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Motion Sickness: Mechanisms, Prediction, Prevention and Treatment

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NORTH ATLANTIC TREATY ORGANIZATION

ADVISORY GROUP FOR AEROSPACE RESEARCH AND DEVELOPMENT

(ORGANISATION DU TRAITE DE L'ATLANTIQUE NORD)

AGARD Conference Proceedings No.372

MOTION SICKNESS: MECHANISMS, PREDICTION, PREVENTION AND TREATMENT

Papers presented at the Aerospace Medical Panel Symposium held in Williamsburg, US from 3 May to 4 May 1984.

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PREFACE

Motion sickness continues to be a significant operational problem in the armed forces of the NATO Countries. Air sickness degrades operational efficiency and raises the cost of flight training programmes by causing delays in training and higher attrition rates. With the introduction of surface effect ships that can achieve speeds up to 80 knots, sea sickness is also expected to be a considerable problem. In the US Space Shuttle Programme, space motion sickness has become a major operational concern. Nearly 50% of the shuttle crew members have experienced some symptoms of motion sickness.

In recent years, some progress has been made in identifying etiological factors that contribute to motion sickness and in treating motion sickness with pharmacological and desensitization techniques. The goal of this symposium was to provide an opportunity for this new information to be presented and discussed in ways that would be of direct operational benefit. This objective was clearly met and, in addition, areas in which further laboratory investigation is essential were identified.

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TECHNICAL EVALUATION REPORT

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1. INTRODUCTION

The Aerospace Medical Panel Symposium on "Motion Sickness : Mechanisms, Prediction, Prevention, and Treatment" was held in Williamsburg, Virginia, USA on 3 and 4 May, 1984. Twenty-one individual papers were delivered that are presented here as an AGARD Conference Proceedings. These papers concerned motion sickness on sea, land, in air, and in space and covered all four of the conference's rubrics.

2. SYMPOSIUM THEME

The Symposium addressed the continuing operational importance of motion sickness in the Air Forces and Navies of the NATO countries. Emphasis was also placed on the growing importance of motion sickness as an aspect of the Space Adaptation Syndrome in the first several days of orbital flight. Individual papers dealt with these themes from the perspectives of neurophysiological causes of motion sickness, prediction of susceptibility, incidences of air, sea, and space sickness, pharmacological and behavioral techniques for decreasing susceptibility, and adaptation and biofeedback techniques for treating airsick pilot trainees.

3. PURPOSE AND SCOPE

The goal of the Symposium was to provide an opportunity to disseminate information about recent advances that have been made within the laboratory and in operational settings concerning the nature and etiology of motion sickness, prediction of motion sickness susceptibility for different exposure conditions, pharmacological, adaptation and biofeedback techniques for preventing or ameliorating motion sickness, and the incidence of motion sickness in different exposure environments. The selection of topics was such as to allow comparison of treatments and selection procedures across different countries and the opportunity to incorporate laboratory findings into operational aproaches to the problems of selection, prevention, and treatment.

4. SYMPOSIUM PROGRAM

The program included twenty-one papers that fell into five genral categories: Physiological Mechanisms of Motion Sickness, Sickness in Motion Simulators, Behavioral and Pharmacological Countermeasures, including Biofeedback, Space Motion Sickness, and Prediction of Susceptibility.

5. TECHNICAL EVALUATION

Motion sickness is an immensely complicated topic. It was not possible with the relatively small number of papers presented and the brief duration of the symposium to deal with the problem both in depth and range. Consequently, the range of topics to be discussed had been narrowed to include those of most immediate operational concern.

Physiological Mechanisms of Motion Sickness. Miller and Wilson (paper 21), presented evidence a) that vomiting could still be elicited in animals after lesions of the uvula and nodulus of the cerebellum and b) that using electrical stimulation they could not localize a vomiting center in the brain stem. These observations call into direct question accepted beliefs concerning the neurophysiological causes of motion sickness. They are signally important in showing that the physiological bases of motion sickness are even less well known than thought before and that great concern must be devoted to characteristics of the test situation, e.g. type and range of provocative stimuli, duration of stimulation, and to establishing the character and extent of lesions in experimental animals.

Insight into the role of vestibular-visual interactions in the elicitation of motion sickness in cat and squirrel monkey was provided by Daunton, et al (paper 31). They showed that in these animals as in man, visual stimulation alone can elicit motion sickness. Interestingly, animals most sensitive to such visual stimulation were generally also most susceptible to combined visuo-vestibular stimulation. This observation has relevance both for developing susceptibility tests as well as physiological theories of the role of visual-vestibular convergence in motion sickness and spatial orientation.

Prediction of Susceptibility. Seven papers (25-31) dealt with possible psychological and physiological correlates of susceptibility to car, air, sea, and space sickness. No relationship was found between cardiovascular changes and motion sickness susceptibility and incidence of car sickness (Vogel, 25). Although correlational analyses suggest that individuals who show greater head and trunk movement when stepping in place tend to be more susceptible (Claussen, 28), Bles, et al (27) studying correlates of chronic sea sickness found as others have, too, that nystagmus and sensation cupulograms are not discriminative nor is the time constant of nystagmus decay after impulse deceleration. However, they found that the susceptible subjects tended to have greater labyrinthine asymmetries during caloric stimulation and tended to be more visually dependent in a tilting room. These findings point to the greater tendency for underlying vestibular abnormalities in the chronically susceptible subjects and accordingly a greater dependence on visual cues for maintaining postural balance.

Lentz et al (29) and Hixon et al (30) described the long-term efforts of the U.S. Navy's Aerospace Medical Research Laboratory in developing predictors of air sickness and in identifying the training situations where sickness usually occurs. There are many important observations from these studies including that most sickness occurs in basic training in VIP, that self-ratings and instructor ratings of motion sickness are very well correlated. Motion sickness history questionnaires and a variety of provocative susceptibility tests involving different patterns of otolithic, canalicular, and visual stimulation also correlate with susceptibility to air sickness. However, the correlations are not large enough to use the procedures as selection determinants. Important characteristics of useful selection procedures were also identified in these papers: a) generalizability of the test, b) generation of few false positives, c) brevity, d) inexpensiveness, and e) with large populations, the capability of decreasing the stressor and with small populations of increasing it to make it more like an operational situation. Lager et al (31) presented evidence suggesting that susceptibility to air sickness may be related to the intensity of illusory motion experienced during Coriolis stimulation, a finding that complements Reason's suggestion that susceptibility may be related to the duration of sensory after-effects because of an increased slope of psychophysical functions relating perceptual magnitude and sensory stimulus magnitude, suggesting in Reason's terminology greater "receptivity" in susceptible subjects.

Motion Sickness has become a significant operational concern in the United States' STS shuttle program. Reschke et al (26) presented a comprehensive analysis of the problem and a summary of their extensive experimental program to identify predictive tests of susceptibility to space motion sickness. As part of this goal, they have used ground based tests to measure susceptibility to motion sickness during exposure to various combinations of provocative visual, canalicular, and otolithic stimulation, they have also measured vestibulo-ocular reflex phase and gain, and postural ataxia. The results of these procedures have been correlated with susceptibility to motion sickness during parabolic flight maneuvers involving alternate periods of microgravity and macrogravity. None of the correlations was sufficiently great to justify rejecting the use of any of the ground-based procedures. Many females were included in the population evaluated and it is important to note that systematic differences in susceptibility were not present between males and females, despite the popular belief that females are more susceptible than men.

Together the findings of the papers in this series can be taken to indicate that a) there are no simple, highly reliable ground-based tests of sea, air, or space sickness, b) that there are few, if any, reliable psychophysiological correlates of motion sickness susceptibility in the normal population, and c) that susceptibility to motion sickness during exposure to provocative stimulation implicating one combination of receptors or receptor systems may not be highly correlated with susceptibility to stimulation of different receptor systems or even the same receptor systems stimulated at different frequencies. In short, it is unlikely that a simple test of motion sickness susceptibility can be useful for more than one "motion environment" and an adequate assessment of susceptibility for a particular motion environment will require more than one test. In the case of space motion sickness, the full range of provocative characteristics of microgravity have yet to be determined; as a consequence, the development of predictive tests is especially difficult.

Motion Simulators. Surface effect ships, largely supported on air, that can attain speeds up to 80 knots will soon be commonplace. Anderson et al (38) have simulated the heave, pitch, and roll motion components in the range to be expected in these ships, 0.05-1.5Hz, and measured movements both of the wave motion simulator and the subjects' heads. These findings are important in showing that when the amplitude of spectral components near 0.16 Hz increases, the liklihood of emesis also increases; in addition, when subjects experienced nausea they also exhibited increased lateral head sway - the reason for this is not known.

Kennedy et al (34) provided a comprehensive summary of information about sickness in flight simulators. Several important conclusions can be made : simulator sickness is motion sickness, more experienced aircrew are more likely to experience sickness, negative transfer to flight is possible, exposure frequency and duration are important, and the greater the fidelity of simulation the more likely sickness will be elicited.

From these papers, it is evident that simulators will continue to be useful in training and that it is valuable to use simulators as experimental tools for making predictions about susceptibility to motion sicksness under operational conditions.

Countermeasures to Airsickness. The symposium was particularly valuable in allowing a comparison of techniques that are being used to treat airsickness in flight training programs in the United Kingdom, The Federal Republic of Germany and the United States. Stott et al (40) described the RAF program on desensitization which is a two part program involving ground-based and, later, in-flight exposure to progressively stressful levels of vestibular stimulation. Prior to 1981 only one form of vestibular stimulation, Coriolis cross-coupled angular acceleration was used, since then two additional patterns, vertical oscillation and angular oscillation, have also been used. The success rate of the program - measured in terms of return to active flying was 70% before 1981, and 84% in 1981-1983. No one who applied for the program between 1981 and 1983 was denied access and there is excellent persistence of training. Biofeedback is not part of the training program.

The German program for treating airsickness (Kessler et al 41) also has an excellent success rate. Since greater emphasis was placed on using selection criteria for participation that stress

strong aptitude for flying and great motivation the success rate has reached 87%. Like the RAF program, it makes use of "vestibular desensitization" training by exposing subjects to progressively greater levels of provocative vestibular stimulation. In addition, it incorporates physical fitness training, relaxation training, cognitive pre-training, and self-control management. It does not use biofeedback.

Jones and Hartman (42) described the USAF program for biofeedback treatment of airsick crew members. This program places great emphasis on pre-screening candidates for high-motivation, providing psychiatric interviews and counselling, giving candidates training in how to perform relaxation exercises, and training them to use biofeedback to control their physiological responses. Candidates are treated by exposing them to progressively greater levels of Coriolis stimulation in a biaxial rotating chair while they are provided biofeedback about several of their physiological responses to the Coriolis stressor. Earlier work in this program using a different type of vestibular stimulation and not involving biofeedback had a success rate of approximately 40%. Since the incorporation of progressively stressful Coriolis stimulation and biofeedback, the success rate has been approximately 75%.

The features that are common to all three programs are the reliance on progressive exposure to provocative vestibular stimulation and the emphasis on the importance of high motivation in the trainees. The slightly higher success rates of the RAF and German desensitization programs compared to the USAF program are likely due to the use of more effective vestibular adaptation paradigms involving additional forms of vestibular stimulation besides Coriolis cross-coupled angular accelerations. No conclusions about the relative contribution of biofeedback in preventing air sickness can be drawn, the USAF program is a treatment rather than an experimental program and control comparisons were not made. It is possible that if biofeedback had been used as part of the RAF and German programs, their success rates would have been even better.

Space Motion Sickness. Valuable information was presented by Homick et al (36) on space motion sickness in STS flights 1-9, and by Oman et al (35) and Money et al (33) on motion sickness in Spacelab 1. From available evidence obtained in STS 1-9, it appears that space motion sickness a) has similar properties to terrestrial motion sickness-except that pallor is less common, presumably owing to the rostral redistribution of body fluids in orbital flight because of the absence of hydrostatic pressure in the circulatory system, b) is evoked by head and body movements, c) is largely over by mission day 4, and d) tends to be helped by antimotion sickness drugs (Homick et al 36).

Excellent in-flight characterization of symptom development and expression was obtained in Spacelab 1. Head movements, especially head movements in pitch, tended to elicit symptoms and astronauts spontaneously reduced their head movements for the first several days; even after three or four days vigorous head movements still elicited symptoms and roll head movements produced oscillopsia; rapid vomiting occurred but the astronauts always had other symptoms of motion sickness beforehand; as with terrestrial motion sickness, vomiting brought symptomatic relief for some period afterwards; no evidence was apparent for fluid shifts being an important etiological factor in space sickness (Oman et al 35).

Money et al (33) attempted to correlate for the Spacelab 1 astronauts their pre-flight motion-sickness susceptibility test scores on four provocative test conditions with their in-flight susceptibility. Little correlation was apparent; and their observations point to the difficulty of attempting such correlations under operational conditions, e.g. the number of participants is small and it is difficult to rank order in-flight susceptibility when activity levels are different and antimotion sickness drugs are being taken on different schedules. An important finding from Spacelab 1 and from STS 1-9 is that motion sickness was not experienced after return to Earth. Moreover, the astronauts seemed less susceptible post-flight than pre-flight to several forms of provocative vestibular stimulation (cf. Homick et al 36; Money et al, 33; and Oman et al 35).

Evidence for the nature of the changes in vestibular and vestibulo-ocular function that occur in microgravity and that may be related to space motion sickness was available from observations in Spacelab 1 and parabolic flight experiments. Von Baumgarten et al (37) found during the SL-1 mission that thresholds for linear acceleration were elevated in-flight and that ocular counterrolling was decreased post-flight. This pattern suggests a decrease in gain of CNS processing of otolith information as adaptation to the space environment proceeds. Some nystagmus responses to in-flight caloric irrigation were also found later in the mission, but the mechanism for this remains uncertain. Using parabolic flight maneuvers to create Gx variations, Vesterhauge et al (24) found an upward-beating nystagmus at high force levels and a slight downward beating nystagmus in 0G; both were inhibited by visual fixation. The presence of these nystagmus patterns points to changes in oculomotor function in the space environment, as does the further finding by Vesterhauge et al that the latency of saccadic eye movements increase in free fall relative to 1G test conditions. Related studies by Lackner and Graybiel (22) indicate that the apparent magnitude and the elicitation of motion sickness by Coriolis cross-coupled angular accelerations are highly gravitoinertial force dependant, 0G test conditions are much less stressful and provocative than 2G conditions for constant levels of Coriolis stimulation. This finding provides an explanation for the decreased in-flight susceptibility of the Skylab astronauts to Coriolis stimulation in the Skylab M-131 experiment.

It is clear from the evidence presented at the Symposium that space motion sickness is likely of multiple etiological origins, that it has similar characteristics to other forms of motion sickness, and that it will likely prove as refractory to prevention and treatment as motion sickness has under operational conditions on land and sea, and in air.

6. CONCLUSIONS

The Symposium despite its brevity was extremely successful in delineating our current knowledge of motion sickness under terrestrial and space conditions. The principal conclusions from the topics covered include:

6.1 Understanding of the physiological mechanisms responsible for the elicitation of motion sickness

is less advanced than generally believed.

6.2 There is little relationship between psychological and psychophysiological variables and susceptibility to motion sickness.

6.3 Space motion sickness seems to be elicited by head movements in microgravity and appears to have similar characteristics to motion sickness observed under terrestrial conditions.

6.4 "Simulator sickness" appears to be a form of motion sickness.

6.5 Programs for treating air sick flight candidates are achieving quite good success rates.

7. RECOMMENDATIONS

7.1 Virtually all aspects of motion sickness and how to deal with it - mechanisms, contributing etiological factors, predictive tests of susceptibility, physiological correlates, adaptation - require additional intensive investigation. Our knowledge is quite restricted at present despite many years of study.

7.2 It would be valuable every several years to hold additional meetings on motion sickness to allow monitoring of progress and reassessment of goals.

Neurophysiological Correlates of Motion Sickness: Role of Vestibulocerebellum and "Vomiting Center" Reanalyzed

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SUMMARY. Unexpected findings were obtained regarding (1) the role of the nodulus and uvula of the vestibulocerebellum in vestibular-induced vomiting and (2) the existence of a readily identifiable, discretely localized "vomiting center". Sinusoidal electrical stimulation of the vestibular labyrinths of decerebrate cats could produce vomiting and related activity similar to that observed during motion sickness. These symptoms occurred in animals with lesions of the posterior cerebellar vermis that included the nodulus and uvula, indicating, by analogy, that these structures are not essential for the development of many symptoms of motion sickness in intact animals. In a second series of experiments, electrical stimulation of the brainstem was used in an attempt to localize a "vomiting center" to a restricted anatomical region. Vomiting proved difficult to produce; a "vomiting center", stimulation of which evoked readily reproducible results, could not be identified.

INTRODUCTION

Our current concept of the neurophysiological correlates of motion sickness is largely based on work done prior to the mid-1950's (reviewed in 8,9). In 1956, Wang and Chinn organized this body of knowledge and proposed that motion-induced vomiting is produced by vestibular signals that traverse the nodulus and uvula of the vestibulocerebellum and then somehow activate the chemoceptive emetic trigger zone in the area postrema which in turn activates the medullary vomiting center (12). However, our recent experiments have raised questions about both the role of the nodulus and uvula in vestibular-induced vomiting and the presence of a discretely localized "vomiting center".

Lesions of the nodulus and uvula have been reported to prevent not only motion-induced vomiting (1, 2, 12) but also prodromal symptoms of motion sickness in dogs (1, 2). Wang and Chinn concluded that lesions including only one third to one half of the entire nodulus-uvula complex were large enough to be effective (12). We re-examined the role of these structures by using sinusoidal electrical polarization of the labyrinths to mimic natural vestibular stimulation in decerebrate cats, some of which had lesions of the nodulus and uvula (6).

The generally accepted concept of a "vomiting center" is based on studies by Borison and Wang who showed that vomiting can be produced by electrical stimulation of a region of the brainstem of decerebrate cats (3) and that large lesions in this area render dogs refractory to emetic agents (11). Their effective stimulating sites were located in the region of the solitary tract and nucleus and nearby lateral reticular formation, over a rostral-caudal distance of about 4 1/2 mm. In a second series of experiments, we also used electrical stimulation of the brainstem of decerebrate cats in an attempt to obtain a more restricted anatomical localization of a "vomiting center" (7).

METHODS

Experiments were performed on unanesthetized, decerebrate adult cats. Animals were chosen at random, without regard to their susceptibility to motion sickness. Initial surgical procedures prior to decerebration were carried out under halothane/nitrous oxide anesthesia, with the exception of one cat that was anesthetized with Nembutal (40 mg/kg i.p.) and decerebrated on the day prior to the experiment. The animals were held in a stereotaxic frame. Blood pressure was monitored, and rectal temperature was maintained between 36 and 38 degrees C. At least 2 hours usually elapsed between decerebration and the start of electrical stimulation.

In the first series of experiments, silver/silver chloride ball electrodes were implanted to stimulate the vestibular labyrinths of 14 cats. Stimuli consisted of polarizing currents, having as a waveform either single sinusoids (0.2-0.6 Hz) or a sum of 10 sinusoids (0.035-0.809 Hz) (13), which were applied for an average of about 6 hours (range 2 3/4 to 8/3/4 hours). Current levels were on average about 2 1/2 times that required for producing reflexly induced eye movements. No facial movements were observed which would have indicated current spread to the facial nerve.

Large lesions of the posterior cerebellar vermis were carefully made in 10 of these cats. Postmortem examination under a dissecting microscope revealed that the lesions always included the nodulus and uvula as well as the pyramis and tuber and usually some adjacent cerebellar tissue. Lesions were almost always complete; only occasionally did a small remnant of tissue remain. The extent of the lesion was confirmed in 3 cats by examining thionin-stained, parasagittal sections (100 μm) of the brain. The lesions in these animals were also found to include the caudal part of the fastigial nucleus, which was probably affected in most animals, and in 1 cat, the caudal part of the nucleus interpositus posterior.

Electrical stimulation of the brainstem was carried out in a second series of experiments using either glass micropipettes, pairs of enameled covered side-by-side tungsten electrodes, or enameled bipolar nichrome wire electrodes identical to those used by Borison and Wang (3). A variety of stimulus parameters and experimental procedures were used in an attempt to produce vomiting (for details see Miller and Wilson (7)). The locations of selected stimulating sites were marked with either Fast Green dye marks (10) or small electrolytic lesions and were later visualized on thionin-stained, frontal sections (100 μm) of the

Table 1. Vomiting and related activity produced by sinusoidal electrical polarization of the vestibular labyrinths, in cats with and without lesions of the posterior cerebellar vermis (data not listed sequentially). Symptoms observed indicated by +; symptoms occurred repeatedly in the majority of animals (++).

Cat	Posterior Cerebellum	Vomit	Retch	Salivate	Pant	Swallow	Lick
1	Intact				+		
2	Intact						
3	Intact						
4	Intact						
5	Removed			++	++	++	
6	Removed	+					
7	Removed					++	+
8	Removed	++					
9	Removed		++		++	++	
10	Removed	+					
11	Removed						
12	Removed						
13	Removed						
14	Removed						

brainstem.

To make it more likely that electrical stimulation would produce vomiting, the opiate antagonist naloxone (Endo Lab., 1.3-3.1 mg/kg i.v.) was administered to some animals. Naloxone was used because it has been shown to increase cats' susceptibility to motion sickness (4).

RESULTS

Electrical Stimulation of the Vestibular Labyrinths

Vomiting, as defined by the expulsion of gastric contents, was produced by vestibular stimulation in 3 out of 14 cats (Table 1). Times from stimulus onset to the first act of vomiting ranged from about 1 to 8 hours. Related activity including licking, swallowing, panting, salivation, and retching was observed in 4 other animals. Symptoms often appeared in a series of episodes; a maximum of 7 episodes of panting, swallowing, and retching occurred in one cat over a 5 1/2 hour period. It was not possible to predict the optimal stimulus condition or duration required for producing vomiting or related symptoms.

Unexpectedly, the 3 cats that vomited all had lesions of the nodulus and uvula. Other symptoms were also more prevalent in lesioned animals. The extent of the lesion in 3 cats is illustrated in Figure 1.

Electrical Stimulation of the Brainstem

A total of 296 tracks was made in 15 cats in grid-like patterns from 2 mm caudal to the obex to 6 mm rostral to it, encompassing the region of the "vomiting center" as described by Borison and Wang (3). Stimuli were applied at depth intervals of 0.5 mm or less in each track. Readily reproducible vomiting could not be produced by stimulating anywhere in this region. Emesis occurred during stimulation in only 3 cats (Table 2). The stimulation sites at which vomiting occurred were not restricted to a single anatomical structure; they were located in the solitary tract and reticular formation ventral to it (Figure 2). In contrast to these 3 instances in which vomiting occurred, numerous additional attempts to produce emesis by stimulating in the same region proved unsuccessful, both in cats that vomited and in other animals.

Emesis occurred reproducibly in one cat immediately after the stimulus was turned off; another animal vomited spontaneously while the current was off. Prodromal signs of vomiting were elicited in most experiments (Table 2).

Table 2. Effects produced by electrical stimulation of the brainstem (data not listed sequentially). Observations indicated by +. Cat 15 vomited spontaneously (indicated by X); cat 14 vomited only when the stimulus was turned off (X).

Cat	Vomit	Retch	Mouth Opening	Salivate	Swallow
1	+	+		+	+
2	+		+		+
3	+		+		+
4		+	+		+
5		+		+	+
6			+	+	+
7			+		+
8			+		+
9			+		+
10			+		+
11			+		+
12			+		+
13			+		
14	X		+		+
15	X				

DISCUSSION

While the vestibular system has long been recognized to be essential for the development of motion sickness (8, 9, 12), the role of some other structures implicated in motion sickness remains to be clarified. We have shown that a transcerebellar pathway involving the nodulus and uvula is not essential for vomiting and related activity that can be produced by electrical polarization of the vestibular labyrinths of decerebrate cats. By analogy, it seems likely that this pathway is also not required for the occurrence of many symptoms of motion sickness in intact animals. Rather, the nodulus and uvula either may form part of one of multiple pathways from the vestibular apparatus to brainstem structures involved in producing symptoms of motion sickness and/or may have an important modulating influence on these structures.

Furthermore, we were not able to obtain a restricted localization of a "vomiting center", despite our efforts to replicate the experimental conditions of Borison and Wang (3). If a well localized coordinating center for emesis did exist, we would expect that its activation by electrical stimulation would produce vomiting more reliably than we found. The absence of a readily identifiable "vomiting center" further complicates the task of determining how certain motion situations can lead to activation of the somatic and visceral effectors that produce vomiting and its related symptoms. In sum, our work has indicated that the neural mechanisms that produce motion sickness are not as well understood as has been assumed.

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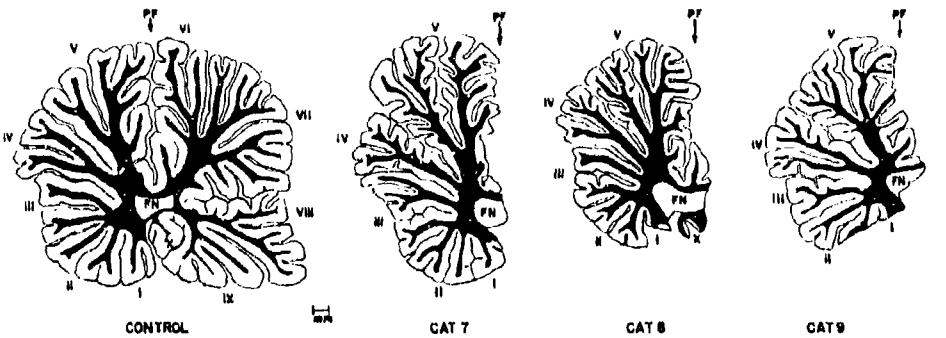

Figure 1. Cerebellar lesions from 3 cats illustrated on parasagittal sections near the midline. The cerebellum from a normal animal is shown on the left. Nomenclature follows Larsell (5). FN = fastigial nucleus, PF = primary fissure. Reproduced from Miller and Wilson (6) with the permission of the publisher, S. Karger AG, Basel.

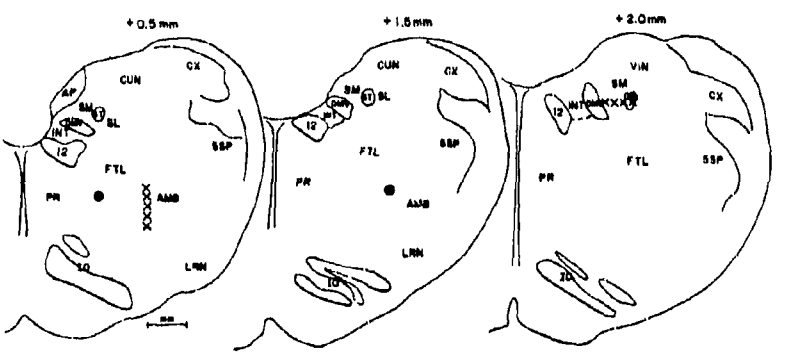

Figure 2. Vomiting only occurred 3 times during electrical stimulation of the brainstem, at sites shown by filled circles on frontal sections at indicated distances rostral to the obex. Retching was obtained in 2 other animals at sites marked with X. Abbreviations: AMB, n. ambiguus; AP, area postrema; CUN, cuneate n.; CX, external cuneate n.; DMV, dorsal motor nucleus of vagus; FTL, lateral tegmental field; INT, n. intercalatus; IO, inferior olive; LRN, lateral reticular n.; PR, paramedian reticular n.; SL, lateral solitary n.; SM, medial solitary n.; ST, solitary tract; VIN, descending vestibular n.; 5SP, spinal trigeminal n.; 12, hypoglossal n. Reproduced from Miller and Wilson (7) with the permission of the publisher, Elsevier Science Publishers B. V., Amsterdam.

DISCUSSION

GUEDRY: Please compare the stimuli used in your study and in the study of Wang and Chinn.

MILLER: Both Bard and his colleauges (1945, 1947) and Wang and Chinn (1956) tested their animals using swings, presumably without restraining the head of the animal.. Bard et al swung their animals using a frequency of 0.29 Hz and Wang and Chinn used 0.22 Hz. Their stimuli would have affected both the semicircular canals and otolith organs. In our preparation (Miller and Wilson, 1983a), stimulating electrodes were placed in the vestibular labyrinths after removing part of the cochlea. Polarizing currents were applied which had a waveform consisting of either single sinusoids (0.2-0.6 Hz) or a sum of 10 sinusoids (0.035-0.809 Hz) (Wilson et al 1979). Current amplitudes were slightly in excess of the threshold for eliciting reflexly-induced eye movements. Stimulation of this type causes sinusoidal modulation of the firing rate of primary vestibular afferents (1). In our preparation, afferents from both the semicircular canals and otolith organs were presumably modulated in a synchronous pattern that would not be experienced during natural vestibular stimulation. Such an unusual pattern of afferent activity may produce an intralabyrinthine sensory conflict leading to the development of symptoms of motion sickness. 1. Ezure K., Cohen A.S., Wilson V. J. Response of cat semicircular canal afferents to sinusoidal polarizing currents: Implications for input-output properties of second-order neurons. J. Neurophysiol. 49 (1983) 639-648.

CLAUSSEN: I congratulate you on your findings. The results you have shown are very important and are matching with our findings in many patients. We observe major cerebellar lesions and some circumscribed brain stem infarctions without affecting the motion sickness system. Arisal of motion sickness depends on dynamic interferences of different spatial information processing systems.

MILLER: Thank you for your interesting comments. We too believe that motion sickness is best explained by a sensory conflict theory.

CRAMPTON: Early motion sickness experiments on the cerebellum and area postrema were performed on dogs. Later evidence controverting early data are from cat. Is there a possibility that there are species differences?

MILLER: There is no a priori reason why the neural control system for motion sickness should be fundamentally different in cats and dogs. Perhaps in dogs the nodulus and uvula and area postrema have a more important tonic facilitatory influence on the mechanisms that produce motion sickness and/or perhaps these structures are part of a parallel mechanism that is more important in dogs than in cats. It is also possible that earlier investigators might have produced motion sickness in more of their animals if they had used more provocative stimuli or longer post lesion exposure periods. Bard (1945) comments that swings having a longer radius than the one he used produce at least double the incidence of vomiting in dogs. In spite of not having optimal stimulus conditions, Bard (1945) reported that out of 7 dogs with lesions of the nodulus and uvula, 2 vomited once during post lesion testing and 1 of these animals also regularly licked and salivated. In regard to the emetic responses of animals following lesions of the area postrema, Wang and Chinn (3) reported that 2 out of 12 dogs vomited, Brizzee and co-workers (2) found that 3 out of 8 squirrel monkeys vomited, while Borison and his colleagues (1) recently reported that 4 out of 5 cats vomited in comparison to 4 out of 10 intact cats. In addition, you can see from Dr. Claussen's remarks during the discussion period that he has obtained findings similar to ours with patients having major cerebellar lesions and circumscribed brainstem infarctions. I would also like to point out that our attempts to obtain a restricted anatomical localization of a so-called "vomiting center" were carried out in decerebrate cats, the same preparation used in Borison and Wang's original study (1949). 1. Borison H.L., McCarthy L.E., Borison R., Mandal A.K., Fisk T.J. Motion sickness is not prevented by chronic ablation of area postrema in cats. Fed. Proc. 43 (1984) 504. 2. Brizzee K.R., Ordy J.M., Mehler W.R. Effect of ablation of area postrema on frequency and latency of motion sickness-induced emesis in the squirrel monkey. Physiol. Behav. 24 (1980) 849-853. 3. Wang S.C., Chinn H.I. Experimental motion sickness in dogs. Functional importance of chemoceptive emetic trigger zone. Am. J. Physiol. 178 (1954) 111-116.

Influence of Gravitoinertial Force Level on Apparent Magnitude of Coriolis Cross-Coupled Angular Accelerations and Motion Sickness

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Summary

The Skylab astronauts showed a great decrease in susceptibility to motion sickness during exposure to Coriolis cross-coupled angular accelerations when tested in orbital flight. In fact, none of them reached a motion sickness endpoint inflight although each of them had preflight. We have been attempting to determine whether this decreased susceptibility is related entirely to adaptation or in part to changes in vestibular and sensory-motor function that occur virtually immediately in the microgravity conditions of orbital flight. To resolve this issue we have tested subjects separately in the free fall and high force phases of parabolic flight maneuvers and measured 1) susceptibility to motion sickness during Coriolis stimulation as a function of force level and 2) the perceived intensity of Coriolis cross-coupled angular accelerations as a function of force level. The findings are clear cut: subjects exhibit fewer and less severe symptoms of motion sickness when tested in free fall than they do for the same Coriolis stimulation in 1G; they exhibit much earlier and much more severe symptoms when tested in 2G. Ratings of the apparent intensity of Coriolis stimulation show the same pattern: subjects find that executing head movements in free fall at a particular velocity of rotation is much less stressful than in 1G; in 2G, the perceived intensity and associated discomfort are greatly increased. We conclude that part of the Skylab astronauts' inflight decrease in susceptibility to Coriolis stimulation was related to alterations in vestibular and sensory-motor control that occur immediately during exposure to microgravity force levels.

Introduction

We describe here how variations in gravitoinertial force level affect the experienced magnitude of Coriolis cross-coupled angular accelerations and the elicitation of symptoms of motion sickness. Cross-coupled stimulation of the semicircular canals occurs when a rotating individual makes head movements out of the plane of his rotation. The intensity of stimulation is dependent on the rotary velocity of the body, ω_1, the velocity of the head movement, ω_2, out of the plane of body rotation, and the angle, ϕ, between the ω_1 and ω_2 axes. Descriptions of the physical basis of Coriolis cross-coupling effects have been provided in particularly useful form by Guedry and Benson (1), Benson (2), Guedry (3) and Jones (4). Because of cross-coupling, a rotating individual who makes a head movement will experience aberrant motion of his head about an axis roughly orthogonal to ω_1 and ω_2. For example, an individual who tilts his head toward his right shoulder while being rotated counterclockwise at constant velocity will experience a forward pitching motion about the transverse plane of his head. It has long been known that Coriolis stimulation, when intense, will elicit dizziness, nausea and vomiting. The ability to withstand exposure to Coriolis cross-coupled angular acceleration has formed the basis for a test of motion sickness susceptibility that has been of value in predicting susceptibility during aerial maneuvers, the Coriolis Sickness Susceptibility Index Test or CSSI test (5).

The CSSI test was one of the procedures conducted as part of the Skylab M-131 experiment on vestibular function in weightlessness (6,7). Eight of the nine astronauts who participated in the three manned Skylab missions were evaluated with the CSSI test preflight, inflight, and postflight. The first inflight tests for the different astronauts took place between mission days 8 and 12. At the time of their first inflight evaluation and during subsequent inflight tests, all of the astronauts showed a marked decrease in susceptibility compared to their preflight scores. Even when the velocity of the rotating chair was increased beyond the ground-based test velocities to 30 rpm, all of the astronauts completed the maximum possible number of head movements in the test without reaching a motion sickness endpoint; in fact, all of them were virtually symptom free. The decreased susceptibility of the Skylab astronauts to Coriolis cross-coupled angular accelerations persisted into the postflight period; only over a period of days, and even in some cases weeks, did susceptibility on the CSSI test gradually return to preflight level (6,7).

The origin of the decreased susceptibility to cross-coupled angular accelerations inflight has significance for understanding the etiology of space motion sickness and for gaining insights into the nature of vestibular function in the altered gravitoinertial conditions of space flight. One question of immediate concern is whether the decreased inflight susceptibility resulted from some form of adaptation process, an adaptation which once achieved then persisted for some period postflight and gradually decayed, or whether it resulted at least in part from immediate changes in vestibular function related to the effective lifting of the G force in free fall.

In an experiment relevant to this issue, Miller and Graybiel (8) found that in the free fall phase of parabolic flight maneuvers some subjects show a decreased susceptibility to motion sickness during the CSSI test while others show an increase. Many individuals, however, are susceptible to motion sickness during parabolic flight maneuvers simply as a consequence of exposure to periodic variations in gravitoinertial force level, even when they are seated with their heads stationary in relation to the aircraft. It is not known whether the subjects tested by Miller and Graybiel (8) who showed increased sensitivity on the CSSI test are among those individuals who are susceptible to motion sickness during

exposure to the parabolic flight force variations independent of Coriolis stimulation, and whether those who showed a decreased susceptibility are insusceptible to the force variations alone.

To resolve this issue we have measured the basic susceptibility of subjects during parabolic flight maneuvers and then have determined how their susceptibility to motion sickness during exposure to cross-coupled angular accelerations relates to gravitoinertial force level. In other assessments, we have had subjects rate the apparent intensity of cross-coupling in situations involving comparable Coriolis stimulation but different gravitoinertial force levels.

Experiment 1

Susceptibility To Motion Sickness During Coriolis Stimulation As A Function of Gravitoinertial Force Level

Materials and Methods

Subjects: Eight individuals took part including one of the authors and seven college students who were paid for their voluntary participation. All had met the medical requirements and undergone the physiological training procedures necessary for taking part in parabolic flight experiments. Each had normal otolithic and canalicular function as measured by tests of ocular counterrolling, ataxia, modified Fitzgerald-Hallpike caloric irrigation, and thresholds for perception of the oculogyral illusion.

Subject Categorization Each subject was categorized in terms of his susceptibility to motion sickness in parabolic flight maneuvers. This was done as follows: in one of a subject's first two flights he was seated with his head restrained and his eyes covered, in the other flight his head was restrained but his eyes were open and he had full sight of the aircraft. Each of these flights lasted 40 parabolas. If a subject scored a total of between 0 and 4 motion sickness points in the two flights, he was assigned to Category I (insusceptible to motion sickness during exposure to periodic variations in gravitoinertial force level; 5-12 points, Category II (moderately susceptible); and 13 or more points in Category III (highly susceptible). The scoring system for assigning motion sickness points was developed by Graybiel, Wood, Miller and Cramer (9) and is presented in Table 1. Four of the eight subjects fell in Category I and four in Category III.

Table I

DIAGNOSTIC CATEGORIZATION OF DIFFERENT LEVELS OF SEVERITY OF ACUTE MOTION SICKNESS

Category	Pathognomonic 16 points	Major 8 points	Minor 4 points	Minimal 2 points	AQS* 1 point
Nausea syndrome	Vomiting or retching	Nausea+ II, III	Nausea I	Epigastric discomfort	Epigastric awareness
Skin		Pallor III	Pallor II	Pallor I	Flushing/Subjective warmth ≥ II
Cold sweating		III	II	I	
Increased salivation		III	II	I	
Drowsiness		III	II	I	
Pain					Headache ≥ II
Central nervous system					Dizziness Eyes closed ≥ II Eyes open III

Levels of Severity Identified by Total Points Scored

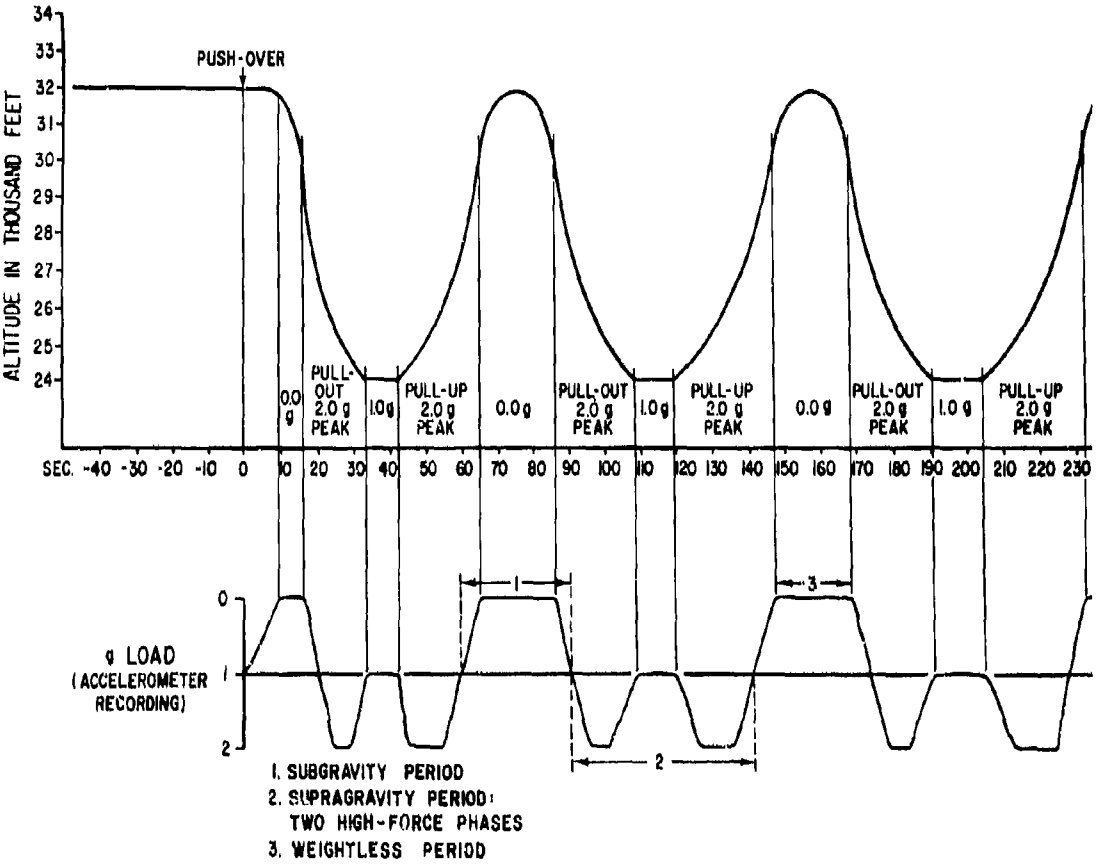
Frank Sickness	Severe Malaise	Moderate Malaise A	Moderate Malaise B	Slight Malaise
(S)	(M III)	(M IIA)	(M IIB)	(M I)
≥ 16 points	8 - 15 points	5 - 7 points	3 - 4 points	1 - 2 points

*AQS = Additional qualifying symptoms. + III = severe or marked, II = moderate, I = slight.

Apparatus: A servo-controlled Stille rotating chair was mounted in the mid-region of the fuselage of the Boeing KC-135 aircraft used in our experiments.

Parabolic Flight Profile: Figure I is a schematic illustration of the flight pattern of the KC-135 aircraft during parabolic maneuvers. The aircraft is flown in a parabolic path to generate alternating periods of increased gravitoinertial force, approximately 2G peak, and of free fall (0G). There are two high force periods in each parabola, and a free fall period lasting approximately 20 sec. In our experiments, the aircraft flies a total of 40 parabolas during each mission. The parabolas are flown consecutively except for turnarounds to gain additional airspace or breaks to assist motionsick subjects.

Figure I



Procedure: Each subject was tested under three conditions involving clockwise rotation at 20 rpm: 1) in the laboratory, 2) in the free fall phases of parabolic flight, and 3) in the high force phases of parabolic flight. In these conditions, the subject was required to make tilting head movements to a tape recorded 1 beat/s cadence. The subject was always maintained at constant velocity for at least 60s before head movements were initiated. The movements involved were a variation on the CSSI test procedure: the subject ventriflexed his head forward in pitch until it touched his chest and then dorsiflexed it until it touched a padded head rest: movement amplitude was 90°, one cycle of movement was completed in 2s for a movement frequency of .5Hz. Eight cycles of movement were carried out, then there was a rest period before the next set of movements, this procedure was repeated until either a motion sickness endpoint of severe nausea was reached or the subject had made 320 cycles of head movement. On the ground, 40s periods separated sets of 8 head movement cycles; in the parabolic flight tests, the minimum separation was 40-45s and the maximum separation was sometimes as long as several minutes or more in the case of a turn around. This maximum interval varied non-systematically across subjects and across test conditions. In parabolic flight, head movements in the microgravity test conditions were initiated in each parabola when a digital accelerometer indicated 0.0G' in the high force condition, when 1.8G had been attained. Ground-based laboratory testing always preceded the parabolic flight evaluations, the order of subject testing in parabolic flight was balanced across subject categories and force levels.

Results

All of the subjects showed dramatically greater susceptibility to motion sickness during Coriolis stimulation when they were tested in the high force phase of flight compared with their susceptibilities in free fall and in the laboratory. Moreover, all of the Category I subjects also showed a marked decrease in susceptibility in free fall compared with their laboratory results; two of the Category III subjects also showed a substantial decrease in free fall while two were more susceptible. Table 2 presents a summary of the data in terms of the total number of motion sickness points scored and the total number of head movement cycles achieved according to subject category and test condition.

It is notable that when tested in free fall 3 of the 4 Category I subjects completed the full 320 cycles of head movements without scoring any motion sickness points, the remaining Category I subject had some symptoms but completed the test. Only one of these subjects had completed the 320 cycles of head movements on the ground and none of them had been symptom free.

Table II

SUSCEPTIBILITY TO MOTION SICKNESS DURING EXPOSURE TO A CONSTANT LEVEL OF CORIOLIS CROSS-COUPLED ANGULAR ACCELERATION AS A FUNCTION OF GRAVITOINERTIAL FORCE LEVEL. THE AVERAGE NUMBER OF HEAD MOVEMENT CYCLES COMPLETED (320 = MAXIMUM ENTRY) AND THE AVERAGE NUMBER OF MOTION SICKNESS POINTS SCORED (16 = MAXIMUM ENTRY) DURING TESTING ARE INDICATED. THE EMESIS ENTRIES INDICATE THE TOTAL NUMBER OF SUBJECTS WHO VOMITED IN EACH CONDITION.

Subjects	Gravitoinertial Force Level	Head Movement Cycles	Motion Sickness Points	EMESIS
Category I	1 G	186	8	0
(N=4)	0 G	320	2	0
	2 G	77	10	1
Category III	1 G	122	10	1
(N=4)	0 G	141	8	0
	2 G	24	16	3

Experiment 2

Apparent Intensity Of Coriolis Stimulation As A Function Of Gravitoinertial Force Level

Materials and Methods

Subjects: Fifteen individuals took part including one of the authors. All had met the medical requirements necessary for parabolic flight experiments and were without sensory-motor anomalies.

Procedure: The same apparatus and aircraft were used as described above in Experiment 1. Prior to the onset of parabolic maneuvers, the subject was blindfolded and accelerated at $15°/s^2$ to a constant angular velocity of 120°/s, this velocity was maintained for the duration of the test. During straight-and-level flight, the subject was required to execute a total of three rapid tilting movements of the head: the subject tilted his head to his chest (movement time approximately 1s) kept it there for 10s and gradually returned it to the upright avoiding disturbance. This procedure was repeated twice more while the subject paid careful attention to the experienced magnitude of the Coriolis forces acting on his head during and after the forward pitch movement and the level of subjective discomfort associated with the movement. The subject was instructed to give each of these experiences the reference value 10 and to use smaller or larger numbers as appropriate to rate the levels of cross-coupling intensity and discomfort experienced during head movements in subsequent parabolic maneuvers.

During parabolic flight, the subject was required to make one cycle of head movement in the initial high force phase and one cycle in the free fall phase of each parabola. The subject tilted his head to his chest in approximately 1s kept it there for 10s and returned it gradually to the "vertical". The head movements made in high force levels were initiated when a digital accelerometer indicated at least 1.8G, the low force ones at 0.0G. After the completion of each test head movement the subject gave numerical magnitude estimates of the cross-coupling and the discomfort experienced. If there was a turn around period of straight-and-level flight during a subject's test parabolas, he was required to make an additional 1-g force level, head movement to help maintain his rating standard. The subject was tested until he either reached a motion sickness endpoint of nausea or had rated 30 parabolas.

Results

The experienced magnitude of a constant level of Coriolis cross-coupled angular stimulation was highly dependent for each subject on gravitoinertial force level. In free fall, relative to straight-and-level flight there was a significant decrease in ratings of apparent intensity, $p < .001$; by contrast, during exposure to high force levels there was a great increase in apparent intensity, $p < .001$. This pattern was characteristic of every subject and all of them also remarked on the great differences experienced.

The same pattern appeared in the ratings of apparent discomfort associated with head movements. The head movements in free fall were reported to be much less stressful than those in straight-and-level flight, and those made in 2G were rated as much more stressful than the 1G standard, $p < .001$ for both

comparisons. This pattern was characteristic of every subject.

Table 3 summarizes the experimental findings for apparent intensity and for relative stressfulness of cross-coupling as a function of gravitoinertial force level.

Table III

MAGNITUDE ESTIMATIONS OF SUBJECTIVE INTENSITY AND STRESSFULNESS OF CONSTANT LEVELS OF CORIOLIS CROSS-COUPLED ANGULAR ACCELERATION AS A FUNCTION OF GRAVITOINERTIAL FORCE LEVEL. THE 1 G TEST CONDITION SERVED AS THE STANDARD AND WAS ASSIGNED 10 AS A REFERENCE VALUE. STANDARD DEVIATIONS IN PARENTHESES. N = 15.

	GRAVITOINERTIAL FORCE LEVEL		
	1 G	0 G	2 G
APPARENT MAGNITUDE	10	2 (±1.3)	25 (±3.6)
APPARENT STRESSFULNESS	10	1 (±1.1)	28 (±4.2)

Discussion

The results of our two experiments show unequivocally that the apparent intensity and the relative provocativeness of constant levels of Coriolis stimulation are gravitoinertial force dependent. This finding provides an explanation, at least in part, for the decreased susceptibility of the Skylab astronauts when tested with the CSSI procedure inflight: the same patterns of Coriolis stimulation are less provocative in free fall than on the ground; accordingly, in the absence of other stressful vestibular stimulation, it may be expected that astronauts will be less susceptible to Coriolis stimulation after entry into weightlessness. In addition, however, the continued decreased susceptibility of the Skylab astronauts postflight suggests that some form of vestibulo-motor adaptation also took place inflight. We have described elsewhere how and why this adaptation may occur (10).

Over the past few years, there have been several indications that vestibular responsivity to angular acceleration is gravitoinertial force dependent. Lackner and Graybiel (11) found that the frequency and amplitude of nystagmus elicited in blindfolded subjects by constant levels of angular acceleration were diminished in free fall and enhanced during exposure to greater than 1G force levels. Bludworth, Reschke, and Homick (12), Vesterhauge, Manasson, Johansen, and Zilstorff (13), and de Jong, Oosterveld and Lavooy (14), have recently made similar observations. Together these findings suggest that the gain of the vestibulo-ocular reflex (VOR) diminishes in free fall.

The present findings of a decreased apparent intensity and a decreased provocativeness of Coriolis cross-coupled stimulation of the semicircular canals in free fall relative to terrestrial force levels are in accord with such a decrease in the VOR. The reason for the decrease is uncertain. It has been suggested that the semicircular canals may under some circumstances, such as Z-axis recumbent rotation, be sensitive to linear as well as angular accelerations (15,16). In addition, it is well established that otolithic input can modulate the activity of cells receiving afferents from the semicircular canals. (17,18,19,20,21,22,23,24). This latter possibility seems at present a more likely basis for the effects of gravitoinertial force level on responsivity to angular acceleration. In this context, it should be noted, too, that Igarashi (25) has shown that if the otolith organs are ablated, the intensity of pendular rotation nystagmus is diminished.

Several other factors may influence the apparent intensity of Coriolis cross-coupling accelerations in addition to variations in otolith organ activity related to gravitoinertial level. During exposure to force levels greater or lesser than Earth gravity, alterations also occur in many other aspects of sensory-motor control. These include, for example, changes in the intensity and distribution of touch and pressure stimulation of the body surface, alterations in proprioception, and changes in the levels and patterns of muscle activity associated with making particular body movements. In the last few years, there has been increasing evidence that all of these factors participate in a dynamic sensory-motor calibration of the body to terrestrial force levels. During exposure to non-terrestrial force levels, a variety of illusions occur during body movement, the character of these illusions reveals the existence of the sensory-motor calibrations that otherwise would not be recognized as such (26,27,28,29,30). It seems to us quite likely that the dependence of the apparent intensity of Coriolis stimulation on gravitoinertial force level will be related to these wide ranging functional changes in sensory-motor calibrations as well as to alterations in the central interpretation of patterns of semicircular canal activity.

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DISCUSSION

BLES: We know you can motivate the vestibular Corolis effect by adding congruent somatosensory stimulation in which case you would diminish the effect, or adding incongruent somatosensory stimulation which may result in an enhancement of the Coriolis effect. I wonder, if it is possible in your set-up to split out what the influence of the somatosensory information is and what the influence of the otolithic stimulation is?

LACKNER: The story with regard to the influence of the otolith organs in this situation is a very complicated one; in fact, I think many investigators would have predicted just the opposite patterns of results that we have obtained. We have good reason for believing that in addition to the changes in otolith activity there are other factors that change during gravitoinertial force variations in parabolic flight, and contribute to the patterns we have observed, e.g. the loading of the head on the neck changes and the patterns of muscle spindle feedback from the neck musculature are altered. We know, for example, that by vibrating neck muscles to create abnormal levels of spindle activity we can elicit illusory changes in head posture. The point is that we have a sensory motor control system dynamically calibrated to 1G and when we go into high force levels or free fall we are altering much more than just the vestibular receptor system. In fact, skeletal-muscular control and the proprioceptive and somatosensory systems are also being modulated systematically.

MILLER: I know that you've also reported changes in the gain of the vestibular ocular reflex at different force levels. Could you correlate that with the changes you see in susceptibility to cross-coupling?

LACKNER: The changes that we saw in cross-coupling would be in accord with the decreased gain of the VOR that we observed in free fall. From this standpoint the relative effectiveness of a constant pattern of angular acceleration would presumbly be diminished in free fall and augmented in 2G.

HAWKINS: I would like to ask about the influence of outside visual reference on the effect of Corolis stimulation and the visual confusion which may follow it. Fighter pilots in their combat manouvers frequently make large head movements under very high force levels. I recently saw a case of a pilot who made a fairly gentle pull-up but with a completely empty visual field and he couldn't see his instruments for the next 30 to 40 sec. presumbly due to nystagmus. He did not notice any problems when he had a good outside visual reference. Did your subjects who were making head movements while rotating have a clear visual reference or were they shut in a cabin and unable to see any outside horizon?

LACKNER: Our subjects were blindfolded. In the case to which you refer, one would expect with reduced visual reference and roll head movements to get a rotary nystagmus that would make it very difficult for your pilot to maintain clear view of the instruments, whereas with a full visual field, the nystagmus would be much less. Fred Guedry described an effect several years ago that he referred to as the G excess illusion which I think is related to what you are describing. Essentially in the G excess illusion the pilot is banking his aircraft and there isn't much angular acceleration involved, so there is very little cross-coupling during head movements, but there is a greater than normal G force and this would alter the gain of the vestibulo-ocular reflex, producing rotary nystagmus and apparent deflection of the instrument panels during roll head movements.

GUEDRY: The cross-coupled vector lies in the plane of rotation. Did the head movements of your subjects in parabolic flight involve trunk movement so that a centripetal vector was introduced? The centripetal vector would be aligned with the cross-coupled vector.

LACKNER: We have done the cross-coupling studies both with simple head tilts in pitch, approximately 90° amplitude, and with full head and torso pitch forward. The results are very similar for the two test situations.

JONES: Why is cross-coupled stimulation so rare and so unpredictable in operational high-performance jet flight, given the high-G environment and the frequent head motions of the aircrew?

LACKNER: I think Fred Guedry knows more about this issue than anyone else.

GUEDRY: Most maneuvers in aircraft do not involve sustained high angular velocity spins or turns which are required to induce strong cross-coupled illusory effects from head movements. However, head movements made in a high-G field, 2G and above, can produce disturbing illusory effects often referred to as "G excess" effects, possibly due to excessive feedback from the otolith system in high-G fields. This was shown in several studies in high-speed aircraft making level high-G turns at turn rates so low that cross coupled stimulation of the semicircular canals would be negligible, yet illusory and nauseogenic effects were produced. Experienced pilots and aircrew undoubtedly learn "the feel of maneuvers" and their anticipation of effects from head movements in high-G fields serve to reduce effects. Pilots also sometimes intellectually override such effects, e.g., an experienced pilot reported a 20-30° nose down attitude as a result of a head movement in a 2G field, but said that he was not disoriented because he knew the true condition of the aircraft, which was in a level bank and turn.

VESTIBULAR AND OCULOMOTOR FUNCTION DURING Gz VARIATIONS

by

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SUMMARY

10 normal subjects were exposed to G-force variations during parabolic flights and turns in a SAAB Supporter aircraft. A vertical head drift accompanied by a vertical eye drift was recorded in all subjects. The eye drift was most prominent during the hyper-G phase of the parabolic manoeuvres. Compensatory eye movements were induced by horizontal head rotations. No statistical significant changes could be demonstrated in this reflex. Horizontal oculomotor saccades were induced with a visual distance of ± 10°. A significant increase of the latency time could be demonstrated during the weightless phase of the parabolas. It is concluded that spontaneous eye and head drift and disturbances in voluntary eye movements might contribute to the development of motion sickness during combat manoeuvres and space flight.

INTRODUCTION

During aviation, spatial disorientation might appear whenever a linear acceleration of the aircraft interferes with the perception of the gravitational force. Unexpected and contradictory sensory cues might cause motion sickness. During space missions, the frequent appearance of space motion sickness not only affects the crew member's comfort but interferes with their productivity and the safety of the missions. The free fall phase of parabolic flights is an important tool for the study of immediate physiological reactions to weightlessness. Lackner & Graybiel (1) were the first to report on alteration of the gain of compensatory eye movements elicited by passive rotation in yaw during parabolic flights. Later, our group (2) was able to demonstrate similar alterations in the gain of compensatory eye movements elicited by voluntary horizontal head rotations. In both studies, the gain decreased during hypogravity and increased during hypergravity. Bludworth et al. (cited in 3) found that the gain of the vestibulo-ocular reflex decreased both in hypo- and hypergravity. The aim of this study was to observe whether opening of the eyes in darkness affected the gain variations caused by Gz variations. Further, we wished to study a phenomenon reported by von Baumgarten et al. (4). During rollercoaster flight vertical nystagmus was observed. A vertical eye drift might interfere with horizontal eye movements and by that influence the results of studies of horizontal eye movement phenomena. By itself, a vertical eye drift might contribute to sensory conflicts and cause spatial disorientation and motion sickness. Vertical eye drift might be elicited by a head drift in the opposite direction and serve as a compensatory measure to the head drift. Because of that, we decided to do simultaneous recordings of head and eye drift in pitch.

In most studies dealing with eye movements in yaw and pitch, electro-oculography is the method used for eye movement recording. This technique is based on the existence of the corneofundal electrical potential. Variations in the intensity of light changes this potential and makes it mandatory to perform calibrations in immediate relation to the experiments. Calibration is performed by fast saccadic eye movements between light-emitting diodes. A disturbance of this voluntary eye movement reflex might contribute to disorientation during variations in the Gz forces. Our experimental setup made it easy to evaluate this reflex in the same procedure. For these reasons it became a specific part of the present parabolic flight study.

METHODS

Ten subjects with normal vestibular pretest were selected for the experiments. None of them were professional pilots, but all subjects had some experience as passengers in small aircrafts. This qualification was prefered to avoid anxiety reactions during the flights. A SAAB Supporter aircraft was supplied by the Royal Danish Air Force. It is designated T-17, in daily service it is used as a training and reconnaisance aircraft. It is a small, two seated propeller driven aircraft well fit for aerobatic manoeuvres. In each mission three series of consecutive parabolas were interrupted by one minute 60° turns with a constant G-load of two G. Convenient pauses with straight and level flight were interpolated between stressfull manoeuvres according to the subjects' wishes.

Horizontal and vertical eye movements were recorded simultaneously by means of superficial skin electrodes. DC-amplification was performed with a time constant of ten seconds. Head movements in yaw and pitch were recorded by a angular velocity sensitive device (Ratemeter) mounted in a firm head holder. G-load was recorded by a linear accelerometer. An instrument tape recorder carried by the aircraft recorded the signals. The subjects were adapted to darkness by means of red glasses before and during the flights.

During the first sequence of five parabolas and one minute of two G load, the subjects were instructed to keep their eyes open behind a cover and to keep their heads still. During the next sequence, the subjects performed horizontal head rotations guided by an 0.4 Hz frequency modulated sound signal presented to a set of ear phones from a

tape recorder. During the third and last sequence, the cover was removed from the eyes and the subjects fixed alternately activated red-light-emitting diodes. The duration of each stimulus was randomized. The visual distance between the two diodes was ± 10° horizontally. Eye movement calibration was performed by means of the same equipment.

After return to the laboratory, data were analysed off-line from the tape recordings. Vertical eye movements appeared in the form of vertical nystagmus. Fast components were identified by a computer program and removed from the signal. Slow components were connected with each other by extrapolation. Compensatory eye movements were analysed according to our laboratory procedure described elsewhere (5). Results appeared as gains and phaseshifts of the transfer function between head movement input and oculomotor output. Latency times of saccades were measured and the peak velocity of the saccade computed from a digital differentiation of the eye signal. Vertical head position data were computed by a digital integration of the head velocity signal from the y-axis sensor.

The duration of all separate parabolas were almost exactly 10 sec.

RESULTS

Vertical eye and head drift:

Fig. 1 demonstrates the mean eye and head movement data of all ten subjects as an average of all parabolas flown. The eye position data describe the eye drift in the direction of the slow phase of nystagmus, whenever nystagmus was present. This explains the offset between eye position at time zero and eye position at 20 sec. The averaging was triggered by the sudden transition from weightlessness to high G-load, which appeared by pull-out from the parabola.

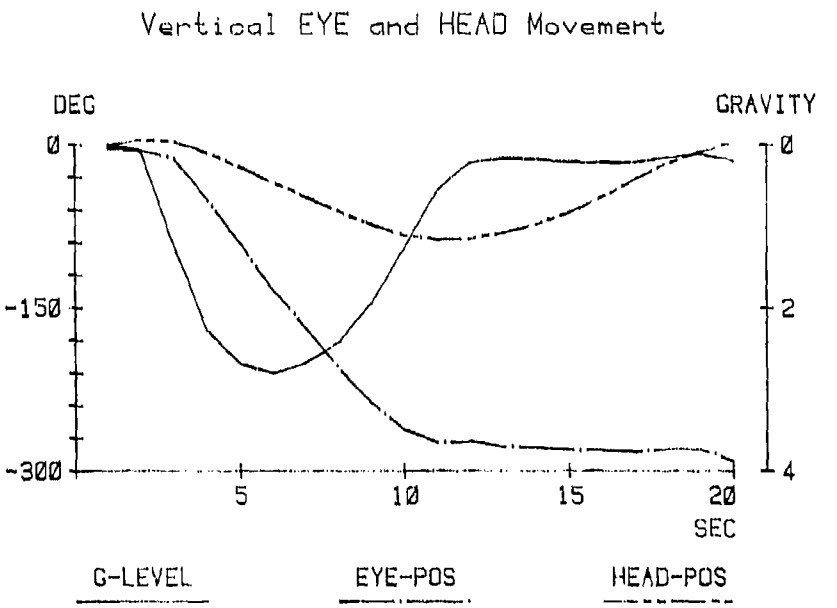

Figure 1. Average eye drift in the direction of the slow nystagmic phase and head position from 10 subjects, 5 parabolas each. Averaging is triggered by the transition from weightlessness to high G-load during pull-out.

All subjects exhibited vertical upward beating (direction of the fast component) nystagmus during high G-load. The nystagmus appeared within the first 1-2 seconds of pull-out from the parabola and disappeared with transition to weightlessness in the next parabola. Careful examination of the original recordings revealed very weak downward beating nystagmus during weightlessness in three of the ten subjects. Slow phase velocity being 2-3 °/sec, the nystagmus was too weak to be recognized as such by our computer analysis.

Head movements were smooth, directed downwards during hypergravity and upwards during hypogravity. This configuration suggests that there is a simple connection between the variations of the weight of the head and the movements. The head reaches it's maximal speed in the downward direction 2.5 sec after the maximal G-load.

Compensatory eye movements:

Results of the compensatory eye movement test appear in tab. 1.

It's obvious that there is no difference at all between gains at 0 G and gains at 1 G. The gain at 2 G is lower than the two other gains computed, though no statistical significant difference can be demonstrated, probability level being above o.o5. Intraindividual differences of phaseshifts are high and no statistical significant G-dependance can be extracted from our results. Spectral purity of the responses is lower in this experiments than those obtained under laboratory conditions. This explains the high variability.

	0 G		1 G		2 G	
	GAIN	PHASE	GAIN	PHASE	GAIN	PHASE
$\bar{X}$	0.83	-21.8°	0.84	-21.4°	0.65	-20.2°
SD	0.20	28.2°	0.23	24.0°	0.25	31.1°
N	10	10	10	10	10	10

Table 1. Gain and phaseshift of compensatory eye movements induced by 0.4 Hz head rotations in yaw at different G-loads. No statistical significant differences can be demonstrated.

Saccadic eye movements:

Results of saccadic eye movement tests appear in tab. 2.

	0 G		1 G		2 G	
	LAT. TIME	PEAK VEL.	LAT. TIME	PEAK VEL.	LAT. TIME	PEAK VEL.
$\bar{X}$	*261 msec*	*374 °/sec*	*227 msec*	*356 °/sec*	237 msec	347 °/sec
SD	*21.5 msec*	*27.0°/sec*	*22.8 msec*	*21.6°/sec*	*28.6 msec*	*33.1°/sec*
N	9	9	9	9	9	9

Table 2. Latency time and peak velocities of horizontal randomized saccades with an amplitude of ± 10° at different G-loads. Italicized figures are statistical significantly different at a probability level below 0.05.

It appeared from tab. 2., that only nine subjects contributed to the results. One of the subject followed his own rythm during flight tests and was omitted from the material. Latency times at 0 G are significantly longer than latency times at higher G. A tendency to higher peak velocities at weightlessness is not statistically significant.

DISCUSSION

Vertical head drift was measured in relation to the earth vertical, we did not do any efforts to subtract the flight profile in order to achieve a measurement of movement in pitch in relation to the aircraft. Flight profile is almost rectilinear, except at maximal and minimal altitude. If the ratemeter recording only described the flight profile, a distinct minimum and maximum would be expected at 5 and 15 sec respectively. The vertical semicircular canals responds to angular accelerations relative to earth vertical. From a recording of rotational rate relative to the aircraft, it would be difficult to predict canalicular vestibular responses. The velocity of the head in pitch is almost exactly in phase with the G-load. This allows us to conclude, that the head movement is a simple consequence of the variations of the weight of the head. The position of the subject in the seat with the head bended a little forward explains the direction of the movements.

Movements of the head in pitch will induce compensatory eye movements in the same axis. However, the eye movements recorded are not compensatory to the head movements and are consequently not caused by the head movements. von Baumgarten et al. (4) conclude, that vertical nystagmus during gravity changes is caused by a central misinterpretation of vestibular information as being caused by involuntary forward or backward tilts. In both cases the utricular receptors would signal a change in the direction of the gravity load. A signal reporting a change of the size of the gravity vector must be substantial different from that. Compensatory eye movements during free fall or during +Gz acceleration should have an upward and downward direction respectively. For that reason, we conclude, that the nystagmus recorded is a relevant central interpretation of a vestibular signal caused by variations of the Gz-load on the utricular receptors. We are unable to explain the non-linearity of the response, the hypergravity response being much stronger that the hypogravity response. In a recent paper, our group has shown that the gain of the compensatory eye movement response to head rotations in yaw varies proportionally to the G-load (2). As discussed below, we were not able to confirm this observation in the present study. Nevertheless, the behavior of the vertical eye drift might be caused by the effects of varying gravity load.

The results of the compensatory eye movement study are difficult to explain in view of the findings from a similar study performed one year before the present study (2). In our first study, subjects performed head rotations with their eyes closed, in the present experiment, eyes were open but covered. All other variables were kept constant. Even the pilot was the same in the two experiments. Four subjects participated in both studies and their results perfectly reflect the different conclusions of the two experiments. Absolute gain values were on average 19% higher in the present study. This difference is statistically significant. In the first study (2) a significant G-dependence of the gain was demonstrated as mentioned above. Gains were 8.5% lower at 0 G and 17% higher at 2 G. The study confirmed the findings of Lackner & Graybiel concerning the oculomotor response to passively induced head rotations. In a recent article Lackner & Graybiel (3) quote Bludworth et al. in a yet unpublished work for having found gain to be decreased during both free fall and at two G force levels. Our experimental design forces us to conclude, that the quantitative and qualitative differences between the results of our two experiments are caused by the difference in the state of vision in the two experiments, closed eyes in the first and open eyes behind covers in the present.

The disturbance in saccadic eye movements demonstrated is statistically significant. It's doubtful whether it can be considered of any significance in aviation or space missions. Somebody might claim that an increased reaction time could be disastrous in high performance fighter combats, but usually negative G-forces are avoided under these circumstances. In the neurological clinical practice, disturbances in saccadic function are interpreted as

a sign of brain stem lesion. Indeed, no such lesion was present in the subjects. Our knowledge of brain stem circulation is rather insufficient. Nevertheless it seems probable that the minor impairment of saccadic function seen in this study could be caused by an impairment of brain stem circulation due to the redistribution of blood volume during weightlessness. Further experiments are needed to shed light on this phenomenon.

It was obseved that fixation during saccade tests stabilized eyes in a way that no vertical eye drift could be seen. Head drift disappeared as well.

All together, vertical eye drift, vertical head drift, changes in the gain of compensatory eye movements and disturbances in voluntary saccadic eye movements might cause major disturbances in visual function under flight conditions with shrinking and reexpansion of the gravitational vector. The disappearance of eye drift during visual fixation emphasizes the importance of the state of visual function and no other conclusion concerning oculomotor function during changes in G-vector size can be drawn, than lots of factors might influence function in a way that makes visual fixation ability the crucial factor.

CONCLUSIONS

Following conclusions are drawn from this work:

I: Hypergravity induces a spontaneous downwards directed eye and head drift.
II: Hypogravity induces a spontaneous head drift upwards. An eye drift in the same direction is less pronounced and only present in some individuals.
III: The above mentioned phenomena disappeared with visual fixation.
IV: Eye opening behind a cover increased the gain of compensatory eye movements and made the response less sensitive to gravitational changes compared to results obtained with eyes closed.
V: Latency time of saccadic eye movements is prolonged during short periods of weightlessness.

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DISCUSSION

VON GIERKE: In your paper as well as in the previous paper by Professor Lackner I wonder if not the dynamics of the G-time history has to be taken into account. It appears as if in the parabolic zero G flights the oscillations i.e. the period between zero G and maximum G loading is in the order of 10 to 100 seconds. That is, according to steady state laboratory experiments, the frequency range of maximum vestibular response and motion sickness sensitivity. A statement of increased amplification at 2G should probably be qualified as occurring at 2G peak values at the particular oscillation frequency. Do you agree that the dynamics of the G exposure must be stated, analyzed and taken into account? That the frequency might be just as important as the G amplitude?

VESTERHAUGE: Yes, I agree with you. It should be stated that the experiments were performed at a relatively high G-load frequency (about 0.05Hz) compared to other experiments with longer duration of weightlessness.

VON BAUMGARTEN: I find it very important to look at the first push over and the first pull-out because if you do roller-coaster flight you come into a pattern of your subjects anticipating the next move of the aircraft; especially in the small aircraft, the parabolas are 5 or 10 sec. I see from your diagrams that you did the same thing. You said that head movements were caused by simple mechanical forces on the head. That's a possibility. We have not seen these vertical head movements for the reason that we worked with restrained heads in our studies. I would have explained them as a vestibular reflex. We know there is head nystagmus of some patients in the same direction as the eyes flick and if you put someone on the Barany chair and accelerate him he moves his chin against the direction of rotation; and if you rotate him about the Y axis, I would also expect a head movement.

VESTERHAUGE: I'm happy you say that because we believe it might be a vestibular reflex as well. But it's very difficult to prove that it's not just a consequence of the weight of the head changing with acceleration variations.

VON GIERKE: I have no question, just a comment to the last discussion. In 2G, you know that the spinal column is compressed, you must expect head motion between 0G and 2G of more then an inch. That's just spinal dynamics.

RESCHKE: We have also done some very similar things during parabolic flight. Did you notice a lot of variation in the individual subjects in terms of the gain and phase in the eye movements both in vertical and horizontal? For example,were there different patterns? Overall, generally what we found with the horizontal canal stimulation was that during 2G and 0G there was a decrease in the gain and phase. However, with the vertical canals we found an increased gain in 2G and a decreased gain in 0G relative to 1G but this was a general pattern. None of it was statistically significant and every subject seemed to have their own type of pattern although you could begin to group them. I was wondering if you perhaps find the same thing?

VESTERHAUGE: I would agree with you that there is quite a lot of variation in the data especially in the last experiment where I reported about compensatory eye movement. We had quite a lot of variation but the variation was much less with eyes closed than with eyes covered. We had the same experience in the laboratory that these experiments are better done with the eyes closed because response variation is less than with the eyes open in darkness. I don't know why.

PREDICTION OF THE SPACE ADAPTATION SYNDROME

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INTRODUCTION

Results of Skylab (1,2) confirmed that approximately 25-50 percent of individuals exposed to microgravity develop some degree of space motion sickness during the first 36 hours of space flight. Currently the incident of sickness during the Shuttle flights has exceeded this level (3). Realizing the potential operational impact that this difficulty in adapting to microgravity might have on continued Shuttle missions, NASA has mounted a considerable effort to treat, predict, and explain this space adaptation syndrome. Among these efforts was the development of facilities at the Johnson Space Center to produce motion sickness symptoms from a variety of provocative tests. One of the results of this effort has been the development of a normative data base where a number of individuals have been exposed to each test, making it possible to look at the interrelations of these various tests, as well as, to begin to evaluate their usefulness in prediction.

Prediction, either for practical purposes or heuristic motivation, has been an objective in the research of motion sickness. A variable approach, ranging from the use of questionnaires (4-11), psychodynamics or personality traits (11, 12-22), vestibular function tests (11, 23-39), physiological correlates (11, 40-62) and tests in specific nauseagenic environments (11, 63-72) has been directed toward the question of who will experience motion sickness, under what conditions and to what degree. More often than not, the correlations obtained between motion sickness and the selected predictors have been statistically significant, but low in magnitude and of no practical purpose in establishing predictive susceptibility.

The Neurophysiology Laboratory at the Johnson Space Center is currently tasked with identifying a means of predicting what has been called either space motion sickness or the space adaptation syndrome for the purpose of applying possible countermeasures. Historically, efforts to predict space sickness have not been encouraging, and have been hampered frequently by limited access to the astronaut population and the small number of crew involved in spaceflight. Individual variations in preflight experiences, medications, inflight tasks (i.e., mobility) and personal strategies for symptom management have further compounded our efforts.

Realizing that no single test or battery of tests will yield a prediction index of unity, we have opted to assign a probability value to motion sickness generated in variable accelerative environments. To accomplish this objective, we have accessed the large non-astronaut motion sickness data base mentioned above. The objectives of this study are: 1) To describe the univariate and multivariate relationships of the current battery of provocative and non-provocative measures used by our laboratory; 2) To develop and cross-validate sets of linear equations that optimally predict motion sickness using predetermined sets of tests; and 3) To determine the inherent properties of the various tests in a multivariate setting so that redundant and/or ineffective tests could be eliminated.

METHOD

Tests for motion sickness susceptibility and vestibular function used in this study included: 1) the Coriolis Sickness Sensitivity Index; 2) an off-vertical rotation test; 3) a sudden-stop test with an optokinetic stimulus; 4) a sudden-stop test without an optokinetic stimulus; 5) a staircase velocity test similar to the Coriolis Sickness Sensitivity Index; 6) motion sickness susceptibility during parabolic flight; 7) tests of Vestibular Ocular Reflex phase and gain; and 8) Postural ataxia measurement. In addition to these tests, the subjects were administered two questionnaires. The first questionnaire was designed to obtain a motion experience and symptom history, while the second was an immediate pretest questionnaire to assess health and drugs used during the 24 hours prior to testing. Age and gender (and for the female population, menstrual cycle) were also obtained with this questionnaire. Two multivariate statistical methods were used to meet the analytical objectives of this study . Firstly, a factor analysis was completed to describe the interrelations of the variables between the different tests, and to reduce the dimensionality of the variables. Secondly, Multiple Discriminant Analysis was used to develop and cross-validate optimal weights for each variable.

Subjects

All subjects in the normative data base had passed an Air Force Class III medical examination, and where required for parabolic flight, had completed a course in physiological training for high altitude survival. In addition, prior to any test or set of tests, all necessary documentation required by the NASA-Johnson Space Center Human Research Policy and Procedures Committee were submitted and approved.

In this study, a total of 159 subjects, 121 male and 38 female, were drawn from the normative data base. Their ages ranged from 19 to 58 years. Criteria for selection from the normative population was based on exposure to parabolic flight and completion of substantially all of the tests currently used in the laboratory. With the exception of parabolic flight, if test results were missing for any subject, mean scores were inserted for those individuals. This data treatment was used with less than 2% of the total study population, and in no case did any one subject have more than one set of scores missing.

Tests

Coriolis Sickness Sensitivity Index (CSSI Test). The CSSI test assessed an individual's susceptibility to cross coupled angular acceleration and conflicting sensory input primarily from the semicircular canals and statotoliths using modified procedures described by Miller and Graybiel (73, 74). The test was repeated at different velocities as required for each subject. The repeat tests were used to determine a stable endpoint at the MIII level of malaise (8 or more symptom points). The test was implemented using a 100 lb/ft torque rotator-chair assembly. With the subject blindfolded and seated in the upright position, the chair was accelerated at 6°/sec^2 to a terminal velocity (up to 30 RPM) predetermined by the results of the Motion Sickness Experience Questionnaire. While rotating, the subject executed standardized head movements in each of the four cardinal directions (Sequence: front, right, back, left). Head movements were performed in sets with each set consisting of five head movements (the front movement performed twice). Each set was separated by a 20 second period of no head motions. Subjects were questioned during these 20 second periods for signs and symptoms indicative of early motion sickness. The test was terminated when either the MIII level of malaise was achieved or 150 head movements (30 sets) were completed.

Prior to deacceleration of the chair, the subject was instructed to report any sensation of reversal in the rotational direction and to mark the moment he felt that he had come to a complete stop. The chair was deaccelerated at 6°/sec^2 until it reached 0 RPM. When the subject reported the onset of the reversal sensation, the test operator began timing with a stop watch. Timing was stopped when the subject reported no sensation of movement. This time was referred to as the reversal sensation time and was recorded in units of seconds.

Off-Vertical Rotation (OVR Test). A modified version of the off-vertical rotation test which provides a rotating linear acceleration for otolith stimulation, initially developed by Graybiel and Miller (28, 75, 76), was administered using the 100 lb/ft torque rotator-chair assembly employed for the CSSI test. Subjects were blindfolded with dark, light occluding goggles that permitted the eyes to remain open, and restrained in the chair with lap, shoulder and leg straps. The head was restrained with pads at the base of the skull and a strap around the forehead. With the subject in the upright position, the chair was accelerated at 6°/sec^2 to a terminal velocity of 20 RPM and rotated at 0° tilt for five min. Following stabilization at 0°, the angle of tilt was increased in 5° increments at 5 min intervals. During rotation, the subject was questioned for symptoms indicative of early motion sickness. The test was terminated when the subject reached the MIII level of malaise or the chair had been maintained at 30° tilt for five min.

To terminate the test, the chair assembly was returned to 0° tilt, and maintained at 0° tilt for approximately 1-5 min to allow subject stabilization. The chair was then deaccelerated at 6°/sec^2 to 0 RPM.

Sudden-Stop Test (SST). The sudden-stop procedure (eyes open), a vestibulo-visual test, was administered using a modified version of the sudden-stop test developed by Lackner and Graybiel (64). The test was implemented with the same chair, rotator and restraint system employed for the OVR and CSSI tests. Visual stimulation was provided with an optokinetic field (dark blue cloth drum) which surrounded the chair. Vertical white stripes at 60° intervals on the drum ran from floor to ceiling. The white stripes substended a visual angle of 1.74°. With the subject in the upright position, the chair was accelerated at 20°/sec^2 to a terminal velocity of 50 RPM and maintained at this velocity for 30 sec. At the end of 30 sec, the chair was deaccelerated at 150°/sec^2 to a complete stop and maintained at zero velocity for 30 sec. This profile, representing one trial, was presented to the subject eyes open a maximum of 40 times. The direction of rotation remained constant for 20 trials and was reversed for the subsequent set of 20 trials. After each trial, the subject was questioned for symptoms indicative of motion sickness. The test was terminated when the MIII level of malaise was achieved or 40 trials were completed.

The sudden-stop test (eyes closed) was conducted following the same procedures defined above with the exception of the visual stimulation. In the eyes closed test, the subject was blindfolded with dark, light occluding goggles.

Staircase Velocity Motion Test (SVMT). The Staircase Velocity Motion Test (SVMT) was used to assess each an individual's susceptibility to motion sickness with a modified Coriolis Sickness Susceptibility test procedure in which the cross-coupled angular acceleration experienced progressed from low level to maximum stress stimulation.

Rather than a single fixed constant velocity, a staircase velocity profile was used. The test was performed with the 100 ft/lb torque rotator-chair assembly. Before the rotator was started, the subject was restrained in the chair with lap belts and foot straps. The rotator was then accelerated at 6°/sec^2 to an initial velocity of 1 RPM.

With eyes blindfolded, the subject executed standardized head movements in each of the four cardinal directions. These head movements were performed in sets, with each set consisting of five head movements (front, right, back, left, front). Each set was separated by a 20 sec period of no head motions. The rotator velocity was then increased in 2 RPM steps, with 40 head movements being performed at each velocity step, until the Malaise III endpoint or a terminal velocity of 35 RPM was reached. The number of head movements, RPM level and symptoms when the Malaise III endpoint was reached were recorded, and the rotating chair was deaccelerated at 6°/sec^2 to a complete stop.

VOR. The VOR test was designed to obtain ocular nystagmus gain and phase elicited via sinusoidal angular acceleration from the horizontal semicircular canals and associated CNS structures. Five disposable pregelled infant electrodes were used to record the nystagmus. Prior to electrode placement, the skin was cleansed with an isopropyl alcohol wipe. Electrodes were placed at the outer canthus of each eye, on the center of the forehead (ground), centered above the right eye, and centered below the

left eye. Impedance measurements were recorded using the ground electrode as reference, and electrodes with greater than 10k ohms impedance were replaced. The subject doned red filtered goggles following the electroding procedure for a 20 minute dark adaptation period. Upon completion of dark adaptation, the subject was seated on a 30 lb/ft torque rotator-chair assembly. For calibration purposes the subject was instructed to visually track 2 alternately flashing LED's located 5 feet from the subject's forehead. These lights substended a visual angle of 10°. When the eye calibration measurement was completed, the red goggles were replaced with dark, light occluding goggles. All measurements were obtained with the eyes opened beneath the light occluding goggles. The subject's head was ventroflexed 30° and held in place with a biteboard attached to the chair.

White noise was provided through headphones, placed over the subject's ears to eliminate auditory cues in the test room. The rotator was computer driven at five separate frequencies one octave apart, ranging from 0.01 to 0.16 Hz. Peak velocity was 60°/sec at all frequencies with acceleration ranging from 3.8°/sec^2 at 0.01 Hz to 60.8°/sec^2 at 0.16 Hz. The subject was given mental task exercises to enhance alertness during nystagmus recording. Upon completion of all test conditions, the light occluding goggles were replaced with the red goggles and a post test eye calibration was performed using pretest procedures.

KC-135 Static Chair Test. The KC-135 static chair test assessed an individual's susceptibility to motion sickness during parabolic flight. The KC-135 aircraft was flown through a series of parabolic maneuvers (typically 40 parabolas per flight) with each parabola comprised of 24 sec of weightlessness and 30-60 sec of a 2-g pullup.

Subjects were secured in a passenger seat aboard the aircraft using the available seatbelts. The subject's head was immobilized using a soft neck brace. Dark, light occluding goggles were positioned over the subjects eyes to eliminate visual cues. Symptoms of motion sickness were reported after each parabola to the test operator. A symptom tally or score sheet was maintained for the duration of the flight. Due to the nature of this test, subjects were not removed from the provocative stimulus regardless of symptom level.

Postural Equilibrium. Postural equilibrium was tested by a modified and shortened version of a standard laboratory method developed by Graybiel and Fregly (77). Metal test rails of four widths, 1.90, 3.17, 4.45, and 5.72 cm (0.75, 1.25, 1.75, and 2.25 in) provided the foot support for the subject during this tests. Each subject was fitted with military type shoes to standardize footwear.

Time, the measurement of balance, began when the subject, while standing on the prescribed support with his feet in a tandem heel-to-toe arrangement, folded his arms across his chest. The eyes remained open in the first test series. In the second series, the time measurement was initiated after the subject attained a balanced position, folded his arms and closed his eyes.

During the test session the initial rail width for testing with eyes open was typically 3.17 cm (1.25 in). Three test trials with a maximum required duration of 60 sec each were given. If a cummulative 100 sec score was reached on the first two trials, a third was not performed. A perfect score was 100 sec. If the subject failed to obtain a perfect score, the two largest time values for the three trials was summed to obtain the final score. The choice of the second rail width depended upon the subject's score on the initial support width. If the score was greater than, or equal to 80 seconds, the next smaller support width was used; if this score was less than 80 seconds, the next larger support width was used. Testing on a third rail size was required when both of the two previous support width scores fell either above or below the 80 seconds performance level. Testing with eyes closed followed the same procedure except that a larger rail support, 5.72 cm (2.25 in) was typically used first. Eyes closed tests always followed testing with eyes open. All tests were conducted with normal laboratory illumination, and the subject facing a blank, white wall.

Variables

From the tests and questionnaires described above, 27 variables were derived and used in the data analysis. The 27 variables were further broken down into 24 test variables, and three criterion variables. Of the 24 test variables, 6 variables were obtained from the judgment of three raters who assigned individual subjects into categories of motion sickness. The test variables were used to predict the criterion variables.

Last Variables

The 24 test variables included the subjects age and gender, as well as, three variabes each from the CSSI test, SSTEO, SSTEC and SVMT. Two variables each were derived from the OVR and RAIL tests, and four from the VOR. For the CSSI test the three variables included: 1)CSSC, the subjects' MIII score; 2) CSR(n), the susceptibility level assigned (i.e., severe, moderate low) by one of the three individual raters.; and 3) CSCEN, the value which indicated whether or not the subject reached the MIII endpoint in the CSSI test. If an endpoint was obtained a score of 0 was assigned and 1 if it was not obtained.

The three variables derived from the SSTEO test included: 1) EOSC, the score or number of stops at which the MIII level of malaise occurred; 2) EOPTS, the number of symptom points attained at the conclusion of the tests; and 3) EOR(n), the susceptibility level assigned by one of the three individual raters. The same three variables obtained from the SSTEO test were used for the SSTEC test.

For the OVR test the two variables used in the analysis were the OVRSC and the OVR(n). OVRSC was the score each subject obtained on the OVR test. This score reflected the time in minutes at which the MIII level of malaise occurred. OVR(n) was the susceptibility level assigned by one of three raters.

For the staircase velocity motion tests (SVMT). The three variables included SVHM, SVPTS and SVR(n). Variable SVHM equaled the number of head movements completed when the MIII level occurred. SVPTS equaled the total number of symptom points accumulated by the end of the test, and SVR(n) was the level of susceptibility assigned to a particular individual by one of the three raters.

Variables EORAIL and ECRAIL were obtained from the posture test. EORAIL equaled the score attained during the eyes open postural ataxia test on the 4.45 cm rail width, and ECRAIL that socre attained with the eyes closed and standing on the 5.72 cm rail.

The four variables attained from the VOR measurements reflected phase and gain at two frequencies. VORP1 was the phase angle at 0.01 Hz, and VORP5 the phase at 0.16 Hz, VORG1 was the gain of the nystagmus at 0.01 Hz, and VORG5 the gain at 0.16 Hz.

Criterion Variables

Because of the only microgravity experience of the normative population that could be equaled with the astronaut population is obtained during parabolic flight, the motion sickness response to these flights was used as the prediction criteria. The three variables selected from parabolic flight consisted of: 1) KCPTS, a variable indicating the parabola at which the MIII level of malaise occurred and whether or not the subject vomited; 2) VOMIT, whether or not the subject vomited regardless of the parabola at which vomiting occurred and 3) KCR(n) the category of susceptibility assigned an individual based on one of three raters judgment.

Rater Variables

The variables CSR(n), OVR(n), EOR(n), ECR(n), SVR(n), and KCR(n) were all obtained from three independent raters. The three raters were familiar with each of the 6 provocative tests associated with these variables (CSSI, OVR, SSTEO, SSTEC, SVMT and KC-135), and were told to group the subjects used in this study into three groups of susceptibility for each of the tests. To do this, each rater was presented with a frequency histogram for each of the 6 tests. Figures 1-6 show these histograms. Each histogram is based on the frequency of sickness for the entire normative population . Figure 1 shows the frequency of the MIII score for this population, Figure 2 shows the test duration of the OVR, Figures 3 and 4 are the completed stops for the SSTEO and SSTEC test respectively. Figure 5 indicates the number of head movements completed for all subjects on the SVMT, and Figure 6, the parabolas flown prior to reaching the MIII level during parabolic flight.

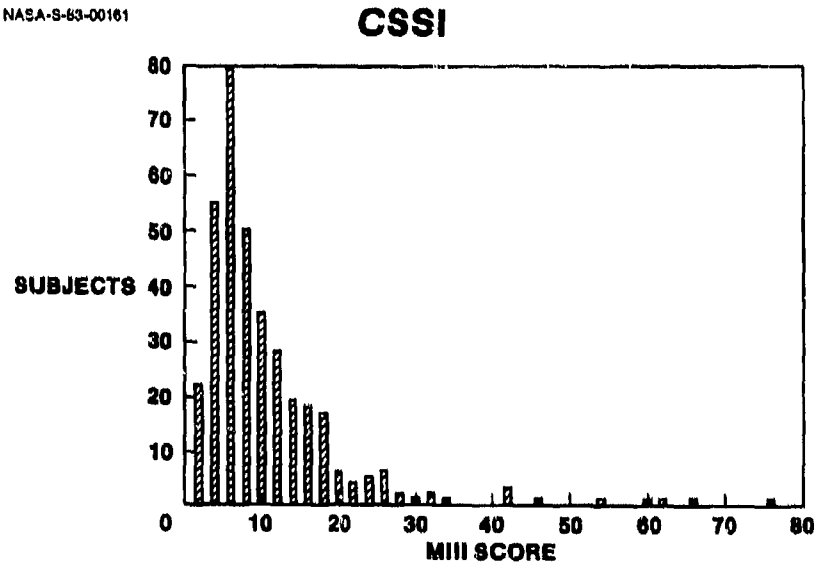

Figure 1

NASA-S-63-00155

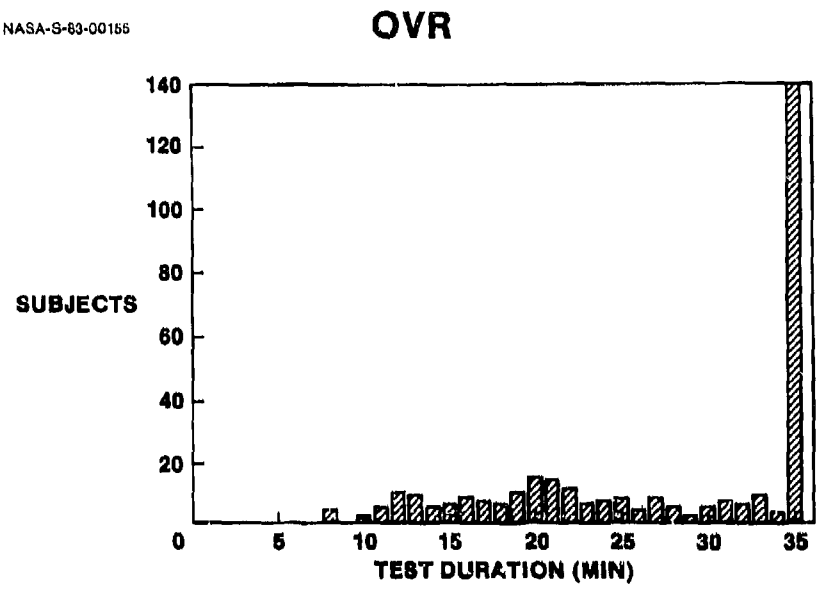

Figure 2

NASA-S-63-00158

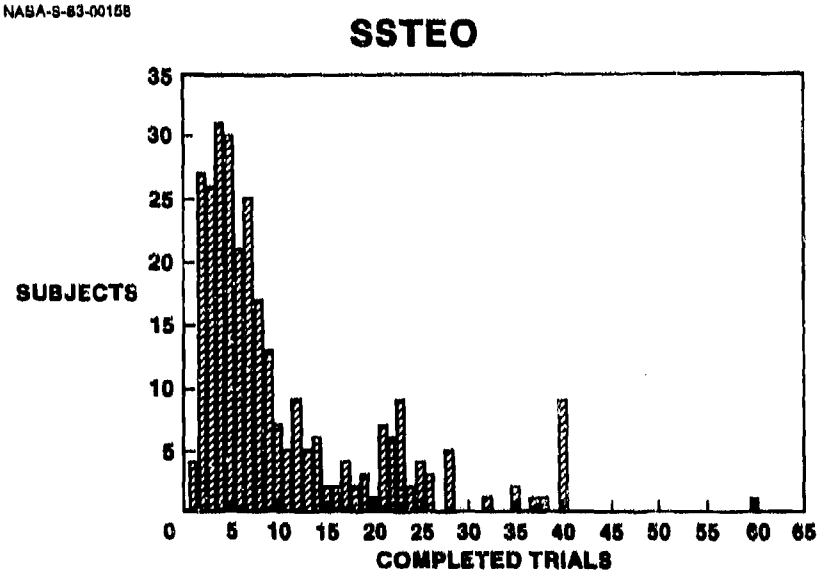

Figure 3

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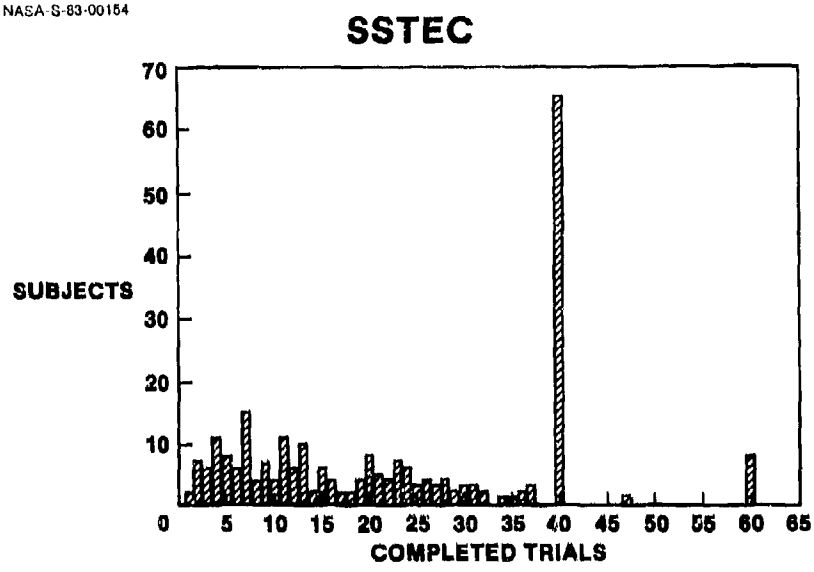

Figure 4

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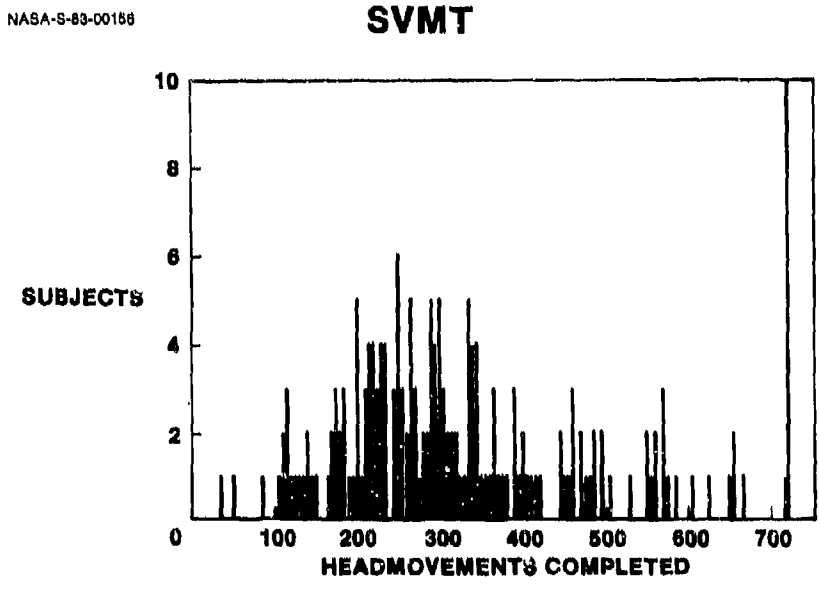


Figure 5

NASA-S-83-00187

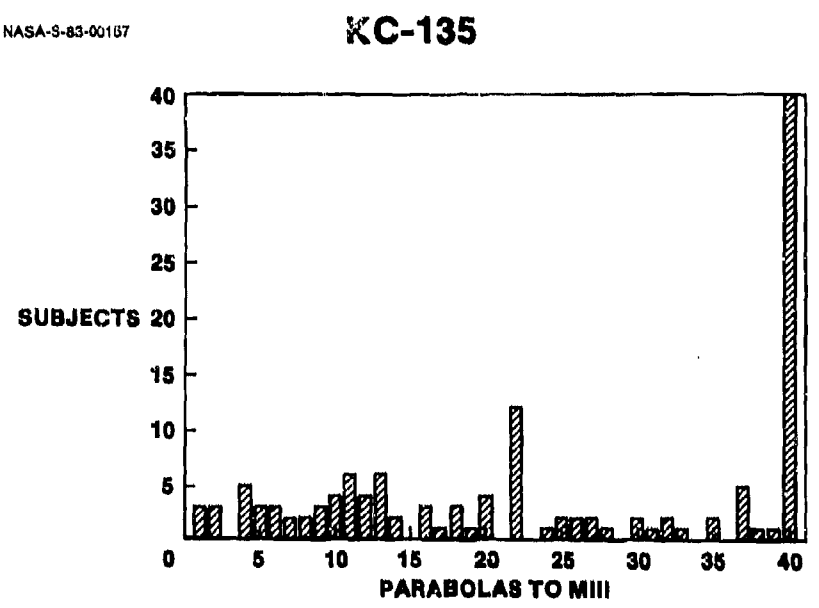


Figure 6

Using the frequency histograms each rater grouped subjects into one of three categories of susceptibility. By prior agreement the levels of susceptibility were identified as low to nonsusceptible, moderately susceptible or severely susceptible. Later the categories for parabolic flight were further reduced to VOMIT or NO VOMIT.

The relationship between each of the three independent raters was so high that only the rating from one of the three was included as the final variable for data analysis purposes.

Data Analysis

Descriptive Analysis

The first step of the data analysis was to try to understand the relationship between variables and to reduce the dimensionality with little or no loss in information. To accomplish this goal, the multivariate statistical method of factor analysis (78) was applied to the 27 variables shown in Table 1. This analysis used all of the variables on 159 subjects and was completed via a Digital Equipment Corporation Computer (VAX 11/780) using a commercial statistical software package (79). Criterion variables were included in the factor analysis to observe the degree of relationship the KC-135 criterion had with other variables.

Initially, the factor analysis computer program calculated the means, standard deviations, and intercorrelations using the scores from each of the 159 subjects. The intercorrelation matrix was a quantitative representation of all possible 378 relationships. The next step in the factor analysis was to reduce the dimensionality of the intercorrelation matrix by determining a smaller matrix (the Factor or F matrix) where each column represented a dimension that was independent (i.e., orthogonal of all other columns and each row contained a correlation coefficient (or factor loading) representing the variables relation to the dimension. Definitions of the dimension (or columns in the factor matrix) were made by noting which variables had the highest correlation in the column. If more than one variable was correlated (or "loaded") on a particular dimension, then only the variable with the highest correlation was used in subsequent predictive analysis. This process assured that all major non-redundant sources of common variance were included in the predictive analysis with a minimum number of variables and that the most representative variables were chosen. Mathematically, the factor analysis consisted of calculating the eigenvalues and eigenvectors of the intercorrelation matrix (R) and then calculating a matrix F such that $FF'=R$, where the diagonals of R contained the squared multiple correlation.

Because the squared multiple correlation used in the study was a measure of what each variable had in common with all other variables, the process explained all of the common variance. Those variables that had a relatively low squared multiple correlation were not discarded from subsequent analysis since they had some unique variance that could have been useful in prediction.

In short, the factor analysis was used not only to describe the variables but assisted in the selection of variables for the predictive phase of the analysis.

Predictive Analysis

In trying to access differences between groups for predictive purposes, it was obvious they varied in many ways and therefore required some form of multivariate statistical analysis. It has been noted (80) that the reduction of multiple measurements to a single weighted composite is the key to much of multivariate analysis. This is particularly true in discriminant function analysis. By assigning appropriate weighting coefficients, multiple correlated measurements could be converted to a single score thus reducing the mutlivariate problem to a univariate one. In 1936, Fisher (81) considered the problem of determining a linear combination of variables that would, better than any other combination, discriminant between two chosen groups. By better discriminant he specifically meant the ratio of between groups variance to within group variance (i.e., the familiar F ratio in analysis of variance) would have a larger value than any other linear function of the same variables. Fisher called this optimal linear combination the discriminant function. Other statisticians, especially Rao (82, 83) independently extended Fisher's work to more than two groups showing the procedure was mathematically optimal, and that after one linear combination was calculated successive optimal linear combinations (called multiple discriminant functions) could be extracted from successive residuals. Mathematically this involved finding the eigenvalues and eigenvectors of a matrix BW^{-1} (or a matrix of F ratio) where B was the between group matrix and W was the within group matrix, in contrast to factor analysis where the W matrix was used. With this accomplished, it became possible to examine the relative merits of each variable at each step and discard those variables that did not add appreciably to the prediction.

With this method, quantitative and qualitative criterion groups were identified on the basis of actual motion sickness responses to the KC-135 flights. Groups identified were no-vomit, vomit; low, moderate, or severe sickness; and sick or not sick. Using stepwise multiple discriminant analysis (2), predictor variables were differentially weighted to optimally predict the criterion of susceptibility. Each individuals scores or ratings on the predictor variables were multiplied by the appropriate weighting coefficient for a particular group and then summed to get a single composite score for the group. Individuals were then assigned to the different susceptibility groups for which the composite score was highest. Part of the subjects were used to develop the weighting coefficients and part were used to cross-validate the predictions.

Predictor variables in this study initially consisted of the first 24 variables shown in Table 1. For one phase of the discriminant analysis all variables were used. The final set of discriminant analysis utilized 9 variables that were the best "marker" variables for each of the factors in the factor analysis.

Ideally, we would like to have data on each of the tests so that we could have rated astronauts responses to Shuttle flights and determine the degree to which the developed equations would predict inflight responses. Unfortunately only the CSSI has been given preflight to all Shuttle crewmen. With this in mind, all 159 subjects were used to develop weighting coefficients for age, sex, and CSSI scores to predict low, moderate, or severe responses to the KC-135 flights. All crewman for the first nine Shuttle flights were rated in terms of the inflight sickness they actually experienced. All crewman's age, sex and CSSI scores were then inserted into the equations and assigned to the group (low, moderate, or severe) for which the composite score was the highest. These results were then tabled to calculate the percent correctly assigned.

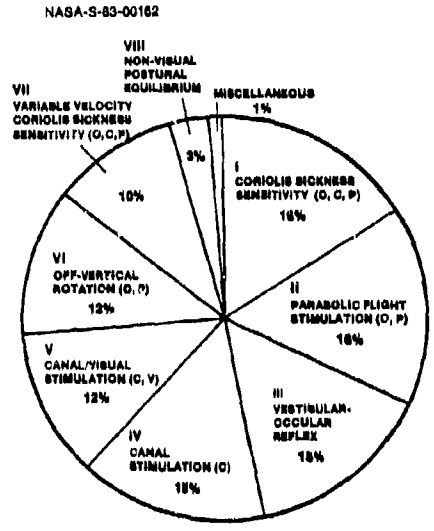
In short, by applying the method of stepwise multiple discriminant analysis to a random sample drawn from the data base, the objective was to:

1) establish equations for group with different levels of susceptibility
2) use the equations to predict motion sickness
3) delete those variables, where possible, that did not contribute to the prediction
4) cross-validate the coefficients by using the equations to assign new randomly drawn normative subjects to susceptibility groups and determining the percent of "hits" and "misses"
5) apply the method to the currently limited inflight data to determine the level of predictibility.

RESULTS

Shown in Table 1 are the means, standard deviations, and intercorrelations of 27 variables. Although three ratings were collected for each of the provocative tests, only one rating was used for each variable in the factor analysis since the ratings were so highly correlated with each other. The average inter-rater reliability for the CSSC, OVR, SSTEO, SSTEC, SV, and KC-135 was .88, .95, .94, .88, .98, .91, and .89, respectively.

Table 2 shows the rotated factor matrix with the squared multiple correlation of each variable with all other variables shown in the last column. The highest correlation (or factor loading) in each column has been underlined to highlight it as the defining variable. Each column represents a dimension and each value in the row is the correlation (or factor loading) of the variable with the dimension. The last row across the bottom gives the precent of common variance associated with that particular dimension (or column). The results indicate that nine dimensions account for all of the common variance associated with the 27 variables. Figure 7 provides a pictorial view of the results of the factor analysis.



INDEPENDENT FACTORS ASSOCIATED WITH VARIABLES USED IN THE STUDY

Each factor is named and shows the percent of common variance accounted for by that dimension. One variable from each factor was used in the discriminant analyses. O, C, P, & V stand for the type of stimulation i.e., ootolith, canal, propoceptive, and visual.

Figure 7

Results of the attempt to predict and cross-validate the degree of susceptibility to KC-135 parabolic flights are shown in Tables 3 and 4. Subjects were selected at random to develop the equations (labeled "original cases") and the remaining cases (labeled "cross-validation cases") were classified using the equations. Table 3 shows the means and standard deviations for each of the susceptibility groups (low, moderate, and severe) for both the original and cross-validation samples. Table 4 shows the discriminant function weighting coefficients developed from each of the original cases in Section I. Section II of Table 4 shows the results (in terms of numbers and percent) of applying these coefficients to each of the original sample groups. Subjects were always predicted to be members of the group that had the highest weighted score for the subject. For example, the low susceptible group actually had 48 cases but 7 of these cases had a higher weighted composite score using the coefficients for the moderate group and were therefore predicted (in this case, misclassified) to be in moderate group. Notice if the results were perfect, all cases would have been on the diagonals. The results indicated correct prediction on the original cases of 69, 50, and 61 percent for the low, moderate, and severe groups, respectively. The total correct for the original sample was 64 percent. Finally, Table 4, Section III is identical to Section II except the equations were applied to the cross-validation sample. These results showed correct predictions of 59, 50, and 40 percent, respectively, with the total correct dropping to 50 percent for all three groups.

Tables 5 and 6 have the same information and are set up similar to Tables 3 and 4, except the prediction is simplified to predict if the individual will or will not get sick rather than the degree of susceptibility. In this case, 58 percent of the not-sick and 63 percent of the sick were correctly predicted for the original samples for a total correct of 60 percent. On cross-validation, 67 percent and 70 percent of the not-sick and sick respectively were correctly predicted with a total correct of 68 percent.

The final tables (Tables 7 and 8) are similar to the others, except the attempt was to predict no vomiting or vomiting on any of the parabolic flights flown. For the original cases, 64 percent of the no vomit and 89 percent of the vomit cases were correctly categorized for a total correct of 74 percent. On cross-validation, this reduced to 61 percent and 58 percent for a total correct of 60 percent. For each cross-validation subjects, two weighted scores were calculated by multiplying his measurements by the weights and assigning him to the group having the largest score.

The results of applying the equations developed for age, sex and CSSI scores to the ratings of inflight responses was essentially negative. Of 32 inflight crew experiences, 18 were actually rated as low, 11 as having moderate symptoms, and 3 as severe. The equations misclassified 16 in the low group as moderate and misclassified all 3 severe symptom individuals, (predicting 2 as moderates and 1 as low). Of the 11 individuals actually rated as inflight moderates, the equations predicted 10 of them correctly. In short, the equations overly assigned individuals to the moderate group.

DISCUSSION

Descriptive Analysis

With respect to the means and standard deviations, presented in Table 1, the results showed considerable variability on the CSSC. Part of this resulted from some 33 individuals who never reached endpoint in the test. The effect of this on the total analysis is not known, although, it probably contributed some additional error to this variable making it somewhat less useful. The intercorrelations given in Table 1 show generally low correlations and while their overall description is best seen in the factor matrix, the intercorrelations are useful in looking up some specific univariate relations. With this number of cases, a correlation of .19 is significantly different from zero.

The best explanation of the information content of intercorrelation matrix was obtained from the rotated factor matrix shown in Table 2. In fact, any correlation can be reproduced from the factor matrix by cross multiplying each element in the two rows representing the correlation of interest and summing these products across the columns. As noted in the table, the last column contains the squared multiple correlations representing the relationship of a particular variable to all of the other variables in the matrix. Background variables (age and sex), questionnaire responses, the non-provocative rail tests, and the higher frequency VOR were not correlated with any of the other tests. This suggested that from a general prediction viewpoint, these variables should not be discarded as they might contain some uniqueness that could aid in prediction beyond what the provocative test provide. For this reason they were used in the development of prediction equations.

The Coriolis sickness sensitivity dimension represented by Factor I showed the effects of both constant and staircase velocity as evidenced by SVHM having significant loadings on both Factor I and Factor VII. Also seen on this dimension (Factor I) was the score variance for the off-veritcal rotation, as well as for both the visual and non-visual part of the sudden-stop test. This probably reflects that the same fundamental provocative procedures are used for each of these test.

Prior to the study, it was thought by some that since all of the provocative test use the same rotational procedure they would be highly related and perhaps some could be dropped. If this were true, Factor I would have shown higher loadings on each of the procedural variations than was actually the case. The analysis showed that each procedural variation was sufficiently unique and orthogonal to make it a potentially useful predictor. With respect to objective scores (as opposed to ratings), the results indicated higher loadings for scores except for the Sudden-Stop Test with both eyes open and closed.

Of some interest was the lack of any significant correlation of any single test with the criterion selected for this study, i.e., the responses to the KC-135 parabolic flights. This suggests that a low prediction is almost inevitable unless some optimal combination of the independent predictors is possible that collectively captures what little relation that does exist. While this study used KC-135 reactions as a criterion since it does include a limited zero gravity experience, its emergence as an independent factor suggest it may be more useful to think of it as simply another provocative test in predicting some other experiences of motion sickness.

In summary, the factor analysis indicated which variables should be included in the predictive analysis to represent each of the factors. In addition, the results indicated some variables that were not loaded on any dimension and should be included in prediction because of their uniqueness and their potential contribution.

Predictive Analysis

In general, the level of predictions in the study were better than chance but they were not high. The fact that the predictions exceeded chance expectations was encouraging since there was virtually no significant correlations with the criterion. This lack of correlation with the criterion also explains why it was not useful in this particular study to determine relative contribution of each variable using the discriminant functions. One way of doing this was to systematically drop the last variable and observe the percent change. The classification function given in the tables do not indicate the order that each variable was utilized in the discriminant analysis. For example, the variables in Table 4 were successively added as follows: OVRSC, AGE, EOR1, VORP1, ECR1 and CSSC. The first discriminant selected and classified all subjects using only OVRSC into low, moderate and severe with a percent success of 62, 10, and 65, respectively. AGE was the next variable added to OVRSC and it improved the success rate to 43, 40, and 60, respectively. The next variable added to the others was EOR1 and this changed the success rates to 56, 30, and 60. This process of gradual improvement for different categories continued until it reached 69, 50, and 61 for the CSSC. In short, the relative contribution was not clear cut except to a particular low, moderate, or severe category. Similarly, the variables selected by the analysis process from one discriminant analysis was not related to another discriminant analysis. An exception to this was that OVRSC was selected as first or second in each of the three prediction.

In using multiple factor analysis, the concern was to describe common elements, or what Thurstone (7) called the "invariant structure", and the correlation coefficients were easily interpreted. In multiple discriminant analysis the concern was to maximize differences between groups and the coefficients, while mathematically optimal, had little meaning from an interpretation veiwpoint. For this reason, no attempt was made to interpret the weighting coefficients, however, a computer program was written to use the coefficients on future laboratory subjects to continue the validation process.

The property of the multiple discriminant analysis to capitalize on any difference between groups required special attention to make sure the number of original cases was sufficiently representative to eliminate chance difference between variables. Randomization of the original and the cross-validation population was used to assure that the cross-validation sample was represented statistically by the original population or a significant loss in prediction would have occurred. While some shrinkage did occur, it was well within expectations. Finally, as the number of variables used in discriminant analysis increase the theoretical prediction increases. For example, some initial runs using 43 variables and 60 cases showed 95 percent correct predictions on the original cases. However, this was due to increased curve fitting to chance points that disappeared on cross-validation. The number of cases required to establish validity increases dramatically as number of variables increase. For this reason, factor analysis was an important step before discriminant analysis as it both reduced the number of variable and assured any new variable was independent of those previously selected.

The results of this study suggest that no single motion sickness test, provocative or otherwise, can be used to predict susceptibility in a novel motion environment. Currently, our program employs only the CSSI test preflight to test these crewmembers who will fly. As might be expected the CSSI has not been effective in predicting susceptibility aloft. We are now modifying our preflight testing to replace the CSSI test with the OVR. However, based on the findings presented here, the OVR alone should not provide a higher index of success than the CSSI. We believe that a small battery of tests is required, and until an expanded program is implemented, our predictive capability will not improve.

SUMMARY

A study was completed (a) to describe the univariate and multivariate relationships of provocative measures used by the neurophysiology laboratory, (b) to use normative subjects to develop and cross-validate sets of linear equations that optimally predict motion sickness in parabolic flights, and (c) to evaluate the possibility of reducing the number of measurements required for prediction.

After describing the variables verbally and statistically for 159 subjects, a factor analysis of 27 variables was completed to improve understanding of the relationships between variables and to reduce the number of measures for prediction purposes. The results of this analysis showed that nine factors account for 100 percent of the common variance and that none of variables are significantly related to the responses to parabolic flights.

Using the results of the factor analysis, a set of variables were selected to predict responses to KC-135 flights. A series of discriminant analyses were completed using part of the subjects to develop sets of predictive linear equations and part to cross-validate the equations. Results indicated that low, moderate, or severe susceptibility could be correctly predicted 64 percent and 53 percent of the time on original and cross-validation samples, respectively. Similarly, sickness or non-sickness was 60 and 68 percent correct on original and cross-validation samples, respectively. Predicting vomiting or no vomiting on the KC-135 flights was 73 and 62 percent correct on original and cross-validation samples, respectively. Finally, equations developed on the normative group for sex, age, and Coriolis Sickness Sensitivity Index were not effective in predicting crewman's space adaptation to the first nine Shuttle flights.

Both the factor analysis and the discriminant analysis provided no basis for reducing the number of tests. The results suggested that the provocative test are relatively independent and that all should be retained until new criterion measures are available to evaluate them.

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TABLE 1

INTERCORRELATIONS, MEANS AND STANDARD DEVIATIONS OF VARIABLES USED IN THE FACTOR ANALYSIS*

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	MEAN	S.D.
SEX	1.00																											1.23	.42
AGE	-.06	1.00																										30.55	7.20
CSSC	.02	-.11	1.00																									16.87	18.52
CSR1	-.06	.18	-.86	1.00																								3.33	.73
CSCEN	-.03	-.17	.48	-.50	1.00																							.21	.41
OVRSC	-.06	-.19	.41	-.45	.29	1.00																						27.79	7.96
OVR1	.06	.16	-.40	.43	-.31	-.94	1.00																					1.99	1.04
EOSC	-.04	.04	.42	-.35	.26	.27	-.28	1.00																				10.81	9.19
EOPTS	.01	.00	-.05	.04	-.19	-.04	.14	-.36	1.00																			8.84	2.69
EOR1	-.01	.04	-.30	.25	-.27	-.20	.29	-.76	.60	1.00																		3.05	1.02
ECSC	-.10	.05	.33	-.34	.14	.24	-.25	.50	-.24	-.40	1.00																	22.89	13.65
ECPTS	-.02	-.04	-.26	.21	-.06	-.08	.15	-.43	.41	.46	-.67	1.00																6.43	4.17
ECR1	.00	-.01	-.23	.25	-.12	-.13	.20	-.42	.39	.51	-.87	.79	1.00															2.36	1.31
SVHM	-.03	-.06	.58	-.51	.32	.43	-.39	.27	.02	-.13	.25	-.12	-.12	1.00														258.13	193.30
SVPTS	.00	.06	-.08	.03	-.14	.09	-.05	-.19	.26	.25	-.13	.23	.22	.46	1.00													6.82	3.85
SVR1	.00	.15	-.20	.17	-.26	-.03	.07	-.25	.25	.28	-.19	.24	.26	.25	.87	1.00												2.32	1.40
QUESX	-.06	-.01	.09	-.15	.07	.09	-.09	.00	.00	.05	.09	-.06	-.05	.03	.06	.10	1.00											2.87	3.43
QUESS	.00	.13	.08	-.02	.09	.04	-.01	-.05	.01	.08	-.05	.11	.10	.15	.10	.07	-.01	1.00										3.02	4.62
EORAIL	-.03	-.07	-.04	.07	-.09	-.01	.04	.06	-.03	.00	.05	-.14	-.09	-.04	-.06	-.04	.01	-.04	1.00									86.46	26.92
ECRAIL	.14	-.15	.00	-.04	.02	-.05	.07	.07	.14	.02	-.06	-.04	.06	-.03	-.01	.01	.00	-.15	.21	1.00								41.98	30.55
VORP1	-.04	.04	-.02	-.05	-.11	.10	-.06	.02	.13	.09	-.05	.15	-.19	.23	.47	.42	.01	-.07	.04	.16	1.00							26.79	23.94
VORG1	-.08	.04	-.06	.00	-.11	.04	-.04	.05	.05	.06	-.01	.10	.15	.21	.43	.41	.01	.04	-.04	.08	.80	1.00						.18	.18
VORP5	.07	.05	.01	-.02	-.12	.06	-.02	.00	.05	.08	-.02	.08	.08	.20	.26	.26	.02	-.11	-.04	.05	.52	.38	1.00					2.42	4.08
VORG5	-.08	.19	-.05	.05	-.08	.02	-.03	.00	.04	.07	.00	.09	.10	.05	.28	.26	-.03	.09	-.04	-.02	.48	.47	.23	1.00				.29	.51
KCPTS	.03	-.20	-.22	.23	-.15	-.26	.25	-.28	.10	.19	-.20	.07	.12	-.19	-.01	.08	.10	-.03	.00	.09	-.13	-.10	-.17	-.10	1.00			1.70	.87
VOMIT	.07	-.17	-.13	.13	-.03	-.19	.17	-.23	.05	.17	-.15	.07	.09	-.14	-.04	.02	.09	-.01	.03	.09	-.17	-.13	-.19	-.12	.89	1.00		1.36	.48
KCR1	.03	-.15	-.24	.28	-.24	-.25	.25	-.32	.13	.23	-.19	.07	.12	-.17	.05	.13	.01	-.04	-.05	.00	-.14	-.10	-.18	-.09	.89	.71	1.00	2.02	1.10

*N = 159

TABLE 2
ROTATED FACTOR LOADINGS

VARIABLE NAME	VARIABLE NUMBER	I	II	III	IV	V	VI	VII	VIII	IX	R^2*
SEX	1	.04	.01	-.06	.03	-.01	.05	.02	.10	-.25	.14
AGE	2	-.15	-.20	.10	-.07	.02	.20	.02	-.26	.12	.27
CSSC	3	.94	-.07	-.01	-.14	-.09	-.13	-.10	-.15	-.05	.82
CSR1	4	-.85	.11	.02	.15	.04	.20	.04	-.07	.06	.80
CSCEN	5	.50	-.10	-.18	.02	-.16	-.17	-.04	.20	-.02	.41
OVRSC	6	.30	-.12	.05	-.05	-.05	-.91	.04	-.04	.12	.92
OVR1	7	-.26	.11	-.03	.10	.14	.91	-.02	.06	-.04	.92
EOSC	8	.33	-.17	.03	-.24	-.67	-.06	-.07	.00	.28	.75
EOPTS	9	.06	.07	.10	.23	.56	.06	.13	-.03	.13	.49
EOR1	10	-.16	.09	.06	.25	.94	.10	.07	-.01	.03	.79
ECSC	11	.21	-.11	-.12	-.87	-.14	-.08	-.01	.06	.35	.87
ECPTS	12	-.11	-.00	.07	.73	.28	.02	.10	.01	.08	.69
ECR1	13	-.08	.05	.13	.94	.26	.06	.04	-.08	.04	.90
SVHM	14	.58	-.07	.19	-.11	-.03	-.22	.41	-.14	.04	.69
SVPTS	15	.01	.02	.37	.10	.15	-.05	.90	-.12	-.05	.86
SVR1	16	-.11	.09	.36	.12	.15	.03	.76	-.15	-.04	.82
QUESX	17	.12	.11	-.02	-.06	.08	-.07	.08	.10	.06	.15
QUESS	18	.08	-.02	-.06	.09	.07	.01	.09	-.18	.07	.16
EORAIL	19	-.04	-.02	.02	-.09	.02	.01	-.05	.18	-.04	.18
ECRAIL	20	.08	.07	.10	.08	-.02	.06	-.02	.34	-.01	.23
VORP1	21	.03	-.06	.94	.09	.01	-.04	.15	.20	.02	.77
VORG1	22	-.02	-.03	.80	.05	-.02	-.03	.16	.09	.09	.72
VORP5	23	.02	-.13	.52	.01	.07	-.01	.07	.06	-.05	.38
VORG5	24	-.05	-.06	.50	.02	.03	.00	.09	-.06	.09	.32
KCPTS	25	-.12	.98	-.10	.06	.06	.10	.02	.09	-.01	.94
VOMIT	26	-.02	.86	-.17	.06	.08	.05	.02	.19	.00	.85
KCR1	27	-.18	.87	-.07	.02	.12	.09	.03	-.11	-.04	.85
PERCENT		16	16	15	15	12	12	10	3	1	
CUMMULATIVE %		16	32	47	62	74	86	96	99	100	

*R^2 = Squared multiple correlation i.e., the relationship of this variable to all other variables in the matrix. Note 1-R^2 is the amount of variance that is either unique, error of measurement, or both.

TABLE 3

MEANS AND STANDARD DEVIATIONS OF VARIABLES USED TO PREDICT DEGREE OF SUSCEPTIBILITY (LOW, MODERATE, OR SEVERE) TO KC-135 PARABOLIC FLIGHTS

I. ORIGINALS CASES:

VARIABLES	GROUPS					
	LOW		MODERATE		SEVERE	
	MEAN	S.D.	MEAN	S.D.	MEAN	S.D.
SEX	1.19	.39	1.40	.52	1.30	.47
AGE	30.46	7.08	27.00	4.69	27.56	5.61
CSSC	18.38	20.61	23.39	27.23	9.60	14.84
OVRSC	29.67	7.16	28.50	3.28	23.26	6.90
EOR1	2.94	.78	3.40	.84	3.43	.79
ECR1	2.64	.76	3.10	.87	3.09	.90
SVMPTS	7.00	3.87	7.40	4.00	6.82	3.73
ECRAIL	40.96	30.04	44.40	35.76	49.87	31.38
VORP1	29.23	23.52	18.30	24.55	20.75	24.61
NUMBER CASES	48		10		23	

II. CROSS-VALIDATION CASES:

VARIABLES	GROUPS					
	LOW		MODERATE		SEVERE	
	MEAN	S.D.	MEAN	S.D.	MEAN	S.D.
SEX	1.26	.44	1.12	.34	1.20	.41
AGE	32.64	6.92	34.37	9.63	28.50	6.32
CSSC	20.65	18.78	18.50	16.21	9.07	6.18
OVRSC	28.74	7.74	28.75	8.58	25.35	9.04
EOR1	3.02	.84	3.19	.83	3.45	.76
ECR1	2.71	.80	2.75	.77	2.80	.89
SVMPTS	6.76	3.87	5.94	4.15	6.95	3.99
ECRAIL	41.95	30.37	23.44	23.23	49.05	30.69
VORP1	31.48	23.74	20.28	23.96	27.43	23.63
NUMBER CASES	42		16		20	

TABLE 4

PREDICTION OF DEGREE OF SUSCEPTIBILITY
(LOW, MODERATE, OR SEVERE) TO KC-135 PARABOLIC FLIGHTS

I. CLASSIFICATION FUNCTION:

VARIABLES	WEIGHTING COEFFICIENTS		
	LOW	MODERATE	SEVERE
AGE	1.048	.977	.942
CSSC	.170	.195	.161
OVRSC	.827	.796	.705
EOR1	6.382	7.286	6.835
ECR1	3.782	4.455	4.313
VORP1	-.013	-.039	-.034
CONSTANT	-45.069	-46.850	-41.103

II. ORIGINAL NUMBER AND PERCENT OF CASES CLASSIFIED INTO GROUPS:

A. NUMBER OF ORIGINAL CASES:

		PREDICTED			
		LOW	MODERATE	SEVERE	TOTAL
ACTUAL	LOW	33	7	8	48
	MODERATE	2	5	3	10
	SEVERE	4	5	14	23

TOTAL NUMBER OF CASES = 81

B. PERCENT OF ORIGINAL CASES:

		PREDICTED		
		LOW	MODERATE	SEVERE
ACTUAL	LOW	69%	15%	17%
	MODERATE	20%	50%	30%
	SEVERE	17%	22%	61%

TOTAL % CORRECT = 64%

III. CROSS-VALIDATION NUMBER AND PERCENT OF CASES CLASSIFIED IN GROUP:

A. NUMBER OF CROSS-VALIDATION CASES:

		PREDICTED			
		LOW	MODERATE	SEVERE	TOTAL
ACTUAL	LOW	25	5	12	42
	MODERATE	7	8	1	16
	SEVERE	7	5	8	20

TOTAL NUMBER OF CASES = 78

B. PERCENT OF CROSS-VALIDATION CASES:

		PREDICTED		
		LOW	MODERATE	SEVERE
ACTUAL	LOW	59%	12%	28%
	MODERATE	44%	50%	6%
	SEVERE	35%	25%	40%

TOTAL % CORRECT = 53%

TABLE 5

MEANS AND STANDARD DEVIATIONS OF VARIABLES USED TO
PREDICT SICK OR NOT-SICK ON KC-135 PARABOLIC FLIGHTS

I. ORIGINAL CASES:

VARIABLES	GROUPS			
	NOT-SICK		SICK	
	MEAN	S.D.	MEAN	S.D.
SEX	1.20	0.41	1.21	0.41
AGE	32.10	8.06	28.86	6.64
CSSC	18.87	18.24	15.44	17.26
OVRSC	28.47	7.62	27.15	7.25
EOR1	2.95	0.82	3.15	1.05
ECR1	2.12	1.24	2.18	1.44
SVMPTS	7.12	3.76	7.07	3.83
ECRAIL	42.43	30.66	39.97	31.31
VORP1	30.03	23.37	25.32	23.57
COUNTS	48		38	

II. CROSS-VALIDATION CASES:

VARIABLES	GROUPS			
	NOT-SICK		SICK	
	MEAN	S.D.	MEAN	S.D.
SEX	1.23	0.43	1.30	0.46
AGE	30.76	5.68	30.06	8.11
CSSC	20.08	21.45	11.34	15.44
OVRSC	30.09	7.14	24.46	9.46
EOR1	2.78	1.15	3.40	1.03
ECR1	2.30	1.29	2.96	1.09
SVMPTS	6.61	3.97	6.23	4.01
ECRAIL	40.26	29.61	44.26	30.63
VORP1	30.57	23.96	19.04	24.25
COUNTS	42		30	

TABLE 6

PREDICTION OF SICK OR NOT-SICK IN RESPONSE
TO KC-135 PARABOLIC FLIGHTS

I. CLASSIFICATION FUNCTION:

VARIABLES	NOT-SICK	SICK
AGE	.774	.706
OVRSC	.720	.679
VORP1	.027	.020
CONSTANT	-23.792	-20.361

II. ORIGINAL NUMBER AND PERCENT OF CASES CLASSIFIED INTO GROUPS:

A. NUMBER OF ORIGINAL CASES:

		PREDICTED		
		NOT-SICK	SICK	TOTAL
ACTUAL	NOT SICK	28	20	48
	SICK	14	24	38

TOTAL NUMBER OF CASES = 86

B. PERCENT OF ORIGINAL CASES:

		PREDICTED	
		NOT-SICK	SICK
ACTUAL	NOT SICK	58%	42%
	SICK	37%	63%

TOTAL % CORRECT = 60%

III. ORIGINAL NUMBER AND PERCENT OF CASES CLASSIFIED INTO GROUPS:

A. NUMBER OF ORIGINAL CASES:

		PREDICTED		
		NOT-SICK	SICK	TOTAL
ACTUAL	NOT SICK	28	14	42
	SICK	9	21	30

TOTAL NUMBER OF CASES = 72

B. PERCENT OF ORIGINAL CASES:

		PREDICTED	
		NOT-SICK	SICK
ACTUAL	NOT SICK	67%	33%
	SICK	30%	70%

TOTAL % CORRECT = 68%

TABLE 7

MEANS AND STANDARD DEVIATIONS OF VARIABLES USED TO
PREDICT NO VOMIT AND VOMIT ON KC-135 PARABOLIC FLIGHTS

I. ORIGINAL CASES:

VARIABLES	NO VOMIT		VOMIT	
	MEAN	S.D.	MEAN	S.D.
AGE	1.13	.34	1.30	.46
SEX	31.00	6.77	29.55	6.89
CSSR	20.50	21.19	12.87	13.55
OVRSC	30.00	7.08	24.89	7.89
EOR1	2.85	.93	3.29	1.10
ECR1	2.15	1.32	2.59	1.46
SVMPTS	7.23	3.79	5.86	4.68
ECRAIL	40.33	31.25	39.41	32.23
VORP1	26.61	23.28	20.94	24.14
# OF CASES	39		27	

II. CROSS-VALIDATION CASES:

VARIABLES	NO VOMIT		VOMIT	
	MEAN	S.D.	MEAN	S.D.
SEX	1.26	.44	1.26	.44
AGE	31.76	8.05	28.42	5.77
CSSR	17.60	17.71	14.30	20.05
OVRSC	28.31	7.96	26.52	8.43
EOR1	2.96	1.02	3.26	1.06
ECR1	2.34	1.25	2.45	1.31
SVMPTS	6.76	3.86	7.29	3.11
ECRAIL	39.61	29.17	61.03	30.69
VORP1	31.94	23.96	21.78	23.40
# OF CASES	62		31	

TABLE 8

PREDICTION OF VOMIT OR NO VOMIT
TO KC-135 PARABOLIC FLIGHTS

I. CLASSIFICATION FUNCTION:

VARIABLES	WEIGHTING COEFFICIENTS	
	NO VOMIT	VOMIT
SEX	7.604	8.298
OVRSC	.641	.555
EOR1	2.517	2.925
SVMPTS	.185	.122
VORP1	.042	.032
CONSTANT	-19.394	-18.493

II. ORIGINAL NUMBER AND PERCENT OF CASES CLASSIFIED INTO GROUPS:

A. NUMBER OF ORIGINAL CASES:

		PREDICTED		
		NO VOMIT	VOMIT	TOTAL
ACTUAL	NO VOMIT	29	10	39
	VOMIT	8	19	27

TOTAL NUMBER OF CASES = 66

B. PERCENT OF ORIGINAL CASES:

		PREDICTED	
		NO VOMIT	VOMIT
ACTUAL	NO VOMIT	74%	26%
	VOMIT	30%	70%

TOTAL % CORRECT = 73%

III. CROSS-VALIDATION NUMBER AND PERCENT OF CASES CLASSIFIED IN GROUP:

A. NUMBER OF CROSS-VALIDATION CASES:

		PREDICTED		
		NO VOMIT	VOMIT	TOTAL
ACTUAL	NO VOMIT	41	21	62
	VOMIT	14	17	31

TOTAL NUMBER OF CASES = 93

B. PERCENT OF CROSS-VALIDATION CASES:

		PREDICTED	
		NO VOMIT	VOMIT
ACTUAL	NO VOMIT	66%	34%
	VOMIT	45%	55%

TOTAL % CORRECT = 62%

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DISCUSSION

CLEMONT: Did I understand that it was a factoral analysis which included 27 variables performed on data from 150 subjects? Is this right? A factoral analysis is very sensitive to huge errors.

RESCHKE: In fact, we had over 40 variables that we used in the analysis and we began to selectively drop these out. I didn't point out as the slides progressed when we were predicting vomit, no vomit, we were doing so, I believe, with only four different variables for the 159 subjects. This is the direction we wanted to go. We were attempting to get away from curve fitting.

CLEMONT: If the number of subjects is small with regard to the number of variables analyzed, then the results of cross-sections usually are disappointing.

RESCHKE: That's absolutely true because you design the equations on the original sample.

CLEMONT: Some of the variables are qualitative, most of them, severe, medium and high. Did you try to find an optimal scoring system of these qualitative variables or did you score them arbitrarily 0, 1, 2?

RESCHKE: All of the scoring for motion sickness responses were done using the diagnostic categorization chart that Dr. Lackner showed earlier this morning.

LACKNER: You have a great deal of data on a large number of both male and female subjects. One of the firmly entrenched views in the literature is that there are major differences in susceptibility between men and women, with women being more susceptible. I think you have data that speaks to this issue and I wonder if you would describe it?

RESCHKE: That issue was really not part of the study but we do have correlation data that suggests, yes, there is a sex difference and yes, there is not. It is very test specific. Once again, the general roles have taken a tumble and there are no specific trends for males and females.

PREDICTION OF SEASICKNESS SUSCEPTIBILITY

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SUMMARY

Thirty-nine subjects, suffering from chronic seasickness, and 21 controls have been submitted to several tests in order to find parameters for the prediction of seasickness susceptibility. Routine ENG examination revealed a labyrinthine predominance of more than 30% with caloric irrigation in about 15% of the seasickness susceptibles, suggesting a higher incidence of chronic motion sickness susceptibility in subjects with a labyrinthine imbalance than in normals. Cupulometry revealed identical slopes of the sensation cupulogram for both groups. No difference in slope was found for the nystagmus cupulogram either. The time constant of the 'velocity storage mechanism' also covered the same range for both groups. Stabilometry performed in a tilting room suggested that seasickness susceptibles are more visually oriented than the controls as revealed by the visually induced postural instability.

INTRODUCTION

At the Vestibular Department of the Free University Hospital in Amsterdam, 50 subjects from the Royal Dutch Navy have so far been examined with respect to their high susceptibility to seasickness. These subjects have been submitted to a routine vestibular ENG examination and to stabilometry in a tilting visual surround. During the first examinations Coriolis techniques were used too, but soon cupulometry was preferred.

Cupulometry as a test to assess motion sickness is well known in the literature. De Wit (1953) reported that the cupulogram could discriminate between seasickness-susceptible (steep slope of the cupulogram) and non-susceptible subjects (shallow slope). Similar results were reported by Aschan (1954) and Krijger (1954), who both showed that experienced fighter pilots had a particularly shallow slope of the cupulogram. Dobie (1974) could not reproduce these findings in a study on 1,000 pilots.

These differences in the slope of the cupulogram reflect differences in the time constant of the central vestibular system (Raphan et al. 1979): the steeper the cupulogram, the longer the time constant. Since adaptation results in a shortening of this time constant and chronic seasickness is assumed to be a non-adaptation phenomenon (or non-habituation phenomenon), large time constants are expected with seasickness susceptibles, which is in line with the findings of e.g. de Wit. Shortening of the time constant for sensation and nystagmus after strong vestibular stimulation has been used as a sign of adaptation in a study on seasickness susceptibility (Van Maanen 1965).

In our test battery we also incorporated stabilometry in a tilting room, since this test does not deal with the semicircular canals as does cupulometry, but more with otolithic-visual interactions which are similar to what may happen on a ship. In this test we measure postural stability of a subject standing on a firm horizontal support, while his visual surround is sinusoidally tilted. The rationale behind this test is that a conflict about verticality is created between the visual information (the tilted room) and the otolithic information (gravity). If the subject relies on the otolithic information only, he will maintain postural stability, but if he relies on vision a large lateral body sway will be the result following the room tilt.

In close cooperation with the Vestibular Department of the Academic Medical Centre in Amsterdam, 28 young biologists have also been examined, prior to scientific nautical expeditions, to screen them on chronic motion sickness susceptibility. These subjects were submitted to the same test battery.

This study presents a first analysis of the material in order to find parameters which may be useful for the prediction of seasickness susceptibility. We have limited ourselves to the study of two subgroups, one consisting of seasickness susceptibles, including those subjects (N = 39) from the Royal Dutch Navy who suffered from seasickness continuously when the wind speed exceeded 2 on the scale of Beaufort - according to their own reports -, and one consisting of controls including only those biologists (N = 20) who actually participated in an expedition without showing signs of chronic seasickness, which means that they adapted within a few days.

The age range was the same for both groups; the controls, however, consisted of 14 males and 6 females whereas the seasickness susceptibles were only males.

METHODS

Eye movements were recorded using electronystagmography (AC-amplification, time constant 5 s). Routine vestibular and optokinetic examination included a search for spontaneous and provocative nystagmus together with optokinetic nystagmus, smooth pursuit and suppression of the vestibular nystagmus by visual fixation. Caloric irrigation was performed using water of 30 and 44°C. Maximum nystagmus slow phase velocity (SPV) of the response was calculated, after which the response difference percentage of both labyrinths was computed.

Cupulometry was performed with a rotatory chair inside a drum (Tönnies, Freiburg im Breisgau). Subjects were sitting in darkness with their eyes open and signalled direction and termination points of the induced rotation sensation. These data, together with nystagmus and chair velocity, were recorded continuously. Nystagmus and sensation duration were measured after a deceleration of 90°/s^2 from a constant velocity rotation of 6, 14, 30, 60 and 90°/s CW and CCW. Plotting the duration of the sensation or nystagmus against ln ω yields a so-called cupulogram.

The time constant of the 'velocity storage mechanism' (Raphan et al. 1979) was estimated from the nystagmus decay following the stop from the 90°/s constant velocity rotation.

The tilting room (Tönnies, Freiburg im Breisgau) is a completely closed device (2.5 x 2.5 x 2 m), except for a hole in the floor and a door. Lateral tilting is done from the base (sinusoidal tilting with an amplitude of 5° at 0.025, 0.05, 0.1 and 0.2 Hz). Subjects had to stand on a stabilometer (Kapteyn and de Wit 1972) placed on a firm horizontal support in the hole in the floor, and were instructed to maintain an upright stance as much as possible. They were told about the actual stimulus and were given the task to maintain a rod, fixed to the backwall of the room, in the horizontal position by means of a potentiometer they held in their hand. The induced body sway was determined by computing the amplitude of the Fourier component at the stimulus frequency in the left-right stabilogram (Bles et al. 1983), taking weight and height of the subjects into account too.

Subjects were also asked to adjust the rod, which was illuminated by LEDs, to their subjective horizon (SH) in the otherwise completely dark room. The mean SH deviation from the real horizon was computed from 6 trials with initial rod tilt of 15° to the left or right.

RESULTS

Routine examination and caloric irrigation With the controls no abnormalities were observed at the routine examination. This was different for the seasickness susceptibles: in two cases cervical pathology could be established, one case showed slightly impaired smooth pursuit and fixation suppression (0 a .2), and once a congenital nystagmus was observed together with a significant asymmetry (60%) in caloric irrigation. Calorisation had to be interrupted in two subjects because of severe vomiting, and once the use of water was ruled out because of an ear drum perforation. The computed labyrinthine predominance of the remaining 36 calorigrams of the seasickness susceptibles is shown in figure 1, together with those of the controls. It is of interest to note that in the group of these 36 seasick a significant (>30%) difference between the left and right labyrinth is observed 5 times.

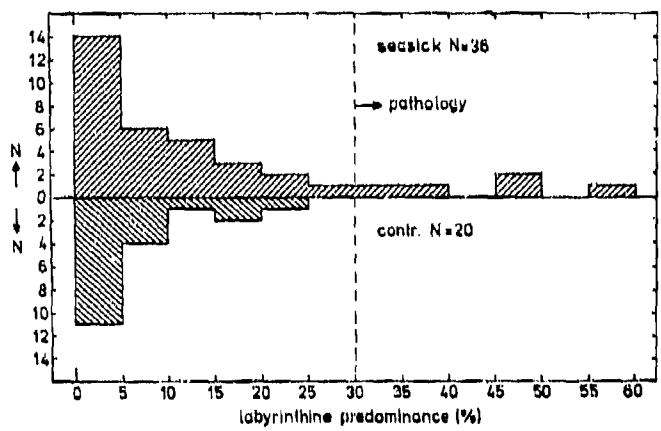

Fig. 1. Histograms showing the distribution of the percentage labyrinthine predominance for the seasickness susceptibles and the controls as obtained with caloric irrigation. A difference of over 30%, found in 5 out of these 36 seasickness susceptibles, is to be considered as pathological.

Cupulometry As described in the introduction, cupulometry was at first not performed routinely with the seasickness susceptibles, and sometimes only the stop from 90°/s was performed. In two cases the cupulometrical procedure had to be interrupted because of vomiting directly after the first stop; in those cases where caloric irrigation had caused severe problems, cupulometry was not performed either, leaving finally 21 complete cupulograms of the seasickness susceptibles. The sensation and nystagmus cupulograms are depicted in figure 2, showing the median values and the 10th and 90th percentiles together with the mean slopes for a large population as determined by Hulk and Jongkees (1948). Apparently, the slope of the cupulogram is the same for both groups, for the sensation as well as for the nystagmus. In figure 3 the computed time constants for the nystagmus decay are shown, indicating no difference between both groups. The largest time constants are, in fact, found with the controls.

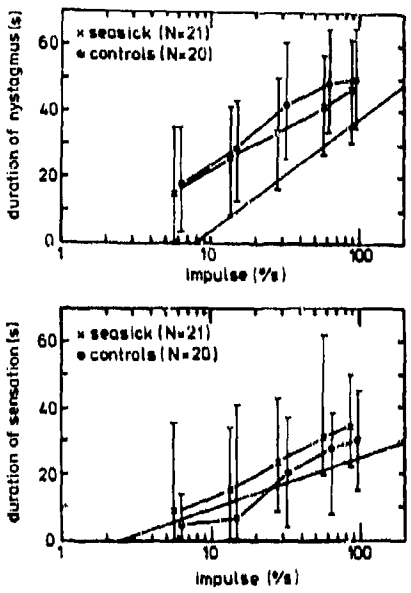

Fig. 2. Sensation and nystagmus cupulogram showing the median values and the 10th and 90th percentiles of the individual data points for the seasickness susceptibles and the controls, together with the slopes for normals as determined by Hulk and Jongkees (1948).

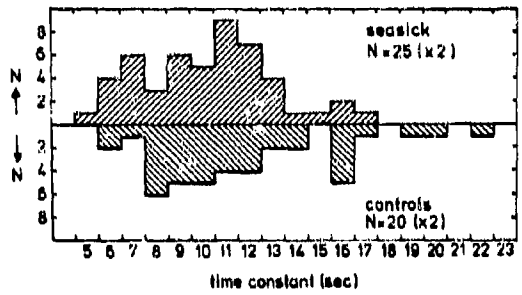

Fig. 3. Histograms showing the distribution for the time constant of the central vestibular system for the seasickness susceptibles and the controls. These time constants were computed from the decay of the vestibular nystagmus after a deceleration of 90°/s² from constant velocity rotation of 90°/s CW and CCW.

As for the shallowing of the slope of the cupulogram after exposure to vestibular stimulation, we had the opportunity to examine 7 controls within a few hours after disembarkation from a nautical expedition, on which they had been troubled on the last days by heavy seas (Beaufort 8-11). The cupulograms (mean values) of these 7 controls obtained before and directly after the expedition are shown in figure 4.

The increase in the duration of the sensation at the weaker impulses is significant, and did result in a shallowing of the cupulogram. One would have expected, however, a decrease at stronger stimuli instead of an increase at weaker stimuli. As for the nystagmus cupulogram, the slope is not influenced by the preceding weeks at sea.

The mean time constant of the nystagmus decay did not decrease either: both shortening and lengthening were observed.

Tilting room All controls (N = 20) and seasickness susceptibles except one (N = 58) were examined in the tilting room. The amplitudes of the induced lateral body sway at 0.015 and 0.2 Hz are shown in figure 5. Obviously, the effect is greatest for the group of seasickness susceptibles, especially for the frequency of 0.2 Hz.

For both groups the SN was within the normal range (2.5° to either side), with two exceptions in the seasickness group of 3.3 and 3.7° tilt; the last one also showing an asymmetry with caloric irrigation of 47%.

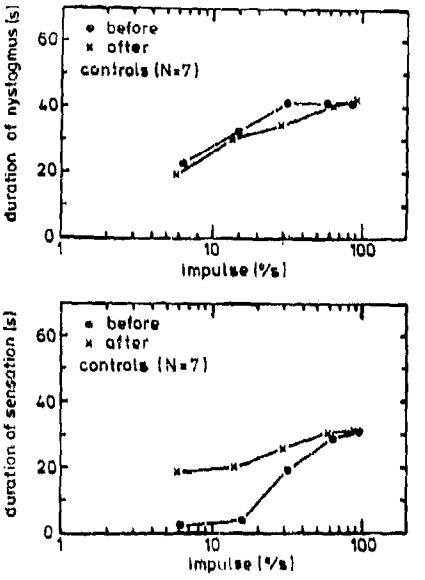

Fig. 4. Sensation and nystagmus cupulograms showing the mean values for 7 controls. Cupulometry was done before and immediately after a nautical expedition.

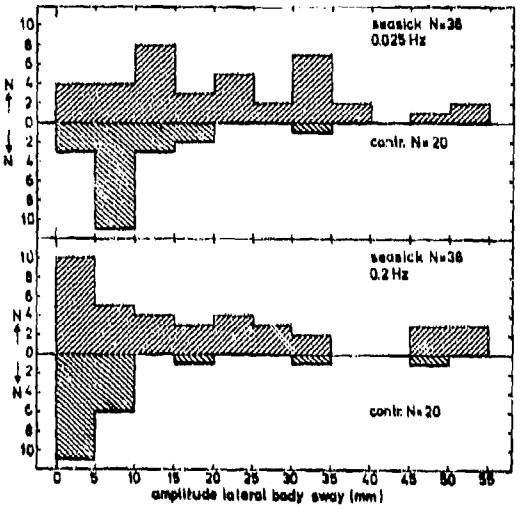

Fig. 5. Histograms showing the distribution of the amplitudes of the visually induced lateral body sway for the seasickness susceptibles and the controls. The tilting room was sinusoidally driven with an amplitude of 5° to either side at 0.025 Hz (upper panel) and at 0.2 Hz (lower panel).

DISCUSSION

Because of the plasticity of the equilibrium system mostly several tests at different times are necessary before a satisfactory ranking of susceptibility to motion sickness can be obtained (e.g. Graybiel and Lackner 1980). The tempting question is whether such a possibility to habituate can be predicted already from the behavior of the parameters used to describe the results of one single test.

Unfortunately, the present study does not reveal a parameter which can be used to predict susceptibility to chronic seasickness with sufficient accuracy. There are, however, some interesting findings in this study. For instance, it was not expected that the routine vestibular examination would reveal such a high

percentage of abnormal values, such as the over 30% of labyrinthine asymmetry found with caloric irrigation in about 15% of the seasickness susceptibles.

It may be assumed that these subjects have adapted to this vestibular imbalance by relying on visual information. Such a shift in favor of the visual system may be useful in normal daily life, but not useful when the visual information is misleading, which may be the case aboard a ship. A similar shift in favor of vision may be observed in patients with cervical pathology or labyrinthine impairment (Bles et al. 1983). Van Maanen (1965) showed that a vestibular imbalance does not necessarily lead to chronic seasickness. Our findings suggest that the incidence of chronic seasickness may be higher in this group than in healthy subjects.

Unfortunately, we could not reproduce the cupulometric findings of de Wit (1953). We found, like Dobie (1974), no difference in slope for seasickness susceptibles and controls. This is difficult to explain, since the attractive ideas about shortening of the time constant have been experimentally verified in several studies on motion sickness for sensation (de Wit 1953; Aschan 1954; Krijger 1954; Preber 1958, and van Maanen 1965) and for nystagmus (Krijger 1954; Preber 1958, and van Maanen 1965). The discrepancies may be due to experimental procedures (which is hard to believe), or perhaps our subjects were 'less' chronic than those in the other studies (which is also hard to believe). Dispersion of the data points may be of interest, since de Wit (personal communication) did not incorporate those cupulograms with to much dispersion. This well-known problem of dispersion is less evident in the determination of the time constant of the nystagmus decay. Unfortunately, here too we were able to demonstrate neither a difference in the magnitude of the time constants for both populations, or a shortening after vestibular constraint. More careful analysis of the nystagmus decay at other impulsive stimuli may perhaps reveal different values for the time constant because of the occasional presence of secondary after-nystagmus at these strong impulses.

Our findings in the tilting room suggest that seasickness susceptibles are more visually oriented than the controls although here, too, a clear overlap exists. As stated already, a similar behavior has been found in patients with a vestibular deficiency (Bles et al. 1983), and in patients suffering from post-concussional dizziness (de Wit and Bles 1975). It is noteworthy that the seasickness susceptibles also show postural imbalance at the lower stimulus frequencies, whereas the vestibular patients usually learn to adapt at these frequencies, only showing imbalance in the higher frequency region for a longer period of time (Bles et al. 1983).

The findings in the tilting room are interesting from the point of view that in this test the otoliths should be involved more than the semicircular canals. Moreover, the test condition resembles the real conditions better than cupulometry does. It should be noticed that the earlier investigators were well aware of the fact that they used cupulometry in spite of the fact that seasickness at that time was thought to be due to overstimulation of the otolithic system (cf. Reason and Brand 1975, p. 189); de Wit (1953, p. 31) noticed this duality but propagated cupulometry, while he did not succeed in finding a reliable test for examining the otolith apparatus at that time.

As a conclusion of this preliminary analysis it may be stated that 1) the slope of the sensation cupulogram is not a good parameter for predicting seasickness susceptibility, 2) a vestibular imbalance may enhance susceptibility to seasickness or, more generally, motion sickness, and 3) interactions of the otolithic and visual systems may be organized in a different way in seasickness susceptibles. The fact that examination with the tilting room is essentially an examination on sensory interactions, whereas current concepts about seasickness are mostly in terms of sensory rearrangement (Reason and Brand 1975), makes it worthwhile to proceed with the research on seasickness susceptibility in sensory interactions. Moreover, motion sickness is not only due to visual-vestibular interactions but depends also on somatosensory information (Bles 1981); it must be borne in mind that a large lateral postural sway may also be due to non-adequate somatosensory information.

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DISCUSSION

CLAUSSEN: I was very interested in seeing your first slide and the slide where you showed the caloric test evaluation from the susceptible persons to motion sickness and the normals. You said, there were more vestibular asymmetries in the pathological cases. How did you find that these were vestibular imbalances and not brain stem imbalances? Does your formula allow this discrimination?

BLES: We cannot be completely sure but we have at least no indiction that there is a brain stem malfunction in these patients. The routine ENG examination tests like OKN, smooth pursuit, and fixation suppression provided no evidence for asymmetric lesions in brain stem or cerebellum.

JONES: In the Air Force, we are not greatly concerned with seasickness, but I was wondering if there might be any difference between vestibular ocular conflict among people who work inside the ship and those who work on the deck?

BLES: As far as I've been told by these subjects, there is quite a difference being in the ship or on the deck. Mostly the symptms are less when they are up on the deck.

VON BAUMGARTEN: Bilateral asymmetries could play a role in space motion sickness as well.

BLES: Thank you for your comment.

VON GIERKE: Did you consider the inverse test having the room stationary and oscillating the subject's platform.

BLES: Quantitative analysis with stabilometry is very difficult if not impossible because of the induced complex body movements when the platform is tilting. Preliminary experiments with the platform coupled to the tilting room showed that healthy subjects have severe problems in keeping the upright position and that all patients had to be supported which means that the test did not have a diagnostic value anymore. Semistatic tests in which the subjects control the tilt of the platform seem to be more promising. The analysis of these data is not yet completed.

Laboratory Tests of Motion Sickness Susceptibility

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SUMMARY

This paper reviews some of the laboratory tests of motion sickness susceptibility that have been evaluated over the years at the Naval Aerospace Medical Research Laboratory in Pensacola. The discussion focuses on 1) the procedures used to rate the extent of sickness; 2) how the intent of testing influences the outcome; 3) the problem of measuring adaptative potential; 4) aftereffects; and 5) the relationship of these tests to success in flight. Individual tests which are discussed include: Brief Vestibular Disorientation Test, Coriolis Sickness Susceptibility Test, Sudden-stop Vestibulovisual Test, Tilted-Axis Rotation Test, and the Visual/Vestibular Interaction Test.

INTRODUCTION

This paper is a review of five laboratory tests of motion sickness susceptibility that have been evaluated over the years at the Naval Aerospace Medical Research Laboratory in Pensacola. These tests, involving Coriolis stimuli, off-vertical rotation, visual/vestibular interactions, were developed with the objective of predicting individual susceptibility to airsickness and space sickness. However, there is much work left undone and this short review reflects some thoughts on both past accomplishments and future directions.

CORIOLIS (CROSS-COUPLED ANGULAR ACCELERATION) STIMULUS TESTS

Brief Vestibular Disorientation Test (1, 2, 3, 4, 5, 8, 9, 12, 13)

More subjects have taken this test than probably any other laboratory test of motion susceptibility. The Brief Vestibular Disorientation Test (BVDT) involves passively rotating an erectly seated S, with eyes closed, at a constant 90°/s. After 30 s at constant velocity the S makes 45° head movements (Fig. 1) every 30 s according to the following order: head right, upright, head left, upright, head right, upright, head left, upright, head forward, upright. The total time of rotation is 5 1/2 minutes. Following the BVDT each S completes a brief self-rate questionnaire concerning his reaction to the test, and is rated by observers for signs of motion sickness.

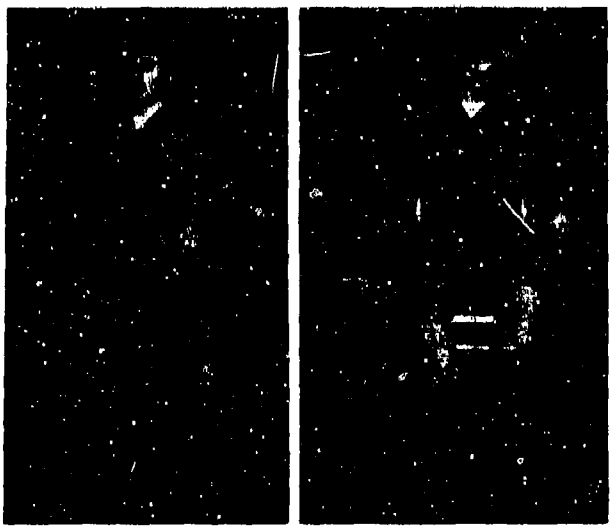


Figure 1
(a) (b)
Brief Vestibular Disorientation Device
Subject's head in the upright (a) and left-tilted (b) positions

Data (13) from a group of 552 student Naval Flight Officers (non-pilot category) is shown in Fig. 2. It is clear from this figure that rater (observer), self-rate, and follow-up (aftereffect) scores are strongly skewed toward high scores (high susceptibility). Due to the nature of this type of distribution this test may be useful in detect-

ing extremely susceptible individuals but is probably not useful in establishing even a rank order among most individuals of average susceptibility. This particular group of students is the subject of the next paper which traces their inflight incidence of airsickness through three phases of training. As you will see, their inflight airsickness does not correlate highly with their BVDT scores. Some of this low correlation is likely due to the skewed BVDT distributions and perhaps a different statistical approach (e.g., point biserial analysis) would improve these correlations. In one of the initial studies describing development of this test Ambler and Guedry (5) found that the BVDT correlated significantly with later separation from flight training for any reason (.165), tension or airsickness (.272), airsickness only (.413).

The low correlation with inflight airsickness is in part due to a) the brief one-shot test exposure which lacks the ability to estimate adaptative potential, and b) the somewhat mild stimulus which produces a highly skewed distribution of scores designed primarily to detect the extreme reactor.

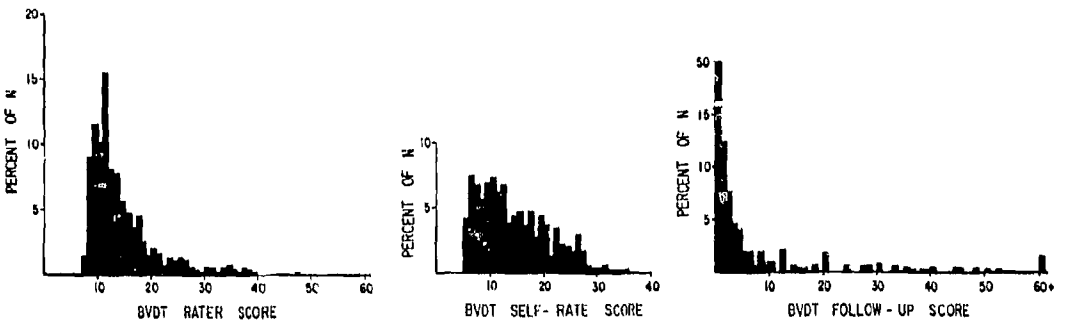


Figure 2

BVDT rater, self-rate, and follow-up score distributions (N=552)

Coriolis Sickness Susceptibility Index (CSSI) (14, 15, 16, 17)

Prior to discussing the Coriolis Susceptibility Index (CSSI), I want to mention an earlier test (the Dial Test) which had some influence in CSSI development and if it had received additional attention, could have evolved as a major test in this area.

The Dial Test (10) was an attempt to force specific head and body movements (Coriolis stimuli) and to relate a measure of performance to this stimulus/response complex. Figure 3 shows the response sequence required during rotation (7.5 rpm) on the Slow Rotation Room. In the initial report describing the Dial Test, Kennedy and Graybiel compared three groups of subjects: 100 incoming flight students, 40 experienced aviator pre-flight instructors, and 25 test pilots. The test produced sickness in 70, 30, and 5 percent of the respective groups (vomiting in 10, 0, and 0).

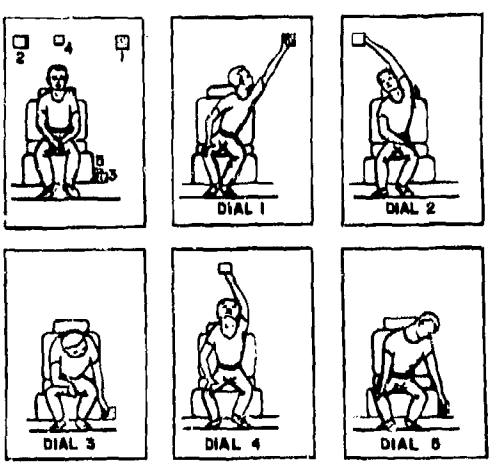


Figure 3

Dial Test -- Dial setting sequence

One difficulty with the Dial Test, and other procedures being used at the time, was the great range of symptom expression and the lack of a method to grade or rate the elicited motion sickness symptoms. Some investigators were using vomiting as an endpoint; however, this proved unacceptable to both subjects and observers particularly with repeated exposures. To remedy this situation Dr. Graybiel devised a method for grading the severity of motion sickness (7). This method for grading symptoms underwent several refinements and was combined with a set of head and body movements (Coriolis stimuli) to produce "a provocative test for grading susceptibility to motion sickness yielding a single numerical score." This test procedure has been generally called the Coriolis Susceptibility Index or CSSI (pronounced sissy).

The CSSI test required a seated subject to make 90° head movements in four quadrants according to the following order: front, upright, pause; right, upright, pause; back, upright, pause; left, upright, pause; front, upright, rest (Fig. 4). The chair velocity was determined by several preliminary tests and questionnaires and was limited to one of the following constant velocities (2.5, 5, 7.5, 10, 12.5, 15, 20, 25, 30 rpm). The CSSI scores were computed

by multiplying the number of head movements at the testing rpm by a factor E which was the average relative stimulus effect of a single head movement. In a separate study, Miller and Graybiel (17) found that the E factor could be expressed as a linear function of chair velocity (log x log) and that the duration of the test was usually less than 15 minutes. Fig. 5 shows a distribution of CSSI scores for 250 normal subjects (aviation related personnel). Inspection of the distribution reveals a strong skew toward high scores. Remember that with this test a low CSSI score indicates high motion susceptibility whereas a high CSSI score indicates considerable immunity to motion sickness. The distribution of scores on this test seems to suggest that it would be best suited for detecting individuals who are relatively resistant to motion sickness.

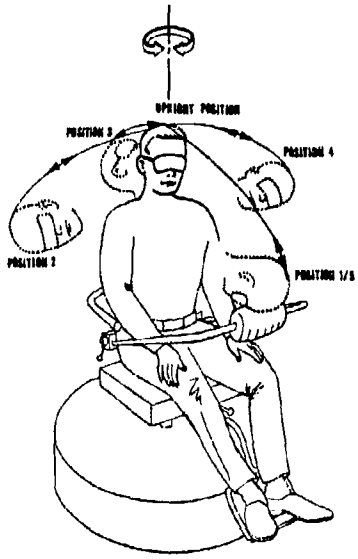


Figure 4

Diagram of standardized procedure for making each sequence of head movements to and from tilt position 1 through 5 during chair rotation

To summarize these two approaches to Coriolis stimulus testing: the BVDT involves rating the degree of symptom expression to a nonvariable physical stimulus set (10 head movements over 5 1/2 minutes), the CSSI involves always taking the subject to a selected symptom level and then rating physical stimulus on the basis of its average vestibular stress value (E factor times the number of head movements). As currently designed each procedure results in a strongly skewed distribution of scores. The BVDT may better detect an extremely susceptible individual and the CSSI may better detect an extremely resistant individual. Neither test attempts to provide a measure of adaptative potential. Adaptative potential is a factor that will have to be measured if we are to improve these rating methods; however, the problem is how to do this both accurately and with a short period of testing. It is my opinion that the two or three repeated exposures will not provide an adequate estimate of adaptability. However, a second exposure to a cross-coupled stimulus will probably yield a better estimate of current susceptibility since it will not be contaminated by the unexpectedness of the experienced motion (occasionally a 'fear' reaction).

VISUAL-VESTIBULAR CONFLICT TESTS

Visual-Vestibular Interaction Test (12, 13, 19)

In the Visual-Vestibular Interaction Test (VVIT) the erectly seated S is passively and sinusoidally oscillated at 0.02 Hz with a peak angular velocity of $\pm$ 155°/s while he attempts to retrieve data from a visual display. The axis

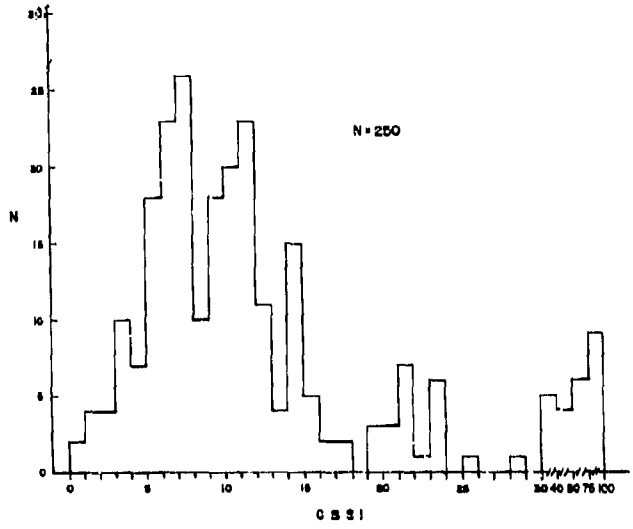


Figure 5

Distribution of Coriolis Sickness Susceptibility Index (CSSI) among 250 normal subjects.

of rotation is vertical and the S is encapsulated within a chamber (Fig. 6) which remains completely dark until presentation of the visual display (Fig. 7). Subjects are instructed to use the coordinate system to find the corresponding digit embedded within the matrix. Once the digit is located, the S reports it along with the next two digits below it. Coordinates are issued via a tape recording every 7 s, with a total of 42 commands. Following the test each student completes a brief questionnaire concerning his reaction to the test and two observers rate the magnitude of overt motion sickness signs. The rater, self-rate and follow-up scores on this test are almost identical to those used with the previously mentioned BVDT procedure. The resulting distributions are shown in Figure 8 and again they are skewed toward higher scores (stronger signs/higher susceptibility).

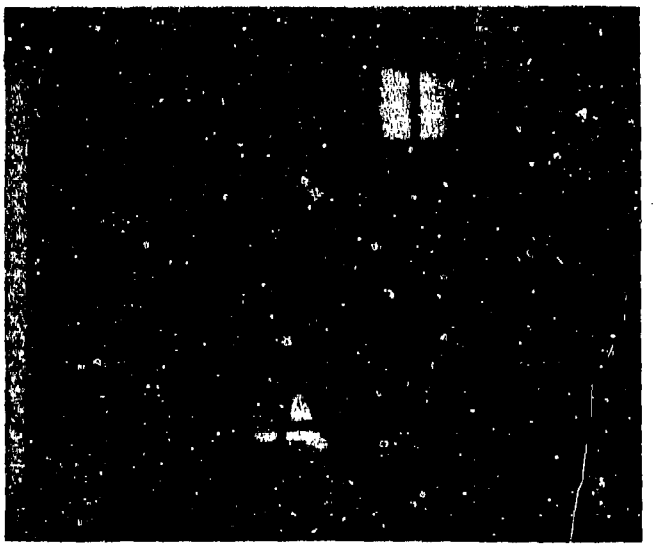


Figure 6

Visual-Vestibular Interaction Test Device
During testing the black shroud completely occluded the subject's external visual reference.

	A	B	C	D	E	F	G	H	I	J	K	L
1	7	1	1	8	2	4	3	1	6	6	9	4
2	6	4	4	2	4	3	1	8	9	7	4	1
3	2	2	3	4	7	8	6	5	1	4	8	5
4	9	9	5	4	6	2	7	3	8	3	7	9
5	8	1	4	3	6	5	7	7	1	4	2	6
6	7	4	7	1	8	1	9	6	3	2	8	5
7	1	7	6	7	6	4	9	5	4	8	3	7
8	7	1	3	3	4	8	9	4	2	5	6	8
9	6	2	1	6	7	3	8	9	7	2	6	6
10	1	7	5	9	9	1	5	6	6	3	5	8
11	9	3	6	7	3	2	2	8	4	5	2	5
12	2	7	8	2	9	9	3	4	1	5	1	7

Figure 7

VV1T Visual Display

It may be interesting to note that during development of this test it was found that the display complexity played an important role in establishing the nauseogenic quality of the test. For instance, using the same physical vestibular stimulus, a 3 digit display was typically not nauseogenic whereas a 7 digit display was somewhat nauseogenic and the 12 x 12 matrix was quite nauseogenic (12% abort the 5 minute test). One would suspect that this test would be useful in detecting those individuals who get motion sick while reading in a moving vehicle (e.g., navigation duties); however, it has a generally low correlation with reported inflight airsickness. I should note that with repeated exposures (ten sessions) I have personally adapted fairly rapidly to this stimulus situation whereas I have had only limited success adapting to a cross-coupled stimulus with much more exposure. I am particularly enthusiastic about this procedure since it offers a situation where the rate and/or severity of sickness can apparently be altered by changing a static display without necessitating changes in the motion condition - in other words, we may be able to change display dynamics; however, we probably won't be able to change aerodynamics.

The Sudden-Stop Vestibulovisual Test (6, 11)

The Sudden-Stop Vestibulovigual (SSV) test involves accelerating ($15^{\circ}/sec^{2}$) a subject to a constant velocity ($300^{\circ}/sec$), holding at that velocity for 30 sec and then rapidly decelerating (1.5 sec) to a stop followed by a 30 sec rest. This basic sequence is repeated 20 times with eyes blindfolded then an additional 20 times with eyes open and

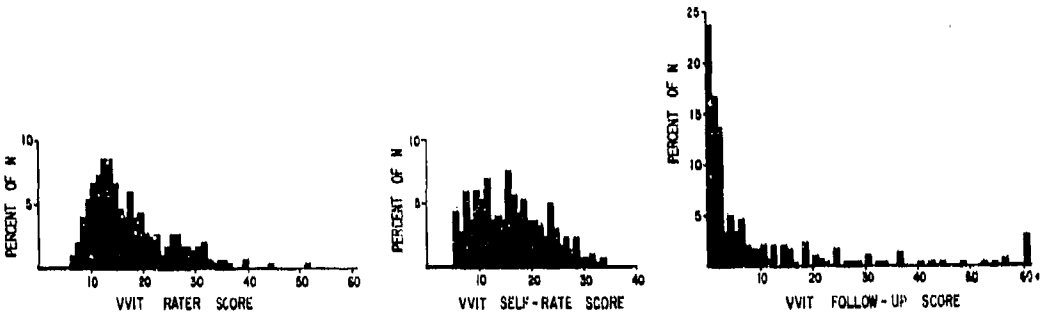


Figure 8

VVIT rater, self-rate, and follow-up score distributions

then if necessary another 20 times again with eyes open, but using the opposite direction of rotation. In the eyes open condition the subject views a dark cylindrical surround which has 6 vertical white stripes (Fig. 9). Each individual continues exposure until they reach the slight "nausea" endpoint as defined by the diagnostic grading procedure developed by Graybiel, et al. (7,14,16,17). When this point is reached each subject receives a score which is one-half the number of stops with eyes covered plus the number of stops with eyes open plus twice the number of stops after the direction of rotation has been reversed. Since this procedure has only recently evolved, a normative data base on a large population is not yet available. When more data are collected with this procedure, the arbitrarily assigned weights for the different stop procedures can be better evaluated. This test also seems to have a novel (possibly fear) component which is present on first exposure (7/14 aborted during eyes closed) but which is less evident on the second exposure (1/14 aborted during eyes closed).

Because we normally function with our eyes open, particularly in motion situations, I propose that continued work on visual-vestibular interaction tests will prove to be the best predictors of motion sickness in most human performance systems.

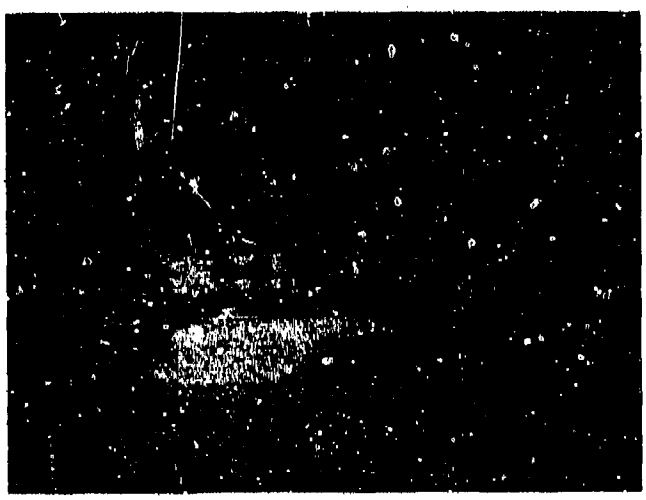


Figure 9

Test chamber and rotator used for the Sudden-stop Vestibulovisual Test

OFF-VERTICAL TESTS

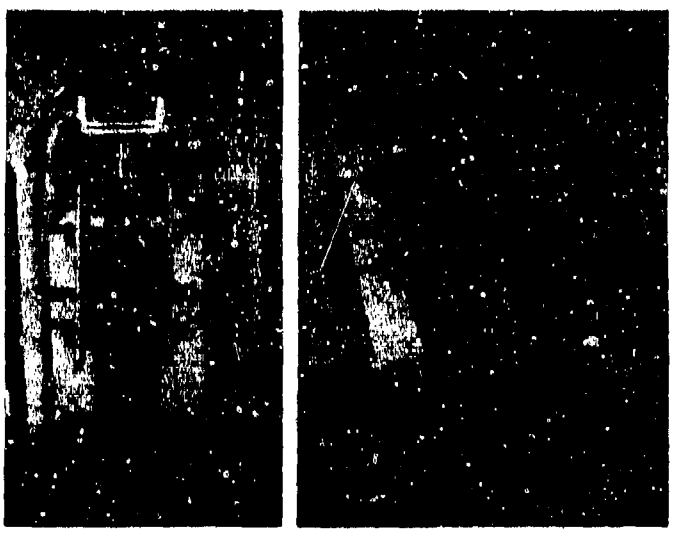
Tilted Axis Rotation Test (TART) (12)

In the TART, the erectly standing S is securely fastened in a litter device capable of rotation about an axis that can be tilted relative to gravity (Fig. 10). The S is blindfolded and tested in a darkened room. In the first trial, the S is accelerated at $25^{o}/s^{2}$ in a clockwise (CW) direction with the axis of rotation vertical, i.e., aligned with gravity. The acceleration is terminated upon reaching $60^{o}/s$ (10 rpm), and this constant velocity was maintained for 90 s and then the S is decelerated at $25^{o}/s^{2}$ to a stop. The second trial is identical to the first, with the exception that rotation is in a counterclockwise (CCW) direction. In the third and fourth trials the axis of rotation

is tilted 30° off-vertical (Fig. 10) and, with the axis remaining tilted, the rotation velocities and accelerations described in Trials 1 and 2 are repeated. The S is always stopped in the nose-up position. In the fifth and sixth trials the S remains tilted at 30° off-vertical and again is accelerated at 25°/s². A constant velocity of 102°/s (17 rpm) is used for this pair of trials. The interval between trials is approximately 5 minutes. Following the test each subject completes a brief self-rate questionnaire concerning his reaction to the test and is rated by observers for signs of motion sickness. The rater and self-rate procedures are identical to those used in the previously mentioned BVDT and VVIT. Since it is not uncommon for subjects to terminate the TART prior to its completion, the rater and self-rate scores were weighted with respect to the number of trials completed. Rater and self-rate scores of individuals completing six trials were multiplied by 0.65, since approximately 65 percent of a random unselected group of subjects completed six trials. In a similar manner the scores of individuals completing five trials were multiplied by 0.73, four trials were multiplied by 0.90, and three trials were multiplied by 0.98. Subjects who were unable to complete an off-vertical trial (third trial) were assigned their raw scores. This method of weighting rater and self-rate scores on the TART is arbitrary and may need future revision. Data distributions for an airsick group (N=47) and an unselected or 'comparison' group (N=80) are shown in Fig. 11.

Another off-vertical procedure (18) has been used to generate motion sickness symptomology at Pensacola; however, it has not been administered to a large normative population. Miller and Graybiel (1970) rotated a seated subject at one of several selected velocities (2.5, 5, 10, 15, 20, 25, 30, 40, 45 rpm) and after 60 seconds tilted the rotating chair at 5°/sec to a tilt position selected from among 2.5, 5, 7.5, 10, 15, 20, or 25 degrees (Fig. 12). The rotation continued for one hour or until moderate malaise was elicited. With the limited number of individuals tested with this procedure, it appears that the test duration varies between 5 and 20 minutes depending on the extent of the off-vertical axis.

It may be interesting to note that the first procedure (TART) appears to be more nauseogenic than the second procedure. This difference is most likely due to the fact that the second procedure uses constant rotation for up to one hour to elicit symptoms whereas the TART uses a short series of accelerations/decelerations. In a blindfolded subject, the strongest otolith-canal conflict would be associated with decelerations and therefore the increased number of decelerations in the TART probably accounts for its increased nauseogenic value. In general, these off-vertical procedures do not seem to elicit a strong 'fear' reaction on initial exposure.



(a) (b)

Figure 10

Tilted-Axis Rotation Device: (a) vertical position; (b) 30 degrees off-vertical

GENERAL DISCUSSION

Desirable traits for a motion sickness susceptibility test

a. Any laboratory test of motion sickness susceptibility will be judged primarily on its ability to generalize to other exposure situations. The premise that motion sickness is a personal trait which should basically generalize across motion conditions is a most important concept and is the basis of much of our testing although there are questions about idiosyncratic susceptibility to particular motion stimuli. With a group of unselected subjects, correlations between the BVDT, VVIT and TART were fairly high (r ≈ 0.5) and statistically significant (12). However, with airsick referrals the intertest correlations (rater and self-rate) were low and generally not significant. Because these tests are

intentionally fairly mild and designed to detect susceptible responders they lack resolution among airsick referrals. It is possible that resolution among airsick referrals could be improved by adjusting the difficulty of each test in an effort to elicit measurable reactions which would better resemble a normal distribution. In this regard, taking every individual to a selected symptom level might improve intertest correlations.

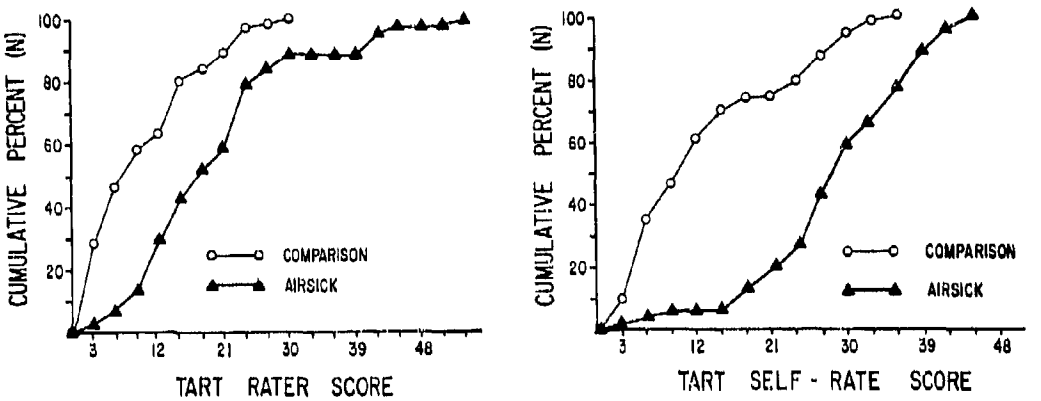


Figure 11

Cumulative percent distributions for TART rater and self-rate scores.

In general, all of these tests have had fairly low correlations with field conditions. How can prediction or generalizability be improved? For a mass testing situation (i.e., all pilot candidates), one would have to consider reducing the false positive predictions and therefore an even milder test may better identify the really extreme responder. Although the incidence of false negative predictions would be high in this case, if the identification of positive cases were always correct, then selection personnel would surely be quite interested. If testing is limited to small groups or individuals, then test development should probably focus on approximating the field condition (both stimuli and duration of exposure) as closely as possible which ideally will reduce the generalizability problem. Accuracy of testing might also be improved by developing objective physiological monitoring of symptoms instead of relying on observer ratings or self-rate reports. It is my opinion that although these systems would be nice, they are not yet needed. Although the methods of subjective observation are not technologically impressive, they are more than adequate for identifying the <u>major</u> sickness symptoms which tend to affect performance and motivation.

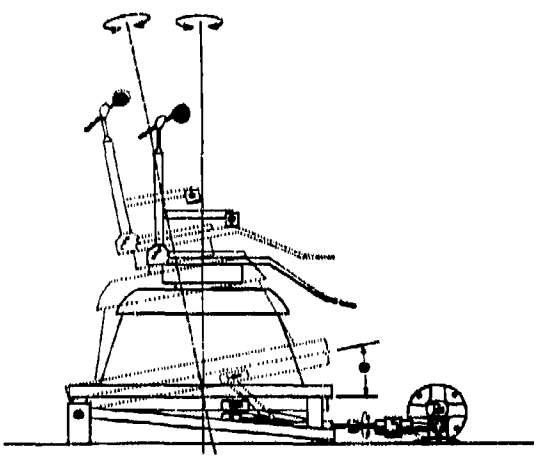


Figure 12

Diagram of apparatus used in off-vertical rotation test.

b. A laboratory test of motion sickness susceptibility needs a measure of adaptative potential. In most cases where these tests have been administered to the same subjects on a repeated basis the testing objective was not to measure adaptative potential but to measure test-retest reliability or to serve as the basis of evaluating drug effectiveness etc. and the intervals between exposures have been long in an effort to minimize adaptative shifts. The time involved in repeat exposures is for many users unacceptable and if for

no other reason, I would guess that this approach to measuring adaptative potential will not survive. A quick measure of adaptative capacity might ultimately be obtained by measuring a CNS perceptual aftereffect which superficially may not seem directly related to the vestibular system (i.e., visual spiral aftereffect); however, this will not be easily accomplished.

Many people overlook the possibility of estimating adaptative potential by measuring the magnitude and duration of aftereffects during recovery from a single exposure. One problem with this approach is its dependence on a truthful subject report. I believe more effort will be made to measure aftereffects particularly because of the numerous reports of sickness and aftereffects following flight simulator exposure. This area will also receive attention due to the increasing concern for the protection of our human subjects once they depart the testing environs.

c. A third trait that is desirable for a laboratory test of motion sickness is a short administration time. In situations where large numbers of flight candidates are being tested, 20-25 individuals must be tested in no more than 3-4 hours. This factor loses importance in situations limited to small groups or individual subjects.

d. Ideally the perfect motion sickness susceptibility test would not need specialized equipment or highly trained personnel - and thus the cost of administration would remain low. This is the least important factor and could be overlooked if the other factors can be maximized.

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DISCLAIMER

Opinions or conclusions contained in this report are those of the authors and do not necessarily reflect the views or endorsement of the Navy Department.

DISCUSSION

KUEHN: I'm interested in your remarks on the complexity of a target and proclivity to motion sickness. I've always thought that distraction of the type you indicated reduced incidence of motion sickness, yet you indicate that it might be worse. This has implications for space motion sickness. Perhaps some of our space protocols should be more simplified than they are.

LENTZ: Yes, it seems crucial. As you increase display complexity it appears that you also increase motion sickness incidence (example 3 digits - 7 digits - 12 x 12 matrix).

JONES: Our experience has been that some fliers getting airsick try to ignore their premonitory symptoms and thus find themselves..rather suddenly vomiting. This is the antithesis of what our lab was teaching, that they should attend to their symptoms and diminish them by relaxation procedures. Could this effect at least partially account for your finding that complexity of visual task was positively associated with motion sickness?

LENTZ: In many cases a susceptible individual concentrating on performing the matrix task without error and having no error still exhibited very strong nauseogenic responses. In sum, just concentrating on the task doesn't seem to alleviate the sickness much.

MONEY: I understand that the Israeli airforce uses a technique whereby early in the selection process the candidate prospects are put into a transport aircraft.

LENTZ: If we look at people who are not sick and I'm talking about the P3 aircraft now, I don't have the information for all of the different squadrons, then only 17% of those are getting sick when they get into the fleet readiness squadron in the P3 aircraft.

RESULTS OF A LONGITUDINAL STUDY OF AIRSICKNESS INCIDENCE DURING NAVAL FLIGHT OFFICER TRAINING

by
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SUMMARY

This paper outlines the results of a longitudinal study of airsickness in a large sample population of Naval Flight Officers (NFOs) being trained to perform various nonpilot flight duties prior to assignment to operational fleet squadrons. The study has concentrated on the acquisition of airsickness data on an individual student basis as training progressed from the basic/primary level through the advanced/secondary level to the fleet readiness squadron phase for each of the major NFO training pipelines. The primary objectives of the study were to define the incidence of airsickness in each of the training squadrons and to identify differences in the motion stress exposure associated with the different pipelines that can affect decisions on the initial selection and assignment of NFO candidates. A secondary objective was to relate the inflight airsickness data to the results of several short tests of motion reactivity given to a segment of the study population prior to their beginning flight training.

INTRODUCTION

This paper is a summary of a series of research reports (7-13) dealing with a longitudinal study of airsickness in Naval Flight Officer (NFO) students being trained to perform specialized nonpilot flight duties aboard various fleet aircraft. As a matter of background the longitudinal study originated as a result of numerous airsickness problems and questions that were directed to this activity by training command personnel responsible for delivering qualified NFOs to the fleet, by flight surgeons responsible for the medical management of naval aviation aircrews, and by career naval aviators and flight officers experiencing chronic airsickness difficulties during performance of their fleet flight duties. Training command personnel raised questions concerning the overall cost of the airsickness risk to the NFO training program. Specific problems included degraded flight performance of airsick students, the need to repeat hops when performance was inadequate, loss of personnel and training time due to airsickness-related attrition, the usage of airsickness medication over an extended period of the training program, and the occasional graduation of airsick NFO students who were able to complete the training program but could not perform adequately in the fleet. Concern was also expressed about the need for laboratory tests to medically screen airsickness susceptibles early in the training program to reduce the costs of mid- or late-term attrition.

Similar questions were raised by flight surgeons who were dealing with airsick flight personnel. They were interested in more specific knowledge of the profile of airsickness during NFO training and on into the fleet; the basic causes of airsickness; the probability of eventual adaptation to flight given by a particular history of motion sickness; the use of medication, especially with provocative hops, to assist in the adjustment period; and the probability of recurrence of motion sickness with new fleet assignments. They also were interested in the availability of preflight laboratory tests that might identify individuals in need of early treatment and/or alternative naval service, and in special clinical tests that would aid in a comprehensive evaluation of specific airsick cases. In addition, this activity was often contacted directly by fleet aircrew suffering repeated airsickness difficulties who raised questions similar to those of the flight surgeons.

Airsickness problems have long existed in military aviation and are neither new nor unique to the NFO population. During World War II, Hemingway (6) reviewed numerous field studies conducted by the military which indicated a high incidence of airsickness during various phases of flight training for both pilot and nonpilot aircrew groups. In this and later reviews (1, 18, 20) it was shown that though the pilot and nonpilot groups were both at risk relative to airsickness, the latter group generally suffered the highest incidence rate. Recognition of airsickness as a continuing biomedical problem is also marked by efforts that have been taken to develop desensitization procedures for susceptible military aircrew (2, 3, 4, 5, 14, 16).

Since few operational data were available on airsickness problem during NFO training, a first step in addressing some of the above stated questions and problems involved describing the incidence and severity of airsickness normally experienced by the NFO population. To this end, a longitudinal study of airsickness in a large sample NFO population was initiated to follow students through the basic (primary level), advanced (secondary level), and fleet readiness squadrons comprising the NFO training syllabus. The primary objectives of the study were to define the relative magnitude of the airsickness problem during each phase of training on an individual squadron basis; and to identify differences in motion stress exposure associated with the different pipelines that can affect decisions on the initial selection and assignment of flight personnel. The study also gained a secondary objective through the cooperation of the training command who allowed a large segment of the NFO study population to be exposed on a

one-time noninterference basis to several short laboratory tests of motion sickness reactivity prior to beginning flight training. The objective here was to obtain some insight into the avenues that might be followed in the future to develop and validate tests of motion reactivity that will have high predictive value in the early identification of airsick susceptible individuals. In this respect, the inflight airsickness data collected during the longitudinal study were intended to serve the dual function of defining the magnitude of the NFO airsickness problem and establishing validation criteria for measurement of the relative effectiveness of candidate motion reactivity tests.

PROCEDURE

A block diagram of the NFO training pipelines included in the study is shown in Figure 1. All NFO candidates receive their basic flight training in Training Squadron TEN (VT10) prior to being selectively assigned to one of four advanced pipelines that lead to type-specific training in 14 different fleet readiness squadrons. Advanced training in the Mather Air Force Base (MAFB) pipeline leads to Fleet Readiness Squadron (FRS) training in the P-3 aircraft. In Training Squadron EIGHTY SIX (VT86), students who follow the Advanced Jet Navigation (AJN) pipeline receive FRS training in attack/antisubmarine aircraft including the A-6, EA-6, and the S-3; while those who follow the Radar Intercept Officer (RIO) pipeline are assigned to F-4 or F-14 FRS fighter squadrons. Those students that follow the Airborne Tactical Data Systems (ATDS) pipeline receive FRS training in E-2 squadrons. Upon completion of FRS training, the graduate NFOs are generally assigned to an operational squadron for fleet duty.

The study was initiated in VT10 where the incidence and severity of airsickness that occurred on each hop by each participating student was documented by means of a questionnaire (7) with separate sections for the student and instructor evaluations of the students airsickness reactions on the given hop. In general, each hop (a formally defined component of the squadron flight syllabus with a specific training mission or objective) involved a single flight of the student. However, there were rare occasions when a student flew two different hops on a single flight. On the student component of the questionnaire, the students were asked to rate their airsickness symptoms as not present, mild, moderate, or severe with these responses scored (weighted) on an integer scale of 0 to 3, respectively. A second question asked the students to provide corresponding scaled judgments for the amount of inflight performance degradation that they may have experienced as a result of airsickness. A third question addressed the number of times vomiting occurred on a given hop with zero, one, two, or three or more vomiting incidents being scored on a 0 to 3 scale, respectively. The instructor questionnaire required the instructor to make similar judgments on the same three items.

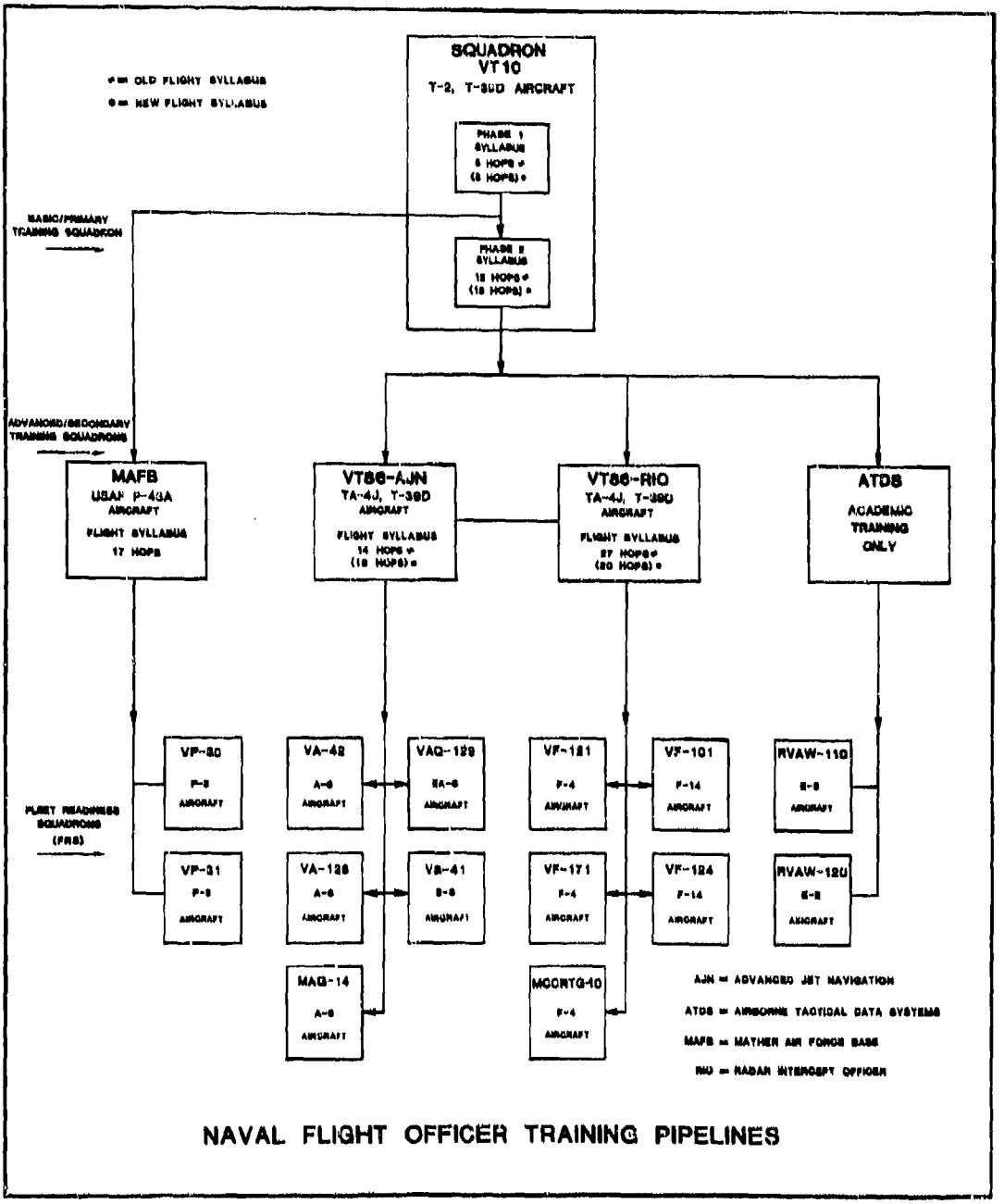
The same student/instructor questionnaire used to evaluate the incidence and severity of airsickness during basic training in VT10 was also used in the VT86-AJN and VT86-RIO advanced training pipelines. For the MAFB pipeline and for all of the individual fleet readiness squadrons, a modified questionnaire of near identical form was utilized to collect corresponding data on an individual hop basis with the exception that only the students rated the incidence and magnitude of their airsickness experiences. Throughout the course of the study, emphasis was placed on ensuring the participating students that their questionaire responses would be treated in confidential fashion.

As outlined in the first report (7) of the longitudinal study, the questionnaire responses were then computer-stored on an individual- student/individual-hop basis for each squadron involved in the study. The same computer file structure was also used to store the results of several laboratory-conducted motion reactivity tests given to a large segment of the NFO study population prior to their beginning flight training in VT10. These data included results from a motion sickness history questionnaire (19); a Brief Vestibular Disorientation Test (15); and a Visual/Vestibular Interaction Test (15).

As the students progressed through the basic, advanced, and FRS phases of NFO training, the computer-stored questionnaire data were extracted on an individual student basis and used to calculate unweighted and weighted indices that could be used to gauge individual susceptibility to airsickness during each phase of training. The function of these indices was to allow comparisons to be made among different squadrons and among different training pipelines. In addition, they served the further function of relating an individual's airsickness during basic training with subsequent airsickness in advanced and fleet readiness squadrons. For each student unweighted flight indices were calculated for the airsickness, vomiting, and performance degradation elements of the questionnaire as follows:

$$\text{UNWEIGHTED FLIGHT INDEX} = \frac{\text{Number of Hops Response Experienced}}{\text{Total Number of Hops Flown}} \times 100$$

where no weight was given to the severity of the response; i.e., attention was given only to the fact that a response such as airsickness occurred on a flight without regard to its severity. Accordingly, the unweighted indices simply represent the percentage of the total hops flown in a given squadron where the denoted response such as airsickness or vomiting occurred.



NAVAL FLIGHT OFFICER TRAINING PIPELINES

FIGURE I

Block diagram showing the major training pipelines followed by Naval Flight Officer (NFO) students as they progress through the NFO flight program. All students receive basic (primary level) flight training in Training Squadron TEN (VT10) and then are assigned to one of four advanced (secondary level) squadrons prior to receiving type-specific training in one of fourteen Fleet Readiness Squadrons (FRS). The MAFB pipeline leads to FRS training in the P-3 aircraft; the VT86-AJN attack pipeline to A-6, EA-6, and S-3 FRS training; the VT86-RIO fighter pipeline to F-4 and F-14 FRS training; and the ATDS pipeline to E-2 FRS training.

The weighted indices were derived similarly with the exception that the 0 through 3 integer values used to scale the magnitude of a given questionnaire item on a given hop was incorporated into the calculations. For example, if a student reported that on a given hop he was not airsick, he would be assigned a response rating of 0; if he reported experiencing mild, moderate, or severe airsickness, he would receive a response rating of 1, 2, or 3, respectively, for that particular hop. The response ratings received on each hop flown in a given squadron were then summed and used to calculate a weighted index that was normalized to have a maximum value of 100 as follows:

$$\text{WEIGHTED FLIGHT INDEX} = \frac{\text{Sum (Individual Flight Response Rating)} \times \frac{100}{3}}{\text{Total Number of Hops Flown}}$$

To illustrate, a student who reported being mildly airsick on half of his hops and not airsick on the remainder would have an unweighted airsickness index of 50.0 and a weighted index of 16.7, while a student who was severely airsick on half of his hops and not airsick on the remainder would also have an unweighted index of 50.0 but his weighted index would rise to 50.0. The instructor questionnaire data were also used to separately calculate instructor-based unweighted and weighted flight indices for each individual student.

RESULTS AND DISCUSSION

Over the course of the longitudinal study, airsickness data were collected on a total of 28,383 hops flown by 796 students as they progressed through the NFO training program. A summary of the incidence of airsickness, vomiting, and inflight performance degradation due to airsickness as reported by the participating students is presented in Table I for each phase of training. In this table, incidence is expressed as the percentage of the total hops flown in a given phase where the denoted airsickness-related response was reported to have occurred without reference to the response magnitude. For the advanced and FRS phases, separate breakdowns are given for the principal training pipelines as well as a subtotal that combines the pipeline data.

TABLE I

Summary listing of the percent incidence of airsickness, vomiting, and performance degradation due to airsickness reported by the NFO population during the basic, advanced and FRS phases of flight training for different pipelines. Incidence is expressed as the percentage of the total hops flown in a given phase of training where the denoted airsickness event occurred.

Phase of Training	Number of Students	Total Hops Flown	Airsickness Percent-hops	Vomiting Percent-hops	Perf.Degrad. Percent-hops
Basic Training					
VT10	796	10,759	19.4	9.2	12.7
Advanced Training					
VT86-AJN (Attack)	226	3,385	10.7	4.1	4.3
VT86-RIO (Fighter)	185	4,120	16.9	7.5	5.6
MAFB (P-3)	132	1,794	2.6	0.2	0.5
Subtotal	543	9,299	11.9	4.9	4.2
FRS Training					
Attack	120	3,269	9.2	3.9	4.1
Fighter	89	3,661	4.7	2.1	2.2
P-3	128	900	15.8	4.7	8.3
E-2	35	495	4.0	0.6	3.0
Subtotal	372	8,325	7.6	3.0	3.6
Total - All Phases	796	28,383	13.5	5.9	7.3

As shown in Table I, the highest incidence of airsickness problems occurred during basic training in VT10 as would be expected. Of a total of 10,759 hops flown, airsickness, vomiting, and performance degradation occurred on 19.4, 9.2, and 12.7 percent, respectively, of the flights. During advanced training, corresponding figures for all pipelines combined declined to 11.9, 4.9, and 4.2 percent, respectively. For the final FRS phase of training, a further decline to 7.6, 3.0, and 3.6 percent, respectively, was noted. These raw data show a general decline in airsickness difficulties as training progresses with the incidence during the FRS phase being roughly one-third the incidence during basic training which should be expected as the result of some adaptation to flight stress. The totalized data shown at the bottom in Table I indicates that 13.5 percent of the 28,383 hops flown by 796 NFO students involved airsickness. This is similar to the incidence data reported by McDonough (17) where 15.6 percent of 4,534 flights flown by navigation students involved airsickness.

Although the subtotal data presented in Table I for the advanced and FRS phases shows this gradual decline in incidence as training progresses, considerable variations occur when the pipelines are treated independently. For example, during advanced

training in the attack pipeline, airsickness incidence (10.7 percent) was approximately half the 19.4 percent basic training incidence while the fighter pipeline incidence (16.9) percent showed a much slighter decline. However, when the FRS phase of training was encountered, the attack pipeline incidence (9.2 percent) remained near its advanced phase level while the fighter pipeline incidence (4.7 percent) fell to below one-third its advanced level. The most significant difference occurred in the P-3 pipeline where airsickness incidence fell to 2.6 percent during advanced training but rose to 15.8 percent during FRS training.

To further define these pipeline differences, a Kruskal-Wallis one-way analysis of variance by ranks test was utilized to compare the performance of the NFO students across the four different pipelines. The results of this test are summarized in Table II where the rows with the BAS-prefix represent the unweighted airsickness, vomiting, and performance degradation flight indices received during basic training in VT10 for each pipeline; the rows with the ADV-prefix represent the corresponding flight indices received during advanced training; and the rows with the FRS-prefix the same for fleet readiness squadron training. The rows with the MEAN-prefix represent the simple mean of the flight indices received by an individual during the basic, advanced, and FRS phases. For each of the pipelines, the mean, standard deviation of the observations, and number of students included in the analysis are separately tabulated for each flight index variable.

TABLE II

Results of a nonparametric Kruskal-Wallis one-way analysis of variance comparison of the unweighted airsickness flight indices received by the students in the four major training pipelines. See text for details.

Flight Index variable		VT86-AJN			VT86-RIO			MAFB			ATDS		
	H	Attack Pipeline			Fighter Pipeline			P-3 Pipeline			E-2 Pipeline		
(Unweighted)	Stat.	Mean	S.D.	N	Mean	S.D.	N	Mean	S.D.	N	Mean	S.D.	N
BAS-Airsick	29.2*	17.0	15.3	115	12.5	14.0	84	27.3	23.4	120	20.7	16.4	34
BAS-Vomit	9.2	8.5	13.4	115	5.1	9.5	84	11.7	15.9	120	10.6	13.5	34
BAS-Perf.Degr.	19.2*	9.9	10.7	115	6.9	9.5	84	17.2	19.3	120	16.3	18.3	34
ADV-Airsick	64.1*	10.0	11.8	112	15.3	18.4	84	2.6	5.5	108	--	--	--
ADV-Vomit	43.7*	4.0	7.6	112	6.2	12.6	84	0.2	1.1	108	--	--	--
ADV-Perf.Degr.	34.1*	3.4	6.1	112	3.8	6.2	84	0.5	1.8	108	--	--	--
FRS-Airsick	27.5*	12.4	19.7	115	7.2	17.2	84	16.4	19.2	124	4.6	10.0	34
FRS-Vomit	13.8	6.0	15.1	115	3.2	10.0	84	4.6	11.8	124	0.7	2.3	34
FRS-Perf.Degr.	13.7	4.8	11.1	115	3.4	12.2	84	9.0	16.0	124	3.7	17.2	34
MEAN-Airsick	8.5	13.1	11.6	115	11.7	13.5	84	16.1	13.3	124	12.7	11.9	34
MEAN-Vomit	2.3	6.2	9.4	115	4.8	8.6	84	5.6	8.2	124	5.7	7.2	34
MEAN-Perf.Degr.	13.9#	6.1	6.9	115	4.7	7.4	84	9.2	10.6	124	10.0	16.1	34

= Significant beyond the .01 level; * = Significant beyond the .001 level.

The Kruskal-Wallis H-statistic corrected for tied scores is shown in the data column at the left in Table II where the assumption is made that H is distributed like chi squared with three degrees of freedom for all flight indices except those associated with advanced training. For these indices, only two degrees of freedom are involved since the ATDS pipeline received only academic-related training in the advanced phase. As shown by the significance symbols located adjacent to the H-statistic, the unweighted and weighted airsickness indices showed dissimilarities in the pipeline populations that were significant to the .001 level or better for all three phases of training. For the vomiting indices, differences occurred in only the advanced phase. In the case of the performance degradation indices, differences occurred during both basic and advanced training and were also reflected in the mean indices.

Prior to further discussion of these pipeline differences, reference will again be made to Figure 1 to describe some fundamental differences in the flight syllabi and student flow associated with the four different advanced training pipelines. As schematized by the two blocks drawn within the VT10 block at the top in Figure 1, the flight syllabus in this squadron was subdivided into two sequential phases. All NFO students, with the exception of those to be assigned to the MAFB advanced training pipeline, flew both phases of the flight syllabus. For those students following the MAFB pipeline, only the first phase was flown prior to assignment to advanced training. At the time the longitudinal study was initiated, the VT10 syllabus consisted of five hops in the first phase and 13 hops in the second phase. Midway in the study, the flight syllabus was modified to provide eight hops in the first phase and 13 hops in the second phase. Similar changes occurred in the VT86-AJN and VT86-RIO flight syllabi at about the same time while no changes occurred in the 17 hop MAFB flight syllabus. In subsequent discussion, the original and modified flight syllabi for these squadrons will be referred to as the "old" and "new" flight syllabi, respectively.

The incidence of airsickness in VT10, VT86-AJN, and VT86-RIO on an individual hop basis is displayed in Figure 2. The top three graphs (A, C, and E) pertain to the old flight syllabi associated with these squadrons and the bottom three (B, D, and F) to the

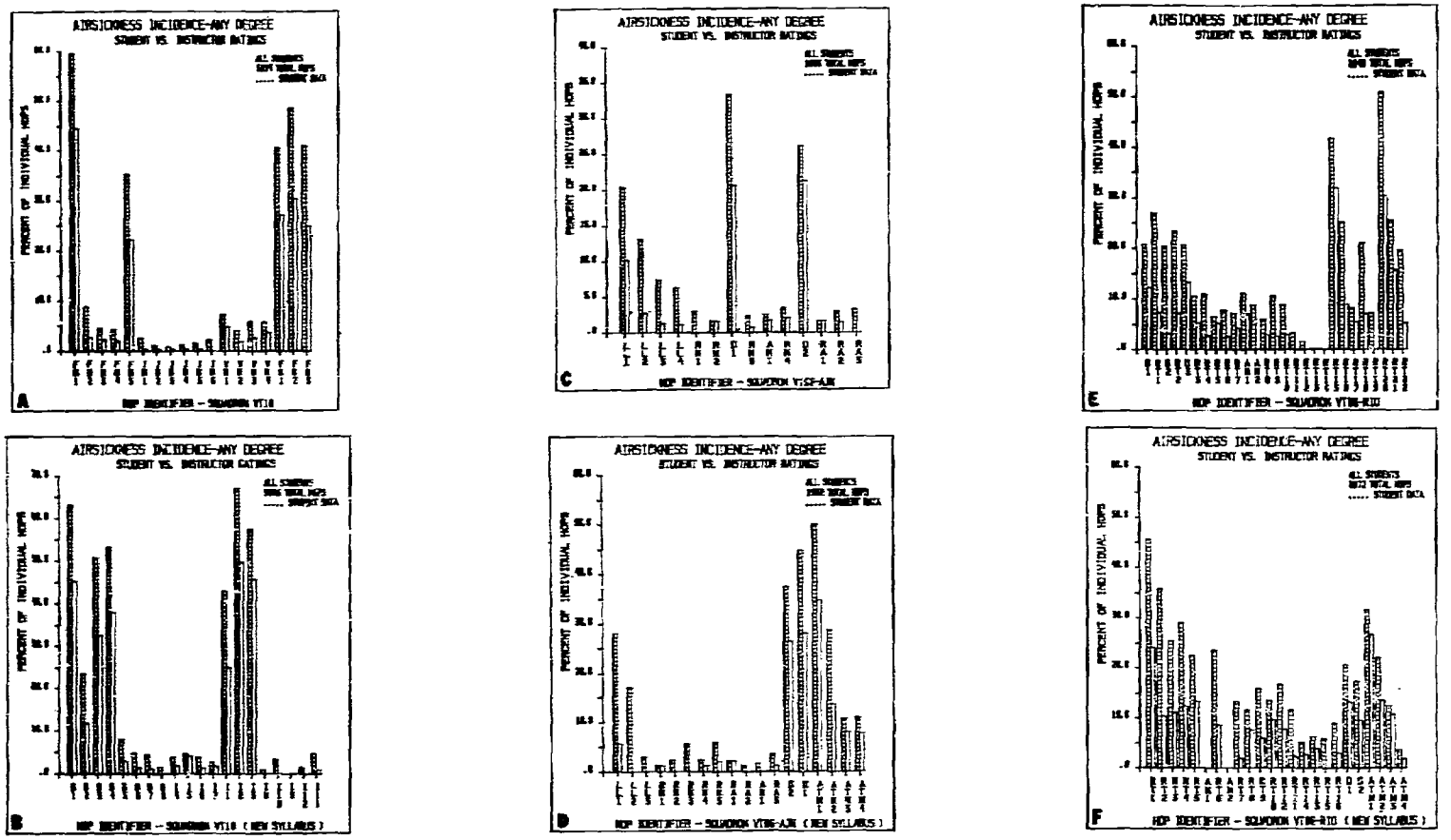


FIGURE 2

Percent incidence of airsickness reported to have occurred on the individual hops comprising the flight syllabi associated with VT10, VT86-AJN, and VT86-RIO. The top three graphs (A, C, and E) pertain to the student population who flew the old flight syllabi and the bottom three (B, D, and F) to those who flew the new flight syllabi. In each graph, two bars are shown above each hop identifier. The left (dotted) bar represents the percentage of the hops flown where the students reported experiencing airsickness and the right (clear) bar represents the percentage of the same number of hops where the instructors reported that the students experienced airsickness. The left-to-right sequence of hops shown in each graph corresponds in general to the sequence that the hops were actuallly flown by the NFO students.

new flight syllabi associated with the same three squadrons. For each graph, incidence is expressed as the percentage of the total hops flown of a given classification where airsickness (mild, moderate, or severe degree) was reported to have occurred. The dotted interior bars represent the incidence as derived from the student judgments and the adjoining clear bars the incidence as derived from the instructor judgments. The left-to-right sequence of hops identified at the bottom in each graph corresponds in general to the sequence the students flew the hops.

Examination of Figure 2A shows that during the first phase of VT10 training, composed of hops FM1 through FM5, two of the five hops involved relatively high motion stress with nearly 60 percent of the students reporting airsickness on FM1 and over 35 percent on FM5. Figure 2B shows that four of the eight hops (B1 through B8) comprising the first phase of the new VT10 flight syllabus involved a relatively high degree of airsickness. For the second phase under both the old and new flight syllabi, a smaller proportion of the total hops flown produced corresponding motion stress. Since the students following the MAFB or P-3 pipeline flew only the first phase of VT10, it would be expected that their VT10 airsickness indices would be higher than those students who flew both phases of the VT10 syllabus. This difference is the primary reason for the pipeline differences noted in the basic training rows of Table II.

In the case of the airsickness measures associated with advanced training, the data of Table II are distinguished again by the MAFB pipeline which had the least difficulties with airsickness. This would be expected since the MAFB flight syllabus involved training in the large, relatively stable P-43A with most hops involving straight and level flight. However, when the MAFB students reached the FRS phase of their training which involved long duration missions in the P-3 aircraft, airsickness rose considerably as reflected by the raw incidence data of Table I and the FRS flight index data of Table II. In effect, the MAFB group flies relatively few hops during basic training and receives only a mild exposure to motion stress during advanced training. A measure of airsickness susceptibility will thus not arrive until the FRS phase of training is reached. Since only a relatively few hops are flown in the P-3 FRS squadrons compared to the fighter and attack pipelines, there is a hazard that airsick susceptibles in the MAFB pipeline may not be identified until they receive their initial fleet assignments. Accordingly, when validated laboratory-based tests of airsickness susceptibility are finally developed, high priority must be given to their early application to the MAFB pipeline.

Examination of Figure 2 relative to the incidence of airsickness as a function of progress through the flight syllabus associated with a given squadron shows a general trend for a relatively high incidence rate for the first few hops of the syllabus. However, there is no pronounced trend for airsickness incidence to gradually decrease as training progresses within a squadron. Instead, as shown for all squadron data presented in Figure 2, airsickness incidence actually rose to a quite high level for certain hops flown toward the end of the syllabus. The high incidence rate for these hops is accounted for by their related flight missions which usually involved aerobatics or advanced tactical maneuvering. In effect, conclusions concerning airsickness adaptation of the NFO population as a function of flight exposure must be carefully weighed in relation to the motion stress level of each hop flown within a given flight syllabus.

Referring once again to Table I, these incidence data provide background data on the overall incidence of airsickness during the different phases of NFO training. However, no information is provided by these data relative to the wide variations always present in individual susceptibility to airsickness nor to the relative contribution of different students to the overall magnitude of the airsickness problem. To provide some insight into this problem, the questionnaire data were analyzed to determine the number of students who experienced repeated airsickness during the course of their training in selected squadrons. To emphasize the multiple contributions of a small number of students to the overall airsickness problem, the airsickness data derived from both the student and instructor questionnaires have been plotted in cumulative frequency distribution form in Figure 3 for VT10, VT86-AJN, and VT86-RIO. In this figure, the deviation between the student and instructor distributions reflects the tendency for the instructors to underestimate the incidence of airsickness using the student judgments as reference. This point is also demonstrated on an individual hop basis by the Figure 2 data. The percentage of the total number of students who were considered to have never experienced airsickness is represented in each Figure 3 graph by the intersection of the distribution curve with the ordinate axis. These distribution data graphically illustrate the point that a small number of airsick susceptible students make a most significant contribution to the hop incidence data of Table I.

Further insight into the overall incidence of airsickness and the multiple contributions of certain students is provided by the data listed in Table III. In this table, data columns 1 and 2 represent the number of students included in the study population and the total number of hops they flew, respectively, with separate listings for VT10, VT86-AJN, and VT86-RIO for both the old and new flight syllabi. Data columns 3-5 describe the percentage of the total hops flown in a given squadron where airsickness, vomiting or performance degradation was involved. (These data are of the same form as those presented in Table I.) Data columns 6-8 list the percentage of the students who reported experiencing airsickness, vomiting, or performance degradation on one or more hops. Data columns 9-11 list the percentage of the total number of students who were responsible for fifty (50) percent of the hops flown where airsickness, vomiting, or performance degradation was reported, i.e., those students who suffered

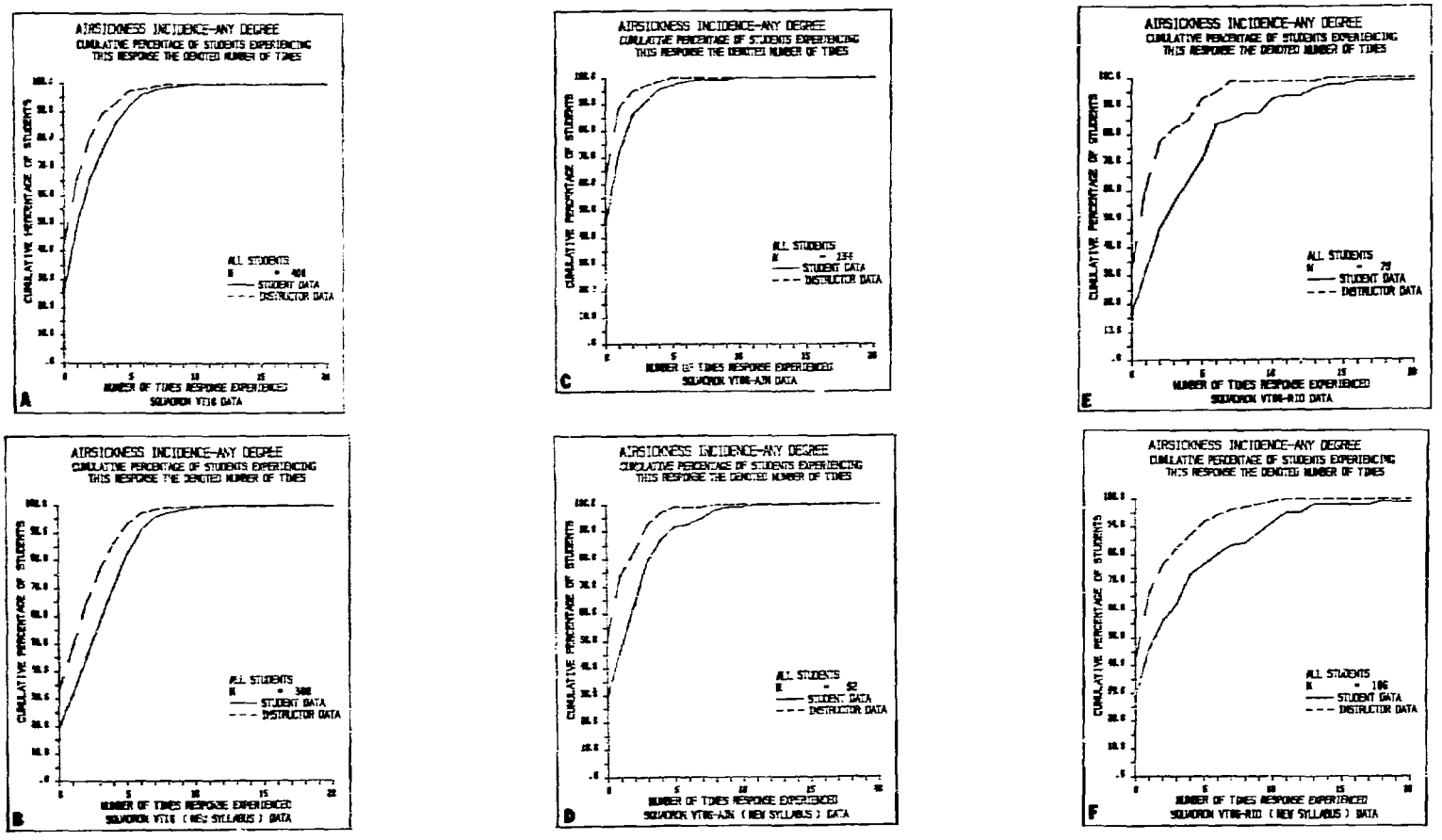


FIGURE 3

Normalized cumulative frequency distribution of students experiencing airsickness a different number of times during the course of their flight training in a given squadron. The top three graphs (A, C, E) pertain to the student populations who flew the old flight syllabi associated with VT10, VT86-AJN, and VT86-RIO and the bottom three (B, D, F) to those who flew the new syllabi. The solid-line distributions are based upon student data and the dashed-line distributions upon instructor data. The deviation between the two distributions reflects the tendency for the instructors to underestimate the incidence of airsickness as compared to the judgments of the students.

repeated airsickness experiences and fell into the upper portions of the Figure 3 distributions.

TABLE III

Incidence of airsickness during basic training in VT10 and advanced training in VT86 for the attack (AJN) and fighter (RIO) pipelines. Columns 1 and 2 represent the number of students studied in the squadron and the number of hops they flew. Columns 3, 4, and 5 represent the percentage of the hops flown where airsickness, vomiting, and inflight performance degradation occurred; columns 6, 7, and 8 show the percentage of the squadron students who reported experiencing the denoted response one or more times; and columns 9, 10, 11 show the percentage of the students that accounted for fifty percent of the total hops flown where the denoted responses occurred.

Squadron	Total No. Stud.	Total Hops Flown	Percent of Total Hops Flown Where Response Occurred			Percent of Students Experiencing Response One or More Times			Percent of Students Causing 50% of Hop Responses		
	(1)	(2)	Air Sick (3)	Vomit (4)	Perf. Degr. (5)	Air Sick (6)	Vomit (7)	Perf. Degr. (8)	Air Sick (9)	Vomit (10)	Perf. Degr. (11)
VT10 #	408	5,394	16	7	11	74	39	59	19	10	14
VT10 *	388	5,365	23	11	15	81	53	67	24	14	18
VT86-AJN #	134	1,833	9	4	3	55	28	31	13	8	8
VT86-AJN *	92	1,552	13	5	5	71	36	41	12	9	11
VT86-RIO #	79	2,048	10	6	4	83	47	48	19	8	12
VT86-RIO *	106	2,072	18	9	7	72	46	43	15	10	9

= Old Flight Syllabus * = New Flight Syllabus

From data columns 6-8, it can be seen that for the denoted basic and advanced training squadrons, the number of NFO students experiencing airsickness one or more times ranged from 55 to 83 percent of the total squadron population which again is comparable to that reported by McDonough (17) who found that 65.7 percent of a navigation student population experienced airsickness one or more times during training. Corresponding ranges were 28 to 53 percent for the vomit measure and 31 to 67 percent for the performance degradation measure. However, the incidence of airsickness shown in data columns 3-5 is not at all evenly distributed over the population represented in data columns 6-8. This is pointedly illustrated by data columns 9-11 which show that a relatively small proportion of students contributed most significantly to the overall incidence data. For example, column 9 indicates that half of the hops flown in the old VT10 flight syllabus where airsickness was reported to have occurred was caused by only 19 percent of the students. This figure ranged from 12 to 24 percent across the denoted squadrons. The contribution of students who suffered repeated experiences of airsickness was even more marked for the vomit measure where the percentage of the students accounting for half of the flights where vomiting occurred ranged from 8 to 14 percent of the total population. Corresponding ranges were 8 to 18 percent for the performance degradation measure. In effect, if the overall magnitude of the airsickness problem during NFO training is to be significantly reduced, then attention must be given to developing selection tests that have the potential to identify this most susceptible component of the NFO population prior to the time they begin flight training.

In the previous reports (7-13) detailing the results of the longitudinal study, correlation matrices were developed using a Spearman rank correlation analysis based upon corrected tied scores to explore the many relationships that existed among and between the flight airsickness indices and the laboratory motion reactivity test scores. One point of concern addressed in the report (13) dealing with the students who successfully completed the entire NFO training program involved the relationship between the airsickness experienced by a given individual during basic training with the airsickness he experienced during later phases of training. To this end, a Spearman rank correlation analysis was performed to determine the relationship between the unweighted airsickness indices received in basic training with the same indices received during advanced and FRS training for each of the major pipelines. The results of this analysis, presented in Table IV, show that the strongest relationship existed for the VT86-AJN and VT86-RIO pipelines where the correlation coefficients were in the range of .51 to .61 and significant to the .001 level or better. For these pipelines, it is probable that the airsickness experiences of a given student during basic training will carry over into the advanced and FRS phases. In the case of the MAFB or P-3 pipeline, a significant relationship between basic and advanced training was not realized. Again, this is accounted for by the low motion stress associated with advanced flight training in the P-43A aircraft at MAFB. However, a significant correlation was achieved for the FRS phase of this pipeline.

As described in the Procedure section, a large sample of the NFO population was given several laboratory tests of motion reactivity prior to beginning flight training. In the report (13) dealing with airsickness problems during FRS training, a tabulation was presented (Ref 13, Table VIII) of the Spearman rank correlation coefficients between certain of these tests and the unweighted airsickness indices received during each phase of training for each of the pipelines. Table V shows the results of this analysis for all pipelines combined where separate listings are provided for three sets of tests. The first set involved a two-part motion sickness history questionnaire describing motion sickness incidence and exposure where the first part (variable 1) pertained to experiences prior to age 12 and the second part (variable 2) to experiences following age 12. The sum of these two scores is separately listed (variable 3) in Table V. The second set of tests pertain to the Brief Vestibular Disorientation Test (BVDT) which is

TABLE IV

Spearman rank correlation coefficients expressing the relationship between airsickness experienced during basic training with airsickness experienced during the following advanced and FRS phases for different pipelines.

Basic Training Pipeline	Flight Index	Advanced Training ADV-Airsick	FRS Training FRS-Airsick
VT86-AJN	BAS-Airsick	.58*	.51*
VT86-RIO	BAS-Airsick	.61*	.53*
MAFB	BAS-Airsick	.13	.38*
Combined	BAS-Airsick	.24*	.48*

* = Significant beyond the .001 level.

TABLE V

Spearman rank correlation coefficients expressing the relationship between selected laboratory motion reactivity test scores and unweighted airsickness indices received during different phases of training.

Laboratory Test Variables No.	Test Name	Airsickness Indices (Unweighted)-All Pipelines Combined Basic Training	Advanced Training	FRS Training	Mean Index
1	MS History:Part 1	.41*	.19#	.26*	.40#
2	MS History:Part 2	.47*	.20*	.36*	.48*
3	MS History:Sum	.48*	.23*	.36*	.50*
4	BVDT:Rater Score	.37*	.16	.26*	.38*
5	BVDT:Self-rating Score	.37*	.20*	.32*	.41*
6	BVDT:Post-rating Score	.28*	.14	.25*	.31*
7	BVDT:Sum Scored	.42*	.26*	.33*	.46*
8	VVIT:Rater Score	.22#	.12	.14	.23#
9	VVIT:Self-rating Score	.23#	.23#	.28#	.30*
10	VVIT:Post-rating Score	.22	.05	.21	.24
11	VVIT:Sum Score	.27#	.15	.21	.29*

= Significant beyond the .01 level; * = Significant beyond the .001 level.

based upon cross-coupled angular acceleration stimuli produced by paced head motions on a rotating chair. The BVDT-Rater score (variable 4) involves the motion reactivity signs judged to be present by observers following the test; the BVDT Self-rating (variable 5) and Post-rating (variable 6) scores involve the rating of similar symptoms by the subject immediately following and 24 hours after completing the test. The sum of these three BVDT scores is represented by variable 7. The Visual/Vestibular Interaction Test (VVIT) is based upon the visual scan, acquisition, and identification of a matrix type numerical display while undergoing sinusoidal rotation. The symptoms were rated in a fashion similar to those of the BVDT with the related test scores listed as variables 8-11 in Table V.

All three of the motion sickness history scores showed significant correlations with the airsickness indices received during each phase of training. However, the strongest relationship existed during the basic phase with the sum score (variable 3) displaying the strongest relationship. In the case of the four components of the BVDT, significant correlations existed with all airsickness indices except those received during advanced training. Again, the sum BVDT score (variable 7) showed the strongest relationship. The VVIT test scores showed a weaker relationship to the airsickness indices compared to the

other two tests. However, the self-rating score (variable 9) was significantly correlated with all four indices. Though most of these test variables have statistically significant correlations to the listed airsickness indices, the correlated coefficients are not at all adequate for prediction applications. (An important point in evaluating the relative magnitude of the correlation coefficients presented in Table V is that the data are based upon only those NFO students who successfully completed the entire NFO training program. The analysis does not include those students who attrited from the program or those who decided to not continue their voluntary participation in the longitudinal study). That is, until an individual test or test battery is developed with much higher correlation coefficients, too many students will be rejected who could have successfully completed the program or vice versa.

A last point involves the need for inflight validation data to establish the relative strength of each candidate test undergoing development. Just as the individual motion reactivity tests must be designed to eliminate any bias that may be introduced by the student, so must the method used to document the actual incidence of airsickness during a given flight. In this respect, heavy dependence must be placed on the flight instructor to gauge the incidence and severity of airsickness experienced by a given student. Although the instructor will obviously identify an overt sign such as vomiting, it might be argued that there would be too many limitations imposed on his judgments where airsickness occurred with less obvious signs and symptoms.

The data of this study (1-6), however, have shown a high degree of correlation between the student and flight instructor ratings of airsickness present on a given hop. In Table VI, Spearman rank correlation coefficients adjusted for tied scores are presented which show the close relationship between student and instructor ratings (unweighted flight indices) of airsickness incidence as judged to have occurred in different training squadrons. For all three response variables, airsickness, vomiting, and performance degradation, the student and instructor ratings are significantly correlated to the .001 level or better. The correlation coefficients range from 0.85 through 0.97 for the vomiting response as would be expected. Equally important, the student and instructor ratings are highly correlated in the range of 0.69 through 0.86 for the airsickness measure as well. The weighted flight indices, though not listed in Table VI, also show correlation magnitudes of equal or slightly greater magnitude. In this respect, it would appear that instructor-based judgments of airsickness incidence and severity can well serve as validation criteria for identification of candidate tests with the highest potential for optimizing the aircrew selection process.

TABLE VI

Spearman rank correlation coefficients showing the close relationship between the student and instructor ratings of airsickness (unweighted flight indices) judged to have occurred during basic training in VT10 and advanced training in VT86-AJN and VT86-RIO for both the old and new flight syllabi populations.

Student Data Squadron Flight Indices	Instructor Data Airsickness Flight Syllabus Old	New	Vomiting Flight Syllabus Old	New	Perf. Degradation Flight Syllabus Old	New
VT10						
Airsickness	.80*	.79*				
Vomiting			.93*	.94*		
Perf. Degradation					.71*	.75*
VT86-AJN						
Airsickness	.71*	.69*				
Vomiting			.92*	.87*		
Perf. Degradation					.55*	.61*
VT86-RIO						
Airsickness	.77*	.85*				
Vomiting			.95*	.96*		
Perf. Degradation					.63*	.48*

* = Significant beyond the .001 level

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DISCLAIMER

Opinions or conclusions contained in this report are those of the authors and do not necessarily reflect the views or endorsement of the Navy Department.

DISCUSSION

CURRAN: What is the percentage overall NFO P-3 attrition for FRS? What is the incidence of motion sickness for NFO's in P-3 fleet operations, specifically low altitude, race track pattern?

GUEDRY: We do not have information on attrition rate in P-3 FRS but believe that it was relatively low in our study because we had a good return rate on our questionnaires. In answer to the second question, we have heard that the incidence of motion sickness in P-3 fleet operations involving low altitude race track patterns is high and we have included this as part of a proposal for study in the next fiscal year.

SUSCEPTIBILITY OF CAT AND SQUIRREL MONKEY TO MOTION SICKNESS INDUCED BY VISUAL STIMULATION: CORRELATION WITH SUSCEPTIBILITY TO VESTIBULAR STIMULATION

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SUMMARY

This paper describes experiments in which the susceptibility of both cats and squirrel monkeys to motion sickness induced by visual stimulation is documented. In addition, it is shown that in both species those individual subjects most highly susceptible to sickness induced by passive motion are also those most likely to become "motion" sick from visual (optokinetic) stimulation alone.

INTRODUCTION

It is well known that symptoms of motion sickness, as well as illusions of self-motion (circularvection and linearvection), can be elicited in human subjects by visual stimulation alone (1, 4, 5). Visual stimulation has also been shown to be effective in modifying the sickness-inducing effects of vestibular stimulation (2, 8). Further, in recent electrophysiological studies in animals it has been demonstrated that neural activity in the vestibular nuclei is modulated in a similar way by actual passive sinusoidal angular or linear acceleration of the animal and by visual stimulation which simulates those motions (3, 10). These findings suggest that vision should play an important role in the production of motion sickness in animal subjects as well as in human subjects, and, as in humans, the effects of visual stimulation should be greatest in those animals most susceptible to motion sickness produced by vestibular stimulation.

With the exception of the report of motion sickness in one squirrel monkey exposed to sinusoidal yaw-axis optokinetic stimulation (6), the susceptibility of animals to visually induced motion sickness has not been documented, nor has the relationship between susceptibility to motion sickness induced by visual vs. vestibular stimuli been addressed. The studies reported here were designed to investigate these factors in two species, the cat and the squirrel monkey. In these studies animals were subjected to passive acceleration provided by a two-pole swing (cats) or to passive rotation (monkeys), and to visual (optokinetic) stimulation which simulated these motions. Levels of susceptibility to visual stimulation alone were compared with those for the same animals exposed to the associated vestibular stimuli to obtain a better understanding of the role of vision in the production of motion sickness. The data were also analysed to determine how consistent the trait of susceptibility is across different stimulus conditions.

METHODS

Cats.

Twenty mature female cats were exposed to two conditions of visual and vestibular stimulation while free to move within a clear Plexiglas cage (44 cm X 16 cm X 21 cm). Animals were exposed to motion for a period of 20 min or until retching/vomiting plus 5 min, whichever period was longer. A period of not less than 30 days intervened between each of the tests.

Combined visual-vestibular stimulation was provided by a two-pole swing with a radius of 1.8 m, a frequency of 0.37 Hz, an arc of 1.0 rad, and a vertical displacement of 0.9 m. This swing was suspended within a large box-like enclosure, the interior of which was covered with patterned wallpaper and illuminated with a 100 watt bulb. A one-way vision port for observation of the animal was situated at one end of the box. The swing was manually pushed to provide the vestibular stimulation.

Visual stimulation alone was provided by the same two-pole swing and enclosure used in the combined stimulus condition, but in the Visual Only Condition the swing holding the cat remained stationary, while the enclosure was swung at a frequency of 0.28 Hz, with an arc of 1.0 rad. The visual stimulation was thus nearly, but not exactly, the same in the Combined Visual-Vestibular and the Visual Only Conditions. For the Visual Only Condition both observation ports were covered with one-way vision material.

Four additional motion sickness-inducing conditions involving visual-vestibular stimulation were used in an assessment of each subject's level of susceptibility to motion sickness. In Condition 1, a turntable was used to rotate the animals at 120 deg/sec. During rotation the cage holding the animal was tilted 7.5 deg above and below the horizontal plane at a frequency of 0.6 Hz. In Condition 2 the cage holding the animal was suspended from the end of a tilting beam which oscillated over a vertical distance of 2.1 m at 0.12 Hz. In Condition 3 the tilting beam was used to provide vertical oscillations at 0.42 Hz with a displacement of 1.0 m. A two-pole swing similar to that described for the Combined Visual-Vestibular Condition was used to provide the stimulus for Condition 4. In this condition the swing had a radius of 3.7 m, a frequency of 0.27 Hz, an arc of 1.5 rad, and a vertical displacement of 1.0 m. Both visual and vestibular stimulation were provided in these conditions, since the animals

could view the room through the Plexiglas cage during each of these tests. In all of these test situations retching/vomiting was detected by visual observation.

Monkeys.

Squirrel monkeys were exposed to the visual and vestibular stimulation while free to move in a clear Plexiglas cage (52 cm X 23 cm X 30 cm). Each test session lasted until 5 min after the time of retching/vomiting, or for a maximum of 30 min if vomiting did not occur. An interval of at least 30 days without testing was maintained between experimental sessions.

In the Combined Visual-Vestibular Condition the animals were rotated by a turntable (Goerz Model 611) while able to view the interior of the test room (a 2.3 m cube). The center of the turntable was located 1 m from the nearest corner on a room diagonal. In this condition the animals could see the observers and other contents of the test room, and therefore were exposed to very complex visual stimulation during rotation.

In the Visual Only Condition visual stimulation was provided by an optokinetic drum, the inside of which was covered with alternating white and dark green stripes, each subtending a visual angle of approximately 6.5 deg. In this condition the animal remained on the stationary turntable while the optokinetic drum rotated around the animal, providing optokinetic stimulation.

Two additional conditions (Vestibular Dark and Fixed Visual-Vestibular), studied extensively in another experiment (2), were used in the assessment of susceptibility to visual and vestibular stimulation. In the Vestibular Dark Condition the turntable holding the animal was rotated in the dark, and the animal received no visual stimulation. In the Fixed Visual-Vestibular Condition the optokinetic drum was coupled or fixed to the turntable and rotated with the animal, so that no optokinetic stimulation was produced by the rotation of the turntable.

Two different angular velocities were used to assess the effects of frequency of visual stimulation and amplitude of vestibular stimulation on relative provocativeness of the stimuli and on the correlation between susceptibility to visual vs. vestibular stimulation. Each monkey was exposed to each stimulus condition at both 60 and 150 deg/s. Motion sickness was assessed by determining latencies to retching/vomiting by audio monitoring.

RESULTS

Cats.

Two cats out of the 20 tested (10%) were made motion sick to the point of retching/vomiting under the Visual Only Condition. The latencies to the first retching/vomiting episode for the two cats were 2 min and 19 min. Five of the 20 animals (25%) were made motion sick by the combined visual-vestibular stimulation. The latencies to the first retching/vomiting episode ranged from 5 min to 17 min. The two animals which became sick in the Visual Only Condition were also made sick by the combined visual-vestibular stimulation. However, the remaining three animals which were susceptible to combined visual-vestibular stimulation were not made sick by visual stimulation. Thus, although neither stimulus produced high rates of sickness in these cats, of those animals which were made sick by combined visual-vestibular stimulation a subset was made sick when exposed to visual stimulation alone.

To determine whether there is a relationship between susceptibility to visual and to vestibular stimulation, two comparisons were made. One comparison involved determining whether the animals which retched/vomited to visual stimulation were those individuals with the shortest latencies to retching/vomiting in the condition involving combined visual-vestibular stimulation. If the animals which retched/vomited in the combined condition are ranked in order of ascending latencies, with a rank of 1 being the shortest latency (5 min) and a rank of 5 being the longest latency (17 min), it was found that the animals which retched/vomited to visual stimulation were not those ranked 1 and 2 on the combined stimulus, but rather those ranked 3 and 4. Thus, the two cats which retched/vomited to visual stimulation were not the most susceptible animals if latencies to retching/vomiting on the combined visual-vestibular test are used as the criterion of susceptibility.

Another measure of susceptibility is available, however, since all of these animals had been tested for motion sickness in the four additional conditions involving rotation, vertical oscillation, and swinging. If animals are ranked on the basis of each of their responses in five repeated trials on each of the four additional motion sickness tests (a total of 20 test sessions), with Rank 1 assigned to the animal having the highest number of test sessions in which retching/vomiting occurred, then the two animals which vomited to visual stimulation were ranked 1 (vomited 10 times) and 2 (vomited 5 times) for susceptibility. By this measure, the animals made sick by optokinetic stimulation were indeed the most susceptible cats.

Monkeys.

The percentage of monkeys retching/vomiting in the Visual Only and Combined Visual-Vestibular Conditions and the median latency to the first sickness episode are shown in Table 1 for both angular velocities. The high percentage of animals vomiting to visual stimulation alone was quite unexpected on the basis of the data from the cat and from human studies, both of which typically show that susceptibility to visual stimulation is much lower than that to combined visual-vestibular stimulation. In this study the incidence of vomiting at the lower velocity was the same with visual and combined stimulation ($p > .50$), but the latency to sickness was shorter in the Visual Only Condition ($p < .01$). At the higher velocity, where greater vestibular effects would be expected, the incidence of sickness was greater ($p < .05$) and the latency to sickness was shorter in the Combined Visual-Vestibular Condition than in the Visual Only Condition ($p < .05$).

Table 1. Median Latency to Retching/Vomiting and Percentage of Animals Sick in Each of the Test Conditions (N = 27).

ANGULAR VELOCITY	MEASURE	VISUAL ONLY	COMBINED VISUAL-VESTIBULAR
60 deg/s	MDN. LATENCY (min)	10	25
	%-age SICK	74	70
150 deg/s	MDN. LATENCY (min)	11	8
	%-age SICK	81	100

To assess whether the highly susceptible animals were more likely to become sick when exposed to the optokinetic stimulus, those animals most and least susceptible to sickness in the Combined Visual-Vestibular Condition were selected as representative of the extremes of susceptibility induced by rotation. Animals with the 7 shortest and 7 longest (highest and lowest 25%) latencies from the extremes of the retching/vomiting latency distribution for each angular velocity were taken as representive of the least and most susceptible subjects for that velocity. The mean and median latencies to retching/vomiting to optokinetic stimulation at each angular velocity were then calculated for these highly susceptible and resistant animals. These data are shown in Table 2. Animals classified as susceptible to the combined visual-vestibular stimulation had shorter median latencies to retching/vomiting induced by optokinetic stimulation than did those classified as resistant. This relationship occurred for tests run at both 60 deg/s ($p = .04$) and 150 deg/s ($p = .03$) showing that animals susceptible to combined visual-vestibular stimulation were also those most susceptible to visual stimulation.

Table 2. Mean and Median Latency (min) to Retching/Vomiting Induced by Visual Stimulation at 60 and 150 deg/s for Animals Selected as Susceptible and Resistant on the Basis of Combined Visual-Vestibular Stimulation.

MEASURE	60 deg/s		150 deg/s	
	Resistant	Susceptible	Resistant	Susceptible
MEDIAN	26.0	7.3	22.0	4.7
MEAN	20.3	10.8	20.9	10.0

This conclusion is based upon an analysis of the relationship between the latencies to retching/vomiting for animals representing the extremes of the susceptibility spectrum, i.e., those which were either very susceptible or very resistant. Such an analysis could be misleading, since any relationship could depend predominately upon extreme ranges of susceptibility, and thus might not reflect accurately the responses of the entire population of subjects.

To examine this issue further, we obtained correlations between sickness latencies from all monkeys across the four different sickness-inducing conditions, including Visual Only, Combined Visual-Vestibular, Vestibular Dark and Fixed Visual-Vestibular Conditions. The correlations between latencies obtained during visual stimulation alone and those obtained during the three other conditions of stimulation are shown in Table 3. These results indicate that at 60 deg/s (upper portion of the table) the level of sickness evoked in individual animals by the optokinetic stimulation is predicted better by the response of these animals to the Fixed Visual-Vestibular Condition than it is by the response to the Combined Visual-Vestibular or the Vestibular Dark Conditions. Conversely, sickness evoked by the optokinetic stimulus at 150 deg/s (lower portion of the table) is predicted better by the data obtained from the Combined Visual-Vestibular Condition than by those obtained in the other two conditions. Predictions about susceptibility to visual stimulation based on data obtained during vestibular dark stimulation, a condition not involving visual stimulation, is poor for both angular velocities.

Table 3. Correlations Between Latencies to Retching/Vomiting (N = 27).

60 deg/s				
	VESTIBULAR DARK	COMBINED VISUAL-VESTIBULAR	FIXED VISUAL-VESTIBULAR	VISUAL ONLY
VESTIBULAR DARK	1.00	0.16	0.19	0.08
COMBINED VISUAL-VESTIBULAR	—	1.00	0.28	0.36
FIXED VISUAL-VESTIBUALR	—	—	1.00	0.53 *
150 deg/s				
	VESTIBULAR DARK	COMBINED VISUAL-VESTIBULAR	FIXED VISUAL-VESTIBULAR	VISUAL ONLY
VESTIBULAR DARK	1.00	0.20	0.34	0.13
COMBINED VISUAL-VESTIBULAR	—	1.00	0.74 *	0.56 *
FIXED VISUAL-VESTIBULAR	—	—	1.00	0.41

* $p < .05$

DISCUSSION

These studies indicate that in animal subjects, as in man, motion sickness can be elicited by visual stimulation alone, a condition which involves no direct stimulation of the vestibualar and organs by passive motion. This study has also shown that a subject's susceptibility to sickness induced by optokinetic stimulation is predictable from information about that subject's susceptibility to other motion conditions. In general these data indicate, as do data from studies with human subjects, that those individuals that are highly susceptible to motion sickness induced by passive motion are more likely to become sick, and/or become sick more rapidly, to visual stimulation alone, than are subjects that are relatively resistant to sickness induced by passive rotation.

While relative susceptibility in this population is predictable between some conditions, the determinants of this prediction are not clear. Current theories of motion sickness (7, 9) suggest that sickness-evoking properties of a situation depend upon, or evolve from, a complex interaction of stimulus characteristics and/or effects. Presumably these interactions among the visual, vestibular, and proprioceptive systems occur while the individual is maintaining postural and oculomotor control and performing goal-directed behaviors. The fact that, as shown above, correlations exist between different conditions at the two different velocities of stimulation implies that at the lower velocity, production of sickness in the Visual-Vestibular Fixed and Visual Only Conditions had some particular combination of visual-vestibular-proprioceptive factors in common. At the higher velocity the relationship among these factors was closer in the Combined Visual-Vestibular and Visual Only Conditions.

These data show that the particular combination of visual-vestibular-proprioceptive factors which produce motion sickness may be quite different under conditions of stimulation with similar motion components. It thus seems obvious that improved prediction of susceptibility to motion sickness will require extensive analysis of the specific components of motion stimulation which produce that sickness. In addition, these experiments have shown that both the cat and squirrel monkey, like man, are susceptible to motion sickness induced by visual stimulation alone. The fact that the squirrel monkey appears to be highly susceptible to visually-induced motion sickness suggests that this animal may be useful for more detailed analyses of the role of visual input in the production of motion sickness and for the assessment of parameters critical to successful prediction of susceptibility across sickness-inducing conditions.

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DISCUSSION

KENNEDY: Your prediction relationships are likely to be higher if you correct the correlations by the attenuation occasioned by the expected unreliability of the criterion. The latter may be best estimated by Guedry's data which suggest r =.50 is reasonable.

DAUNTON: The unreliability of the criterion (vomiting) is not due to unreliability of the measurement but is an inherent response unreliability. We regard the uncorrected correlations as good descriptors of the relationships among the variables reported here. On the other hand, it is of interest to know what the correlations might be without these unreliabilities, and we should examine such corrections.

OMAN: Do you have any data on test/retest reliability of any of the individual treatments (tests) you used? How does this compare with similar tests involving humans?

DAUNTON: As occurs with humans, some individuals respond on each exposure to motion while others respond on some tests but not others. As a general rule, we expect an analysis would show that animals are consistent in their responses on about 75% of the tests.

GUEDRY: Did you say that the animals were free to move within their container in all of the stimulus conditions you used?

DAUNTON: Yes, the animals move freely in the container-which was the same size in all of the conditions.

SUSCEPTIBILITE AUX CINETOSES ET AMPLITUDE PERCUE DES ILLUSIONS SENSORIELLES

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RESUME

Les accélérations de Coriolis provoquées par le départ et l'arrêt d'une centrifugeuse entraînant de fortes sensations illusoires de déplacement angulaire. L'amplitude de ces sensations a été étudiée dans deux groupes de sujets, réceptifs et non réceptifs au mal des transports. On a également fait varier l'axe d'application du facteur de charge par rapport à la tête. Des différences d'amplitudes notables ont été relevées entre les deux groupes pour l'ensemble des protocoles étudiés. L'orientation du facteur de charge influence fortement l'apparition des symptômes de cinétose.

INTRODUCTION

Malgré les très nombreuses études qui ont pu lui être consacrées, le mal des transports possède encore quelques aspects relativement mal connus. Parmi ceux-ci le problème des différences de susceptibilité, vis à vis des stimulations provocatrices, observées en pratique courante d'un individu à l'autre, n'a pas encore reçu de réponse satisfaisante.

Pour tous ceux qui ont eu à sélectionner des personnels dans ce domaine, il est en effet classique de constater que la sensibilité aux stimulations habituellement employées se distribue d'une manière très erratique dans la population.

Certaines études (3) ont pu montrer que le mal des transports était moins fréquent dans une population de pilotes que dans la population générale. Toutefois, on peut voir des pilotes, pourtant très habitués aux stimulations vestibulaires aéronautiques, révéler une grande sensibilité aux épreuves de Coriolis alors que d'autres sujets, sans expérience particulière, démontrent une immunité remarquable vis à vis des mêmes contraintes.

Les notions de réceptivité et d'adaptabilité, avancées par REASON (17) pour expliquer ces différences, constituent une hypothèse de travail intéressante.

Cependant la susceptibilité au mal des transports doit être, dans la plupart des cas, déterminée à l'aide d'épreuves provoquant l'apparition du syndrôme. Plusieurs difficultés surgissent alors :

- C'est en premier lieu la difficulté de porter un jugement objectif sur la sévérité du malaise engendré par les stimulations, surtout chez des sujets non coopérants.

- C'est aussi le problème bien connu de la spécificité des stimulations. L'expérience des vols spatiaux a clairement mis en évidence que l'on peut être affecté en impesanteur tout en résistant parfaitement aux épreuves de laboratoire sur terre.

Il n'est donc pas étonnant que de nombreux auteurs se soient attachés à relier la susceptibilité au mal des transports avec des variables physiologiques ou psychologiques plus générales.

On peut ainsi citer dans le domaine cardio-vasculaire les études rapportées par CRAMPTON et plus tard JOHNSON et coll.(8). Certaines corrélations psychologiques ont également pu être avancées par COLLINS et LENTZ (4). MONEY (13) et REASON (17) font la revue de ces corrélations.

Bien évidemment ce sont les caractéristiques du fonctionnement de l'appareil vestibulaire qui ont principalement retenu l'attention de la majorité des auteurs. On pense bien sûr aux travaux de l'école Hollandaise avec VAN EGMOND, GROEN et JONGKEES qui ont développé les techniques de cupulométrie, utilisées par certains auteurs pour prédire la susceptibilité au mal des transports.

Des études plus récentes, menées par BENSON (2) et surtout DOBIE (5) ont clairement démontré qu'il n'existait pas de corrélation significative entre les caractéristiques du cupulogramme et la susceptibilité individuelle aux cinétoses.

De même, toutes les études menées ultérieurement, en particulier par CLARK et STEWART (3) sur les seuils de perception des rotations, LENTZ sur l'analyse du nystagmus rotatoire (11), n'ont pas permis de mettre en évidence des caractéristiques propres au fonctionnement vestibulaire qui soient liées à la susceptibilité au mal des transports. Finalement les seuls résultats positifs dans ce domaine sont ceux rapportés par REASON (16) sur certaines variables psychophysiques et sur l'estimation de la perception de vitesse angulaire post-rotatoire.

Pour REASON la réceptivité au mal des transports dépendrait en quelque sorte de la façon dont le système nerveux central "coderait l'énergie des stimulations sensorielles". Ces résultats permettent éga-

lement à CLARK (3) d'émettre l'opinion que "les épreuves donnant une indication de la façon dont le système nerveux central traite l'information vestibulaire semble avoir une valeur dans la prédiction du mal des transports".

Certains résultats portant sur le jugement du déplacement de la verticale perçue au cours de lancements en centrifugeuse, lorsqu'existent des accélérations de Coriolis, semblent apporter un nouveau support expérimental à ces affirmations.

METHODES

L'expérimentation a été conduite sur la centrifugeuse du Laboratoire de Médecine Aérospatiale dans la configuration nacelle libre. Cette configuration implique l'existence de fortes accélérations de Coriolis au départ et à l'arrêt de la centrifugeuse. Des sensations illusoires sont donc perçues par les sujets. Ces illusions consistent en une sensation de bascule plus ou moins importante dans le plan sagittal du corps.

Sujets expérimentaux

Douze sujets volontaires ont subi l'ensemble des trois protocoles constituant l'expérimentation.

Les sujets avaient été classés en deux groupes distincts selon leur réceptivité aux effets des accélérations de Coriolis.

Un premier groupe composé d'individus résistants à ce type de stimulation (NR = Non Réceptifs) a été recruté parmi des personnels n'ayant pas ou peu d'histoire de mal des transports, malgré leur expérience des stimulations vestibulaires.

Le deuxième groupe de sujets (R = Réceptifs) a été sélectionné sur des critères d'exposition aux accélérations sur un fauteuil tournant et sur leurs antécédents de cinétose.

Aucun des sujets ne présentait d'antécédents de troubles vestibulaires pathologiques. Une exploration fonctionnelle des canaux semi-circulaires a été réalisée pour chacun d'entre eux lors d'épreuves rotatoires. Ces épreuves réaliséessur un fauteuil tournant CONTRAVES-GOERZ (PITTSBURG) ont permis de calculer, pour chaque sujet, les caractéristiques de gain, de phase et de prépondérance labyrinthique du réflexe vestibulo-oculaire. Ces valeurs sont pour l'ensemble des sujets dans le cadre de la normalité.

La plupart des participants dans les deux groupes étaient déjà familiarisés avec les lancements en centrifugeuse.

Dispositif expérimental

Le dispositif expérimental utilisé pour l'essai a été installé dans la nacelle "universelle" de la centrifugeuse du Laboratoire.

Un cadre métallique sur lequel reposait un matelas coquille était fixé sur le plancher de la nacelle, de façon à ce que la tête du sujet se trouve à l'intersection de l'axe de rotation de la nacelle (X nacelle) et du rayon médian du bras (Y nacelle). Les pieds du sujet se trouvaient ainsi à l'arrière de la nacelle par rapport à la direction du lancement. Avant chaque série d'essais, le sujet était soigneusement installé et l'on faisait alors le vide dans le matelas. Deux sangles immobilisaient le corps au niveau du thorax et du bassin. Une sangle frontale assurait la contention de la tête dans un repose tête en forme de U.

Les paramètres physiques du lancement étaient recueillis au moyen de chaînes de mesure d'accélérations linéaires et de vitesses angulaires. Les capteurs étaient situés au plus près de la tête du sujet et dans le bras de la centrifugeuse.

Un système optique de projection d'image couplé à un levier de commande permettait au sujet d'indiquer la position perçue de l'horizontale et de la verticale au cours du lancement.

Un repère lumineux orthogonal, constitué d'une barre pour l'horizon et d'un point pour la verticale, était projeté sur un écran hémi-cylindrique de 38 cm de large et de 140 cm de diamètre. Au repos, le repère était calé de façon à indiquer la verticale et l'horizontale vraie. Un système de rappel permettait de ramener automatiquement le dispositif à cette position lorsque le sujet n'exerçait pas de pression sur le levier.

L'écran dont le diamètre était disposé selon l'axe sagittal du sujet comportait, sur un fond blanc, des stries noires de 0,6 cm de large. Les repères de position étaient portés tous les dix degrés à partir de la verticale. La tête du sujet était placée au centre géométrique du dispositif. La distance des yeux à la surface de projection était donc d'environ 70 cm.

Un potentiomètre solidaire du système optique permettait de connaître le sens et de mesurer la valeur du déplacement angulaire du repère lumineux lorsque le sujet agissait sur le levier de commande.

Pendant les essais le sujet était mis en condition de "vision stabilisée" (référence visuelle interne). C'est-à-dire qu'il ne disposait d'aucune référence visuelle liée au repère terrestre.

La surveillance médicale était assurée au moyen d'une caméra vidéo en basse luminance (AATON) ainsi que par un contact auditif (interphone deux voies). De plus une surveillance constante de l'électrocardiogramme était pratiquée tout au long du lancement sur un moniteur vidéo de contrôle.

L'ensemble des signaux en provenance de la nacelle et du bras transitait par les contacts tournants de la centrifugeuse jusqu'au poste de contrôle. Un enregistreur magnétique SCHLUMBERGER A 4660 assurait l'acquisition de ces données ainsi que celles des signaux d'une base de temps codée. Simultanément les signaux d'entrée étaient contrôlés en temps réel sur un oscilloscope 14 voies. Les six paramètres principaux étaient envoyés en relecture sur un enregistreur graphique GOULD.

Protocole expérimental

Chaque groupe de sujets, réceptifs ou non-réceptifs aux effets des accélérations de Coriolis (R, NR) a été soumis à la même série d'essais.

A l'issue d'expériences préliminaires, trois protocoles d'essais avaient été retenus.

Les protocoles faisaient respectivement varier le niveau de l'accélération en plateau, la pente de mise en accélération, la position de la tête du sujet relativement au facteur de charge. L'effet de la variation de position de la tête n'a été examiné qu'en combinaison avec la variation du niveau d'accélération.

Les essais ont donc été divisés en protocoles "Niveau" (avec un protocole "X-Niveau" et un protocole "Z-Niveau" suivant l'orientation de la tête) et en protocole pente (position selon l'axe X).

L'organisation matérielle des essais impose que chaque protocole soit appliqué successivement aux sujets. C'est ainsi que les essais avec le facteur de charge orienté selon l'axe X de la tête ont été systématiquement effectués avant les lancements en position "Z". Chaque protocole comportait 3 traitements différents.

Afin d'éliminer un éventuel effet d'ordre, il a été procédé à une permutation circulaire de l'ordre de présentation des différents traitements à l'intérieur de chaque protocole. Les cellules de trois ainsi constituées ont été répliquées quatre fois pour tenir compte du nombre de sujets. Un intervalle minimum de 7 jours a été respecté entre chaque protocole.

Protocoles Niveau

Les deux protocoles "Niveau" (Z-Niveau et X-Niveau) comportaient chacun trois profils d'accélération différents, présentés successivement au sujet à environ 5 minutes d'intervalle.

Les profils consistaient en une mise en accélération selon une pente constante, identique pour chaque essai (33°/s) avec une valeur terminale de l'accélération fixée à 2,3 ou 4 g (N_2, N_3, N_4). Le plateau était maintenu pendant 20 secondes. La centrifugeuse était alors arrêtée selon un profil de décélération maintenu constant d'un lancement à l'autre.

Chaque protocole niveau était précédé d'un lancement d'entraînement correspondant à un profil N_3. Ce lancement portait donc à 4 le nombre total des lancements de centrifugeuse pour chaque protocole de ce type.

La différence entre les protocoles "Z-Niveau" et X-Niveau" vient donc de la variation de l'orientation de la tête relativement à l'axe d'application du facteur de charge.

La condition "X" signifie que c'est l'axe X de la tête qui coïncide avec l'axe d'application du facteur de charge. (C'est-à-dire toujours l'axe X de la nacelle). Dans ce cas le sujet est complètement allongé parallèlement au plancher de la nacelle.

Pour la condition "Z", c'est selon l'axe Z de la tête qu'est appliqué le facteur de charge. Dans ce cas, le sujet est placé dans une position demi-couchée où l'axe Z de la tête est perpendiculaire au plancher de la nacelle.

Protocole pente

Dans le protocole pente la valeur de l'accélération atteinte en plateau était de 4 g quel que soit l'essai.

Les pentes de mise en accélération et de décélération pour les trois lancements constituant ce protocole étaient classées en pente faible, pente moyenne et pente forte (P_1, P_2, P_3). L'asservissement de la centrifugeuse est effectué en vitesse. C'est donc la valeur de la pente de mise en vitesse angulaire qui est prise comme référence pour déterminer les trois traitements P_1, P_2 et P_3. Ces valeurs ont été respectivement fixées à 14°/s², 24°/s² et 34°/s² pour les départs, 14°/s², 18°/s² et 24°/s² pour les arrêts.

Comme dans le protocole Niveau, l'accélération en plateau était maintenue pendant 20 secondes. Il n'était pas pratiqué de lancement de démonstration préalablement à l'application de ce protocole.

Tâche et consignes du sujet

La tâche et les consignes données aux sujets restaient identiques quel que soit le protocole.

Le sujet était d'abord familiarisé au repos avec le maniement du levier de commande du système optique. La tâche qui lui était assignée consistait à indiquer en permanence la direction de l'horizontale ou de la verticale perçue tout au long du lancement. Il devait donc agir sur le repère lumineux orthogonal de façon à compenser le déplacement angulaire perçu de son corps par rapport aux références de verticale et d'horizontale. Les consignes données avant chaque essai précisaient que seuls les mouvements angulaires survenant dans le plan sagittal du corps devaient être pris en compte.

Les sujets recevaient également la consigne de noter l'apparition de symptômes de cinétose pendant ou entre les lancements. Les symptômes devaient être rapportés verbalement au fur et à mesure de leur apparition.

Le critère d'arrêt impératif de l'expérimentation avait été préalablement défini comme l'apparition de nausées franches mais d'intensité modérée.

A l'issue de chaque lancement, il était demandé au sujet de commenter les sensations perçues et d'essayer de préciser les symptômes éventuels de cinétose.

Lorsqu'un protocole avait été complété ou lorsqu'un sujet avait demandé l'arrêt de l'expérimentation, il était immédiatement procédé à une évaluation du niveau de malaise. Cette évaluation portait sur les symptômes subjectifs (nausées, pesanteur ou inconfort épigastrique) et sur les symptômes objectifs (pâleur, sueur etc...).

Traitement des données

Les signaux acquis sur support magnétique ont été traités par le centre de calcul. Après numérisation des bandes, les signaux ont été traités sur l'ordinateur IBM 370/3031 par le programme PAM.PGM.FM. Le graphiquage des données sur table BENSON a été effectué à l'aide du programme GRAPH 2000.

RESULTATS

Les illusions de déplacement angulaire perçues par les sujets se sont révélées très caractéristiques. Lors des essais en "X", une sensation de bascule coordonnée du corps dépassant rarement 90°, est perçue à l'arrêt comme au départ. Il s'agit d'une bascule vers l'avant au départ et vers l'arrière à l'arrêt. Par contre, les sensations perçues lors des essais en "Z" sont qualitativement plus désorientantes. L'analyse détaillée concernant ces illusions est présentée par ailleurs (9).

Les résultats obtenus dans le domaine des cinétoses ont permis de justifier "a posteriori" la répartition selon la réceptivité effectuée "a priori" en deux groupes.

Le groupe de sujets réceptifs a expérimentalement démontré sa sensibilité aux effets des accélérations de Coriolis en centrifugeuse.

Le tableau I résume les principaux symptômes observés lors des protocoles niveaux pour les différents sujets du groupe réceptif. Tous les sujets de ce groupe ont été plus ou moins sévèrement affectés par les lancements successifs. On note accessoirement que les lancements avec le facteur de charge selon l'axe X de la tête (Niveau X) se sont montrés nettement moins provocateurs que lorsque le sujet a la tête redressée (Niveau Z). Dans cette dernière configuration, 3 sujets sur 6 ont atteint le critère d'arrêt de l'expérimentation dès le troisième lancement.

Par contre, le tableau II montre clairement que les sujets non réceptifs (à l'exception d'un) n'ont été affectés par aucun des protocoles. Les résultats observés dans le protocole pente sont parfaitement identiques à ceux des protocoles niveaux.

A partir des données recueillies à l'aide du potentiomètre solidaire du dispositif optique, les valeurs de la sensation de déplacement angulaire du corps perçues lors des départs et des arrêts de la centrifugeuse, ont pu être déterminées pour chaque sujet et chaque lancement.

Les valeurs moyennes selon les différents protocoles, traitements et groupes, sont présentées au tableau III.

L'examen direct des valeurs moyennes met en évidence d'assez importantes différences entre sujets "A" et "MR". Toutefois on note également que les valeurs d'écart-type sont généralement élevées. Ceci traduit des variations inter-individuelles fortes à l'intérieur des groupes, par ailleurs de faible effectif. Si on examine les données brutes, il apparaît cependant un fait évident : tous les sujets du groupe réceptif perçoivent des sensations de grande amplitude surtout lors des arrêts de la centrifugeuse.

TABLEAU I

MANIFESTATIONS DE CINETOSE OBSERVEES
DANS LE GROUPE DES SUJETS RECEPTIFS.

Protocoles "Niveau"

Condition / SUJETS	PROTOCOLE X N		PROTOCOLE Z N		CATEGORIE
	Nombre de Lancement	Symptômes	Nombre de Lancement	Symptômes	
DUS.....	4	Néant	4	Nausée ++ Paleur ++ Sueurs +++	Réceptifs.
COR.....	4	inconfort épigastrique - sueur	3*	Nausée +++ Sueurs ++ Paleur ++ Tétanie	
MAR.....	4	Nausée + Paleurs + Sueurs +	3*	Nausée +++ Paleur ++ Sueurs ++	
CHA	4	Néant	4	Légères pesanteurs épigastr. chaleur	
LEG.....	4	inconfort épigastrique - paleur	3*	Nausée++ Paleur++ Sueur++	
VAL.....	4	inconfort épigastrique léger.	4	inconfort épigastrique marqué	

* - Arrêt pour malaise.

TABLEAU II

CINETOSES OBSERVEES DANS LE GROUPE
DES NON RECEPTIFS

Condition / SUJETS	PROTOCOLE X N		PROTOCOLE Z N		CATEGORIE
	Nombre de Lancement	Symptômes	Nombre de Lancement	Symptômes	
MIG	4	Néant	4	Néant	Non Réceptifs
HAL.....	4	-	4	-	
LEJ.....	4	-	4	-	
DUC.....	4	-	4	-	
CLE.....	4	-	4	Nausée + Paleur + Sueurs +	
DOL.....	4	-	4	Néant	

TABLEAU III

VALEUR MOYENNE ET ECARTS-TYPES DES AMPLITUDES MAXIMALES PERCUES A L'ARRET ET AU DEPART

NR : Non réceptifs (6 sujets)

R : Réceptifs (6 sujets)

$\bar{X}$: Moyenne (en degrés)

S : Ecart type

* - Moyenne sur 5 sujets.

Groupes / Conditions Expérimentales		DEPART		ARRET		PROTOCOLE
		NR	R	NR	R	
X N_2	$\bar{X}$	23,83	33,10	34,5	59,67	X NIVEAU
	S	15,55	25,13	33,29	27,29	
X N_3	$\bar{X}$	25,83	54,50	30,83	79,50	
	S	17,99	18,35	20,05	31,05	
X N_4	$\bar{X}$	35,5	53,00	30,66	81,00	
	S	19,69	22,58	12,24	38,57	
Z N_2	$\bar{X}$	23,33	*41,40	35,5	*59,40	Z NIVEAU
	S	10,94	18,11	17,18	21,67	
Z N_3	$\bar{X}$	36,00	*50,20	44,00	*78,60	
	S	14,62	27,19	19,83	19,44	
Z N_4	$\bar{X}$	29,66	*44,80	47,75	*76,20	
	S	20,81	17,06	7,15	19,44	
P 1	$\bar{X}$	23,67	50,50	21,17	85,00	PENTE
	S	17,14	23,35	13,89	40,51	
P 2	$\bar{X}$	26,67	53,67	24,17	91,50	
	S	18,74	23,64	19,16	31,84	
P 3	$\bar{X}$	27,33	47,33	32,00	93,17	
	S	17,14	22,74	28,11	37,92	

La représentation des amplitudes moyennes de déplacement perçues à l'arrêt et au départ, selon les protocoles et traitements (fig. 1,2,3) permet de mieux se rendre compte de la nature du phénomène.

La figure 1 considère les résultats obtenus avec le protocole pente. Les deux groupes de sujets apparaissent ici clairement distincts avec des amplitudes perçues beaucoup plus élevées pour les sujets réceptifs, principalement lors des arrêts de la centrifugeuse. Il faut rappeler que dans ce protocole le sujet subit l'augmentation du facteur de charge selon l'axe X de la tête. La situation n'est pas désorientante et les sensations de déplacement du corps sont bien définies.

Une série de tests de t menée sur les différents traitements montre qu'il existe à l'arrêt une différence très significative entre les deux groupes de sujets. Pour les départs, seule la pente moyenne passe le seuil de signification de 5 %, la pente faible le manquant de très peu (t = 2,21 pour 10 ddl).

Lorsque l'on considère les protocoles niveaux, on retrouve les mêmes caractéristiques que pour le protocole pente. La figure 2 présente les valeurs moyennes obtenues avec le protocole X Niveau. Là aussi, il existe des différences importantes, surtout lors des arrêts entre les deux groupes de sujets.

Ces différences à l'arrêt de la centrifugeuse sont statistiquement significatives pour les niveaux 3 et 4g. Par contre, au départ, seul le niveau 3 g est significativement différent selon les groupes au risque 5 %.

Ces résultats sont également obtenus, quoique un peu moins nets, avec les lancements en configuration Z (fig.3). On peut ici penser que le caractère perturbant des illusions sensorielles pour les sujets réceptifs altère leur capacité à décrire clairement leur sensation. Comme dans le cas précédent, seuls les niveaux 3 et 4 g sont significativement différents entre les deux groupes à l'arrêt. Au départ, on ne met pas en évidence de différence statistique.

Dans tous les cas, on peut noter que les amplitudes perçues au départ sont nettement inférieures pour les deux groupes, à celles de l'arrêt. Cette donnée pourrait être interprétée dans le sens des résultats obtenus par GUEDRY et BENSON (6). Toutefois les calculs d'intensité de la stimulation permettent de montrer que, dans nos conditions expérimentales, l'arrêt et le départ de la centrifugeuse ont une dynamique difficilement comparable.

DISCUSSION

Les résultats obtenus lors de la présente étude sont à rapprocher des estimations de vitesse angulaire post-rotatoire, rapportées par REASON pour deux groupes de sujets sélectionnés à l'aide d'un questionnaire sur le mal des transports (15). Pour différentes impulsions de vitesse, le groupe de sujets réceptifs montrait une "réponse sensorielle au-dessus du seuil" significativement plus élevée que le groupe des non-réceptifs.

Dans notre expérimentation, c'est l'amplitude d'une sensation illusoire induite par les accélérations de Coriolis qui est prise en considération.

Toutefois si l'on considère également le temps d'établissement au maximum de l'illusion, on peut se rendre compte que la "vitesse moyenne d'établissement" est également plus élevée chez les réceptifs que chez les non-réceptifs.

Ce type d'étude, où les variables étudiées représentent l'image centrale de la stimulation vestibulaire, diffère sensiblement des techniques de cupulométrie.

En effet, comme le souligne REASON il s'agit d'une représentation du codage nerveux de l'intensité de la stimulation, alors que la cupulogramme repose sur une notion de temps.

La faible effectif de la population étudiée doit cependant inciter à la prudence. La claire démonstration faite par DOBIE (5), à l'aide d'une grande population, de l'absence de corrélation entre le cupulogramme et la susceptibilité au mal des transports ne doit pas être perdue de vue.

Il existe de plus des difficultés inhérentes à l'estimation subjective de l'amplitude d'un déplacement angulaire. GUEDRY (7) a très clairement souligné ces problèmes. Les consignes données au sujet ne portaient, en fait, pas sur l'amplitude du déplacement du corps. C'est la direction de verticale perçue que le sujet devait indiquer. Dans certaines illusions de mouvement, on conserve, en effet, une notion relativement bien définie de la direction du "haut" et du "bas" (10). L'expérience a montré que si cette estimation du haut était relativement aisée dans la configuration "X", elle devient beaucoup plus difficile, surtout lors des arrêts en "Z".

Ce fait, rapproché de l'incidence forte des cinétoses dans cette dernière position n'est, en soi, pas surprenant. Il est bien connu depuis les travaux de MANNING et STEWART, confirmés par de nombreux auteurs, que l'apparition des symptômes de cinétose est dépendante de l'orientation du sujet par rapport à la stimulation.

De même, BENSON, a étudié depuis longtemps l'effet de l'orientation du vecteur gravité sur les réponses post-rotatoires obtenues pour différents axes (1).

Dans notre cas, il faut également tenir compte d'un facteur complémentaire, qui est la variation de l'intensité du vecteur gravitaire appliqué, sans que sa direction soit modifiée.

Vu sous l'angle de l'intégration multi-sensorielle (15) on est donc tenté de penser que la stimulation utriculaire qui se produit lorsque le sujet est en position couchée (X), participe à l'atténuation des symptômes et au caractère peu désorientant des illusions.

Il faut alors faire l'hypothèse que la stimulation canalaire liée aux accélérations de Coriolis est identique dans les deux configurations.

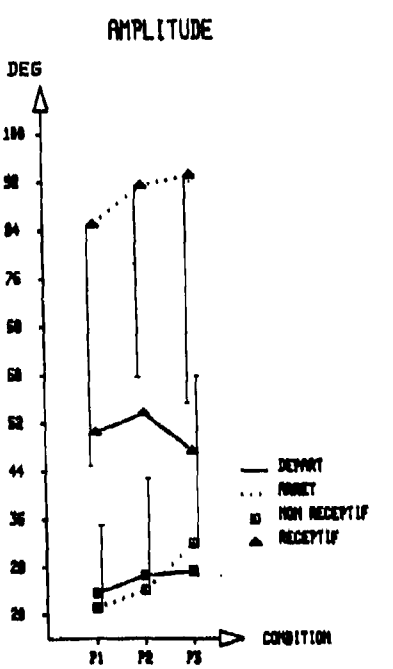

Figure_1
Amplitudes moyennes perçues au départ et à l'arrêt pour le protocole "pente" (l'écart type est figuré uniquement pour les arrêts).

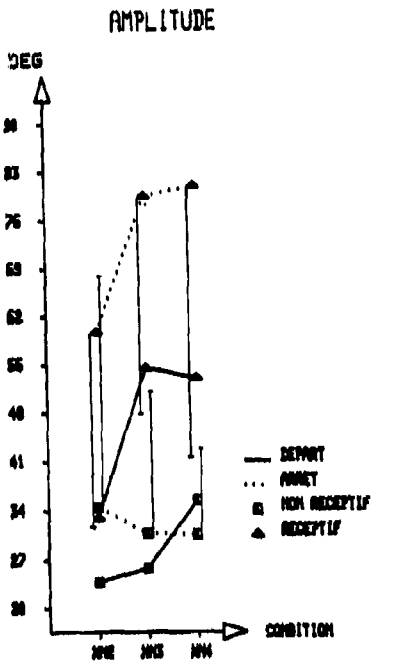

Figure_2
Amplitudes moyennes perçues au départ et à l'arrêt pour le protocole "X niveau" (l'écart type est figuré uniquement pour les arrêts).

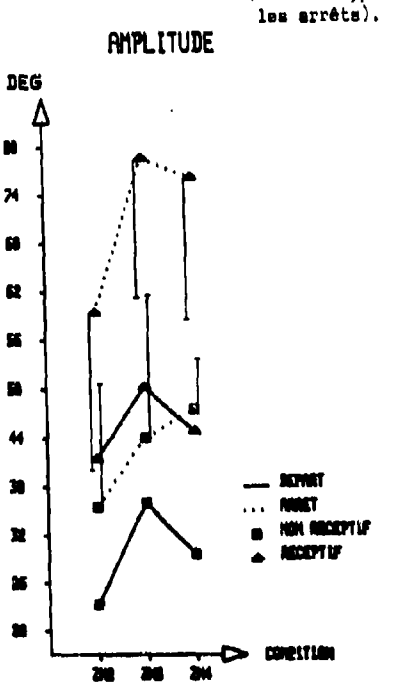

Figure_3
Amplitudes moyennes perçues au départ et l'arrêt pour le protocole "Z niveau" (l'écart type est figuré uniquement pour les arrêts)

D'un autre côté, il faut également remarquer que lorsque le sujet est en position "Z", seule la macula sacculaire est stimulée par les variations du facteur de charge associées à l'effet de l'accélération de Coriolis sur les canaux. Dans ce cas, le contenu informatif du message sensoriel afférent est probablement différent de celui qui est produit par la double stimulation utriculaire et sacculaire en "X"

Quels que soient les mécanismes impliqués à l'origine des phénomènes observés, il est utile de chercher à comprendre comment peuvent s'intégrer ces résultats dans les modèles conceptuels du mal des transports. Le modèle proposé par OMAN (14) fait le lien entre les modèles d'orientation spatiale et ceux supportant la "Théorie des Conflits". Il est à ce titre particulièrement intéressant. Cet auteur distingue trois points clés pouvant influencer l'importance des conflits sensoriels et leur traduction sous forme de cinétoses. Ces points clés sont respectivement, le "gain" des capteurs (matrice S), une matrice K représentant le "modèle dans le modèle" et enfin une matrice T représentant la sensibilité du sujet aux conflits.

D'une manière ou d'une autre, si l'on prend en compte nos résultats, les coefficients des matrices pondérant la variable d'état (estimé d'orientation) et ceux réglant la sensibilité au conflit (matrice T) doivent se trouver liés.

CONCLUSION

Les réponses concernant les amplitudes perçues de déplacement angulaire, lors des départs et des arrêts de centrifugeuse, diffèrent sensiblement selon la sensibilité au mal des transports du groupe de sujets étudié. Ces amplitudes, évaluées par un jugement d'orientation de la verticale par rapport au corps, sont beaucoup plus importantes dans le groupe de sujets classés "Réceptifs".

En fonction de l'orientation du facteur de charge et du protocole d'essai, ces résultats sont retrouvés plus ou moins nettement, mais d'une manière constante. Les variations de l'orientation du facteur de charge par rapport au sujet jouent considérablement sur l'apparition des symptômes de cinétose. La contribution utriculaire aux caractéristiques peu désorientantes des illusions et à la faible incidence de cinétose dans la position couchée (X) est hautement probable.

D'une manière générale, il semble que le système d'orientation spatiale des sujets non réceptifs soit plus à même de "filtrer" les messages sensoriels destinés aux centres nerveux supérieurs.

Conceptuellement, il semble donc exister un lien entre l'image centrale de l'intensité du mouvement illusoire et la sensibilité aux conflits générateurs de mal des transports.

En pratique, les structures qui pouvaient rendre compte de ce type d'interactions sont encore loin d'être identifiées et celles-ci restent donc très hypothétiques.

De plus, si les observations réalisées au cours de cette étude peuvent présenter un intérêt dans la compréhension des mécanismes régissant la susceptibilité aux cinétoses, leur portée pratique en matière de prédiction reste faible.

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DISCUSSION

MONEY: Did you by any chance apply this test to the French cosmonaut. If so, what sort of results did he show?

LEGER: An experiment was designed to see what kind of effect reclining a pilot when sustaining G loads would have and we just bumped into this effect in susceptible and non-susceptible subjects.

GUEDRY: The resultant force was being aligned with the Z axis of the subject, is that it, and during deceleration the subject was then setting the vertical or horizontal?

LEGER: The subject was through the start, acceleration, and the stop suppose to constantly align the target lights with the horizontal and vertical.

OMAN: You presented data for the two different populations, the receptive and non-receptive. For an individual subject how consistent were his magnitude estimates and was there any learning effect?

LEGER: We took a great deal of attention to avoid order effects. The susceptible subjects all exhibit, especially at the start, very large displacements.

OMAN: I guess what I'm really asking is does a susceptible subject always show a very large magnitude estimate? How much of your overall variances is due to the differences between subjects versus the variability within an individual subject?

LEGER: Yes, the same subject usually has very consistent patterns. Sometimes you get some susceptible subjects with small responses at the start but when they are very susceptible they are large at the start.

PREFLIGHT AND POSTFLIGHT MOTION SICKNESS TESTING OF THE SPACELAB 1 CREW

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SUMMARY

The four Spacelab 1 payload crew members, as experimental subjects, were exposed to a variety of motion sickness tests. Contrary to expectation, the crew member who was most susceptible to these tests was the least susceptible to space motion sickness, and the crew member who was most susceptible to space motion sickness was one of the least susceptible to these tests. On the third day after returning from the mission, one of the preflight tests (KC 135) was repeated, and all of the crew members were found to be non-susceptible. Statements of generalities will have to wait for the accumulation of more experimental subjects.

INTRODUCTION

In order to increase understanding of space motion sickness (1), the susceptibility to motion sickness in the four Spacelab 1 payload crew members was measured using a variety of motion stimuli on Earth and in aircraft. It was thought that a comparison of susceptibility to these stimuli with susceptibility to the stimulus of spaceflight might reveal something about the nature of space sickness and might even suggest a technique for predicting susceptibility to space sickness or a technique for effective prehabituation. (In this context, the term "prehabituation" refers to the acquisition of adaptive resistance, by repetitive exposure to a motion stimulus on Earth, with resulting resistance to space sickness.)

In general, it was expected that "Subjects who show consistently low susceptibility to motion sickness in the full range of provocative tests ..." (2) would tend to show low susceptibility to space motion sickness also.

PROCEDURES

All four payload crew members were exposed to all of four different formal tests of susceptibility preflight, and their susceptibilities were also assessed by two additional informal tests and by their responses to a questionnaire. Their susceptibility to space motion sickness was assessed during the mission, and one additional test of susceptibility was also performed shortly after landing from the mission.

1. The Space Sled Simulator:

The subject was in a seated position in an enclosure on a sled that moved along rails (Figure 1). The movement was oscillatory so that the subject was accelerated from side-to-side (along his Y axis) sinusoidally at 0.2 Hz with a peak acceleration of 0.135 G. The subject was blindfolded and used a joy stick to indicate his perception of the sled's velocity. He abstained from alcohol or relevant drug ingestion for the 24 hours preceding the test, and he was instructed to describe verbally any symptoms experienced and to call the test to a halt when he experienced slight but unequivocal nausea. If the subject did not reach that level of sickness and did not halt the test, it was stopped after an arbitrary maximum time of 10 minutes. After stopping, the subject's nausea, pallor, sweating etc. were recorded and the degree of sickness was estimated according to the scale of Graybiel et al. (3).

Figure 1. The Space Sled Simulator, DCIEM Toronto.

2. The Precision Angular Mover (PAM):

The subject was restrained in a seated position in a closed capsule (Figure 2) and he was rotated at 20 rpm about an Earth-horizontal axis that passed through his head's Y axis; that is, he was tumbled head-over-heels (4). The instructions for the subject regarding the end point etc., and the scoring of the sickness, were the same as for the Space Sled Simulator test (above) except that the subject continuously counted out loud, backwards by twos from 1000 (this provided "alerting" and was also an indicator of the subject's well-being). During the rotation the subject kept his eyes open and viewed the inside of the rotating capsule (stabilized vision, (4)).

Figure 2. The Precision Angular Mover, DCIEM Toronto.

3. Ingestion of Deuterium Oxide:

The subject drank 2 ml of deuterium oxide per kg of body weight, and then lay supine for 15 minutes. At 15 minutes he assumed a (i) left-side-down orientation, at 25 minutes (ii) right-side-down orientation, at 35 minutes (iii) supine, at 45 minutes (iv) left-side-down, at 55 minutes (v) right-side-down, at 65 minutes (vi) sitting upright, and then starting at 75 minutes, with changes every 10 minutes as before, the orientation sequence (i) to (vi), above, was repeated. At the end of each orientation period, the degree of "motion sickness" was estimated according to the scale of Graybiel et al. (3). The instructions to the subject regarding alcohol, drugs, the end point etc. were the same as for the Space Sled Simulator test (above), except that the stimulus was halted by maintaining an upright posture.

4. Head Movements During "Zero g" Parabolas:

These flights are known to be provocative of motion sickness (5). In preparation for these formal tests in April 1983, the subjects had refrained from flying zero g parabolas for the previous three months. They were restrained only by a locked but loose lap strap in a seat in NASA's KC 135 aircraft (Figure 3). The aircraft flew 20 consecutive "parabolas" such that periods of near weightlessness of approximately 25 seconds were achieved (Figure 4). In fact the aircraft followed a free-fall trajectory and can only be said to be flying an ellipsoid (M. Lampton, personal communication). However, the flight path is at least similar to a parabola, and since the term "parabola" is in common use in this context, it will continue to be used here.

Figure 3. NASA's KC 135 aircraft.

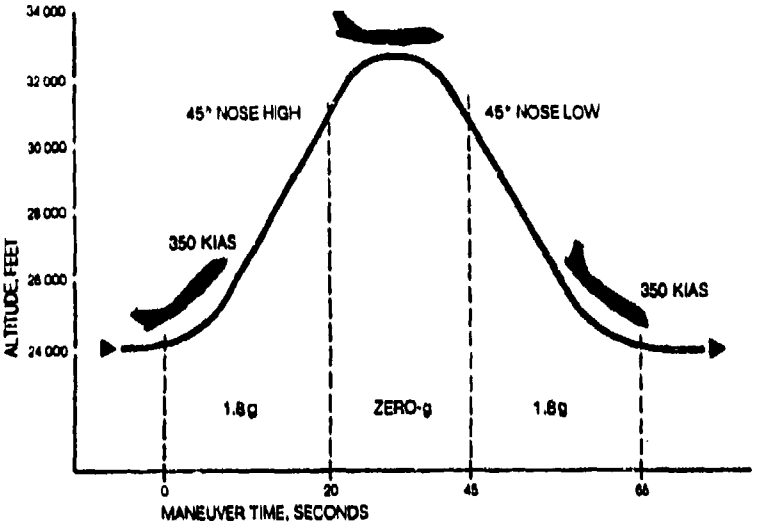

Figure 4. Flight path of KC 135 aircraft in producing weightlessness.

During each 25-second weightless period, starting with the first such period, the subject hung onto the arm rests with the hands only, elbows out, and feet on the floor, and with eyes open he made head movements in time with audio signals from a tape recorder (beeps every 1.5 seconds), touching his head to the seat's head rest, then to his knees, the head rest, etc., with a complete cycle of these nodding movements every 3 seconds. The head movements continued only for the duration of each weightless period, and during the hypergravity parts of the flight, the subject kept the head upright and motionless. The degree of motion sickness was scored according to the technique of Graybiel *et al.* (3) after each parabola.

In this test, the subject stopped making head movements at a milder end point, epigastric discomfort (before nausea), because the flight continued in spite of motion sickness so that the provocation could not be completely stopped by any individual who began to suffer early in the flight. This KC 135 test was also repeated with the payload crew on the third day after returning from the Spacelab 1 flight, to see whether the space flight would have an influence on the susceptibility to the test.

5. Miscellaneous Activity During "Zero g" Parabolas:

In addition to the above four formal tests of susceptibility, records were kept of the crew members' susceptibilities on an earlier KC 135 flight (March 1979) for H-reflex testing, circularvection testing, and "floating familiarization" during the same parabolic manoeuvres. On one such flight for each subject, if the subject did not reach the level of "stomach awareness" in the course of his other activities, then he made the stereotyped head movements until that level was reached or until 15 parabolas of stereotyped head movements had been completed. In preparation for these "miscellaneous" flights, some of the subjects were permitted to take antimotion sickness drugs. These KC 135 flights were made four years before the formal zero g parabolic tests (without drugs) described in Procedure 4.

6. Reversing Prisms:

Motion sickness signs and symptoms were also recorded during head movements while wearing left-right reversing prisms, a procedure employed to allow the subjects to become motion sick slowly for the purpose of allowing each individual to observe at leisure his own buildup of symptoms and his own recovery when motionless. The movements made were not standardized, and each subject decided for himself how much walking and how much head moving to do. The degree of sickness experienced was recorded.

7. Questionnaire:

The crew members also completed questionnaires that indicated their other experiences with motion sickness, from childhood to the present.

8. Motion Sickness During Space Flight:

The experiences with motion sickness during the Spacelab 1 flight were carefully monitored, recorded, and assessed.

9. Head Movements During "Zero g" Parabolas -- After the Mission:

On the third day after landing from the Spacelab 1 mission, the four crew members were tested again in the KC 135 aircraft as described in Procedure 4 (20 parabolas with stereotyped head movements during the weightless periods). This was done to determine whether the 10 days of weightlessness had influenced susceptibility to motion sickness from this stimulus.

RESULTS

1. The Space Sled Simulator:

None of the four subjects showed any signs or reported any symptoms of motion sickness in response to this stimulus. This stimulus must therefore be considered insufficiently provocative (subthreshold) for these four subjects. Three other payload specialists, who were similarly candidates for the Spacelab 1 flight assignments, were also tested at the same time, and two of these three did report mild but definite symptoms on the sled. Nevertheless, for purposes of revealing differences between the four who flew on Spacelab 1, the test was not revealing.

2. The Precision Angular Mover (PAM):

This test was imposed on the subjects twice: in February 1979 and in April 1983. The severity of the resulting sickness was expressed in total "Graybiel points" (3) per minute of stimulus, since a subject who reached a given level of sickness in a shorter time was considered to be more susceptible than a subject who reached the same level of sickness but required a longer time to do so. The results are indicated in Table 1.

Table 1. Severity of sickness in response to horizontal axis rotation on the PAM, "Graybiel points" per minute.

Subject	Feb 79	April 83
A	1.8	0.9
B	0.2	0.2
C	0.6	0.5
D	2.0	1.8

3. Ingestion of Deuterium Oxide:

None of the three subjects who took this test showed any signs or reported any symptoms of motion sickness in response to this stimulus. As with the space sled simulator, this heavy water stimulus must be considered insufficiently provocative (subthreshold) for these subjects, although previous less-provocative heavy water tests (6) on ten normal subjects resulted in six who did suffer sickness. Of the seven payload crewmembers who were offered this test, three declined to volunteer for it, and one of these three (Subject C) was assigned to the mission so that only three of the four SL 1 crew were tested. The fourth payload specialist who took this test (but did not fly on SL 1), Subject L, did show definite motion sickness signs, and reported definite symptoms, as a result. the heavy water positional nystagmus was rated moderate in Subject A, strong in Subject B, strong in Subject D, and very strong in Subject L.

4. Head Movements During "Zero g" Parabolas:

For these formal tests in the KC 135, in March/April 1983, the subjects were requested to abstain from any alcohol consumption or relevant drug ingestion for the previous 24 hours. All subjects observed the drug restriction, but they all failed to receive the request regarding alcohol restriction and all reported modest alcohol consumption during the previous evening. Subjects A and B completed the full 20 parabolas with head movements and had essentially no signs or symptoms. Subject C completed the full 20 parabolas with head movements and reported no symptoms but showed some slight sweating after the 20th parabola. Subject D showed signs and reported symptoms as follows (signs and symptoms were unchanged for subsequent parabolas unless indicated):

Parabolas 1 to 4: no signs or symptoms
5: possible increased salivation
8: cold sweating 1, pallor 1, salivation 1
10: flatulence at that time only
11: facial pallor increased to level 2 (moderate)
13: mild epigastric awareness
15: pallor lessened to level 1, salivation reverted to normal
16: flatulence at that time only
20: epigastric awareness gone, cold sweating gone, but pallor 1 still present

The relative susceptibilities to these tests, in order of decreasing susceptibility, was judged to be DCAB.

5. Miscellaneous Activity During "Zero g" Parabolas:

These flights took place on Tuesday 13 March 1979 (one flight, 40 parabolas) and on Friday 16 March 1979 (morning trip 60 parabolas, afternoon trip 40 parabolas).

Subject A: On the March 13th flight this subject was wearing the Transderm Scop scopolamine anti-motion sickness skin patch. He completed the 40 parabolas of other activities without any motion sickness, and did no stereotyped head movements. For the March 16th flights he took oral scopolamine/

dexadrine and again suffered no motion sickness, even after doing 15 parabolas of stereotyped head movements on the morning flights. During the afternoon flight he suffered a faint headache that could have been related to the motion experienced.

Subject B: This subject used no drugs for any of these flights. On the March 13th flight he experienced some stomach awareness briefly, a little burping and "slight flatulence". On the March 16th morning flight he experienced only some belches, and his stereotyped head movements on that flight elicited only two more belches. On the March 16th afternoon flight he experienced no motion sickness.

Subject C: This subject used no drugs for any of these flights. He reported no motion sickness on the March 13th flight or on the March 16th morning flight, and on the March 16th afternoon flight he reported only a stomach awareness that was not changed by the stereotyped head movements.

Subject D: This subject took oral scopolamine/dexadrine before the March 13th flight and before the March 16th afternoon flight, but no drug before the March 16th morning flight. On the March 13th flight he reported no motion sickness. On the March 16th morning flight, he was noticeably inactive for many parabolas and he reported stomach awareness before starting his stereotyped head movements on the forty-first parabola. He vomited after only three of the head movements during that parabola (parabola 41). On the March 16th afternoon flight, after taking scop/dex at noon, he volunteered to do the head movements again and completed 15 full parabolas of (smaller than standard) head movements without any sickness.

The relative susceptibility shown on these three flights, was judged to be, in order of decreasing susceptibility, DBC(A). Subject A is bracketed because no measure of his susceptibility without drugs was obtained at this time.

6. Reversing Prisms:

It was difficult to assess susceptibility to this stimulus, since the subjects were free to walk and make head movements at rates and frequencies of their own choosing. However, the signs and symptoms were recorded as they occurred, and the observers formed a subjective impression of how much activity was required to provoke a given level of sickness in each subject and how much adaptation to the new visual situation was achieved.

Subject A: This subject, with relatively modest activity while wearing the prisms, suffered sweating, epigastric awareness, increased salivation, epigastric discomfort, belching, lethargy, intense epigastric discomfort (description resembles what is called "nausea" by others), and slight pallor. Walking was "with difficulty". At times, active movements were stopped to ameliorate symptoms. When the prisms were removed after 69 minutes, the subject's visual world appeared stable when head movements were made (suggesting that little adaptation had occurred).

Subject B: This subject suffered sweating, subjective temperature increase, belching, flatulence, yawning, epigastric awareness, dry lips, epigastric distress, slight nausea, and mild apathy. At times, active movements were stopped to ameliorate symptoms. When the prisms were removed after 74 minutes, the subject's visual world appeared stable when head movements were made (suggesting that little adaptation had occurred).

Subject C: This subject suffered "possible" epigastric awareness and sweating. The observer felt (subjective impression) that he was more ataxic than the others when walking with prisms on. When the prisms were removed the subject's visual world appeared stable when head movements were made (suggesting that little adaptation had taken place).

Subject D: This subject was judged to have experienced the most activity (walking and head movements) while wearing the prisms. He suffered brief epigastric awareness, subjective temperature increase, flatulence, belching, and "possible" pallor. He learned to navigate without intellectualizing, and kept walking for the entire period of prism wearing. When the prisms were removed the subject's visual world appeared unstable when head movements were made, and "reversed" horizontal nystagmus appeared prominently. The instability of his visual world, and the reversed direction of horizontal nystagmus, appeared occasionally for "up to 4 hours later". This subject was judged (subjective impression) to be the least susceptible to sickness from reversing prisms stimulation, and the fastest to adapt. Such relative ranking of susceptibility to this stimulus must be considered a general impression rather than a measurement.

The relative susceptibilities to the prisms test, in order of decreasing susceptibility, was assigned ABCD.

7. Questionnaire:

The four-page questionnaire is simply too voluminous to be reproduced here. As might be expected, all of these four subjects had experienced a wide variety of motions with very little resulting motion sickness. All of them rated themselves "less susceptible than most", which appears to be a reasonable rating for all of them, considering people in general.

On the basis of their amounts of reported exposure to motion and the amount of sickness reported, it could be decided that their relative susceptibilities, in order of decreasing susceptibility, was DABC, although this relative rating is based on very little evidence.

8. Motion Sickness During Space Flight:

The motion sickness experience of these four subjects in space flight, Spacelab 1, is described in detail in paper 35 of this symposium (7). It should be remembered that Subjects A and D took antimotion sickness drugs (scop/dex, in this case 0.4 mg scopolamine plus 2.5 mg dexadrine) before launch and

regularly thereafter, whereas Subjects B and C took no drugs before the launch and took them (Subject B took scop/dex and Subject C took promethazine 25 mg plus ephedrine 25 mg) only after motion sickness was well developed. Consistent with the many tests reported here, the subjects had received extensive classroom instruction and practical experience with motion sickness so that, although they could not all be described as motion sickness experts, they could be considered reliable and knowledgable observers of motion sickness.

To summarize the detailed account (7) of the spaceflight experience with motion sickness, Subjects A, B, and C vomited repeatedly, whereas Subject D was without symptoms, and their relative susceptibilities in spaceflight, in order of decreasing susceptibility, was judged to be BACD.

9. Head Movements During "Zero g" Parabolas -- After the Mission:

This stimulus, on the 3rd day after landing from the Spacelab 1 mission, provoked no signs or symptoms of motion sickness in any of the four crew members. After the 20 parabolas of stereotyped head movements, an additional 20 parabolas were flown for the purpose of other experiments, and the four crew members had no sickness during those parabolas either, in spite of much floating about.

DISCUSSION

The results are summarized in Table 2.

Table 2. Relative susceptibility of the four crew members, A, B, C and D, to the various stimuli, in order of decreasing susceptibility.

STIMULUS		SUSCEPTIBILITY most ----- least
Sled	Feb 79	- no sickness -
PAM (horiz axis)	Feb 79	D A C B
PAM (horiz axis)	Apr 83	D A C B
KC 135 (miscell)	Mar 79	D B C (A)
KC 135 formal	Mar 83	D C A B
Questionnaires		D A B C
Reversing prisms	Feb 79	A B C D
Space flight	Nov/Dec 83	(B)(A)(C)(D)
KC 135 postflight	Dec 83	- no sickness -

( ) indicates the crew member used antimotion sickness drugs

It is, of course, impossible to draw conclusions from an experiment with variable results and a subject population of only 4, especially since the day-to-day intrasubject variation in susceptibility is significant (R. Kennedy, personal communication). However, the number of subjects will increase with the Spacelab 4 and D1 missions, and with the flights of the Canadian payload specialists, and valuable insights can be anticipated after these flights have been completed.

In many of the preflight motion sickness experiences of the Spacelab 1 crew, it was "splitting hairs" to say that one subject was more or less susceptible than another. However, it does seem clear that Subject D was the most susceptible subject overall on the many preflight tests, and the least susceptible to space motion sickness. This finding was opposite to the kind of finding anticipated, even though anecdotal descriptions of similar findings have been encountered. Also, Subject B was one of the least susceptible subjects on the preflight tests and the most susceptible to space motion sickness. The possibility that resistance to space motion sickness is actually associated with susceptibility to other forms of motion sickness should perhaps be given serious consideration, although it should be remembered that all four of these subjects are less susceptible than most people in a general population. It is commonly held (8) that people who are extremely susceptible in one environment can be expected to be extremely susceptible in other environments also, but predictions regarding persons of more moderate susceptibility are less certain.

Any interpretation of the spaceflight results is complicated by the fact that the different tasks of the different crew members required different amounts of bodily movement and head movement at different times of the "day". Such interpretation is also complicated by the use of antimotion sickness drugs, especially since Subjects A and D took the drugs prophylactically and Subjects B and C took them only after motion sickness was well developed. It is perhaps possible that antimotion sickness drugs are spectacularly effective against space motion sickness in some, or one (Subject D), of the subjects and near-useless in some others. If this were so, it would seem to be impossible to reveal anything about a subject's "susceptibility to space motion sickness" by drug-free tests on Earth, if he took the spectacularly effective drug just before launch. In the three subjects who suffered space motion sickness, however (A, B, and C), the drugs that were used appeared to be only moderately helpful, and sickness occurs routinely in spaceflight in spite of standard medications (9,10,11).

The exceptional preflight test was the reversing prisms test, in which Subject D was judged to be least susceptible to the sickness and most adaptable to the new "environment". Unlike the findings in all the other preflight tests, the relative susceptibilities of the four subjects to this test (ABCD) was similar to the relative susceptibilities to space motion sickness (BACD, using drugs), which is a finding consistent with the suggestion (12) that the unusual loss of gain in the vestibulo-ocular reflex in weightlessness might be important in space motion sickness. Possibly, people who find that a change in the gain of the vestibulo-ocular reflex is provocative, and who adapt slowly to such a change, are people who are susceptible to space motion sickness. In considering such a possibility it is important to remember that the suggestive evidence is miniscule, complicated, and barely visible through the obscuring fog.

It was noteworthy that all four subjects suffered no motion sickness at all from head movements on the KC 135 zero g parabola tests on the third day after returning from the mission. This postflight result was unlike the results from the preflight parabolic stimuli, and it might be suggested that the adaptation to the weightless environment during the mission conferred immunity to the parabolic stimulus. It has been reported that space flight has conferred resistance to the Coriolis stimulus also (13), but such transfer of habituation did not, apparently, prevent the sea sickness that has been reported (anecdotally) in several astronauts who returned from space by capsules that landed in the sea. If reliable records of such sea sickness were available, it would be interesting to know whether the sea sickness occurred even in astronauts who spent enough time in space to become adapted (4 days or so) or whether it occurred only in those who were in space for shorter times.

Another possibility, concerning the lack of sickness in the postflight KC 135 parabola tests, is that random day-to-day variability in susceptibility produced that unusual result. Further tests of later Spacelab crews should settle the matter. If in fact the adaptation to spaceflight causes immunity to the KC 135 parabola stimulus, then it would seem reasonable to hope that adaptation to the KC 135 parabola stimulus might cause immunity to the spaceflight stimulus, but such a process of prehabituation would possibly be a peculiar one since the people who were suffering no apparent sickness might be benefitting from extensive exposure to the KC 135 stimulus, and the people who were suffering sickness from the exposure might be the ones who do not need the exposure (such as our Subject D).

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DISCUSSION

LENTZ: Can you tell us a little bit more about the duration of exposure on your reversing lenses portion back in February ' 79.

MONEY: I believe one of the subjects stoped after about 70 min. but it was intended to be about an hour and a half. Only if the subject decided he didn't want to endure any more of the nausea did it end much before that. I would like to add that we repeated the prisms goggle test two other times and by and large the results were the same. In February '79 we went about 90 min., the other times we went longer. As a matter of fact, one of the principal objectives for using the prisms goggles test was to acquaint our subjects with the difference between their responses in a quick test and to prolonged stimulation. The responses and the ordering across all three tests were pretty consistent. The most susceptible subjects may have exchanged, A and B may have changed places on some of the other two tests but by and large the results were consistent.

UNIDENTIFIED SPEAKER: What was the head position of the subjects during your heavy water tests.

MONEY: The subjects were supine for 15 minutes. Then they spent 10 minutes on the left side down and 10 minutes on the right side down, etc. This went on for approximately 2 hours. None of the 4 who flew actually showed any symptoms although they did show very strong positional nystagmus and one of the other subjects who has not yet flown did show good strong motion sickness symptoms as well as nystagmus. We'll have to wait until he flies to know whether there is anything interesting.

LACKNER: Did you have the opportunity to test the astronauts post-flight on provocative motion sickness tests?

MONEY: Postflight, we had access to the crew for only one provocative test, the KC 135 test. On that test, all four subjects were entirely immune and also floated about during subsequent parabolas, thoroughly enjoying the experience.

BLES: Why didn't you do post-flight tests with drugs?

MONEY: It was hoped that none of the subjects would take drugs before flight. As it turned out, two of them (because of requirements of other activities) did take antimotion sickness drugs just before launch.

VON GIERKE: If one believes in the conflict theory of motion sickness, it appears to me that in the prism goggle test one should not measure the degree of initial disturbance but the time required to adapt/reorient to the reversed perception. Do you have a measure of this time?

MONEY: We did record the apparent speed and extent of that reorientation and, in fact, our subject D was the one who adapted most quickly and completely. He learned to negotiate corners without intellectualising, and often wearing the goggles he experienced illusory movement of his visual world when making head movements. This was the subject who proved to be non-susceptible to space motion sickness.

GUEDRY: Did any of the tests, other than the reversing lenses test involve goal directed behaviors?

MONEY: The only other motion exposure that involved goal directed behavior was the informal part of the KC 135 flights, when the subjects floated about to get familiar with locomotion in weightlessness.

CLAUSSEN: Which gross modal do you presently build up as a consequence of your findings?

MONEY: We could not derive a gross model from our limited number of subjects, but the findings were consistent with the idea that susceptibility to space sickness involves sensitivity to retinal slippage (or the resulting reorganisation centrally) and inability to adjust VOR gain quickly. This model is still viable, but it is not likely to be the core of the problem.

KENNEDY: The criterion data show that the astronauts all took drugs during flight. Were they the same drugs, same time, etc? Thus the conclusion could be that drugs interact with individual and or predictive values of ground tests.

MONEY: Subjects A and D took scopolomine plus dexaedrine just before launch and regularly thereafter. Subject D was non-susceptible to space sickness whereas Subject A vomited repeatedly. Subjects B and C, both of whom vomited repeatedly, took drugs only after their space sickness was well established (B took Scopdex, C took promethazine plus ephedrine). It is possible that D had an unusually beneficial response to scopdex.

SIMULATOR SICKNESS: REACTION TO A TRANSFORMED PERCEPTUAL WORLD VI. PRELIMINARY SITE SURVEYS

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ABSTRACT

There have been numerous recent documented and anecdotal reports of aircrews experiencing psychophysiological disturbances, visual illusions and sickness following the use of flight simulators. Symptoms of simulator sickness occur not only during flight, but in some individuals, have lasted up to several hours post exposure. Furthermore, simulator aftereffects may be delayed; some aircrews report symptom onset as late as eight to ten hours post utilization. Incidents of simulator sickness have been documented in fighter, attack, patrol and helicopter simulators. These occurrences have been reported in both motion-base and fixed-base simulators, to pilots and other aircrewmen, as well as instructors. Preliminary data suggest that more experienced aircrewmen are at greater risk and that such factors as wide field-of-view and visual/inertial lag contribute to the problem. Simulator sickness represents a major obstacle to obtaining the full training potential from the vast inventory of flight simulators currently in use and under development. Obviously, the learning capability of an individual who is suffering discomfort generated by a simulator is greatly compromised. Moreover, there is the possibility that the visual and proprioceptive cues responsible for simulator sickness may contribute to negative transfer of training in actual flight.

Data on pilot experience and exposure factors, symptomatology, scores on postural disequilibrium tests, video-game performance and engineering design aspects in two different Navy helicopter simulators are presented, along with a brief review of past simulator sickness studies.

INTRODUCTION

Since World War II, training in simulators has become more popular, due to their economies in equipment and fuel, plus their attendant advantages of maintenance, availability and safety. Orlansky and String (1,2) have provided eloquent summary statements of their effectiveness. New types of simulators, such as those for training air combat maneuvering, air cushion vehicles and Skylab crews, seem to be in great demand. Unfortunately, there has been a recent increase in reports of discomfort and distress associated with the use of flight simulators.

Since the phenomenon of simulator sickness was first reported by Havron and Butler (3) and Miller and Goodson (4), a large body of anecdotal and documented evidence has accumulated. This evidence suggests that simulator sickness symptomatology resembles motion sickness and other forms of distress which occur after exposure to altered and rearranged sensory information (5).

Humans, along with other species, adapt biologically to ecological changes; otherwise, they do not survive. Sometimes, this adaptation involves long-term evolutionary modifications of structure and function. However, less permanent modifications occur which capitalize on the plasticity of the human central nervous system. These short-term changes may be considered under the general rubric of adaptation to the environment; but persons who study learning, habituation, acclimatization, adjustment, compensation, satiation, and other time-course events may be examining similar processes. These short-term changes in human behavior and performance make simulator sickness an important problem.

It is axiomatic that motion is the basis for motion sickness; and the constellation of symptoms which occur under some force environments illustrates that this is an ecological change to which humans have not yet adapted. Man-made systems, such as ships and aircraft, have introduced new force environments more rapidly than would be the case for most ecological changes.

Opinions or conclusions contained in this report are those of the authors, and do not necessarily reflect the view or the endorsement of the Navy Department.

It is our view that motion sickness is a normal consequence of exposure to certain moving environments (6). The incidence, time course, symptom mix, et cetera, follow certain rules, some of which are known. Frequently, if the stimulus parameters of the force environment are sufficiently specified, our technology can predict the resulting incidence of sickness (7). It follows that, to the extent that the real system produces motion sickness, a simulator which replicates the real environment is liable to induce the same responses. However, when a simulator produces effects which are dissimilar from those which ordinarily occur in the aircraft, then the adequacy of the simulator must be challenged.

REPORTED CASES

Simulator sickness is a problem of recent vintage. Its occurence was first reported in aircraft simulators (3), then in driving simulators (8). Although trainers/simulators have been around for several years, it is only recently that wide field-of-view (FOV) visual systems have been incorporated into simulator design. It appears that the onset of simulator sickness corresponds closely to the introduction of wide FOV visual displays. This is not to say that wide FOV visual displays are the causal factor. As will be seen, and as has been documented elsewhere (5), 1983), the issue is far more complex.

Unfortunately, the data on simulator sickness is sparse. With respect to flight simulator sickness, nearly all the documented cases are found in military technical reports of limited distribution. A brief review of these studies follows:

Havron and Butler (3) published the first report of simulator sickness occuring in a flight trainer. In their study of the 2-FH-2 helicopter hover and autorotation trainer, they noted that 77% of the individuals exposed to the trainer experienced some type of symptomatology. They also noted that some effects lasted several hours following simulator flight and that there were delayed effects. In a later study of the same simulator, Miller and Goodson (4,9) found that 60% of the instructor pilots reported symptomatology, as compared to only 12% of the student pilots. This finding suggests that experience may be an important factor. Miller and Goodson also reported the occurrence of delayed effects in one instructor pilot who became "so badly disoriented in the simulator that he was later forced to stop his car, get out, and walk around in order to regain his bearings enough to continue driving" (3, p. 244).

One of the first attempts to document the problem in the Air Force was reported recently by Kellogg, Castore and Coward (10). They surveyed 48 pilots using the Air Force Simulator for Air-to-Air Combat (SAAC) and found that a majority (88%) had experienced some symptoms of simulator sickness (primarily nausea) during SAAC training. Of particular interest were the F-4 pilots, who reported delayed perceptual aftereffects occurring 8 to 10 hours following simulator flight. These included sensations of climbing and turning while watching TV, or experiencing an 180-degree inversion of the visual field while lying down. The authors cogently suggested that "the users of such (wide field-of-view) simulator should be aware that some adjustment may be required by pilots when stepping back into the real world from the computer-generated world."

In a study of flight simulator motion sickness conducted for the Canadian Department of National Defence, Money (11) reported that nearly half of the pilots using the Aurora simulator experienced sickness ranging from slight discomfort to mild nausea.

An investigation of simulator sickness in the Navy's 2E6 Air Combat Maneuvering Simulator (ACMS) found that 27% of the aircrews using the ACMS reported varying degrees of symptoms (12). The more experienced aircrews (over 1500 flight hours) had a higher incidence of symptoms than the less experienced flight crew. Dizziness was the most frequent symptom, followed by vertigo, disorientation, "leans," and nausea. The incidence of symptomatology was greater in pilots than in radar intercept officers (RIOs). The authors suggested that one reason for the reduced levels of simulator sickness found in the 2E6, relative to the Air Force SAAC, may have been the less intensive schedule of simulator time. Exposure duration and frequency appear to be potentially important variables, as has been found in other environments that produce motion sickness (7).

Frank (13) has reported that almost one out of every 10 individuals using the F-14 simulator (2F112) experienced symptoms of simulator sickness, and that close to 48% of the 21 aircrew sampled using the E-2C simulator (2F110) reported symptoms. Crosby and Kennedy (14) have documented cases of simulator sickness in the P-3C simulator (2F87), particularly at the flight engineer's position.

Tables 1 and 2 represent an attempt to collate the literature on simulator sickness occurrences in flight trainers. Table 1 presents a rudimentary categorization of the characteristics of four simulators where "sickness" has been recorded in formally reported studies. Table 2 summarizes the findings of the seven studies performed on the four simulators presented in Table 1.

Table 1. Flight simulator characteristics

Designation	2FH2	2E6	SAAC	2F87
Aircraft	Bell HTL	F4/F14	F4	P3-C
Type	Helicopter	Fighter	Fighter	Patrol
Mission	Hover train	Air/air combat	Air/air combat	Flight train
Base				
Type	Fixed	Fixed	Motion	Motion
Degree-of-freedom	-	-	6	6
Max g	-	-	.2	.8
Enhanced*	-	Yes	Yes	-
.2-.4Hz Component?	-	-	Yes	Maybe
Noise simulated	Yes	Yes	Yes	Yes
Vibration simulated	Yes	Yes	-	-
Cockpit				
Type	Open	Enclosed	Enclosed	Enclosed
No. crew stations	2	2	1	3
Display				
Type	Project	Project	CIG	CIG
Medium	Screen	Dome	8 CRTs	3 CRTs**
Source	Point source	Pt. source TGT proj	CIG & model	CIG & model
Content	Sky/earth	Sky/earth targets	Sky/earth targets	Sky/earth
Luminance	"Dim"	Mesopic	Mesopic	Mesopic
Resolution	"Blurred"	Soft	Good	High
Motion range	6DOF	6DOF	6DOF	6DOF
FOV Horizon (deg)	260	350	296	48***
Vertical	75	150	180	36***
Lag Visual	2-3xNorm	+.20"	+.28"	+.15"?
Inertial	-	-	-	-
Typical miss. length	-	30-50min.	45-60min.	4 hrs.

* Use of g suit, g seat, dim lights, etc.
** Pilot = 2; co-pilot = 1; flight engineer = 0.
*** One window.

Although it is tenuous to generalize from the studies reviewed, the following points emerge:

1. The reported cases are divided about evenly among fighter, transport a helicopter aircraft simulators.

2. Symptomatology has been reported in both fixed-base and moving-base simulators.

3. Flat-screen, dome and computer image generation (CIG) visual systems are all implicated.

4. Wide field-of-view is implicated

5. The greater the intensity or duration of simulator exposure, the greater the likelihood of symptom occurrence.

6. Visual screen illumination was dim or at mesopic levels.

7. Little attention has been paid to the role of visual and inertial lags. There are several anecdotal reports of excessive and noticeable lags. Comments have been made that simulator latencies are "out of specification."

8. There was more sickness in experienced pilots than in students.

9. Incidences ranged from 11% to 88%.

10. Adaptation occurred or was mentioned in 38% of the studies.

Table 2. Flight simulator studies

Authors	Hartman & Hatsell (1976)(15)	Kellogg et al. (1980)(10)	McGuinness et al. (1981)(12)
Type study	Survey	Survey	Survey
Focus of study	Incidence of sim. sickness	Incidence of sim. sickness	Incidence of sim. sickness
Simulator type	Fighter	Fighter	Fighter
Simulator designation	SAAC	SAAC	2E6
Trial duration	Unknown	2.5 hours	.5-1 hour
No. trials	-	5	1
Subjects Who	IPs	Pilot	Pilot
No.	114	48	66
Symptoms			
How obtained	Quest/int	Interview	Questionnaire
Onset	During	During/post	During/post
Max duration	-	All week	6 hours
Max % with symptoms	52	88	27
Max % quitting*	-	-	+
% Reporting			
Vomiting	2		
Nausea	14	79	9
Dizziness	-	7	17
Ataxia/kinesthetic	-	60	10
Sweat	-	54	
Pallor	-	-	-
Visual	50	71	8
Headache	32	-	6
Drowsiness/fatigue	32	-	-
Disorientation	52	-	11
Attentional	-	35	-
Habituation/adaptation	-	Some	-
Experience effects*	No	-	+
Instr/stud effects*	No	-	-

* + = Instructor or experienced person with greater effects.

11. Vomiting was rare.

12. Nausea, dizziness and ataxia were the most commonly reported symptoms.

13. Reporting of symptoms is not complete. For example, sweating, which is ordinarily associated with motion sickness, is not mentioned by the pilot unless he is queried, because he believes it is due to the heavy workload.

14. There are possible negative implications of simulator sickness which can be grouped into three broad categories:

a. Compromised Training. First, symptomatology may interfere with learning in the simulator through distraction. Secondly, since humans are flexible, trainees may adapt to unpleasant perceptual experiences. If new learned processes are not similar to responses required in flight, then the new responses comprise negative transfer to in-flight conditions.

b. Decreased Simulator Use. Because of the unpleasant side effects, simulator usage may decrease, or persons may lack confidence in the training that they receive in such simulators.

c. Simulator Aftereffects. The exposure to the simulator may result in aftereffects, or post-effects. These are not unlike the post-effects of other devices; but their relevance to safety (e.g., driving home) is not known.

Table 3 presents the remaining documented reports of simulator sickness.

Table 2. Flight simulator studies (cont.)

Authors	Havron & Butler (1957)(3)	Miller & Goodson (1958)(4)	Ryan, Scott & Browning (1978)(16)	Crosby & Kennedy (1982)(14)
Type study	Evaluation	Field exp	Field exp	Field exp
Focus of study	Training effectiveness	Etiology of mot. sickness	Motion effects	Off-axis viewing
Simulator type	Helicopter	Helicopter	Patrol	Patrol
Simulator designatioon	2FH2	2HF2	2F87	2F87
Trial Duration	30 minutes	Unknown	4 hours	4 hours
No. Trials	12	4	1	1
Subjects Who	Inst/stud	Inst/stud	Inst/stud	Flight eng
No.	36	10+	47	20+
Symptoms				
How obtained	Quest	Q/int*	Quest	Quest/int*
Onset	Dur/post	Dur/post	-	Dur/post
Max duration	24 hrs	-	-	-
Max % w. sympt	78	60 Inst 15 Stud	11	50
Max % quitting	-	-	-	-
% Reporting				
Vomiting				
Nausea	+			
Dizziness	+			
Ataxia/kinesthetic		+	11	50
Sweat				
Pallor				
Visual	+			
Headache	+		6	
Drowsiness/fatigue		+		
Disorientation				
Attentional	+			
Habituation/adaptation	Some	-	No	-
Experience effects**	-	-	No	-
Instr/stud effects**	+	+	No	-

* Symptomatology either not evaluated or not evaluated in detail.
** + = Instructor or experienced person with greater effects.

EXPERIMENTAL PLAN

Because of the sparse data on simulator sickness and its possible negative implications, the U.S. Navy has developed a protocol to systematically survey its flight trainers in order to: (a) ascertain the frequency of occurrence of the various symptoms; (b) determine if there are human performance side effects due to simulator exposure; and (c) determine the magnitude and duration of the effects. When collected, these data will be used to develop simulator design and procedural use methodologies for the amelioration of simulator sickness. These data will also assist in defining the etiology of simulator sickness and contribute to a further understanding of the mechanisms involved in motion sickness.

FIELD EXPERIMENT

Two U.S. Navy helicopter simulators were investigated in a pilot effort to test the protocol procedures and to determine if any changes in pilot performance, postural steadiness or symptomatology occurred as a result of simulator exposure.

MATERIALS AND METHODS

Subjects

Thirty-six designated Naval aviators flew the SH-3 helicopter simulator and 28 different Naval aviators flew the SH-2 helicopter simulator. All subjects were in good health at the time of testing and were qualified helicopter pilots.

Table 3. Simulator sickness incidence reports

Authors	Type report	Simulator	Focus of report
Sinacori (1967)(17)	Incidence	V/STOL	Simulation techniques
Kellogg, Castore & Coward (1980)(10)	Information	SAAC (F4)	Simulator sickness
Money (1980)(11)	Incidence & recommendations	CF140 (Aurora)	Simulator sickness
USN Message (1980)(18)	Requirements	2F87 (P-3)	Visual display upgrade
Wenger (1980)(19)	Incidence	2F87 (P-3)	Simulator sickness
Frank (1981)(13)	Incidence	2F110 (E-2)	Simulator sickness
Frank (1981)(13)	Incidence	2F112 (F-14)	Simulator sickness
Kennedy (1981)(20)	Incidence & recommendations	2F87 (P-3)	Simulator sickness
USN Message (1981)(21)	Guidelines	2F112 (F-14)	Aircrew readjustment
Frank & Crosby (1982)(22)	Incidence	2F117A (CH-46)	Psychophysiological disturbances

Simulators

The SH-2 simulator (Device 2F106) had a 144° X 32° (H X V) field-of-view which was generated by a "Vital III" calligraphic night CIG. The display was a four-window, three-channel, folded on-axis virtual image. The SH-3 simulator (Device 2F64C) had a 130° X 30° (H X V) field-of-view which was generated by a "Vital IV" calligraphic dusk/night CIG. The display was a seven-window, five-channel, folded on-axis virtual image CRT display. Both simulators had a synergistic, six-degree-of-freedom, 60-second motion-base system. The flight scenarios were relatively constant across subjects. The scenario consisted primarily of familiarization rides around the "local" simulated work area. The average exposure duration for each subject in the simulator was two hours.

Motion Sickness Questionnaire

The Pensacola motion sickness questionnaire (MSQ) was used to determine each subject's past motion-exposure history and susceptibility. It is an omnibus anamnestic form that has been item analyzed, empirically validated and cross-validated against a laboratory procedure for the prediction of motion sickness. MSQ scores are related to flight training success (23,24).

Symptomatology Categorization

Figure 1 presents the symptomatology diagnostic categorization worksheet utilized in this study. In an effort to improve its precision and utility, Wiker, Kennedy, McCauley and Pepper (25) expanded the five-point scale (three degrees of malaise, plus vomiting and vestibular sickness) used in the Pensacola diagnostic worksheet, to the seven-point scale shown in Table 4. Wiker et al. found that the seven-point scale was easy to use by different raters and yielded an inter-rater reliability of r = .95. Moreover, there is a high correlation (r = .80) between subject recordings of their symptoms and experimenter observations of vomiting.

DIAGNOSTIC CATEGORIZATION WORK SHEET

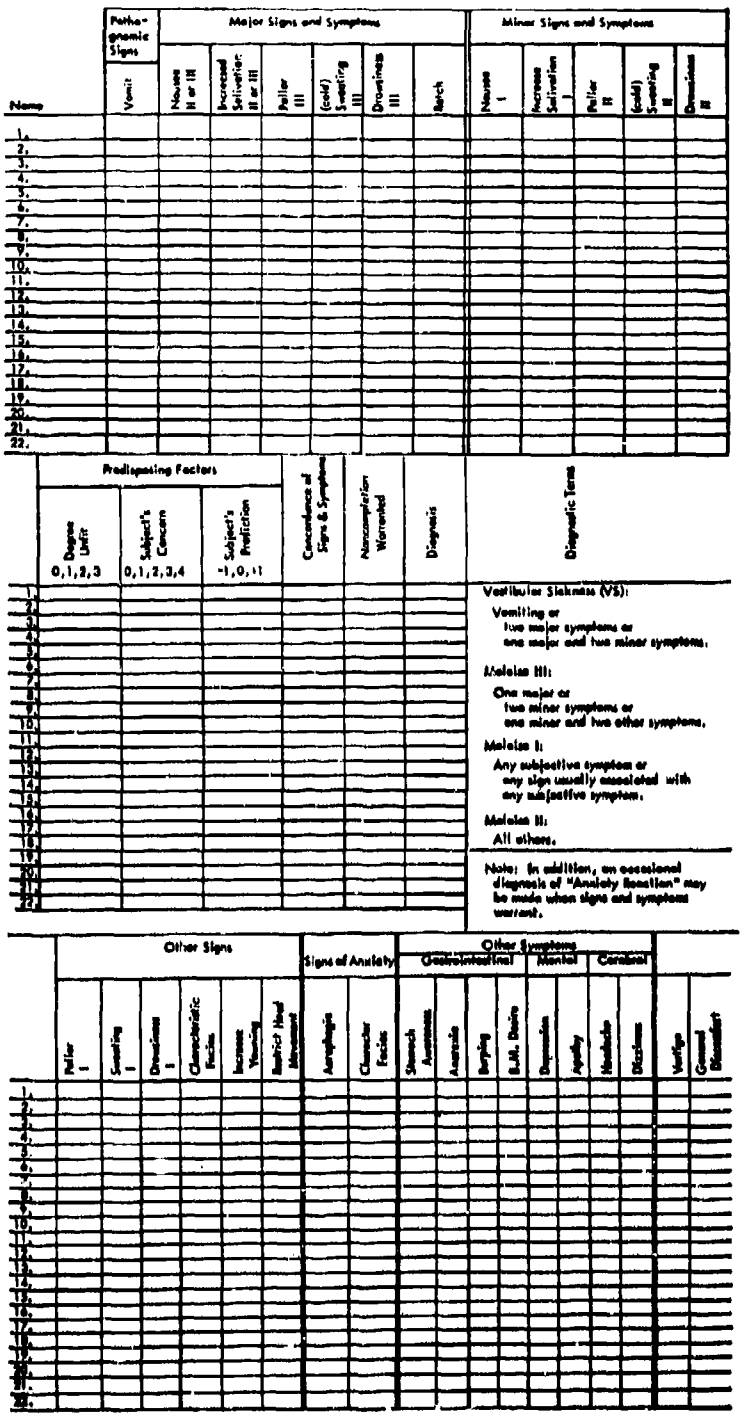

Figure 1. Diagnostic categorization worksheet. From Wiker et al. (25).

Table 4. Diagnostic criteria
for levels of motion sickness severity

7	Experimenter's report of emesis
6	Two major symptoms (including retch and subject's report of emesis)
5	One major and two minor symptoms
4	One major symptom alone or Two minor symptoms or One major and one minor symptom or One minor plus four other symptoms of which two (or more) are stomach awareness, sweating, drowsiness or pallor (depending upon whether pallor is scored)
3	One minor plus other symptoms
2	More than two other symptoms are reported
1	Any symptom related to motion sickness is reported
Ø	No symptoms are reported

Performance Testing

A commercially available Air Combat Maneuvering (ACM) Atari^{tm} video game (CX-26Ø1 #24) was used to test for possible psychomotor performance decrement as a result of simulator exposure. The ACM game is a two-dimensional pursuit tracking task which has been shown to be stable (26), related to other traditional tests of manual control (27), and appears to be a useful test for the measurement of pilot skills (28).

Postural Equilibrium/Ataxia

Two postural equilibrium tests were administered four times each, before and following simulator exposure, to each subject. These tests were:

1. Walk-Heel-to-Toe-Eyes-Closed (WHTEC). Subjects were started in an erect heel-to-toe position with arms folded across the chest and eyes closed. They were then asked to walk 1Ø heel-to-toe steps without side-stepping, at a rate that was neither too slow nor too fast as exhibited by the examiner. Subjects were stopped and their scores recorded when they either side-stepped, fell, or completed the 1Ø steps.

2. Stand-on-Preferred-Leg-Eyes-Closed (SOPLEC). Subjects were asked to choose their "preferred" or "best" leg, and to stand on that leg only in the erect position for a maximum of 3Ø seconds or again, until they side-stepped or fell.

Procedure

The MSQ, postural equilibrium tests and the ACM performance test were administered to all subjects prior to their entering the simulator. Thirty-one subjects "played" the ACM video game before taking the postural equilibrium tests and 33 the reverse. Each subject received four trials of 2.25 minutes on the ACM performance test before and after simulator exposure.

Following simulator exposure, subject reports and objective experimenter recordings of motion/simulator sickness signs and symptoms were performed. Postural equilibrium tests and ACM performance tests were also administered.

RESULTS

There were no significant differences in results between the experiments conducted at the two simulators, and so the data were pooled for purposes of analysis.

The results show that pre- and post-ataxia scores were not significantly different (P=.5), nor were video game performances (P=.5). Symptomatology scores are shown in Table 5. Thirteen percent of the pilots reported symptomatology related to discomfort of considerable magnitude. Nearly 40% reported two or more symptoms, and 80% indicated that they experienced one or more symptoms. MSQ scores were mildly ($p = .1Ø$) predictive of those subjects who had greater difficulty.

Table 5. Simulator/motion sickness symptomatology scores for the SH-2 and SH-3 helicopter simulators (N = 64)

	Symptom Rating	Frequency SH-2	Frequency SH-3
None	Ø	7	6
	1	11	16
	2	5	1Ø
	3	4	3
	4	Ø	1
	5	1	Ø
	6	Ø	Ø
Emesis	7	Ø	Ø

DISCUSSION

The symptomatology data in Table 5 are in concert with those of other studies and clearly demonstrate that simulators can alter an individual's physiological state. Whether such physiological alterations are of import, and what the exact nature of that alteration is, cannot be determined from this study. However, the aftereffects reported by Miller and Goodson (4) and Kellogg et al. (1Ø) clearly support the notion that perceptual sensory rearrangement can occur, and that such rearrangement may compromise saftety.

Personal reports by pilots to one of the authors (TB) reinforce this observation. One pilot reported that he experienced disequilibrium on the evening following a four-hour helicopter simulator (CH-53) ride, while he was in a movie theater. The disequilibrium occurred when the scene panned a landscape. In a second case (much like that reported by Miller & Goodson, 4), an aviator experienced a feeling of detachment while driving home about half an hour following a four-hour helicopter simulator (CH-53) exposure. He "found it mandatory to pull off the side of the road to avoid being a hazard to the normal flow of traffic," until he regained his awareness.

A third, and more telling, anecdote came from an instructor pilot with more than 5ØØ hours in a simulator. He claimed that while attempting to land a real helicopter in a clearing, he had the illusory feeling of being in the simulator, and recalling that "...simulators land high...," had trouble setting his aircraft down. Just as he was about to hand over control to his co-pilot, the vehicle made contact with the ground, and the disorientation subsided.

The tests of ataxia and psychomotor tracking performance failed to show simulator effects. While the reliabilities of the tests we used were marginal for these purposes ($\bar{r}$ = .45), Crosby and Kennedy (14) found significant ataxia problems in aircrew following exposure to the P-3 simulator (Device 2F87), with similar tests. However, the aircrew in that study, were in the simulator about four hours--considerably longer than in the present study. Thus, while better tests are desirable, it is our speculation that the postural equilibrium tests are probably sensitive enough to measure any meaningful effect generated by a simulator.

As expected, video game performance improved from Session 1 to Session 2. This improvement may have masked any simulator effect. Because scores change over sessions with motor skills tests (due to insufficient subject time), it may be better to emphasize cognitive ability tests in future studies. However, should post-effects turn out to be smaller than the session-to-session improvement in a battery of stabilized performance tasks, whether motor or cognitive, it might be possible to argue that there is mimimal disruption of the constructs they measure.

In the present study, the same experimental protocol was applied to two similar flight simulators. As evidenced in Table 5, the results from each closely paralleled, and we feel confident making comparisons between them. When different protocols are applied to different simulators, as has been done in the past (cf. Tables 1 and 2), comparisons are impossible. For example, two additional helicopter simulators, similar in detail to those reported on above, were surveyed for simulator sickness. The motion sickness symptomatology form administered was developed by the local flight surgeon and differed from that administered here. Sixty-four Marine pilots responded to a questionnaire after having "flown" in either the CH-46 helicopter simulator (Device 2F117A) or the CH-53 simulator (Device 2F121). The subjects were not required to sign the questionnaires and were about evenly distributed as "nuggets" (first tour) and very experienced pilots. Half of the subjects reported some side effects and 13 responded with dizziness. Dizziness appeared to be more prevalent in those individuals with less flight hours. The higher incidence of symptomatology reported in the CH-46 and CH-53, compared to the SH-2 and SH-3, is partly due, we believe, to the anonymity of the motion sickness form and the fact that a

more relaxed criterion was employed. In a different survey, conducted by the local squadron on the same Marine simulators, when subjects were asked whether they had experienced simulator sickness (yes or no), only 12% responded "yes." This latter incidence rate corresponds well with the finding of our field study.

GENERAL SUMMARY AND CONCLUSIONS

The present field study was designed to test an experimental protocol for the assessment of simulator sickness. It was found that a seven-point motion sickness symptomatology scale, postural equilibrium tests and MSQ were useful in determining the magnitude of the effects. Although the ACM video game did not detect any performance decrement, it was noted that it may be difficult to show effects, even in stable motor-skills tests, if means increase with practice. It was suggested that performance tests which asymptote more rapidly (e.g., cognitive and information processing) may be better candidates for future studies.

It is obvious from this pilot effort that the nature of the stimulus must be defined. Future studies should include scenario definition, measurements of visual and inertial lags and the resonant heave frequency of the simulator. Individual differences in pilot experiences must also be obtained. (A detailed listing of likely causal factors can be found in Frank et al., 15). In addition, aftereffects need to be systematically evaluated. It is also obvious from this study and the literature that simulator sickness is a problem. How large and how serious a problem it is has yet to be determined.

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DISCUSSION

LENTZ: Do you intend to take your test battery to the flight line (after an actual flight) and compare these results to the simulator?

KENNEDY: Yes, if possible. It is definitely a good idea to compare the post-effects of several moving environments. It would not surprise me if they occur.

CURRAN: Does your survey of simulators include both fixed and moving base?

KENNEDY: Yes. Simulators are divided about evenly between moving and fixed base; helicopter and fixed wing; point source vs. computer generated imagery; U.S.Navy vs. Marine Corps.; very wide (300°) vs. not so wide (120°) FOV.

LACKNER: Could you give us a summary of the difference in incidence in sickness for the trainees versus the experienced pilots?

KENNEDY: If you have lots of experience in aircraft, it sometimes places you in a less good position than if you have none, there are a series of incidences that have been reported on the real hazard of lots of exposures in simulators. The problem is yet to be fully sorted out.

SPACE MOTION SICKNESS MONITORING EXPERIMENT: SPACELAB 1

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SUMMARY

Symptoms and signs of space motion sickness and fluid shift were observed by 4 specially trained crewmembers on the physically demanding 10 day flight of Space Shuttle/Spacelab 1 launched on 11/28/83. Anonymous but detailed firsthand reports are presented. Three crewmen experienced persistent overall discomfort, and vomited repeatedly. Symptom pattern was generally similar to that seen in the individuals preflight, except that: prodromal nausea was brief or absent in some cases; facial pallor and cold sweating were usually absent; one subject experienced uncomfortable "stomach elevation". However, symptoms were clearly modulated by head movement, were exacerbated by unfamiliar visual cues, and could be reduced by physical restraint providing contact cues around the body. Drugs known to be effective in preventing motion sickness were judged helpful in limiting symptoms, including vomiting. Results support the view that space sickness is a form of motion sickness.

CONTENTS

BACKGROUND

Since the first report of "seasickness"-like symptoms by the Soviet Cosmonaut Titov in 1961, the incidence of similar symptoms have exceeded 30 percent in the Soviet Vostok/Voskhod spacecraft. Although there were no reports of sickness from astronauts in the smaller Mercury or Gemini vehicles, the incidence in the larger Apollo, Skylab, Space Shuttle and Soviet Soyuz/Salyut vehicles has approached 50% (Refs. 1, 2). The malady has become generally known as "space sickness", or more recently, "space adaptation syndrome". A close association between rapid head and body movements and the development of symptoms has been noted, which may explain the apparent lack of sickness in Mercury and Gemini, since crewmen could not move about inside the cabin of these vehicles. Susceptibility to space sickness has been highest during the first several days on orbit. On Apollo 10, one crewman executed deliberate head movements in an attempt to hasten adaptation on the first two days of the mission, but was forced to stop within a minute by developing symptoms. Movements were still nauseogenic on the seventh day, although stomach awareness was not produced until 5 minutes of head movements were completed. On Skylab, controlled provocative motion sickness tests were conducted for the first time (Ref.1). However, the testing did not begin until the 5th day of the mission, and by then most crewmen were virtually asymptomatic, even when making head movements in a rotating litter chair, a

stimulus which most of them had found quite provocative both preflight and postflight.

As we know it on earth, motion sickness is a condition characterized by stomach discomfort, nausea, vomiting, pallor, cold sweating, and other autonomic manifestations, and it is generally accepted that virtually everyone is susceptible provided the stimulus is appropriate and lasts for a long enough time (Ref. 3). Unfortunately, the physiological mechanisms underlying motion sickness remain poorly understood. Nonetheless, behavioral evidence derived from the wide variety of different situations which make us motion sick have offered some important clues (Ref.4), and have led to the development of "sensory conflict" hypotheses to explain the disorder: Sickness has been noted (Refs. 2, 5) to consistently occur in situations were man is passively exposed to certain real or apparent motion stimuli, or to conditions of "sensory rearrangement" in which the "rules" which define the "normal" relationship between body movements and the resulting neural inflow to the central nervous system have been systematically changed. Apparently, whenever the central nervous system receives sensory information on the orientation and movement of the body which are unexpected or unfamiliar in the context of previous sensory-motor experience, and this condition occurs for a sufficient interval of time, motion sickness can be expected to result. Motion sickness may therefore be a manifestation of a prolonged "overload" condition in brain centers which process body movement control and spatial orientation information. The physiological processes responsible for coupling brain areas responsible for movement control and those which mediate symptoms and signs of motion sickness have not been determined. However, it has been suggested (Ref. 5) that sensory conflict must in some sense be continuously functionally "averaged", normally subliminally, by physiological processes which determine the dynamic of symptoms and signs when conflict exceeds normal levels. This would account for the significant delay frequently seen in the first appearance of motion sickness symptoms, and the subsequent perseveration of symptoms and sensitivity to further stimulation observed after the stimulus is removed.

The notion that space sickness is simply another form of motion sickness is an attractive one. In the absence of gravitational loading, the four otolithic masses of the inner ear gravity sensitive organs would be expected to collectively assume new positions relative to the underlying sensory cells. The pattern of neural information flowing to the brain during all head movements would seem totally "unfamiliar", until the new pattern is learned by experience. To the extent that visual and tactile/proprioceptive orientation cues become ambiguous in weightlessness, one would expect episodes of disorientation to occur, and to be potentially provocative. Therefore, from the perspective of the "sensory conflict" hypothesis, the appearance of motion sickness symptoms during the first few days in weightlessness is hardly unexpected. However, exposure to weightlessness has other profound physiological implications as well. For example, removal of the normal gravitational load on the cardiovascular system produces an additional set of symptoms and signs (stuffy noses, puffy faces, spindly legs) associated with interstitial "fluid shift" from the legs to the head and thorax. Available evidence indicates that these physical signs of fluid shift are apparent in all crewmen to some degree immediately upon entering zero gravity, and are believed to be present throughout the entire period in weightlessness.

Based on available descriptions, the symptoms and signs of space sickness and motion sickness have generally been thought to be so similar that, as noted by Benson (Ref. 6), "in the absence of evidence to the contrary, parsimony dictates that space sickness must be considered as just another form of motion sickness". However, other hypotheses exist (reviewed in Ref. 7). For example, it has been speculated that perhaps fluid shift might induce nausea and vomiting through a direct effect on the central nervous system via increased cerebrospinal fluid pressure or a change in its chemical consituency. Alternatively, increased labyrinthine fluid pressure or constituency might produce pathological changes in the vestibular organs themselves. Evidence in favor of these notions is scant. However, it is important that these alternative hypotheses be ruled out. The impact of space sickness on crew efficiency now demands that more effective methods for prevention and treatment of space sickness be quickly developed. To proceed on a scientific basis, it is essential that the hypothesis that space sickness is a form of motion sickness not simply be accepted without systematic collection and scrutiny of appropriate scientific evidence. If space sickness is motion sickness, one would anticipate that the sickness intensity would modulate depending on the time history of head movements, and perhaps depending on the particular type of head movements which were made. As with other forms of motion sickness, one would expect visual, tactile, and proprioceptive cues to play a significant role in creating symptoms, and that when these cues were properly manipulated, symptoms could be alleviated. Existing crew reports generally have tended to support these expectations. However, compelling evidence has not yet been systematically collected and documented.

Research on space sickness has been complicated by a number of realities: Crewmen generally have operational responsibilities which make it impossible for them to prevent head and body movements prior to vestibular and motion sickness tests. Crewmen are legitimately concerned that their participation in such tests might subsequently compromise their physical effectiveness. For the same reasons, many crewmen take anti-motion sickness drugs prophylactically, (although the effectiveness of these drugs against space sickness has not yet been definitively established). Although crewmen have frequently described their symptoms in detail in medical debriefings, these reports have an anecdotal character, and have been regarded as sufficiently personal in nature that relatively few details have been documented in the open scientific literature. Although briefed on the problem preflight, most crewmen in the US and Soviet programs have lacked credible preflight experience and interest in the physiology and psychology of motion sickness and spatial orientation, and have been unfamiliar with the professional vocabulary of these disciplines. It is therefore not surprising that occasionally their reports have created some degree of confusion within the scientific and clinical communities. Recently, a serious effort has been made by the NASA Johnson Space Center to collect and interpret reports from Shuttle crewmen. Some of the results of this effort are presented at this meeting (Ref. 8).

Now that Spacelab is operational, investigators have the opportunity to more thoroughly train participating crewmen to perform research on spatial orientation, movement control, fluid shift, and motion sickness on orbit. In 1976, NASA selected a team of investigators from the Massachusetts Institute of Technology (USA) the Canadian Defence and Civil Institute of Environmental Medicine, and McGill University (Canada) to develop a group of interrelated experiments on human spatial orientation, vestibular function, and motion sickness on three Spacelab missions (Ref. 1). Complementary experiments in these disciplines are also being developed for the same missions by other investigator teams from the USA and Europe.

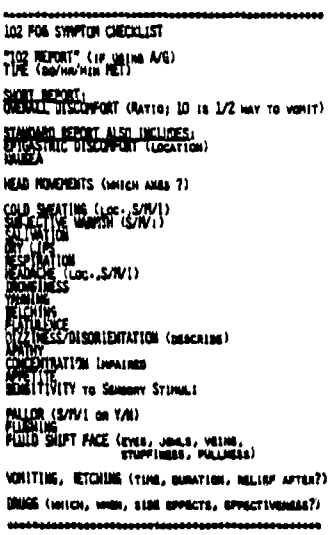
This report presents the operationally relevant results from space sickness monitoring and provocative testing experiments developed by the MIT/Canadian team, and flown on the ten day flight of Spacelab 1, launched from the Kennedy Space Center on November 28, 1983 in the orbiter "Columbia". As part of the experiment, accelerometers were used to continuously record head movements in two subjects, in order to quantitatively examine the hypothesis that the level of sickness is related to the level of activity in some subjects. Analysis of this portion of the data is not yet complete, and will be published elsewhere. Results of related motion sickness susceptibility tests conducted pre and post flight on this same mission are described in a second report at this meeting (Ref. 9). When considered together with the large body of results (in preparation) from the other Spacelab 1 vestibular experiments on visual/vestibular/tactile interaction, vestibulo-spinal responses, perception of limb position, and motion thresholds as well as experiments on mass perception, cardiovascular function and neuroendocrine regulation, these results may provide additional insight regarding the etiology of space sickness.

METHODS

Subjects were the 4 males, aged 35 - 53 at the time of flight, with no history or evidence of central or peripheral vestibular disease, as determined by a clinical otoneurologic exam. Two were professional NASA Scientist Astronaut/Mission Specialists, one of whom had flown in space previously as Science Pilot on the 59 day Skylab III mission in 1974. The other two were Payload Specialists, selected for the flight by the mission Investigators, and included one of the authors (BKL), an MIT vestibular researcher. All four were pilots. Three were current in high performance jet aircraft. All considered themselves "less susceptible than most people to motion sickness". For the reasons given earlier, our subjects were not naive, and functioned as observers.

All four crewmen (henceforth denoted as Subjects A- D) were asked to observe the time course of symptoms and signs of space sickness and fluid shift, the relationship of these to head movements, and the effect of visual, tactile, and proprioceptive spatial orientation cues on sickness intensity. Our principal objective was to obtain and document firsthand as complete a picture as possible of space sickness as it occurred on a physical activity intensive, multi-disciplinary Spacelab mission, while preserving the anonymity of results from the individual subjects. We asked Subjects B and C, who wore the head mounted accelerometers for extended periods, to make detailed reports on symptom status whenever symptoms changed, for subsequent correlation with the head movement records. For this purpose, a pocket voice recorder (Pearlcorder M420) was carried by each of the two, and a "symptom checklist", reproduced in Figure 1, was provided. Subjects A and D wore the accelerometers only occasionally, and were asked to use the checklist to make appropriate notes for use in postflight debriefing. (For various technical and programmatic reasons, objective physiological recording of space sickness signs was not attempted on Spacelab 1, except that a stethoscope was used by two crewmen on occasion to monitor abdominal sounds. However, on subsequent flights of this experiment, facial skin pallor and temperature will also be monitored.)

FIGURE 1: Symptom Checklist
Provided to subjects for documentation of symptoms and signs of space sickness and fluid shift.



Although we emphasized the importance of systematic reporting, we anticipated that if a crewman was to be able to provide frequent reports, often circumstances would not allow time to evaluate all the items on the checklist. In this situation, the subjects were asked to provide only a "short report", consisting of a single number. This was a numerical magnitude estimate of the intensity of overall discomfort. Subjects employed a method of reporting (Ref. 10) which was designed to produce a ratio scale. Instructions to the subjects, as they appeared in the experiment procedures, were as follows: "Pick a sensation magnitude of overall discomfort in the middle of the "moderate" range, halfway to vomiting. Call this standard "10". Estimate the magnitude of overall subjective discomfort with respect to it. If no sensation, say "absent". If just noticable, say "threshold".

When time permitted, subjects were to proceed to the remainder of the checklist and log the presence and where appropriate the intensity (on a 4 level absent/slight/moderate/intense, "S/M/I" scale) and location ("loc.") of 20 individual symptoms and signs. In constructing the checklist, which underwent some evolution

during the course of training, we included the individual elements of the Pensacola Diagnostic Criteria for Acute Motion Sickness (Ref. 11), plus additional items which experience has taught us are regularly seen, particularly in long duration motion sickness. In training, emphasis was placed on developing a consistent vocabulary to describe symptoms and signs.

"Nausea" was defined as that unpleasant sensation which, in its most intense form, is usually associated with the act of vomiting. One of our subjects (C) had very little lifetime experience with vomiting, and therefore was unsure how to distinguish "nausea" from "epigastric discomfort" or general "queasiness".

"Epigastric discomfort" was any sense of discomfort in the stomach, abdomen, or substernal (i.e. lower esophageal) areas which was not considered "nausea". "Epigastric awareness" was any sensation drawing attention to these epigastric areas which was not uncomfortable. Our subjects felt "awareness" referred to a very low level of epigastric discomfort, because they generally felt "awareness" could become unpleasant. For some, the distinction between "discomfort" and "nausea" was clear; for others it was not. We therefore encouraged them to augment reports of epigastric awareness, discomfort or nausea with any other comments which better defined the nature of their sensations.

"Slight" was to correspond to subjective threshold, or just above. "Intense" was the most severe level encountered, usually just prior to vomiting. "Moderate" was the range between slight and intense. Our subjects were instinctively precise individuals, and occasionally commented that they found it difficult to determine when a sensation changed levels under these definitions, and would spontaneously adopt a magnitude estimation method of reporting instead.

"Respiration" referred to respiration changes, including the slow, deliberate breathing pattern some of our subjects employed because it made them "feel better".

"Dizziness" referred to any uncertainty in orientation which outlasted head movement. "Vertigo" was a feeling of spinning or movement in a definable direction.

"Sensitivity to sensory stimuli" referred to the state of mind where sounds and voices, odors, heat, cold or tightness of clothing seem unusually strong, bothersome, or repulsive.

"Pallor" was judged visually, either using a mirror in the Shuttle Orbiter mid-deck, or by asking another subject to act as observer. Even after training, our subjects did not always have confidence they could describe pallor reliably on an absent/slight/moderate/intense basis, and felt that reporting "present", "absent", or "changed" would be better. We warned them that pallor might be masked in weightlessness by facial plethora resulting from fluid shift.

In addition to symptom monitoring, Subjects B and C agreed to attempt to make some deliberately provocative head movements in order to study specific stimulus/response relationships. The protocol consisted of a "susceptibility test" followed by a "symptom comparison" test, which explored the influence of eye closure and the axis of head movement. These tests were originally scheduled for the end of the "scientific working day" on the first, fourth, and ninth days of the mission. The susceptibility test was conducted as follows: If the subject was asymptomatic at the start, he would strap into a seat and cautiously make forehead to knee head movements (to a 1.5 sec. per movement cadence provided by a metronome) for 7 head movements, then rest for 10 seconds while making a symptom report, and then repeat until the very first symptom or until 5 minutes had elapsed. If symptoms were present at the start of the session, the susceptibility test was to be skipped, and the subject was to perform the "symptom comparison" test. The comparison test <u>required</u> a slightly symptomatic subject so that the head movement stimulus/symptom response relationship would be immediately obvious. Subjects first made up to 7 forehead to knee movements eyes closed, then repeated this eyes open, and ranked the two conditions in terms of capacity to provoke an increase in symptoms. After a pause for recovery, subjects then were to make 20 second intervals of 90 degree head movements (eyes open) successively in pitch, yaw, and roll, with rests in between, and then rank these movements in terms of provocativeness. The final decision whether or how far to proceed with these provocative tests was left entirely in the hands of the subjects. In flight the crewmen themselves would be in the best position to know whether symptoms could be constrained to levels such that they would not jeopardize their physical capacity.

Experiment training was conducted at intervals during 1979-1983, and consisted of:

1. Formal lectures at our institutions in Boston, Montreal, and Toronto on vestibular physiology, spatial orientation, and motion sickness, totalling approximately 50 hours, as part of the overall training for the MIT/Canadian experiments.

2. Practical training on evaluation of motion sickness symptoms and signs resulting from "chronic" stimulation achieved by wearing left/right vision reversing goggles while walking about the laboratory for several hours. Two sessions were conducted using a protocol similar to that described in Ref. 10, and a third session as described in Ref. 12, in which subjects used anti-motion sickness drugs. In these sessions, emphasis was placed on recognition of the various signs of motion sickness, and the order in which they appeared, and also on evaluating the dynamic relationship between the onset of head movements, the rise of discomfort and nausea, and the subsequent decline of these symptoms when head movements ceased.

3. Less formal practical training in evaluation of symptoms and signs while serving as subjects in the various preflight susceptibility tests as described in Ref. 9. Stimuli consisted of horizontal axis rotation (pitch mode), horizontal linear acceleration (y axis), forehead to knee head movements made while seated during the weightless phase of parabolic flight in the NASA KC-135, and the Brief Visual/Vestibular Interaction Test (Ref.13). To provide experience with some of the sensory aspects of orthostatic fluid shift which cannot be achieved in parabolic flight, subjects were exposed to 30 minutes of 10 degree head down bedrest. Photographs of their faces were taken at the end of the test.

4. A practical session in parabolic flight, during which some of the known effects of visual, vestibular, and

tactile cues on spatial orientation, disorientation and zero-g locomotion were demonstrated.

5. Practical training in the use of the accelerometer headband and belt mounted recorder/battery pack worn to document crew head movement.

The crew gained additional relevant experience as a result of lectures and tests related to the vestibular and cardiovascular experiments of the other investigators in the US and Europe. The Operational Medicine branch at the Johnson Space Center conducted modified CSSI tests (Ref. 14) on the crew in order to facilitate individual selection of an anti-motion sickness drug for use in flight.

NASA policy required two of our subjects to take anti-motion sickness drugs prophylactically as soon as they reached orbit. At the request of the life sciences investigators working on the mission, the other two attempted to remain unmedicated. Partly as a result of their training, NASA mission managers were content to leave the decision whether and how to treat space motion sickness up to the individual crewmembers, with the Flight Surgeon available for consultation if requested. Drugs chosen for use were 0.4 mg scopolamine/2.5 mg dexedrine in capsules for subjects A,B, and D, and 25 mg promethazine/ 25 mg ephedrine for subject C. Note that dexedrine was here used in half the 5.0 mg conventional dose so as to reduce possible side effects (e.g. insomnia) when multiple doses are taken. In addition, as indicated below, 2 subjects chose to evaluate the effects of a 10 mg. dose of metoclopramide (Reglan) on orbit.

RESULTS AND DISCUSSION

A. PREFLIGHT TRAINING AND TESTING

Table 1 shows the distribution of symptoms and signs observed in the four subjects during 6-9 preflight training/testing sessions. (Sessions in which subjects used anti-motion sickness drugs were excluded). By comparing columns, one can infer the apparent susceptibility of the individual subjects to this particular group of stimuli, and by comparing rows for a given subject, one can determine a subject's most frequently observed preflight symptoms and signs. It is significant that in conducting our training, we decided that we would never deliberately take a subject to vomiting. We felt this policy was appropriate in order to insure the continued cooperation of our subjects. In horizontal axis rotation and parabolic flight testing, we used the first appearance of unequivocal nausea as the test endpoint, and Malaise III (Ref. 11) was used in the CSSI tests conducted by NASA-JSC. When wearing the prism goggles, subjects only rarely went much beyond 10-12 on the "discomfort scale" described earlier. Had we chosen more provocative stimuli, or pushed our subjects harder, the results undoubtedly would have changed somewhat, and perhaps influenced our subjects' impression of what their "usual" motion sickness symptoms and signs were to some degree. In the context of the flight experiment results, it is significant that Subject B reported "sweaty palms", and was among those subjects reporting "substernal pressure", "constricted feeling in the chest", and/or a "tight throat" in some of these preflight tests.

TABLE I

FREQUENCY OF SYMPTOMS AND SIGNS DURING PREFLIGHT TRAINING/TESTING
(sessions: 2-4 parabolic flights, 1-2 prism goggles, 2 horiz.axis rot., 1 CSSI)

Subject Code	A	B	C	D
Total # sessions	7	9	8	6
Modality:				
Epigastric discomfort	5	4	4	6
Nausea	2	2		4
Vomiting				1
Cold sweating	5	5	4	2
Subjective warmth	2	1	1	2
Salivation (increase)	1			3
Dry lips		4		1
Respiration (change)	1	1		
Headache	2	1		3
Drowsiness	1	1		1
Yawning		2		
Belching	4	4		2
Flatulence		2		1
Pallor	5	2	2	4

Subjects A and B experienced significant symptoms while wearing prism goggles during the 1-3 hour test sessions, such that they were forced to stop their active head movements and close their eyes. In these individuals, we usually saw a pattern of response generally similar to that of 8 other subjects described in Ref. 10: Overall discomfort increased during each controlled head movement sequence, and decayed between sequences to a level which itself gradually increased with time, as if reflecting the cumulative effect of all previous head movement sequences. As time went on, the number of head movements which could be tolerated without stopping usually decreased.

After experience in training with the "overall discomfort" magnitude estimation technique, subjects A,B, and D felt that although the exact quality of the discomfort sensations each experienced was probably different between them, the scale had at least face validity, and in terms of functional capacity at any level on the numerical scale, they all had quite similar inclinations: At a relative discomfort intensity of 5, they would start limiting provocative head movements, but try to press on with assigned tasks. When the intensity rose to the 8-12 range, it was judged "definitely time to stop". Above this level, subjects feared symptoms would avalanche quickly, and it would be hard to avoid vomiting.

B. FLIGHT EXPERIMENT RESULTS

Science operations were conducted 24 hours a day on two alternating 12 hour shifts. Our subjects worked in pairs in the Spacelab module, a cylindrical pressurized workshop installed in the payload bay, and connected to the orbiter crew compartment by an open tunnel. One of the two orbiter pilots continuously monitored the mission status from the orbiter flight deck, while off duty crewmen slept and ate in the shuttle orbiter "mid-deck", located beneath the flight deck.

The investigators and the flight surgeons on the ground maintained a 24 hour listening watch on the voice communication channel, and monitored all available video transmissions. Fluid shift facies were evident in all crewmen when first seen on the television monitor an hour into the flight. Not surprisingly, their appearance compared well with with photographs taken previously on the ground during 10 deg. head down tilt. During the first two days, it was obvious that all subjects usually preferred to move relatively slowly, and generally maintained an upright posture with respect to the floor of Spacelab. Subject B was occasionally seen severely restricting his head movements.

It was not until after the landing that the investigators learned that during the early days of the mission, Subjects A,B, and C all experienced space sickness and all vomited repeatedly. Only Subject D was free of significant symptoms.

Believing that the principal objective of the experiment was to provide anonymous but well documented, firsthand case histories of space motion sickness, we have chosen to present the flight experiment results using transcripts from the pocket voice recorder tapes, the air to ground voice communication channel, and postflight debriefings. The latter were conducted at intervals during 5 days immediately after landing at NASA's Dryden Flight Research Center where the subjects remained to undergo life science postflight testing. As the on-orbit and postflight transcripts together totalled several hundred pages, we here present what we believe are the most significant excerpts. In some cases, we have paraphrased comments, as indicated [within brackets] either to improve clarity, reduce jargon and idiom, or to provide context or anonymity. Occasional reference is made to other experiments; details on these experiments are available in Ref. 15. The time and date of comments recorded during the mission are indicated using a standard NASA convention for denoting Mission Elapsed Time ("MET"). This best illustrated by example: An event occuring on the very first flight day, i.e. within the first 24 hours of MET is said to occur on Mission Day zero ("MD 0"). The time of an event occuring 14 hours, 7 minutes into the mission is denoted as 0/14:07. An event occurring exactly 24 hours later (on MD 1) would be shown as 1/14:07. Three orthogonal axes of head motion are referred to in "aeronautical" notation: with the origin at the center of the head, the X axis points through the nose, the Y axis through the right ear, and the Z axis towards the feet.

Overview: Subject B

Subject B was able to frequently log symptoms and signs as they occurred. The time course of his magnitude estimates of overall discomfort are shown in Figures 2 and 3 for the first two days of the flight, when space sickness was most intense. A score of 20 indicates a vomiting episode. The curves between the individual data points were interpolated based on additional notes made at the time, and by Subject B himself postflight. Scop/dex was taken at the times indicated by diamonds, and the period of presumed maximal effectiveness of the dose taken is indicated by horizontal bars. Metoclopramide was taken at the times indicated by triangles. An appreciation of the relationship between Subject B's activities, medications, and the rise and fall of overall discomfort can be gained by examining these figures together with reference to portions of Subject B's recorded commentary. For purposes of brevity, we have here omitted many of Subject B's reports on status of the individual symptoms and signs. A detailed discussion of symptoms and signs experienced by this crewman is presented later.

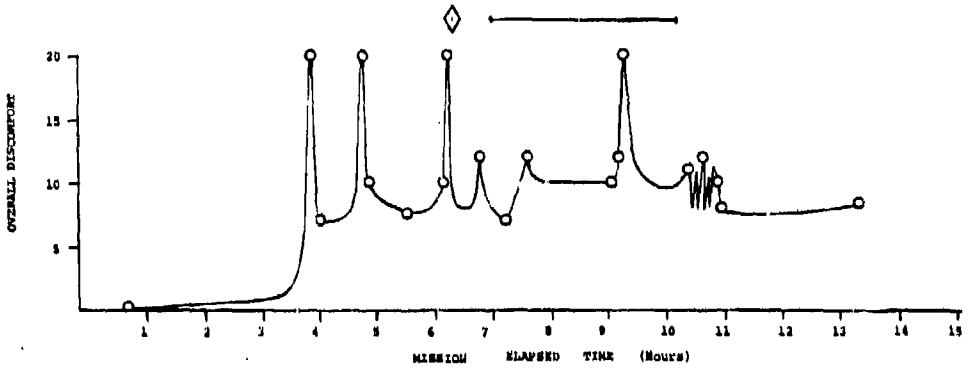


FIGURE 2 Magnitude Estimate of Subjective Discomfort vs. Time for Subject B
First Day on orbit. A score of 20 indicates vomiting. Additional details in text above.

Subject B noticed the inverted attitude of the orbiter cabin during the launch and ascent to orbit, despite

the thrust of the engines. After entering weightlessness when the main engines cut off, he was surprised to find that he continued to feel inverted. This sense of inversion persisted, despite his deliberate cognitive and physical efforts to reverse it, for roughly the next hour, and disappeared only after he unstrapped from his seat and moved about the mid deck area. (Somewhat similar illusions, typically lasting several hours, have been reported by Soviet cosmonauts; Ref. 16) Subject B and the rest of the crew then changed clothes, reconfigured the mid-deck, and prepared to enter and activate the Spacelab:

Subject B, 0/1:51 "..no restrictions of head motion, just normally taking it easy."

0/2:25 ".. noticing a little bit of stomach awareness occasionally, head movements have been restricted somewhat."

0/2:55 "Noticing a little bit of stomach awareness occasionally. Head movements have been restricted.., and a little bit of flatulence.."

0/3:55 "Just after Spacelab ingress. Did a couple of large maneuvers and immediately went to feeling just a little bit of stomach discomfort to nausea and several minutes later I vomited in a bag.."

0/4:45 "Again, virtually without warning - it's just a couple of seconds, really, no stimulus other than just active motion in the Spacelab. Vomited a second time...but felt immediate relief.. there was no real cold sweat or any [severe] stomach awareness ahead of time."

0/5:23 "..fullness of the head, and a little bit of stomach awareness occasionally on rapid head movement..trying to keep the head movements down to a minimum."

0/6:09 "again an incident of vomiting. There is virtually little warning before hand. Immediately after, I felt quite a bit better. I had possibly a little bit of cold sweating, clammy hands, but no pallor, and there has just been a constant level of stomach awareness. The vomiting episodes come on without any warning whatsoever. It's just one minute you're kind of hanging in there hacking it; the next minute you just really feel lousy, and you can tell that you have to throw up. You become nauseated; you go from about a 10 to an 18 inside of a minute. I'm currently sitting somewhere around a 10 I guess. I am able to work, but have to slow down, and don't really feel like making too many head movements. I've had a container and a half of water. At 0/6:09, I decided to go ahead and take one scop/dex... My only other sensation is a moderate sense of stomach awareness which is pretty continuous. A while back I tried to close my eyes, but that didn't seem to help at all..."

0/7:09 "..I've been restricting my head movements. Pitch axis seems to be by far the most provocative.. Just occasionally a slight amount of dizziness as I move my head very rapidly, but it doesn't persist."

0/7:37 "I have an experiment to do; running around a ratio scale 7 until I set up the second TV camera, and after floating down the module, I got up to about a 12, so I'm going to rest a minute without making head movements.... Closing my eyes definitely does not help."

0/9:11 " again virtually without warning - I had maybe two or three minutes warning - I was sitting in about a ratio scale of 10. There hadn't been any changes in symptoms for an hour and a half. So after doing some work, I realize that it was going up to about a 12, so I tried to get in the corner with some tactile cues, but had about a minute's warning. Realized that I was going to vomit again...feels much better once it's over with....my clammy hands are going away, but I still have stomach discomfort, and again it's localized up near the sternum. I call it moderate all the time, and it gets uncomfortable or severe just prior to vomiting. Very clear to tell just exactly when it happens... Head movements...again, it was fast rotations and pitches. I had just finished moving the foot restraint from one of the racks on the port side to the starboard side...There was a little cold sweating, a little yawning, a little bit of belching several minutes before.. It's difficult to get everything squared away so that you can burp properly".

In postflight debriefing, Subject B added "I noticed early on that I was really restricting my head movements, as you saw on TV during the Hop and Drop [experiment, 0/10:00-11:00]...Lots of times, I'd just look over and reach to grab something, or [my shift partner] would hand something to me, and I wouldn't even turn my head at all.

(In the "Hop and Drop" experiment (Ref.1), calf muscle electrical activity is monitored during unexpected "falls" and rhythmical hopping. In the absence of gravity, a "downward" acceleration is provided by adjustable elastic bungee cords which run from a torso harness to the deck of the Spacelab.)

"[During the bungee cord harness operations, starting at 0/10:20] I wasn't feeling great at all. One g hops and drops bothered me; I was getting quite a bit of oscillopsia [(apparent motion of the "seen" world)] in the Z axis when I was hopping, and that was kind of disturbing, and the 1-g drops, I didn't like at all. In fact.. I only did ten of them in [the Hop and Drop experiment]. The 2/3 g and 1/3 g were alot better, but I was [working] at about half speed [so I could] keep working and not get really sick again...I did all 15 of each of those, but it just took a long time. I'd go through a set, and stop and wait awhile, and let things calm down, and then start up again."

During the Hop & Drop operations at 0/10:47, Subject B noted that when he stopped moving, his symptoms moderated back to the preexisting baseline typically over 2-3 minute time course.

At 0/13:20, because he was suffering significant symptoms, Subject B reported "Skipping the [scheduled provocative] motion sickness test. Still feeling between a 7 and a 10. Unable to make any pitch head movements whatsoever."

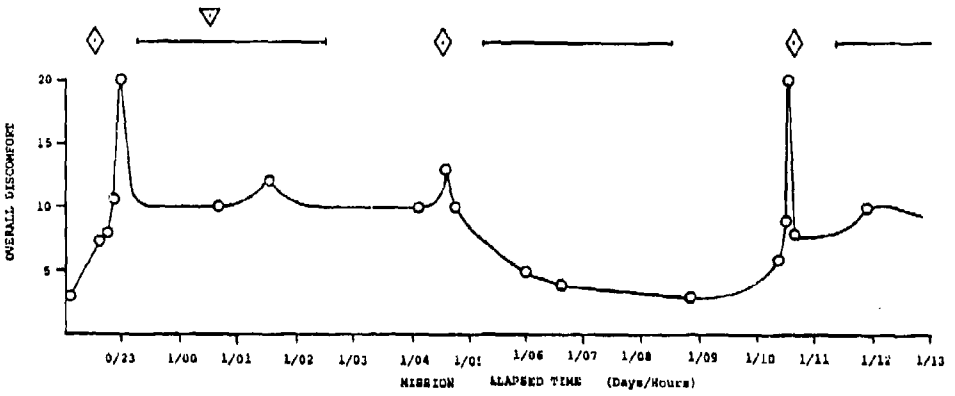


FIGURE 3 Subjective Discomfort vs. Time for Subject B, Second Day on orbit

Subject B obtained 7 hours of interrupted sleep. The following morning, he reported:

> 1/00:29 "During the night it seemed to really help to have my head velcroed down to the pillow..An hour and a half ago, just prior to the blood draws, I vomited again after taking ..scop/dex. I'm about to take a Reglan here in a couple of minutes.."
>
> 1/01:28 "Doing the blood work, trying to hold my head pretty well still and steady. Symptoms have been reasonably constant...Lately I've noticed a sort of tightening up in my lower abdomen after taking the Reglan.
>
> 1/4:33 "[While operating the Drop and Shock experiment, I] did alot of pitching head movements looking up at the control panel and down at the [Stimulus Isolation Unit]. Going to take a breather here: I've got myself up to about a 12- 14, and got cold, sweaty hands. Took a Scop/dex about 5 minutes ago. (yawn) and as you can tell there's some yawning... I'm just trying to remain quiet here for 5 or 10 minutes before we try to get on with the last part of [Drop and Shock].
>
> Subject B, 1/5:58 "Been [sitting here quietly as the subject in Drop and Shock] doing the..static recruitment curve, and have been in the harness [bungeed to the deck] for about the last hour or so. Having some tactile load, I have been able to keep still, and keep my eyes closed. Has helped immeasurably."
>
> 1/6:36 (after performing the Hop and Drop Experiment again) "Ratio scale of 4 and I've got slight stomach awareness occasionally on rapid head movements. Negative nausea. Except for the hopping and dropping, head movements have been really nil for the last hour and a half. It's been very comforting to stand in the harness, ..strapped down, and being able to close my eyes and not move. In fact, I almost fell asleep."

Note that, in contrast to his earlier reports, closing the eyes now seemed palliative. However, here full body restraint was provided by the experiment harness - with eyes closed, the subject could be sure he was not moving.

> 1/8:53 "the last couple of hours have been pretty steady; I've been floating around a 3 or so on the ratio scale..."

Subject B recalled that at 1/9:32-:47, while performing a Mass Discrimination experiment, he found the relatively small yawing head movements required to look alternately at his left hand holding a test mass and then to his right while logging the experiment results were mildly provocative.

> 1/10:30 "I was working getting set up for the Awareness of Position Experiment, and I was up to about an 8-10. Almost without warning, within one or two minutes to the endpoint, I got another vomiting episode...Immediately after the vomiting, most of the symptoms subside, and I feel quite a bit better
>
> 1/10:37 I went down to the mid-deck and took a scop/dex...Right now I'm sitting at about an 8, and am about to do the Awareness of Position Experiment.

(In the Awareness of Position Experiment, the subject is strapped blindfolded to a flat surface at the aft end of Spacelab, and after several minutes rest, is asked to point to various preestablished targets and to judge his limb position.)

> 1/11:55 Awareness of position experiment [floating eyes closed with] loose straps increase symptoms from an 8 to a 10.

Subject B elaborated in the debriefings "I was not [feeling too] keen during [the Spatial Awareness

experiment]. I was feeling kind of grim at the end of that, and I hadn't [actively] been moving for 20-30 minutes. But [loosely tethered], just the sensation that you could be moving and not detecting it was kind of grim, which is why I didn't like the eyes closed that much either.

On the third day, Subject B took a Reglan and a scop/dex on arising, and later noted that his abdomen was slightly sore when palpated. He repeated the scop/dex at 5-6 hour intervals that day. Discomfort magnitude estimates ran in the range of 1-3, with occasional slight increases due to twinges of stomach awareness on rapid head movements.

> 2/5:55 "..head movements don't seem to be much restricted. I've been able to come into the module upside down and float every which way today."
>
> 2/12:55 "Yaw is no problem, roll and pitch there is a little funny sensation in the head".
>
> 2/13:20 (his final report for the day) "No new symptoms, no problems. Have been translating between the flight deck and the Spacelab module at rapid rates. No real need to keep head movements to a minimum. Although I do notice that still pitch and roll head movements are more distressful than yaw."

By the fourth day, Subject B was "generally feeling quite good" until near the end of the day, when discomfort was again running in the 2-3 range. He was able to deliberately provoke further symptoms with head movements at the end of the fourth working day when he completed the "symptom comparison" test protocol:

> 3/2:48 "Feeling pretty good, just an occasional burp. No problems with head movements or motion whatsoever."
>
> 3/10:54 (while about to do a Symptom Comparison test) "Overall discomfort is about 2-3, stomach awareness, and it's high up, just under the sternum. No nausea. Head movements have not been really bothersome today except for several times when I made short reports. I have pretty much good mobility...A little bit of dizziness if I roll my head...ooh, that's not very good, I still don't like to roll my head very much. Pitch motions give me a little bit of disorientation. But the roll definitely is disorienting."
>
> 3/11:00 Symptom Comparison test results: Forehead to knee movements slightly more provocative when made eyes open. Subjective axis ranking: roll worst, pitch, forehead to knee, yaw least.

On the fifth day, symptoms remained at a very low level until the end of the day, when symptoms briefly rose slightly.

> 4/3:31 "I've been belching, and even though I feel comfortable moving about, after a rapid series of motions or head movements or something, I still get a few twinges of stomach awareness...In general it's slight, it doesn't persist very long, and I generally feel reasonably good.
>
> 4/5:17 "Still a little bit of fullness in the stomach and a feeling of pressing up in the sternum. No nausea. Head movements haven't been very much restricted, although again, I can tell that if I move alot, I don't really like it, and I want to keep my head from moving too rapidly."
>
> 4/8:37 (after a Rotating Dome experiment) " I was looking in the mirror doing some head rolls, looking at my ocular counterrolling..and after I finished that, I had a twinge of slight stomach awareness..I think I'll just keep my head quiet for a minute or so."

After the 5th day, Subject B experienced no further activity related symptoms. However, his sense of "fullness" beneath the sternum, headache, head fullness and congestion persisted. On the 9th day (8/23:30), he again performed the "Provocative Test" protocols, and was able to perform all the required head movements without any significant symptoms. However, slight oscillopsia was reported during rolling head movements.

Overview: Subjects A,C,& D

Turning now to the other subjects, Subject A summarized his experience at debriefing this way: " On day 1 [MD 0], I was not feeling too bad. I was under 0-G, and I knew I had to be just a little bit careful what I was trying to do.. then it began to get just a little bit worse. Day 2 was no better, if anything a little bit worse, and on the afternoon of day 2, I was really sick...I'd taken 2 scop/dex on the first day, and 2 on the second day, and [on the afternoon of the second day] I took a Reglan..and very shortly thereafter threw up. I vomited 4 times that afternoon...And although I never threw up on days 3 and 4, I was really feeling sorry."

Subject C: "When we did the activation of Spacelab,.. I [stayed] up a little longer than originally scheduled because it was really interesting to see, and also quite an emotional moment when the hatch [to the Spacelab module went] open and you go down the tunnel. When I went to bed, I felt a little queasy, but the real problem..was [our] hectic [next shift], when we had to run [the Rotating] Dome [visual/vestibular interaction experiment] and the whole [European Vestibular Experiment], with the many [technical] problems we had. I simply threw up twice... I remember I really didn't like ..to [float] through the tunnel.. Very automatically, you are trying to reduce the amount of head movements to a minimum...I agree with [Subject A] that you can still work quite effectively even if you are very sick, as I was. At the end of [my first full shift] I took one [promethazine/ephedrine], the only one I took during the entire flight,.. just before I went to bed. I thought that [would be] the best time to take it, because all the biological measurements were complete for that day...I felt much better the next day, and I was in absolutely good shape by the third day.... [On our first full shift] I think I did pretty much what I was supposed to do. In terms of speed, I think I could do it. I am [happy] to know that even if you subjectively don't feel too well, you can do [the work]. It wasn't really so bad. I probably should tell you that most of the time, the problems were really bearable...When I threw up, it was just a matter of a few seconds. You loose a minute or two.. after that, you feel much better. So in operational terms, I think I would probably do it exactly the same way [again].

0/5:45 "..A few minutes a go, I had to vomit. It came all of a sudden, without any warning, when I tried to eat an apple". Subject B's description: "We were sitting in the mid-deck. I'd just come back and we were talking. He was eating an apple. And right in the middle of eating the apple, he said ' aw, gee !', threw the apple in the air, and vomited just like that ! It was almost without warning."

0/14:39 " About 25 minutes ago, I came back from the middeck after returning the Plant Carry On Container, and as I came back into the Spacelab, all of a sudden I had to vomit a second time... Right now, I am feeling pretty good...about 4-5".

3/10:50 "I'm fully adapted now, and don't have any problems anymore. It's great to be here; I feel real fine."

3/16:24 "We stopped the [Rotating Chair vestibular experiment] effort when we started the sinusoids..because I think it is very [provocative] for the time being, and I don't want to run into trouble."

3/21:40 "Okay, I'm doing [the Provocative susceptibility test], and I'm doing the first couple of head movements, and I know already now that I won't do much more, because that is really a killer. I'm basically over the problem, but I don't want to get it back...Oh, it's bad. Okay this is a symptom report of the eyes closed [comparison test], and I think it feels a little easier, but it is definitely not pleasant to do, and I think I'll stop here."

4/10:27 "I have no motion sickness problems, I'm fully adapted, but I still don't want to make rapid head movements. That is still no go."

In debriefing, Subject C added: "I'd say it was about halfway through my fourth flight day that I really felt comfortable, didn't restrict my head motionsanymore. I was able to tumble in[to the Spacelab], do flips and summersaults. From then on it was really alot of fun. The third day was ok. I had no problem working. I was starting to enjoy it. But it wasn't the ball of fun that it was the last 5 or 6 days."

Subject D, who experienced no significant symptoms, said: "In my case, I took scop/dex prophylactically, as planned. I took one shortly after [orbital insertion], and one at 5 hours, MET, just before I went to bed. Took one on arising, which would have been about 0/11:00, and I took one an hour before supper at the end of that day, about 24 hours into the mission. I then took one upon arising at 1/8:00, and [I may have taken] one on arising 24 hours later. I had no symptoms.."

Etiologic Factors:

1. Head Movements:

In debriefing, we asked our subjects whether there was any specific activity which seemed to trigger vomiting or and increase in symptoms, and why their incidence of vomiting so high, as compared to previous missions ? For those subjects who experienced symptoms, they had no doubt that symptoms were modulated by the head movements associated with physical activity.

Subject A responded "[the stimulus for vomiting] is just continued activity. If I had stopped early enough - maybe 30 or 45 minutes before - and gone to my bunk, closed the door, and been quiet I think the symptoms might well have been suppressed... [On a 0-20 scale] the point of no return is maybe 15-17. I'm guessing a little bit, because I never really had much opportunity to explore just how far I could go and still return....I can't pick out anything that was individually provocative more than another.." "All kinds [of motion were provocative], not just pitch, roll, and yaw. Pure translation, maybe a little less, but you've got the visual effect... When a person gets over the threshold, it is hard to get back. When you get over the threshold, you better stop and do something promptly, like taking a scop/dex, getting quiet, and getting dark.. To me it is always a help to get in darkness...What I would have liked to do is to get in my bunk, strap down - if I had a good way to do it - and rest quietly for a half an hour or more. Then I think the symptoms would have never developed seriously."

Subject B added "I [agree] that if the timeline had allowed it, and I'd had time to sit there and take it easy and rest for 20 or 30 minutes, then I wouldn't have thrown up so much, and I would have been able to keep [my overall discomfort scores] lower on the scale...."

"It was a general pattern of activity, [which led to vomiting].... and you kind of felt bad at that activity level, but you said 'well, I don't seem to be getting any worse, so I'll keep on going', and then all of a sudden you said "oh, no, I'm feeling bad". A couple of deep breaths and it's in the bag.. What I was anticipating was that I would gradually feel worse, and get to a stage where I could stop and not vomit again, which happened [eventually]: Day 2, I picked up the time course of what was going on, and realized that if I went to a 12, I'd better stop <u>right there</u>. I could go between an 8 and a 12. If I hit 12, it was just: absolute stop and sit there for awhile. It was the only thing I could do....Day 2, I realized the time course of this.. you make a bunch of head movements or something, and you don't notice much change in the symptoms until boom, you're gone. So I guess I became more sensitive to the change. You allow for the time lag. [I began to use more] anticipation...[and I took drugs]".

Subject A again: "You wake up and you go all day, and then you quit with very small breaks. Occasionally you stop to fix a lunch, and sit down and eat it. The only stationary activity that I can remember was when I was taking pictures during a nighttime through the back window, and I had my head in this big [cloth skirt], and I surprised myself. I really found that somewhat quieting, and felt good after that... Fifteen minutes is a long enough period to begin to have some response to [counter] the provocative nature of your preceeding work."

"As soon as you'd throw up, you'd feel a little bit better, and the first thing you'd do would be to get it cleaned up and disposed of. Nobody else is going to do that work for you - you sure don't want anybody to.

And that's additional activity right then. You flounder around and get all that taken care of, and get right back to it."

"It is an extremely important question..how much disturbance the protocol or the experiment operation itself caused in terms of making crewmembers feel uncomfortable either all the way to complete sickness, and to what degree you think that effected conduct of the ..experiments. ..After thinking about it, I am convinced that..the experiment individual protocol.. - the actual 'Rotating Dome', or the 'Hop and Drop', or the 'Drop and Shock' [experiment protocols] - were not particularly provocative in terms of a five minute operation. But it is substantially more provocative to actually set up all the hardware. We started right from the ..ingress into Spacelab going at nearly full speed.... I started off feeling pretty good on [the first day], and it was sort of steady downhill from that point on, right through day two. And so it is my opinion that all the set up, all the action, all the head movements that are required that is the most provocative of all the activities. For the normal crewmember, you are very apt to make him sick if you continue the activity in a volume the size of Spacelab. If your objective is to get the work done, you can work through it. In our case, we essentially accomplished everything that was listed on our flight plan. But it was certainly an unpleasant experience - you had to force yourself to work through it. It was not easy to do, but you can get it accomplished. Do you accomplish it in the 'optimum' manner ? I don't think so. You are doing things more mechanically. You say: 'now I've got to go get this piece of hardware, and I've got to plug it in there, and I've got to put the [Hop and Drop handle] up there, and wait till it drops', and you do it. But you're not thinking about any of the other factors that have to do with, say, any of the small effects that you might ought to be looking for: perturbations in the experiment, or have I forgotten anything, or do the electrodes really look good ? Have I checked the impedances ? Are the amplitudes correct on the oscilloscope ? Because you are not fully up to speed, you're ..at a point where you are feeling lethargy, and you don't feel like you're ready to charge [ahead] at a normal rate. It does effect how well you interpret all the rest that's going on around you... and it will effect how well you do it. The mechanical part, you can plow ahead and get it done...I think it does effect the quality of.. the data. On a multidisciplinary [mission], somebody is going to have to decide whether you want to push crewmembers that hard, knowing that the normal crewmember is very likely to be sick. Or do you want to slack off, and let them go at a [natural] speed. In my own case, I think the appropriate kind of timeline, which I would have preferred to have, would have been... lax enough to say 'if I don't feel real prime, we've got time for me go lay down in the bunk for 15-30 minutes....It's a trade off between disciplines.

Subject D added: "You know, we ran on a timeline [which had built in an extra 25% time factor to allow for our unfamiliarity with weightlessness during] the first day or two, and..that was about right. [But] there was no way we could have gone through there at full speed.

Subject B: "There are things you've got to go do. You make really slow movements. I was keeping my head really lined up with my body. I didn't need a [neck] collar or anything to tell me that I wasn't about to make rapid head movements. We were forced [by the timeline] to keep making head movements. Even when I was making head motions, I felt I was sitting around a 10 or so, and it would be almost without warning that it would zoom up; it wasn't any one set of head motions."

As noted in the Overview, Subject B experimented on occasion with specific head movements, and noticed that movements in pitch seemed particularly provocative on MD 0. This observation is generally consistent with Soviet reports (Ref. 17). On MD 3, however, he found that rolling movements were the most disturbing. On MD 8, symptoms of space sickness could not be provoked by head movements, but oscillopsia was particularly prominent in roll. Our subjective impression, which must be confirmed by the head mounted accelerometer data, is that when working, crewmen make yawing head movements very frequently, and certainly make many more head movements in pitch than in roll. As the crewman's experience in zero g develops, adaptation to space sickness might be expected to occur first to those head movements which are most frequently made.

2. Visual Orientation Cues

In the brief weightlessness of parabolic flight, visual cues are known (Refs. 18, 19) to play an important role in spatial orientation: there is a general tendency to feel that one's own feet define the direction of "down", particularly when they are in view, and there are no familiar visual cues present defining a "vertical".

As noted earlier in the Overview, Subject A found closing the eyes made him feel better, as did Subject B, provided that he had tactile cues which indicated his body was not moving. Subject B noticed occasional oscillopsia on hopping and head turning. Oscillopsia gradually faded during the mission, but Subject B noticed it was still present in roll on MD 8. All of our subjects agreed vision played a major role in spatial orientation on orbit, and the symptomatic subjects felt that unfamiliar visual cues were disorienting and potentially provocative.

Subject B recalled that seeing a familiar visual scene inverted was bothersome: "My bunk was the bottom one, and [the two of us who used it] were hanging upside down like a bat hanging from the ceiling. I found that distressing for the first couple of days..I'd look out there in the morning and I'd see the orbiter [mid deck area] all upside down. That was very disconcerting." Subject C agreed with this assessment.

Subject B felt that "[Early in the mission] I really needed a good vertical feeling, a good optical "down". It was really distressing when [Subject D] came floating into the [Spacelab] module upside down and tumbling and things - that really didn't sit too well with my own perception of Spacelab and the way things ought to be..." " I ..felt like I needed a real visual 'down', and it was the floor...and I didn't really have one of my own."

Asymptomatic Subject D observed that "Spacelab is very familiar. It's got strong vertical orientation, so seeing another crewman [in an unusual orientation], if I was in the normal orientation to my mind had no effect whatsoever on me at all. [But] when I had rotated [in] the Spacelab or [in] the orbiter into some other attitude,..and had decided to make the ceiling the floor... seeing another crewman in the other orientation, the 'proper' orientation.. tended to break the illusion... The architectural 'imprint' of Spacelab [in your mind] is very strong, because you have worked in it for five years...".

In a lifetime of experience on earth, one becomes quite used to seeing other people in the familiar, erect orientation with respect to gravity. We have frequently observed in parabolic flight, where there is no gravitoinertial "down", that if one sees another person in an "unusual" orientation with respect to one's own body, unless other strongly oriented visual cues are present, it is not unusual to experience a sudden "reorientation" illusion, such that the seen person's feet suddenly define the perceived direction of "down", and the observer suddenly feels in an unusual orientation with respect to him. Subject B apparently experienced a similar visually triggered reorientation illusion, and that it was somewhat provocative is hardly surprising in the context of conflict theories for motion sickness. In other forms of motion sickness, the motion cue stimulus/ symptom response relationship is usually only apparent when the subject is already experiencing symptoms. Subject D obviously experienced the illusion, but only when he was inverted with respect to the Spacelab floor. He was asymptomatic, and therefore presumably should not find the illusion provocative. The implication of reports such as these is obvious: Early in the flight when crewmen are at risk of space sickness, all crewmembers, symptomatic or not, should try to remain in "familiar" one g orientations when practicable so as to reduce the frequency of reorientation illusions in all crewman. Travel through areas with ambiguous visual horizontals and verticals, such as the Spacelab tunnel (or analagous tunnels in a space station) should be avoided in the early days on orbit.

Note that later in the mission (3/11:07) when Subject B had become largely asymptomatic, he reported "Today for the first time I was really able to change my feeling of orientation at will. For the first several days, it was very important to maintain myself upright with respect to the Spacelab. Today...even without drugs I was able to obtain any orientation I felt like."

Subject C noted that "[working upside down was] alot of fun at the end of the mission, but for the first two days, it is probably a smart idea to create an 'optical' down, because all the labels are oriented the same way, the displays are made to be read that way..."

Moving about freely and efficiently in weightlessness apparently can trigger visual reorientation sensations with some frequency. Subject D commented that for him, "One of the delightful illusions is to turn the rooms on their sides or upside down - to reorient 'down'... The tunnel is a big help for that, because it gives you a good place to change orientation without being aware of it. Even accidentally, you were frequently caught working on a task or something, and apparently reorienting your 'down' without thinking about it, and then turning away, and finding that the whole room was completely cattywampus to what you thought it was..."

If a view of the earth is available, it may also play a role in visual reorientation episodes. At 8/14:43, Subject B commented [My shift partner] and I felt it was very difficult... to make the ceiling be "down" unless you really cheat by looking [up] out the window and see the earth down below and then you say ' yes, [the ceiling] is down'.

On earlier Shuttle missions, there were occasional reports that if the earth was seen in an unfamiliar or unexpected orientation, it could produce and increase in symptoms, and in one case may have provoked vomiting. Similar sensations have been noted after a period of window viewing when shifting the gaze back inside the vehicle. On Spacelab 1, our subjects occasionally experienced reorientation illusions when they looked at the earth, but did not find them particularly provocative. However, the science crew had little time to look at the earth until the second half of the flight. Subject B: "when I was looking at the earth... I wanted to orient myself so that I was looking at [it] from above somehow. So I would twist myself around so that the earth was along my [+] Z axis. So I'd first orient myself at a window and get set there..[so] that whatever I was looking at was vertical, and the earth was down below.." " [When I looked back inside the Spacelab] I never got a twinge. ...I didn't look out the first couple of days..[Early in the mission,] I'd catch a glimpse of the earth, and I knew I just didn't want to know or care what the spacecraft attitude was. I wanted to really concentrate on the inside surrounds.

3. Tactile/Proprioceptive Cues and Passive Body Restraint:

In parabolic flight, touch and pressure cues, also termed "agravic contact cues" (Ref. 18), are known to have a profound effect on spatial orientation. As noted in the Overview, our three subjects who experienced symptoms felt that forces passively applied to the body providing touch and pressure cues indicating that the body was not moving were very helpful in alleviating symptoms.

Subject B noted at 0/10:20 (during Hop and Drop, after stopping 1 g hopping due to increasing symptoms) "it appears that putting tactile cues on did tend to help to some extent, so I'll just stand here a little bit with the bungees on and see if that helps....it appears that free floating with a little bit of tactile feeling on the feet is probably about the most beneficial; if I get (the bungees) up too tight, it doesn't feel right, and if you're just floating, it doesn't feel right either, but a little bit of tension on the feet seems to help me feel better".

However, after further experience, he reported that "wedging in is better. The feeling of tactile cues around your body, that you are not floating off into space..You don't need to go to the [trouble] of putting on the harness and attaching the bungees..." " What I really felt was best was getting back in the [aft] endcone of the [Spacelab] module [and wedge myself in between the endcone wall and the last Spacelab rack.] I had good pressure on both sides of me to give me good stability, and it wasn't just against my back. And I was out of the volume. One of the most distressing things seemed to be that big volume."

Subject B tried an experiment to see if the same effect could be achieved if he actively applied the tactile forces himself: At 3/11:03 (after completion of the provocative testing, while feeling "about a 5") he noted: "It's usually more provocative trying to hold yourself down against something flat rather than it is just to

wedge yourself into the corner or get a bunch of tactile cues around your body. I try to press myself down into the sitting position, and it feels very awkward and uncomfortable and provokes slight stomach awareness."

None of our subjects tried strapping into a spacecraft seat. However, Subject C recalled that when he strapped into the Body Restraint System seat used in the European Vestibular Experiments, he "immediately felt better."

Subjects A and B sought relief by wedging themselves into their bunks. Subject B: "I would get in the bunk, and bring my knees up to my chest, and push them against the door, and push my back up against the wall, and get myself really set in there. (Subject A "So did I") " That felt alot better - I liked that. You knew you weren't moving and rotating around. Closing my eyes and floating was really bad." " I slept like that for two nights. Good parts of the night, I'd wake up from a fitful sleep, and you'd say: 'something doesen't feel right', and kind of scrunch around and get [re]wedged in." "During the night, it seemed to really help to have my head velcroed down to the pillow - that made me sleep alot better". "First couple of nights, I didn't [read] or anything [in the bunk.]. Just crawled in there and tried to go to sleep. Kind of uncomfortable, and it took me awhile to go to sleep, with all the strange feelings. But by the 3rd day, I'd get the [crew activity plan book], the stereo, and make some notes."

At 3/21:55, Subject B observed: "[I] had a good night sleep last night with ear plugs, and for the first time last night I was able to sleep free floating without having to be tied down with the head band and body straps - so it's much better sleep".

Subject D commented "I think the bunks are deficient in their design.. they have six long smooth sides, they don't provide any real means of wedging yourself in...Find a place where you can wedge yourself.. where the muscles in your legs and your back can just relax. Subject A added: "[the bunks] have two straps, which are intended to work. But by the time I pulled the strap over to make it tight, the velcro misses.

If our subjects' experiences are representative, then it seems clear that appropriately designed body restraints can be of value in alleviating symptoms of space sickness. The design objective need not be to provide an artifical "gravity" cue so much as to provide comfortably firm immobilization of the trunk, upper legs and - optionally - the head. As such a restraint is most useful in treatment, it must require a minimum of physical activity to deploy and strap into. The restraint should be located away from windows and oriented so that all objects viewed by the subject are seen in their familiar orientation. Ideally, it might be located so that a crewman using it could accomplish useful manual tasks if he felt up to it. Flight deck seats are inappropriate unless the eyes are kept closed because of the large number of windows. Mid-deck seats, perhaps equipped with thigh, waist, and shoulder straps could conceivably be used for body restraint. Unfortunately, these seats impede normal activities, and are normally stowed after reaching orbit. The physical movement required to set one up again is a negative factor. With thoughtful modifications and physical reorientation, the existing mid-deck bunks could probably made to serve this purpose better. Another possibility is the zero-g toilet, which is in an enclosed area, and is currently equipped with straps and thigh bars to restrain the lower body. An additional torso strap might be helpful. In Spacelab, the corners between the racks and the endcones are demonstrably usable, and allow a crewman to remain in the laboratory and in touch with activities there. Using them instead of the bunks eliminates two trips through the tunnel, which can be provocative. In the design of space stations, some forethought should obviously be given to placement of such restraint areas. On the basis of the foregoing, one would expect that the elastic neck restraint cap ("MPSA") and the foot insole counterpressure device (Ref. 17) and the "Penguin" elasticized suit (Yegorov) being evaluated by Soviet cosmonauts should be less effective than "wedging in". These devices, which provide tactile cues and/or passive stress on anti-gravity muscles by loading the body in a head to foot direction do not prevent body movement with respect to the spacecraft.

4. Epigastric Discomfort:

There have been reports in the Soviet literature (e.g. Ref. 16, p.5) that cosmonauts experienced "an unpleasant sensation of heaviness in the epigastric region, and a feeling of 'elevation' of the stomach in the inital stage [of sickness]". In previous NASA flights, there have been a few reports of "wet burping". Interpretation of subjective reports is complicated by the fact that some subjects experiencing motion sickness symptoms under conditions which are <u>not</u> associated with weightlessness <u>also</u> frequently report "substernal pressure" and "constricted" feelings in the chest (Ref. 4, p. 46). In our study, Subject B was one of these.

Subject A, in debriefing, commented that "Your stomach feels like it really has shifted up - your guts move up into your thoraic cavity... You have the same sense in a zero-G airplane; exactly the same feeling.. It's not particularly uncomfortable, but it can be a contributing factor to the whole 'sickness syndrome'." "[When I vomited],I was surprised at the volume of liquid that I had available to throw up. I thought that after throwing up once that I couldn't have anything else in my stomach, but I did, basically liquid." "..after you feel better, you can burp. There is no problem with it."

Subject B logged belching as a symptom during the first 5 days of the mission, with particular frequency during the first 2 days. He also noted repeatedly, beginning at 0/07:09, and continuing through his 5/10:50 report that he had stomach awareness "very high up in the sternum. It feels like the whole stomach has shifted up". In postflight debriefing, he commented that "I did have a fullness in the upper stomach, and I had this for the first 3 or 4 days. It was like everything in my stomach was up underneath my sternum; a very uncomfortable feeling. It felt like lots of times I wanted to burp, which is one of my symptoms, but [frequently] I just couldn't burp because [it felt like] the air bubble was in the middle of my stomach and all the fluid was around both ends. It was really uncomfortable, and I couldn't seem to get rid of that sensation at all." Subject B felt that this discomfort contributed to the value of his overall discomfort scores shown in Figures 2 and 3. This discomfort was accentuated at 1/00:30, when Subject B took a Maglan dose, and subseqently noted a feeling of "tightening up" in his abdomen. On the third day, he noted that his stomach and abdominal muscles felt somewhat sensitive to palpatation, and this disappeared by the middle of the 5th day. However, at 8/23:20, when performing the provocative motion sickness test, he reported "it is pretty difficult to touch my forehead to my knee because [my stomach] muscles are tender down there, and I

don't want to bend that much."

During the debriefing, Subject D did not recall feeling that his stomach had lifted up, or that he had any difficulty burping, or a bubble in the middle of his stomach or 'wet' burps. " Two or three days towards the beginning, I may have had a 'sour stomach'. At the time, I think I partially attributed that to the [hydrogen contaminated] water, but it wasn't like the stomach awareness or discomfort [of motion sickness]. [But] it could have been the one motion sickness symptom that I had. [The hydrogen in the water] ..didn't taste bad... The hydrogen continued through the mission on an off, but it was worst on days 2 and 3. The hydrogen continued..but this sour stomach did not."

It would surprising if there was not some rostral shifting of the abdominal organs on orbit due to the absence of normal gravitational loading, given that the stomach and intestines are normally mechanically suspended by the omentum and mesentery. Shift of interstitial fluid into the thoracic cavity might also be a contributor to the subjective effect. Mechanical stimulation of abdominal organs can trigger nausea and vomiting in certain situations, and abdominal gas and overeating are familiar causes of stomach discomfort and nausea. In the extreme, gross distention of the stomach or duodenum by very high pressures can lead to vomiting (Ref. 20). Also, under normogravic conditions, gas introduced into the stomach by swallowing or digestion might physically be expected to rise to the fundus and esophagus, and eventually be relieved by burping. However, in weightlessness, the absence of buoyant forces on gas bubbles could conceivably result in a tendency for gas bubbles to remain trapped in the stomach, and for burping to produce gastro-esophageal reflux, "wet burping", and sensations of heartburn and stomach discomfort in some subjects. The behavior of the abdominal organs and of food, fluid and gas within has not yet been systematically studied in weightlessness, but perhaps should be. Although the evidence relating the etiology of space sickness to other forms of motion sickness is now strong, we believe it is important to determine whether special "gut" factors exist in space sickness which are unique to weightlessness, and the extent to which these contribute to nausea, vomiting, and other symptoms in individual cases. However, it seems to us unlikely that these factors play a dominant role in space sickness, given the demonstrated importance of head movements and various orientation sensory cues in determining the time course of malaise. As Subject C put it "you would think that the motion sickness problem [then] should have the same time course as the fluid shift problem, but it simply does not."

Effectiveness of Pharmacological Countermeasures:

Drugs which have been employed in an attempt to prevent or control space sickness have generally been those known to be effective against motion sickness on earth. However, their efficacy has proven very difficult to evaluate under operational conditions. For example, 3 of 5 Skylab astronauts who took drugs on MD 0 experienced increased symptoms later that same day (Ref.1). Placebo effect is also a potential problem, because subjects are aware of exactly which drugs are being taken. It may be that the use of drugs simply raises the sickness threshold, and under uncontrolled conditions, crewmen simply make a greater number of provocative head movements before becoming frankly ill.

Table II summarizes the anti-motion sickness drug doses taken by our subjects on orbit:

TABLE II
PHARMACOLOGICAL COUNTERMEASURES AGAINST SPACE SICKNESS
SPACELAB 1

Key:
S/D = 0.4 mg scopolamine/2.5 mg dexedrine
P/E = 25 mg promethazine/25 mg Ephedrine
REG = 10 mg metoclopramide (Reglan)

Subject A:	S/D	0/00:25	Subject B:	S/D	0/06:09	Subject D	S/D	0/00:20
	S/D	0/05:30		S/D	0/22:30		S/D	0/05:00
	S/D	0/21:15		REG	0/00:30		S/D	0/10:00
	S/D	1/02:20		S/D	1/04:30		S/D	1/00:00
	REG	1/06:30		S/D	1/10:36		S/D	1/08:00
	REG	1/10:15		S/D	1/21:05		S/D	2/08:00?
	S/D	1/20:20		REG	1/21:05			
	S/D	2/02:11		S/D	2/02:14			
	S/D	2/07:15		S/D	2/08:30			
	S/D	3/01:20						
			Subject C:	P/E	1/01:10			

Although all 4 of our crewmen took drugs, and 3 subsequently experienced symptoms, the 3 symptomatic crewmen had the distinct impression that certain of the drugs they took were helpful in reducing their overall discomfort. Also, data on the relative frequency of vomiting episodes with and without scop/dex is consistent with the notion that this drug, known to be effective against other forms of motion sickness, reduces the incidence of vomiting in space sickness: As indicated in Table II, three subjects took scop/dex frequently over a 2-4 day period. If experience in 1- g is a guide, scop/dex is most effective in the period 3/4 hr until 4 hr after administration. 3 out of 4 of Subject A's vomiting episodes, and 5 out of 6 of Subject B's episodes (see Figs. 2 & 3) took place outside of this period of presumed effectiveness. For Subject B, the single exception was on MD 0, when he vomited some three hours after taking scop/dex. Subject A took scop/dex at 1/02:20, and vomited at about 1/04:00. Subject D, who also used scop/dex, was asymptomatic the entire time. Subject C had not taken any drugs prior to his 2 vomiting episodes on MDO. He subsequently took promethazine/ephedrine once at the end of his second full working day. His impression was that it "certainly had some effect" in reducing his symptoms before he retired.

In evaluating drug effectiveness, we believe it is important to keep in mind that the protection conferred by

a drug is a matter of degree, and no drug has been found for motion sickness, let alone space sickness, which acts as a "silver bullet", totally preventing sickness in everyone. Given this, we believe that our 3 subjects' experiences - at least with scop/dex, and possibly with promethazine/ephedrine - are encouraging. Considering the number of scop/dex doses taken in succession, it is also noteworthy that significant side effects (other than dry mouth) were not reported.

Metoclopramide HCl 10 mg. (Reglan, Robbins) is a dopamine antagonist known to increase the amplitude of gastric contractions and the tone of the esophageal sphincter, relax the pyloric sphincter, and increase peristalsis of the duodenum. Its conventional clinical use is to stimulate gastric emptying. However, its use against motion sickness has been advocated by several workers, and it was informally evaluated on a previous Shuttle flight with some apparent success. On Spacelab 1, however, neither of the subjects who tried metoclopramide were convinced of its effectiveness. Subject A reported that after becoming frankly sick for the first time (at about 1/04:00), about two and a half hours later "I took Reglan..the last scop/dex was four or five hours earlier. I wanted to be sure there was no conflict between the two. I checked for belly sounds, found they were about 1/3 the normal kind of motility and noises, and thought 'o.k., maybe if I take that Reglan, things will subside. Took the Reglan [at about 1/06:25]. No change...and I got sick promptly thereafter[, and again at about 1/08:25] ..I decided that the Reglan maybe hadn't had time to absorb. Tried it one more time [at 1/10:15]; got sick again [about 30 - 45 minutes later]. ...the report [from an earlier shuttle flight] was that its [effect] was almost instantaneous, that you take that stuff and right off you begin to feel better. It didn't [work that way] for me.

Subject B took Reglan twice, in combination with scop/dex. He described his experience this way (c.f. Fig. 2): "The second day, I woke up, and decided to medicate. So I took a scop/dex. About a half and hour later, I went in [to Spacelab] and was getting to do the blood draws, and I realized I wasn't feeling too great. (I don't think the scop/dex really had time to work.) I threw up there. And then I said, 'ok, maybe I'll try a Reglan'... That gave me some uncomfortable feelings in my stomach....like my muscles were really tightening up in my stomach and lower abdomen. I pressed on. And about 5 hours later, I took a scop/dex, which was halfway through the day....I could tell that 5 to 6 hours was the time course.

At 1/8:53, Subject B noted " I haven't really been limiting my motions or activities lately. Nor have I tried to maintain any specified orientation with the Spacelab recently." In the debriefing, he continued: "...all of a sudden I could tell my symptoms were going up and I was feeling worse. [Shortly thereafter, I vomited again] ..Time for another pill. So [I think scop/dex] definitely helped me, and I was able to keep working... The third day, I woke up in the morning, and..I took a Reglan and a scop/dex almost simultaneously...I took two more scop/dex that day, one at mid shift, and one about the time of getting off work.

It should be noted that the effect of Reglan on motility can be abolished by anticholinergic drugs, so taking Reglan in combination with scopolamine may have compromised Subject B's trial to some degree.

Motion Sickness Symptoms and Signs Observed

1. Subject B Summary:

An indication of the individual symptoms and signs experienced by Subject B during the first two days of the mission can be obtained from Table III below. (Nausea and vomiting episodes were not included in Table III, nor was epigastric awareness and discomfort, which was almost continuous during this period.) A comparison with Table I demonstrates that that palmar sweating, dry lips, flatulence, belching, and yawning were seen in both motion sickness and space sickness in this subject. (In preflight testing, we did not systematically ask about the degree of apathy and concentration impairment, although these are frequent symptoms of motion sickness (Ref. 3). However, they were a flight checklist item, and Subject B reported them with some frequency on orbit.)

TABLE III
FREQUENCY OF SYMPTOMS AND SIGNS DURING MD 0-1
(Number of reports in 48 hr. by Subject B)

Headache*	11
Cold sweating (palms)	10
Dry lips	7
Sensitivity to sensory stimuli	7
Flatulence	6
Belching	5
Yawning	5
Apathy	5
Concentration (impaired)	4
Anorexia	4
Dizziness/disorientation	3
Subjective warmth	3
Drowsiness	2
Respiration changes	0
Pallor	0

* Note: Subject B reported headache due to extended wear of accelerometer headband

Our subject who had experienced space sickness on Skylab said that his symptoms and signs in Spacelab were similar, except that he had not vomited on his previous space flight.

It is interesting that drowsiness, which is frequently observed in other forms of motion sickness, was not mentioned as a prominent symptom. We suspect that any tendencies toward drowsiness may have been cushioned by the anti-motion sickness medications taken by our subjects, by their demonstrably high level of motivation and interest in completing the experiments, by their frequent constant voice communication with the ground, and because they frequently worked interactively with other crewmen.

Overall, our 3 symptomatic subjects felt that the majority of the symptoms and signs they experienced were similar to those encountered in preflight training. However, significant exceptions were also noted which are discussed below.

2. Nausea and Vomiting:

Subjects A and B vomited repeatedly in the Spacelab, but apparently always had sufficient warning to move out of the field of view of the video cameras.

Subject A reported: "It gradually built up. I could feel it coming. Finally I'd have to decide 'o.k. well, I'm just not going to be able to suppress it anymore', and so I would stop for five minutes, throw up, take care of the bag, and get back to it...It was never unexpected; I was never caught unawares."

However, subject B was surprised that the crescendo of prodromal nausea prior to vomiting seemed to occur surprisingly quickly, as compared to his previous experience:

Subject B: "Unlike here on the ground where things tend to building up - I've been sick alot on the ground for different reasons...it's different in the sense that you are sitting there and you are not feeling really great but you've been feeling that way for an hour or an hour and a half, and things haven't really changed, and then all of a sudden, it's..a one or two minute warning. You say: 'I've got to stop doing what I'm doing', and then it's like you've gone over the dam..and you reach for the bag. It's over in a minute. And I felt much better every time after I threw up... And then get back to work.. Yes, it did [feel like a normal nausea sensation], but it was really fast. If I have stomach flu, you just sort of sit there and feel nauseous for hours, and you're not sure if you are going to throw up or not. It wasn't this way. I wasn't nauseous, I just had stomach discomfort. It just didn't feel right, and I was doing things to keep my activity down. But I don't think I really slowed down until 2-5 minutes before I vomited. And at that point, you say: 'Oh, I think I better take it easy' and then the next thing you know, you've gone beyond the point of no return."

Subject C had no experience on which to base a comparison, but he also reported little warning before his episode of vomiting: "I have really no way to tell you, because in spite of all the things I have done in my life, [prior to this flight] I have never managed to vomit [when motion sick]. And [the space sickness] was probably the only two times in my life that I [recall] vomiting ever...I cannot tell you [if there was nausea associated with it]. The way it feels is: just a few seconds before it starts, you feel this stuff sitting [in your throat]..You bite it a little while, and then give up. And the moment you [vomit] you feel much better.

All three subjects who vomited always experienced relief afterwards. The shortest time between vomiting episodes reported by Subject B was approximately 50 minutes.

"Sudden" vomiting with only very brief prodromal nausea has been occasionally reported on earlier missions (Ref. 7), and has lead to (unpublished) speculation by some that since vomiting in space sickness can apparently have no prodrome at all, that therefore the etiology of vomiting in space sickness must somehow be totally different than in motion sickness. However, we believe several facts argue against this view: First, all three of our symptomatic subjects reported prodromal overall subjective discomfort prior to the onset of nausea and vomiting. The extent of this prodrome is exemplified by Subject B's data in Figures 2 and 3. Second, there is some evidence (Ref. 21) that "avalanching" of symptoms just prior to vomiting is more characteristic in individuals who are highly resistant to motion sickness stimuli. (Indeed, on the basis of the preflight data in table 1 for Subjects A,B, and C, in which Subject C appeared least susceptible and Subject A more susceptible, one might have predicted the individual differences in avalanching pattern seen.) Thirdly, "sudden" vomiting of the type described by our subjects is also reminiscent of that frequently observed with relatively long duration provocative stimulation, for example, as in seasickness on ocean liners. Ocean travel and seasickness is much less common today than it once was. The majority of recent clinical and research experience with motion sickness involves stimulation over minutes to hours, not days, and use symptom endpoints short of vomiting. Certainly our subjects' impression of what constituted their "normal" prodromal pattern undoubtedly was determined by their previous experience and the type of stimuli we used in training. Half a century ago, ships' medical officers frequently noted that sudden vomiting was quite typical of seasickness on long passages. In his classic clinical paper Desmoes (Ref. 22) noted that "vomiting is very often projectile in character, and there may be little or no nausea preceding". In 108 cases reviewed by Maitland (Ref. 23), 34 vomited without reporting nausea first. Hill (Ref. 24) agreed with this assessment. It is interesting that Subject A, who felt his space sickness nausea built up in a more or less expected fashion, had previously served in the U.S. Navy, and had experienced symptoms while on a destroyer in a hurricane. Our MIT research group's recent experience in testing with other long duration, "chronic" provocative stimuli has been that after the onset of first symptoms, the "dynamic" of the subject's response changes with time such that he becomes much more sensitive to stimulation. Our subjects make intervals of head movements over a 1-2 hour period while wearing prism goggles or while in a rotating chair. After several symptomatic intervals, subjects generally find they can make far fewer head movements without vomiting. We believe that "sudden vomiting" is to be expected in space sickness, and that additional research is needed to better define the difference between motion sickness responses to short and long duration stimulation.

3. Cold Sweating:

Cold sweating is a consistent symptom of motion sickness (Ref. 3), and was frequently seen in Subjects A, B,

and C in preflight motion sickness tests. It has apparently been reported Soviet cosmonauts suffering space sickness (Ref. 17).

Subject A reported: "I sweat almost zero; I could have easily worn one shirt for the whole flight". On the basis of his preflight experience (Table I), Subject A was slightly surprised at his lack of sweating, given the intensity of his other symptoms. Subject B consistently reported "cold, clammy hands" as he had in several preflight tests, but whereas sweating on the backs of his hands, arms, and forehead had been noted on occasion in training, on orbit, sweating on the remainder of his body apparently remained at insensible levels. However, the appearance of sweat is notoriously dependent on environmental factors. It may be relevant that the mid-deck was subjectively cold during the first two days of the mission, and the Spacelab module was cold (approx. 68 deg. F) and dry throughout the entire flight. In some of our preflight test situations , the temperature and humidity of the environment could not be well controlled, and in one case (the cabin of the device used for horizontal axis rotation) was quite warm, and conducive to thermal sweating. With respect to Subject B, it may be pertinent that the physiology and time course of palmar sweating is known to be somewhat different than that seen on the "thermal" sweat areas of the remainder of the body (Ref. 25).

4. Pallor:

Facial pallor is one of the most consistent signs observed in motion sickness (Refs. 3 & 4), was seen frequently in our training and testing preflight, particularly in subjects A and D, and has been reported present in Salyut/Soyuz crewmembers (Ref. 17). However, it is reportedly difficult to observe visually on-orbit (W. Thornton, personal communication). This could perhaps be due to changes in skin circulation patterns caused by fluid shift. Therefore, we were not particularly surprised that pallor was not noted visually in flight. Subject B checked for pallor in himself on 10 occasions during the early days of flight, always with negative results. However, in postflight debriefing, Subject A, reported "I definitely saw [Subject B] with pallor while 'in extremis'. I might very well have had [it] also. [Subject B] definitely had pallor around and associated with vomiting episodes."

We believe it is important to verify that some degree of vasoconstriction tends to occur as a consistent feature of space sickness, just as it does in motion sickness. Sensitive electrooptical instrumentation suitable for monitoring skin pallor in ambulatory subjects is under development at MIT for use in this experiment on future flights (Ref. 26).

5. Headache

Headache had been occasionally noted as a preflight motion sickness symptom in Subjects D,A, and B (in decreasing order of frequency - see Table I). On orbit, Subjects B and C both reported slight to moderate headaches, but believed that these were largely due to the occipital accelerometer package they wore, which was held in place by a cloth headband passing over the brow. The headache typically developed after several hours of wear, did not modulate with other symptoms, and was present throughout the mission whenever the accelerometers were worn. (The accelerometer headbands had been noted to produce headaches during preflight experiment simulations; they were not worn during preflight motion sickness tests. The headband is being redesigned for future missions.)

Subject D, who wore the accelerometers only briefly, reported "I had headaches, but I have headaches, so it's not really unusual. Headaches right across the top of my head. I had it a couple of days in the afternoon, but it wasn't really until [mission days] 2-3. I wouldn't neccessarily attribute this to the effects of fluid shift. I get headaches at home like that."

In debriefing, Subject A noted that "Sometimes in the morning I'd wake up with a little bit of a headache. But as soon as I began any kind of activity, moving around, it disappeared. I never took any aspirin or Tylenol at all during the flight." Question: Could the headache be due to closing off of sinuses ? Answer: "It's possible. I always used a little touch of Afrin, which I use frequently at home [most nights]. That always works perfectly and opens up my nasal passages very nicely."

6.Anorexia:

Anorexia is frequently seen in motion sickness, and is also potentially a side effect of taking dexedrine. Symptomatic subjects experienced anorexia early in the flight, as indicated by the subjects' debriefing comments:

Subject A: "I've got listed all I ate in the first 4 days. It was probably only about 100 calories total. I wasn't starving, I just didn't want to eat. I had a piece of bread and a banana, and one or two other items. That was it. But after that it was nearly normal. .But I never ate lunch for ten days. Crackers or something but that was it. We were too busy."

Subject B: " I started out, didn't eat anything the first day. Halfway through the second day, I think I ate two Saltine crackers. I drank alot of water. I was continuously drinking water. Every time I'd throw up, it was water that was coming back . At the end of the second evening, I ate about half a meal - chicken a la king or something. It slowly picked up from there. The first couple of days, we didn't really eat lunch. We could never find 10-15 minutes even to go back there and get things prepared."

Subject C skipped breakfast on first morning, and drank very little during the first 2 days.

Subject D said: "I don't eat lunch, anyway. But I ate 2/3 to 3/4 of what was packed for each meal for every meal..right from day zero, on.

Weight losses on orbit as a percentage of body weight varied from 6.6% in Subject A to 4.1% in Subject D.

Anorexia early in the mission may have been a contributing factor.

7. Abdominal Sounds

Considerable experimental evidence (reviewed in Ref.3, page 58) indicates that gastric hypomotility is consistently seen in motion sickness. A reduction of bowel sounds has been reported in space sickness by Thornton (personal communication). On Spacelab 1 an electronic stethoscope was carried, and taped to the abdomen when used. Subject A reported that when he was experiencing some symptoms on flight day 2, he "checked his belly sounds, and judged them to be roughly 1/3 normal." On day 3, he judged his sounds as normal. Subject B reported that "On the evening of the second day, I listened to my bowel sounds, and concluded they sounded normal. This was the second evening, before I ate some dinner. Prior to this, I hadn't had the time or the inclination to listen." He rechecked his sounds on the third day, with the same result.

Additional Symptoms and Signs Observed associated with Weightlessness:

1. Fluid Shift

Our subjects expected to observe symptoms and signs of a rostral shift of blood and interstitial fluid immediately upon reaching orbit. We asked them: when did you first notice the symptoms and signs of fluid shift ?

Subject A: "Minutes. It was just like hanging upside down..[It was] not particularly uncomfortable...

Subject B: "Within 10-15 minutes". Question: Did you notice them less as time passed ? Answer: "Not until about day 6. First 4-5 days, I felt really puffy, congested, and the same way every day. I was looking for it to level out, but it wasn't until about day 6 that I noticed that it was still there, but it didn't seem as prominent - you get used to it." "...I had alot of congestion. It was like I had a bad head cold. Started using Afrin about the 3rd day, and used it about every 18 hours for almost the whole mission. And I could tell when it wore off. Within 20 minutes, I'd get totally stuffed up and [would sound that way when I talked]."

Looking in the mirror, Subject B described his fluid shift signs at 0/10:54 as "fluid shift - most of it is around the jowls and cheeks,..there is some increased puffiness under the eyes, right up at the top of the nose, and an increased fullness in the skin up by the sideburns near the ears." His report at 8/23:30 was essentially identical.

Subject C: "In my case, [fluid shift] was really dramatic.. it was uncomfortable. And also, if I took a looked in the mirror and saw my own face, I was shocked. It was really puffy. It persisted basically through the entire mission. It [slowly got] a little better, but I still looked different at the end of the mission... I had no problems with [stuffy] nose, throat, or things like that...But I could also feel it..up in my head...its a real feeling of fullness, everywhere."

Subject D: "I was aware of fluid shift from appearances, ..and looked to see how it looked...I don't remember feeling a particular uncomfortable fullness of the face. My nose was stuffy, but there was also alot of lint and [debris] floating around... My nose was not draining. But my membranes were swollen, and this is probably as good an indication as any that it was not what was in the air as much as it was increased fluid in the head. ..sinus [ache] and fullness of the head [feel like] the same thing really."

It has been speculated that perhaps space motion sickness could be somehow associated with decreased pressure in the middle ear due to functional blockage of the Eustacian tubes by fluid shift pharyngeal edema. Also, if exposure to weightlessness were to somehow cause pathologic changes in the inner ear, as as been suggested by some, one might expect concomitant changes in auditory function as well. All subjects denied any problems with hearing or Eustacian tube function. Subject A commented " My sense of taste and smell were largely absent for the first 3-4 days. But nothing [wrong with] hearing. As a matter of fact, we'd do a little pressure test or change for one thing or another, and I could feel my ears pop, with quite normal Eustacian tube clearing. No problems."

2. Back Pain

Subjects B and D experienced significant back pain during the mission:

Subject B noted at 0/7:37 that his "whole spine feels distended; muscles in the back are a little stiff..". This stiffness progressed into moderate back pain which persisted at least through MD 4.

Subject D felt no sense of spine distension but admitted that he didn't specifically look for it. However, he had "definite back pain, for the first 2-3 days. Lower back pain..in the lumbar curve. I have mild sciatica, and do back [abdominal] exercises [at home], and it takes care of it very nicely... The first couple of days, you were stiff alot of places, cause you were doing alot of things that you weren't used to doing.. and it would be hard to say that I didn't have a [slightly] stiff neck during those first few days. No abdominal muscle pains...The business of maintaining posture while using two hands - that's why your muscles are so tired - is a very stressful thing."

When debriefing, Subject A said he did not feel any back pain, muscle pain, or that spine felt stretched. Subjects A and B had no previous history of back injury or significant back pain.

CONCLUSIONS:

In this paper, we have tried to present a detailed description of 3 cases of space motion sickness as they occured on Spacelab 1, drawing heavily from the firsthand narratives provided to us inflight and immediately postflight by our subjects themselves. These sickness monitoring and provocative testing experiments are scheduled to fly on several other Spacelab missions, so this report should be regarded as only preliminary. Analysis of data from the head mounted accelerometers worn by 2 of our subjects is not yet complete. Hence, we are reluctant to draw strong conclusions based on the experience of only 4 individuals. However, our subjects had been specifically trained in motion sickness symptom monitoring, and their patterns of response to various provocative stimuli well established in preflight testing. They were knowlegable in the physiology and psychophysics of spatial orientation and vestibular function, so we believe their reports will be of particular interest and utility to the scientific community. Hence we are making their reports and our preliminary interpretations available to aerospace medical specialists now.

Three of our four subjects experienced significant symptoms which were generally similar to their own motion sickness symptoms preflight. Our subjects quickly learned to limit their head movements, and attributed their sickness directly to the high level of physical activity demanded of them during the first days of this science oriented mission. They believed that had there been sufficient flexibility in retimelining their activities during this period, they might have been able to pace themselves better physically, and perhaps have avoided vomiting. As it was, our subjects elected to press on, and all 3 vomited several times during the first two days of the flight. All three felt better after vomiting, and noted that if the objective is to get the work done, one can put up with vomiting occasionally. Although they all experienced a prolonged general malaise, it is significant that two of our three subjects experienced only a short period of intense prodromal nausea prior to vomiting. Although additional research on this point is desirable, there is evidence that a sudden "avalanche" of symptoms is characteristic of long duration motion sickness, and also of relatively resistant subjects, (which these two were). Other symptoms and signs observed included anorexia, flatulence, belching, yawning, sensitivity to sensory stimuli, mild apathy and impaired concentration, and subjective warmth. Persistent headache was reported by two subjects, but was probably attributable to the accelerometer headband. One subject (of two) reported via stethoscope a reduction in abdominal sounds. In the symptomatic subjects, pallor was notably visually absent, as was cold sweating from thermal areas. We tentatively attribute these latter observations to the presence of physiological fluid shift, and to the cool, dry environment of Spacelab, respectively. Drowsiness was not conspicuous. One subject experienced a persistent, uncomfortable feeling of stomach elevation and some difficulty in burping early in the mission. In 1-g motion sickness tests, this subject had reported "substernal pressure" and a "constricted feeling in the chest". However, in 0 - g, his sensations could also be attributable to a rostral shift of abdominal organs and interstitial fluid and/or trapping of gas in the stomach. The possible contributory role of these "gut" factors in individual subjects needs to be investigated further. Our subjects denied having any difficulty with hearing or with clearing their ears, although all reported a persistent feeling of head fullness and congestion, and several used nasal decongestants through much of the flight.

Symptoms diminished by the end of the third day on orbit, although they could still be elicited with vigorous head movement through the fourth or fifth day. Deliberately provocative head movements made by one subject indicated that - at least for this subject - pitching and rolling head movements were particularly disorienting and provocative early in the mission, but that later on, pitching movements became less troublesome.

Although we have deliberately not discussed readaptation to the terrestrial environment in this paper, it is significant that upon reentry and landing, subjects initially experienced significant oscillopsia for several hours, and were ataxic for several days postflight. However, they had no "earth" sickness upon their return. A period of readaptation is to be expected if adaptation to weightlessness involves learning a new set of rules relating body movement and sensory inflow.

The fundamental question we asked our subjects to consider in postflight debriefings was: is space sickness a form of motion sickness ? Their answer - and ours - is: very clearly, yes. Even without the head mounted accelerometer data to reference, it was abundantly clear from our subjects' reports that head movements associated with physical activity precipitated an increase in symptoms, as expected. Drugs known to be effective against motion sickness were taken by all, and were judged effective against space sickness, although - as might be expected - they did not provide everyone with total immunity. Visual and tactile/proprioceptive cues exacerbated symptoms, also as expected. To avoid creating provocative visual reorientation illusions in themselves or their companions, to the extent practical, all crewmen (including the asymptomatic) should avoid assuming unfamiliar orientations with respect to the spacecraft interior and to each other during the first several days on orbit. Both subjects who slept "like bats hanging from the ceiling" in inverted mid-deck bunks found this orientation distressing. The majority of our symptomatic subjects felt that travelling through the tunnel from the Orbiter to the Spacelab was provocative. We believe this may be because the tunnel lacked a well defined "ceiling" and "floor". Tactile and proprioceptive contact cues provided around the body by wedging into a corner or a bunk were clearly palliative, as was closing the eyes, provided that these contact cues were simultaneously present. (In this regard, several deficiencies in the present design of the bunks were noted.) These findings have clear implications with respect to future space station design.

In all these observations, we see precious little evidence to support any of the various "fluid shift" hypotheses. Although not all of these hypotheses can be formally ruled out due to the limited set of observations made, we believe the reports of our trained observers on Spacelab - 1 completely support the view that space sickness fundamentally is motion sickness, and therefore is only a normal human response to an abnormal gravitoinertial environment.

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DISCUSSION

UNIDENTIFIED SPEAKER: Were you able in the observed discomfort either with the prism goggles or in the flight to see the share of peripheral vision versus full visual field?

OMAN: The field of view was not large with the prism goggles, maybe 50° x 30°. Nonetheless, it was quite provocative. I would think if we had the appropriate equipment, a helmet mounted display system with a very wide field of view, we could make people sicker even faster. Our subjects adapt to the very narrow view of the prism goggles in terms of achieving their orientation information.

UNIDENTIFIED SPEAKER: I have difficulty in your expression "space sickness is a motion sickness", since the proving experiment was not done keeping someone absolutely quiet in space and seeing whether he gets sick. I can identify at least three different conflict situations that already exist in space without any movement.

OMAN: You distinguish between static space sickness and kinetic space sickness and that's a useful distinction. I think you are right. In speaking in very generic terms of space sickness as motion sickness, I think I choose my words very carefully in saying the evidence is consistent with the view that space sickness is motion sickness. I believe there is precious little evidence to the contrary.

SPACE ADAPTATION SYNDROME: INCIDENCE AND OPERATIONAL IMPLICATIONS FOR THE SPACE TRANSPORTATION SYSTEM PROGRAM

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SUMMARY

The space adaptation syndrome (SAS), is an operationally relevant biomedical problem for manned space flight. On the basis of past experience, it is clear that SAS presents a potential threat to the well-being and optimal operational performance of space flight crewmembers. In an effort to develop better methods for the prediction, prevention, and treatment of SAS, investigators at the NASA-Johnson Space Center have initiated a systematic, long range program of operationally oriented data collection on all individuals flying Space Shuttle missions. Preflight activities include the use of a motion experience questionnaire, laboratory tests of susceptibility to motion sickness induced by Coriolis stimuli and determinations of anti-motion sickness drug efficacy and side effects. During flight, each crewmember is required to provide a daily report of symptom status, use of medications, and other vestibular related sensations. Additional data are obtained postflight. During the first nine Shuttle missions, the reported incidence of SAS has been 48%. A wide range in severity of symptoms has been reported with general malaise, anorexia, nausea, and emesis being the most frequently described. As in the past, self-induced head motions and unusual visual orientation attitudes appear to be the principal triggering stimuli. Anti-motion sickness medication, although used by a high percentage of crewmembers, has been of limited therapeutic value. Complete recovery from symptoms has occurred by mission day three or four. Also of relevance is the lack of a statistically significant correlation between the ground based Coriolis test and SAS. The episodes of SAS reported thus far have resulted in no impact to Shuttle mission objectives and, with the exception of a one-day postponement of a scheduled space walk on the fifth Shuttle mission, no significant impact to mission timelines. Additional data collection activities are planned for future Shuttle flights.

INTRODUCTION

Space motion sickness, recently renamed the space adaptation syndrome (SAS), is a special form of motion sickness that is experienced by some individuals during the first several days of exposure to the microgravity space flight environment. The syndrome may include such symptoms as depressed appetite, a nonspecific malaise, lethargy, gastrointestinal discomfort, nausea, and vomiting. As in other forms of motion sickness, the syndrome may induce an inhibition of self-motivation which can result in decreased ability to perform demanding tasks in those persons who are more severely affected. The syndrome is self-limiting. Complete recovery from major symptomatology, in other words adaptation to the space flight environment, occurs within two to four days. After complete adaptation occurs, crewmembers appear to be immune to the development of further symptomatology. This finding was eloquently demonstrated by provocative rotating chair tests conducted inflight during the Skylab missions (1).

The overall incidence to date of SAS in the U.S. and Soviet manned space programs is summarized in Figure 1. Data from the U.S. program are based upon inflight reports and postflight crew debriefings and are reasonably accurate. The Space Transportation System (STS) Program results include missions 1-9. (Test activities and results related specifically to the STS Program are discussed in greater detail later in this report.) Data on the Soviet space program were derived from various U.S. National Technical Information Service (NTIS) English translations of available Soviet literature, personal communications with Soviet vestibular investigators, and other open literature.(2).

During the first two U.S. flight programs, Mercury and Gemini, no occurrence of SAS was reported. In retrospect, this finding is attributed largely to the design of the Mercury and Gemini spacecraft which restricted the mobility of crewmembers as well as their ability to see outside. With movements restricted, limited exterior vision and relatively stable internal spacecraft visual orientation cues, crewmembers did not encounter the SAS symptomatology that was to be experienced in later programs.

These factors changed with the Apollo and Skylab programs because the spacecraft had more interior volume, thus allowing increased mobility of the crew. Because of this mobility, the incidence of vestibular and other sensory rearrangement problems leading to SAS was heightened. The Skylab program enabled NASA scientists to investigate SAS in a more systematic manner. A number of quantitative vestibular function and motion sickness susceptibility tests were conducted with the Skylab astronauts preflight, inflight, and postflight. Several reports detailing the findings from Apollo and Skylab have been published (1, 3, 4, 5).

As indicated by Figure 1, SAS events occurred somewhat early in the history of the Soviet space program. This finding is in all probability related to the earlier use by the Soviets of relatively larger spacecraft. It is obvious from available data that the frequency of occurrence of SAS has been approximately equal in the U.S. and Soviet manned space programs.

In an effort to resolve the SAS syndrome, or at least minimize the operational impact of the syndrome, NASA has significantly expanded its research and development efforts in this area. As part of this expanded effort, investigators at the NASA-Johnson Space Center have initiated a systematic, long range program of operationally oriented data collection on all individuals assigned to STS flights. A primary objective of this program, which began with the first STS flight (STS-1), is to conduct inflight observations, supported by a series of preflight and postflight data collection procedures, on

the crewmembers in an effort to begin validating ground based tests which may be predictive of susceptibility to the SAS syndrome. An additional objective is to implement crew testing procedures which would enable acquisition of data to be used in validating countermeasures for the syndrome.

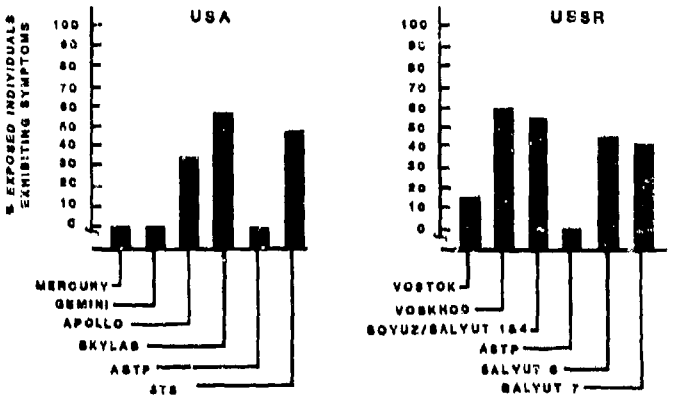

Figure 1.

Incidence of space adaptation syndrome in the U.S. and U.S.S.R. manned space programs.

METHODS

Preflight

Part of the required crew preflight activity was based on guidelines set forth in a NASA medical operations policy for the prophylaxis and treatment of SAS with anti-motion sickness drugs. This policy stated that astronauts with a positive history of SAS or with no space flight experience would be premedicated with a properly selected anti-motion sickness drug. Premedication was operationally defined as taking the prescribed drug either prior to launch or immediately after the first inflight orbital correction maneuver. This maneuver occurs about 10 minutes after orbital insertion. The policy further stated that astronauts who had previously flown in space with no symptoms of SAS were not required to be premedicated. Any individual who experienced SAS was to be administered appropriate inflight treatment with anti-motion sickness drugs. The policy required preflight side effects screening and efficacy testing with one or more anti-motion sickness medications.

Approximately three to six months prior to flight, each of the crewmembers completed a questionnaire designed to elicit pertinent information regarding past experiences with various types of motion environments and responses to those environments.

Also during the three to six months before flight each of the crewmemebers were tested at least one time for suceptibility to experimentally induced motion sickness in the Johnson Space Center Neurophysiology Laboratory. A standard Coriolis Sickness Susceptibility Index (CSSI) test, originally developed by Miller and Graybiel (6), was used. This procedure requires the performance of head movements while rotating at a constant velocity in a servo-controlled chair. The test was terminated when the blindfolded crewmember reached the Malaise III level (8 symptom points) of motion sickness or performed 150 head movements, whichever occurred first. This test served two purposes. First, it provided a ground based susceptibility data point against which inflight susceptibility could be compared. Second, it provided a baseline for subsequent evaluations of anti-motion sickness drug efficacy. During this test session the crewmembers were instructed on the self-recognition and reporting of motion sickness symptoms. They were also instructed on the use of a microcassette recorder and symptom checklist which were to be usad inflight for symptom reporting.

In accordance with the medical operations policy for the use of anti-motion sickness medications, the majority of crewmembers were screened for side effects with one or more medications. This screening was typically done under operational conditions. For example, the crewmember would use a medication while working in the Shuttle simulator. Verbal reports of any side effects experienced were given to a flight surgeon and documented. The medication most frequently evaluated in this fashion (and the most preferred medication) was oral scopolamine (0.4 mg) plus dexedrine (5.0 mg). A recently developed transdermal (skin patch) method of administering scopolamine was evaluated by a few crewmembers. Also, an oral combination of promethazine (25 mg) plus ephedrine (25 mg) was evaluated by a few individuals.

Crewmembers who were required to be premedicated for flight were tested in the Neurophysiology Laboratory to evaluate the efficacy of the preferred medication in preventing or minimizing motion sickness. The CSSI test procedures described above were used. In a few cases where the initially preferred medication produced questionable results, the test was repeated with the same medication or different medication. A minimum of two weeks was maintained between the rotating chair tests to minimize adaptation effects.

Inflight

A microcassette tape recorder and symptom checklist were stowed onboard the STS Orbiter. The flight crewmembers were required to use the recorder and checklist during a designated time (pre-sleep

period) each mission day to report on any symptoms or sensations that had been experienced during that day.

Postflight

Questions pertaining to SAS, vestibular sensations, and performance were asked of each crewmember on the day of landing and during postflight medical debriefings. Several crewmembers voluntarily repeated the CSSI test on the day of landing or within one or two days after landing. The purpose of repeating the CSSI test postflight was to determine what effect adaptation to space flight might have on susceptibility to Coriolis motion sickness upon return to 1-g.

RESULTS

Preflight

The motion experience questionnaire indicated that all of the crewmembers on STS missions 1-9 had minimal history of susceptibility to terrestrial forms of motion sickness. The questionnaire revealed that a few had experienced some motion sickness symptomatology during past exposures to aerobatic flight, parabolic flight, and heavy sea conditions. The questionnaire results did not correlate with the actual incidence of SAS symptomatology reported by this group of 29 crewmembers.

The mean preflight baseline CSSI score (without medication) for the 29 crewmembers was 28.7 (S.D. = 21.6) on a scale of 0-100 where a score of 100 means extreme resistence to the CSSI test. In contrast, the mean CSSI score for a normative population of 497 non-astronaut individuals at the NASA Johnson Space Center is 14.0 (S.D. = 14.3). Thus, it is evident that the typical astronaut is less susceptible to the CSSI test than the average non-astronaut; however, the range of susceptibility within the astronaut population is large.

The frequency of occurrence of motion sickness symptoms experienced by the STS crew population during the preflight CSSI test is summarized in Figure 2. The symptoms shown are those which were being manifested by the crewmember at the end of the test. It may be seen that increasd body warmth (TMP), mild nausea (NSA), sweating (SWT), and facial pallor (PAL) were the most commonly occurring symptoms. Symptoms such as persistent dizziness (DIZ), drowsiness (DRS), and increased salivation (SAL) occurred much less frequently. The pattern of symptomatoloy expressed by the SAS astronaut population with the CSSI test is highly similar to the pattern exhibited by a normative population.

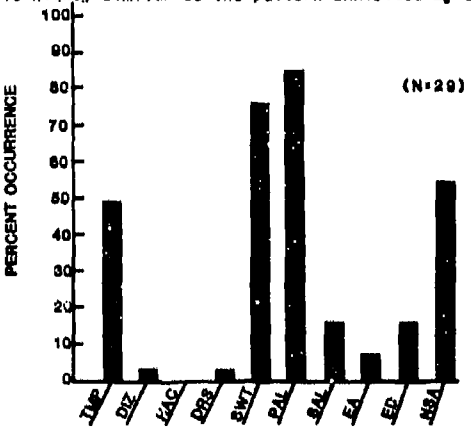

Figure 2.

Frequency of occurrence of motion sickness symptoms reported by the Shuttle Transportation System (STS) crewmembers during the preflight Coriolis sickness susceptibility index (CSSI) test. Symptoms shown are those which were present at the time the test was terminated.

Complete data are not available with regard to the results of preflight anti-motion sickness drug efficacy testing. However, where drug efficacy data were obtained with the CSSI it was found that the CSSI score was raised by an average of 50%. The drug test results were highly variable with some crewmembers demonstrating a two-fold or more decrease in their susceptibility with the drug tested while others showed no improvement over baseline.

Inflight

As already indicated by Figure 1, 48% of the crewmembers of STS missions 1-9 reported symptoms that were interpreted as being SAS. Table 1 provides an overall comparison of the preflight CSSI test results with SAS. The mean preflight CSSI score for the group of astronauts who experienced SAS was 27.4, while the mean for the group who did not experience symptoms was 30.9. This difference suggests that the CSSI test may be mildly predictive of SAS. However, the CSSI score ranges and standard deviations for the two groups are large. The difference between the two groups is not statistically significant. In a further attempt to establish a relationship between the CSSI test and SAS, the crewmembers were ranked on a four point scale of inflight level of severity of symptoms. The statistical correlation between the CSSI scores and assigned rankings was near zero.

Predominant inflight symptoms reported by the 14 affected crewmembers are summarized in Table 2. A comparison of Table 2 with Figure 2 reveals striking differences in the pattern of symptomatology generated inflight versus during the ground based CSSI test. The symptoms of subjective warmth, sweating, and pallor which were dominant CSSI test symptoms were almost nonexistent inflight. Only a few crewmembers reported seeing pallor in another crewmember. In contrast, anorexia, headache, malaise, lethargy,

general stomach discomfort and vomiting were dominant inflight symptoms. One or more episodes of inflight vomiting were experienced by 13 of the 14 crewmembers who had symptoms. The vomiting episodes often occurred abruptly with little or no prodromal nausea, although a general stomach fullness or discomfort generally prevailed prior to vomiting. In most instances, vomiting resulted in quick relief from the uncomfortable stomach sensations, although, for some crewmembers the discomfort would gradually return.

TABLE 1

SPACE ADAPTATION SYNDROME	Yes	No
N	14	15
% of Total	48	52
CSSI SCORE		
$\bar{X}$	27.4	30.9
S.D.	20.8	20.9
Range	8.4-64.5	11.2-90.0

Group mean comparison of preflight CSSI test scores and reported inflight space adaptation syndrome (SAS) events for the 29 different individuals who flew during the first nine STS missions. The between group difference is not satistically significant.

TABLE 2

SYMPTOM	PERCENT OCCURENCE
Nausea	25.0
Abdominal Fullness/Discomfort	17.0
Vomiting	42.0
Anorexia	40.0
Lethargy	40.0
Malaise	43.0
Headache	45.0
Pallor	5.0
Sweating	0

Space adaptation syndrome symptoms reported by crewmembers of the first nine STS missions. The data were derived from inflight and postflight debriefings. Due to minor variations in reporting procedures these data are relatively accurate approximations rather than absolute values.

The specific nature and time course of symptomatology has been highly individualistic during the first nine STS missions. A few crewmembers have reported that symptoms appeared within the first one to two hours of the mission. Others did not become aware of symptoms until the second day of flight. In general, symptoms begin during the first day of flight, plateau between 24-48 hours and gradually diminish between approximately 48-96 hours. During this time the symptoms may wax and wane in severity. Unquestionably, head and body movements contribute to the symptomatology. A general impression of the typical time course of the more prominent symptoms of SAS is given in Figure 3.

Anti-motion sickness and/or anti-emetic medication was used by 21 of the 29 individuals who flew during the first nine STS missions. The oral scopolamine plus dexedrine combination was the most frequently used with 17 crewmembers taking one or more doses during the first few days of flight. The first scopolamine plus dexedrine dose was generally taken about ten minutes after orbital insertion, however, on three occassions crewmembers took the medication about two hours before launch. The transdermal scopolamine skin patch, an oral promethazine plus ephedrine combination, compazine suppository and a promethazine suppository were each used one time. Oral metaclopramide was used by four crewmembers in an effort to restore gastric motility and alleviate nausea and vomiting. Thirteen of the 14 crewmembers who experienced SAS, used medication during the course of their symptomatology. One symptomatic crewmember elected not to use medication.

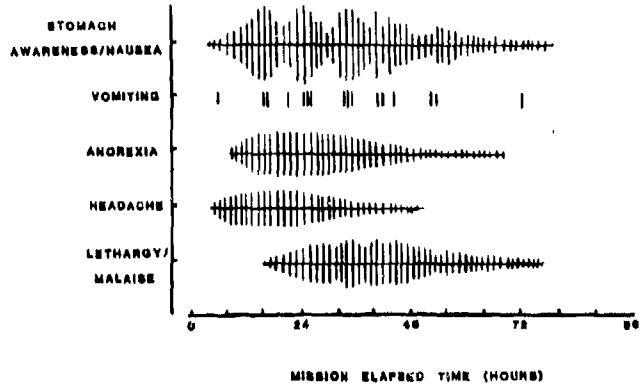

Figure 3.

Illustration of the approximate time course of major symptoms of space adaptation syndrome.

Postflight

Two of three crewmembers who repeated the CSSI test immediately postflight demonstrated a marked resistence to motion sickness relative to their preflight baseline susceptibility. Because of their busy postflight schedules it was not possible to conduct additional tests on these individuals to determine the time course of return to baseline susceptibility.

With one exception, a crewmember who reported a transient vertigo, no significant vestibular disturbances were experienced as a result of exposure to gravito-interial forces during re-entry and landing. None of the crewmembers experienced any motion sickness at any time post-landing. A number of crewmembers experienced postural equilbrium disturbances immediately after landing, however, these disturbances were never severe as evidenced by the fact that they all were able to walk unaided from the Shuttle. Normal gait and postural control was recovered within hours to a few days after landing.

DISCUSSION

STS Observations

The incidence of SAS during the first nine STS missions was not unexpected when considering past space flight results. The relatively large habitable volume of the STS orbiter (and in the case of STS-9 the Spacelab module), plus the fairly ambitious schedule of operational activities during the first several days in flight, are conditions condusive to precipitating symptoms in susceptible individuals.

The CSSI test which involves exposure to Coriolis stimulation clearly did not predict susceptibility to SAS. This outcome was suggested as early as 1974 when similar preflight CSSi test procedures failed to correlate with SAS during the Skylab missions. The Skylab data, however, could not be interpreted accurately because of confounding variables. The larger sample size obtained during STS flights 1-9 unequivically rules out the use of a single CSSI test for predicting susceptibility. These data and previous similar data underscore the difficulty in predicting susceptibility to SAS with a single test procedure. Additional data must be collected with several different test methods in an attempt to establish a composite index or susceptibility profile. In this regard the CSSI test was discontinued and new preflight data collection procedures were implemented beginning with the STS-11 flight. These procedures include an off-vertical rotation test which provokes motion sickness by otolith stimulation and a sudden-stop test which involves vestibular and visual stimulation. Both procedures are modifications of techniques previously developed by Graybiel and his colleagues (7,8).

In assessing the effectiveness of medications utilized inflight, it must be recognized that they were medications usually taken after orbital insertion and may have had insufficient time to reach a therapeutic level before the individual was stressed. Orally administered scopolamine normally requires 60-90 minutes to reach its peak effectiveness. Some crewmembers were already beginning to move about in the vehicle within that period of time. On the basis of available data it cannot be determined whether or not affected crewmembers would have had more severe symptoms if they had not used anti-motion sickness medication. Verbal reports suggest that the medication was having some positive effect, although, the time course of action seemed to be delayed. Reduced gastric motility apparently interferred with absorption of oral medications. Clearly, additional ground based and flight data must be collected to establish more efficient drugs and drug administration strategies. Because preflight drug efficacy testing has not had a major influence on the actual drug used inflight, such testing with the rotating chair was discontinued after the STS-9 mission.

Alterations to Mission Timelines Induced by SAS

On the basis of available information, a definitive statement of the effects of the SAS on mission operations and crew performance cannot be made. No quantitative testing of crew performance has been conducted during any of our past space flights. Also, specific and complete information on meaningful mission timeline alterations induced by SAS is not readily available.

In general, however, it is known that the overall impact of this syndrome on mission operations in the U.S. space program has been minimal. Planned crew activities on only four missions (about 10% of all flights to date) have been altered by the syndrome. A planned extra vehicular activity (EVA) on Apollo 9 was postponed one day in order to allow the crewmember scheduled to do the EVA an opportunity to fully recover from symptoms. The crew of the Skylab 3 mission went into a "powered-down" mode (i.e. reduced their workload) during approximately the first 36 hours of flight because of SAS symptomatology. A scheduled light work load day was traded with a busy work load day to allow the crew of the STS-3 mission to overcome symptoms. Lastly, a planned EVA on STS-5 was postponed one day to ensure that an affected crewmember was fully recovered from symptoms of SAS. (The STS-5 EVA was ultimately cancelled, not because of crew health, but because of failures in both EVA suits.)

None of the four events cited above had a deliterious impact on the successful accomplishment of mission objectives. Likewise, on all other missions where SAS symptomatology occurred, no degradation in mission objectives occurred despite the fact that the operational efficiency of affected crewmembers was probably impaired by some unknown amount. A significant factor is that with larger, cross-trained crews on STS, temporary performance inefficiencies experienced by part of the crew can be compensated for by other unaffected crewmembers. Such trade-offs may not be possible if the entire crew is moderately to severely affected by SAS symptoms. On the basis of past experience the likelihood of an entire STS crew being affected is remote.

General Considerations

The precise mechanisms underlying SAS are not fully understood, however, most investigators in this area agree that the syndrome has its origin in the vestibular system. It is generally believed that the combination of varying degrees of microgravity induced alterations in vestibular (especially otolith), proprioceptive and somatosensory function plus unaltered visual function, causes sensory-perceptual conflict of the type originally described by Reason and Brand (9). The importance of vestibular function and the probable role of sensory-perceptual conflict are underscored by reports that head movements and unusual visual orientation attitudes within the spacecraft both trigger and aggravate SAS symptomatology. Pronounced headward shifts of body fluid occur in microgravity. Investigations to date have failed to provide clear evidence for implicating fluid shifts in the SAS syndrome (10, 11, 12).

In addition to the present lack of understanding of the etiology of SAS, issues of greater operational significance remain unresolved. Paramount among these are the issues of prediction, prevention, and treatment. Ground-based techniques for the a priori identification of persons susceptible to this syndrome have not yet heen validated. Effective and operationally acceptable countermeasures have not been developed. Anti-motion sickness medications have been used with some regularity in the U.S. program, but their therapeutic value has been unsatisfactory. Nonpharmacological approaches, for example adaptation training or biofeedback training, have not matured to the stage where they can be applied in a routine fashion to astronaunts. In short, SAS remains an operationally relevant biomedical problem for manned space flight and has demonstrated potential for affecting the well being and optimum performance efficiency of flight crewmembers.

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DISCUSSION

BURCHARD: What was the impact on the vomiting eposides as far as food and water intake was concened?

HOMICK: The data we have in this area were rather sketchy and depend entirely on post flight suggestive reporting from the crewmen. Generally speaking, during the first days of flight when many of the crewmen were not feeling well, there was an obvious tendency not to eat very much solid food and a number of crewmen existed entirely on liquids. I can't recall any specific information about the relationship between ingesting food relative to a vomiting incident although certainly on some occasions crewmen have reported shortly after consuming some liquid or some food to have it come back up again.

MONEY: How effective is metaclopromide against motion sickness on Earth, and how effective is it in space? Who coined the term "Space Adaptation Syndrome"?

HOMICK: Data from ground based studies are very limited. One study done at the Johnson Space Center showed that metaclopromide was ineffective as a countermeasure for motion sickness induced by exposure to Coriolis cross coupling stimuli in a rotating chair. Metaclopromide used during space flight has produced mixed results. A few crew members experienced relief from nausea and vomiting with the drug. Other experienced no beneficial effect. In the latter cases, the crewmen were also using scopolamine, a pharmacological antagonist of metaclopramide. The term "Space Adaptation Syndrome" was coined by NASA management personnel. The term was originally coined as a substitute for the term space motion sickness. However, the term is now generally used to describe the entire spectrum of physiological responses to weightless space flight that may have some potentially negative consequences for the crew, thus, cardiovascular deconditioning may be considered a part of the space adaptation syndrome. Space motion sickness is now generally considered a subset of the space adaptation syndrome.

ANALYSIS OF HEAD MOTION DURING SIMULATED, ROUGH WATER OPERATION OF A 2200 TON SURFACE EFFECT SHIP

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SUMMARY

Nineteen Navy volunteers were exposed, for periods up to 48 hours, to simulated motion environments predicted for a 2,000,000 Kg (2200 ton) surface effect ship. Surface effect ships, which are supported by a cushion of air, operate at high speeds and produce motion strongly influenced by the dynamics of the air cushion. The motions of both the environment and the head of each volunteer were measured during scheduled 5 minute intervals and the relationship of head motion to impending emesis was investigated. The time series data and the frequency spectra were examined to identify variability in head response resulting from: repetitions with the same subject, repetitions with different subjects, repetitions with a subject in different positions, repetitions simulating different ship operating conditions, repetitions with and without pitch and roll motions, differences between well and sick subjects, fatigue, and progression to emesis. Heave, pitch and roll motions in the range of 0.05 to 1.5 Hz were simulated. Results of the analysis indicated that a correlation between spontaneous head motion and motion sickness exists. Additionally, the results demonstrate the utility of studying the effects of motion in a controlled laboratory environment. The methodologies developed may readily be extended to other ship motion problems.

BACKGROUND

A new ship technology (1,2) which may produce large scale surface effect ships (SES) that can operate at speeds up to 150 Km/hr (80 knots) in the open seas is under world-wide development. The SES is supported primarily on a large plenum of air, resulting in reduced hydrodynamic drag and high operating speeds. The motions characteristic of these ships represent a significant departure from the motions produced by present ships of comparable displacement. The spectrum of the heave motion, which depends upon the wave encounter rate and the ship's natural heave frequency, extends to higher frequencies than conventional ships. Pitch and roll amplitudes are reduced. The effects of this motion on crew personnel may be different from that experienced on conventional ships.

One of the principal complications resulting from ship motion is motion sickness. Many investigations of motion sickness have been reported in the literature (3), but specific procedures for predicting tolerance to complex ship motions have never been successfully formulated. Thus, although the motion of an SES can be predicted from the engineering design, the effects of the motion on the ship's occupants is largely conjectural until the craft has been built and tested at sea.

The U.S. Navy initiated a program for investigating crew habitability of a representative large scale SES. Nineteen novice U.S. Naval enlisted research volunteers, specially selected for hazardous duty biodynamic research, were exposed to heave, pitch and roll motions while inhabiting a closed cab for periods up to 48 hours. The cab motions were defined using a computer simulation based upon the engineering design of a 2,000,000 kg (2200 ton) SES, and upon performance measurements taken from an operational 45,000 kg (100 ton) prototype SES (4,5). Five minutes of simulated data were generated for three different SES operating conditions (Table I). In each condition, the heading of the ship with respect to the wave velocity was 135° (i.e., the waves were approaching the ship from the starboard bow). During each exposure, the cab was driven using the simulated data from one sea state, with the five minute segment of motion being repeated for the duration of the exposure. Of primary interest in the study were the changes in visual-motor functions, cognitive functions, physiological stress, sleep, and clinical medical effects caused by the motions experienced by the subjects. These results have been reported previously (6,7,8,9). A secondary objective of the investigatio was the measurement of the motions of test subjects' heads, and the correlation of those head motions with cab motion and with susceptibility to motion sickness. Analysis of the motion data shows that previous research reporting a relationship between sinusoidal motion and incidence of motion sickness may be extended to motions with complex spectra. It also shows that subjects experiencing nausea exhibit characteristic head motions not seen in well subjects. This paper summarizes the results of the head motion study.

TABLE I. TEST CONDITIONS

Simulated Vessel Speed km/hr (knots)	Sea State	Wind knots (km/hr)	Average Wave Heights meters (feet)	Average Wave Period seconds
150 (80)	3	15 (28)	0.75 (2.5)	4.3
110 (60)	4	18.5 (34)	1.21 (4.0)	5.2
75 (40)	5	21 (39)	1.73 (5.7)	6.0

NOTE: SES heading with respect to wave velocity was 135°.

EXPERIMENTAL PROCEDURES

The original experimental schedule required that 12 volunteer test subjects be exposed, in pairs, to three separate 48 hour test segments. In each segment, two subjects lived in a small (2.5m X 2.5m X 2.5m) cab which moved with heave, pitch, and roll approximating the motion of the center of gravity of a 2200-ton SES in one of the three operating environments (Table I). The motions and cabin arrangements have been fully described previously (4,6).

In the tests, head motion was to be measured every 12 hours using a mouth-mounted package of accelerometers. In each measurement period the subject was to sit for five minutes facing the bow, sit for five minutes facing to starboard, and finally stand for five minutes facing to starboard. The data would be recorded on analog tape and selected portions of it later digitized for comprehensive analysis.

As often happens in experimental research, frequent deviations from the original schedule were required (9). The high incidence of emesis shortened the exposures for many subjects, and prevented some from continuing to the more severe conditions. Frequently, complaints of motion sickness symptoms shortened or eliminated the measurements of head motion. Equipment malfunctions required that some tests be run at other than the prescribed operating conditions. As a result, the data had to be carefully reviewed to determine how meaningful analysis could be performed; some of the original comparisons planned were found to be impossible, but the possibility of other approaches was opened for consideration. The eventual analysis of head motions using the available data included:

1. Long-term repeatibility of a subject's mechanical response to different exposures to the same cab motions.

2. Differences in a subject's response as the length of continuous exposure to motion increased.

3. Differences in a subject's response as he approached emesis.

4. Differences in a subject's response for one exposure to motion in which emesis occurred compared with a second exposure to the same motion in which emesis did not occur.

5. Comparison of a subject's response for each of the three body positions assumed during measurement of head motion.

6. Differences between subjects undergoing identical cab motions.

7. Comparison of the same subject's response for each of the different cab motions (sea states 3, 4, 5).

8. Differences in a subject's response resulting from attenuation of the cab's heave acceleration.

Special instrumentation was required for measuring head motion. Each subject was fitted individually with specialized anatomical mounts to accommodate six accelerometers for measuring three dimensional motion of the head. These mounts and associated straps, harnesses and hooks required to hold them in place were developed and fabricated at the Naval Biodynamics Laboratory (NBDL), formally NAMRL Detachment, and have been described previously (10,11). The instrumentation packages are designed to measure linear and angular acceleration in three dimensions. The package consists of a T-shaped aluminum plate on which six accelerometers are mounted. The positions and alignments of the accelerometers are selected, within the geometrical restrictions of the package, to minimize computational errors incurred during data reduction. The instrumentation package is fastened to the subject using a stainless steel bite plate which is custom molded to fit the subject's upper jaw. This fixture assures accurate and repeatable positioning of the instrumentation for every test.

In order to make valid comparisons of the motions of different test subjects, it is necessary to represent the acceleration data in terms of the subject's anatomy. The head coordinate systems are defined by stereoradiographic measurements (10,12) made at NBDL for each test subject prior to his first exposure to SES motion. Briefly, the procedure for defining the coordinate system requires that a subject wear, firmly fixed to the mouth mount, a dummy instrumentation package containing lead markers at carefully measured locations. Lead markers are also used to highlight specific features of his bony anatomy. The test subject is then positioned at the perpendicular intersection of the cones of radiation from two x-ray tubes, and exposures are made as nearly simultaneously as possible. Using a computational procedure (12) the geometry relating the location of the instrumentation with respect to the bony anatomy is determined from the two x-ray images. The geometrical relationships are then used to represent the measured acceleration data in a standard coordinate system that is common to all test subjects, and whose origin and orientation are defined with respect to prominent skeletal landmarks. Thus, differences in motions observed between test subjects indicate that the subjects are moving differently, not that there are merely differences in instrumentation setups.

Head motion was measured with piezoresistive accelerometers which produced differential output voltages proportional to the accelerations present. Each signal was amplified by a differential amplifier, encoded to an FM format using a voltage controlled oscillator (VCO), and recorded on instrumentation quality analog magnetic tape. The tape was transferred to an NBDL digitizing system in which the FM formatted signals were converted back to voltage amplitudes, and then converted to binary code using an analog to digital converter. Band-pass of the system was limited to DC to 100 Hz by the VCOs. Each signal was sampled 200 times/second and the corresponding digital values written to a magnetic tape suitable for processing on a UNIVAC 1100 digital computer. The subsequent computer analysis consisted of scaling the data to engineering units, smoothing the data using digital filters, transforming the acceleration data to a standard coordinate system in the subjects' anatomies,

determining the spectral content of the signals using fast fourier transform routines, and plotting all data of interest in easily understandable formats. The comparative studies were performed using the graphic output.

The data channels were calibrated at the start and end of each motion exposure by substituting a nine-level precision voltage staircase in place of each accelerometer. These calibration signals were also digitized, and used to scale the accelerometer data to engineering units (meters/second^2).

DATA ANALYSIS

In a typical experiment, the test subject wore a standardized instrumentation package rigidly fastened to his upper jaw. Six miniature linear accelerometers mounted in the package produced electrical signals which were recorded on analog tape, digitized, and used to reconstruct the three dimensional acceleration of the head. Processing of the data required careful execution of a number of related operations, as outlined in Table II. The following major functions were performed.

1. Pre-experiment calibration was performed using on-site facilities located at NBDL. For the SRS simulations, the following functions were performed.

a. Before they were used in the experiments each standard accelerometer package was calibrated at NBDL under computer control, using a spinning rate table to apply precise accelerations to the package. The resulting accelerometer voltages were measured to determine the calibration curve and direction of the sensitive axis for each accelerometer. Calibration errors were less than 0.3% of full scale (0.3 m/s^2, or 0.03g for the accelerometers measuring data in this paper). The precise location of each accelerometer was determined by the manufacturer during assembly of the package. Reference 11 describes the calibration process more completely.

b. Stereographic x-rays of each subject were taken to determine the three-dimensional location of the instrumentation package relative to a coordinate system defined by bony anatomical landmarks visible in the x-rays of each subject's head.

2. Accurate calibration information, generated at the time of the experiment, was required for each data channel. The following steps were required.

a. Before and after each series of experiments, a nine-step staircase of precisely measured voltage levels was substituted for each sensor output, and recorded on the analog tape.

b. After the experiments were complete, the calibration signals recorded on tape were digitized under computer control.

c. The digitized staircase signals were converted to the standard NBDL data format.

d. The relationship between acceleration and the resulting digital number was calculated using the sensor calibration information from step 1a, and the staircase calibration from step 2c.

3. Sensor data were recorded and analyzed as follows:

a. Five minute intervals were scheduled during which a subject in the moving cab would wear his mouth mounted instrumentation package. The signals produced by each accelerometer were recorded on analog tape. Cab motion was also recorded using both NBDL instrumentation and signals from the cab sensing electronics.

b. After the data collection was complete, selected signal data recorded on tape were digitized under computer control. All channels were sampled at 200 points/second for five minutes. The high sampling rate was selected to avoid folding 60 Hz noise into low frequency data.

c. The digitized signal data were converted to the standard NBDL format.

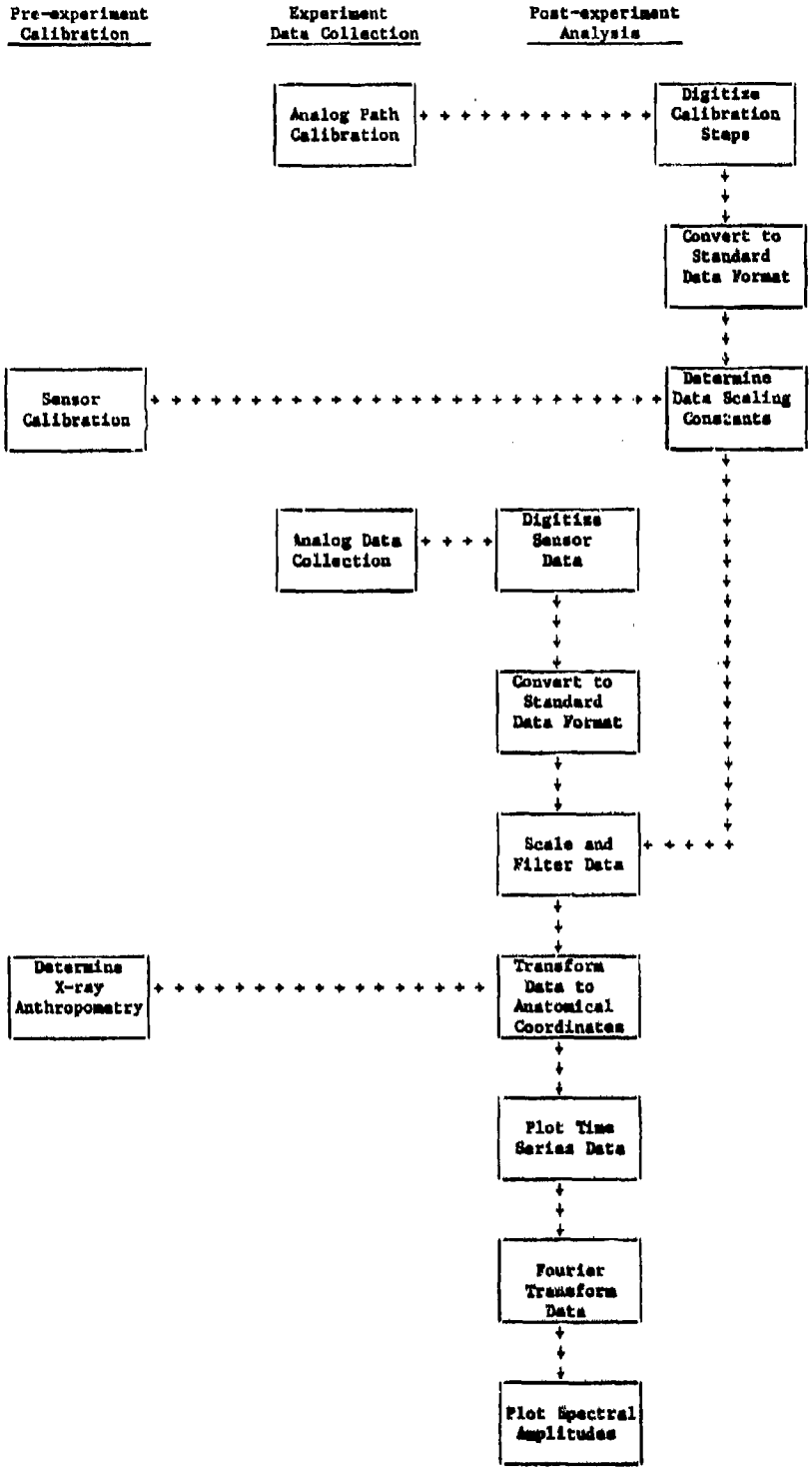
d. The data were filtered, condensed, and scaled from volts to physical units. Filtering was done with four cascaded, second-order, linear, recursive digital filters. Two were low-pass filters with a cutoff frequency of 5 Hz and a damping ratio of 0.7. The third was a high pass filter with a cutoff frequency at 0.05 Hz. The fourth was a notch filter at 1.960 Hz, designed to remove the response to a structural resonance of the motion simulation system. Data for each channel were compressed from 200 points/second to 40 points/second after being filtered. The scaling information determined in step 2d was used to convert the data to physical units (e.g., m/s^2).

e. The data measured by the mouth-mounted accelerometer package were processed to determine the motion of the subject's head, specified in the standard coordinate system defined by each subject's anatomical landmarks. The origin of the coordinate system was the center of the line connecting the ears (left and right auditory meatuses). The X-axis projected forward through the point midway between the eyes (left and right infraorbital notches); the Y-axis projected through the left ear; and the Z-axis extended upward, mutually perpendicular to the X and Y axes. Considerable effort was spent evaluating signal processing algorithms which could separate angular acceleration information from signal noise. None were successful, primarily because head angular accelerations were so small (generally less than four rad/sec^2).

f. The time series data for the head linear accelerations and the cab motions were plotted in a strip chart format.

TABLE II

FLOW OF DATA THROUGH ANALYSIS SYSTEM



g. A fourier transformation was calculated for 2048 points from each sensor. A cosine taper was used to smooth the first and last 10 percent of the data to reduce the errors caused by wraparound discontinuities.

h. The spectral amplitudes were plotted in the format of the figures shown in this paper. Both the amplitude and frequency scales are linear. Plot scales were selected for each variable which would provide the greatest resolution for a particular sea state condition. Spectral amplitudes, rather than power spectral densities, were plotted. To convert data in this paper to the equivalent power spectral density, the amplitude value plotted should be squared, and divided by 0.0125 Hz.

RESULTS

The experimental design called for the evaluation of human response to motion simulating the SES operating conditions summarized in Table I. The spectral content of the cab vertical heave, angular pitch, and angular roll motions measured for sea states 3, 4, and 5, respectively, are shown in Figures 1a, b, and c. As the conditions progressed from sea state 3 to sea state 5, the amplitude of the spectral peaks increased very modestly (note the change in plot scales), but the peaks shifted significantly to lower frequencies as the wave encounter rate decreased.

McCauley and Kennedy (13) reported maximum incidence of motion sickness centered at frequencies of 0.16 Hz for sinusoidal heave motion. Figure 1a shows negligible motion at 0.16 Hz for sea state 3; Figure 1b shows considerable motion at 0.16 Hz about the pitch axis for sea state 4; and Figure 1c shows substantial heave, pitch and roll motion at 0.16 Hz for sea state 5. The incidence of emesis was examined to determine if McCauley and Kennedy's findings may have application to the complex spectra present in Figures 1a, b, and c.

Incidence of Emesis

Thirteen subjects experienced at least one episode of emesis during a collective total of 79 exposures to simulated ship motion. In some cases, pitch and roll motions were not present, heave motions were attenuated, or the experiment was stopped early for equipment repair. However, each of the 79 exposures could be classified by matching the spectral content of the cab heave motion with Figures 1a, b, or c. The percentage of exposures resulting in emesis was calculated. As summarized in Table III, the collective incidence of emesis for these subjects was 17% for sea state 3, 50% for sea state 4, and 64% for sea state 5. Five of the thirteen subjects who vomited during sea state 3 or 4 conditions did not participate in a sea state 5 run, possibly biasing the 64% incidence of emesis for sea state 5 toward the low side. These results provide very convincing, if heuristic, support for the hypothesis that, even for complex motions, spectral components near 0.16 Hz dramatically increase the incidence of motion sickness.

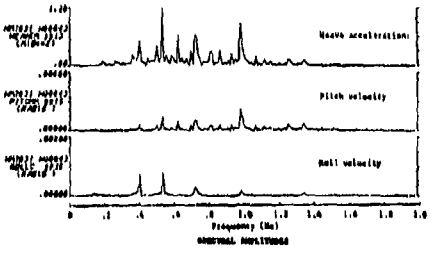


Figure 1a. Cab motion for sea state 3 conditions.

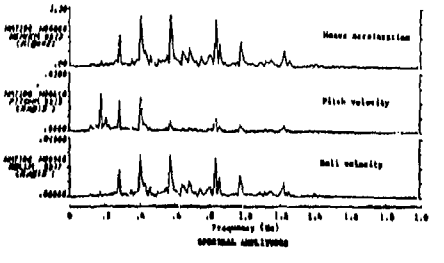


Figure 1b. Cab motion for sea state 4 conditions.

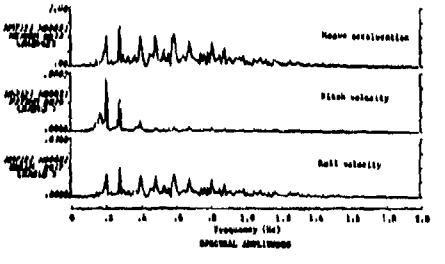


Figure 1c. Cab motion for sea state 5 conditions.

TABLE III

INCIDENCE OF EMESIS GROUPED BY SEA STATE CONDITIONS

Sea State	Total Exposures	Exposures Resulting in Emesis	Incidence of Emesis
3	23	4	17%
4	22	11	50%
5	14	9	64%

Head Motion Measurements

The original experimental design included provisions for analyzing the mechanisms of motion sickness. Mouth-mounted instrumentation was provided for measuring the three-dimensional motion of a subject's head while in the moving cab. The protocol called for measuring each subject's head motion for five minutes while seated facing the bow, while seated facing starboard, and while standing facing starboard. The measurements were to be repeated up to four times for each exposure to motion.

A review of the instrumentation and medical logs showed that 322 5-minute segments of head motion were recorded. However, instead of three motion conditions (sea states 3, 4, and 5), equipment malfunctions resulted in the addition of six alternate motion environments, including combinations of attenuated heave accelerations and elimination of pitch and roll motion. Further examination of the instrumentation and medical logs showed that test subjects experiencing discomfort or nausea frequently requested that the head motion measurements, which required placing a steel bite plate in their mouth, be cancelled. As a result, most of the head motion data was collected from subjects who were not feeling sick, and the few measurements from subjects approaching emesis could not always be compared directly with a large group of other subjects.

Baseline Response (Subject 43)

Subject 43 was selected as a baseline subject. He participated in 12 separate tests with no reports of nausea and no emesis, and appeared to be a consistent performer throughout the program. Unique head motion related to motion sickness, or to susceptibility to motion sickness, probably would not be observed in Subject 43. The following excerpts from the run log are reported to help the reader understand the data in the context of the experiment (times are noted using the 24-hour clock convention).

29 August

2100: Pre-run examination Subject 43: weight 141 lbs; temperature 98.4°F; pulse rate 76; blood pressure 118/68. No unusual activity or excessive stimulation. Coordination okay, mood good.

Note: Two other subjects also participated in the test. Subject 50 initially accompanied subject 43 in the cab. Subject 48 was on-site in a standby status, and later replaced subject 50 in the cab.

2256: Motion started.

2326: Motion stopped. One hydraulic drive pump developed bearing noise, and is turned off.

2342: Motion restarted with no roll or pitch motion, heave acceleration attenuated to 0.19g rms.

30 August

0024: Subject 50 reports nausea for the past 10-15 minutes.

0032: Subject 50 resting on table, head in hands.

0035: Subject 50 reading bag, not watching radar.

0040: Subject 50 appears to be retching? No, just holding bag at ready.

0050: Subject 50 asks to be released, about to vomit. Cannot look at task, and "everything spinning around".

0052: At test director's direction, stop was made by Subject 50 using abort switch in moving cab. Subject 50 exits.

0058: Subject 50 expressed and fulfilled a need to self-induce vomiting.

0123: Subject 48 placed in cab as replacement for continuation of run. Subject 43 is still asleep.

0131: Motion restarted. Subject 43 asleep.

0158: Subject 48 reports stomach awareness.

0206: Subject 48 vomits.

0208: Subject 48 reports severe nausea.

0210: Subject 48 retches again.

0217: Subject 48 requests release.

0220: Motion stops. Subject 48 exits. Subject 43 asleep.

0231: Motion restarted. Subject 43 alone for remainder of test.

0835: Motion stopped for unknown reasons. Subject 43 unaffected, and performing routinely.

0843: Subject 43 released from cab pending investigation of motion failure. Subject 43 reports strong vertical phantom motion, but no rotation. Mood good, general condition good.

0930: Subject 43 feeling normal.

1023: Motion restarted with Subject 43 alone.

1102: Subject 43 seated, facing bow; head acceleration recorded.
1112: Subject 43 seated, facing starboard; head acceleration recorded.
1118: Subject 43 standing, facing starboard; head acceleration recorded.

2100: Subject 43 seated, facing bow; head acceleration recorded.
2106: Subject 43 seated, facing starboard; head acceleration recorded.
2111: Subject 43 standing, facing starboard; head acceleration recorded.

31 August

0936: Subject 43 seated, facing bow; head acceleration recorded.
0942: Subject 43 seated, facing starboard; head acceleration recorded.
0947: Subject 43 standing, facing starboard; head acceleration recorded.

2108: Subject 43 seated, facing bow; head acceleration recorded.
2113: Subject 43 seated, facing starboard; head acceleration recorded.
2119: Subject 43 standing, facing starboard; head acceleration recorded.

2124: End of motion.

2130: Post-run examination, Subject 43: weight 146 lbs (5 lbs gained during run); temperature not taken; pulse rate 60; blood pressure 132/78. Subject 43 slept and ate well and feels fine except for the feeling that he is "going up and down".

Sea state 5 motion was simulated in the above experiment, with pitch and roll velocities set to zero, and the heave acceleration attenuated to 0.19g rms. Comparing the heave spectrum for the test, shown in Figure 4, with the heave spectrum for the standard sea state 5 condition, shown in Figure 1c, confirms that the two spectra are similar except for the amplitude attenuation. Figures 2a, b and c show the spectral components of the linear acceleration along the subject's head X, Y and Z axes, respectively, sampled at various times while the subject was in a seated position. Because no pitch or roll motion is present in the cab, the head X and Y accelerations are relatively small. The spectral peaks which occur in the X direction (Figure 2a) correspond to the peaks in the cab heave acceleration (Figure 4). The larger peaks seen in the measurements made at 12 hours indicate the subject's head was not level, and the accelerometers were sensing a component of heave acceleration along the X axis of the head. The motion measurements made at 12 hours occurred approximately 30 minutes after a 2 1/2 hour interruption of motion for equipment repair, and may indicate the subject is becoming reacclimated to motion. The seated subject's acceleration along the head Z axis (Figure 2c) was similar to the cab heave acceleration (Figure 4).

Similar results were observed for the subject in the standing position. Accelerations along the head X axis (Figure 3a) still appeared to be a result of the head not being level. As the run progressed, the amplitude of response along the X axis diminished, suggesting that some adaptation may have taken place for the subject in the standing position. Accelerations along the Y axis (Figure 3b) were very small, and along the Z axis (Figure 3c) were similar to the cab heave acceleration (Figure 4).

Analysis of the data measured during this run shows no surprises, and can be explained in terms of the cab motion. There appears to be little, if any, adaptation to motion which influences the head response. Adaption, if present, is limited to the angle at which the subject holds his head relative to the heave motion.

Effects of Nausea: Subject 59

Subject 59 was exposed to a modified sea state 4 motion in which the heave acceleration amplitude of the cab was attenuated to 0.19g rms. The spectral amplitudes of the cab motion, shown in Figure 7, indicate the measured roll velocity was almost zero. The problem was not documented, but the suspected cause is instrumentation error. No other indications of zero pitch and roll motion were found.

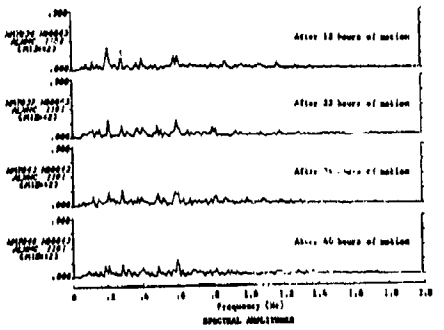


Figure 2a. Head x response of a seated subject, sampled during a 48 hour test. (No motion sickness).

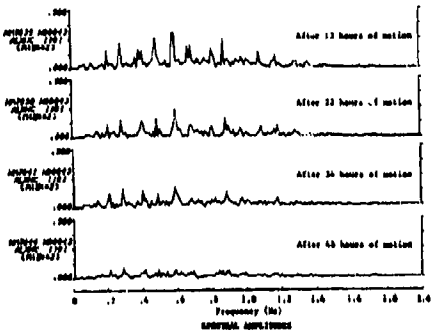


Figure 3a. Head x response of a standing subject, sampled during a 48 hour test. (No motion sickness).

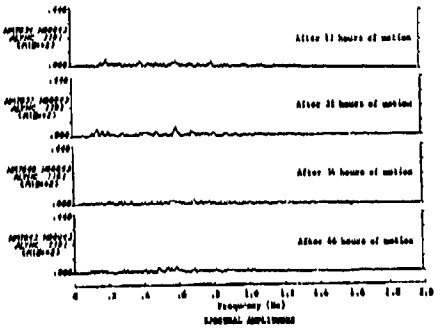


Figure 2b. Head y response of a seated subject, sampled during a 48 hour test. (No motion sickness).

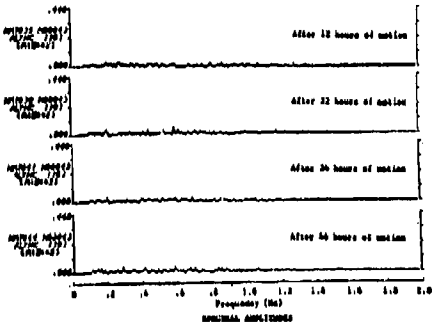


Figure 3b. Head y response of a standing subject, sampled during a 48 hour test. (No motion sickness).

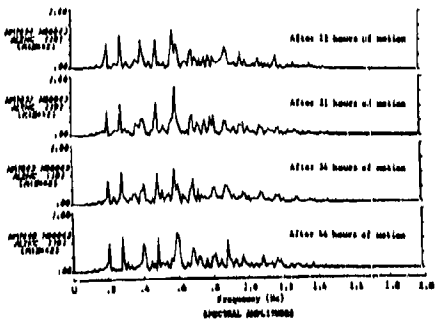


Figure 2c. Head z response of a seated subject, sampled during a 48 hour test. (No motion sickness).

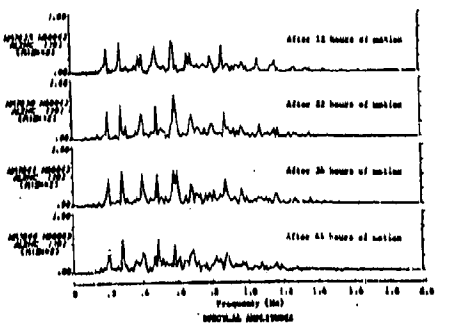


Figure 3c. Head z response of a standing subject, sampled during a 48 hour test. (No motion sickness).

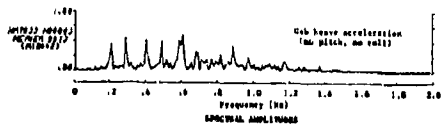


Figure 4. Cab motion for Figures 2a, b, c and 3a, b, c.

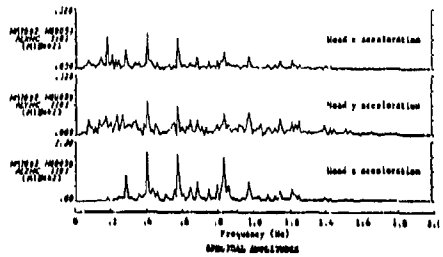


Figure 5a. Head x,y,z response of subject 59, seated, facing the bow (normal) condition).

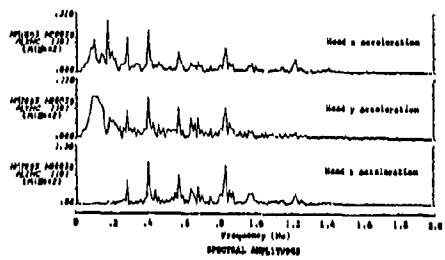


Figure 5b. Head x,y,z response of subject 59, seated, facing the bow (nausea condition).

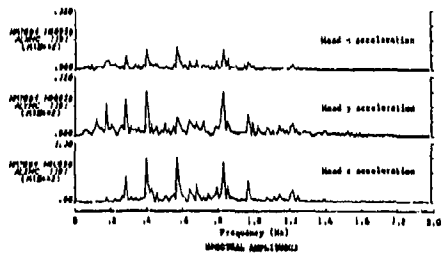


Figure 6a. Head x,y,z response of subject 59, standing, facing starboard (normal condition).

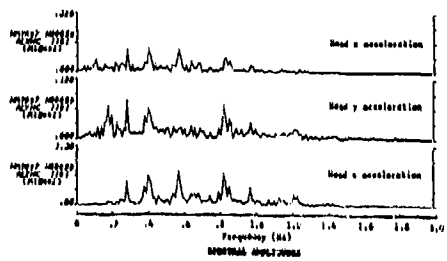


Figure 6b. Head x,y,z response of subject 59, standing, facing starboard (nausea condition 1).

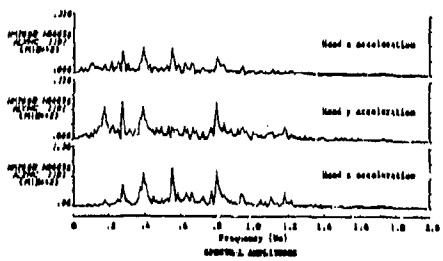


Figure 6c. Head x,y,z response of subject 59, standing, facing starboard (nausea condition 2).

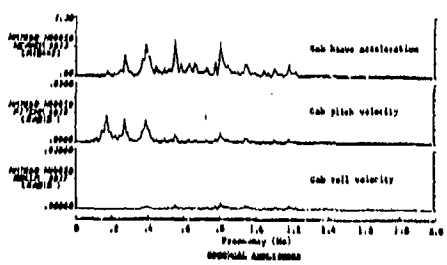


Figure 7. Cab motion for Figures 5a,b and 6a, b, c.

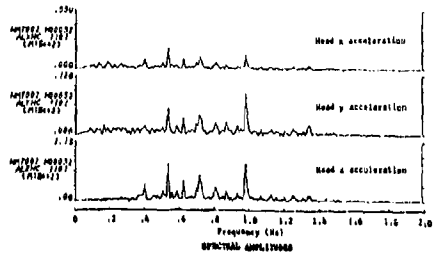


Figure 8a. Head x,y,z response of subject 52, seated, facing bow (normal condition).

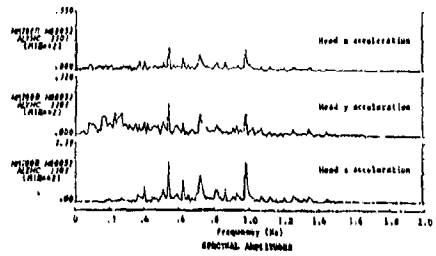


Figure 8b. Head x,y,z response of subject 52, seated, facing bow (nausea condition).

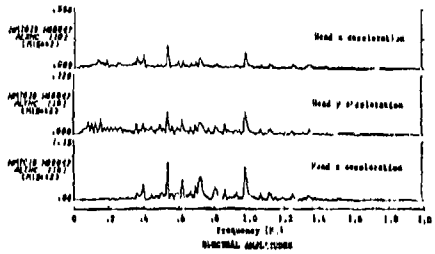


Figure 9a. Head x,y,z response of subject 47, seated, facing bow (17 minutes before emesis).

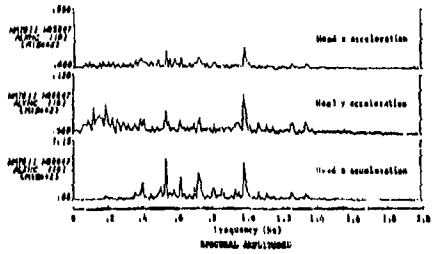


Figure 9b. Head x,y,z response of subject 47, seated, facing starboard (11 minutes before emesis).

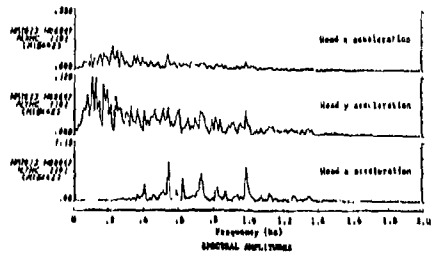


Figure 9c. Head x,y,z response of subject 47, standing, facing bow (1 minute before emesis).

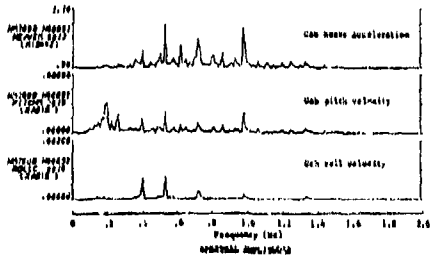


Figure 10. Cab motion for Figures 8a, b and 9a, b, c.

The motion of the subject's head was measured twice; once 14 minutes after the cab motion started, and again 5 hours later. During the second set of measurements, the subject experienced nausea, permitting within-subject comparisons to determine if head motion may be altered when nausea is present. The subject completed the entire run without vomiting.

The response of the seated subject, facing the bow, without nausea is shown in Figure 5a, and with nausea in Figure 5b. The most significant difference in the two measurements is the increased motion below 0.2 Hz in the X and Y directions when nausea was present. In a second comparison, the response of the standing subject, facing starboard, without nausea is shown in Figure 6a, and two consecutive periods with nausea in Figures 6b and c. Very clearly, the motion below 0.2 Hz increases along the Y axis when nausea is present.

For this subject, the presence of nausea is accompanied by increased motion below 0.2 Hz along the head Y, and possibly Z, axes, even though the motion of the cab remains unchanged. Additionally, the frequencies involved are the same as those identified by McCauley and Kennedy (13) as producing the highest incidence of motion sickness.

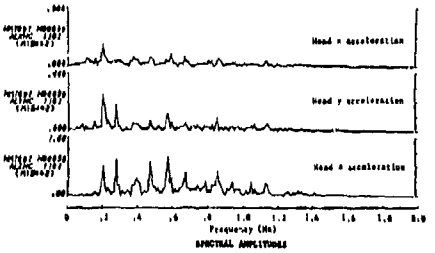


Figure 11a. Head x,y,z response of subject 59, standing, facing starboard (3 minutes before emesis).

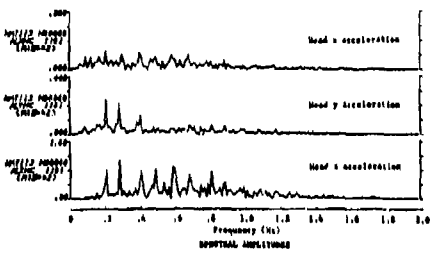


Figure 11b. Head x,y,z response of subject 60, standing, facing starboard (emesis in other tests).

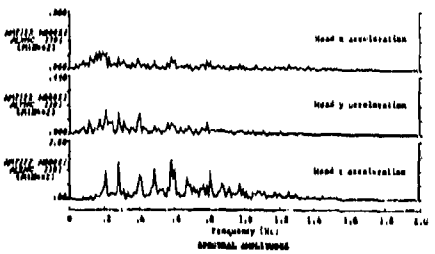


Figure 11c. Head x,y,z response of subject 51, standing, facing starboard (no history of emesis).

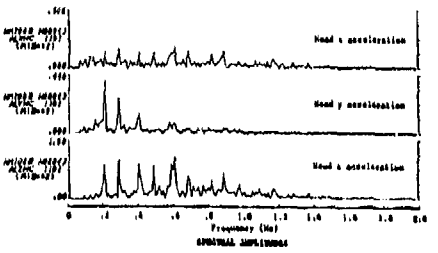


Figure 11d. Head x,y,z response of subject 43, standing, facing starboard (no history of emesis).

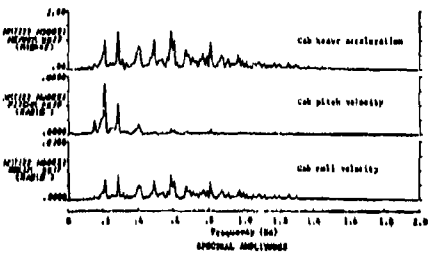


Figure 12. Cab motion for Figures 11a, b, c, d.

Effects of Nausea: Subject 52

Subject 52 was exposed to a modified sea state 3 motion in which the heave acceleration of the cab was attenuated 20%. The spectral amplitudes of the cab motion, shown in Figure 10, indicate considerable unwanted roll motion below 0.2 Hz for the cab. The cause of the problem is unknown, but it did not occur in later tests. Subject 52 reported periodic developing nausea throughout the run, attributed to lack of adequate ventilation, poor tasting water, and confinement. During the test, his partner in the cab vomited ten times. Subject 52 definitely felt he ate and drank less than normal. The motion of the subject's head was measured twice; once 14 minutes after cab motion started, and again 23 hours later. At both times the subject reported that burning head pains developed beneath the head mount on the right side, then spread throughout the area of the mount. Upon tightening the mount straps, a sharp pain developed in the corner of the right upper incisor which lasted several minutes. Subject 52 requested cancellation of the last head motion measurement (standing position) and termination of the run because of severe burning head pain and developing nausea. After the straps were loosened, the head pain and nausea disappeared, and he was able to remove the mount assembly as rehearsed. Upon removal from the cab, he had no dizziness or other problems. Within several minutes he developed a sense of extreme heaviness of legs, body and arms, but denied feeling light headedness, dizziness, numbness or tingling.

The motion of the subject's head at the start of the run (Figure 8a) was compared with his motion immediately prior to termination of the run (Figure 8b). During both measurements, the subject was seated, facing toward the bow. There are substantial components of acceleration below 0.2 Hz along the Y axis when nausea is present (Figure 8b) which were not present in the earlier motion (Figure 8a). The change in head motion when nausea is present is similar to that previously observed for Subject 59 under different motion conditions.

Onset of Emesis: Subject 47

Subject 47 was exposed to the same modified sea state 3 motion previously described for Subject 52. The spectral amplitude of the cab motion is shown in Figure 10.

The head motion measurements began 57 minutes after the start of cab motion. The first head response, 19 minutes before emesis, (subject in a seated position, facing the bow), shown in Figure 9a, exhibits a slight increase in low frequency amplitudes in the head Y acceleration. The head response 12 minutes before emesis (subject in a seated position, facing starboard) contains still larger low frequency amplitudes as shown in Figure 9b. After measurement in the first two positions was complete, the subject began feeling warm. Cab temperature was 77.8° F. Finally, the head response one minute before emesis (subject in standing position, facing the bow), shown in Figure 9c, was predominately low frequency motion in the Y axis. One minute after the last measurement was complete, the subject vomited on the floor. Subject 47 continued with the run for an additional 22 hours, and experienced nine more episodes of vomiting. Head motion measurements for the following day were cancelled by the medical monitor.

Data from Subject 47 demonstrate very dramatically the progressive increase in low frequency lateral motion that accompanies the progression of nausea to emesis. The motion along the Y axis of the head is characteristic of all three subjects who reported nausea while head motion was being recorded.

Comparison of 4 Subjects

Four subjects, for whom head motion was recorded, were exposed to the standard sea state 5 motion (Figure 12). Subject 59 vomited three minutes after head motion was measured. Subject 60 vomited during a different exposure to sea state 5. Subjects 51 and 43 did not experience emesis during the entire program. The motions of the four subjects were compared to determine if distinguishing characteristics could be identified which may be used to predict susceptibility to motion sickness. None were found.

More surprisingly, the characteristic low frequency components of Y acceleration previously observed during nausea were not present for Subject 59 just prior to emesis, while he was in a standing position, facing starboard (Figure 11a). The characteristic head motion was seen in the seated position several minutes earlier (not shown), and in a previous run involving Subject 59 in both seated and standing positions (Figures 5b, 6b). No explanation for the absence of low frequency lateral head motion was found. Subject 59 reported that the mouth mount which he had been wearing for 13.5 minutes before vomiting, caused the sickness, not the motion.

Head motions for Subjects 60, 51 and 43 (shown in Figures 11b, c and d, respectively) standing, facing starboard, were puzzling. Head X and Y accelerations for all three subjects showed small, but measurable, low frequency components (near 0.1 Hz) which were not present in the cab motion (Figure 12). The mechanism which produced the low frequency motion is unknown.

CONCLUSIONS

The relationships between moving environments with complex motion spectra below 1 Hz, the three-dimensional motion of a subject's head, and the presence of nausea were examined. Unfortunately, sufficient information for determining cause and effect relationships is not available. First, the history of the evolution of head motion from a normal to a nauseated state is not known since measurements were made only during widely separated intervals, primarily because of the discomfort caused by the instrumentation. Second, the head angular accelerations, which were small but possibly significant, could not be determined from the data recorded. Third, position and orientation of the head, which are not completely defined by acceleration, could not be determined. Finally, at the time of the experiment, it was not known that the characteristics of the head motion were changing, and no special attention was given to documenting the subject's awareness of motion discomfort. Nonetheless, the following important conclusions are made:

1. Observations indicate that the incidence of emesis increases as the amplitude of the spectral components near 0.16 Hz increases for complex spectra.

2. Observations indicate that nausea due to motion sickness is usually, although not always, accompanied by increased lateral acceleration of the head with a spectral content less than 0.2 Hz. The mechanism which causes this is not known.

3. Instrumentation used to measure head motion contributed to nausea. Instrumentation not mounted in the mouth would be more acceptable to test subjects, and could probably collect more data with less inconvenience.

4. Angular accelerations of the head were extremely small, and could not be measured with the instrumentation used.

5. Relationships between motion and motion sickness can be determined much more effectively if the motion data are collected, analyzed and reported while the experiment is in progress. The low data rates involved make real-time analysis feasible with state-of-the-art equipment now available.

The experimental results reported in this and related papers demonstrate the utility of studying the effects of motion on people in a controlled laboratory environment with repeatable motion conditions. It is expected that the methodologies developed for the SES simulation may readily be extended to other ship motion problems. Examples include:

1. Motions which are known to cause a problem on ships can be reproduced with the simulator. The spectral content of the motion can be modified to determine which components of complex motion are the principal cause of the problem, and sensitivity to small changes in amplitude and frequency of specific spectral components may be studied.

2. The effect of moving a workstation from one location to another in a ship can be studied by making the appropriate modification to the simulator motion.

3. Human response to motion of new ship designs can be evaluated by using the predicted motion to control the motion simulator.

4. Performance measuring equipment can be evaluated before it is taken out to sea.

5. Contours which relate frequency, amplitude and exposure time to performance can be determined for use as guidelines for ship design engineers.

6. Criteria which relate ship motion to crew performance and disruption of shipboard activity can be developed as guidelines for ship operations.

7. Habituation to specific motions can be evaluated.

8. Effects of anti-motion sickness measures can be evaluated.

9. Fatigue from specific motions can be evaluated.

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The volunteers were recruited, evaluated and employed in accordance with the procedures specified in the United States Secretary of the Navy Instruction 3900.39 Series and the Bureau of Medicine and Surgery Instruction 3900.6 Series. These instructions are based upon voluntary consent, and meet or exceed the provisions of prevailing national and international guidelines concerning human experimentation.

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DISCUSSION

OMAN: 1) Have you tried to quantitatively evaluate O'Hanlon and McCauley's simple frequency model against your data, or is your observation that as the low frequency spectral peak decreases (with increasing sea state), sea sickness increases as per McCauley a qualitative guess? 2) Are your findings re head acceleration: -apparent "adaptation" of asymptomatic subject over 25 hours in Y axis acceleration - increased low frequency Y axis acceleration when other subjects become sick suggestive that asymptomatic subjects work to keep their head vertical, and don't care about it as they get sick, and asymptomatic subjects acquire "sea legs"?

ANDERSON: The data reported represents only three different motions, each with relatively few sample points. No attempt was made to determine an algorithm for combining multiple, low-frequency components to predict motion sickness. However, the presence of two spectral components in sea state 5 is correlated with increased incidence of motion sickness. Sea states 3 and 4 are both complex motions, but each has only one major spectral component below 0.4 Hz. The incidence of motion sickness predicted by the Hanlon/McCauley model agrees with the sickness observed for sea states 3 and 4. Although the lateral head motion appears to be consistent with other stimuli which produce motion sickness, it was not anticipated in a predominantly heave environment. Consequently, we did not collect any information which would define mechanisms that produced the motion. For future work we are developing a computer-based system to collect and analyse data while the experiment is in progress. As a subject response changes he/she may be observed on closed circuit TV, or may be queried via an intercom to the cab, and we anticipate having a very adaptive and flexible test environment in which unexpected relationships can be identified and studied very rapidly.

VON GIERKE: Where was the center of rotation for the cab motions? Does the increase in head Y-motions indicate a decrease in the body's compensation for the forced oscillations?

ANDERSON: Cab motions were measured on the heave, pitch and roll control circuits; on the heave, pitch and roll assemblies; and on a work table in the cab. Heave motions at the three locations agreed. Pitch and roll measured at the table had large errors and could not be used for analysis. Center of rotation was about 18 inches beneath the floor at the cab. Cause of the increased y-motions of the head is an unanswered question. However, whether the subject faced forward or starboard seemed to have no effect on the head motion, suggesting the lateral head motion was not closely coupled with the angular motion of the cab.

A DOUBLE BLIND COMPARATIVE TRIAL OF POWDERED GINGER ROOT, HYOSINE HYDROBROMIDE, AND CINNARIZINE IN THE PROPHYLAXIS OF MOTION SICKNESS INDUCED BY CROSS COUPLED STIMULATION

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SUMMARY

A double blind laboratory trial was conducted to study the relative effectiveness of powdered ginger root (1G), hyoscine (0.6 mg), cinnarizine (15 mg) and a placebo in increasing the tolerance of subjects to the development of motion sickness symptoms induced by cross coupled stimulation. Tests were carried out at weekly intervals on sixteen subjects two hours after taking each drug. In order to assess the effect of each drug on performance, a range of tests was carried out in the period between ninety minutes and two hours after taking the drug.

The study confirmed the effectiveness of hyoscine in delaying the onset of motion sickness symptoms and showed cinnarizine to be similarly effective. However it failed to substantiate a previous report that powdered root ginger is of value in the prophylaxis of motion sickness. Significant differences in the results of performance tests were found only after the administration of hyoscine, which produced a small decrease in subjective alertness and a reduction in the velocity of saccadic eye movements.

INTRODUCTION

Drugs that are of value in the prophylaxis of motion sickness belong to a variety of pharmacological groups. Anticholinergic drugs (hyoscine, atropine), anti-histaminics (promethazine, cyclizine, cinnarizine) and sympathomimetics (amphetamine, ephedrine) have all been shown to be effective in delaying the onset or preventing the occurrence of motion sickness (1,2). All these drugs exert their effect principally through their action on the central nervous system and may, in consequence, show side effects that limit their usefulness. Indeed, a pharmacological action on the central nervous system may well be an essential requirement of any drug which is to be active against the full syndrome of motion illness.

A recent paper by Mowrey and Clayson (3) which reports a prophylactic effect on the development of motion sickness following the administration of powdered ginger root is of particular interest. Powdered root ginger is not known to have any action on the central nervous system. It contains a number of aromatic oils, zingiberene, gingerol and terpene derivatives, whose pharmacological actions are those of a carminative (4). They act directly on the gut to relax the cardiac sphincter and possibly to promote onward movement of the gut contents.

Some therapeutic benefit of cinnarizine in a single dose of 150 mg was noted in a comparative trial of sixteen drug combinations by Wood and Graybiel in 1968 (1) but its effect was small compared with hyoscine or promethazine. Its value in seasickness was demonstrated during sea-going trials by Hargreaves (5) at a lower dose level of 75 mg t.d.s. It has in recent years gained wide acceptance in the prophylaxis of seasickness on account of having a long duration of action and a lower incidence of drowsiness as compared with hyoscine and with other anti-histamines. However no other laboratory trial of the anti-motion sickness properties of cinnarizine at currently recommended dose levels has been carried out.

There is ample experimental evidence for the effectiveness of hyoscine in motion sickness prophylaxis (reviewed in 2). It was included in the present trial as a standard against which to compare both therapeutic effectiveness and the incidence of side effects.

METHOD

Sixteen fit male volunteers took part in the trial. In order to assess their susceptibility to motion sickness, subjects on entry to the trial completed the Reason motion sickness questionnaire (6). No selection of subjects was made on the basis of motion sickness susceptibility. However, one subject was replaced in the trial because the preliminary test failed to produce any symptoms of motion sickness. The sixteen subjects had a mean age of 28.4 yrs (range 20-44), and the motion sickness questionnaire indicated susceptibilities ranging from the 2nd to the 93rd percentiles.

The Motion Stimulus. Motion sickness symptoms were induced by cross-coupled (Coriolis) stimulation of the semi-circular canals (7). Subjects were seated in an enclosed cab mounted on a horizontal turntable. The cab was internally illuminated and allowed its occupant no view of the outside world. The subject was required to make head movements either forward or backward in pitch or to the left and right in roll through an angle of 45 degrees from the vertical, the limiting positions being determined by head rests mounted within the cab. The subject followed a pre-recorded sequence of instructions which asked him to make a head movement every three seconds to or from each head position in randomised order, each sequence lasting for 30 seconds. The turntable was accelerated at a rate of 0.1 deg/sec^2 for 15 min. After this time the angular velocity, which had reached 90 deg/sec, was held constant for a further 5 min.

Following each head movement sequence, the subject was asked to rate his symptoms. The rating system used was a seven point scale, in which a rating of one indicated no symptoms of motion sickness, and a rating of seven indicated the level of motion sickness symptoms at which he wanted the motion stopped. The intermediate points on the scale were allocated as the subject saw fit. The run was stopped either when the subject reached a symptom rating of seven or at the end of twenty minutes.

One week before starting the trial proper each subject was given experience of the motion stimulus profile. He was thus able to practice rating his motion sickness symptoms and to find a level of symptoms, that fell short of frank vomiting, to which he would be willing to progress in the subsequent four test runs. He was also urged to withdraw from the trial at this point if he had any doubt about completing the remaining four test sessions. In the event, none did so and all 16 subjects completed the trial.

Two hours before the start of the test, subjects took one of the following: 0.6 mg hyoscine hydrobromide, 15 mg cinnarizine, 1 g powdered ginger root, 1 g lactose (placebo). The drug was contained in two gelatine capsules. All capsules were made identical in appearance by the inclusion of lactose and small quantities of caramel and cocoa colouring. The order of administration of the drugs was randomised using non-repeating 4 x 4 latin squares. Neither the subject nor the experimenter knew which drug had been taken on any particular occasion.

In order to avoid variations due to possible effects of circadian rhythm on performance, and to reduce the possibility of adaptation to the motion stimulus, each subject was tested at the same time of day at intervals of 7 days over a four week period.

Saccade Measurement

The subject, seated in the dark, faced a black screen which carried a horizontal row of nine red light emitting diodes (LEDs). The central LED was situated at eye level two metres in front of the subject, and the LEDs on either side subtended 5, 10, 15 and 20 degrees to left and right. Eye movements were recorded electro-oculographically. Head movement was restricted by means of a bite bar. The subject was instructed to gaze steadily at the centre light and to shift his gaze as rapidly as possible to whichever peripheral light appeared. Under microcomputer control (HP 85) the central LED was illuminated for a period which varied randomly between 3 and 6 seconds, after which time one of the peripheral LEDs appeared. This event simultaneously initiated analogue to digital conversion of the electro-oculographic (EOG) signal for the following one second period at a rate of 500 samples/second. A total of 40 saccades were stored on disc for subsequent analysis.

Computer analysis of each saccade determined its velocity from the slope of a straight line fitted to the saccadic portion of the EOG record between 10% and 65% of its total amplitude. The analysis also measured saccade latency - the time from the appearance of the peripheral light to the start of the saccade, and saccade deficiency - the extent to which the primary saccade fell short of the desired amplitude of eye movement. The EOG record for each saccade was plotted, overlaid by the idealised saccade as derived by the computer. Those saccades in which the computer algorithm had failed were excluded from further analysis. Average values for saccade velocity, latency and primary saccade deficiency were computed for the 5°, 10°, 15° and 20° deflections, left deflections and right deflections being considered together.

Subjective and Objective Measures of Psychological and Physiological Function

The following psycho-physiological performance tests, selected on the basis of their likely sensitivity to the effect of the drug on the CNS, were carried out between 90 minutes and 2 hours after taking each drug.

Subjective Estimation of Alertness. The subject was asked to make a mark on a line 100 mm in length at a point to the left of centre if he felt more drowsy than normal and to the right if more alert.

Ocular Accommodation. Near point was measured for each eye separately, using the RAF Near Point Rule. This consisted of a card bearing letters in N5 type mounted on a rule. One end of the rule was positioned under the subject's eye and the card was moved slowly towards the subject until he reported that the print appeared blurred. The distance of the card from the eye was read from the scale. The near point for each eye was noted, and the mean of the two measurements calculated.

Missing Digit Test. The task utilised an Apple II computer which presented sequentially upon the screen a randomly ordered series of nine of the ten digits 0-9, at a rate of one per second, followed by an audible tone. The subject was required to identify the missing digit in the series, and to respond by depressing the appropriate key on a numerical key pad. The percentage of correct responses in twenty trials was computed, as well as the average response time for the correct responses.

Critical Flicker Frequency. The flicker fusion frequency was measured using a portable Flicker Fusion Test Meter (Gaedev Ltd). It consisted of a closed metal tube 20 cm long, having an eyepiece at one end and an orange light emitting diode at the other. The subject was instructed to look with his right eye at the slowly flickering light. The rate of flicker was gradually increased until the subject reported that the light no longer appeared to flicker. The rate of flicker was increased beyond this point, and then slowly decreased until the subject reported that he could discern flickering. This sequence was repeated five times, thus obtaining ten estimations of the flicker fusion frequency. The mean and standard deviation of these readings were calculated and noted.

The Digit Symbol Substitution Test. The subject was given a sheet of paper on which were 200 digits arranged in 10 rows. At the top of the page was a key which assigned a symbol to each digit. The subject was required to use the key in order to write the appropriate symbol below as many digits as he could in two minutes. The subject's score was the average number of correct substitutions in two such trials.

In order to minimise learning effects during the test runs, each subject was given eleven practice sessions in which he carried out the various performance tests. The final practice session included, in addition to the performance tests, measurement of saccade velocity and exposure to the cross-coupled stimulus test profile.

RESULTS

Prophylaxis of Motion Sickness

Statistical analysis was based on the time taken for the subjects to reach a symptom score of 7 on every trial, analysis was based on the time taken to reach the highest symptom score that was achieved on all 4 runs. Following this procedure, a score of 7 was used for 11 subjects, 5 for 1 subject, and 4 and 3 for 2 subjects each. An analysis of variance was used to test for difference between drug treatments. A breakdown of the order effect revealed that it was linear and represented an improvement in time of 2.0 minutes from the first to the fourth run.

The treatment means of individual subject times corrected for order were compared using the Newman-Keuls shrinking range test. This indicated that hyoscine was more effective than the other treatments in delaying the symptoms of motion sickness ($p<.01$) and that both ginger and cinnarizine were better than placebo ($.01<p<.05$). However there was a difficulty in accepting this analysis. The correction for order did not appear to have adequately represented the order effect on all subjects. Indeed it might be unreasonable to expect a consistent order effect when the scores were subjective and differed from subject to subject. Accordingly, analysis of covariance was used to correct for a differential linear (by subject) order effect. The results are summarized in Table 1. This analysis showed that hyoscine was still better than the other three treatments ($p<.01$), that cinnarizine was better than both the other two ($.01<p<.05$), but there was now no significant difference between ginger and placebo. Thus, the correction for differential order, had completely removed the significant difference between ginger and placebo in the original analysis.

TABLE 1

	PLACEBO	GINGER 1G	CINNARIZINE 15 mg	HYOSCINE 0.6 mg
Time to final symptom rating (mins) - corrected for individual order effect	11.09	10.96	12.41*	14.32**
Saccade velocity (deg/s)	303.5	299.6	298.9	284.8*
Subjective alertness (0-10)				
- before motion	5.31	4.99	4.98	4.51*
- after motion	4.21	4.37	4.57	4.09
Near point (cm)	16.34	17.31	16.75	17.81
Missing digit task				
- % error	23.75	25.31	24.69	24.38
- Response time (s)	1.35	1.35	1.32	1.32
Critical flicker frequency (Hz)	34.24	34.65	34.00	33.89
Digit symbol substitution	87.03	86.63	87.31	86.44

* $P<0.05$ ** $P<0.01$

This analysis appeared more reasonable than the first: the effects of hyoscine and cinnarizine were unchanged, while the effect of ginger, which relied to some extent on two subjects, both of whom had ginger on their final trial and placebo on their first or second, was lost. Unfortunately, three other subjects who performed well with ginger in spite of taking it on an early trial were found by this analysis to be associated with an order effect that represented a deterioration in performance with time. It is slightly unsatisfactory that an effect should be lost so completely in this way, but it reflects the non-orthogonality of differential order with the treatments. Even on the original analysis, however, only 11 of the 16 subjects performed better with ginger than with placebo, this figure reducing to 8 on the second analysis. This compares with 13 for cinnarizine on both analyses and all 16 for hyoscine, also on both analyses.

The conclusion, therefore, is that hyoscine had a beneficial effect in increasing subjects' tolerance to motion sickness induced by cross-coupled stimulation, and cinnarizine a smaller beneficial effect, whereas ginger was not distinguishable from the placebo.

Subjective and Objective Measures of Performance

Ocular Saccades. The data analysed were mean saccadic velocity, mean latency and mean saccade deficiency for four different angles of eye movement. A multivariate analysis of variance was applied to each measure, the four angles being treated as separate variables.

In the analysis of saccadic velocity the values obtained for 5 deg saccades were omitted as they were significantly more variable than those at the other three angles. The results for the remaining three angles were meaned. Analysis of variance revealed a small order effect, but there was no evidence of a differential order effect of the type observed in the motion sickness analysis. The larger treatment effect was entirely explained by the low mean velocity with hyoscine which was less than both placebo ($p<.01$) and the other two treatments (both $.01<p<.05$). All comparisons between means were made, as before, using the Newman-Keuls procedure.

No drug effects on saccade latency were observed, nor did the multivariate analysis indicate any effects on saccade deficiency, though a large value of saccade deficiency was found for 10 degree saccades following hyoscine. Thus the suggestion that hyoscine might be having an effect here cannot be rigorously demonstrated.

Subjective Estimate of Alertness. Mean scores for the four treatment conditions both before and after exposure to the provocative motion stimulus are presented in Table 1, where the scores have been adjusted to remove small order effects. Hyoscine was shown to have a marginally significant ($p=0.05$) effect in inducing drowsiness or lowering alertness compared with placebo. No effect was detected with the other preparations. In addition, the subjective measures exhibited a significant shift towards drowsiness following the test ($p<0.05$), but as this was equally evident in all experimental conditions, this reflects the increase in drowsiness induced by the test procedure.

Other Tests. Statistical analysis showed no significant difference between treatments in the results of near point estimation, missing digit task, critical flicker frequency or the digit symbol substitution test.

DISCUSSION

The principal finding from this experiment was the absence of any significant therapeutic or other effect of powdered ginger root. This is in contrast to the study of Mowrey and Clayson (3) who demonstrated prophylatic benefit from powdered ginger root in excess of both dimenhydrinate and placebo.

There are several differences in experimental method between the two studies that may account for the disparate findings. In the study by Mowrey and Clayson subjects were selected on the basis of a questionnaire for their high susceptibility to motion sickness. Each subject was tested once only at 20-25 minutes after taking one or other of the drugs. Subjects were not forewarned that the test would induce motion sickness and were asked to report only those symptoms referrable to the stomach using an arbitrary numerical rating. The intensity of the nauseogenic stimulus, delivered by means of a chair rotating about an off-vertical axis, was evidently fairly high since most subjects tolerated the stimulus for less than 6 minutes.

It is likely that the 20-25 minute interval between the administration of a drug and exposure to provocative motion was too short for dimenhydrinate to have reached its full therapeutic level in the body (8) and may account for the small degree of benefit derived from this drug. Conversely, it could be argued that in testing subjects at two hours after drug ingestion, as was done in the present trial, any therapeutic effects of ginger, which probably exerts its effect locally on the stomach, may already have waned. Some difference in result may be a consequence of not selecting motion sickness susceptible subjects for the present trial. However, the results show no evidence that those eight subjects whose tolerance showed an increase following ginger were more or less susceptible to motion sickness as indicated by their score on the Reason motion sickness questionnaire. Likewise, Hargreaves (5) found a similar subjective benefit from cinnarizine among those with a history of seasickness as compared with the total study group. The wide range of motion sickness susceptibility of the subjects influenced the choice of a continuously incrementing turntable speed in the stimulus used to induce motion sickness. By use of this profile susceptible subjects did not become unwell before making a reasonable number of head movements while relatively resistant subjects eventually reached a stimulus level at which they also were rendered motion sick. Diversity of motion sickness susceptibility and limitations on the number of potential experimental subjects made it preferable to test each subject repeatedly under all four drug conditions. This allowed within-subject statistical comparisons to be made. Even so, valid conclusions were only reached by elimination of order effects which varied from subject to subject.

While subjectively assessed drowsiness was detected following hyoscine, the objective measures of performance used in the trial yielded no significant differences between treatments. Loss of ocular accommodation following hyoscine has been reported as a prominent side effect in some subjects who received hyoscine for prolonged periods either orally (9) or by means of a transdermal patch (10). Hyoscine has also been shown to impair short term memory of items in a vocally presented list (11). Some performance decrement might therefore have been expected in the execution of the missing digit task, in which numbers were presented visually. The digit symbol substitution test has been used as a performance test to detect the central nervous system effects of several benzodiazepine drugs (12). The test is akin to writing in an unfamiliar script. For the test to give useful results a large number of preliminary practice sessions are necessary to reduce learning effects, so it may be that the eleven practice sessions employed in the present study were insufficient to yield a test of optimal sensitivity.

The effect of drugs on saccadic eye movements has been studied in relation to alcohol, marijuana, methadone and the benzodiazepines (13,14,15,16). Saccade latency - the time taken to initiate a saccade following a visual stimulus - is a measure of the sensory component of the saccadic response. Saccade velocity on the other hand is an attribute of the motor component of the response. Certain drugs affect predominantly one or other component (17). The reduction of saccadic velocity found in the present study following hyoscine administration has not previously been described. This effect of hyoscine is similar to that produced by the benzodiazepine group of drugs and by alcohol. Reduction in saccade velocity is probably an indication that these drugs are exerting a depressant effect on an area of the pontine reticular formation responsible for generating the saccadic pulse (18,19).

CONCLUSIONS

The trial has confirmed the efficacy of hyoscine in the prophylaxis of motion sickness but has not shown any beneficial effect following the administration of powdered ginger root. Cinnarizine has also been shown to be effective at a relatively low dose level. The prophylaxis provided by 15 mg cinnarizine was somewhat less than that following 0.6 mg hyoscine.

While there was some evidence, both subjective and objective, of the CNS depressant effect of hyoscine, no such effects were found following cinnarizine. Due care however is necessary in extrapolating this negative finding to higher dose levels of cinnarizine or to situations in which a high subject performance level is vital.

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DISCUSSION

KENNEDY: Was your digit symbol test scored in terms of percent correct as opposed to number correct?

STOTT: No, it was scored on the basis of the number of substitutions that they made excluding those substitutions that were incorrectly made but, in fact, I think I'm right in saying that almost nobody made an incorrect substitution and really it was a measure, if you like, of hand-eye coordination.

ENSCHKE: I would just like to make a comment about the observation which you reported where the subjects appeared to become more sensitive to the motion with each successive test. I was wondering whether or not you noticed anything in their demeanor which would suggest, in fact that increased sensitivity was really a reflection of their learning that they really didn't like the motion test and perhaps were more eager to end it. I think that's something we all have to be very careful about in doing studies like this.

STOTT: I think your interpretation is exactly correct. I've nothing very much more to add, except that because we used a balanced design this fact did not matter.

THE CURRENT STATUS OF THE RAF PROGRAMME OF DESENSITISATION FOR MOTION SICK AIRCREW

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SUMMARY

The RAF Motion Sickness Desensitisation Programme has been in operation since 1966 and has to date treated 151 aircrew.

The programme consists of a ground phase and a flying phase. Since January 1981 the programme has been located entirely at Farnborough. Additional motion stimuli have been incorporated into the ground phase of treatment and the flying phase is now carried out in a high performance aircraft, the Hunter T7.

Comparison of the results of follow up for the period 1981-83 with those for 1974-80 indicates an improvement in overall success rate and shows a significant increase in the number that progress to fly in the demanding motion environment of fast jets.

INTRODUCTION

Airsickness is a common problem in early flying training. Rubin (1), in a survey of airsickness in US trainee aircrew in 1942 quoted an incidence of 11%. He also noted that, while the general failure rate in training was 36%, the failure rate among those who developed airsickness was 52%. In a similar survey among UK trainee pilots in 1974, Dobie (2) classified airsickness according to its effect on performance. He found that 38.7% of aircrew suffered from airsickness at some stage in training and in 14.6% it was sufficiently severe to impair the student's performance and his ability to absorb instruction.

The pattern of airsickness during the course of pilot training is characteristic. The incidence of symptoms on early sorties may be 20-30% despite relatively unprovocative manoeuvres. This incidence falls as adaptation occurs to the motion stimuli of flight, but shows transient increases when more provocative manoeuvres such as spinning and aerobatics are introduced (3). Motion sickness also affects trainee navigators and tends to make its appearance at the start of low level navigation training. A further group of aircrew in whom motion sickness is a problem are engineers and electronics operators in maritime reconnaissance aircraft in which long periods of flying are carried out at low level.

In all these groups the normal pattern is of gradual spontaneous desensitisation. In some motion sick individuals a useful reduction in incidence may be gained by the use of prophylactic drugs during the early period of training. Hyoscine 0.3 - 0.6 mg is the most commonly used drug for this purpose, though cinnarizine has been found useful for long duration flights. In pilots the use of such drugs is, of course, prohibited for solo sorties.

In a small proportion of subjects, either on account of a high susceptibility or a slow adaptive response, motion sickness persists, erodes confidence and impairs ability. This is reflected in poor performance in training and an increased likelihood of suspension. The recognition of a student's continuing problem with motion sickness can only occur after a reasonable period has been allowed for spontaneous adaptation, by which time a large financial investment has been incurred. Furthermore, susceptibility to motion sickness is no indicator of a student's inherent ability at the airborne task; motion sick aircrew once treated can go on to become not merely useful but outstanding pilots or navigators.

The RAF programme of desensitisation treatment for chronic airsickness was started by Dobie in 1966 (2), and has to date treated 151 subjects. Over the 10 year period 1974-1983, 57% of those treated have been pilots, 27% navigators and 14% aircrew from maritime reconnaissance aircraft. This distribution is probably not a true indicator of motion sickness incidence within these various categories but reflects also the degree to which a continuing susceptibility to motion sickness is compatible with the airborne task. Though primarily a problem affecting aircrew in training, there is sufficient anecdotal evidence to suggest that motion sickness represents a continuing problem for some aircrew. How many continue to experience symptoms and to what degree is not yet known.

In January 1981 a number of changes were made to the desensitisation programme. Responsibility for the programme was transferred entirely to the RAF Institute of Aviation Medicine (IAM) at Farnborough where the initial assessment and the ground phase of treatment are carried out and the flying phase is conducted by the Medical Officer Pilot using the Institute's Hunter T7 aircraft.

This paper outlines the current practice in the desensitisation programme, and compares the results of follow up in those treated between 1981 and 1983 with those who underwent desensitisation treatment between 1974 and 1980.

DESENSITISATION PROCEDURE

Initial Assessment

There are no absolute pre-conditions to entry to the desensitisation course, but advice from the student's flying training school is taken on his prospects of success once he is free of motion sickness. Also, we would wish to exclude those who, allowing for the dispiriting effects of motion sickness, think they have made a mistake in their choice of a flying career.

Initial assessment at the IAM is made over a three day period. A detailed history of motion sickness is taken and inquiry made for relevant psychological factors. Routine vestibular function tests are carried out to exclude any unexpected abnormality. Assessment is made of the subject's susceptibility to motion sickness using three different provocative stimuli - cross-coupled stimulus, 0.3 Hz $\pm$ 0.25 G linear G_z oscillation, and 0.02 Hz, $\pm 150^{o}$/sec angular oscillation accompanied by a visual search task (4). The subject's likely rate of adaptation is assessed using a cross-coupled stimulus of gradual onset. This test is repeated on three successive days at the same time of day using identical stimulus profiles.

Ground Based Desensitisation

The same types of motion sickness-inducing stimuli, cross-coupled, sinusoidal linear G_z oscillation, and 0.02 Hz angular oscillation are used during the ground phase of treatment.

Cross-coupled stimuli have formed the basis of ground based desensitisation since the start of the programme in 1966. The subject, seated in an enclosed cab over the axis of the spin table, makes head movements in pitch and roll while rotating in yaw. The speed of rotation is increased in 1 rpm steps, and the subject makes five to twenty head movement sequences at each speed of rotation (each sequence involves 8 head movements at 3 sec intervals). The cab is either illuminated or in darkness, alternating every 5 sequences. Every 30 secs, after each sequence, the subject gives his well-being rating (WBR) on a scale from 1-6, defined as follows:

Rating	Symptoms
1	No symptoms. 100% well
2	Malaise. Mild symptoms. No nausea.
3	Mild nausea $\pm$ other symptoms.
4	Moderate nausea + other symptoms.
5	Severe nausea and stomach awareness.
6	Vomiting.

Because of the tendency for symptoms of motion sickness to cascade, the session is ended as soon as the subject reports a WBR of 4.

For each session of cross-coupled stimulation an estimate of the stimulus dose is obtained by adding the products of rotational speed in revolutions per minute and the number of head movement sequences made at that speed. Because sessions are generally terminated at the same degree of subject malaise, graphs of this derived figure plotted over the course of treatment give an indication of progress.

Since mid 1981 the two metre stroke vertical vibrating platform, operating at 0.3 Hz and ± 0.25 G_z and 0.4 Hz, ± 0.4 G_z, has been used as a motion sickness provoking stimulus. The stimulus is intensified by excluding external earth fixed visual reference and by making the subject carry out a visual search task, a modified version of that described by Moore et al (4). The subject looks at a 12 x 12 array of numbers, the rows of which are referenced by the randomly ordered digits 1-12, and the columns by letters A-L also in random order. Given a letter and a number, he has to return the referenced number. Two such arrays are used, one just above the subject's line of sight, the other on his knee. The subject uses each array alternately and has to make head movements in doing so.

Over the three week period of the ground phase subjects have twice daily sessions, up to a third of which may be carried out on the vertical vibrator if assessment has shown them to be sensitive to this stimulus. In addition, one session per week is carried out on the turntable, oscillating in yaw at 0.02 Hz $\pm 150^{o}$/sec, during which the subject carries out the same visual search task but without head movements, reading from a single array of numbers fixed to the turntable.

Not all aspects of aircraft motion can be reproduced on the ground and this phase of treatment is regarded only as an essential precursor to a period of graded remedial flying.

In the treatment of aircrew from maritime reconnaissance aircraft a flying phase of treatment appropriate to that type of aircraft is not available. For this group the ground phase of treatment is extended to four weeks and contains additional sessions on the vertical oscillator.

Airborne Phase of Desensitisation

The airborne phase of desensitisation follows the ground phase without a break and consists of 10-15 hours flown in the Hunter T7 with the Medical Officer Pilot who is also a qualified flying instructor. The flight envelope of the Hunter is extensive and allows a graded build-up of motion stimulus from physiologically undemanding straight and level flight through to advanced aerobatics and high speed low level navigation.

The syllabus is divided into initial and advanced phases and is adapted for each individual from a number of specific exercises. For both pilots and navigators the initial phase is similar, but their advanced phases differ in emphasis. Progress through the course is entirely dependent on the subject's rate of adaptation, and he is under no pressure to achieve any particular objective during each sortie.

Initial phase

(i) Familiarisation

(ii) Straight and level flight at various speeds

(iii) Effects of controls

These early sorties contain no extremes of attitude or acceleration and teach the subject the differences between the basic handling of the Hunter and his training aeroplane.

(iv) Low speed handling

(v) Flying in manual control

(vi) Turns at 30°, 45° and 60° angle of bank

(vii) Circuits

Circuit flying in the Hunter is demanding with a high workload, and the rapidly changing angles of bank and linear accelerations are provocative stimuli. They are introduced at the end of other exercise sorties as the individual's adaptation increases.

(viii) Introduction to instrument flying

One of the advantages of the Hunter in this role is the ability to operate above the weather, which frequently entails an instrument departure and recovery. Instrument flying is introduced on the course as and when necessary, taking due account of the provocative effect of an absent visual horizon and of turbulence often encountered in low level cloud.

The initial phase normally takes about 5 flying hours during which the subject often retains some sensitivity to airborne provocative motion. Once the subject can tolerate 60° angle of bank, level turns, and circuit flying with no ill-effect he moves on to the advanced phase.

Advanced Phase (Pilots)

During the advanced phase the subject learns to fly the aircraft to its limits and gains increasing confidence as he learns to enjoy flying again.

(i) Maximum rate level turns

Medium speed entries to maximum rate level turns, stabilised turns and reversals maintaining the light buffet are practised. As well as extending his desensitisation the subject learns to handle the Hunter in the light buffet which is necessary to fly good aerobatic manoeuvres.

(ii) Aerobatics

Aerobatics are introduced gradually and are built up from the maximum rate level turn and the basic barrel roll. Throughout a barrel roll the subject maintains a visual reference on the horizon thus assisting his orientation. Only when he is confident and tolerant of these manoeuvres is the loop introduced during which he loses sight of the horizon for about one third of the manoeuvre. Once the subject has mastered the loop, he progresses very rapidly through the repertoire of roll off the top, horizontal and cuban eights, Derry turn, wing-over, slow roll, vertical roll and hesitation manoeuvres. By the end of the course he should be capable of flying a 20 minute sequence linking basic and advanced manoeuvres.

(iii) Practice diversion and forced landing procedures

(iv) Low level navigation (420 knots)

High speed, low level navigation exposes the pilot to turbulence and to large acceleration (all turns being done at 60° angle of bank ± 20). It is an effective means of increasing his airborne mental capacity in a demanding environment. The first introduction is done over the gentle undulating terrain of Southern England at 500 ft agl, but then he progresses to flying at 250 ft agl in the mountainous terrain of Wales.

(v) Formation flying and tailchasing

(vi) Pairs low level attack

This is an extension of basic low level navigation to demonstrate the operational and tactical environment. It provides much provocative stimulus involving rapid head movements to maintain the good lookout essential for tactical integrity and aggressive flying.

(vii) Landaway navigation exercise

This provides the final confidence boost of his rehabilitation when the subject flies the Hunter to his home base and is able to restore self-esteem by showing off to his colleagues. The importance of this should not be underestimated.

Navigators

Navigators present a slightly different problem. It is well known that motion sickness afflicts the passengers far more than the driver of a vehicle (5). The navigator spends a lot of time with his head down in the cockpit and at low level he is subjected to turbulence, high G, and large accelerations without the benefit of being able to anticipate the aircraft behaviour. During the initial phase the navigator also learns the basic handling of the aircraft so that he gains the benefit of anticipating aircraft motion. Cockpit management and map reading tasks are introduced later as the subject's adaptation develops.

The advanced phase for navigators is necessarily biased towards low level navigation and attack profiles and ends with 1 v 1 air combat manoeuvres.

(i) Introduction to low level navigation at 420 knots

(ii) IP to target runs

(iii) Simulated attack profile

(iv) Low - high navigation

(v) Pairs low level attack

(vi) Procedural diversion and instrument approach procedures

(vii) Landaway navigation exercise

(viii) Air combat manoeuvres, 1 v 1

Assessment of Progress

During the rehabilitation flying, the subject uses the same well being rating as he has used during the ground desensitisation.

Because of the individual variability between subjects and the absence of any common provocative motion it is difficult to quantify the stimulus in absolute terms. However, each sortie is assessed retrospectively and given a Provocation Index, also on a 1-6 scale, according to the following scheme.

Provocation Index	Example of manoeuvre
1	Straight and level; up to 45° angle of bank turns
2	Up to 60° AOB turns; circuits, radar approach
3	Practice forced landing; introduction to aeros; advanced turns
4	Basic aerobatics; max rate level turns
5	Advanced aerobatics; low flying
6	Aerobatic sequence; attack profile

The subject's well being rating and the Provocation Index for each sortie are plotted graphically and provide an indication of the progress being made by the subject and the degree of flying continuity he has received.

RESULTS

Some small degree of selection of subjects for the course may take place before referral by the training units, but poor progress in training is an almost inevitable accompaniment of continuing motion sickness and due account is taken of this. During the past three years no cases have been refused admission to the desensitisation programme following assessment. One subject finally decided to withdraw from training during the assessment period, the principal reason for which was not related to motion sickness.

Although there is some correlation between the susceptibility to motion sickness induced by the three ground based stimuli some individuals show wide differences in susceptibility. Figure 1 shows the results of treatment with cross-coupled and linear G_z stimuli in one subject who showed a high sensitivity to cross-coupled stimuli and adapted slowly but who proved relatively insensitive to the linear G_z stimulus. By contrast, the subject illustrated in Figure 2 desensitised rapidly to cross-coupled stimulation.

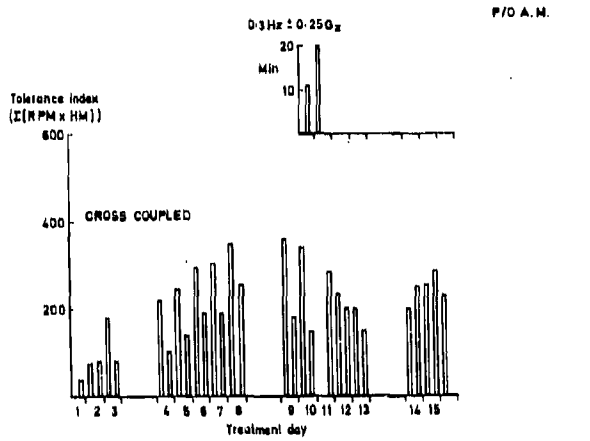

Figure 1. Record of ground-based desensitisation of subject showing high sensitivity and slow adaptation to cross-coupled stimulation but relatively low sensitivity to the linear G_z stimulus.

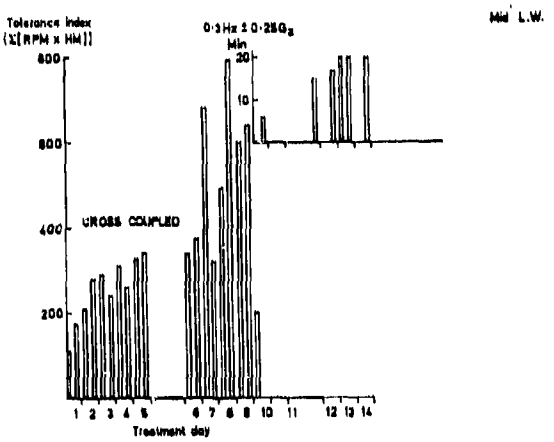

Figure 2. Record of ground-based desensitisation of subject who, despite good adaptation to cross-coupled stimulation, showed no consequent adaptation to the linear G_z stimulus.

The linear G_z stimulus is not introduced until at least the second week of the ground phase of treatment when subjects have shown some increase in tolerance to the cross-coupled stimulus. No increase in tolerance to the linear G_z stimulus above the baseline level established at initial assessment has been observed as a result of this period of cross-coupled treatment. The two stimuli appear to be independent of each other in this respect. Preliminary observations, however, on the results of once weekly exposure to low frequency angular oscillation accompanied by visual search suggests that with this stimulus some increase in tolerance is transferred from that acquired on exposure to cross-coupled stimulation.

Progress during the ground phase of treatment is considered satisfactory if subjects are able to make 20 head movement sequences while rotating at 10 rpm, and can survive 20 minutes of 0.3 and 0.4 Hz G_z oscillation without developing more than mild symptoms of motion sickness. Occasionally the period of ground based treatment is extended if these goals are not achieved, but the rate of progress and degree of acquired tolerance during this phase of treatment are poor indicators of ultimate success in overcoming airsickness.

Progress during the flying phase varies between individuals. Typically there is gradual increase in tolerance as the motion sickness provoking content of successive sorties is increased (Figure 3). Occasionally improvement in tolerance appears to be more abrupt (Figure 4). Rarely, there is no clear indication of an adaptive response during the flying phase (Figure 5).

On completion of the desensitisation course, subjects resume flying training no matter what the apparent outcome of treatment. Even at this stage the prediction of success is not easy. In fact, all those subjects whose records are shown in Figs 1-5 currently continue to fly, the most recent of them having been treated 18 months ago.

In analysing the results of follow-up surveys for the periods 1974-1980 and 1981-1983 subjects have been assigned to five categories detailed below.

Classification of effect of therapy

a.	Successfully desensitised.	Completes flying training.	Progresses to fast jets.
b.	Successfully desensitised.	Continues flying training.	Progresses to multi-engined aircraft or helicopters on completion.
c.	Successfully desensitised.	Fails to complete flying training for reasons other than motion sickness.	
d.	Motion sickness recurs.	Completes flying training.	Problem with motion sickness resolved when re-roled to different aircraft type.
e.	Motion sickness recurs.	Fails to complete flying training on account of motion sickness.	

This scheme reflects the fact that success or failure is not necessarily absolute and that the degree of success will only emerge over the course of subsequent training. A student pilot or navigator may successfully overcome his motion sickness only to fail later in training for lack of ability. Alternatively the treatment may not achieve full desensitisation and the problem may only be resolved when the subject is re-roled to a less provocative type of flying.

Results for the seven year period 1974-80 are compared with those for the period 1981-83 in Table 1.

Table 1

Category	1974-1980	1981-1983
a	6 (13%)	10 (31%) *
b	25 (54%)	13 (41%)
c	1 (2%)	4 (12.5%)
d	7 (15%)	2 (6%)
e	7 (15%)	3 (9%)
Total	46	32

* $p < 0.05$

If those in categories a, b and c are to be regarded as therapeutic successes, then the success rates are 84% for the period 1981-83, compared with 70% for the period 1973-80. The results show a significant increase ($p < 0.05$) in the proportion of aircrew that progress to fast jet flying (category a). Of the seven pilots who, since 1981, have reached category a, three have been prizewinners either for aerobatics or for low level navigation at graduation from advanced flying training. Another recent prizewinner during advanced flying training, albeit in helicopters, is classed in category d - a treatment failure. He nonetheless acquired sufficient benefit from the desensitisation course to complete basic flying training and later to excel in the less provocative environment of helicopters.

Results of desensitisation treatment in those aircrew from maritime reconnaissance aircraft who received only the ground based phase of desensitisation indicate that all have experienced benefit from treatment and the 5 subjects in this group seen since 1981 all continue to fly. Follow-up data for the equivalent group treated between 1974-80 are incomplete but 2 out of 7 treated in this period are known to have failed in training on account of motion sickness.

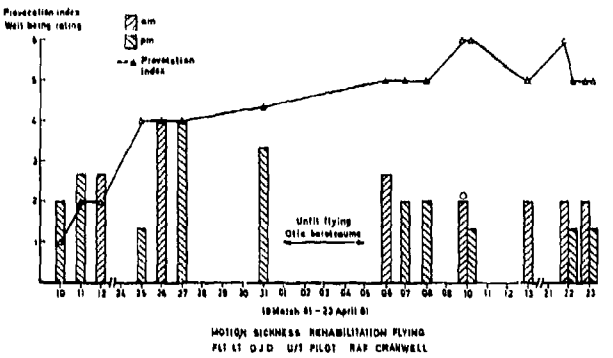

Figure 3. Record of airborne phase of desensitisation in a typical subject. Despite an increase in provocative content of the sortie as the course proceeds, there is a steady improvement in tolerance from the fifth sortie onwards.

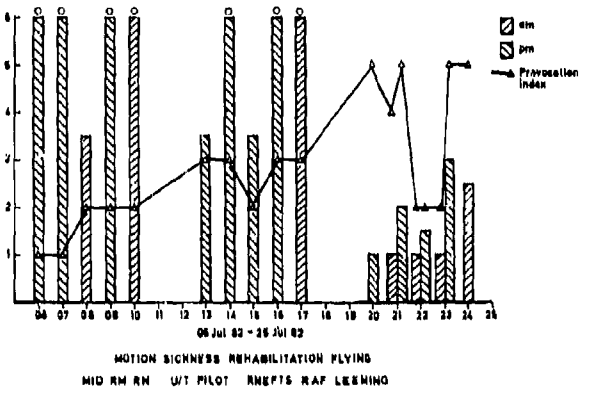

Figure 4. Record of airborne phase in a subject who showed an abrupt increase in tolerance after 10 sorties.

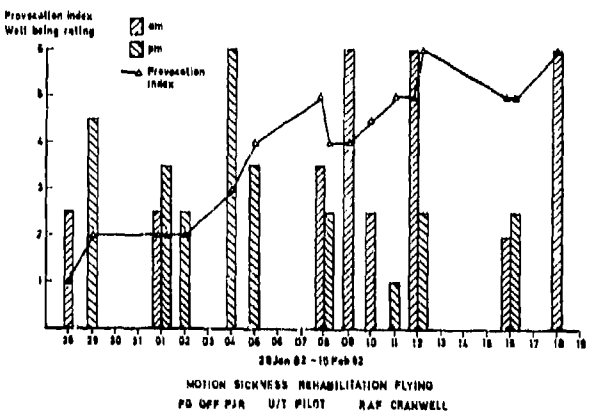

Figure 5. Record of airborne phase in a subject who failed to adapt during the course and was subsequently re-roled to helicopters.

DISCUSSION

While there may be a number of factors that contribute to the problem of motion sickness in any one individual, the approach to treatment is fundamentally a physiological one. The ground phase of treatment is designed to reproduce on the ground features of the neural mismatch that are generated in the airborne environment and allow the subject to be exposed to them in a controlled and gradually incrementing manner.

The desensitisation that results from repeated exposure to a provocative stimulus tends to be fairly specific to that type of stimulus. We have found that tolerance acquired to the cross-coupled stimulus does not result in increased tolerance to a linear G_z stimulus. It might similarly be argued that a cross-coupled stimulus is not representative of the motion stimulus of an aircraft. However at the level of canal-otolith mismatch, there are sufficient similarities to expect some transfer of tolerance to occur. Many aerobatic manoeuvres, while not producing cross-coupling, generate a rotational signal from the semicircular canals which is not accompanied by the degree of gravitational vector rotation that terrestrial motion rules would dictate. An equivalent neural mismatch is produced by the cross-coupled stimulus.

There is perhaps a more direct similarity between aircraft motion in low level turbulence and the vertical oscillation at 0.3 and 0.4 Hz used in the ground phase of treatment. Laboratory studies have shown the incidence of motion sickness to be inversely related to frequency of G_z oscillation in the frequency band 0.2 to 0.6 Hz (6). Because the stroke of the vertical oscillator is limited to 2m, the maximum nauseogenic capability of the machine is achieved at about 0.4 Hz.

The nauseogenic stimulus of 0.02 Hz angular oscillation when accompanied by a visual search task has not hitherto been used intensively during the ground based phase. Motion sickness due to this stimulus results from a visual-vestibular mismatch. There is some indication that tolerance to this stimulus is increased by the cross-coupled stimulus in which, when the cab is illuminated, there is also generated a visual-vestibular mismatch.

Despite the inclusion in the ground phase of additional motion stimuli designed to reproduce the nauseogenic features of aircraft motion, the value of a flying phase of treatment remains paramount. There are inevitably aspects of aircraft motion that cannot readily be reproduced on the ground and subjects need to acquire the confidence that they are resistant to sickness in the air, rather than in a set of laboratory machines. Indeed, subjects do not find themselves to be totally immune to airsickness at the start of their flying phase and it is still necessary to build up tolerance gradually.

In the earlier years of desensitisation treatment it was considered important that "no attempt should be made ... to carry out types of manoeuvre beyond the scope of the normal training syllabus" (2). With the use of the Hunter T7 for rehabilitation flying this principle has been abandoned. The student progresses as far as he is able, both in terms of the provocative aircraft manoeuvres that he is able to tolerate and secondarily in extending his confidence and mental capacity to meet the demands of flying a high performance aircraft. The increase in the proportion of those treated who progress to fast jet aircraft can be ascribed in large measure to the use of the Hunter aircraft. Having successfully adapted to this aircraft the student does not need to confine his aspirations, nor the expectations of his instructor, to a flying career in the less provocative environment of helicopters or transport aircraft.

One advantage that has resulted from locating the flying phase at Farnborough is that the student is removed from the pressures of the training environment and this allows therapy to be conducted in a relaxed manner. Though the Medical Officer Pilot is also a qualified flying instructor the aim is to establish a therapist/patient relationship in place of the instructor/student relationship of flying training.

The extent to which adaptation is lost during periods off flying is not fully known. Some aircrew report a recurrence of motion sickness symptoms on resumption of flying after a long break, but tolerance appears to be regained more rapidly and a pattern of chronic motion sickness does not recur. Over the past three years, 7 aircrew have returned for a one week period of 'top-up' ground based treatment, most at their own request, prior to resumption of flying after a break of a few months. It is frequently observed at the start of 'top-up' treatment that no loss of tolerance to cross-coupled stimuli has occurred since the end of the initial ground phase of treatment, despite having had no similar treatment in the intervening period. In addition, this group has reported no problems with airsickness when they resumed flying, a finding which cannot wholly be ascribed to the additional period of ground based treatment. Thus adaptation to provocative motion may be retained for longer periods than has previously been thought.

CONCLUSIONS

Several changes were made in January 1981 to the RAF Motion Sickness Desensitisation Programme, notably the addition of a 0.3 Hz linear G_z provocative stimulus to the ground based treatment, and the use of a high performance aircraft, the Hunter T7, for the airborne phase of desensitisation. In addition, the flying phase of treatment was moved away from the training environment and the whole programme based at Farnborough.

The results of follow-up comparing the periods 1981-83 and 1974-80 suggest that there has been an improvement in overall success rate and show a significant increase in the proportion progressing to fast jet flying.

It is suggested that the improvement in results is a consequence of changes made in the programme in 1981.

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DISCUSSION

The discussion of this paper follows paper 42

PSYCHOLOGICAL COMPONENTS IN THE DEVELOPMENT AND PREVENTION OF AIR SICKNESS

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Summary

Extensive behavior analysis is made on the basis of 23 case studies of motion sick aircrew members. Situational context variables and their interaction with individual dispositions, behavior regulation patterns, coping mechanisms and self control techniques are discussed. It seems that sensitivity to motion ist not the only criterion in the development of motion sickness. Description of countermeasures and their effects are presented. Daily exercise of various techniques of behavior regulation to build up successive adaptation to motion stress will enable persons to cope with motion sickness producing situations inflight.

Introduction

First, training and therapy measures would normally begin when sufficient diagnostic explanations ans precise indications are present. From the standpoint of the training institution for aircrew of the German Air Force ist is enough when:

1. There is an appearance of manaifested air sickness (AS) in three flights.
2. There is an exclusion of organic or toxic induced changes, respectively, the dysfunction of the equilibrium sensory system.
3. There is at least average aptitude for flying.
4. There is a strong flying motivation.

Then this member is recommended for an anti-airsickness training program (AATP). This AATP has become institutionalized in the selection process of flight applicants.

Since this criteria has been put to use, the success quota has improved drastically to 87% in 1980-83, compared to 57% between 1974-1979. Since the training methods have basically stayed the same, from what we see, the difference can only be interpreted as being a result of the efficiency of the selection criteria. In spite of this relatively high success rate, we are not pleased with our present understanding of this problem; especially, the diagnostic explanation of the type of AS and the determining factors. An extensive training program lasting four to five weeks would not be necessary when a reliable prognosis ans preventive measures are present. Through past cases it has been found that without the vestibular organs there would be no AS. The organ itself, with its specific functions, does not sufficiently explain the development of AS. Persons who are susceptible to strong vestibular stimuli and/or sensory conflict fo not get airsick in every case. Nor do those of lesser susceptibility, who become unexpectedly sick and interrupt and/or end their flight training. Most probably other psycho-physiological processes and functional systems play an important role. Kamiya (1982) points out that there must be a wider spectrum of the central-nervous system functions, while the vestibular system alone can not produce such widely ranging effects. It is possible that there are psychological processes with the psychological correlates, which constitute an adequate frame of reference for the explanation of the development of AS. The roles of adaptation, and the learning processes are of relative importance (Kamiya, Cramer, Money, Gardner 1982); when hardly anybody has the experience. The relationship of the importance of these factors in the development of AS has not been addressed in current research. A theoretical analysis is necessary to structure the results from the various aspects of this area of research. There are least three sources of variance (Roesler 1983):

1. Behaviour characteristics (dimensions)
2. Biosignals (biomedical data)
3. Situations

We can expect that the biosignals wil indicate certain regulatory processes, which are directed with central regulatory systems. The regulatory systems are activated through different stressors of the organism, which is represented in different reaction patterns. The function of the regulatory system is dependant on specific individual characteristics. Observation shows specific individual regulatory systems. A multi-varied approach is important to find the variations, covariations and dissociations of signals. The objective should be to detect covariations between behavior dimensions and physiologically determindes regulatory systems. The only problem in doing this having a representative number of persons, situations and signals. In our case, we did not have the numbers, so we are doing this in 23 cases; from these, 11 were from the United States of America. The goal of this paper is to present ideas for documentation and a description of these regulatory processes. It is possible that a hypotheses could then be drawn from this approach. This approach comes from the results of the diagnostic phase before AATP, and also from information within the training program.

Procedure and Results

Our clients suffering from AS were examined to rule out any pathological factor prior to being referred to us. We found that 21 persons (91.3 %) were given medication, to no effect, before taking part in the program (see Figure 1)

	Student Pilots	Squadron Pilots	WSO	BTO	Flight Surg.	N	AATP Successful	AATP Unsuccessful
N	18	1	2	1	1	23	20	3
Highly Susceptible	11	1	-	-	1	13	12	1
Lesser Susceptible	2	-	2	1	-	10	8	2
Medication not helpful	16	1	2	1	1	21	19	2
no medication	2	-	-	-	-	2	1	1

Fig.: 1 Descriptive variables of 23 airsick cases (1980 - 1983)

The AATP consists of three phases:

Anti-Airsickness Procedure Phases
Phase 1 Background Assessment 1. Laboratory Tests of Motion Reactivity Assessment of psychophysiological indicators of motion sickness 2. Behavior Analysis Assessment of psychological indicators of motion sickness Education in Self-Control and Stress Coping Techniques 1. Education 2. Coping Training - Physical Fitness Training (including "space wheel" familiarization) - Relaxation Training (combination of gressive relaxation and autogenic training) - Mental Practice (according to the syllabus)
Phase 2 Adaptation, Desensitization and Self - Control Management 1. Rotating Chair 2. Visual-Vestibular Interaction Device 3. Spatial Disorientation Simulator 4. Exercise Unit ("Space Wheel") 5. Cognitive Self-Control of Inflight Stress Situations
Phase 3 Inflight Desensitization and Evaluation 1. Three to five flights with increasing motion stimulation and work load 2. Use of self-management technique for inflight symptom control

Background Assessment (Phase 1)

A collection of psychological data was performed according to the behavior formula of Kanfer and Phillips (1970): "Who was experiencing what, under which circumstances, with what effect, and what did the person do to overcome it."

This also includes the questions for personality dimensions, specific behavior characteristics and situations. These methods come from:

1. Biographic analysis
2. Exploration
3. Personality tests
4. Performance tests
5. Sate of being scales
6. Flight instructor questioning
7. Symptom specific tests

Participants in the training program show, through the wide variety of personality traits, an uncertian picture. More than half are highly susceptible to motion. They are as successful in the program as those of lower susceptibility (see Figure 1). All scores of state anxiety (Spielberger 1980) were very high in flights where AS occured, even though only 20% admit that anxiety could be a determining factor. The situations in which AS occur can be seen in the following:

1. Steep turns
2. Rapid Altitude changes and stalls
3. Workload pressure
4. Changes in g-load
5. Georgraphic and spatial disorientation
6. Turbulence
7. Flight termination
8. Other than inflight (i.e., walking to aircraft, flight gear issue)

Information about the development of AS ans its dynamic process is unprecise. We assume that responsibility lies in the inaccurate self-perception as dissimulation tendencies. The results of behavior analysis done in the training phase are much more perceptible. Through the training program, the willingness and ability for self-perception is advanced.

Education in Self Control and Stress Coping Techniques

1. Education

The first parts begins with a discussion of the individual test results. This should improve self exploration and remove dependancy of behavior controlling stimuli conditions. The role of the trainer is as a nondirective advisor who leads discussions according to the rules of client centered therapy (Rogers 1951). This part can be understood as the "educational phase" according to (Meichenbaum 1978).

2. Coping Training

- Physical Fitness Training

The self perception in situated stress reactions is improved with the progress in daily fitness training. Aside from that the practice of relaxation training, after fitness training, is easier due to a better discrimination of muscular tensing and relaxing. A more regular breathing is also induced.

- Space Wheel Training (Rhoenrad)

Observations of behaviour in unusual motion situations is very helpful. The ability to differentiate between AS and individual stress symptoms is improved. There is a rapid adaptation and habituation to the specific motion profile of this device. Desensitization effects are limited on this device.

- Relaxation Training

Relaxation training consists of a tape (Kemmler 1980) which incorporates parts of the Progressive Relaxation Technique of (Jacobson 1938) and the Autogenic Training of (Schultz 1973). The program is shorened through certain relaxation habituations. This shortened form is used for individual operational situations. The main effects can be observed in the learning process. First, there is a sensitization of body perceptions and reactions including emotions and cognitions, later we can affect a desensitization. After these relaxation techniques have been introduced clients are more and more able to

describe their individual activities of so called "naive state regulation" (Nitsch et al. 1979). They recall using muscular tension against the symptoms, as well as difficulties in breathing. They are able to tell S - R sequences, the increase of nervousness and the development of emotional cognitive self-evaluation, as well as the anticipation of AS. It seems that some develop the symptoms independent of motion. Rather, it comes from their high level of stress. Mild motion stimuli could be a trigger. However, specific flight maneuvers are also responsible for AS. These stress reactions in the sense of negative self perception, fear of achievement (apprehension of failure, threat of health integrity) serve as reinforcement in the development of AS. Continual self observation in relaxation training and discussions with the trainer lead to an ability to clearly differentiate between the emotional and cognitive processes. The danger to flight career and to the low level of self control in the flight situation lead many to a new state of extreme subjetive helplessness. This could also lead to flight phobias, depressive reactions and psychosomatic disturbances. Napalkov phenomena are also observable.

- Mental Training (Cognitive Pretraining)

The person is lead through three steps of an imaginative thought process, under intensive reproduction of sensoric and motoric motion sensations (Kemmler 1979). The confrontation with tasks performed during flight maneuvers is a further behavioral diagnostic analysis. Noted is the beginning of performance dificits, rise of incompetence, emotional and cognitive processes of self evaluation and the anticipation of AS as reinforcement. As exact self-perception and evaluation from the person is necessary for list of flight stress situations and for the majority, AS is a learned process that follow a paradigm of classical conditioning (see Figure 2). The visible dramatic peak in AS is when the classical conditioning process is initiated by unconditioned situations in which the person can not cope or adapt. Later, we often find an reinforcement of AS incidenne by self stimulation processes (operant conditioning).

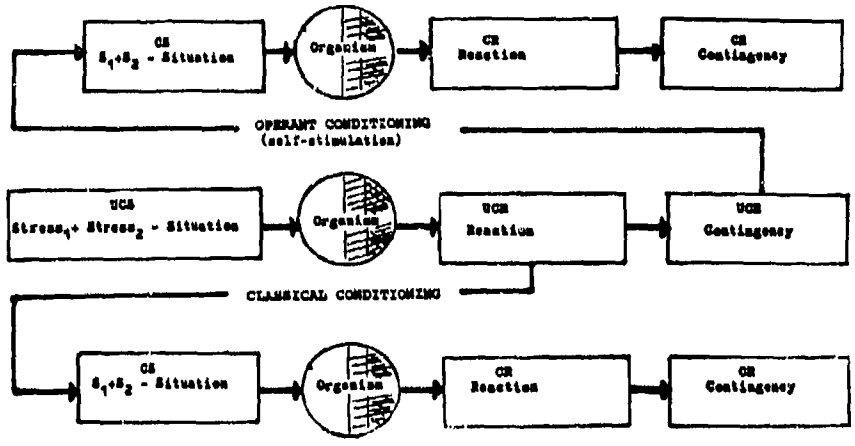

Fig.2 Model of the development of motion-sickness as a learning process

S_1: motion stressors
S_2: Person-/situation-specific stressors

CS: conditioned stimulus
UCS: Unconditioned stimulus
CR: conditioned reaction
UCR: Unconditioned reaction

Organism: stress moderator system

Adaptation, Desensitization and Self-Control Management (Phase 2)

This phase in the AATP contains measures of adaption under simulated flight conditions.

Desensitization

Desensitization follows a "level" adaptation system and a situation resulting in many movements (i.e., space wheel, rotation chair, modified Stille-Werne rotation device, spatial disorientation simulator, and centrifuge). The most economical would be desensitization under specific conditions. It makes more sense to have a wide spectrum of provocative situations, due to the various flight conditions. One special problem is that of the forward-downward movement; there are no adequate simulators.

We started to use the centrifuge, but the manpower requirements make this approach not cost effective. The desensitization procedure makes one aware of earlier false coping methods (i.e., bad eating habits, muscular tension, changes in respiration rates, and fixation on the symptoms).

Phenomenologically interesting is the appearance and process of failure as in cognitive worry and in emotionality. Fear of failure leads to an enforced perception of the physiological arousal and it distracts from the task at hand. The performance level then decreases. The excitement is then interpreted as the reason for the anxiety. This decrease in performance leads to anticipation of failure, which the arousal strengthens and reinforces. The higher the probability that AS will occur, the closer the person gets to the AS triggering situational condition. The important moderator variables of the organism seem to be the type of achievement motivation, the sensitivity to loss of competence and the inadequate self regulation.

The Phase two procedures follow the well know "coping modes" of stress research conducted by Lazarus and Launier in 1981. These include:

1. Seeking of information (reduction of dissonant information)
2. Direct action
3. Action inhibition
4. Intrapsychic processes for problem regulation

We have found that the countermeasures used are highly dependent on the situational context and the individual moderators.

Self Control Management

Desensitization is extended to self regulation. Therefore, the development of a working sequence of self control is necessary (Kanfer and Phillips 1970). Individuals learn the following:

1. Self monitoring of behavior in problem situations,
2. Self evaluation of coping techniques,
3. And self reinforcement of effective behavior. (See Figure 3)

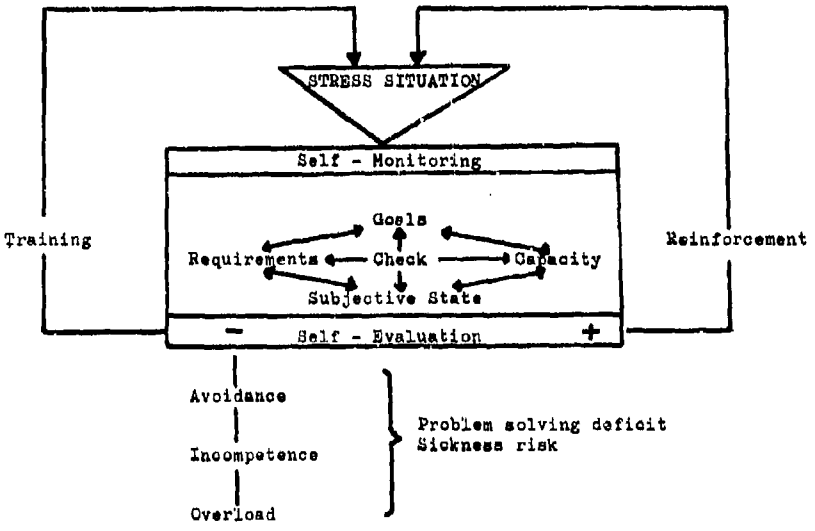

Fig. 3 Model of Self Control Management

The self control management procedure not only addresses stress conditions, it can also highlight the different emotional and cognitive processes that the client perceives as threatening his ability to cope. The cognitive structure of the task load and the stress factors (including motion stress) and the organization of the individual's behavior in connection with the anticipation of problem solving and timely regulation are of primary importance in this training.

Inflight Desensitization and Evaluation (Phase 3)

Transition Flights

The ability to apply the training and the evaluation of its effectiveness is done in three to five flights. The first flight (30-45 minutes) ist for students familiarization only.

Candidates (students) must apply self-regulation techniques inflight. In the second flight (45-60 minutes) definite tasks and flight maneuvers are assigned. The third flight (60-90 minutes) consists of a demonstration of emergency procedures and aerobatic fight maneuvers. When there are problems which cannot be controlled, the flight is repeated. Almost all the candidates have been observed with mild symptoms, but in each case he has successfully coped with them. When uncontrollable symptoms (vomiting) occur in more than two flights, then the candidate is attrited from the training program. Weather induced delays during the AATP are very detrimental to satisfactory progress, especially in the transition flight phase.

Conclusion

The accent of our training program is on psychological intervention techniques. In order to determine the psychological aspect of AS and to modify it, we have developed a program that makes possible the adaptation to unusual inflight motion conditions through better self-regulation.

Important information about behavior structure, stress factors and conditioned stress reactions in the development of AS was found during the training process. Autonomic arousal processes with their perceptions, cognitive and emotional processes and their subjective evaluation seem to play as large a role as unusual physical and physiological stimulus situations. The combination of physiological adaptation and psychological intervention appear to be a highly effective anti-airsickness training program within specific limits (see Fig.4).

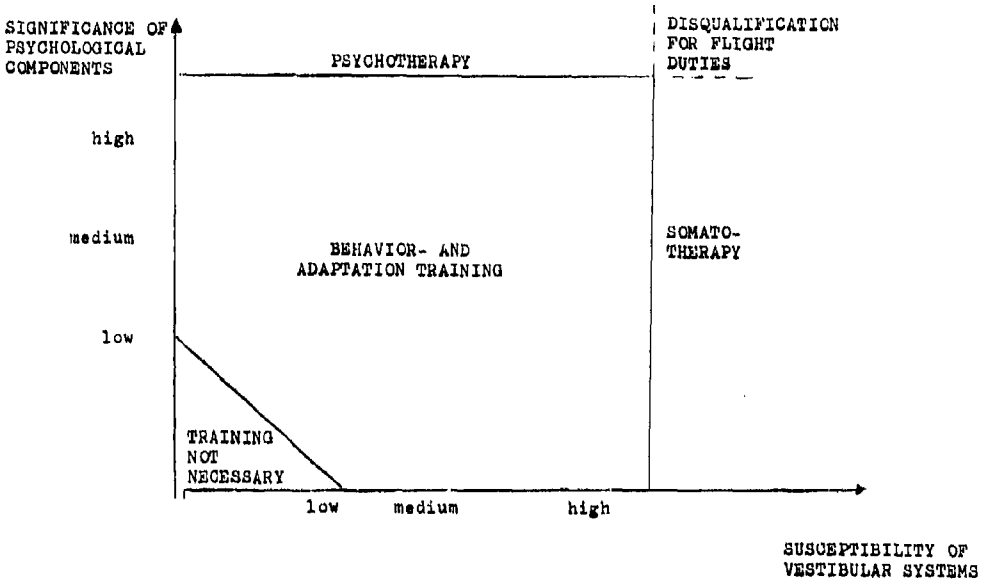

Fig. 4: Countermeasures of air-sickness as a function of psychological and somatic components

This can be seen through other authors (Levy, Jones and Carlson 1981, Ceresia 1983, Bagshaw and Stott 1983, Marsha and Rocco 1983, Lochridge and Giles 1983). It is a fact that the physiological stimuli conditions were not very extensive (Kammler, Houk, Bellenkes and Guedry 1983). This could be seen as the reason for the presence of psychological determinants in the development of AS. In order to deal with the stress conditions, psychological coping mechnisms (self-regulation) must be given equal significance and consideration. The combination of these coping methodologies leads to sensitization and ultimately to a desensitization of perceptions. The instruction about personality traits, behavior characteristics and stress reactions give the individual an adequate frame of reference for problem orientation and activity management.

Physical fitness training and relaxation training seem to accentuate the emotional reactions. As for the relaxation procedures, different techniques may have to be used. This is due to there being no one technique being better than another. Each is highly individualistic in nature and are considered successful when the candidate is able to control his autonomic responses. Therefore, it is necessary to define individual arousal parameters and to decide upon an adequate relaxation technique. The importance of biofeedback techniques has been proven (Levy, Jones and Carlson 1981; Cowings and Toscano 1982); however, this importance should not be overestimated (Gardner 1982). The technical equipment does not guarantee effective self control in a real situation and it does not replace the experienced clinical specialist. Mental training, desensitization, and self control management improve the perception of cognitive processes and the discrimination of cognitions versus emotions.

The cognitive structuring of the training situation leads the candidate to an effective transfer to reality.

In conclusion, there are the following main results:

1. Diagnostic relevant results has not only been found in the diagnostic phase but during the AATP, due to a decrease of dissimulation tendencies and an increase of adaequate self perception of internal, emotional and cognitive processes.

2. Desenditization and Self Control Management does not primarily regulate or desensitize provocative motion stimuli but internal perceptions. The effect of this AATP consists in a habituation of self perception.

3. Adaptation as well as non-adaptation seems to be to us an outcome of conditioned learning processes, most of which are established by classical conditioning and reinforced by self- stimulation processes (operant conditioning).

4. Responsible for adaptation or non-adaptation are moderator variables in the organism. Besides physiological factors psychological factors has to be considered, among which achievement motivation, sensitivity to loss of competence and inadaequate self regulation seem to be most relevant.

5. Prediction of motion sickness would probably be much more valid and reliable when we add psychologically defined scores to the scores of susceptibility based on laboratory motion tests.

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DISCUSSION

The discussion of this paper follows paper 42

BIOFEEDBACK TREATMENT OF AIRSICKNESS: A REVIEW

by

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The affect of significant and persistent airsickness on the capability of operational pilots is well known. The effect of airsickness on the progress of student pilots, who are affected much more frequently, is equally well known. For years, our predecessors have had few options in dealing with airsickness which would give such fliers a fair chance to adjust to the stresses of flight, in order to avoid disqualifying them from further flying. The cost of replacing a trained operational flier is substantial, perhaps representing over a million dollars. The cost of disqualifying a student pilot or navigator is less, about $15,000 for a pre-solo student pilot, but this loss is still significant because of the numbers of fliers involved. In addition, there is the human side of the problem. Consider the plight of the operational pilot, who not only must suffer through the physical unpleasantness, but also is confronted with a probable end to his or her career. Consider also the plight of the student flier, who not only experiences the episodes, but also may feel himself or herself disgraced in front of peers, and must abandon a much-dreamed-of future having high career potential. Heretofore there was little to do once a short course of medications proved unequal to the clinical situation. Now we have the alternative of biofeedback treatment.

Let us first dispose of the review of the literature which our title says that we will present. We used the USAFSAM scientific literature database computerized search system and queried the three major databases: MEDLINE, PsychoINFO, and NTIS for the period 1963-1983, using a two-keyed code, biofeedback/aircrew, with each keyword represented by four synonyms. This search yielded 17 articles, including two papers by Cowings and Tusçano (1, 2) acquired three times, two papers (3, 4) from our own laboratory acquired twice, and one paper by Graybiel (5) saying that biofeedback doesn't work when subjects are exposed to a questionable provocation. If you drop back to one keyword, biofeedback, and our four synonyms, voila!--the Tower of Babel! We obtained 1400 references documenting every possible conclusion. We do not recommend this latter exercise to the practitioner of aerospace medicine who needs to know if there is a way to medically manage or modify airsickness without drugs.

What is biofeedback? It is a process which addresses internal physical states and events. Having stated this definition, we must step back briefly and observe that nature clearly limits our awareness of internal states and events for entirely appropriate biological reasons: if it were not so, our consciousness would be bombarded with a cacophony of stimuli which only rarely would have any usefulness to us. Consider the beating heart. If one tries hard enough, one may "sense" it vaguely, but even so, what is one to do with this information once registered?

The aim of biofeedback is to assist the patient in focusing on the awareness of some cluster of internal states or events, in developing skills to modify or moderate that cluster, and then, in the service of biologic utility, to allow that awareness to fade away, so that the cluster once again has been internalized, while the skills for its modification or moderation are maintained. It is the enhancement and control of biological awareness as a state of consciousness (6). The end product, then, is self-regulation of internal events or states which previously resulted in distress. The process is much like that of learning a motor skill, wherein the conscious awareness of the sequence of motor events is high early in the learning, but progresses to an internalized, coordinated response as proficiency is achieved. Those of you who have learned to ride bicycles will recognize this sequence. Feedback is crucial to both of these processes.

We may make some other observations about biofeedback as a therapeutic tool. It appears that:

—Biofeedback can enhance progressive relaxation

—Biofeedback can reduce anxiety symptoms

—Biofeedback makes the patient more willing to accept the therapist's interpretations and suggestions

—Biofeedback helps the patient feel that his problem is under his own control, increases his self-confidence, and gives him hope for the future

—Biofeedback is more effective under those conditions which facilitate transference; the therapist must convey warmth, genuine concern and empathy

—Biofeedback works best when the therapist serves as a facilitator to help the patient integrate his understandings and skills into a new approach to the problem.

Is biofeedback a "necessary and sufficient" process in the sense that the biologist describes necessary and sufficient stimulus? Clearly not. Many things go on during biofeedback treatment. Consider the following, which must be co-processes:

—Assertiveness training may be an important aspect

—Acquisition of more generalized coping skills may occur

—Concerned attention from a health care provider may be important

—Systematic desensitization while treating the anxiety may play a role

—Positive change in lifestyle may occur.

Is biofeedback the only approach? No, there are other ways to alter psychophysiologic functions. Barber (7) identifies hypnosis, autohypnosis, autogenic training, autosuggestion, direct suggestion, meditation, hatha yoga and relaxation training. Relaxation may be the key work here; relaxation is clearly important as an inherent part of biofeedback therapy. Relaxation techniques are the one part of the treatment process which the patient can take home and practice, and those patients who do so improve their potential for a successful therapeutic outcome. However, one should note that in the two laboratories where airsickness is the center of attention, biofeedback or autogenic feedback training which includes biofeedback is used. We know of no systematic studies on or applications of the other approaches. On the other hand, a clinical study by Lochridge and Giles (8) reported 93% success with a combination of Bárány chair stimulation and simultaneous relaxation techniques when applied to a population of military student pilots with recurrent airsickness during their first few flights. These students received one to three one-hour sessions of simultaneous vestibular stimulation and relaxation training, between which their flying training schedule continued uninterrupted.

Does biofeedback treatment for airsickness always work? No, it fails approximately one time out of four, if one uses return to the cockpit as the measure of success. Our experience seems to be like that of the U.S. Navy (9), where biofeedback is used to treat a variety of problems associated with anxiety about flying, and at the German Institute of Aviation Medicine (10), where a variety of stress reactions in military pilots are treated.

As another way of looking at the utility of biofeedback in treating airsickness, consider two studies from the Autogenic Feedback Laboratory at NASA Ames Research Center. In the first study (1), subjects were assigned to one of four groups based on motion sickness susceptibility and were treated as follows:

	SUSCEPTIBILITY: HIGH	SUSCEPTIBILITY: MODERATE	
AFT	N = 6	N = 6	treatment and exposure to Coriolis stimulation
No AFT	N = 6	N = 6	exposure to Coriolis stimulation

The outcome of the study was as follows:

	SUSCEPTIBILITY: HIGH	SUSCEPTIBILITY: MODERATE
AFT	tolerated Coriolis stress longer	tolerated Coriolis stress longer
No AFT	no change	some habituation for Coriolis stress

In the second study (1), subjects were assigned to one of three groups based on motion sickness susceptibility, with the following treatment and results:

TREATMENT	RESULTS
AFT	tolerated Coriolis stress longer
"Sham" Rx	no change
None	no change

The point being pursued in the last few paragraphs is that all of us have some awareness of internal states and processes. Individual differences are large, but for each of us that potential for the development of self-regulation can emerge--and under a variety of conditions. It appears that the acquisition of that skill proceeds more efficiently when biofeedback of the processes involved is used.

It is appropriate at this point to describe the program at the USAF School of Aerospace Medicine which uses biofeedback in the treatment of chronic, severe airsickness. Levy et al. (3) describe the major steps in the program:

—Candidates for therapy are identified by operational personnel

—They are carefully screened during an intake interview to insure high motivation, and to rule out intercurrent medically disqualifying physical conditions; medical consultation is obtained if indicated

—Intrapsychic factors which might interfere with treatment are explored, including ambivalence or anxiety about flying

—The biofeedback techniques and instrumentation are fully explained

—The patient is instructed in relaxation exercises, given written instructions, and told to practice in his or her room for a day or two prior to treatment

—Treatment sessions are given in a biaxial Coriolis stimulating chair which rotates at up to 20 rpm and can tilt 40 degrees to the left or right while rotating

—Two sessions per day are given, for a maximum of twenty sessions.

The focus is on the recognition of early symptoms of motion sickness, and the use of relaxation techniques, monitored by the feedback of sweat rate and of surface skin temperature (both from the fingers of the left hand) to let the patient know when the relaxation techniques are working.

Under this regime, the patient learns to attend to the premonitory symptoms, rather than trying to suppress or ignore them. He or she learns which relaxation technique works best: deep muscle relaxation, active or passive mental imagery, abdominal breathing similar to yogic breathing, or attention primarily to the feedback dials or tone. Once a particular method is identified as effective, its use is encouraged. First the patient learns to relax and make the sickness subside, then the technique is refined and accelerated until it takes only a few seconds rather than several minutes, and finally it is practiced while mentally diverting exercises are performed simultaneously. Since there is some accommodation to the concomitant vestibular stimulation, the chair must be rotated faster, and the lateral motions must be given more suddenly and closer together in order that the threshold of symptoms is stimulated. The patients are <u>not</u> conditioned not to get sick; they are taught how to control the symptoms when they occur.

We had a protocol in the mid-1970s which emphasized relaxation and conditioning to vestibular stimuli. This study was never reported, but was able to return about 40% of the patients to flying duties. The current study, which will reach its end when the two-year followup of the last subject is ended in June 1984, is currently about 75% successful. Thus, use of the biofeedback modalities in the dynamic Coriolis chair environment was much more effective in returning our fliers to operational flying than was the relaxation training and desensitization alone.

Active control of airsickness by biofeedback training is possible, and offers a new approach to the control of this distressing syndrome beyond passive accommodation and beyond medications.

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DISCUSSION

PRICE: I'd like to ask all three speakers if they plan to follow up long-term the people they have considered successes because some of the experiences in this business, behavior modification and bio-feedback, are that the patient may relapse several years down the road and once again have the same kinds of reactions to whatever stress it is. So I would ask you if you have plans for follow-up of your successes.

JONES: We have followed these people for two years and that is about as far as we're going to go with it. We have no evidence of changes after the first year follow-up and it's very difficult to keep up with 54 air force pilots. We have some who have gotten out of the service, for instance.

KEMMLER: We have the experience that the more time passes the better they are in flying. As we look at the people we had in 1963, 19 of 23 are still doing well.

STOTT: I have showed you fall-out data for the past ten years. It's extremely reliable for the past three years. Perhaps a little bit deficient in the preceding time but we continue to follow up for at least five years, at least that's our intention. It's done on an informal basis at present simply because aircrew let us know what is happening to them and we're very grateful.

CRAMPTON: Colonel Jones, have you done any control experiments with biofeedback, that is, taking the signals coming on the moisture and temperature of the hand and giving the patient either bogus information or information that will accentuate the response?

JONES: No, we have not done that and the reason is an ethical one. We are these peoples last chance to fly and since we have to coordinate with the air training command and scratch 5 extra flights out of their tight budget, to experiment within our own parameters and find out that something wasn't working might cost that man his career. We are rather in a position of the people that discovered you could stop rheumatic fever with pencillin and how in the world do you use some other drug when you've got one that works all the time. I hope in the future to be able to contract with the training command to give me two chances at an individual so we can try something else, maybe try a shorter period or a more simple technique. I appreciate what's behind your question and I'm as curious about that as you are, but we have not used any double yoke subjects or sham signals or anything else.

KEMMLER: Dr. Stott, I noticed that in the photograph of your accelerator the subject is keeping his head bent anywhere between 30 to 45° so I would like to ask you what kind of stimulus you think the vestibular system received? Combined X and Z stimulation, or was the head fixed in your experiments?

STOTT: No, we deliberately don't fix the head. Our intention is that the subject should make head movements while he is undergoing oscillation.

UNIDENTIFIED SPEAKER: Dr. Stott, I think that as far as humanitarian concerns, the desensitization of the crew is very important and praiseworthy, I also think that the pilot's ability has nothing to do with his susceptibility to motion sickness. However, operationally speaking don't you think that those pilots that came to look for assistance because they were unable themselves to adapt are going to during their career meet with circumstances for which they have been unprepared by the program?

STOTT: Once a pilot has successfully desensitized he rarely comes up against motion sickness problems abruptly later on in his career. As we see it, the problem is the people we see are very slow adaptors. The training course is not really related just to introducing motion stimulation in a graded fashion, it's related to introducing flying skills in a graded fashion and a pilot may suddenly be asked after relatively unprovocative sorties to do spinning, or acrobatics may be introduced. We feel that the important thing is that these stimuli should be introduced at the rate the trainee can absorb them physiologically. Once that is done it has not in any way been our experience that trainees suddenly come up against something for which they have not been prepared.

GUNDEY: Relaxation training and psychotherauptic training does not seem to be part of the RAF program yet the percent success seems to be at least equivalent to the others. I do know that Dobie when he started the program had informal psychotherauptic methods that he used.

STOTT: The psychological aspects of our treatment are not strongly emphasized, largely because we feel that motion sickness is a physcological event although there may be additional psychological components. We feel if we can convince people that physiologically they are desenitized to motion sickness, then psychological elements in most cases will fall into place quite naturally. We still include in our assessment program a battery of psychometric tests but we are impressed by the poor correlation that exists between these various measures and tend only to use them to indicate what sort of person we are dealing with, and to do nothing more formal then that.

42-5

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14. Abstract

Motion sickness continues to be a significant operational problem in the armed forces of the NATO Countries. Space motion sickness has emerged as a major operational concern in the short duration space shuttle flights, with nearly 50% of all crew members experiencing some symptoms. The 21 papers included in these proceedings concern recent developments in understanding etiological factors contributing to air, sea, car, simulator and space sickness, identifying neurological mechanisms mediating motion sickness, developing predictive tests of susceptibility, and preventing and treating motion sickness. These papers and their accompanying discussions highlight the different stages of progress and understanding that have been achieved in these sub-areas of motion sickness research; they also identify laboratory findings that can be incorporated in programs for alleviating motion sickness under operational conditions.

Additional Keywords:
NATO ; aerospace medicine ;
; feedback ; disseminating ;

AGARD Conference Proceedings No.372 Advisory Group for Aerospace Research and Development, NATO MOTION SICKNESS: MECHANISMS, PREDICTION, PREVENTION AND TREATMENT Published November 1984 188 pages Motion sickness continues to be a significant operational problem in the armed forces of the NATO Countries. Space motion sickness has emerged as a major operational concern in the short duration space shuttle flights, with nearly 50% of all crew members experiencing some symptoms. The 21 papers included in these proceedings concern recent developments in understanding etiological factors contributing to air, sea, car, simulator and space P.T.O	AGARD-CP-372 Motion sickness Aerospace medicine	AGARD Conference Proceedings No.372 Advisory Group for Aerospace Research and Development, NATO MOTION SICKNESS: MECHANISMS, PREDICTION, PREVENTION AND TREATMENT Published November 1984 188 pages Motion sickness continues to be a significant operational problem in the armed forces of the NATO Countries. Space motion sickness has emerged as a major operational concern in the short duration space shuttle flights, with nearly 50% of all crew members experiencing some symptoms. The 21 papers included in these proceedings concern recent developments in understanding etiological factors contributing to air, sea, car, simulator and space P.T.O	AGARD-CP-372 Motion sickness Aerospace medicine
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Papers presented at the Aerospace Medical Panel Symposium held in Williamsburg, US from 3 May to 4 May 1984.

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