH-60G operational flight trainer for the PAVE Hawk helicopter. A sample of 13 experienced pilots executed a full range of flight tasks over several sessions in the simulator. The number of SS symptoms reported on later days were significantly fewer than the number reported on the first day of testing. Uliano et al. (1986) required 25 experienced pilots to operate the Vertical Take-off and Landing (VTOL) simulator which represents the SH-60B Seahawk helicopter. Each pilot flew the same flight paths, and performed the same tasks under the same experimental conditions, in counter-balanced order, over 3 days. SS was reported to be significantly worse on day 1 than day 2, and significantly worse on day 2 than day 3. The authors interpreted these results as evidence of adaptation to the simulator.

Besides reviewing the SS literature, Wright (1995) reported on his interviews with Army helicopter flight instructors. These instructors trained helicopter pilots daily. Yet, when introduced to a new simulator, they experienced SS symptoms. After a few days the symptoms disappeared or at least subsided to a minor and tolerable level. These instructors also reported that after several months away from the simulator, they had to readapt as if for the first time. Then readapt they did, again, in a few sessions. Wright interpreted these statements as evidence of adaptation to a novel (simulated) motion environment.

All of the studies cited above involved aviators adapting to a helicopter flight simulator of some kind. Lampton et al. (1995) reported evidence of adaptation to an M-1 tank driver trainer. They collected data from 115 trainees, all of whom had no prior experience driving a tank. Over the course of several training sessions the amount of SS the trainees experienced decreased. The symptom scores, as measured using the SSQ, were significantly higher after the first training session than after the remainder of the sessions. These results were interpreted as adaptation to the simulator.

Reports and manuals that provide guidelines for the detection and treatment of SS acknowledge adaptation as the best current solution to the problem of simulator-induced discomfort (e.g., Kennedy et al., 1987; Lilienthal et al., 1987; NTSC, 1988). As with MS, almost all participants eventually adapt to a simulated motion environment. Guidelines often describe procedures to employ during simulator-based flight training to encourage a rapid and reasonably comfortable adaptation period. For example:

Adaptation of the individual is one of the strongest and most potent fixes for simulator sickness... Do not schedule simulator hops for greater than two hours for any reason. (Kennedy et al., 1987, pp. 12, 17)

Persons new to the simulator, and particularly persons with extensive flight time, are at most risk... Decrease the field of view during nauseogenic hops (e.g., initial hops)... Go on instruments. (Lilienthal et al., 1987, pp. 277, 279)

Brief simulator flights (short hops with gentle maneuvers) separated by one-day intervals will facilitate adaptation to simulator motion and help prevent sickness, especially during the early stages of simulator training for novices and for experienced pilots with little simulator training... Do not slew while the visual scene is turned on... If all else fails, turn off the motion base or the visual scene and conduct instrument training. (NTSC, 1988, pp. 6-7)

Susceptibility

SS is not only polysymptomatic; it is polygenic (Kennedy & Fowlkes, 1992; Kennedy & Frank, 1985). Kennedy & Fowlkes listed 13 factors that are implicated in causing SS. These factors were subdivided into three categories: individual variables, simulator variables, and task variables. In an exhaustive review, Kolasinski (1995) described 40 factors that are associated with SS—also categorized as individual, simulator, and task variables. Pausch et al. (1992) reviewed several factors that evoke SS, with special emphasis given to simulator design issues.

Gender. As with MS (e.g., Reason & Brand, 1975), reviews of SS reported that females are more susceptible than males (e.g., Biocca, 1992; Kennedy & Frank, 1985; Kolasinski, 1995; Pausch et al., 1992). The precise reason for this is unknown. Reviewers have cited at least three possible explanations: hormonal differences, FOV differences, and biased self-report data. The hormonal hypothesis is the same as that advanced in the MS literature—females are more susceptible to SS during a portion of their menstrual cycle. This hypothesis is not without its doubters (e.g., Biocca; Pausch et al.). More likely, some think, is the fact that females have a larger effective FOV, and larger FOV is associated with greater SS (e.g., Biocca; Kennedy & Frank; Pausch et al.). Finally, those data upon which gender differences are based are self-reports. Males, it is suggested, may be more likely to under-report the severity of their discomfort (e.g., Biocca; Kolasinski).

However explained, reports of gender differences in SS continue to emerge. Hein (1993) reported the results of 22 separate studies, involving 469 participants, over the course of 6 years. All studies took place in a fixed-base, automobile-driving

simulator. Hein stated that gender differences in susceptibility to SS were among the most consistent results. "At all ages, female drivers are more susceptible than male drivers" (Hein, p. 610).

Age.

Walt Disney World's "Mission: Space" thrill ride left some older riders gulping, "Houston, we have a problem." In the past eight months, six people over 55 have been taken to the hospital for chest pain and nausea after going on the \$100 million ride... It is the most hospital visits for a single ride since Florida's major theme parks agreed in 2001 to report any serious injuries to the state... Last December, Disney began placing barf bags in the ride... (Schneider, 2004, p. B2)

Reviewers have reported that susceptibility to SS varies with age in the same way that MS varies with age (e.g., Biocca, 1992; Kennedy & Frank, 1985; Kolasinski, 1995; Pausch et al., 1992; Young, 2003). That is, below age 2 infants are generally immune. Susceptibility is at its highest level between ages 2 and about 12. There is a pronounced decline between ages 12 and 21. This decline continues, though more slowly, through adulthood until about age 50, after which SS is very rare. These claims are based on the self-report data reviewed by Reason and Brand (1975) for MS.

Perhaps the reason reviewers are forced to report conclusions based on decades-old self-report surveys of MS symptoms, is because so little research has been performed examining the effect of age on susceptibility to SS. Very few researchers have attempted to investigate the relationship between age and SS more directly. Braithwaite and Braithwaite (1990) administered questionnaires to 230 pilots attending training in a simulator for the Lynx attack helicopter. All were males. Age ranged from 23 to 42 years with a mean age of 32. There was no relationship found between age and reported SS.

Warner et al. (1993) assessed SS in two wide-FOV F-16 flight simulators. Twenty-four (24) male pilots participated in total. Sixteen (16) were active-duty military pilots of mean age 28.6 years (the "younger group"). Eight (8) were older active-duty military pilots and former military pilots of mean age 52.1 years (the "older group"). The task was a challenging 50-minute flight through a long, narrow, twisting canyon in each of the two simulators, in counter-balanced order, two weeks apart. One pilot from the younger group ($1/16 = 6.25\%$) terminated a session prematurely due to severe SS. Three pilots from the older group ($3/8 = 37.5\%$) terminated a session prematurely due to severe SS. The discomfort ratings (early version SSQ) collected from pilots who terminated prematurely were significantly higher than those from pilots who completed the flight. Among those pilots who completed the flight, there was no significant difference in discomfort ratings between the younger and older groups. Among those pilots who completed the flight, there was also no significant difference in postural equilibrium (SOLEC, WOFEC) between the groups.

Hein (1993) reported the results of 22 separate studies, involving 469 participants of both genders and a wide range of ages, over the course of 6 years. All studies took place in a fixed-base, automobile-driving simulator. Hein stated that age differences in susceptibility to SS were among the most consistent results. “Younger, male drivers adapt easily. Older drivers and women are severely susceptible to simulator sickness” (Hein, p. 611).

Age and experience. Among those (like the present author) who have been involved in the simulator-based training of large numbers of aviators, it is common knowledge that older participants are more susceptible to SS. Further, the small amount of evidence that does exist tends to support these anecdotal observations. Yet researchers investigating SS rarely even aggregate their data by age. Given the importance of age in both behavioral science and medical science research, this appears to be a glaring omission. Then, to confuse matters further, reviewers of the SS literature continue to repeat the conclusions published by Reason and Brand (1975) that sickness decreases with age and eventually almost disappears. Why is this so?

This is because researchers are convinced that the demographic variable that influences aviator SS is experience as measured in flight hours, not chronological age. Data are frequently aggregated by the flight hours of the participants. Researchers reviewing the literature discuss the impact of aircraft flight experience on SS. This view is also entirely consistent with the sensory conflict theory, where experience in a particular motion environment is central to the explanation.

However, among aviators age (in years) and experience (in flight hours) are strongly linked. Magee et al. (1988) reported a statistically significant correlation between age and flight hours ($r = 0.67$). The present author (Johnson, in preparation) has also found a significant correlation ($r = 0.75$) between these variables. This is because “As is common in most professions, piloting experience tends to accumulate with age” (Tsang, 2003, p. 525). Thus, disentangling age from experience is a knotty problem when examining SS among aviators (see Tsang, 2003).

It would not, in principle, be such a difficult problem to assess the effect of age upon SS if non-aviators were used as research participants. The present author predicts that among adult non-aviators, SS will increase with age rather than decrease. The chief methodological problems to be solved in order to perform this research would be practical ones. First, gaining access to a sufficiently large sample of non-aviators of a wide range of ages. Second, gaining access to a flight simulator for a period of time sufficient to collect the requisite large amount of data.

Experience. It is universally understood within this research community that the more experienced aviators are more susceptible to SS than novices. For example, this understanding has been acknowledged in at least 12 reviews covering the period from 1984 to 2003 (Benson, 1988; Crowley & Gower, 1988; Kennedy et al., 1987; Kennedy & Fowlkes, 1992; Kennedy & Frank, 1985; Kolasinski, 1995; Lilienthal et al., 1987;

McCauley, 1984; Mooij, 1988; Pausch et al., 1992; Young, 2003; Wright, 1995). In addition, some empirical evidence of this relationship has already been described earlier in the reports by Crowley (1987), McGuinness et al. (1981), and Miller and Goodson (1958, 1960).

Braithwaite and Braithwaite (1990) found a statistically significant positive correlation between experience as measured in flight hours and SS among pilots training in a Singer-Link simulator for the Lynx attack helicopter. That is, pilots with a greater number of flight hours reported greater SS. Gower and Fowlkes (1989b) assessed SS (early version SSQ) among 87 Army aviators training in a UH-60 helicopter simulator. They found a significant positive correlation between flight hours and SSQ scores. Gower et al. (1989) collected data from 57 aviators with flight experience ranging from 450 to 7,000 flight hours. The pilots were taking currency and refresher training in a 2B31 simulator for the CH-47 cargo helicopter. The authors found no correlation between flight hours and SSQ scores. Gower et al. (1987) assessed SS among 127 Apache aviators with flight experience ranging from 150 to 8,400 flight hours. All pilots were training in the AH-64 CMS built by Singer-Link. Again, the authors found no significant correlation between flight hours and reported SS symptoms.

Magee et al. (1988) assessed SS among a group of 42 male C-130 pilots and flight engineers operating a CAE C-130 simulator. Twenty-six (26) participants (the “experienced group”) had flight hours ranging from 845 to 10,000 (median 3,166). Sixteen (16) participants (the “novice group”) had flight hours ranging from 50 to 4,340 (median 1,465). There was no significant difference between the two groups in measured SS, either immediately after the simulator session or later. Also, a partial correlation of flight hours against measured SS, with age held constant, showed a small (0.03) and statistically insignificant result.

Silverman and Slaughter (1995) collected data from 13 aviators as part of an operational test of a MH-60G PAVE Hawk simulator. The participants’ total flight experience ranged from 350 to 15,327 hours. The authors reported that there was no statistically significant correlation between reported SS and either total flight hours or flight hours for the specific MH-60G helicopter. Uliano et al. (1986) assessed SS among 25 male helicopter pilots. Their flight experience ranged from 360 to 2,860 hours (mean 1,071). All participants operated the VTOL simulator, which represented the SH-60B Seahawk helicopter. Aviators with fewer than 900 flight hours experience reported significantly less SS on all measures than those with 900 or more flight hours.

Lerman et al. (1993) collected data from 59 male armor Soldiers operating a tank driver trainer. The authors found no significant correlation between amount of prior tank driving experience and SS symptoms.

Sensory conflict theory states that SS is caused when there is a difference between the current pattern of sensory information and what is expected on the basis of past experience. Thus, this theory predicts that the more flight experience an aviator

has acquired, the greater will be the disparity between his or her neural store and the pattern presented by the flight simulator—since a simulator cannot perfectly simulate flight—and the more SS will be reported. This is the explanation given when statistically significant differences are found between highly experienced aviators and novices or students.

Prior history of motion sickness. Generally speaking, in the behavioral sciences past behavior is the best predictor of future behavior. It follows that people who have a history of prior episodes of MS or SS will be more likely to experience SS in future simulator-based training. Two reviewers reported that there is empirical evidence in support of this generalization (Kennedy et al., 1987; Wright, 1995). Kennedy, Fowlkes, et al. (1992) discussed some of the methodological issues involved in using the Motion History Questionnaire (MHQ) to predict sickness scores in a simulator.

Braithwaite and Braithwaite (1990) reported that among their sample of helicopter pilots training in a Lynx simulator, there was a significant positive correlation between self-reported prior history of motion sickness (MHQ) and SS. That is, those with a history of MS were more likely to experience SS in the helicopter simulator. Gower and Fowlkes (1989a) reported a significant positive correlation between past history of MS as reported on the MHQ and reported SS while training in the Cobra FWS. Gower and Fowlkes (1989b) also reported a significant positive correlation between reported history of MS (MHQ) and SS among helicopter pilots training in a UH-60 simulator. Gower et al. (1989) found this same statistically significant relationship between MHQ scores and early-version SSQ scores for aviators training in a simulator for the CH-47 cargo helicopter.

Gower et al. (1987) collected data from 127 rated aviators training in the AH-64 CMS. They found a significant positive correlation between prior history as reported on the MHQ and SS as reported on a MS questionnaire. Kennedy et al. (1988) reported the results of surveying 1186 pilots training in 10 Navy simulators. Five of the simulators were FW and five were RW. They reported a small, but statistically significant, positive correlation between MHQ scores and SS symptoms. Warner et al. (1993) did not find any significant relationship between MHQ scores and SS symptoms for 24 pilots operating two F-16 simulators. Twenty-four (24) participants, however, is usually too small a sample size for a meaningful study of the correlates of SS.

Lampton et al. (1995) reported this same relationship for a sample of 115 male trainees operating an M-1 tank driver simulator. Trainees were asked, “Have you ever experienced motion sickness (such as in a car or bus, on a plane or train, on an amusement park ride, seasickness, etc.)?” Twenty-two percent (22%) responded in the affirmative. Those answering yes were significantly more likely to score higher on the SSQ. Lerman et al. (1993) assessed 59 male armor Soldiers during tank driver training in a Link tank simulator. The authors reported a significant positive relationship between prior history as measured by the MSQ and SS as measured by a MS questionnaire.

To summarize, two reviewers as well as eight of nine research studies document that a prior history of MS is positively correlated with SS. Past behavior is the best single predictor of future behavior.

Miscellaneous: illness, drugs, sleep, fatigue. There are several health-related conditions that are known to influence susceptibility to SS. As with MS, there is the pathology of an absent or nonfunctional vestibular system. Persons with this pathology (“labyrinthine defectives”) are incapable of experiencing either MS (e.g., Benson, 1978; Reason & Brand, 1975) or SS (e.g., Kennedy & Frank, 1985; Pausch et al., 1992).

It is widely understood among the research community that individuals should not participate in simulator-based training unless they are in their usual state of health and fitness. Individuals in ill health are more susceptible to SS (e.g., Kennedy et al., 1987; Kennedy & Fowlkes, 1992; Kolasinski, 1995; McCauley, 1984; NTSC, 1988; Pausch et al., 1992; Wright, 1995). Symptoms that make individuals more vulnerable include hangover, flu, respiratory illness, head cold, ear infection, ear blockage, and upset stomach. Individuals exhibiting these symptoms should not participate in simulator-based training or simulator-based research (Kennedy, Lane, et al., 1993). Similarly, it is widely known that certain medications, drugs, and alcohol can increase an aviator’s susceptibility to SS (e.g., Biocca, 1992; Kennedy et al., 1987; Kennedy & Fowlkes; NTSC; Young, 2003).

Reviewers have stated that fatigue and sleep loss also predispose an individual to SS (e.g., Kennedy et al., 1987; NTSC, 1988; Pausch et al., 1992; Wright, 1995). Gower and colleagues (Gower & Fowlkes, 1989a; Gower & Fowlkes, 1989b; Gower et al., 1987) have repeatedly reported a significant inverse relationship between the numbers of hours slept the previous night and SS as measured on an early version of the SSQ. That is, the fewer the hours slept, the greater the SSQ score. Gower et al. (1989) reported a significant negative biserial correlation between self-reported “enough sleep” (yes or no) and SS. That is, those aviators who reported that they had not had enough sleep last night, scored higher on the SSQ. This relationship between fatigue/sleep and SS is no trivial result. In military aviation training it is common for aviators to be less than fully rested during initial, advanced, or recurrent training.

Simulator variables. There are several simulator factors that have been implicated as causal in SS. Arguably the two most thorough reviews of these factors can be found in Kolasinski (1995) and Pausch et al. (1992). The review presented below is not an exhaustive listing of known simulator variables.

Wide FOV visual displays have long been associated with increased susceptibility to SS (Hein, 1993; Kennedy & Fowlkes, 1992; Kolasinski, 1995; McCauley, 1984; Pausch et al., 1992). This is because with a wider FOV there is a greater perception of visual flow orvection. Another visual factor with a long history of association with SS is known as off-axis viewing, design eye point, or viewing region (Kennedy & Fowlkes; Kolasinski; McCauley). Every visual flight simulator has a design eye point. This is the location within the cockpit where the visual display can be viewed

best and where the pilot should keep his or her head positioned. Moving one's head away from the design eye point, or optimal viewing region—by slouching or leaning forward, for example—will not only guarantee a poorer visual image, but will increase one's likelihood of experiencing discomfort. Perhaps the oldest visual factor known to evoke SS (e.g., Miller & Goodson, 1958, 1960) is optical distortion caused by misaligned or poorly calibrated optics (Ebenholtz, 1992; Kennedy & Fowlkes; Kolasinski; Lerman et al., 1993; McCauley). Finally, the general issue of cue asynchrony (visual delay, transport delay, asynchronous visual and motion systems) has been investigated as a source of SS, but with equivocal results (Hein, 1993; Kolasinski; McCauley; Pausch et al; Uliano et al., 1986).

Task variables. Not surprisingly, what the participant does while in the simulator, and what is done to him or her, can have a marked impact upon susceptibility to SS. These task factors were particularly well presented in the reviews by Kolasinski (1995) and McCauley (1984). The review of task variables presented below is not exhaustive.

First in importance is session duration (Gower & Fowlkes, 1989a; Gower et al., 1987; Kennedy & Fowlkes, 1992; Kolasinski, 1995; McCauley, 1984; Wright, 1995). The longer the period of time spent operating the simulator, the greater the likelihood of significant discomfort. Another important factor is use, by the instructor, of the freeze/reset command (Gower et al., 1989; Gower et al., 1987; Kennedy & Fowlkes; Kolasinski; McCauley; Wright). The more often the instructor freezes the pilot in mid-flight—to prevent a crash or provide instruction, for example—the more likely the pilot will experience SS. Other unusual or unnatural maneuvers, such as moving forward/backward in time or flying backwards, are also associated with increased risk of discomfort (Kolasinski).

Maneuver intensity (aggressive, dynamic, or violent maneuvering) has been implicated in SS, both in flight simulators (McCauley, 1984; Wright, 1995) and automobile simulators (Hein, 1993). Also, the height above terrain at which pilots fly has been shown to vary inversely with discomfort level (Gower et al., 1989; Kennedy & Fowlkes, 1992; Kolasinski, 1995; Wright). Flying close to the ground (nap of the earth) causes more SS than flying at altitude. This is usually explained in terms of greater perception of visual flow, caused by greater visual detail or density, at lower height above terrain. Degree of control has been associated with increased susceptibility to SS (Kolasinski; Pausch et al., 1992; Riccio & Stoffregen, 1991). The pilot in control of the simulator tends to report less discomfort than a passive passenger. Finally, head movements increase susceptibility to SS (Kennedy & Fowlkes; Kolasinski; McCauley; Riccio & Stoffregen). This last point has long been a part of simulator-trainee lore. Participants, who find themselves vulnerable to SS, quickly learn to keep their heads stationary.

Simulator Sickness, Performance, and Training

Performance. Does SS harm the flight performance of experienced aviators while in the simulator? Does exposure to a simulator temporarily harm the cognitive,

perceptual, or psychomotor performance of the participants? These are not subjects that have received a large amount of research attention.

Silverman and Slaughter (1995) stated that 67 percent of the helicopter pilots in their experiment reported modifying their flight control inputs at some point during the simulator sessions to alleviate the symptoms of SS. Pilots reported that they “slowed control inputs” or “transferred controls” or “closed my eyes during rapid aircraft movements” (p. 16). Uliano et al. (1986) had 25 experienced male helicopter pilots perform a series of tasks in the VTOL simulator. All pilots were to perform both an air taxi task and a slalom task according to prescribed standards. Performance in executing these tasks to standards was measured in three spatial dimensions (x, y, z). The authors found that there was a statistically significant negative correlation between the amount of SS reported and performance on the air taxi task. Specifically, the sicker were the aviators, the greater the percentage of time they flew out of tolerance in x, y, or z. The authors did not find a statistically significant relationship for the slalom task. Warner et al. (1993) assessed 24 pilots flying two F-16 flight simulators through a challenging 50-minute course. They collected 18 objective measures of piloting performance (e.g., airspeed limits, height above ground level, etc.). These they correlated with SSQ scores. The authors found no consistent relationship between SS scores and piloting performance.

As part of their larger survey of Navy simulators Kennedy et al. (1988) performed tests of cognitive, perceptual, and psychomotor capabilities. Three tests (Pattern Comparison, Grammatical Reasoning, Speed of Tapping) were administered both before and immediately after simulator exposure.

Pre- versus post-performance changes were studied in only six different simulators. In no simulator were group performances poorer after exposure, and indeed, most changes showed learning effects from the first (pre) to the second (post) session. Based on interpolations from other experiments on nonpilot subjects, these changes appear within the range of improvements due to practice which are to be expected over two sessions. (Kennedy et al., 1988, p. 5)

Kennedy, Fowlkes, et al. (1993) measured performance on three tasks (Pattern Comparison, Grammatical Reasoning, Finger Tapping) both before and after simulator exposure for 411 aviators engaged in simulator-based training. These data were compared to that from a control group of 16 aviators who were not exposed to a simulator between the first (pre) and second (post) test. Both groups showed improvement (a practice effect) from the pre-test to the post-test for all three tasks. However, the improvement shown by the control group was greater than that shown by the simulator-exposed group. This was a small, but statistically significant, difference. In other words, the simulator exposure attenuated the size of the practice effect for the simulator group. Uliano et al. (1986) tested 25 experienced male helicopter pilots on a grammatical reasoning task both before and after a 40-minute simulator flight. They

reported that there was no statistically significant effect of the simulator flight on performance of the grammatical reasoning task.

Based on the limited evidence that exists, it appears that simulator exposure has little or no effect on the cognitive, perceptual, or psychomotor abilities of aviators. These results are consistent with a larger set of results from the MS literature.

Training. With the exception of theme parks, simulators are used for training important and often dangerous skills—such as flying a helicopter or driving a tank. Does SS harm this training? For anyone who has experienced simulator-induced discomfort, it certainly appears reasonable to suggest that SS may interfere with training. But does it? What is the evidence?

The fear that SS would limit the usefulness of simulators for flight training has been in existence since the very beginning (Miller & Goodson, 1958, 1960). In fact, Miller and Goodson reported that use of the device they evaluated was discontinued. Recall also that two of McCauley's four points concerned this issue (McCauley, 1984). He warned of compromised training and decreased simulator use caused by SS.

When researchers review the literature of SS, the possibility of compromised training and/or decreased simulator use is a common feature. At least 15 times between 1986 and 1997 researchers have mentioned this potential problem of simulator-based training (Casali & Frank, 1988; Crowley, 1987; Crowley & Gower, 1988; Kennedy et al., 1988; Kennedy et al., 1987; Kennedy, Fowlkes, et al., 1992; Kennedy, Lane, et al., 1992; Kolasinski, 1995, 1997; Lampton et al., 1995; Lienthal et al., 1987; Mooij, 1988; Pausch et al., 1992; Wright, 1995; Uliano et al., 1986). Wright (1995) goes farther than other reviewers, however, by describing some of the evidence concerning SS and training.

Although studies indicate that sickness can occur, little—if any—research has investigated whether such sickness has an impact on training effectiveness. (Kolasinski, 1997, p. 151)

Given the primacy of this issue since 1958, it is remarkable how little empirical evidence there is on the subject. Chappelow (1988) administered questionnaires to 271 Royal Air Force pilots training in either of two air combat simulators. Respondents who had reported sickness symptoms were asked to assess the effect of the experience on their willingness to use the simulator in the future. A total of 214 pilots answered this question. Four percent (4%) reported that the experience decreased their willingness to use the simulator again. Sixty-eight percent (68%) responded that it had no influence. Twenty-eight percent (28%) stated that the experience increased their willingness to use the simulator again, because they said it provided good training and was fun to operate.

Gower and Fowlkes (1989a) assessed the effect of SS on training by asking their sample of AH-1 pilots whether simulator-induced discomfort hampers training. They

found two related results. First, there was a statistically significant positive correlation between SSQ scores and agreement with the statement that “discomfort hampers training.” That is, the aviators who reported the most SS were more likely to agree that discomfort harms training. Second, only 8 percent of their sample agreed, “discomfort hampers training.” Four percent (4%) were neutral on the question. Eighty-eight percent (88%) disagreed with the statement. It should be noted that these results were the self-reported opinions of Army aviators. No grades, test results, set-backs, training hours required, or other performance measures were presented to show in an objective fashion that, in fact, those participants experiencing more discomfort learned less than their non-sick counterparts.

Gower and Fowlkes (1989b) asked the same questions of their sample of UH-60 pilots and found the same pattern of results. First, there was a statistically significant positive correlation between SSQ scores and agreement with the statement that “discomfort hampers training.” Second, this was the opinion of a small minority of their sample. Only 1 person (1%) of the 86 who answered this question agreed that discomfort disrupts training. Fifteen percent (15%) were neutral. Eighty-four percent (84%) disagreed with the statement. Again, no data on performance during training were collected that would bear on the issue of SS and amount learned.

Gower et al. (1989) found the same pattern of results with their sample of helicopter pilots training in the CH-47 flight simulator. There was a significant positive correlation between SSQ scores and agreement with the statement that “discomfort hampers training.” Again, only 1 person (1.5%) agreed with the statement. Two people were neutral (2.9%). Of the total of 68 responses to this question, 65 (95.6%) disagreed with the statement. Finally, as before, no performance data were presented as to SS and amount learned during training.

The results of these four questionnaire studies are clear. The vast majority of the aviators surveyed stated that the discomfort-producing potential of the devices did not detract from the training provided. However, a small minority of aviators—those experiencing the most sickness—held the opposite opinion. Given the centrality of this issue for simulator-based training, more research should be undertaken. Measures of performance in learning the required program of instruction should be correlated with measures of SS such as the SSQ. In agreement with the quote from Kolasinski (1997) above, the present author knows of no published research devoted to this question.

Treatment

As with MS, the surest treatment for SS is simple adaptation. Nearly everyone will adapt to a particular simulator eventually. To aid adaptation to a new simulator, aviators should begin with brief simulator hops, flying gentle maneuvers, with subsequent hops separated by one-day intervals (NTSC, 1988). In this context, “brief” means less than one hour, with breaks as needed. The maximum duration of any simulator session should never exceed two hours. Several other guidelines exist and will be described later in this report.

For those pilots who cannot adapt to a simulator, "...anti-motion sickness medication may be considered for the simulator period" (Crowley, 1987, p. 357). Drugs previously used to control the symptoms of MS, such as hyoscine hydrobromide and dimenhydrinate (Dramamine), have also proven effective for relief of SS (Benson, 1978; Reason & Brand, 1975; Regan & Ramsey, 1996). In the world of flight training, it is no secret that some aviators with a history of discomfort self-medicate with MS drugs prior to a simulator session. However, no drug can reduce the occurrence of SS for everyone. Further, every drug has side effects. For example, scopolamine administered as a treatment for SS is known to have side effects that could negatively affect learning (Crowley, 1990). An aviator with severe, intractable SS should visit his or her flight surgeon.

Theory

SS is a form of MS. The two major theories that exist to explain MS are also used to explain SS. By far the more common is the sensory conflict theory (Benson, 1978; Reason, 1970, 1978; Reason & Brand, 1975). Virtually all research reports mention the sensory conflict theory by one of its names. Most authors employ it in the explication of their results. Early examples of how this theory has been applied to SS can be found in Kennedy and Frank (1985), McCauley (1984), and Reason and Brand. The major competitor is the postural instability theory (Riccio & Stoffregen, 1991; Stoffregen et al., 2000; Stoffregen & Smart, 1998). For a more detailed description of these two theories please see the discussion presented in the Motion Sickness section above.

Sensory conflict theory. The sensory conflict (SC) theory states that sensory inputs from the eyes, semicircular canals, otoliths, proprioceptors, and somatosensors are provided in parallel both to a neural store of past sensory patterns of spatial movement and to a comparator unit. This comparator unit compares the present pattern of motion information with that pattern expected based on prior motion history and stored in the neural store. A mismatch between the current pattern and the stored pattern generates a mismatch signal. This mismatch signal initiates both SS and the process of adaptation.

According to the SC theory, when an aviator is operating a new simulator the pattern of motion information presented by the senses is at variance with past experience in the flight environment. This conflict between the current sensory pattern and that pattern expected based upon past experience causes SS. That is, there is a conflict between the current novel motion environment and past experience. However, with continued sessions operating the device the relative mismatch between current pattern and stored patterns decreases until one has adapted. Flight simulators attempt to simulate flight—that is, to trick the human perceptual system. However, no device can perfectly simulate all the physical forces of flight. It is this inability to simulate flight perfectly that causes SS in experienced aviators.

However, one need not be an aviator to know the discomfort of SS. Anyone with a normal vestibular system is susceptible to SS when operating a novel motion simulator. The key concept is the mismatch between the novel motion environment (the current pattern of sensory stimulation in the simulator) and prior motion history (the patterns of sensory stimulation resident in the neural store). As the reader can see, the SC theory explains SS in exactly the same fashion it explains MS—only the motion environment has changed.

Postural instability theory. The PI theory notes that sickness-producing situations are characterized by their unfamiliarity to the participant. This unfamiliarity sometimes leads to an inability of the participant to maintain postural control. It is this postural instability that causes the discomfort—until the participant adapts. That is, a prolonged exposure to a novel motion environment causes postural instability that precedes and causes the sickness.

PI theory states that there are individual differences in postural stability. Further, an imposed motion presented by a simulator can induce postural instability. The interaction of the body's natural oscillation with the imposed oscillation created by the simulator leads to a form of wave interference effect that causes postural instability. This instability is the proximate cause of SS. Experimental evidence in support of this theory—from participants exposed to simulated motion—has been reported (Stoffregen et al., 2000; Stoffregen & Smart, 1998). The PI theory explains SS in exactly the same fashion it explains MS—only the nature of the novel motion environment has changed.

SS, age, and theory. The SC theory and the PI theory make different predictions in some instances. A few examples of these differences are presented earlier in this report in the Motion Sickness section. One issue on which these two competing theories make diametrically opposite predictions concerns the effect of age on susceptibility to SS.

The SC theory states that MS in all its forms must decline with age after about age 12. The reasons for this are that life experiences provide the neural store with a wealth of prior sensorimotor patterns of motion memories and also that receptivity (the strength of the mismatch) declines with age. The SC theory predicts that SS will decline with age. However when research shows that SS increases with age, these results are dismissed as being the product of a confounding with flight experience. Age and flight experience are strongly correlated among pilots. The SC theory predicts that with increasing flight hours the relative mismatch between the sensorimotor pattern of aircraft flight and that of simulator “flight” will be greater and will, therefore, engender more SS. However, this interpretation only exists because the overwhelming majority of simulator research has taken place in the world of aviator training—a world where older aviators are also more experienced aviators. The SC theory would have to predict that a large sample of adult non-aviators of widely different ages would show *decreasing* SS with increasing age.

The PI theory would have to make exactly the opposite prediction. Unlike the SC theory, the PI theory is stated in a way that allows it to be scientifically tested and falsified. According to this theory, SS is caused by postural instability. Postural stability among adults is known to decline with increasing age (e.g., Kane et al., 1994; Lyon, 2003, October). Therefore, PI theory would predict that a large sample of adult non-aviators of widely different ages would show *increasing* SS with increasing age. Further, within any age cohort this theory predicts that greater instability will be associated with greater SS. So this theory not only makes a general prediction concerning age, but also makes a prediction concerning specific aged adults.

It is not an everyday occurrence in science that two competing theories make precisely opposite predictions. The test suggested above could add to the theoretical understanding of all motion sickness phenomena. Again, the most difficult parts of this experiment would be to gain access both to a large sample of adult non-aviators, as well as to the simulator itself.

Guidelines for Reducing Simulator Sickness and Risks from Aftereffects

Several authors have taken the time to publish guidelines in an effort to reduce the rate of SS among trainee populations (Braithwaite & Braithwaite, 1990; Crowley & Gower, 1988; Kennedy et al., 1987; Kolasinski, 1995; Lilienthal et al., 1987; McCauley, 1984; NTSC, 1988; Wright, 1995). Arguably the most thorough set of guidelines are those by Kennedy et al. and Wright. These authors not only provide guidelines, but also explain the reasons for the guidelines and the evidence supporting them. If the reader does not have time for a detailed presentation, the best and most entertaining single source is the field manual published by the Naval Training Systems Center (NTSC).

The temptation to include guidelines of one's own is almost impossible for authors to resist. This is not only because SS is so discomforting to one's trainees, but also because some policies and procedures are clearly better than others. So in the interests of preventing future discomfort the current author will list some suggestions. This is by no means an exhaustive listing.

General rules.

- Simulator flights should not be scheduled on the same day as aircraft flights.
- Arrive for simulator training in your usual state of health and fitness.
 - Avoid fatigue or sleep loss, hangover, upset stomach, head colds, ear infections, ear blockages, upper respiratory illness, medications, and alcohol.
 - If you have been sick recently and are not fully recovered, reschedule your simulator training.
- Persons who are new to the simulator, or who have not operated it in months, are at risk.
- Do not schedule simulator sessions for greater than two hours for any reason.
 - Use breaks, time-outs extensively.
 - The more nauseogenic the session, the shorter the session should be.

- Aggressive, violent maneuvers, near ground level, are more nauseogenic than high, straight-and-level flight.
- Adaptation is one of the most potent fixes for SS.
 - In order to optimize adaptation, there should be a minimum of one day between simulator sessions, and a maximum of seven days.
 - Begin with short sessions, using non-nauseogenic maneuvers.
 - Minimize rapid gain and loss in altitude; minimize abrupt or continued roll; minimize porpoising.
 - Fly the most provocative tasks at the end of the session.
- Minimize head movement, particularly when new or dynamic maneuvers are being trained.
- Tell your instructor if you are experiencing discomfort.
- The instructor should avoid, or at least minimize, use of the freeze command.
 - Have the pilot close his or her eyes before using the freeze command.
 - Have the pilot close his or her eyes before resetting the simulator to another location. Or, turn off visual display before reset.
- The instructor should turn off visual display and turn on cabin lights before asking the pilot to exit the simulator.
- The instructor should decrease the field of view (turn off side displays) during early sessions, nauseogenic maneuvers, or if the pilot shows any symptoms of discomfort.
 - Or, go on instruments at the first sign of discomfort.
- Avoid high-risk activities for at least 12 hours after simulator training.
 - High-risk activities include flying, climbing, driving, riding motorcycles, riding bicycles, or diving.
 - Use handrails to help maintain balance when going up or down stairs.

Suggestions for Future Research

This review has uncovered at least two areas where further research into the subject of SS is clearly warranted.

- *The effect of SS on training.* As this review has shown repeatedly, one of the key arguments offered for studying SS is the potential for compromised training. However, there is virtually no evidence to support this argument. There is no evidence showing a statistically significant and substantial difference in the amount learned as a function of reported level of discomfort. Given that most simulator-based research takes place at aviation training sites, this oversight is particularly curious. This research topic is important and should be examined in a quantitative empirical fashion.
- *The effect of chronological age on SS.* Does increasing adult age make one more susceptible to SS or less susceptible? Are older aviators more susceptible to SS because they are older, because they have more flight experience, or some combination of both? Perhaps the best reason to investigate this subject parametrically is because the two leading theories of SS make precisely opposite

predictions. The SC theory predicts that SS will decrease with increasing chronological age. The PI theory predicts that SS will increase with increasing chronological age. Thus, performing this research has the added benefit of increasing our theoretical understanding of SS.

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