

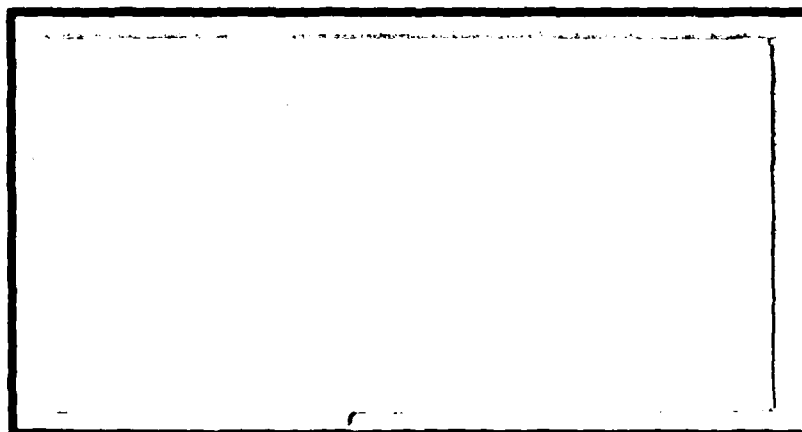
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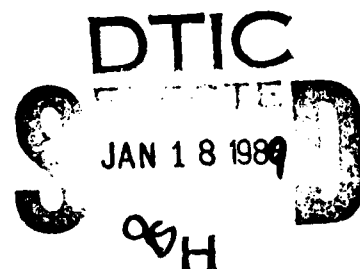
AFIT/GEO/ENG/88D-4

A STUDY OF MOTION SICKNESS:
MATHEMATICAL MODELING
AND DATA ANALYSIS

THESIS

Mark F. Scott
Captain, USAF

AFIT/GEO/ENG/88D-4



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A STUDY OF MOTION SICKNESS: MATHEMATICAL
MODELING AND DATA ANALYSIS

THESIS

Presented to the Faculty of the School of Engineering
of the Air Force Institute of Technology
Air University
In Partial Fulfillment of the
Requirements for the Degree of
Master of Science in Electrical Engineering

Mark F. Scott, B.S.
Captain, USAF

December, 1988

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Preface

This study developed a mathematical model relating an individual's level of motion sickness with the biophysiological data collected on that individual. It also evaluated the effect of the drug phenytoin (marketed as Dilantin) on the evolution of motion sickness.

The results of this research were very promising. Using commercial software, mathematical models were developed that correlated with the symptom levels actually reported by test subjects. The drug phenytoin appeared to delay or even prevent the onset of motion sickness, depending on the individual being tested.

In performing the research and writing this thesis, I've had alot of help from others. I'm deeply indebted to my faculty adviser, Dr. Matthew Kabrisky, for his advice and encouragement, and for taking time out of his busy schedule to carefully proofread my thesis many times. I'm also grateful to Dr. William Chelen for creating much of the AFIT motion sickness equipment, for developing the experimental procedure used in this study, and for having the patience to tutor me in the basics of biomedical engineering. I'd like to thank Captain Roy Morales, my thesis partner, for his assistance and good humor throughout this study. I also want to thank Dan Zambon of the Information Sciences Laboratory for helping me use the software essential to my thesis. A word of thanks is also owed to Jerry Hild and Leroy Cannon of the Aero/Astro Engineering Lab for their technical assistance during the experimental portion of this research. I'd like to thank Captain Vicky Robinson, my study partner, for helping me survive the first two quarters of this graduate program. Finally, I wish to thank my wife [REDACTED] and my son [REDACTED] for their understanding on those many nights when I was busy with work.



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Abstract

Eleven male test subjects were given the drug phenytoin in a double-blind, placebo-controlled crossover experiment. One volunteer with a history of drug allergies developed a reaction to phenytoin and was dropped from the experiment. The remaining volunteers were then rotated in a motion chair while eleven of their physiological parameters were measured. The drug appeared to delay or even prevent the evolution of motion sickness, depending on the test subject.

Previous AFIT motion sickness models were analyzed. Using Barron Associates' Abductive Reasoning Mechanism (ARM) software, new motion sickness models were developed from the 1986 and 1987 AFIT test data.

The biophysiological data collected in 1988 were analyzed for trends. The average heart rates of subjects on the placebo increased during the course of motion sickness, while the average heart rates of subjects on dilantin stayed constant. The electronystagmograph, electrosplanchnograph, and thoracic respiration signals, when measured in rms voltages, increased with the evolution of motion sickness. The facial and peripheral photoplethysmograph data were considered unreliable.

The ARM software was again used to develop motion sickness models from the 1988 test data. During each motion sickness experiment, the test subjects would periodically describe their level of motion sickness, using a ten level scale. A motion sickness model was created with the data from the subjects taking the placebo. It calculated a level of motion sickness within one level of the reported level of motion sickness for only 27% of the time. The 1988 model using the data from the subjects on dilantin had a one level accuracy of 76%.

A STUDY OF MOTION SICKNESS: MATHEMATICAL MODELING AND DATA ANALYSIS

I. Introduction

Background

Motion sickness is a major problem for military aerospace operations. Between 20 and 30 percent of Great Britain's Royal Air Force pilot trainees showed symptoms of airsickness during early sorties (1:1144). Motion sickness also affects navigators, flight engineers, and electronics operators (1:1144; 10:470). Although often a byproduct of such aerial maneuvers as spins and aerobatics, motion sickness can also occur during lengthy, low altitude flights (10:472). Simulator sickness has been reported by pilots during or after flight simulator training (9). Motion sickness has also occurred in space, where "four out of every 10 astronauts become sick during space shuttle missions" (2:51).

Motion sickness develops when an individual is exposed to unfamiliar motion stimuli. The provocative movements are normally real, as in the case of aerial aerobatics. However, as exemplified by simulator sickness, they can also be apparent, and without any physical motion.

The four main characteristics of motion sickness are nausea, vomiting, pallor, and cold sweating (10:469). However, the following symptoms are also associated with motion sickness - dizziness, a spinning sensation, disorientation, tingling, fatigue, warmth, coldness, lightheadedness, dry or acid mouth, salivation, stomachache, headache, anxiety, and apathy (5:24).

The currently accepted explanation for motion sickness is the sensory conflict theory (1; 2; 10). It assumes that the brain is constantly receiving information on the

position and movement of the body. These data can come from the visual system, or from the vestibular organs of the inner ear (10:476). Sensors in muscles of the neck, arms, legs, and other parts of the body also provide the brain with positioning data known as proprioceptive information (2:53). Motion sickness occurs when these sources of information conflict with the brain's memory of normal motions (1:1144; 11:477). For example, suppose an airline passenger is looking at a wall, devoid of any visual reference. If the plane ride is turbulent, the vestibular organs will register sharp motions. While the visual picture conforms with the lack of motion normally expected by the brain, the information provided by the vestibular organs does not, and motion sickness can result.

Treatment of Motion Sickness

Motion sickness has been treated in several ways. The most promising methods of treatment are drugs, biofeedback, and desensitization.

Certain drugs can mitigate the symptoms of motion sickness. Unfortunately, most of the presently used drugs have a depressant effect, along with other undesirable side effects (10:491). As a result, anti-motion sickness drugs are not usually taken by flying personnel (10:491).

However, some recent reports may pave the way for an increased use of drugs in motion sickness treatment. The side effects of the drug scopolamine have been reduced through the use of the transdermal therapeutic system of administration (18). In a pilot study involving the use of the anticonvulsant drug phenytoin, a "greater than six times increase in tolerance to motion stress was obtained", and without any of the side effects of traditional motion sickness drugs (6).

Biofeedback has also been successfully used to treat airsickness. In one study, a drug-free, biofeedback-based treatment program returned 84 percent of chronically airsick flying personnel to flying status (24). Treatments consisted of motion chair rides, with the patient monitoring and controlling changes in his electromyographic

potential, surface skin temperature, and galvanic skin reflex. NASA researchers have combined feedback with self-suggestion exercises to control such physiological functions as "heart rate, skin conductance, depth and rate of respiration, and the flow of blood to the hands." (23:36).

Desensitization therapy helps airsick aircrew by exposing them to additional motion stimuli. This method of treatment is based upon the sensory conflict theory, and assumes that continued exposure to provocative motion will result in new sensory patterns becoming "incorporated into a postulated neural store of such patterns" (1:1144). Thus, the brain's memory of motions will have a better chance of recognizing the sensory information produced in subsequent flying episodes, and motion sickness will be less readily provoked. The Royal Air Force Motion Sickness Desensitization Program has had an 84 percent success rate with chronically motion sick flying personnel for the period 1981 to 1983 (1:1148). The program consisted of both a ground phase of treatment, using a motion chair, as well as a flying phase.

AFIT Motion Sickness Research

AFIT has been researching motion sickness for five years. In 1983, Earl and Peterson assembled the rotating chair facility used to induce motion sickness. They also created a biophysical data acquisition system by combining commercial physiological monitoring equipment with physiological sensors that they themselves had designed and constructed (12). In 1984, Fitzpatrick, Rogers, and Williams attempted to automate the data collection process by developing the software and hardware necessary to integrate a MASSCOMP MC5500 computer into the system (13). In addition, they manufactured two new sensors for measuring gastrointestinal electric potential and eye motion (13: 2-3).

Jarvis and Uyeda continued studying motion sickness in 1985, and were the first AFIT researchers to report any experimental results (22). The researchers relied on strip chart recorders and magnetic tape for data collection because of difficulties

with the MASSCOMP computer. They were joined by Dr. William Chelen, M.D., who redesigned and rebuilt many of the physiological sensors. (Dr. Chelen has since become a permanent member of the AFIT motion research program.)

In 1986, Hartle, McPherson, and Miller moved the rotating chair and its support equipment to a location providing a better working environment. They eliminated the use of the MASSCOMP computer for data collection, preferring to rely on magnetic tape and strip chart recordings. Despite these time-consuming changes, they were able to experimentally identify several physiological trends associated with the evolution of motion sickness (21:102-103; 27:87). They used statistical software to analyze their data and to develop equations predicting the levels of motion sickness experienced by a test subject during the course of an experiment (21:97; 25:59; 27:84).

In 1987, Drylie, Fix, and Gaudreault continued the collection of motion sickness data. They improved the system by standardizing test procedures and increasing the reliability of certain physiological sensors. The researchers also added a differential stethoscope for monitoring gastro-intestinal sounds, a bank of low pass filters to reduce incidental electrical noise, and a 16-channel strip chart recorder (14:7). They used a Zenith 248 computer and commercial signal processing software to digitize, display, and numerically analyze the experimental data.

The 1987 research team was innovative in their results as well as in their procedures. Drylie and Gaudreault reported additional conclusions concerning motion sickness trends (11; 17). Fix developed a new equation for correlating physiological data with a volunteer's subjective sense of malaise (14:23). He also created a neural network simulation as another method of predicting a test subject's level of motion sickness.

Summary of Current Knowledge

Electroencephalograph (EEG) The EEG is used to observe the electrical activity of the brain (29:92). Brain waves recorded at the surface of the scalp vary in intensity from about 5 to 200 microvolts, and in frequency from about 0.1 Hz to 100 Hz (29:92). Four brain waveforms have been described in the literature - the delta wave (0.2 - 3.5 Hz), the theta wave (3.5 - 7.5 Hz, 50 - 200 microvolts), the alpha wave (7.5 - 13 Hz, 10 - 100 microvolts), and the beta wave (13 - 30 Hz) (29:92).

The AFIT research teams have recorded EEG activity which appears to specifically accompany motion sickness. In 1986, Hartle, McPherson, and Miller reported "distinctive brain wave patterns" appearing with the onset of motion sickness, including an unexpected pattern in the 0.1 Hz frequency range (21:46). Drylie, Fix, and Gaudreault also noted low frequency EEG signals in the 0.1 Hz range (17:28). However, only one of their subjects had EEG signals with an amplitude change similar to that reported a year earlier (17:30). Gaudreault hypothesized that the low frequency brain wave activity was due to hyperventilation, since most of the amplitude changes occurred late in the experiments, and usually after the subjects had ended their head movements (17:40).

Electrocardiograph (EKG) The EKG measures the electrical potentials generated by the heart (29:72).

The AFIT studies have reported a few instances of sinus arrest, when a test subject's heart rate will drop to 30 to 35 beats per minute (21:69). Hartle, McPherson, and Miller observed three cases of sinus arrest during experimentation (21:70). The only case of sinus arrest reported in 1987 occurred when the subject was recuperating after the experiment (17:28). To prevent any possible danger to the test subject, AFIT researchers have always ended the experiment when sinus arrest appeared (4:12).

Electronystagmograph (ENG) The ENG measures the changes in potentials generated by movements of the eyeball (29:98). AFIT researchers have used one pair of ENG sensors to measure horizontal eye movements, and a second set to measure vertical eye motion (21:40).

AFIT researchers have been collecting ENG data for the past three years. In 1987, Drylie observed that the rms values of the ENG signals would increase in amplitude with the evolution of motion sickness (11:44). However, the changes in signal strength may have been a sweat artifact (3). The 1986 team recorded ENG data on magnetic tape, but did not analyze their results (21:51). In 1985, Jarvis and Uyeda furnished some spectral analyses of ENG data without comment (22:84).

Gastro-Intestinal Measurements AFIT researchers have used different sensors for measuring gastro-intestinal activity. In 1985 and 1986, an electrogastrograph (EGG) and an electointestinograph (EIG) were used. The EGG supposedly detected the electrical activity of the stomach, while the EIG presumably measured the activity of the small intestine (22:60-61). Drylie, Fix, and Gaudreault changed the measuring equipment used in 1987, due to questions concerning the validity of the EIG and EGG data (3). A phonosplachnogram was attached to the test subject's central abdominal region, and recorded bowel sound activity (14:13). An electrosplachnogram made electrical measurements of the gastro-intestinal tract (17:35).

Jarvis and Uyeda noted that abdominal electric potential increased with the evolution of motion sickness. They observed that the amplitude increases of the EIG signals "ranged upwards of nearly fourteen-fold, with the maximum average increase approximately 400%" (22:95).

Hartle, McPherson, and Miller also found trends in gastro-intestinal activity during motion sickness. They noted that the abdominal electric potentials reflecting the activity of the small intestines "increased by about 400 percent" (21:80). Hartle

observed that the potentials corresponding to stomach activity "increased by about 600 percent" (21:83). He distinguished between the collected EIG and EGG data by noting that "the EGG signal has a higher amplitude of activity in a lower frequency range than EIG" (21:85).

Finally, the 1987 team observed that the amplitude of the electrosplanchnogram signals "increased significantly" during motion sickness experimentation (11:41). Gaudreault found that the phonosplanchnograph recordings "revealed a decrease in gastro-intestinal noise during the evolution of motion sickness for all subjects tested" (17:37). He concluded that the noise reduction "verifies that mechanical activity decreases as the frequency of the electrical activity increases in the gastro-intestinal tract" (17:37).

Galvanic Skin Response (GSR) GSR has been defined as "the dynamic (decreasing) variation of skin resistance between two points on the skin in response to a stimulus" (29:57).

AFIT researchers have continually found that skin conductivity increases during the course of motion sickness (11:35; 21:68; 22:81). The trend has been ascribed to the increased sweating that accompanies the development of motion sickness (21:68; 22:81). However, the skin conductivity increase is primarily due to pseudomotor activity and capillary vascular activity (3). Other investigators have also noted the relationship between skin conductivity and motion sickness (32).

Pallor A photoplethysmograph is used to "optically measure blood flow volume (skin pallor) changes at desired locations in the body" (22:57). In 1985 and 1986, the AFIT researchers used both facial and finger photoplethysmographs (21:51; 22:57). Drylie, Fix, and Gaudreault used only a facial photoplethysmograph in 1987 (17:17).

AFIT test data agree with the generally accepted notion that skin pallor

changes accompany the development of motion sickness (5; 26). Jarvis and Uyeda found that their experiments "essentially confirmed that pallor increases in the majority of subjects and generally precedes the onset of severe motion sickness" (22:87). However, since their equipment probably constricted vascular activity, their results are in doubt (3). Hartle, McPherson, and Miller also found that "pallor increases with the onset of motion sickness" (21:74). The 1987 team collected only two sets of photoplethysmograph data because of changes in equipment and procedure (17:26). However, Gaudreault was able to note that the data showed "both subjects becoming more pale, especially during severe malaise" (17:40).

Respiration Respiration can be measured in two different ways. A pneumograph will detect chest expansion and contraction (29:100). A spiograph will measure lung volume by responding to air flow during inspiration and expiration (29:101).

AFIT researchers have measured changes in both thoracic and abdominal (diaphragmatic) respiration during motion sickness. However, equipment problems have hampered the collection and interpretation of the respiration data.

In 1985, Jarvis and Uyeda used circumferential belts employing strain gauges to measure respiration (22:60). They presented spectral analyses of a few of their pneumograph recordings without drawing any conclusions (22:84).

Hartle, McPherson, and Miller changed the equipment used to measure respiration. They turned to a pair of pneumographs developed by Dr. Chelen (21:30). One pneumograph was used to measure abdominal respiration, while another detected thoracic respiration (21:41). In addition, they calibrated the strain gauge respiration measurements by comparing them with actual breathing volume data obtained by having the test subjects blow into a spirometer (21:41).

Both the 1986 and the 1987 research teams found a correlation between respiration and motion sickness. Hartle noted that, as the motion sickness symptoms developed, "the individuals had higher thoracic respiratory and diaphragmatic volume".

indicating that the subjects were taking larger and less frequent breaths (21:79). However, he felt that the use of the spirometer, which measured abdominal respiration, was an incorrect method of calibrating the diaphragmatic respiration measurements (21:74). In 1987, Drylie observed that, while the frequency of breaths did not change during an experiment, the volume of each breath increased significantly (11:39). He interpreted these data as indicating that "a person begins to hyperventilate as he gets sick" (11:39).

Temperature AFIT researchers have used thermistors to measure the peripheral skin temperature of their test subjects during motion sickness experiments.

The motion sickness research teams have differed in their interpretation of skin temperature data. In 1985, Jarvis and Uyeda observed that, as motion sickness evolved over time, temperature followed increasing linear, decreasing linear, or cyclical trends (22:90). Miller decided a year later that "temperature does not provide a really strong basis for incorporating it into the family of real good predictors of motion sickness" (27:65). Hartle felt that the temperature readings could have been adversely affected by the room environment (21:72).

In 1987, Drylie reported that "subject temperature did not change significantly" during the course of an experiment (11:37). However, Fix used temperature changes in his equation for predicting subjective levels of motion sickness (14:16).

Problem Statement

This study was designed to collect and analyze physiological data during the evolution of motion sickness. In particular, it intended to examine the effect of the anticonvulsant drug phenytoin on motion sickness. This research effort also attempted to develop an improved mathematical model for estimating an individual's level of motion sickness.

Scope

This research effort continued the study of motion sickness at AFIT. It was limited to:

1. Collecting motion sickness data on no more than 20 test subjects. These volunteers were male military personnel. Female subjects were not accepted, since the phenytoin might affect any potential pregnancies.
2. Analyzing the effect of an anticonvulsant drug (phenytoin) on motion sickness.
3. Improving on the mathematical models presently used to predict a test volunteer's subjective sense of motion sickness.

Assumptions

The assumptions underlying this research effort were:

1. Motion sickness has a neurological basis. It results from the brain's inappropriate attempts to cope with perceived changes in the body's position. The concomitant brain wave changes suggest a seizure phenomenon as the underlying cause of the physiological manifestation of motion sickness. Thus, an anticonvulsant drug such as phenytoin, which suppresses brain seizures, may also suppress motion sickness.
2. As motion sickness evolves in an individual, it produces measurable changes in certain bodily processes. The amount of change in these physiological parameters can be correlated with the level of motion sickness experienced and reported by the individual.
3. Motion sickness induced in a motion chair is equivalent to that observed in the real world.

4. Physiological changes measured in these motion sickness experiments resulted only from motion sickness, and were not due to any anomalies in the measuring equipment.

Equipment and Materials

Equipment This experiment used the following equipment:

1. A powered rotating chair, equipped with a speed control console and the following sensors:
 - (a) A Marshall Electronics Astropulse 90 blood pressure cuff for measuring blood pressure;
 - (b) Two pneumographs for measuring abdominal and thoracic respiration;
 - (c) Three photoplethysmographs for measuring skin pallor;
 - (d) An INTECH Systems DIF-STET differential stethoscope (phonosplanchnograph) for recording audible gastrointestinal mechanical activity;
 - (e) Two electrosplanchnographs for electrical measurements of the gastrointestinal tract;
 - (f) A galvanic skin response (GSR) sensor for measuring skin conductivity;
 - (g) Two electronystagmographs for measuring horizontal and vertical eye movement;
 - (h) An electrocardiograph for measuring heart rhythm;
 - (i) Five electroencephalograph channels for measuring brain wave activity;
 - (j) A ballistocardiograph for measuring the force of cardiac motions.
2. Recording instrumentation, consisting of:
 - (a) A SOLTEC model 8K26 series 16-channel strip chart recorder;

- (b) A Kyowa Dengyo RTP-610A 14-channel Beta tape recorder;
 - (c) A Zenith 248 personal computer for real-time and statistical data analysis.
3. Commercial software, including:
- (a) DATAQ Instruments' CODAS for digitizing and displaying waveforms;
 - (b) MacMillan Software's Asystant for numerical and statistical analysis;
 - (c) Barron Associates' Abductive Reasoning Mechanism (ARM) software for developing mathematical models using the collected motion sickness data.
4. Other equipment, consisting of:
- (a) A 16-channel low pass filter bank constructed by Dr. Chelen;
 - (b) A Spiropet pocket spirometer;
 - (c) A Cyborg Thermal P642 digital thermometer;
 - (d) A wireless FM microphone for recording a test subject's symptom reports;
 - (e) An Ace elastic bandage and manual blood pressure cuff for pallor calibration;
 - (f) A portable tape recorder, used to play a tape directing the test subject's head movements during each experiment;
 - (g) A motion sickness bag.

Materials Materials used in this research effort included:

1. The anticonvulsant drug phenytoin;
2. Disposable Medtronic Medical "Huggables" Infant Monitoring Electrodes for use as electronystagmograph electrodes;
3. Disposable ConMed Adult ECG Electrodes for use as electrocardiograph and electrosplanchnograph electrodes;

4. Alcohol pads for cleaning a test subject's skin prior to the placement of the electrodes;
5. Platinum subdermal electrodes for use as electroencephalograph electrodes;
6. Beta format video tapes for 14-channel instrument recordings;
7. Floppy diskettes, used with software for data analysis;
8. Subject questionnaires and histories.

Other Support

Dr. William Chelen (M.D., B.S.E.E.) was a key member of this research team. He provided the necessary "corporate knowledge" for the effort, since he has been continuously involved with the AFIT motion sickness research program since 1985. He constructed most of the physiological sensors used with the motion chair, including the very special extended frequency range EEG amplifiers. He was responsible for screening potential test subjects. Finally, he monitored the physical well-being of the volunteers during and after each experiment.

II. Experimental Procedure

The procedure used in this experiment was designed to gauge the effectiveness of the drug Dilantin (generically known as phenytoin) in preventing and treating motion sickness. It also provided data useful in developing mathematical models relating the levels of motion sickness experienced by a test subject with the biophysiological data collected on that individual during the course of an experiment.

The procedure consisted of three parts - the screening of potential volunteers, the actual induction of motion sickness, and the collection of data.

Screening

The screening phase eliminated those potential test subjects having abnormal reactions to motion, or whose health could have suffered as a result of participation in the experiment. It included a *motion susceptibility trial*, medical history interviews, and physical examinations.

The susceptibility trial consisted of a ride in AFIT's powered rotating chair at 12 to 20 rpm for several minutes. The chair is located in an air-conditioned environment. An audio tape player directed the potential test subject to perform a sequence of head tilts (i.e., right, left, or forward) as the chair was spinning. The test subject was periodically asked by the experimenters to report his symptoms, and to rate his sense of physical well-being on a scale of 1 (normal) to 10 (emesis). Dr. Chelen was present to monitor the health of the test subjects during all of the susceptibility trials.

Once an individual was determined to have a normal susceptibility to motion sickness, he was then asked to complete a questionnaire on his past experiences with motion sickness. A personal and family medical history form was also filled out to identify "familial or genetic disorders and evidence of chronic and systemic disease" (5:12).

After a screening physical examination by Dr. Chelen, all test subjects were then sent to the Wright-Patterson AFB hospital for a complete blood count (CBC). They also underwent a general battery of blood biochemistry, blood lipids and cholesterol testing, urinalysis, and liver function studies to minimize the possibility of blood or kidney disease (5:12).

The volunteers then had their physical performance and cognitive abilities tested. This information provided the baseline data for comparison with later performance-cognition tests, to evaluate any possible side effects resulting from the drug treatment. The tests used were developed by the Air Force Aerospace Medical Research Laboratory, and were implemented as personal computer software. They consisted of a probability monitoring task to test visual perception, a grammatical reasoning task to measure reasoning ability, and an unstable tracking task to test manual response speed and accuracy (31:11).

The screening phase of the experiment concluded with individual meetings between Dr. Chelen and the accepted test subjects. Dr. Chelen informed each volunteer as to what he would experience during the actual experiment and answered any questions. Each subject then received a Subject Consent Form which described the experiment in writing. (The consent form emphasized that the volunteer could withdraw from either the study or an individual motion chair ride at any time.)

Induction of Motion Sickness

The drug Dilantin was administered in a double blind, placebo-controlled crossover technique. Each test subject passing the screening phase was given either the active agent phenytoin, or a dextrose placebo, at two different times at least one week apart:

Each treatment will be administered in unmarked capsules in two lots of five capsules, lot A and lot B. Each subject will be issued a complement of each lot. The identity of each lot, either phenytoin or placebo, will be known only to the principal investigator (Dr. Chelen). The subject will

randomly decide the order in which he will take a single lot prior to each of the two experimental sessions and not reveal until after both sessions what the order was (5:7).

Phenytoin or placebo treatment began the day before each motion chair ride. The test subjects would normally take their first capsule in the late afternoon, with the chair rides usually occurring 24 hours later. The remaining four capsules would be ingested as follows - one with dinner, one with a snack before going to bed, one with breakfast, and one with lunch.

Each instrumented chair ride began with Dr. Chelen asking the test subject whether he had experienced any adverse side effects after ingesting the drug doses. Dr. Chelen then performed a physical examination on the volunteer to determine his ability to participate in the experiment and to also verify the absence of drug-related side effects. If no problems were observed, the volunteer was directed to retake the performance-cognition tests.

Various biophysiological sensors were then attached to the test subjects and calibrated. Pallor was calibrated by first removing blood from the volunteer's hand using an elastic wrap and a blood pressure cuff, and then applying adhesive-attached sensors. The sensors were then calibrated at maximum pallor. The cuff was released after several minutes, enabling the sensors to be calibrated at maximum perfusion. Adhesive silver-silver chloride surface and subdermal electrodes were applied to the abdomen, trunk, and head after those areas had been cleaned with alcohol pads. After the pneumographs and other sensors were attached, the subject was assisted into the motion chair. The volunteer then breathed into a spirometer to calibrate his respiratory volume, and had his eyelids taped shut to eliminate visual references and blinking during the chair ride. Blood would be drawn from the subject during this time, in order to measure his phenytoin serum levels.

The experiment began with the chair being rotated at a velocity between 12 and 20 rpm. (The specific speed was based on the duration of the test subject's

susceptibility trial, in an attempt to have the experiment last between 10 and 30 minutes (3).) The test subject's head movements were again directed by the audio tape player. Dr. Chelen constantly monitored the volunteer's physical state, and the subject was regularly asked about his symptoms. The experiment continued until motion sickness symptoms had fully evolved into emesis.

After the volunteer had experienced frank motion sickness to emesis, the motion chair was decelerated. Although the chair soon stopped, the subject remained sitting until his physiological indicators had stabilized to pretest levels. All electrodes and sensors were then removed, and the subject interviewed about his test experiences. Finally, with Dr. Chelen's permission, the subject was released.

Data Collection

A Kyowa Dengyo 14-channel FM recorder was used to collect the biophysiological data on FM tape. A SOLTEC model SK26 series 16-channel strip chart recorder was used to make a paper recording of some of the data. A Zenith 248 computer was used to digitize and analyze the data, while a 16-channel active filter bank was included in the data collection circuit to eliminate 60 Hz noise.

Data were collected without affecting the test subjects' right to privacy:

All test data will be associated with the subjects' name, but any publication of the data will not reveal the name or any other information about the subject. Data will not be available to anyone but the investigators (5:11).

III. Motion Sickness Models Using 1986 and 1987 Data

AFIT motion sickness researchers have continually tried to develop a mathematical model that would predict a test subject's level of motion sickness to the biophysiological data collected during a motion chair ride. Such a model could be useful in attempts to treat motion sickness through biofeedback. It might also provide insight into the physical nature of motion sickness.

The current research effort into an effective motion sickness model began with a review of previous AFIT representations. Nonlinear mathematical models using data from 1986 and 1987 were then developed with the aid of commercial software.

History of AFIT Motion Sickness Models

From the beginning, AFIT researchers have recognized the value of mathematical models in motion sickness research. In 1983, Earl and Peterson noted the necessity for

formulating a method of calculating a weighted sum of the relevant physiological measurements to determine a so-called 'motion sickness factor'. (This quantity will show the advancement of motion sickness symptoms and may need to be adjusted for each individual being trained.) (12:I-2)

A year later, Fitzpatrick, Rogers, and Williams recommended searching for "predictive relations" in the collected data (13:6-3).

In 1985, Jarvis and Uyeda made the first steps towards developing a mathematical model describing the evolution of motion sickness during the course of an experiment. First, they determined that "two distinct types of individuals" could be distinguished in terms of motion sickness - i.e., nonsusceptible and susceptible subjects (22:105). Second, they noted the following trends with respect to susceptible individuals:

1. A moderate or rapid increase in facial pallor, usually after a slight flushing, to a level of 8% to 20% over the baseline value.
2. Generally, a steady increase in GSR to a level of 12% to 16% over baseline.
3. A significant intestinal activity, usually seen as a 600% to 700% increase in EIG amplitude.
4. A large increase in intestinal activity, seen as a several-fold jump in EGG amplitude (22:105).

Although Jarvis and Uyeda realized the importance of spotting trends in motion sickness data, their conclusions were questionable. As Hartle noted later

The two major problems that could probably lead to errors in their data analysis was the small sample sizes used and the constant changing of experimental protocol ... Because of the evolution of the protocol, there was never really enough standardized test data results to provide a valid statistical analysis. A third problem was the constant changes in the experimental environment caused by the effects of weather and the defective air conditioning and heating system ... (21:28).

Also, their findings concerning intestinal activity were later invalidated when their EIG and EGG measurements were found to be inaccurate (3).

1986 Motion Sickness Equations

In 1986, the AFIT research team of Hartle, McPherson, and Miller each developed motion sickness equations. They hypothesized a linear relationship between a volunteer's level of motion sickness and the biophysiological data collected from a subject during the course of an experiment (21:91; 27:91). As a result, all three researchers used multivariate statistics to develop equations relating a dependent variable (Y, or the symptom level) to a linear combination of independent variables (the biophysical parameters). Hartle's equation was (21:97)

$$\begin{aligned}
 Y = & 595.55 - 0.2268(thor) + 0.1540(fing) \\
 & -0.6581(GSR) + 2.8497(heart) \\
 & +0.2624(temp) - 100.8295(breaths)
 \end{aligned}
 \tag{1}$$

where

Y = "the level of motion sickness experienced by the test subject" (21:98)

thor = thoracic volume (cc)

fing = finger pallor (percent flush)

GSR = galvanic skin response (Kohms resistance)

heart = heart rate (beats per minute)

temp = temperature (degrees Fahrenheit)

breaths = number of breaths per 10 second interval.

McPherson's equation was (25:59)

$$\begin{aligned} Y = & 0.9358 + 0.0095(Thoracic) + 0.1465(Finger) \\ & -0.0004(GSR) + 0.0334(EKG) + 0.2449(Temp) \\ & +0.3696(Breath) \end{aligned} \quad (2)$$

where "the variable Y indicates the possible range of symptom numbers" (25:59).

Miller's equation was (26:84)

$$\begin{aligned} ABS(Y) = & -69.3938 + .0634(t) - .3512(f1) \\ & +.4514(f2) - .00000627(g) - .0179(e1) \\ & -.2006(e2) + .5518(e3) \end{aligned} \quad (3)$$

where

$ABS(Y)$ = the absolute value of the level of motion sickness to be predicted

t = thoracic respiration

$f1$ = finger pallor

$f2$ = facial pallor

g = galvanic skin response

$e1$ = eig

$e2$ = egg

$e3$ = ekg.

The major criticism of the 1986 motion sickness equations is that their creators arbitrarily assumed a linear relationship between the biophysiological parameters

and the evolution of motion sickness. Linear equations have been used to accurately predict who will get motion sick in a real environment, based on experimental data (30). However, a linear relationship will not explain why several AFIT motion sickness test subjects have experienced the "avalanche phenomenon" - a slow evolution of motion sickness, followed by an extremely rapid growth of the illness until emesis. In fact, even Miller noted the "avalanche effect" in some of his test subjects (26:67). McPherson himself admitted that "Even though a linear relationship has been formulated, non-linear techniques should also be explored to determine if a better fitting model is possible" (25:66). Drylie, a member of the 1987 AFIT research team, also recognized the need for a nonlinear motion sickness research model (11:21).

Questions arise concerning the size and validity of the data base used for the 1986 motion sickness equations. Data were collected on only 12 volunteers, and the parameters measured varied from subject to subject (25:85-170).

The GSR data used by the 1986 researchers appear to be incorrect. Hartle stated that "The trend indicates that the subject's resistance decreases as he becomes motion sick" (21:68). This conclusion agrees with the findings of other investigators (11:35; 22:81; 31). However, as Drylie noted a year later, "By examining the 1986 GSR data, it is seen that for some subjects the skin resistance appears to remain the same, or in some instances to increase, while a subject is getting sick and/or sweating" (11:19). In addition, Drylie demonstrated that Miller's equation gave "a slightly better estimation of a person's level of motion sickness" when the GSR data were omitted (11:18).

Other data used in the 1986 motion sickness equation also seem suspect. Both Hartle and McPherson used surface skin temperature data in their formulas. However, since the motion sickness experiments took place in the summer in a room that was not air-conditioned, changes in skin temperature resulting from the experiment could have been overshadowed by the room temperature. When discussing the surface skin temperature data, McPherson noted that "Caution should be taken in

accepting this finding since the experiment was run under less than perfect environmental conditions" (25:36). McPherson and Miller also used heart rate data in their equations, despite a controversy among other researchers as to whether a correlation exists between heart rate change and the evolution of motion sickness (8; 20).

Miller's equation comes in for some additional criticism. He used EIG and EGG data in his formula, but the validity of those data so concerned the 1987 AFIT motion sickness research team that they changed the equipment used to measure intestinal activity (3). Miller also relied on facial pallor data, even though those data were collected on only three subjects (25:85-170). Drylie criticized the 1986 facial pallor data on technical grounds (11:12). First, the red LED used in the 1986 photoplethysmograph was not sensitive to changes in skin pallor. Second, "the adhesive used to hold the sensors in place irritated the skin of many subjects, masking any skin color change due to blood flow change" (11:12).

1987 Mathematical Models

Captain Fix, a member of the 1987 AFIT motion sickness research team, modeled the development of motion sickness with an equation as well as a neural network simulation.

Both the equation and the neural net used the same experimental data. The parameters used were "electrosplachnogram (ESG), electronystagmogram (ENG), thoracic respiration pneumogram (Resp), galvanic skin reflex or skin resistance (GSR), and peripheral skin temperature (Temp)" (14:14). The data were recorded on a Kyowa Dengyo data recorder, and then digitized on a Zenith 248 personal computer using CODAS software (14:14).

The experimental results were processed before being used. The data were first converted to volts, and the GSR and peripheral skin temperature data then calibrated. The GSR, peripheral skin temperature, and thoracic respiration pneu-

mogram data were further processed as (14:16-17)

$$GSR_P = 1 - GSR/GSR_B \quad (4)$$

$$Temp_P = ABS(Temp - Temp_B) \quad (5)$$

$$Resp_P = RMS \text{ } Resp / RMS \text{ } Resp_B \quad (6)$$

where the B subscript described the parameter data during "the asymptomatic period just prior to the start of head motions" (14:17). The ENG and ESG data were

normalized to amplifier gains of 1500 and 600 respectively - that is, if the amplifier gains were different from those values, the output voltage was multiplied by a factor. These signals were then converted to RMS signals with the averaging done over the previous 20 seconds (14:16).

Fix first developed a motion sickness equation. Commercial software (Asys-tant) was used to curve fit each biophysiological parameter with the test data for several volunteers. The resulting equations (one for each test subject) were combined into a single average equation for each parameter. Next, an equation containing all of the parameters was formed by linearly combining the average equations. The final equation was (14:23)

$$\begin{aligned} msick = & F_S(-1.446(RMSESG^2) + 12.97(RMSESG) + 1.141) \\ & + F_N(-.7414(RMSENG^2) + 5.046(RMSENG) + .592) \\ & + F_R(.1848(Resp_P^2) + 3.4587(Resp_P) - 1.537) \\ & + F_G(7.9194(GSR_P^2) + 4.8693(GSR_P) + 1.0488) \\ & + F_T(-1.4104(Temp_P^2) + 13.3469(Temp_P) + .6244) \end{aligned} \quad (7)$$

where

msick = the test subjects' computed level of sickness.

F_S , F_N , F_R , F_G , and F_T are coefficients calculated as follows

1. For each biophysiological parameter

$$StDev = \left(\frac{1}{N}\right) \left(\sum_{i=1}^N\right) (ABS(10 - y_i)) \quad (8)$$

where

N = the number of equations combined in the average equation for that parameter, and

y = the output from the equations fitted for each test subject for that parameter, when the input was the "input parameter value that yielded a report value of 10 from the average equation" (14:21).

2. Each $StDev$ value was then normalized

$$StDev_N = 10/StDev \quad (9)$$

If the average equation did not have an output value of 10, its maximum reported value was substituted for the number 10 in equations (8) and (9).

3. A figure of merit was then derived

$$FOM = (StDev_N)/(R^2) \quad (10)$$

where " R^2 " is calculated during the curve fitting of the parameter with each test subject's data, and is a measure of the reliability of that fitting. (Fix noted that the FOMs ranged from 0.8653 to 4.9116 for all input parameters except the facial photoplethysmograph value, which had a FOM of 136.1 (14:21).)

4. After FOMs for all the parameters were derived, a relative FOM was then obtained for each parameter

$$FOM_R = FOM/FOM_A \quad (11)$$

where " FOM_A " is the average of FOMs for all the parameters to be used in the final composite equation (14:22).

5. Finally, a coefficient for each parameter was then calculated

$$F = FOM_R/K \quad (12)$$

where "K is the number of individual parameters used in the composite equation" (14:22). F_S has been reported to have a value of 0.1569, with $F_N = 0.1359$, $F_R = 0.1964$, and $F_G = 0.4344$ (15).

Fix also used a neural network to define a relationship between a subject's level of motion sickness and the biophysiological data collected for that subject. He simulated a multilayer perceptron on a conventional digital computer, using C source code

The net simulation tested here consisted of 5 inputs, 10 nodes in the next, or first hidden layer, 40 nodes in the second hidden layer, and 10 outputs. The inputs were the same preprocessed physiological data used for the equation model. The outputs correspond to the sickness levels from 1 to 10 reported by the subjects (14:37).

Questions also arise concerning the validity of some of Fix's data. The 1987 galvanic skin reflex or skin resistance (GSR) data appears suspect after reading the following passage from Drylie

The skin resistance measured by the GSR sensors and circuitry has dropped below zero for several subjects this year, but only during the experiment. As this data was obviously in error, the GSR circuitry was modified. Experiments need to be completed with the modified circuitry to determine if the problem has disappeared (11:53).

The 1987 AFIT motion sickness research team, like their counterparts a year earlier, also faced environmental problems caused by a lack of air conditioning in the room containing the motion chair. As Drylie noted, "it was sometimes possible to maintain the temperature in the ideal 22 to 24 degree Centigrade (71 to 75 degree Fahrenheit) range" (11:11). This lack of complete success in controlling the ambient room temperature could have affected the collection of data on the peripheral skin

temperature and other parameters. (The 1988 AFIT research team observed that an elevated ambient temperature in the room containing the motion chair would lead to increased sweating by the test subjects, and result in the body electrodes falling off.) Finally, Fix relied on electronystagmograph (ENG) data. However, the changes in ENG signal strength could have been a sweat artifact (3).

Whatever the cause, Fix's motion sickness equation appears to have been less than 100% accurate. For example, while the test subjects' reported levels of motion sickness ranged from 0 to 10, Fix's "msick" equation had outputs from 0 to about 12 (14:23). In a questionable attempt to improve his equation's reliability, Fix turned to a correcting function, whimsically called "fudge"

One remaining problem: since this equation is an average of several subjects, it may not fit an individual subject. This problem is partly alleviated by using several parameters in the equation, but not completely. Therefore, there is a provision to multiply the output by a constant factor returned by the function fudge(). If the program consistently displays a number that is greatly different from the subject's reports, the operator types the number the subject is reporting. Fudge() then computes a factor that will move the indicator half the distance from the computed value toward the reported value (14:31-32).

Fix found that, on the basis of average absolute errors, the neural net worked about as well as his motion sickness equation (Table 1).

Table 1. Capt Fix's Results - Average Absolute Errors for Neural Net and Motion Sickness Equation (14:44)

Subject	1	2	3	4
Equation	0.9242	1.7651	2.9500	0.7724
Net	0.8100	1.0112	2.9263	0.8956

Unfortunately, Fix did not describe how he calculated average absolute error. Also, these data indicate that Fix tested his model on only four sets of test data. Fix discussed the performance of his neural net

the net could measure the training data sickness level with an average of 0.86. In a very few cases, however, did the correct output node exceed 0.5. In most cases, the values of all the nodes were small, with a peak in the 0.15 to 0.3 range in the neighborhood of the correct response. This poor definition indicates the output regions were not well separated. This may be due to the nature of the data, or the problem may lie with the neural net construction (14:38).

He also suggested improvements to his neural net

The number of nodes in the hidden layers determines the number of output classes (1 through 10) and the number of regions (subject types) per class the net can separate ... Therefore, increasing the number of nodes in those layers might allow the net to separate the report levels better, and incidentally improve the output definition. This approach, however, would increase the training requirement, and the computation time in the indicator program ... It is possible, however, that the net described here is not large enough to handle the problem adequately and that a large enough simulation may not run in real time. A hardware implementation may be required (14:40-41).

This summary of previous work is not intended to denigrate the efforts of the earlier research teams, but rather to emphasize the great difficulty of collecting significant, reliable, and reportable physiological data from highly stressed subjects.

1988 Motion Sickness Models

The 1988 AFIT motion sickness research team decided to model motion sickness using Abductive Reasoning Mechanism (ARM) software provided by Barron Associates (7). This set of computer programs relies on the concepts of abduction and polynomial network theory.

Abductive reasoning can be distinguished from both inductive and deductive reasoning. Abduction has been defined as "the act or process of reasoning from a set of general principles to particulars or other general principles under certainty" (28:22). Thus, it works in the same manner as deduction, and in the reverse direction of inductive reasoning, since the latter goes "from a set of principles and

particulars to general principles" (28:22). Deduction requires that a relationship must exist among separate sets of data. Abduction, on the other hand, needs only a "consistency of the relationships among independent observations of the data"; the exact nature of the relationships can be unknown (28:22). The syllogism is the basic form of deductive reasoning, while abductive reasoning uses abductive functions. An abductive function can be defined as "any function representing the relationships of a set of input variables to a set of outputs" (28:22).

The ARM software relies on an Algorithm for Synthesis of Polynomial Networks (ASPN). ASPN will model any function as a layered network of polynomial elements. This process is in conformance with Kolmogorov's representation theorem, which has been restated by A.R. Barron as

four-layer networks can represent any function provided elements are allowed which implement arbitrary continuous functions of one variable as well as elements which simply implement the sum of several variables (28:34).

The network created by ASPN is called an abductively-synthesized polynomial network or abductive polynomial network (APN) with "each node in the network representing a polynomial equation with the coefficients and network connectivity learned" (28:36). The APN is synthesized after "a tradeoff between model complexity and accuracy, with the assumption that model simplicity will improve the likelihood of closely fitting unseen (new) data" (28:36).

When the ARM software was applied to the 1986 motion sickness data (see Appendix A), the following nonlinear equation was derived relating the level of motion sickness to some of the biophysiological data collected

$$\begin{aligned} level = & -37735.4 + (825.223)(f) + (1668.13)(s) + (4.79697)(t) \\ & +(7.93174)(d) - (37.28)(f)(s) - (27.755)(s^2) \end{aligned}$$

$$\begin{aligned}
& -(0.104811)(f)(t) - (0.103339)(s)(t) + (0.000768)(t^2) \\
& -(0.173303)(f)(d) - (0.171666)(s)(d) - (0.030486)(t)(d) \\
& +(0.000309)(d^2) + (0.633866)(f)(s^2) \\
& +(0.20604)(s^3) + (0.002363)(f)(s)(t) \\
& +(0.000556)(s^2)(t) - (0.000017)(f)(t^2) \\
& -(0.000008)(s)(t^2) - (0.000001)(t^3) \\
& +(0.003924)(f)(s)(d) + (0.000929)(s^2)(d) \\
& +(0.000666)(f)(t)(d) + (0.000652)(s)(t)(d) \\
& -(0.000007)(f)(d^2) - (0.000003)(s)(d^2) \\
& -(0.004809)(f)(s^3) - (0.000576)(s^4) \\
& -(0.000013)(f)(s^2)(t) - (0.000022)(f)(s^2)(d) \\
& -(0.000015)(f)(s)(t)(d) - (0.000003)(s^2)(t)(d) \\
& +(0.000014)(f)(s^4)
\end{aligned} \tag{13}$$

where

level = symptom level

f = finger photoplethysmograph data

s = surface skin temperature data

t = thoracic respiration data

d = diaphragmatic respiration data.

The software discarded the 1986 ekg and GSR data as being unimportant. Modeling statistics are shown in Table 2. Figure 1 is a comparison of the actual symptom levels with the symptom levels computed by entering the data from Appendix A into equation (13).

The ARM motion sickness model developed using the 1986 data was only moderately successful in predicting motion sickness levels. The ARM values were within one level of the reported symptom levels for 60% of the observations, and

Table 2. Modeling Statistics for Equation (13)

Variable	Mean	Sigma	Min	Max
level	5.30	3.23	1	10
finger	71.51	16.15	25	95
surface	91.11	4.91	79.8	97.8
thoracic	244.38	124.52	67	600
dia	219.89	182.74	50	950

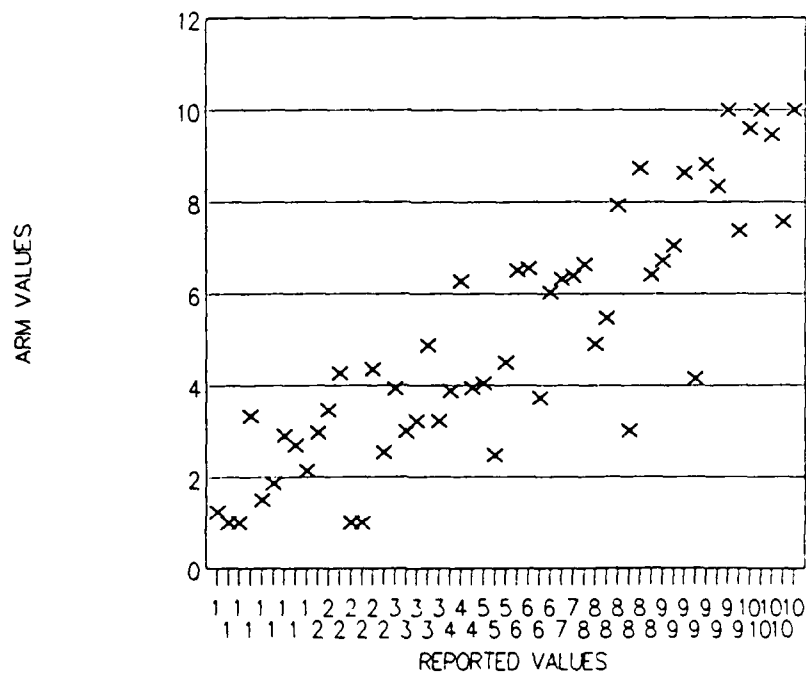


Figure 1. Modeling of 1986 Data

within two levels of the reported symptom levels for 77% of the observations. The poor results may have been due to the possible unreliability of some of the 1986 data. (An attempt was made to use McPherson's (eqn (2)), Hartle's (eqn (1)), and Miller's (eqn (3)) equations with the data used to develop the ARM model. However, Miller's equation could not be tested because McPherson did not provide any eig or egg data in his Appendix B (25:85-170). McPherson's and Hartle's equations were not tested because of an uncertainty as to whether the "Breath" variable in each of their equations could use the respiration data in McPherson's thesis.)

The ARM software was then applied to the 1987 motion sickness data. The data consisted of the first 100 data vectors from Appendix G ("Neural Net Training Data Set") of Fix's thesis (14). The following nonlinear equation was derived relating the level of motion sickness to some of the biophysiological data collected

$$\begin{aligned}
 level = & -3.24664 + (14.5679)(esg) + (14.5334)(eng) \\
 & -(1.07237)(temp) - (20.2803)(esg^2) \\
 & +(2.47056)(esg)(eng) - (8.51878)(eng^2) \\
 & -(7.13422)(esg)(temp) + (2.97328)(eng)(temp) \\
 & +(8.60441)(esg^3) + (1.29536)(eng^3)
 \end{aligned} \tag{14}$$

where

level = symptom level

esg = electroplachnograph data (Note: Fix erroneously labeled the column of ESG data in his Appendix G as eig data)

eng = electronystagmograph data

temp = peripheral skin temperature data.

The software discarded the 1987 GSR and thoracic respiration data as being unimportant. Modeling statistics are shown in Table 3. Figure 2 compares the actual

Table 3. Modeling Statistics for Equation (14)

Variable	Mean	Sigma	Min	Max
level	5.69	3.10	1	10
eng	1.13	0.71	0.231485	3.04202
esg	0.32	0.32	0.010575	1.45433
temp	0.24	0.16	0.019435	0.79045

symptom levels noted in the next 50 data vectors from Fix's Appendix G with the symptom levels computed by entering that data into equation (14).

The ARM motion sickness model developed from the 1987 data seemed to indicate that the 1987 data was better than that collected a year earlier. First, the ARM model using 1987 data (eqn (14)) was simpler than the ARM model for 1986 (eqn (13)). Also, the ARM model for 1987 was much more successful in predicting motion sickness levels. The ARM values were within one level of the reported symptom levels for 64% of the observations, and within two levels of the reported symptom levels for 90% of the observations. (The results could not be compared with Fix's motion sickness equation, since his Appendix G data were intended only for his neural network.)

The next chapter describes the result of using the ARM software with the data collected during the 1988 research effort.

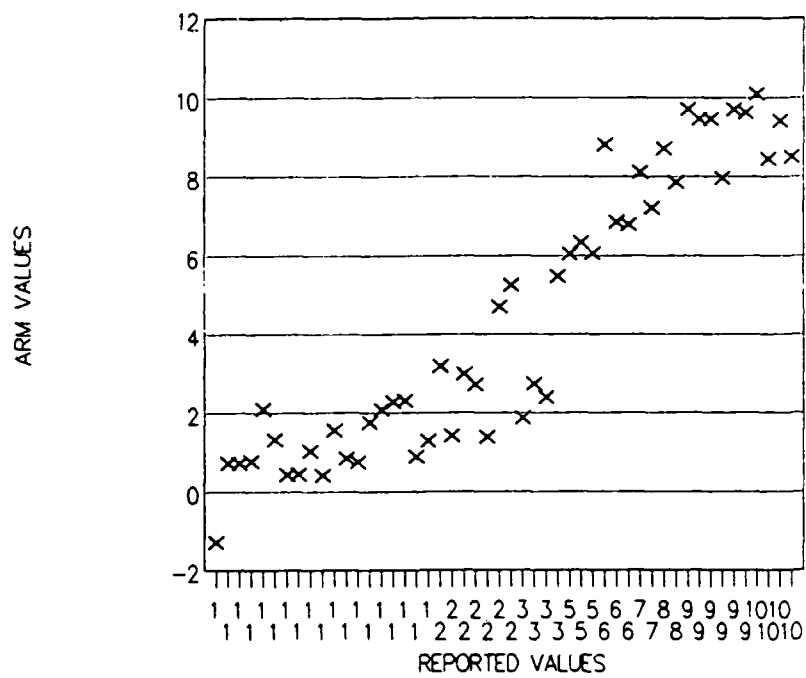


Figure 2. Modeling of 1987 Data

IV. 1988 Result - Data Analysis and Motion Sickness Modeling

Data Analysis - Procedure

At the time of this analysis, data had been collected from eleven test subjects. Major losses of data are described as follows:

1. Subject #5 did not complete either trial. After taking two doses of dilantin, the subject experienced a skin rash that evening. After consulting with Dr. Chelen by telephone, he discontinued treatment. The next morning, the subject experienced a swollen tongue and lack of coordination, and had difficulty driving his car. Fortunately, his symptoms disappeared that evening. The subject had reported on his medical history form that he was allergic to antihistamines, decongestants, and other drugs.
2. Subject #6 completed a trial only while on the placebo. He remained in the motion chair 1 hour, 59 minutes, and 58 seconds (from the beginning of head motions until emesis), even though the chair speed was increased several times from 16 rpm. (In fact, the chair reached a maximum of 32 rpm.) Subject was not given the dilantin treatment, since it appeared that he had successfully adapted to the motion chair ride and head motions while on the placebo. This unusually fast adaptation probably occurred because of the subject's history of participation in aeromedical experiments. Since the subject was not normally susceptible to motion sickness, the data from his motion chair ride were not analyzed.
3. Subject #8 completed three trials; the data from one trial were discarded. The subject remained in the chair 36 minutes and 4 seconds (beginning of head motions until emesis) while on dilantin (trial #1), and 30 minutes and 35 seconds while on the placebo (trial #2). However, lab analysis revealed that the level of dilantin in the subject's blood was so low during trial #1 as to

probably be nontherapeutic. As a result, the biophysiological data collected on the subject during trial #1 were not analyzed. The subject then consented to another motion chair ride while on dilantin. This ride was delayed about three weeks, or until the subject had lost any adaptation to the chair ride and head motions. The data collected on the subject during trial #2 (while he was on placebo) and trial #3 (dilantin) were then analyzed.

Data from both strip chart recordings and magnetic tape recordings were analyzed. In order to compare 1988 results of data analysis with 1987 results, an effort was made to use the 1987 data analysis procedure whenever possible. The current data analysis procedure was also documented in order to allow others to follow it and verify results.

The current data analysis procedure is outlined below:

1. Analysis of strip chart recordings.

- (a) Identify the following times on each strip chart recording:

- i. Control period (subject in motion chair with motion chair at rest).
 - ii. Start of chair motions.
 - iii. Start of head motions. (The data between points i and ii were not analyzed, due to a lack of control over events during this period.)
 - iv. Periods of at least one symptom level, lasting at least eight seconds, and between points iii and viii. These data were later used in the 1988 motion sickness models relating the symptom levels reported by a test subject with the biophysiological data collected on that subject.
 - v. Periods of M I, M IIB, M IIA, and M III motion sickness, using Graybiel's criteria (19:454).
 - vi. Period of frank sickness (defined as the period just before emesis).
 - vii. Emesis.

viii. End of head motions.

ix. Ten minutes of the post-emetic period, separated into ten one-minute intervals. These data were not analyzed in this thesis, because an insufficient amount of data for these periods were collected while the subjects were on dilantin.

(b) Associate the beta numbers of the magnetic tape recordings with the time periods found during step one (using the tape addresses written on the strip chart recordings during the actual trials).

(c) Record the following data for each trial in Appendix B:

i. Time periods found during step (a).

ii. Tape addresses found during step (b).

iii. Symptom levels reported by test subject (from strip chart recording).

2. Analysis of magnetic tape recordings.

(a) Electrocardiograph data. Data were first converted to digital format at 100 samples/second using CODAS software. R to R periods in the EKG records were measured for the periods desired. The average of the R to R periods was then found for each period desired. The reciprocal of this average, which was the average heart rate in beats per minute, was then found for each period desired. Table 4 presents the results for subjects having ingested the placebo; Table 5 presents the results for subjects having ingested the dilantin.

(b) Galvanic Skin Response data. Data were collected for only one subject, and were therefore not analyzed.

(c) Ballistocardiograph data. Data were collected only on the analog strip chart, and were not analyzed.

(d) Phonosplachnograph data. Recordings were listened to, but not analyzed, since interpretation appeared to be quite subjective.

- (e) Abdominal respiration data. Data were not analyzed, due to "the difficulty of subtracting out the thoracic component which is detected by the abdominal sensor due to placement of the strain gauges" (11:37).
- (f) Thoracic respiration data.
 - i. The data were converted to digital format at 100 samples/sec using CODAS software. The desired periods were recorded into separate files.
 - ii. The CODAS voltages were then converted to the correct voltages using an Asystant function developed by the 1987 AFIT motion sickness research team. (Voltages recorded using CODAS have to be corrected so that they equal the voltages shown on the strip chart recordings (16).)
 - iii. The rms voltages were then calculated using the Analysis menu within the Waveform Processor section of Asystant.
 - iv. Table 6 presents the results for subjects having ingested the placebo; Table 7 presents the results for subjects having taken dilantin.
- (g) Electrosplanchnograph data.
 - i. Following Gaudreault's procedure (17:35), only the electrosplanchnograph data from the electrodes placed over the duodenum and right lower quadrant of the abdomen were analyzed. (The data from the electrodes placed over the lower stomach and left lower abdomen were similar, and therefore not analyzed.)
 - ii. The data were then converted to digital format at 100 samples/sec using CODAS software. The desired periods were recorded into separate files.
 - iii. The CODAS voltages were converted to the correct voltages using Asystant.

- iv. The rms voltages were then calculated using Asystant.
- v. Table 8 presents the results for subjects having ingested the placebo;
Table 9 presents the results for subjects having taken dilantin.

(h) Facial photoplethysmograph data.

- i. The left or right facial photoplethysmograph data collected on the Beta recorder were first converted to digital format at 100 samples/sec using CODAS software.
- ii. The data were then converted to the correct voltages using Asystant.
- iii. Following Gaudreault (17:38), the data were filtered with a low pass filter to reduce noise.
- iv. The mean voltages of each desired period were calculated using the Analysis menu within the Waveform Processor section of ASYSTANT. Mean voltages that were increasingly negative would indicate a trend towards increasing pallor (increasing flush), while mean voltages that were increasingly positive would indicate a trend toward increasing pallor (decreasing flush). Mean voltages were used because 0% and 100% pallor calibration were either faulty (i.e., test values falling outside the calibration ranges) or nonexistent.
- v. Table 10 presents the results for subjects having ingested the placebo;
Table 11 presents the results for subjects having taken dilantin.

(i) Peripheral photoplethysmograph data.

- i. The peripheral photoplethysmograph data were analyzed in the same manner as the facial photoplethysmograph data.
- ii. Table 12 presents the results for subjects having taken the placebo;
Table 13 presents the results for subjects having taken dilantin.

- (j) Vertical electronystagmograph data. (Only the vertical electronystagmograph data were analyzed, since it were analyzed in 1987, and "were found to be a good indicator of motion sickness." (11:45).)
 - i. The data were converted to digital format at 100 samples/sec using CODAS software.
 - ii. The data were then converted to the correct voltages using Asystant.
 - iii. Following Drylie (11:44), the rms voltages were then calculated using Asystant.
 - iv. Table 14 presents the results for subjects having taken the placebo; Table 15 presents the results for subjects having taken dilantin.
- (k) Electroencephalograph data. Data were collected on both analog and strip chart and FM recorder. The data were not used in the 1988 motion sickness model for two reasons:
 - i. The EEG data at different motion sickness levels were not as easily comparable as other biophysiological parameters.
 - ii. The successful application of the Abductive Reasoning Mechanism (ARM) software to the 1987 data, when that data did not include EEG data.

Results of Data Analysis

Electrocardiograph data While on the placebo, the subjects had their average heart rates increase as a result of motion sickness. The highest average heart rate (85 beats per minute) was at the M I motion sickness level, and was probably due to anxiety about the motion chair ride. After decreasing from the M I level, the average heart rates then increased an average of 10.8% when the subjects went from frank sickness to emesis. The 1987 AFIT motion sickness researchers also reported an increase in heart rates as motion sickness evolved (17:28).

The subjects' average heart rates followed a different pattern while they were on dilantin. (However, there are less heart rate data for the subjects on dilantin because many of them did not experience all levels of motion sickness.) The heart rates would again increase at the M I level. As the subjects became acclimated to the ride, the heart rates returned to the baseline average and remained there until frank sickness. The heart rates then increased an average of 9.7% when the subjects went from frank sickness to emesis.

Tables 4 and 5 describe the heart rate data collected.

Table 4. Heart Rates of Subjects Taking Placebo (beats/minute)

Subject Number	Control	MI	MIIB	MIIA	MIHI	Frank Sickness	Emesis
1	75	82	88	76	85	86	85
2	86	85	78	78	93	90	87
3	Data not analyzed - no control data on FM tape.						
4	66	70	79	71	62	59	78
5	Data not collected - subject didn't ride.						
6	Data not analyzed - subject adapted to chair ride.						
7	56	91	80	75	82	69	91
8	66	88	75	76	75	57	71
9	67	87	70	92	92	87	76
10	99	105	93	77	96	81	103
11	60	70	70	72	75	64	65
Avge	72	85	79	77	83	74	82

Thoracic respiration data The subjects' thoracic respiration rates, as measured in rms voltages, increased with the degree of motion sickness experienced by the test subjects. This trend occurred whether the subject was on dilantin or the placebo. (In fact, the average rms voltages for the two sets of trials were very similar. For subjects on the placebo, the average rms voltage at emesis was 290% of the average control rms voltage; for subjects taking dilantin, the average rms voltage at emesis was 275% of the average control rms voltage.) In 1987, Drylie had also noted that

Table 5. Heart Rates of Subjects Taking Dilantin (beats/minute)

Subject Number	Control	MI	MIIB	MIIA	MIHI	Frank Sickness	Emesis
1	Data not collected - equipment failure.						
2	70	85	74	81	83	76	100
3	86	69	69	NA	NA	NA	NA
4	68	63	69	NA	NA	NA	NA
5	Data not collected - subject didn't ride.						
6	Data not collected - subject didn't ride.						
7	64	71	74	66	62	78	69
8	62	70	60	63	-	65	75
9	57	59	NA	NA	NA	NA	NA
10	91	107	ND	ND	ND	ND	ND
11	61	87	69	67	64	67	70
Avge	70	76	69	69	70	72	79

Note: NA = Not applicable (subject never achieved sickness level)

ND = No data on FM tape for that period

"motion sickness was well related" to the respiratory rms voltages when the rms levels were divided by the control rms voltage (11:41). Tables 6 and 7 describe the thoracic respiration data collected.

Electrospplanchnograph data The electrospplanchnograph values, as measured in rms voltages, also increased with the evolution of motion sickness. This trend occurred whether the subjects were taking dilantin or the placebo. The results agreed with those reported in 1987 by Drylie and Gaudreault (11:43; 17:35).

Tables 8 and 9 describe the electrospplanchnograph data collected.

Facial and Peripheral Photoplethysmograph data The facial and peripheral photoplethysmograph data appeared to be invalid. They suggested that the subjects' skin color would become flushed during the course of an experiment, and regardless of whether the subject was on dilantin or the placebo. These results contradict actual experimental observations that the subjects' skin color towards pallor during

Table 6. Thoracic Respiration Data of Subjects Taking Placebo (V rms)

Subject Number	Control	MI	MIIB	MIIA	MIH	Frank Sickness	Emesis
1	0.211	0.428	0.332	0.464	0.304	0.641	1.100
2	0.630	0.635	2.010	0.818	2.380	1.510	2.480
3	Data not analyzed - no control data on FM tape.						
4	0.291	0.533	0.610	0.673	0.489	0.743	1.110
5	Data not collected - subject didn't ride.						
6	Data not analyzed - subject adapted to chair ride.						
7	0.187	1.540	0.898	1.120	1.050	0.922	1.300
8	0.261	0.838	0.687	0.616	0.523	0.591	0.793
9	0.170	0.366	0.706	0.533	0.242	0.549	0.263
10	0.192	0.275	0.577	0.419	0.486	0.498	0.901
11	0.374	1.100	0.881	0.443	0.487	0.614	1.090
Avge	0.290	0.714	0.838	0.636	0.745	0.759	1.130

the evolution of motion sickness.

The unreliability of the photoplethysmograph data may have been due to several reasons. The calibration procedure used in the experiments seems faulty, since the voltage values calculated for many of the symptom level periods ended up being lower than the voltage value calculated for 0% pallor (100% flush). During one motion chair ride, the facial photoplethysmographs were observed to respond to variations in the background lighting as the test subject's face was periodically exposed to an overhead light. Finally, the peripheral photoplethysmograph was designed for only one size of finger. Some test subjects complained of a tight fit, and the quality of the peripheral photoplethysmograph data collected on them may have suffered as a result.

Tables 10 and 11 describe the facial photoplethysmograph data collected, while Tables 12 and 13 contain the peripheral photoplethysmograph data.

Table 7. Thoracic Respiration Data of Subjects Taking Dilantin (V rms)

Subject Number	Control	MI	MIIB	MIIA	MIII	Frank Sickness	Emesis
1	Data not collected - equipment failure.						
2	0.599	2.250	2.300	2.730	3.120	3.230	3.110
3	0.467	0.467	0.533	NA	NA	NA	NA
4	0.344	0.246	0.193	NA	NA	NA	NA
5	Data not collected - subject didn't ride.						
6	Data not collected - subject didn't ride.						
7	0.453	0.417	0.888	0.672	0.830	1.190	1.030
8	0.267	1.410	0.747	0.921	NA	0.863	1.360
9	0.132	0.155	NA	NA	NA	NA	NA
10	0.290	0.137	0.775	0.523	0.571	0.301	0.555
11	0.505	0.332	1.210	0.503	0.873	1.470	1.100
Avge	0.382	0.677	0.949	1.070	1.349	1.411	1.431

Note: NA = Not applicable (subject never achieved sickness level)

ND = No data collected, due to equipment failure

Table 8. Electrospinalnograph Data of Subjects Taking Placebo (V rms)

Subject Number	Control	MI	MIIB	MIIA	MIII	Frank Sickness	Emesis
1	0.062	0.171	0.036	0.049	0.064	0.340	0.095
2	0.038	0.055	0.043	0.099	1.060	0.298	0.316
3	0.075	0.073	0.074	0.181	0.632	0.295	0.898
4	0.123	0.242	0.830	1.160	ND	ND	ND
5	Data not collected - subject didn't ride.						
6	Data not analyzed - subject adapted to chair ride.						
7	Data not collected - equipment failure.						
8	0.023	0.124	0.105	0.259	0.320	0.557	0.503
9	0.362	0.061	0.100	0.143	0.099	3.090	3.650
10	0.035	0.071	0.247	0.631	0.680	0.694	0.413
11	0.048	0.047	0.039	0.059	0.029	0.048	0.041
Avge	0.096	0.105	0.184	0.323	0.412	0.760	0.845

Note: ND = No data, due to equipment failure

Table 9. Electroplanchnograph Data of Subjects Taking Dilantin (V rms)

Subject Number	Control	MI	MIIB	MIIA	MIHI	Frank Sickness	Emesis
1	Data not collected - equipment failure						
2	0.036	0.050	2.230	0.135	0.125	0.083	0.770
3	0.019	0.035	0.031	NA	NA	NA	NA
4	Data not collected - equipment failure.						
5	Data not collected - subject didn't ride.						
6	Data not collected - subject didn't ride.						
7	0.041	0.256	0.082	0.033	0.036	0.043	0.049
8	Data not collected - equipment failure.						
9	0.032	0.038	NA	NA	NA	NA	NA
10	Data not collected - equipment failure.						
11	0.020	0.098	0.111	0.100	0.253	1.470	0.745
Avg	0.030	0.095	0.613	0.089	0.138	0.532	0.521

Note: NA = Not applicable (subject never achieved frank sickness)

Table 10. Facial Photoplethysmograph Data of Subjects Taking Placebo (V)

Subject Number	Control	MI	MIIB	MIIA	MIHI	Frank Sickness	Emesis
1	-1.03	-1.61	-1.42	-1.44	-1.50	-1.51	-1.31
2	0.52	-0.29	0.21	0.35	0.20	-0.73	-0.38
3	-0.46	-0.46	-0.46	-0.97	-1.41	-2.60	-2.46
4	-0.83	-0.81	-1.59	-1.85	-0.85	-0.99	-2.00
5	Data not collected - subject didn't ride.						
6	Data not analyzed - subject adapted to chair ride.						
7	0.37	-0.81	-0.48	-1.06	-0.25	-0.03	-0.27
8	1.56	-0.43	-0.51	-0.87	-1.17	-2.00	-2.16
9	-1.20	-1.31	-1.06	-0.34	-0.54	-0.67	-0.57
10	-1.16	-1.92	-1.32	-1.16	-1.57	-1.27	-1.88
11	-0.91	-0.86	-1.72	-2.02	-0.95	-1.49	-1.42
Avg	-0.35	-0.94	-0.93	-1.04	-0.89	-1.25	-1.38

Table 11. Facial Photoplethysmograph Data of Subjects Taking Dilantin (V)

Subject Number	Control	MI	MIIB	MIIA	MIHI	Frank Sickness	Emesis
1	0.76	-0.48	-0.05	-0.27	-0.64	-0.64	-0.51
2	-0.69	-1.68	-0.88	-0.91	-0.97	-0.56	-0.92
3	-0.94	-2.70	-2.07	NA	NA	NA	NA
4	-0.55	-2.21	-1.38	NA	NA	NA	NA
5	Data not collected - subject didn't ride.						
6	Data not collected - subject didn't ride.						
7	0.26	-0.53	-0.67	-0.47	-0.57	-0.40	-0.17
8	0.29	-1.17	-0.23	-0.28	NA	-1.10	-0.38
9	0.14	-2.04	NA	NA	NA	NA	NA
10	0.54	-0.07	0.09	0.42	0.63	0.32	0.03
11	0.47	-0.43	-0.14	-0.38	0.00	0.00	0.16
Avge	0.03	-1.26	-0.67	-0.32	-0.39	-0.48	-0.30

Note: NA = Not applicable (subject never achieved sickness level)

Table 12. Peripheral Photoplethysmograph Data of Subjects Taking Placebo (V)

Subject Number	Control	MI	MIIB	MIIA	MIII	Frank Sickness	Emesis
1	-1.02	-1.10	-1.02	-0.89	-0.87	-0.88	-0.99
2	0.19	-0.68	-0.07	0.25	-0.68	-1.35	-1.16
3	-0.09	-0.09	-0.09	-0.80	-1.16	-2.88	-2.89
4	Data not collected - equipment not used.						
5	Data not collected - subject didn't ride.						
6	Data not collected - subject adapted to ride.						
7	-1.79	-1.69	-1.59	-1.79	-2.07	-2.03	-1.92
8	-2.87	-2.64	-2.69	-2.56	-2.42	-2.18	-2.10
9	Data not collected - equipment failure.						
10	-0.31	-0.05	-1.02	-1.37	-1.18	-1.47	-1.40
11	Data not collected - equipment failure.						
Avge	-0.98	-1.04	-1.08	-1.19	-1.40	-1.80	-1.74

Table 13. Peripheral Photoplethysmograph Data of Subjects Taking Dilantin (V)

Subject Number	Control	MI	MIIB	MIIA	MIII	Frank Sickness	Emesis
1	0.75	-0.43	-0.02	-0.31	-0.45	-0.55	-0.47
2	-0.50	0.03	1.22	-0.12	0.66	0.33	-0.66
3	-0.43	-2.13	-0.50	NA	NA	NA	NA
4	-0.53	0.34	0.92	NA	NA	NA	NA
5	Data not collected - subject didn't ride.						
6	Data not collected - subject didn't ride.						
7	-0.32	-0.73	-1.03	-1.07	-1.08	-0.95	-1.06
8	Data not collected - equipment failure.						
9	-1.65	-1.36	NA	NA	NA	NA	NA
10	0.16	-0.54	-0.88	-1.30	-1.50	-1.49	-1.39
11	-0.42	-0.31	-0.38	-0.47	-0.48	-0.21	-0.30
Avge	-0.37	-0.64	-0.10	-0.66	-0.57	-0.57	-0.77

Note: NA = Not applicable (subject never achieved frank sickness)

Vertical Electronystagmograph data The vertical electronystagmograph data, as measured in rms voltages, increased directly with the degree of motion sickness.

regardless of whether the subject was on dilantin or the placebo. The average rms voltage at emesis for subjects on dilantin was 145% of the average control rms voltage for those taking dilantin. Interestingly, this rms voltage was relatively much lower than for subjects taking the placebo, whose average rms voltage at emesis was 457% of the average control rms voltage for those taking the placebo.

These results agreed with those reported by Drylie in 1987 (11:44-45). Tables 14 and 15 describe the ENG data collected.

Table 14. Vertical Electronystagmograph Data of Subjects Taking Placebo (V_{rms})

Subject Number	Control	MI	MIIB	MIIA	MIII	Frank Sickness	Emesis
1	0.081	0.076	0.066	0.068	0.069	1.630	0.950
2	0.100	0.091	0.118	0.217	0.915	0.199	0.135
3	0.251	0.251	0.251	0.108	0.110	1.430	0.473
4	0.103	0.153	0.191	0.277	0.323	0.237	0.945
5	Data not collected - subject didn't ride.						
6	Data not analyzed - subject adapted to ride.						
7	0.120	0.123	0.146	0.106	0.152	0.129	0.148
8	0.080	0.394	0.284	0.132	0.121	0.218	1.470
9	0.081	0.155	0.161	0.337	0.277	0.376	1.040
10	0.060	0.065	0.135	0.076	0.116	0.156	0.098
11	0.110	0.093	0.256	0.219	0.168	0.315	0.207
Avg	0.109	0.156	0.179	0.171	0.250	0.521	0.607

Motion Sickness Models Using 1988 Data

The 1988 motion sickness data was first separated on the basis of whether the test subject was on dilantin or the placebo. The Abductive Reasoning Mechanism (ARM) software was then applied to both sets of data.

The data for the placebo model consisted of 41 data vectors (see Appendix E). Each data vector consisted of a test subject's stated symptom level, along with the electrocardiograph, electronystagmograph, electrosplanchnograph, and thoracic

Table 15. Vertical Electronystagmograph Data of Subjects Taking Dilantin (V rms)

Subject Number	Control	MI	MIIB	MIIA	MIII	Frank Sickness	Ernesis
1	0.290	0.203	0.128	0.154	0.183	0.444	0.711
2	0.080	0.103	0.067	0.378	0.352	0.117	0.179
3	0.168	0.063	0.059	NA	NA	NA	NA
4	0.223	0.148	0.090	NA	NA	NA	NA
5	Data not collected - subject didn't ride.						
6	Data not collected - subject didn't ride.						
7	0.083	0.266	0.070	0.348	0.080	0.288	0.122
8	0.117	0.246	0.153	0.168	-	0.885	0.697
9	0.140	0.129	NA	NA	NA	NA	NA
10	0.081	0.056	0.153	0.080	0.105	0.074	0.199
11	0.129	0.053	0.101	0.144	0.184	0.314	0.241
Avg	0.146	0.141	0.103	0.212	0.181	0.354	0.358

Note: NA = Not applicable (subject never achieved sickness level)

respiration data associated with that symptom level. The facial photoplethysmograph and peripheral photoplethysmograph data were not used, due to their presumed unreliability. The following nonlinear equation was derived relating the level of motion sickness to the biophysiological data collected

$$\begin{aligned}
 level = & -1.63872 + (0.046845)(ekg) + (55.0861)(eng) \\
 & -(49.5576)(esg) - (1.50992)(tho) \\
 & -(0.518868)(ekg)(eng) + (8.2708)(eng^2) \\
 & +(0.638416)(ekg)(esg) + (77.1585)(eng)(esg) \\
 & +(1.15506)(tho^2) - (11.7564)(eng^3) \\
 & -(1.01402)(ekg)(eng)(esg)
 \end{aligned} \tag{15}$$

where

level = reported symptom level

ekg = electrocardiograph value

eng = electronystagmograph value

esg = electrosplanchnograph value

tho = thoracic respiration value.

The ARM did not discard any of the four sets of input data as being irrelevant. Modeling statistics are shown in Table 16. Figure 3 is a comparison of the actual symptom levels with the symptom levels computed by entering the data from Appendix E into equation (15).

Table 16. Modeling Statistics for Equation (15)

Variable	Mean	Sigma	Min	Max
level	4.71	3.31	1	10
ekg	78.54	10.10	60	103
eng	0.24	0.28	0.0603	1.47
esg	0.35	0.72	0.0229	3.65
tho	0.65	0.45	0.17	2.48

The model developed with the data from subjects on the placebo did a poor job of predicting motion sickness levels. The ARM values were within one level of the reported symptom levels for 27% of the observations, and within two levels of the reported symptom levels for only 59% of the observations.

The data for the dilantin model consisted of only 21 data vectors (see Appendix F). As with the placebo model, the output variable was level, and the input variables were ekg, eng, esg, and tho. The following nonlinear equation was then developed with the aid of the ARM software

$$\begin{aligned} level = & 40.8357 - (4.61778)(ekg) + (176.065)(eng) \\ & + (45.4337)(tho) + (0.099773)(ekg^2) \end{aligned}$$

$$\begin{aligned}
& -(5.15111)(ekg)(eng) + (1789.96)(eng^2) \\
& -(0.715054)(ekg)(tho) - (322.918)(eng)(tho) \\
& +(0.881435)(tho^2) - (0.000576)(ekg^3) \\
& -(4666.85)(eng^3) + (5.23943)(ekg)(eng)(tho)
\end{aligned} \tag{16}$$

The ARM software did not discard any of the four sets of input data for the dilantin model. Modeling statistics are shown in Table 17. Figure 4 is a comparison of the actual symptom levels with the symptom levels computed by entering the data from Appendix F into equation (16).

Table 17. Modeling Statistics for Equation (16)

Variable	Mean	Sigma	Min	Max
level	3.57	3.22	1	10
ekg	71.81	0.08	57	100
eng	0.14	0.08	0.0525	0.378
esg	0.25	0.49	0.0185	2.23
tho	0.98	0.84	0.132	3.11

The model developed with the data from subjects on dilantin did an excellent job of predicting motion sickness levels. The ARM values were within one level of the reported symptom levels for 76% of the observations, and within two levels of the reported symptom levels for 95% of the observations. However, the effectiveness of the model may have been due to the small size of the data base.

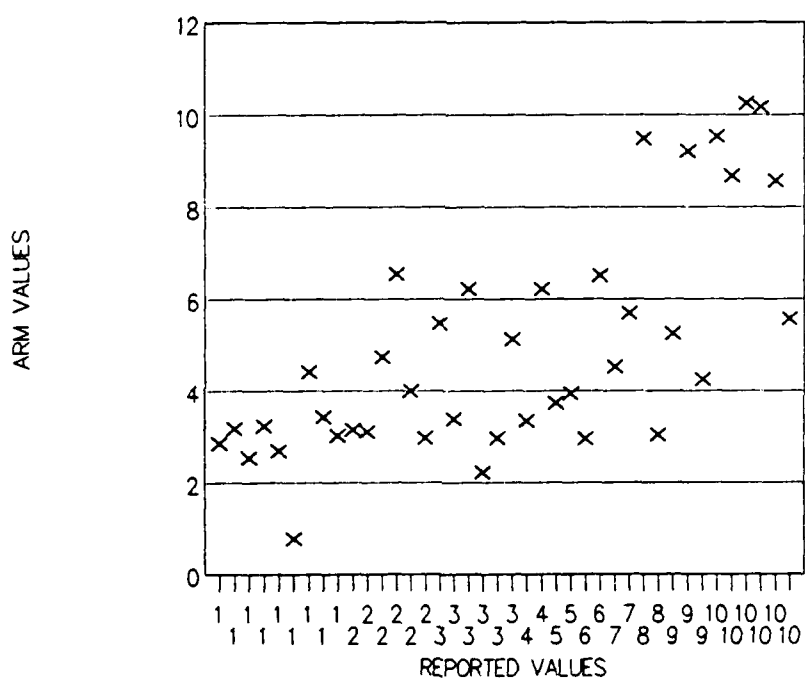


Figure 3. Modeling of 1988 Data (Subjects on the Placebo)

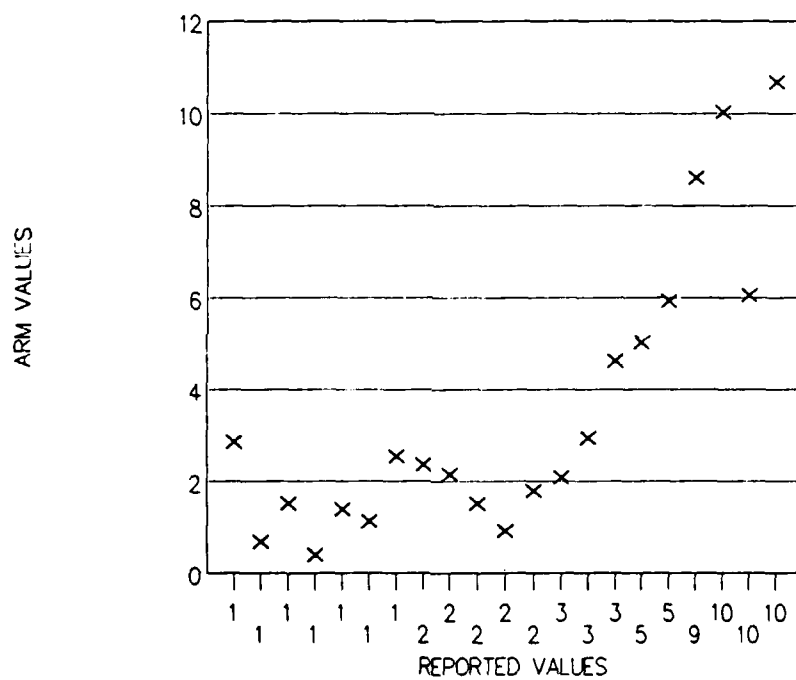


Figure 4. Modeling of 1988 Data (Subjects on Dilantin)

V. Conclusions and Recommendations

Conclusions

Dilantin delayed or even prevented the evolution of motion sickness in nine volunteers tested according to the 1988 AFIT motion sickness research protocol (see Figure 5). Appendix C describes a personal experience with dilantin in reducing the effect of motion sickness.

These results are significant for military aerospace operations. Even if it doesn't completely cure motion sickness, dilantin could "buy time" for military astronauts so that they could effectively function while their bodies were still becoming acclimated to space. Thus, American astronauts could perform military space maneuvers of short duration while their enemy counterparts would still be suffering from motion sickness.

Dilantin did not affect the physical performance and cognitive abilities of the test subjects. The grammatical reasoning (GR) test was given to evaluate reasoning ability. Subjects taking the placebo had an average mean correct response time (mcrt) of 3498.963 msec while completing the GR test, and had an average score of 95.27% correct. Subjects on dilantin had a better mcrt of 3263.366 msec, while their average scores declined only slightly to 95.18% correct. The unstable tracking (UT) test measured manual response speed and accuracy. Subjects that took the placebo had an average 40.6 rms error, with an average of 48.6 total edge violations. Subjects taking dilantin had slightly poorer UT test results, averaging a 42.5 rms error, and 70.0 total edge violations. (Subject #3's UT results were analyzed separately, since he took the test at the low level of difficulty, while everyone else took the test at the medium level. He had a better UT test while under dilantin - 12.9 rms error, versus 15.3 rms error while taking the placebo. He had no edge violations for either trial.) Finally, the subjects were given a probability monitoring test (abbreviated as DM.

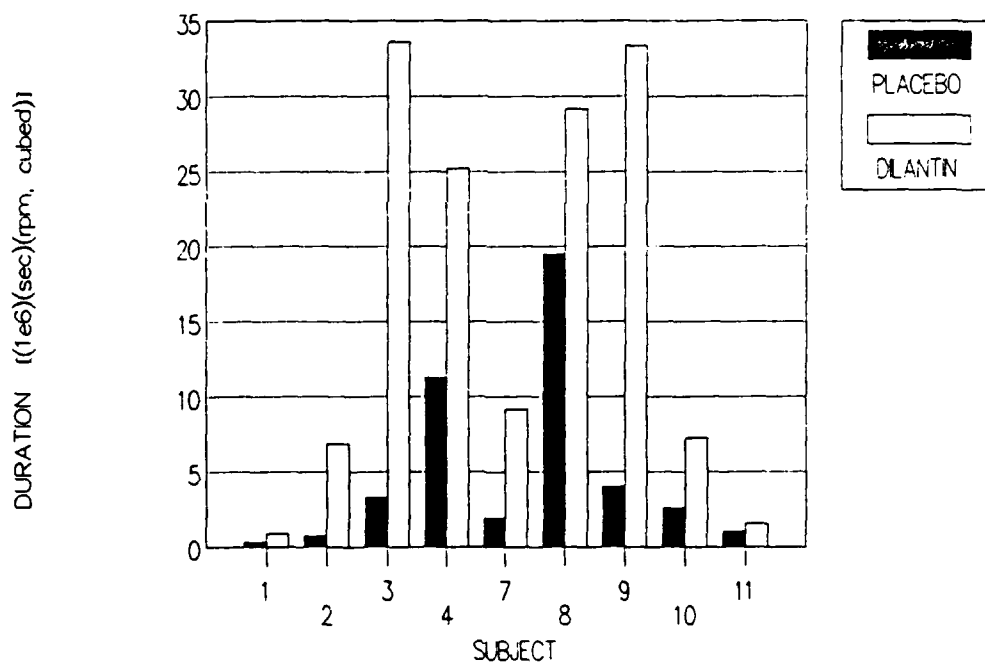


Figure 5. Effect of Dilantin on the Duration of Head Motions

for display monitoring) to test their visual perception. The results of this test were similar. Subjects on the placebo averaged 9.8 correct, 3.9 false, and 0 missed biases. Subjects on dilantin averaged 9.7 correct, 5.9 false, and 0.3 missed biases. The mean response time (mrt) for subjects on the placebo taking the DM test averaged 2.9 sec, while subjects taking dilantin averaged 3.3 sec. The complete performance-cognition test results are presented in Appendix G.

Most subjects taking dilantin did not report any major symptoms before the chair ride.

Subject #5 had the worst symptoms before the chair ride. He complained of skin rash, a swollen tongue, and a decrease in coordination. He discontinued the dilantin treatment after two doses. He had reported allergies to several different drugs on his medical history form.

Four of the remaining test subjects reported lightheadedness. However, this symptom was deemed minor by the subjects themselves, and did not affect the performance-cognition test results. Other symptoms reported by the other volunteers, such as fatigue or diarrhea, could be ascribed to other causes. Subject #4 was asymptomatic before the chair ride.

Table 18 provides a complete description of the subject symptoms before the chair ride, and while on dilantin.

Test subjects, while on dilantin, had symptoms not normally associated with motion sickness (e.g., hunger and thirst). Most of the test subjects, while on dilantin, perceived themselves as undergoing motions other than spinning. Table 19 provides a summary of subject symptoms during the chair ride, while the subjects were on dilantin.

The test subjects taking dilantin did not report any major symptoms after their motion chair ride. Subject #2 did mention a skin rash on his chest that appeared a day after the ride, and in the areas where body electrodes had been placed. Later

Table 18. Subject Symptoms Before Chair Ride, While on Dilantin

Subject	Symptoms
1	Lightheadedness, apathy, constant muffled hearing.
2	Diarrhea, gas (Subject thought due to diet).
3	Diarrhea, gas.
4	No symptoms reported.
5	Skin rash, lack of coordination, swollen tongue. (Subject discontinued treatment after two doses; had a history of drug allergies.)
6	Subject never took dilantin treatment.
7	Lightheadedness, apathy, intermittent muffled hearing, Indecisiveness, drowsiness, indigestion.
8	Fatigue.
9	Lightheadedness; become dizzy after tilting head back and after running. (Subject thought that dizziness might have been due to a sinus infection.)
10	Lightheadedness.
11	Fatigue (Subject reported that he was spending alot of time in the evenings working on his thesis.)

volunteers were directed to wash their chests immediately after the removal of the body electrodes, and no further skin rashes were reported. Even though the dilantin treatment only slightly delayed the evolution of motion sickness in subject #11, it ameliorated his post-chair ride symptoms, as compared to how he felt after his ride while on the placebo. Table 20 further discusses the subjects' reported symptoms after their chair rides.

While on dilantin, the subjects had varying attitudes about the relative provocativeness of the different head motions. Table 21 describes these opinions.

The subjects taking dilantin appeared to adapt differently to motion sickness. Table 22 presents these results.

The electronystagmograph, electrosplanchnograph, and thoracic respiration data appeared to be good predictors of motion sickness. The electrocardiograph

values had a positive correlation with the evolution of motion sickness for those subjects on the placebo. The average heart rates of the volunteers taking dilantin stayed at the control levels during the experiments. The facial and peripheral photoplethysmograph data were unreliable.

The ARM software operated very well. Its operating menus were readily understandable, eliminating the need for instruction manuals. Data bases and variables could be easily created or changed. The software rapidly produced results which could be displayed as usable equations. The Air Force Institute of Technology should continue using this software, along with any other later upgrades provided by its creator (Barron Associates, Inc.).

The motion sickness models created using the ARM software varied in effectiveness. The model created with the data collected in 1988 from the subjects on dilantin appeared to be the best predictor of motion sickness levels. However, the effectiveness of both 1988 models is called into question by the small sizes of their data bases. The various models are compared in Table 23.

Error Analysis

Sources of Experimental Error

1. Sensors. The facial and peripheral photoplethysmographs did not appear to operate as intended. The failure rate for all of the sensors is documented in Table 24.
2. Environmental conditions. Building 640 suffered from air conditioning problems during the summer. The body electrodes would fall off the test subjects whenever room 150 got too hot. In addition, an engineering experiment taking place in one corner appeared to be a source of electromagnetic interference. The EEG data appeared to be most affected by this interference.

3. Electrodes. Some of the body and ENG electrodes had to be replaced because they lost their adhesiveness (most likely due to age).
4. Procedural errors. Until the 1988 motion sickness team had gained sufficient testing experience, the results suffered from misapplied sensors and electrodes. Equipment familiarization and procedural standardization eliminated these problems.
5. Motion artifacts, primarily resulting from sensor wires being allowed to move too freely. Careful use of surgical tape to limit sensor wire movement minimized this problem. The 1988 motion sickness research team also had the test subjects practice the head motions during the control period. This technique allowed the team to discover artifacts associated with movement, and to recognize those artifacts which could not be eliminated by taping down sensor wires.

Sources of Error in Data Interpretation

1. The volunteers' subjectivity in reporting symptoms and symptom levels.
2. The researchers' subjectivity in determining the Graybiel levels of motion sickness experienced by the test subjects during each motion chair ride.
3. When the FM tapes were rewound, they wouldn't always stop at the same recorder address (normally, "0"). However, this was only an insignificant error, since the data were averaged over 8 second periods.

Recommendations

1. Recommended acquisitions.
 - (a) A new motion chair. The present chair appears to be close to the end of its operating life. Its slip rings are starting to fail, causing a loss of

signal channels. Also, regular lubrication is not preventing the chair from making sounds indicative of bearing wear while the chair is spinning.

- (b) Improved photoplethysmograph sensors.
- (c) A peripheral skin temperature sensor should be reintroduced. Room 150 is now air-conditioned, so these data should no longer be affected by environmental conditions. Fix's peripheral skin temperature data (collected in 1987) appeared to be quite reliable.

2. Recommended changes.

- (a) The experimental procedure should be changed to accommodate those situations when the subject has been riding in the chair for at least 30 minutes, yet is still asymptomatic. Under present guidelines, when such a situation occurs, arbitrary decisions are made as to when to increase the motion chair speed and/or end the chair ride.
- (b) The probability monitoring (PM) task used in the performance-cognition tests should be replaced with another test. A majority of the test subjects found the PM task confusing and unenjoyable - opinions which undoubtedly affected the test results.
- (c) The phonosplachnograph data should only be used if someone will actually analyze the data. This sensor is uncomfortable to wear, and often painful to remove.

3. Recommendations for further research.

- (a) The minimum effective dose of dilantin needs to be found. Based on the 1988 data, this dosage level appears to result in a concentration of dilantin in the blood of around 10 ug/ml.
- (b) Both the 1987 and 1988 motion sickness research teams found that ENG signals increased in amplitude with the evolution of motion sickness. How-

ever, it has been contended that this change in signal strength may be due to sweating (3). An effort should be made to resolve this question.

- (c) The accuracy of the 1988 motion sickness models suffered because their data bases were too small. As research with dilantin continues, more data vectors will be collected. The ARM software should then be reapplied to the expanded data bases.
- (d) Subject #7, while on dilantin, reported hearing muffled sounds in a room with many sources of noise. However, he could talk to his wife without any difficulty, when they were the only ones in a room. This interesting anomaly deserves further attention.
- (e) Further researchers need to consider whether, if a test subject took dilantin on the first trial, his performance on the second trial (while on the placebo) was in some part due to an adaptation effect. (The 1988 researchers tried to eliminate this problem by having each test subject's second ride rotate in the opposite direction of his first ride.)
- (f) Given subject #5's reaction, the side effects of dilantin, when taken in small doses, should be carefully observed and correlated with observations in the literature concerning this drug.
- (g) The effectiveness of dilantin in treating motion sickness should be tested under real time conditions - i.e., low level flying in turbulent weather, or zero-g parabola flights.
- (h) Further research is necessary to examine whether a combination of dilantin with some other drug (i.e., dextromethorphan) might increase the effectiveness of dilantin in treating motion sickness.
- (i) Preliminary results with the use of a CO₂ monitor indicate that a test subject's CO₂ concentration in his exhaled breaths declines as motion sickness evolves (most likely due to hyperventilation). This parameter

should be investigated as a concomitant of motion sickness, and any data collected might be added to a motion sickness model data base.

Table 19. Subject Symptoms During Chair Ride, While on Dilantin

Subject	Symptoms
1	Talkative, visualized spinning (had a short ride).
2	Carousel ride sensation, talkative, hungry.
3	Sensations of tumbling backwards, moving end over end, and "tumbling down and to the right". Talkative, hungry, "feels like could go to sleep" (sleepy from boredom).
4	Talkative, hungry.
5	Subject didn't ride in motion chair under dilantin.
6	Subject didn't ride in motion chair under dilantin.
7	Rollercoaster feeling, merry-go-round feeling, "like a pendulum", "like going around a curve".
8	Sleepy from boredom.
9	Talkative, hungry, and thirsty. "Feels like a rollercoaster", "Feels like a carnival ride", "Don't have spinning sensation of last time" (i.e. susceptibility trial), "should've brought a book" (bored), laughing.
10	Subject reported that the instant he did his first head motion, he realized that he was on dilantin; the sensation was "like night and day". During each head motion, he would initially feel a spinning sensation, which was then replaced by a rollercoaster sensation (not the feeling of going down a hill, but that of going around a curve to the right). Subject also reported boredom and reduced sweating (compared to the trial while on the placebo).
11	Normal motion sickness symptoms.

Table 20. Subject Symptoms After Chair Ride, While on Dilantin

Subject	Symptoms
1	No symptoms reported.
2	Allergic to body electrodes. Queasiness that evening while driving over rolling hills and while swinging on a child's swing.
3	No symptoms reported.
4	No symptoms reported.
5	Subject didn't ride in motion chair while under dilantin.
6	Subject didn't ride in motion chair while under dilantin.
7	No symptoms reported.
8	Subject received nontherapeutic dose; no symptoms reported.
9	Subject woke up at 3 AM (about 12 hours after the ride) with an itching sensation; itching relieved by taking a shower. Subject reported that wife had used a different detergent that day while doing the laundry. No further symptoms reported.
10	Not sweating as much as after trial with placebo.
11	Subject reported that he had a quicker recovery after the trial with dilantin than after the trial with the placebo. (He was able to weightlift the evening after the dilantin trial, while remaining nauseous and incapable of any activity the night after the placebo trial.)

Table 21. Subject Feelings About Head Motions, While on Dilantin

Subject	Feelings
1	Coming back up most provocative.
2	Moving head left or right most provocative; moving head down had no effect.
3	Moving head left or right felt worse than moving head downward.
4	Felt dizzy after moving head to the right than to the left.
5	Subject didn't ride in motion chair while under dilantin.
6	Subject didn't ride in motion chair while under dilantin.
7	Moving the head upward was most provocative.
8	First two head motions were the most provocative.
9	Most provocative motion was moving the head upward, after a downward movement. The next most provocative motion was moving the head upward, after moving the head to the right.
10	Moving the head to the left was most provocative.
11	Moving the head upward was most provocative.

Table 22. Change in Symptom Levels, While Subjects on Dilantin

Subject	Changes in Symptom Level
1	Trial ended with emesis (The avalanche syndrome, or the rapid evolution of motion sickness, did not occur).
2	Trial ended with emesis (avalanche syndrome present).
3	Subject at symptom level 1-2 throughout most of the trial (trial began with chair speed at 16 rpm); symptom level 3 at 26 rpm; symptom level 4 at 28 rpm.
4	Symptom levels of 1-3 throughout most of trial, with chair speed at 16 rpm; at symptom level 3 when chair at 20 rpm.
5	Subject didn't ride in motion chair while under dilantin.
6	Subject didn't ride in motion chair while under dilantin.
7	Trial ended with emesis (avalanche syndrome not present).
8	Trial ended with emesis (avalanche syndrome present).
9	Subject at symptom level 1-2 throughout trial.
10	Trial ended with emesis (avalanche syndrome not present).
11	Trial ended with wretching (avalanche syndrome not present).

Table 23. A Comparison of the ARM Motion Sickness Models

Model	Database (# vectors)	Accuracy within 1 level of reported level	Accuracy within 2 levels of reported level
1986	53	60%	77%
1987	100	64%	90%
1988:			
Placebo	41	27%	59%
Dilantin	21	76%	95%

Table 24. Sensor Failure Rate

Sensor	% Failure (# Failures/18 trials)
EEG 4 (EEG 2A)	33.3
EEG 5 (EEG 2B)	27.8
EEG 3 (EEG 1C)	22.2
ESG (RLQ)	16.7
Peripheral Photo.	11.1
Ballistocardiograph	5.6
Abdominal Pneumo.	5.6
EKG	5.6
ESG (LUQ)	5.6
Left Facial Photo.	5.6
Right Facial Photo.	5.6
Thoracic Pneumo.	5.6
EEG 1 (EEG 1A)	0
EEG 2 (EEG 1B)	0
Horizontal ENG	0
Vertical ENG	0
Phonosplachnograph	0

Appendix A. *1986 Motion Sickness Data Used With ARM Software*

Notes:

1. No attempt was made to interpolate symptom levels when they weren't provided in the McPherson thesis (24:85-170). Only those data which had a single symptom level associated with them were used. Observations 1-8 are from subject #2, 9-12 from subject #3, 13-20 from subject #4, 21-25 from subject #5, 26-31 from subject #6, 32-37 from subject #7, 38-43 from subject #9, 44-48 from subject #10, and 49-53 from subject #11. The data from subjects #1 and #8 were not used - subject #1 had no GSR data, and subject #8 had no Surface Skin Temperature data.

2. Thoracic and Diaphragmatic Respiration data were derived as follows - values were given in terms of "vol/#breaths"; the given volume was then divided by the number of breaths to get a value with units "vol/breath", and the result rounded to the nearest whole number.

Table 25. 1986 Motion Sickness Data: Observations 1-35

Observation Number	Finger Photo. (%)	Surface Skin Temp. (F)	EKG (bpm)	Thoracic Resp. (cc)	Dia. Resp. (cc)	GSR (K)	Symptom Level
1	90	97.6	66	167	233	884	1
2	85	97.7	78	130	160	420	2
3	95	97.7	78	167	158	420	3
4	85	97.7	78	130	140	380	4
5	85	97.7	78	170	100	350	6
6	85	97.7	84	160	100	170	7
7	90	97.7	90	170	100	180	8
8	85	97.8	90	142	92	400	9
9	90	79.8	66	186	86	390	1
10	80	79.8	90	175	175	765	2
11	75	79.8	78	360	220	840	5
12	75	80.0	69	313	186	750	8
13	90	88.7	123	400	320	1184	1
14	90	88.5	135	267	400	1104	2
15	90	88.3	126	164	182	854	3
16	85	88.3	132	333	333	854	5
17	85	88.1	120	343	171	854	6
18	85	87.9	132	286	171	854	8
19	80	87.9	126	500	200	854	9
20	80	87.4	120	375	125	854	10
21	80	85	78	163	113	46	1
22	80	85.1	96	100	92	70	2
23	80	85.3	84	144	144	95	6
24	70	85.6	48	189	267	175	8
25	70	86.3	60	213	350	240	9
26	75	94.8	84	67	56	845	1
27	85	94.4	78	125	100	880	2
28	65	94.05	66	160	60	845	3
29	75	93.8	84	350	175	830	4
30	75	93.7	72	250	225	770	8
31	70	93.7	72	240	120	840	9
32	70	93.3	63	157	171	840	1
33	65	93.1	78	260	180	800	2
34	60	93	78	233	100	840	3
35	60	93	84	425	150	800	8

Table 26. 1986 Motion Sickness Data: Observations 36-53

Observation Number	Finger Photo. (%)	Surface Skin Temp. (F)	EKG (bpm)	Thoracic Resp. (cc)	Dia. Resp. (cc)	GSR (K)	Symptom Level
36	25	92.7	66	375	75	890	9
37	30	92.6	69	450	75	880	10
38	80	93.4	120	200	375	563	1
39	65	93.4	114	214	286	563	3
40	50	93.3	117	175	325	600	5
41	50	93.2	102	250	500	600	7
42	55	93.1	120	380	740	563	9
43	40	92.9	108	450	950	700	10
44	80	95.1	96	133	50	750	1
45	55	94.4	84	200	133	900	4
46	55	94.3	90	600	467	854	6
47	70	94.1	84	533	800	854	9
48	70	94	90	375	150	854	10
49	75	88.9	132	150	75	15	1
50	65	88.5	114	111	89	15	2
51	50	88.2	108	150	150	15	8
52	45	88.2	84	117	183	15	9
53	45	88.2	96	75	275	15	10

Appendix B. 1988 Motion Sickness Data

Terminology:

e = estimated

mpe = minute-long period after emesis

* = period used in 1988 motion sickness models

SSL = Subject symptom level (reported by subject)

NR = Not reported

Table 27. Subject 1, Trial 1 (Placebo), 1 Jul 88

Time	Event	Recorder Address	Symptom Level
17:02:06e	Control	172-176	1*
17:03:32e	Start Chair		14 rpm
17:05:49e	Start Head Mtns	193e	1*
17:06:08e	M I	201-205(e)	2*
17:06:18e	M IIB	206-210(e)	3*
17:06:58e	M IIA	226-230(e)	3-8
17:07:16e	M III	233-237(e)	8*
17:07:50e	End Head Mtns		
	Frank Sickness	260-264(e)	
17:08:14e	Emesis	264-268(e)	10*
	1mpe		SSL 8-6
	2mpe		SSL 4-3
	3mpe		SSL 3-1
	4-10 mpe		NR

Table 28. Subject 1, Trial 2 (Dilantin), 8 Jul 88

Time	Event	Recorder Address	Symptom Level
	Control	374-378	1*
16:42:33e	Start Chair	422	14 rpm
16:43:53e	Start Head Mtns	458	
16:45:03e	M I	491-495(e)	1*
16:46:23e	M IIB	531-535(e)	3*
16:47:02e	M IIA	548-552	6*
16:48:21e	M III	590-594	9*
16:49:07e	End Head Mtns		
	Frank Sickness	607-611	
16:49:12e	Emesis	611-615	10*
	1 mpe		SSL 4-7
	2 mpe		SSL 2
	3-4 mpe		NR
	5 mpe		SSL 1
	6-10 mpe		NR

Table 29. Subject 2, Trial 1 (Placebo), 7 Jul 88

Time	Event	Recorder Address	Symptom Level
16:57:43e	Control	303-307(e)	1*
16:58:07e	Start Chair		14 rpm
17:00:56	Start Head Mtns	388	
17:02:31e	M I	434-438(e)	2*
17:04:16e	M IIB	486-490(e)	3*
17:04:27e	M IIA	492-496(e)	3-4
17:04:55e	M III	506-510(e)	6-8
17:05:09	Frank Sickness	515-519(e)	
17:05:19e	End Head Mtns		
17:05:22	Emesis	519-523(e)	
	1- 10 mpe		NR

Table 30. Subject 2, Trial 2 (Dilantin), 13 Jul 88

Time	Event	Recorder Address	Symptom Level
16:07:22	Control	247-251(e)	1*
	Start Chair		14 rpm
16:11:03e	Start Head Mtms	318	
16:19:31	M I	555-559(e)	1*
16:43:35e	M IIB	1252-1256(e)	2*
16:52:17e	M IIA	1493-1497(e)	5*
16:52:21e	M III	1497-1501	5-9
	Frank Sickness	1501-1505	5-9
16:52:43	End Head Mtms		
16:52:43	Emesis	1505-1509	10*
	1 mpe		SSL 4-10
	2-10 mpe		NR

Table 31. Subject 3, Trial 1 (Placebo), 14 Jul 88

Time	Event	Recorder Address	Symptom Level
	Control	298-302(e)	1*
16:23:34e	Start Chair		16 rpm
16:25:05e	Start Head Mtms	384	
16:25:14e	M I	392-396(e)	2*
16:28:21e	M IIB	475-479(e)	3*
16:31:16e	M IIA	558-562	5*
16:33:34e	M III	624-628(e)	7*
		666-670	8*
		696-700	9*
	Frank Sickness	760-764	
16:38:19e	End Head Mtms	763e	
16:38:28	Emesis	764-768	10*
	1-2 mpe		NR
	2 mpe		SSL 2
	4 mpe		NR
	5 mpe		SSL 1
	6-10 mpe		NR

Table 32. Subject 3, Trial 2 (Dilantin), 22 Jul 88

Time	Event	Recorder Address	Symptom Level
	Control	245-249	1*
16:51:20	Start Chair	391	16 rpm
16:52:43	Start Head Mtns	448(e)	
18:10:39e	M I	1148-1152(Tape 2)	2*
18:16:57	M IIB	1358-1362(Tape 2)	3*
18:23:00	End Head Mtns		
18:25(e)	Chair Stopped		

Table 33. Subject 4, Trial 1 (Dilantin), 15 Jul 88

Time	Event	Recorder Address	Symptom Level
	Control	225-229	1*
	Start Chair	298	16 rpm
16:13:08e	Start Head Mtns	330(e)	
16:21:51e	M I	578-582	2*
		678-682	2*
16:57:42e	M IIB	98-102(Tape 2)	3*
17:50:00	End Head Mtns		

Table 34. Subject 4, Trial 2 (Placebo), 25 Jul 88

Time	Event	Recorder Address	Symptom Level
	Control	210-214	1*
16:32:23e	Start Chair	235	16 rpm
16:33:59e	Start Head Mtns	283(e)	
16:38:27e	M I	404-408(e)	1*
16:50:43	M IIB	758-762(e)	2*
16:52:54e	M IIA	821-825(e)	3*
17:08:25e	M III	1266-1270(e)	5*
	Frank Sickness	1374-1378	
17:12:13e	End Head Mtns		
17:12:24e	Emesis	1378-1382	10*
	1-2 mpe		NR
	3 mpe		SSL 3
	4 mpe		SSL 1-2
	5-10 mpe		NR

Table 35. Subject 7, Trial 1 (Placebo), 4 Aug 88

Time	Event	Recorder Address	Symptom Level
	Control	144-148(e)	1*
15:52:26	Start Chair	172	17 rpm
15:53:18	Start Head Mtns	193	
15:53:54e	M I	210-214	2*
15:54:14e	M IIB	221-225	3*
15:55:07e	M IIA	247-251	5*
15:58:20e	M III	340-344	7*
		368-372	8*
	Frank Sickness	382-386	
15:59:55e	End Head Mtns		
16:00:00	Emesis	386-390	10*
	1 mpe		SSL 6
	2 mpe		SSL 5-6
	3 mpe		SSL 5
	4 mpe		SSL 4
	5-10 mpe		SSL 3

Table 36. Subject 7, Trial 2 (Dilantin), 12 Aug 88

Time	Event	Recorder Address	Symptom Level
	Control	339-343(e)	1*
15:17:10	Start Chair	370	18 rpm
15:18:30e	Start Head Mtns	406	
15:22:10		511-515	1*
15:29:45	M I	730-734	2*
15:39:43	M IIB	1015-1019	3*
15:43:20	M IIA	1114-1118	4-5
15:43:26e	M III	1118-1122	7-8
	Frank Sickness	1149-1153	
15:44:40	End Head Mtns	1155	
15:44:40	Emesis	1153-1157	10*
	1 mpe		SSL 5
	2-3 mpe		SSL 4
	4 mpe		SSL 3-4
	5-6 mpe		SSL 3
	7 mpe		NR
	8 mpe		SSL 2
	9 mpe		NR
	10 mpe		SSL 2

Table 37. Subject 8, Trial 2 (Placebo), 17 Aug 88

Time	Event	Recorder Address	Symptom Level
	Control	615-619	1*
14:27:50	Start Chair	650	22 rpm
14:29:05	Start Head Mtns	685	
14:31:25	M I	750-754	2*
14:32:27	M IIB	779-783	3*
		923-927	3*
14:39:07e	M IIA	978-982	4*
		984-988	5*
14:42:15e	M III	1063-1067	6*
		1140-1144	7*
	Frank Sickness	38-42(Tape 2)	10*
14:59:36e	End Head Mtns		
14:59:40	Emesis	42-46(Tape 2)	10*
	1 mpe		SSL 9
	2 mpe		SSL 5
	3 mpe		SSL 3-4
	4 mpe		SSL 2
	5-10 mpe		SSL 1

Table 38. Subject 8, Trial 3 (Dilantin), 9 Sep 88

Time	Event	Recorder Address	Symptom Level
	Control	31-35	1*
16:35:45	Start Chair		22 rpm
16:36:56	Start Head Mtns		
16:40:16		398-402	1*
16:44:28	M I	518-522	2*
16:49:12		654-658	2*
17:22:05	M IIB	79-83(Tape 2)	3*
17:22:27	M IIA	89-93(Tape 2)	4*
	Frank Sickness	94-98(Tape 2)	
17:22:32e	End Head Mtns		
17:22:36	Emesis	98-102(Tape 2)	10*
	1 mpe		SSL 9
	2 mpe		SSL 6-7
	3 mpe		SSL 3
	4-10 mpe		SSL 1

Table 39. Subject 9, Trial 1 (Dilantin), 30 Aug 88

Time	Event	Recorder Address	Symptom Level
	Control	351-355	1*
	Start Chair	365	18 rpm
17:20:11	Start Head Mtns	423	
17:23:06	M I	507-511	2*
18:30:09	End Head Mtns		

Table 40. Subject 9 Trial 2 (Placebo), 8 Sep 88

Time	Event	Recorder Address	Symptom Level
16:44:45	Control	654-658	1*
	Start Chair	703	20 rpm
16:47:33	Start Head Mtns	731	
16:49:29		784-788	1*
16:50:32	M I	814-818	2*
16:51:32	M IIB	845-849	5*
	M IIA	864-868	7*
16:53:15	M III	894-898	9*
16:55:05		945-949	9*
	Frank Sickness	966-970	
16:55:57	End Head Mtns		
16:55:57	Emesis	970-974	10*
	1 mpe		SSL 5-10
	2 mpe		SSL 3-4
	3 mpe		SSL 3
	4 mpe		SSL 2-3
	5-10 mpe		SSL 2

Table 41. Subject 10, Trial 1 (Placebo), 1 Sep 88

Time	Event	Recorder Address	Symptom Level
	Control	399-403	1*
	Start Chair	425-458	
15:37:10	Start Head Mtns	458	20 rpm
15:37:13e	M I	461-465	
15:39:35e	M IIB	531-535	3*
15:41:10e	M IIA	575-579	2-4
15:42:13e	M III	604-608	8*
	Frank Sickness	617-621	
15:42:39e	End Head Mtns	619e	
15:42:50	Emesis	621-625	10*
	1 mpe		SSL 4
	2-10 mpe		SSL 1

Table 42. Subject 10, Trial 2 (Placebo), 16 Sep 88

Time	Event	Recorder Address	Symptom Level
15:47:00	Control	287-291	1*
15:49:10	Start Chair	323	
15:50:25	Start Head Mtns	359	20 rpm
15:54:25	M I	471-475	1*
15:58:35	M IIB	590-594	3*
16:02:48	M IIA	710-714	5*
	M III	782-786(e)	
	Frank Sickness	786-790	
16:05:30	End Head Mtns	792	
16:05:30	Emesis	790-794	10*
	1 mpe		NR
	2-10 mpe		SSL 1

Table 43. Subject 11, Trial 1 (Dilantin), 7 Sep 88

Time	Event	Recorder Address	Symptom Level
	Control	380-384	1*
15:40:46e	Start Chair		14 rpm
15:42:23	Start Head Mtns	527	
15:42:23	M I	527-531	2*
15:43:48	M IIB	567-571	3*
15:44:28	M IIA	586-590	5*
15:47:54	M III	685-689	9*
	Frank Sickness	793-797	
15:51:53	Emesis	797-801	10*
	1 mpe		SSL 9
	2 mpe		SSL 8
	3-6 mpe		SSL 7
	7-8 mpe		SSL 6
	9 mpe		SSL 5-6
	10 mpe		SSL 5

Table 44. Subject 11, Trial 2 (Placebo), 14 Sep 88

Time	Event	Recorder Address	Symptom Level
	Control	417-421(e)	1*
16:17:39	Start Chair		14 rpm
16:18:51	Start Head Mtns	481	
16:19:02	M I	484-488	2*
16:20:44	M IIB	533-537	4*
16:21:45		561-565	6*
16:22:08	M IIA	572-576	7*
16:24:00	M III	626-630	9*
	Frank Sickness	658-662	
16:25:16	End Head Mtns		
16:25:16	Emesis	662-666	10*
	1-5 mpe		SSL 7
	6-8 mpe		SSL 6
	9 mpe		SSL 6-7
	10 mpe		SSL 7

Appendix C. *A Personal Experience With Dilantin*

Ride # 1 - While on Placebo

Ride #1 lasted 4 minutes and 34 seconds, measured from the start of head motions, and until head motions were stopped when the subject began vomiting. (The subject's last stages of motion sickness followed the "avalanche phenomenon" pattern, with the symptom levels changing from 4 to 10 in 43 seconds.) The subject had a spinning sensation throughout the chair ride.

Ride # 2 - While on Dilantin

Ride #2 lasted 41 minutes and 25 seconds.

Before the Ride The subject had diarrhea and a slight gastric disturbance the night before the ride. The subject accomplished the performance-cognition tests before the ride. Compared to the tests taken before ride #1, the subject improved on the grammatical reasoning and display monitoring exercises, while doing slightly worse on the unstable tracking test. The subject felt a numbness in the right portion of his head while performing the performance-cognition tests before ride #2.

While being instrumented, the subject was at symptom level 2. This higher than normal sense of unease was probably due to his memory of having gotten sick in the chair during ride # 1. As a result, the subject had sweaty hands, stomach nausea, and gas.

During the Ride The chair was rotated at 14 rpm in a clockwise direction. When the subject moved his head down, and then back up, he didn't feel he was spinning. When the subject first tilted his head to the right, he felt like his body was moving in a slow, circular fashion. Almost immediately, he felt he was riding on a merry-go-round. However, he felt that the ride was going in a clockwise direction.

if viewed from above (merry-go-rounds normally go counterclockwise). Thereafter, whenever the subject tilted his head either to the left or to the right, he would first note a spinning sensation, and a temporary increase in motion sickness. The spinning sensation would then be quickly (and involuntarily) replaced by the merry-go-round sensation, and the subject's sense of motion sickness would subside. The subject would then relax and await the next head motion.

The subject had other memories about the ride, as well. He commented several times about the chair rotating too slowly, although the speed was checked during the ride. The subject noted that he had eye movements accompanying his head motions. He occasionally belched, and became thirsty during the course of the ride, due to talking.

Towards the end of the ride, the subject's buttocks began to hurt from sitting in the chair. The ride ended when the subject again underwent the "avalanche syndrome", going from symptom level 5 to 10 in 35 seconds. (Subject tried in vain to stop the "avalanche phenomenon" by relaxing his stomach and practicing deep breathing.)

After the Ride Subject did not feel as nauseous after ride #2 as he did after ride #1. He was able to eat and drink without any discomfort. Due to his lengthy stay in the chair, his buttocks and legs ached for a few hours after the ride. He also developed skin rashes on his chest where electrodes had been placed.

Appendix D. *Raw Data For Figure 5*

Subject	Trial With Placebo	Trial With Dilantin
1	17:05:49-17:07:50 (14 rpm)	16:43:53-16:49:07 (14 rpm)
2	17:00:56-17:05:21 (14 rpm)	16:11:03-16:52:43 (14 rpm)
3	16:25:05-16:38:28 (16 rpm)	16:52:43-18:01:18 (16 rpm)
		18:01:18-18:07:00 (20 rpm)
		18:07:00-18:11:58 (22 rpm)
		18:11:58-18:16:57 (24 rpm)
		18:16:57-18:21:43 (26 rpm)
		18:21:43-18:23:00 (28 rpm)
4	16:33:59-17:03:47 (16 rpm)	16:13:08-17:44:00 (16 rpm)
	17:03:47-17:04:37 (18 rpm)	17:44:00-17:50:00 (20 rpm)
	17:04:37-17:12:13 (20 rpm)	
5	Data not collected	Data not collected
6	Data not collected	Data not collected
7	15:53:18-15:59:55 (17 rpm)	15:18:30-15:44:40 (18 rpm)
8	14:29:05-14:59:36 (22 rpm)	16:36:56-17:22:32 (22 rpm)
9	16:47:33-16:55:57 (20 rpm)	17:20:11-17:22:00 (18 rpm)
		17:22:00-18:30:09 (20 rpm)
10	15:37:10-15:42:39 (20 rpm)	15:50:25-16:05:30 (20 rpm)
11	16:18:51-16:25:16 (14 rpm)	15:42:23-15:51:53 (14 rpm)

Appendix E. *1988 Motion Sickness Data From Subjects on the
Placebo*

Number	EKG (bpm)	ENG (V rms)	ESG (V rms)	Thoracic Resp (V rms)	Symptom Level
1	75	0.081	0.062	0.211	1
2	82	0.076	0.171	0.428	2
3	88	0.066	0.036	0.332	3
4	85	0.069	0.064	0.304	8
5	85	0.950	0.095	1.100	10
6	86	0.100	0.038	0.630	1
7	85	0.091	0.055	0.635	2
8	78	0.118	0.043	2.010	3
9	87	0.135	0.316	2.480	10
10	66	0.103	0.123	0.291	1
11	70	0.153	0.242	0.533	1
12	79	0.191	0.830	0.610	2
13	71	0.277	1.160	0.673	3
14	66	0.080	0.023	0.261	1
15	88	0.394	0.124	0.838	2
16	75	0.284	0.105	0.687	3
17	74	0.074	0.182	0.740	3
18	76	0.132	0.259	0.616	4
19	75	0.156	0.274	0.937	5
20	75	0.121	0.320	0.523	6
21	64	0.223	0.255	0.802	7
22	71	1.470	0.503	0.793	10
23	67	0.081	0.362	0.170	1
24	73	0.175	0.064	0.388	1
25	87	0.155	0.061	0.366	2
26	70	0.161	0.100	0.706	5
27	92	0.337	0.143	0.533	7
28	92	0.277	0.099	0.242	9
29	82	0.283	3.090	0.268	9
30	76	1.040	3.650	0.263	10
31	99	0.060	0.035	0.192	1
32	93	0.135	0.247	0.577	3
33	96	0.116	0.680	0.486	8
34	103	0.098	0.413	0.901	10
35	60	0.110	0.048	0.374	1
36	70	0.093	0.047	1.100	2
37	70	0.256	0.039	0.881	4
38	77	0.302	0.039	0.897	6
39	72	0.219	0.059	0.443	7
40	75	0.168	0.029	0.487	9
41	65	0.207	0.041	1.090	10

Appendix F. 1988 Motion Sickness Data From Subjects on Dilantin

Number	EKG (bpm)	ENG (V rms)	ESG (V rms)	Thoracic Resp (V rms)	Symptom Level
1	70	0.080	0.036	0.599	1
2	85	0.103	0.050	2.250	1
3	74	0.067	2.230	2.300	2
4	81	0.378	0.135	2.730	5
5	100	0.179	0.770	3.110	10
6	86	0.168	0.019	0.467	1
7	69	0.063	0.035	0.467	2
8	69	0.059	0.031	0.533	3
9	64	0.083	0.041	0.453	1
10	62	0.108	0.044	0.592	1
11	71	0.266	0.256	0.417	2
12	74	0.070	0.082	0.888	3
13	69	0.122	0.049	1.030	10
14	57	0.140	0.032	0.132	1
15	59	0.129	0.038	0.155	2
16	61	0.129	0.020	0.505	1
17	87	0.053	0.098	0.332	2
18	69	0.101	0.111	1.210	3
19	67	0.144	0.100	0.503	5
20	64	0.184	0.253	0.873	9
21	70	0.241	0.745	1.100	10

Appendix G. *Performance-Cognition Test Results of Test Subjects*

Terminology:

GR: Grammatical Reasoning task; measures reasoning ability.

mcr: mean correct response time.

UT: Unstable Tracking task; measures manual response speed and accuracy.

edge violation: An improper response during the UT task.

PM: Probability Monitoring task; measures visual perceptual inputs (subject must respond to event seen on computer screen).

correct: Subject responded when an event occurred.

false: Subject responded when nothing was occurring.

missed bias: Subject failed to respond when an event occurred.

Table 45. Performance-Cognition Test Results: Subjects 1-6

Subject	Trial With Placebo	Trial With Dilantin
1	GR: 100% correct; 2476.827 msec mcrt. UT: 40.6 rms error; 32 ttl edge violns. PM: 10 correct; 12 false; 0 missed biases; 2.1 sec mrt.	GR: 95.23% correct; 2206.149 msec mcrt. UT: 46.3 rms error; 68 ttl edge violns. PM: 10 correct; 20 false; 0 missed biases; 2.6 sec mrt.
2	GR: 77.35% correct; 2662.487 msec mcrt. UT: 42.5 rms error; 47 ttl edge violns. PM: 10 correct; 10 false; 0 missed biases; 2.7 sec mrt.	GR: 92.30% correct; 2724.979 msec mcrt. UT: 44.9 rms error; 84 ttl edge violns. PM: 10 correct; 8 false; 0 missed biases; 2.8 sec mrt.
3	GR: 94.99% correct; 3923.394 msec mcrt. UT(low): 15.3 rms error; 0 ttl edge violns. PM: 10 correct; 2 false; 0 missed biases; 3.1 sec mrt.	GR: 97.67% correct; 3476.000 msec mcrt. UT(low): 12.9 rms error; 0 ttl edge violns. PM: 10 correct; 0 false; 0 missed biases; 4.5 sec mrt.
4	GR: 100% correct; 6284.68 msec mcrt. UT: 30.7 rms error; 2 ttl edge violns. PM: 10 correct; 0 false; 0 missed biases; 4.3 sec mrt.	GR: 96.87% correct; 4991.516 msec mcrt. UT: 46.9 rms error; 80 ttl edge violns. PM: 10 correct; 2 false; 0 missed biases; 4.3 sec mrt.
5	Not tested	Not tested
6	Not tested	Not tested

Table 46. Performance-Cognition Test Results: Subjects 7-11

7	GR: 95.83% correct; 3089.456 msec mcrt. UT: 40 rms error; 39 ttl edge violns. PM: 10 correct; 0 false; 0 missed biases; 3 sec mrt.	GR: 95.83% correct; 3082.000 msec mcrt. UT: 34.6 rms error; 34 ttl edge violns. PM: 10 correct; 1 false; 0 missed biases; 2.8 sec mrt.
8	GR: 97.43% correct; 3904.763 msec mcrt. UT: 43.2 rms error; 39 ttl edge violns. PM: 10 correct; 0 false; 0 missed biases; 3.5 sec mcrt.	GR: 97.56% correct; 3771.875 msec mcrt. UT: 34.6 rms error; 67 ttl edge violns. PM: 9 correct; 0 false; 1 missed bias; 3.8 sec mcrt.
9	GR: 97.95% correct; 3030.958 msec mcrt. UT: 34.5 rms error; 16 ttl edge violns. PM: 10 correct; 8 false; 0 missed biases; 2.9 sec mrt.	GR: 92.85% correct; 3707.615 msec mcrt. UT: 34.1 rms error; 20 ttl edge violns. PM: 10 correct; 12 false; 0 missed biases; 2.8 sec mrt.
10	GR: 93.87% correct; 3006.369 msec mcrt. UT: 46.6 rms error; 81 ttl edge violns. PM: 8 correct; 1 false; 0 missed biases; 2 sec mrt.	GR: 88.33% correct; 2416.075 msec mcrt. UT: 46.5 rms error; 136 ttl edge violns. PM: 8 correct; 5 false; 2 missed biases; 4.3 sec mrt.
11	GR: 100% correct; 3111.729 msec mcrt. UT: 46.8 rms error; 133 ttl edge violns. PM: 10 correct; 2 false; 0 missed biases; 2.4 sec mrt.	GR: 100% correct; 2994.081 msec mcrt. UT: 43.5 rms error; 71 ttl edge violns. PM: 10 correct; 5 false; 0 missed biases; 1.6 sec mrt.

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Vita

Captain Mark F. Scott was born on [REDACTED]. He graduated from North Salem High School in [REDACTED]. He graduated from the State University of New York at Binghamton with a B.A. degree in Chemistry in May, 1977. After attending Officers' Training School, he was commissioned as a second lieutenant in the United States Air Force in January, 1981.

Captain Scott's first assignment on active duty was with the Navstar Global Positioning System Program Office at Los Angeles AFB. He then attended Ohio State University under the AFIT Undergraduate Engineering Conversion Program, and graduated with a B.S. degree in Electrical Engineering in August, 1984. He was then assigned to the 351st Strategic Missile Wing Technical Engineering Branch, Whiteman AFB, MO. In May, 1987, he was assigned to the Air Force Institute of Technology, Wright-Patterson AFB, Ohio.

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