MOTION SICKNESS INCIDENCE:
EXPLORATORY STUDIES OF HABITUATION,
PITCH AND ROLL, AND THE REFINEMENT
OF A MATHEMATICAL MODEL

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

A series of experiments on human subjects assessed the effects of pitch and roll and habituation on motion sickness incidence (MSI). Pitch and roll angular accelerations, even larger than expected at sea, failed to systematically increase MSI. Habituation was evidenced in susceptible subjects who received consecutive daily 1-hour or 2-hour exposures to vertical motion. Habituation was greater for the longer exposure and the more severe motions.

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A mathematical model describing MSI as a function of the frequency and acceleration of vertical oscillation was refined by including exposure time as an independent variable. Investigation of frequencies of oscillation above .5 Hz confirmed the prediction of the model that MSI continues to decrease as a function of frequency for all frequencies greater than approximately .16 Hz.

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INTRODUCTION

The objective of the present program of research, under
the Office of Naval Research Contract N00014-73-C-0040 to
Human Factors Research, Incorporated (HFR), is to develop
equations for predicting motion sickness incidence (MSI) in
military personnel exposed to sea motion. The results of
this research, however, have potential applicability to vir-
tually all types of transportation vehicles. The first report
under this contract (O'Hanlon & McCauley, 1973) described
research that led to a preliminary mathematical model relat-
ing MSI to vertical sinusoidal motion varying in frequency
(.083 to .50 Hz) and acceleration (.027 to .55 rms g). The
present report discusses a series of exploratory experiments
on topics which were considered to be potentially important
for the prediction of MSI: pitch and roll motions, habitu-
ation, and frequencies above .5 Hz. Also, a more detailed
description of the model is presented, including its assump-
tions, limitations, derivation, and extension to include ex-
posure time.

Stomach awareness, malaise, cold sweating, pallor, nausea,
and vomiting (emesis) are common signs and symptoms of motion
sickness. This syndrome is a normal response to certain types
of motion for individuals who have an intact vestibular sys-
tem. Either angular or linear accelerations, or a combination,
can induce sickness, but the common element seems to be the
repetitive acceleration of the vestibular system. A variety
of species other than man suffers from susceptibility to motion
sickness, including dogs, cats, chimpanzees, seals, birds,
sheep, and even fish. In spite of the prevalence of motion
sickness across species, no apparent purpose, in the teleo-
logical sense, is served by vomiting in response to repetitive
acceleration. Several comprehensive reviews of motion sickness

discuss these issues in detail (Chinn & Smith, 1955; Money, 1970; Reason & Brand, 1975; Tyler & Bard, 1949).

The vestibular system plays a primary role in motion sickness. As early as 1882, William James asserted that individuals with a non-functional vestibular system were immune to motion sickness. Research by Graybiel and his colleagues has clearly demonstrated this immunity in a variety of dynamic environments (e.g., Kennedy, Graybiel, McDonough & Beckwith, 1968). Both the semicircular canals and the otolith organs are implicated in the etiology of motion sickness, but their exact role and their mutual influence remain controversial. Discussions of this issue may be found in Reason & Brand (1975) and in several papers in the NASA Fourth Symposium on the Role of the Vestibular Organs in Space Exploration (Graybiel & Miller, 1970; Guedry, 1970).

Angular acceleration is the primary stimulus to the semicircular canals and linear acceleration is the primary stimulus to the otolith organs. Either angular or linear acceleration can induce motion sickness, and so can a dynamic visual display with implied but not real vestibular stimulation. Reason & Brand (1975) argue for a "neural mismatch" explanation of motion sickness, stating that

> *All* situations which provoke sickness are characterized by a condition of sensory rearrangement involving the vestibular system; that is, a condition where the position and motion information signalled by one or more of our spatial senses [semicircular canals, otoliths, or vision] is in some way discrepant with that signalled by the remainder. (p. 134)

The motion of vehicles on land, sea, or air can be conducive to motion sickness, depending on the environmental forces imparting acceleration to the vehicle. The relationship between the dynamics of the vehicle and the resultant motion sickness is largely unknown (Allen, 1974). Field studies provide one method for investigating that relationship by

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correlating measures of vehicle dynamics with MSI. However, the field study method presents difficult problems of measurement. Ideally, six axes of motion, three linear and three angular, should be quantified. Linear components of angular acceleration, such as pitch motion at the bow of a ship, may be significant if the distance of the individual from the axis of rotation is great. The movement of personnel within the vehicle, therefore, can cause difficulty in quantifying the dynamic stimulus for an individual over time. The problems encountered with the field study method are documented in an unsuccessful attempt to correlate measures of ship's motion with motion sickness (Hanford, Cone & Gover, 1953).

Another approach to the problem of relating motion parameters to MSI is to experimentally control and manipulate the motion of laboratory devices. This method has utilized a variety of devices such as two-pole and four-pole swings, rotating chairs, rotating rooms, and vertical oscillators. Since World War II a number of studies have used these devices and the results have been reviewed by Money (1970). Despite the research effort, our knowledge is far from complete. Benson (1973), in a recent review of the physical characteristics of the motions that induce sickness, concluded that

> There is a paucity of data correlating stimulus parameters to the incidence of sickness....In situations where there are concomitant angular or linear accelerations, as invariably occurs outside the laboratory in transport or fighting vehicles, there is essentially no information which would allow a quantitative assessment of the incidence of sickness in operators or passengers when exposed to a particular vehicular motion. (p. 15)

Vertical linear acceleration, sometimes called "heave," has been implicated as the component of sea motion that is most important in motion sickness, primarily because the level of angular acceleration in pitch and roll is quite low in conventional sea craft, usually less than 5 degrees per second

per second ($°/sec^2$) (Morales, 1949; Sjöberg, 1970). A series of studies on vertical oscillation was begun by Wendt at Weslyan University during World War II, and has been reviewed by Baker (1966), Benson (1973), and Money (1970). The apparatus used in these studies, called the "Wave Machine," was a hydraulically driven, modified elevator with an 18-foot full-wave displacement amplitude. The waveforms were characterized by alternating periods of constant velocity and constant acceleration. According to Baker (1966) and Morales (1949), the displacement waveforms approximated a sinusoid, but the acceleration waveforms were essentially a square wave. Seated subjects were exposed for 20 minutes to frequencies from 13 to 32 cycles per minute (CPM), and accelerations ($\pm a_z$) ranged from .20 g to .65 g. Results of these studies indicated that both frequency and acceleration were important in motion sickness, and that frequencies lower than 32 CPM were more likely to induce sickness. Equipment limitations prevented the independent manipulation of frequency and acceleration, and the relationship between these variables was not discovered.

The first study in the present program of research (O'Hanlon & McCauley, 1973) extended the Wendt-Weslyan data by independently varying the frequency and acceleration of vertical oscillation. Over 300 subjects were exposed to motion for a duration of 2 hours. Frequencies of 5, 10, 20, and 30 CPM (or .083, .167, .333, and .500 Hz respectively) were tested. The frequency of maximum sensitivity to motion sickness was found to be 10 CPM (.167 Hz) in contrast to the Wendt-Weslyan estimate of between 16 and 22 CPM. In addition, MSI was found to increase for all frequencies as a monotonic function of the acceleration. A mathematical model was derived for the prediction of MSI in a 2-hour exposure based on the parameters of frequency and acceleration. However, the data for that model was frequency limited to an upper bound of

.5 Hz and important variables such as exposure time, habituation, and simultaneous angular accelerations were not included in the model.

The purpose of the present experiments was to investigate the effects of the following variables on the incidence of motion sickness: (1) angular motions of pitch and roll up to and beyond the magnitude of angular accelerations expected from sea motion; (2) habituation of the motion sickness response through successive daily exposures to motion; and (3) frequencies of vertical oscillation above .5 Hz. A major goal was to refine the mathematical model for the prediction of MSI by including new data as well as an expression for exposure duration.

GENERAL METHOD

Apparatus

Motion was imparted to the subjects using the Office of Naval Research/Human Factors Research (ONR/HFR) Motion Generator (Buckner & Baker, 1969; Buckner & Heerwagen, 1969; O'Hanlon, Seltzer & Sanderson, 1975). The facility includes a control room and a moving cabin which is mounted on a hydraulically driven piston, capable of a vertical full-wave displacement amplitude (heave) of approximately 20 feet (6.1 meters). Additionally, the Motion Generator provides pitch and roll angular displacement limits of approximately ±15° on each axis. The axes of rotation are approximately 16 inches (40 cm) below the deck of the moving cabin.

The Motion Generator was equipped with a moving cabin of approximately 8 feet x 8 feet x 8 feet (2.4 m) with an insulated partition dividing it into two identical compartments so that two subjects could be exposed to motion simultaneously. Each compartment contained an air conditioning system, an aircraft type seat with headrest and safety harness, a headset mounted on the headrest, a closed-circuit television camera, a fluorescent light, an emesis bag, a symptom-rating chart, and a small response console with five buttons. The five response buttons were numbered, and the symptom-rating chart defined the buttons as follows:

Button Number		Symptom Rating
1	-	No symptoms
2	-	Stomach awareness, feeling slightly "queasy"
3	-	Mild nausea
4	-	Moderate nausea
5	-	Severe nausea, emeis is imminent

The control room contained the communications and control equipment. Parallel communications systems and TV monitors for the two compartments allowed the experimenter to monitor the subjects' progress and communicate with them independently. A sinusoidal drive signal was produced by a function generator (Systron-Donner Corp., Data Pulse 401) and provided the input to the Motion Generator heave servo control system. The frequency and the cabin half-wave displacement amplitude were accurate to within approximately .005 Hz and 2 inches respectively.

Major modification of the ONR/HFR Motion Generator heave system occurred during the time period of the present experiments, substantially upgrading both its frequency and the acceleration characteristics. Appendix A contains information regarding Motion Generator pre- and post-modification drive systems.

Subjects

Students were recruited from four local educational institutions with a combined enrollment of approximately 20,000--the University of California, Santa Barbara; Santa Barbara City College; Brooks Institute of Photography; and Westmont College. Male and female subjects participated in two experiments on habituation; the remainder of the studies employed male subjects exclusively. The subjects were screened by questionnaire for contraindicating medical conditions such as diabetes, heart disease, high blood pressure, and epilepsy. They were paid $10 for an experimental session which lasted either 2 hours or until emesis.

Procedure

The procedures described in this section may be considered "standard" for the three studies reported in this paper. Any deviation from these procedures will be noted.

The two subjects in each session listened to tape recorded instructions and signed informed consent forms. They were seated in their respective compartments of the moving cabin, and the safety harnesses were secured. They were instructed to maintain head position against the headrest and respond to a 1-second tone given each minute over the headset by pressing the appropriate symptom-rating button on the response console. When the compartment doors were secured, no earth-fixed visual reference was available for the subjects, although the fluorescent light enabled TV monitor operation and normal vision within the compartment. The motion was started after a final communications check, and the assigned motion was attained by a gradual increase of the input amplitude over a period of approximately 30-60 seconds. The subjects' symptom development was observed on a strip-chart recording of the symptom ratings and on the TV monitors. If a subject vomited, an automatic motion-stop procedure was initiated, requiring 9 seconds, and he was removed to a recovery bunk. The motion was restarted for the remaining subject as soon as possible, generally within 3 minutes. Although this procedure disturbed the constant stimulus to the remaining subject, the delay did not appear to offer more than a brief respite from motion sickness symptom development. In some studies, noted later, subjects were encouraged to remain in motion for the full 2 hours, even after emesis. After a session, the subjects were requested to remain until fully recovered. A local physician was retained for consultation and for emergency medical care.

In operational sea craft, the angular accelerations of pitch and roll that are imparted to the occupants are concomitant with vertical acceleration or heave. Therefore, accurate prediction of motion sickness incidence in Naval operations must account for these combined effects. Angular acceleration is an effective stimulus for motion sickness when it is cross-coupled with head movements in an orthogonal axis leading to Coriolis acceleration of the semicircular canals (Benson, 1973; Graybiel, 1972; Guedry, 1965). However, the contribution of the angular acceleration of pitch and roll to motion sickness on ships is not well documented. Several investigators (e.g., Morales, 1949; Sjöberg, 1970) have asserted that pitch and roll are relatively unimportant compared to heave motion because the angular acceleration aboard ships is generally very low, on the order of $5°/sec^2$. For example, a roll angle of $10°$ aboard a ship would be very perceptible by otolith and proprioceptive sensors, but because of the long roll period, the angular acceleration and hence the stimulation of the semicircular canals would be low. Consequently, the angular accelerations of pitch and roll per se may play a minimal role in the etiology of motion sickness aboard conventional sea craft. But the vertical linear component of roll and particularly of pitch could be substantial, depending on an individual's location on the ship with respect to the axis of rotation.

The purpose of Study I was to determine how motion sickness incidence is affected by the addition of the angular accelerations, pitch or roll, to a constant vertical motion.

Method

Subjects. The subjects were 325 male college students.

Procedure. A sinusoidal vertical oscillation was defined
by a frequency of .25 Hz and an rms acceleration level of
.11 g yielding a half-wave displacement amplitude of 2.05 feet.
That motion was predicted to give a moderate MSI of 25% in
2 hours, based on the model developed from the earlier work
under this contract (O'Hanlon & McCauley, 1973), although the
effects of this particular frequency had not been previously
observed. There were a total of 15 experimental conditions
of motion (see Table 1)--6 pitch + heave conditions, 6 roll +
heave conditions, and 3 control conditions, pitch-only, roll-
only, and heave-only.

A function generator produced sinusoidal command signals
for the angular motion, and the gain control was increased
until the assigned angular displacement of the cabin was attained
to within 1 degree. Six angular motions were defined on the
basis of a partial factorial design of three frequencies--.115,
.230, and .345 Hz--and three levels of rms acceleration--5.5,
16.7, and 33.3°/sec^2. The six angular motions were super-
imposed upon the standard heave motion and defined as pitch
or roll according to the axis of rotation with respect to the
seated subject. The linear components of the six angular
accelerations were, in all cases, less than .10 rms g accel-
eration at the ear, by calculation for a hypothetical subject
of mean sitting height. The heave-only control condition had
no superimposed angular accelerations; the pitch-only and the
roll-only control conditions had no heave motion.

Eight subjects were scheduled each day and nonsystemat-
ically assigned to one of the 15 motion conditions. Scheduling
continued until a minimum of 20 subjects had been exposed to
each condition. Because of the pitch and roll accelerations
in this study, head restraints, constructed of rubber tubing,
assured that the subject's head remained positioned against
the headrest.

Results and Discussion

The observed MSI in the 15 motion conditions is shown in Table 1. The MSI in the heave-only control condition was 31%, just 6% more than predicted by the model. In contrast, no subjects vomited in the roll-only control condition, and only two (9%) vomited in the pitch-only control condition. Inspection of the six pitch + heave conditions in Table 1 revealed no apparent systematic effect of frequency or acceleration. A chi-square analysis based on an expected MSI of 31% was not significant, $X^2 = 2.17$, df = 5, p > .05, indicating that the addition of the six conditions of pitch motion led to no change in MSI other than what might be expected from the heave motion alone.

Inspection of the six roll + heave conditions revealed a high variability in MSI, with two particularly low values of 14% at .115 Hz, 5.5°/sec^2, and 8% at .345 Hz, 16.7°/sec^2. Yet no systematic effects of frequency or acceleration were apparent. There is no ready explanation for the inversion at a frequency of .345 Hz and an acceleration of 16.7°/sec^2; the reason for the low value is unknown. However, a chi-square analysis supports an interpretation that the results were due to chance variation. The obtained frequency of emesis did not differ significantly from the 31% expected from heave alone, $X^2 = 9.89$, df = 5, p > .05. We are unaware of any vestibular process or theory of motion sickness that would predict a reduction in motion sickness, as in the cells with 8% and 14% MSI, due to the addition of roll motion to heave motion. The overall mean of the MSIs for the pitch + heave conditions was 34% and for the roll + heave conditions, 31%. These data are consistent with the view that the 12 motion conditions of angular acceleration did not differ from the heave-only control condition, and that the inter-cell variation was due to sampling variability.

TABLE 1.

MOTION SICKNESS INCIDENCE (MSI) AS A FUNCTION OF FREQUENCY AND ACCELERATION FOR PITCH + HEAVE, ROLL + HEAVE, PITCH, AND ROLL ALONE. ALL HEAVE MOTIONS WERE AT A FREQUENCY OF 0.25 Hz AND AN rms ACCELERATION OF 0.11 g

EXPERIMENTAL CONDITIONS

Pitch + Heave
rms Acceleration (deg/sec²)

Frequency (Hz)	5.51	16.7	33.3
.115	36% N=22		
.230	40% N=20	40% N=20	
.345	24% N=21	25% N=20	38% N=21

Roll + Heave
rms Acceleration (deg/sec²)

Frequency (Hz)	5.51	16.7	33.3
.115	14% N=21		
.230	43% N=21	40% N=20	
.345	35% N=20	08% N=26	48% N=21

CONTROL CONDITIONS

Pitch-Only
rms (deg/sec²)
33.3

Frequency (Hz)	
.345	09% N=22

Heave-Only
rms g
.11

Frequency (Hz)	
.250	31% N=29

Roll-Only
rms (deg/sec²)
33.3

Frequency (Hz)	
.345	00% N=21

Overall, the most notable, and somewhat surprising, re-
sult from Study I was the failure of pitch or roll to con-
sistently increase the incidence of sickness observed in the
heave motion alone. Whether some complex interaction between
the roll and heave accelerations led to increased variability
in MSI remains a viable question. For example, perhaps the
neural effect of vertical acceleration is modulated by the
average angular position (tilt angle) of the otolith organs
at the time the acceleration is applied. Such an effect could
possibly account for some of the variability observed in these
results. The failure to find a systematic increase in MSI
from pitch and roll supports previous investigators who sug-
gested that the vertical component of sea motion is of pri-
mary etiological significance for motion sickness. This result
also casts doubt on previous suggestions that slight head
movements during vertical oscillation are the basis for motion
sickness (Graybiel & Miller, 1970; Reason & Brand, 1975).

STUDY II
HABITUATION TO MOTION THROUGH DAILY EXPOSURE

Adaptation and habituation are closely related terms, generally referring to a change in response to an input that is constant or repeated. Money (1970) discusses these processes with regard to motion sickness and defines adaptation as a change in the bodily mechanisms which leads to a response decline, and habituation as the acquisition or process of acquring the adaptive change. The term habituation will be used in this report to describe a decline in the incidence of motion sickness with repeated or continued exposure. It is recognized that this definition is not descriptive of the underlying processes. Collins (1973) reviewed habituation of vestibular responses, particularly for angular acceleration, and concluded that mere response reduction is an inadequate conception of vestibular habituation, and that the dynamic processes involved would be better characterized as "active modification" of vestibular responses. A similar viewpoint, expressed by Reason and Brand (1975), is a major feature of the sensory rearrangement theory of motion sickness. An increased research effort on these theoretical issues is needed in order to advance the understanding of motion sickness as well as adaptive processes.

However, practical rather than theoretical considerations motivated the present study because any prediction of MSI for extended operations in a dynamic environment must take into account the degree of habituation that has been acquired. Several important issues arise in the area of adaptive changes in response to motion: the time course of habituation, the persistence of the change (rate of dishabituation), the specificity of the change, and the extent of positive and negative transfer of the habituation to different motion conditions.

Study II was designed as an exploratory investigation of habituation in repeated exposures to vertical oscillation. The study consisted of three experiments examining differences in acceleration, duration of exposure, and sex of the subjects.

Experiment 1

The purpose of Experiment 1 was to observe the effect of repeated daily 2-hour exposures to the same sinusoidal motion on the MSI of susceptible subjects. A decline in the incidence of emesis as a function of days of exposure was considered to be evidence of habituation.

Subjects. Thirty-four out of 54 subjects were selected for susceptibility on the basis of vomiting within 2 hours in response to the standard motion for this experiment. Only 20 of the 34 susceptibles agreed to return for the series of five habituation exposures. The procedure for subject selection was identical for all three experiments in Study II, and the results of the selection process are shown in Table 2.

TABLE 2
DESCRIPTION OF MOTION, SUBJECTS, AND OUTCOME
OF SELECTION PROCESS FOR STUDY II

Experiment No.	Motion			Selection Test				Began Habituation Series	
			Half-Wave Amplitude (ft)	N		MSI		N	
	Freq (Hz)	rms Accl (g)		M	F	M	F	M	F
1	0.25	0.22	4.09	54	--	63%	--	20	--
2	0.25	0.33	6.14	45	9	69%	89%	8	6
3	0.417	0.44	2.94	15	12	40%	58%	4	4

Procedure. A sinusoidal motion at a frequency of .25 Hz and rms acceleration of .22 g, yielding a half-wave displacement amplitude of 4.1 feet, was defined as the standard heave motion throughout the experiment. This motion was predicted, on the basis of the mathematical model (O'Hanlon & McCauley, 1973), to result in a 52% MSI within 2 hours.

An unsuccessful attempt was made to establish a minimum of 5 days between the selection exposure and the first of the five habituation sessions; some subjects began the habituation

series 2 to 3 days after the selection exposure because of conflicts with school class schedules.

   Results. The MSI for the 20 subjects (1 subject = 5%) is shown in Figure 1 as a function of consecutive days of exposure. Although 100% of the subjects had vomited on their selection day, only 75% vomited on Day 1 of the habituation series. This reduction in MSI may have been due to a combination of three factors: (1) residual habituation attribuable to the selection day exposure, (2) nonspecific habituation or reduction in anxiety to the total testing situation, and (3) regression toward mean susceptibility in subjects who were selected for high susceptibility. The habituation series of five daily exposures resulted in a monotonic and negatively accelerating decrease in MSI. However, six subjects (30%) still vomited on Days 4 and 5.
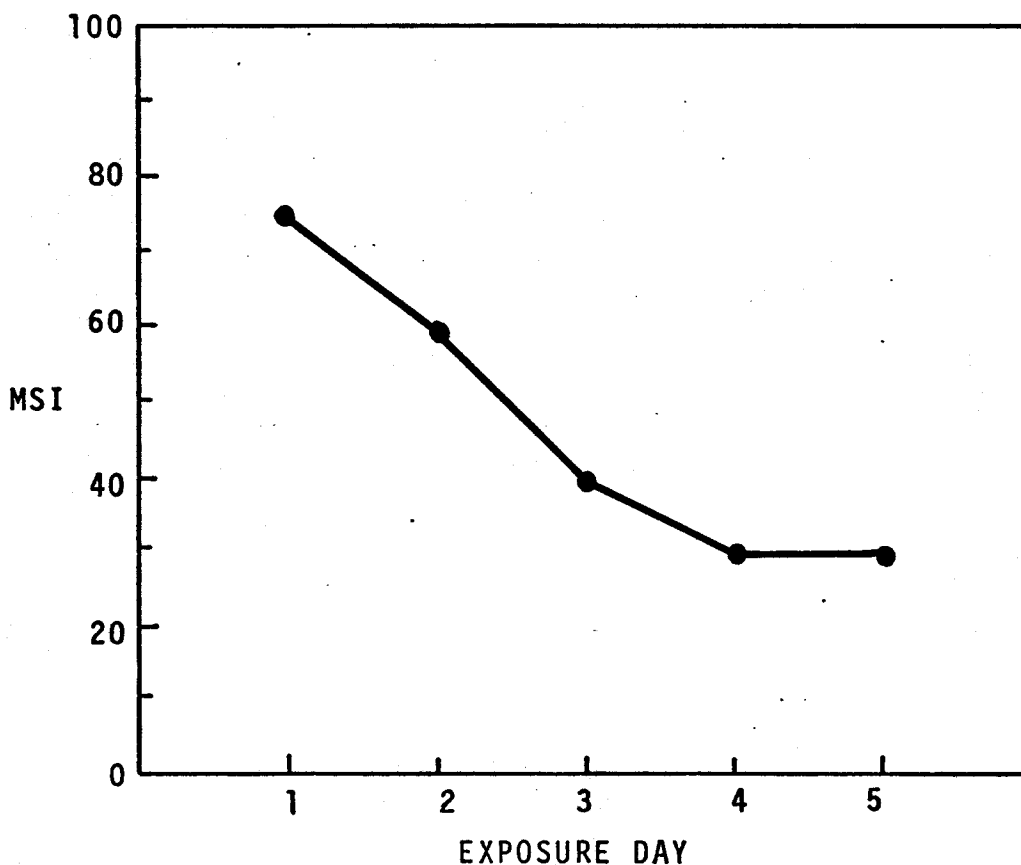

Figure 1. MSI over five daily 2-hour habituation exposures to vertical sinusoidal oscillation at 0.25 Hz and 0.22 rms g in Experiment 1 (N = 20).

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Experiment 2

The primary purpose of Experiment 2 was to determine the effect of the severity of motion on habituation. A secondary purpose was to observe the retention or maintenance of habituation. Experiment 2 also was designed to provide data regarding differential·susceptibility to motion sickness between male and female subjects.

Subjects. There were 8 males and 6 females who participated in the habituation series. These subjects were selected for susceptibility from a total of 31 males and 8 females by the same procedure as in Experiment 1. The results of the selection testing are given in Table 2.

Procedure. The motion for the second habituation experiment was sinusoidal vertical oscillation at the same frequency as Experiment 1, .25 Hz, but at a greater rms acceleration, i.e., .33 g. The 14 subjects who agreed to return (3 to 7 days later) were given a series of five daily 2-hour exposures to the same motion as in the selection test. One week after the final day of the habituation series, the subjects returned for a 2-hour "retention" test, again in the same motion. The purpose of this test was to evaluate the retention of any habituation that may have been acquired during the previous week. The standard procedures were followed with the addition of encouraging subjects to remain in the motion for 2 hours even if they became sick and vomited. If a subject requested to terminate the run after emesis, however, the motion was stopped immediately, the subject was removed, and the motion was restarted for the remaining subject within 3 minutes.

Results. During the course of the habituation series, 5 of the 14 subjects dropped out of the study. One decided the task was too unpleasant, 3 became ill with the flu, and the experimenter terminated 1 subject because of extreme

susceptibility to motion sickness.[1] Nine subjects completed all five habituation exposures and the retention exposure of the following week. Figure 2 shows the MSI for all subjects and for the 9 subjects who completed the series across the six exposures. There was a general decrease in MSI over the 5 days of habituation similar to that shown in Experiment 1. The large decrease in MSI from Day 1 to Day 2 with the loss of only one subject indicates that the habituation effect was not simply an artifact based on non-random subject loss. The small sample size (N = 9 by Day 4) prohibited meaningful

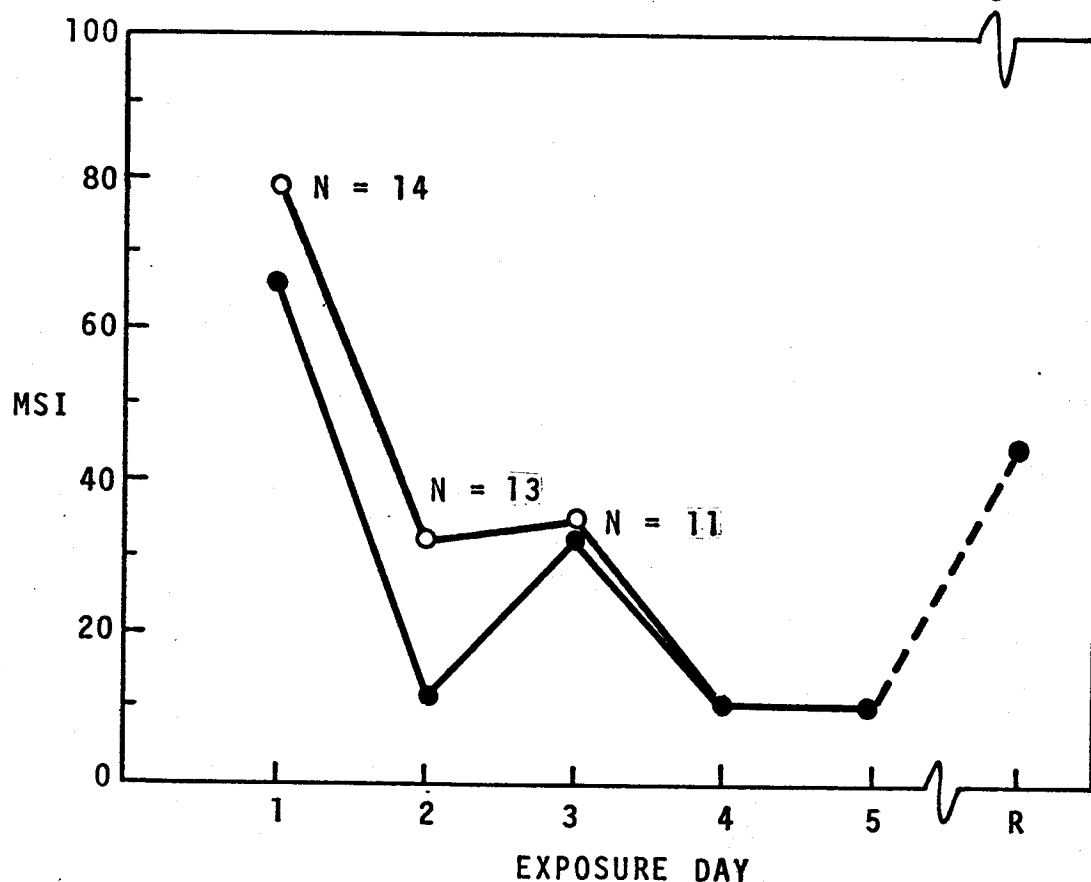Figure 2. MSI over five daily 2-hour habituation exposures and retention of habituation after 1 week. Motion was vertical sinusoidal oscillation at 0.25 Hz and 0.33 rms g in Experiment 2. (N = 9, closed circles.)

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[1]The subject did not request to stop, but the experimenter felt that termination was advisable after 3 days of multiple emesis and no apparent decline in the severity of the motion sickness symptoms.

statistical analysis of these data, but a decreasing suscep-
tibility to motion sickness was evident for the group as a
whole over five daily 2-hour exposures to the same motion
condition.

The retention data were obtained 1 week after the 5th
day of the habituation series. The results, shown as "Ex-
posure Day R" in Figure 2, indicate that some degree of
habituation may have been retained after a week without
exposure to motion. Considering only the 9 subjects who
participated in the retention test, 6 vomited (MSI = 67%) on
Day 1 of the habituation series, 1 vomited (11%) on Day 2,
and 4 vomited (44%) on Day R. Therefore, a maximum of 2
subjects may have been protected from vomiting by retained
habituation. This interpretation must be considered very
tentative due to the limited size of the sample. There were
no significant differences in response between male and female
subjects (see Figure 6).

Experiment 3

Experiment 3 was designed to investigate the effect of
exposure duration on habituation and the retention of habitu-
ation. Again, differential susceptibility of males and fe-
males was a secondary question.

Subjects. A total of 27 male and female students partic-
ipated in the selection tests for the third experiment as shown
in Table 2. Thirteen qualified for the habituation series
based on their demonstrated susceptibility and 8 subjects,
4 males and 4 females, agreed to participate in the series.

Procedure. The selection testing consisted of 2-hour
exposures to vertical oscillation at a frequency of .417 Hz
and .44 rms g acceleration, a motion predicted to yield an
MSI of 52%. The series of five habituation runs consisted
of 1-hour exposures to the same motion as in Experiment 2,
i.e., .25 Hz and .33 rms g acceleration. In both the selec-
tion and the habituation trials, subjects were encouraged but

not required to remain in the simulator for the entire time, even if they vomited. The time interval between selection and the first exposure of the habituation series ranged from 1 to 2 weeks. One week after the last habituation exposure, all 8 subjects were asked to return for a 2-hour retention test.

Results. All 8 subjects completed the habituation series, but only 5 returned for the retention test, 2 males and 3 females. The results were qualitatively similar to Experiments 1 and 2; habituation was evidenced by decreasing MSI with days of exposure (Figure 3). The increase in MSI from Day 3 to Day 4 (20%) was not a meaningful change since it was the
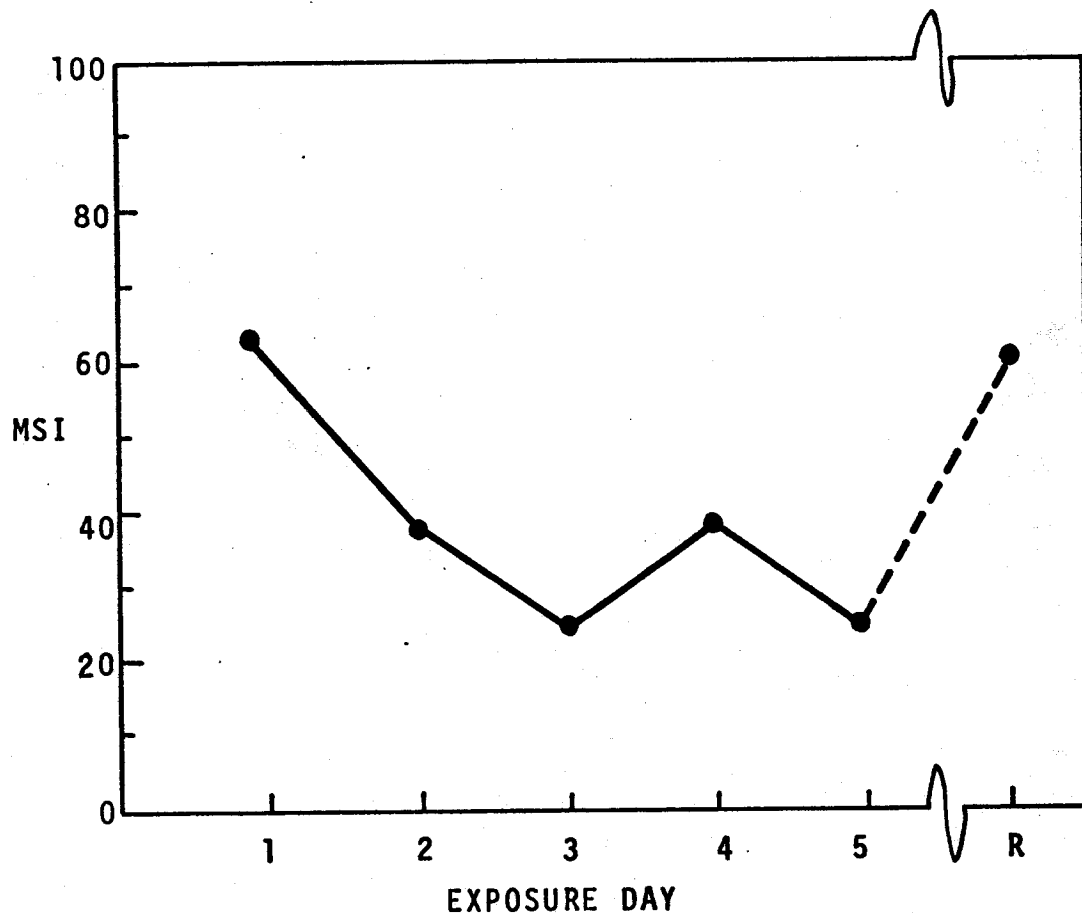
Figure 3. MSI as a function of five daily 1-hour habitu-ation exposures and a 2-hour retention exposure 1 week later to vertical sinusoidal motion of 0.25 Hz and 0.33 rms g in Experiment 3. (Days 1-5, N = 8; Day R, N = 5.)

result of only 1 subject vomiting. In fact, calculating an MSI with sample sizes less than N = 20 must be considered only a very crude estimate of the true population parameter. Again, the differences between males and females did not appear to be significant, but the sample size was too small to make an effective comparison.

The retention data, collected 1 week after the habituation series, indicated that any habituation acquired during the 5-day series of 1-hour exposures was not sustained over a week without exposure to motion. This result cannot be considered firmly established, however, because of the small number of subjects involved (N = 5).

General Discussion

Compilation of the data from the three experiments of Study II indicated that the time course of habituation to motion sickness was a negatively decreasing function of exposures. In all three studies, the greatest decrease in MSI occurred on the 2nd day and habituation continued to be acquired at a slower rate thereafter.

The results of Experiments 1 and 2, which differed only in acceleration, are compared in Figure 4. Greater habituation to motion sickness was acquired in the more severe motion, .33 rms g in Experiment 2, than in the less severe motion, .22 rms g in Experiment 1. One possible explanation is derived from a similar situation discussed by Reason and Brand (1975). They cite evidence that controlled head motion during rotation hastened the development of habituation. In a rotating environment, motion sickness symptoms can be prevented by maintaining a static head position, but this inactivity also precludes habituation. The authors suggest that while increased head movement hastens habituation in a rotating device, it probably has little effect on the rate of habituation "in a situation where one is passively exposed to the motion such as on a ship" (Reason & Brand, 1975). Possibly, exposures

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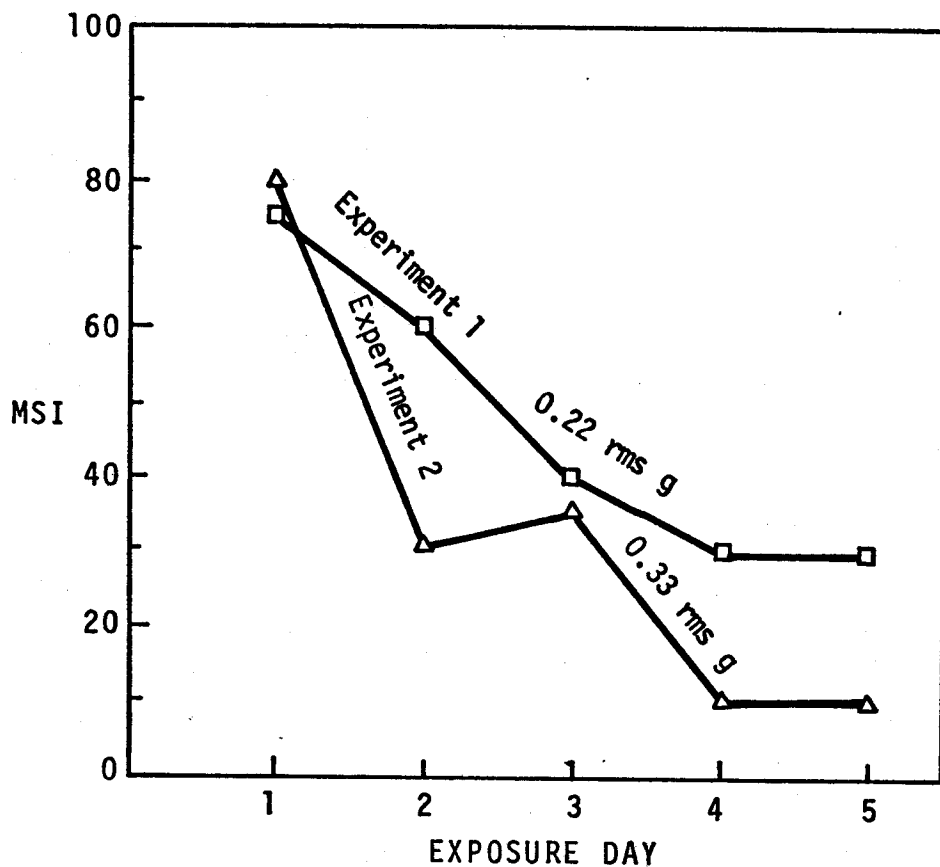
Figure 4. MSI as a function of five 2-hour
habituation trials for Experiment 1 (0.25 Hz,
0.22 rms g) and Experiment 2 (0.25 Hz, 0.33
rms g).

to vertical motions of different severity would have a similar
effect on habituation as head movements do in rotation; the
more severe vertical oscillation would be analogous to greater
head movements during rotation, yielding a greater incidence
of motion sickness but also hastening habituation. Further
research is required in order to more fully understand these
variables.

The data from Experiments 2 and 3, as depicted in Figures
2 and 3, respectively, are based on 5 days of exposure to
the same motion (i.e., .25 Hz, .33 rms g). The only differences
are sample sizes and, more important, exposure times. The
purpose of Experiment 3 was to test the effects of adaptation
to 1-hour of motion per day as compared to 2 hours in Experiment 2.

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By comparing Figures 2 and 3 it can be seen that there are
two differences between them. With the 1-hour exposure
(Figure 3), the initial MSI was lower (63% as opposed to 79%),
and the curve was flatter, yielding a higher MSI on the final
day of the series (25% compared to 11%). On the first day,
the MSI was less with a 1-hour exposure than with a 2-hour
exposure because MSI is a function of exposure time. The
data from the 1st hour of each exposure in Experiment 2 and
from the 1-hour exposures in Experiment 3 are presented in
Figure 5. This presentation equates the data for exposure
time but allows a comparison of the degree of habituation
acquired by exposure to motion for 2 hours per day rather than
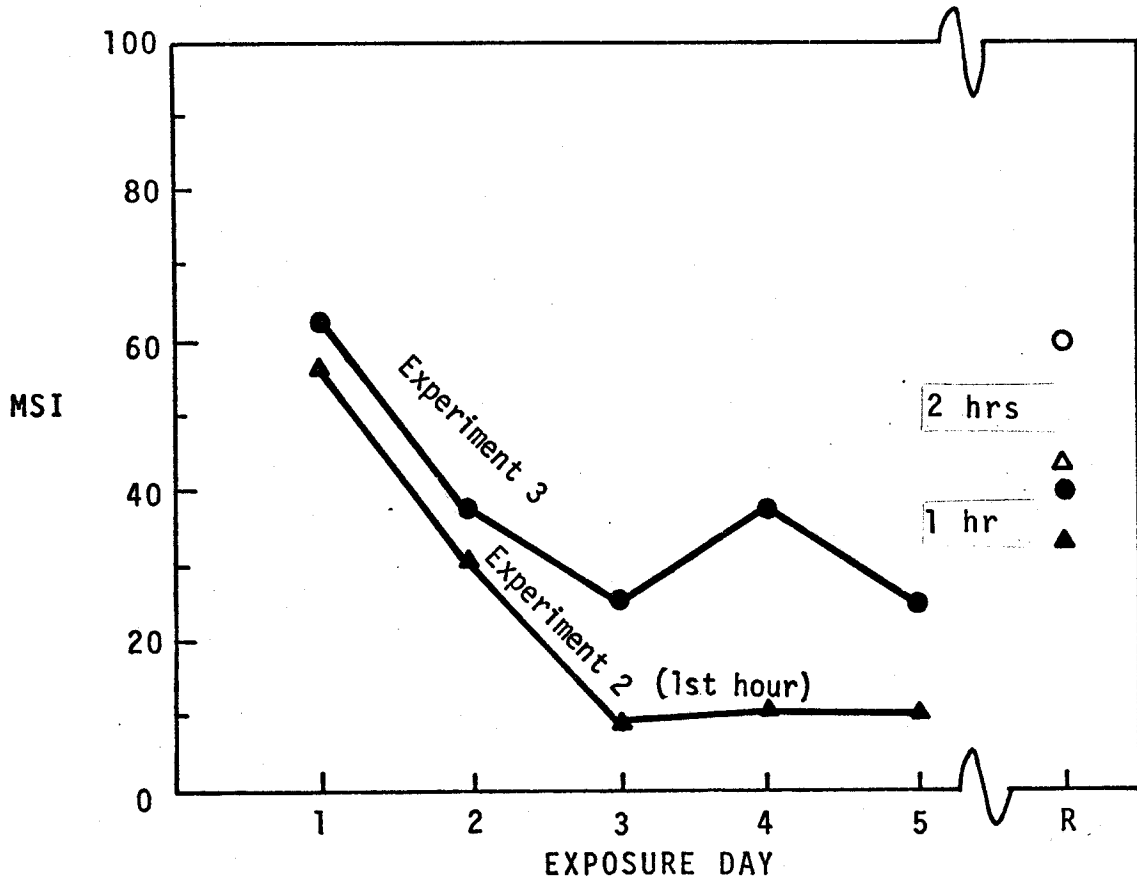
Figure 5. MSI as a function of days of exposure for
the 1st hour of each exposure in Experiment 2 (2-hour
exposure durations) and for Experiment 3 (1-hour ex-
posure durations) and retention data after 6 days
without motion, for the 1st hour (closed symbols) and for
2 hours (open symbols).

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1 hour per day. The initial MSI for the two groups was essentially equal, but by the 3rd day the group with the additional hour of daily exposure had a lower MSI, indicating greater habituation. This same effect is reflected in the retention tests; the longer daily exposure to motion resulted in greater retention of habituation.

Comparison of susceptibility in males and females for the selection runs of Experiments 2 and 3 revealed a combined MSI of 62% for the males and 71% for the females, but this difference was not statistically significant, $Z = .74$, $p > .05$. This result is equivocal with respect to the support of previous findings of sex differences in susceptibility to motion sickness (Reason & Brand, 1975). The habituation data for males and females from Experiments 2 and 3 have been combined in Figure 6. The MSI on the 1st day of the habituation series
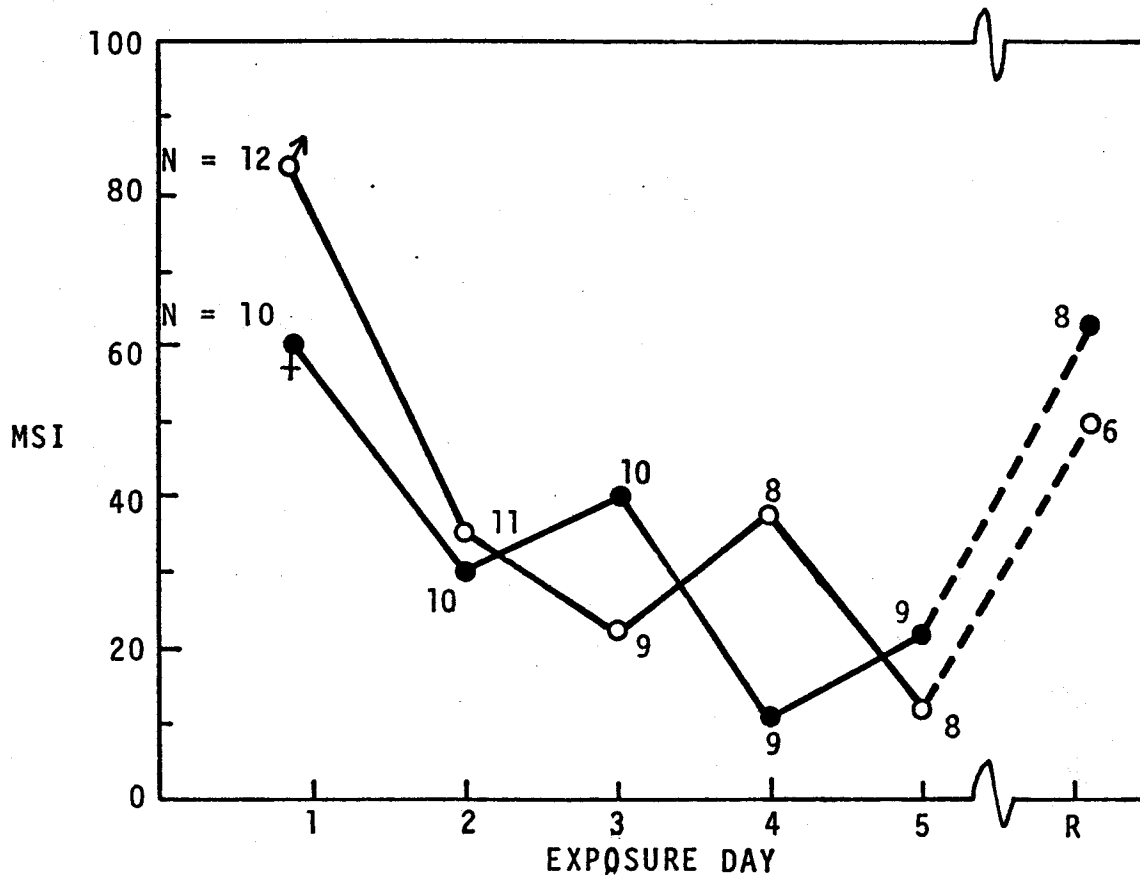
Figure 6. MSI for males and females as a function of days of exposure from Experiments 2 and 3 combined.

was greater for males than females, 83% and 60%, respectively, but the final MSI after five habituation trials was less for the males, 12%, than for the females, 22%. This final difference was not statistically significant, $Z = .54$, $p > .05$, and there are many reversals in the trends. The variation in MSI within the habituation series was apparently due to small sample sizes, precluding a definitive statement about sex differences in habituation.

The data on habituation have shown that five daily 2-hour exposures to a relatively severe motion led to a greater degree of habituation which was better retained than either (a) 1-hour exposures to the same motion or (b) 2-hour exposures to a slightly less severe motion. The current data are not sufficiently extensive to describe the relationships between the number of exposures, the time between exposures, and the length of exposures. Further research on habituation to motion is necessary to quantify the effects of these variables on the acquisition and retention of habituation, and to provide information for developing a comprehensive model for motion sickness.

STUDY III
VERTICAL OSCILLATION AT FREQUENCIES FROM .5 TO .7 HZ

The purpose of this study was to extend the data base for MSI in vertical oscillation to frequencies greater than .5 Hz.

The mathematical model derived from the previous study (O'Hanlon & McCauley, 1973) indicated that approximately .2 Hz was the frequency of maximum sensitivity to motion sickness, with MSI decreasing at higher frequencies, up to .5 Hz. The frequency range from .5 Hz to 1.0 Hz has rarely been investigated. Research with the ONR/HFR Motion Generator and the Wendt-Weslyan Wave Machine have been limited to frequencies below .50 Hz and .53 Hz, respectively, and studies of vibration have traditionally considered 1.0 Hz as a lower bound (Allen, 1971; Hornick, 1972). An excellent review of vibration above and below 1 Hz is given by Guignard and King (1972). Several recent psychological studies from the vibration domain have included values below 1.0 Hz in establishing "equal comfort" contours (Shoenberger, 1975; Yonekawa & Miwa, 1972; Holloway & Brumaghim, 1972). However, this type of study has usually been limited to a low level of acceleration because of the large displacement amplitudes required with frequencies below 1.0 Hz. The modification of the ONR/HFR Motion Generator (see Appendix A) provided the frequency and acceleration capability for extending the investigation of MSI into the region between .5 Hz and 1.0 Hz.

Method

Subjects. The subjects were 101 male students.

Procedures. Four motion conditions were defined by frequency and acceleration as shown in Table 3. The lowest frequency in this experiment, .5 Hz, was equal to the highest frequency previously investigated at this facility. A different motion condition was given each day, and eight subjects per day were scheduled by unsystematic assignment to a motion condition. This procedure was continued until at least 20

TABLE 3

MOTION PARAMETERS, NUMBER OF SUBJECTS, AND
PREDICTED AND OBTAINED MSI IN STUDY III

Condition					
Frequency (Hz)	Acceleration (rms g)	Half-Wave Displacement Amplitude (ft.)	N	Predicted MSI (%)	Observed MSI (%)
1. .50	.55	2.54	24	42	42
2. .60	.55	1.77	22	22	18
3. .60	.44	1.41	25	16	8
4. .70	.55	1.30	24	10	4

subjects were run in each condition. The procedures were stan-
dard: 2-hour exposures, no earth-fixed visual reference, and
symptom ratings each minute.

Results and Discussion

     The MSI for the four motion conditions is given in Table
3, along with the MSI that was predicted from extrapolation
of the model. These results indicate that the mathematical
model based on data up to .5 Hz was reasonably accurate, given
the sample size, for predicting MSI up to .7 Hz. Further
investigation of the effects of frequency up to 1.0 Hz, and
perhaps beyond, is necessary to allow detailed analysis of the
upper-frequency boundary of motion sickness. But, the present
results confirm the prediction of the model, that only relatively
high accelerations (>.55 rms g) would be expected to produce
motion sickness at frequencies above .7 Hz. Accelerations of
this magnitude may produce undesirable effects other than mo-
tion sickness; for example, in broadband vehicle motion of un-
restrained passengers, acceleration peaks could exceed 1.0 g
causing potential bodily injury from free-falling. Jex, DiMarco,
and Schwartz (1974) have characterized this region as the
"terror regime" where criteria other than motion sickness
must be considered of primary importance.

A MATHEMATICAL MODEL FOR PREDICTING THE
EFFECTS OF VERTICAL SINUSOIDAL ACCELERATION

During our experiments, a total of 619 male subjects
have been exposed to vertical sinusoidal accelerations in-
volving 24 combinations of frequency and acceleration (Table 4),
and 212 of those subjects experienced emesis before completing
their 2-hour experimental sessions, at times ranging from
2 minutes to 114 minutes after onset of the motion.

These data alone could be of some utility and interest.
However, it has been our intent to go beyond these data in
an attempt to discover any apparent lawful relationships
among motion sickness incidence, frequency, acceleration
and time, and to offer a mathematical description of the
results. A mathematical model would serve to facilitate accu-
rate predictions of the effects of vertical sinusoidal motion
(both in the sense of interpolating among the various data
points we have obtained, and, with some caution, extrapolating
beyond them); and, hopefully, to provide theoretical insights
regarding the underlying processes of motion sickness. A
mathematical model is developed herein in pursuit of these
purposes.

In the previous report (O'Hanlon & McCauley, 1973) it
was observed that motion sickness incidence for subjects
exposed for 2 hours to vertical sinusoidal motion at various
combinations of accelerations and frequencies can be well
described as a log-normal function of stimulus acceleration,
where the two parameters of the implicit underlying normal
distribution (the mean and the standard deviation) are,
respectively, a function of the stimulus frequency, and a
constant. The mean value specifies the acceleration necessary
at a given frequency to produce a motion sickness incidence
of 50%. The functional relationship between the mean value
and frequency is well described in log acceleration versus

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TABLE 4

SUMMARY DATA FOR ALL VERTICAL, LINEAR, SINUSOIDAL MOTIONS PRESENTED, ORDERED BY FREQUENCY (IN Hz) AND ACCELERATION (IN rms g). INCLUDED ARE TOTAL NUMBER OF MALE SUBJECTS PER CONDITION, DISPLACEMENT AMPLITUDE (D, IN FEET), AND OBSERVED MOTION SICKNESS INCIDENCE (MSI) IN 2-HOUR EXPOSURES

Frequency (Hz)	rms Vertical Acceleration (g)								
	.0278	.055	.111	.170	.222	.234	.333	.444	.555
.083	N=20 D=±4.65' MSI=0%	N=20 D=±9.20' MSI=5%							
.167	N=20 D=±1.15' MSI=0%	N=20 D=±2.27' MSI=10%	N=20 D=±4.58' MSI=30%		N=20 D=±9.17' MSI=60%				
.180				N=40* D=±6.05' MSI=60%					
.200						N=35* D=±6.75' MSI=71%			
.250			N=29 D=±2.05' MSI=31%		N=54 D=±4.09' MSI=63%		N=45 D=±6.14' MSI=69%		
.333		N=20 D=±0.58' MSI=5%	N=26 D=±1.15' MSI=15%		N=26 D=±2.31 MSI=46%		N=32 D=±3.46' MSI=50%		
.417							N=6 D=±2.21' MSI=50%	N=15 D=±2.94' MSI=40%	
.500			N=20 D=±0.51' MSI=0%		N=21 D=±1.02' MSI=14%		N=20 D=±1.53' MSI=25%	N=21 D=±2.05' MSI=33%	N=24 D=±2.56' MSI=42%
.600								N=25 D=±1.42' MSI=8%	N=22 D=±1.78' MSI=18%
.700									N=24 D=±1.30' MSI=4%

*Ninety-minute exposure.

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log frequency coordinates as a concave-up parabola, with its
minimum indicating the frequency which produces the greatest
motion sickness incidence for a given acceleration. These
findings seem to suggest a model in which emesis occurs when,
for given frequencies and exposure times, acceleration ex-
ceeds a threshold value, and in which the distribution of
these threshold values among subjects characterizes a random
variable with a log-normal probability density function.

Having discovered and reported this result, we have
subsequently investigated the time-dependent nature of motion
sickness incidence (see Figure 7). For each frequency and
acceleration, we have observed the cumulative MSI as a func-
tion of time and have found that it too is apparently pro-
portional to a log-normal distribution. That is to say, a
model is implied in which individuals have varying tolerances
regarding the duration of exposure to vertical accelerations,
and there is implied an exposure-time threshold which can be
characterized as a random variable with a normal probability
density function in the log-frequency domain. Furthermore,
in investigating the relationship between tolerances to
acceleration and tolerances to duration of exposure, we have
found (not surprisingly) that greater accelerations are, on
the average, tolerated for shorter durations before emesis
occurs, and vice versa. This result implies a negative cor-
relation between the "acceleration threshold" random variable
and the "time threshold" random variable.

The above observations suggested to us that motion sick-
ness incidence as a function of frequency (f>0), acceleration
(a>0), and time (t>0) might be well described by a two-dimensional
normal distribution, the form of which is given by equation 1:

$$MSI = \frac{100}{2\pi\sigma_a\sigma_t\sqrt{1-\rho^2}} \int_{-\infty}^{\log_{10}a} \int_{-\infty}^{\log_{10}t} \exp\left\{\frac{-1}{2(1-\rho^2)}\left[\left(\frac{x-\mu_a(f)}{\sigma_a}\right)^2 - 2\rho\left(\frac{x-\mu_a(f)}{\sigma_a}\right)\left(\frac{y-\mu_t}{\sigma_t}\right) + \left(\frac{y-\mu_t}{\sigma_t}\right)^2\right]\right\} dy\,dx = 100\ \Phi(a,t) \quad (1)$$
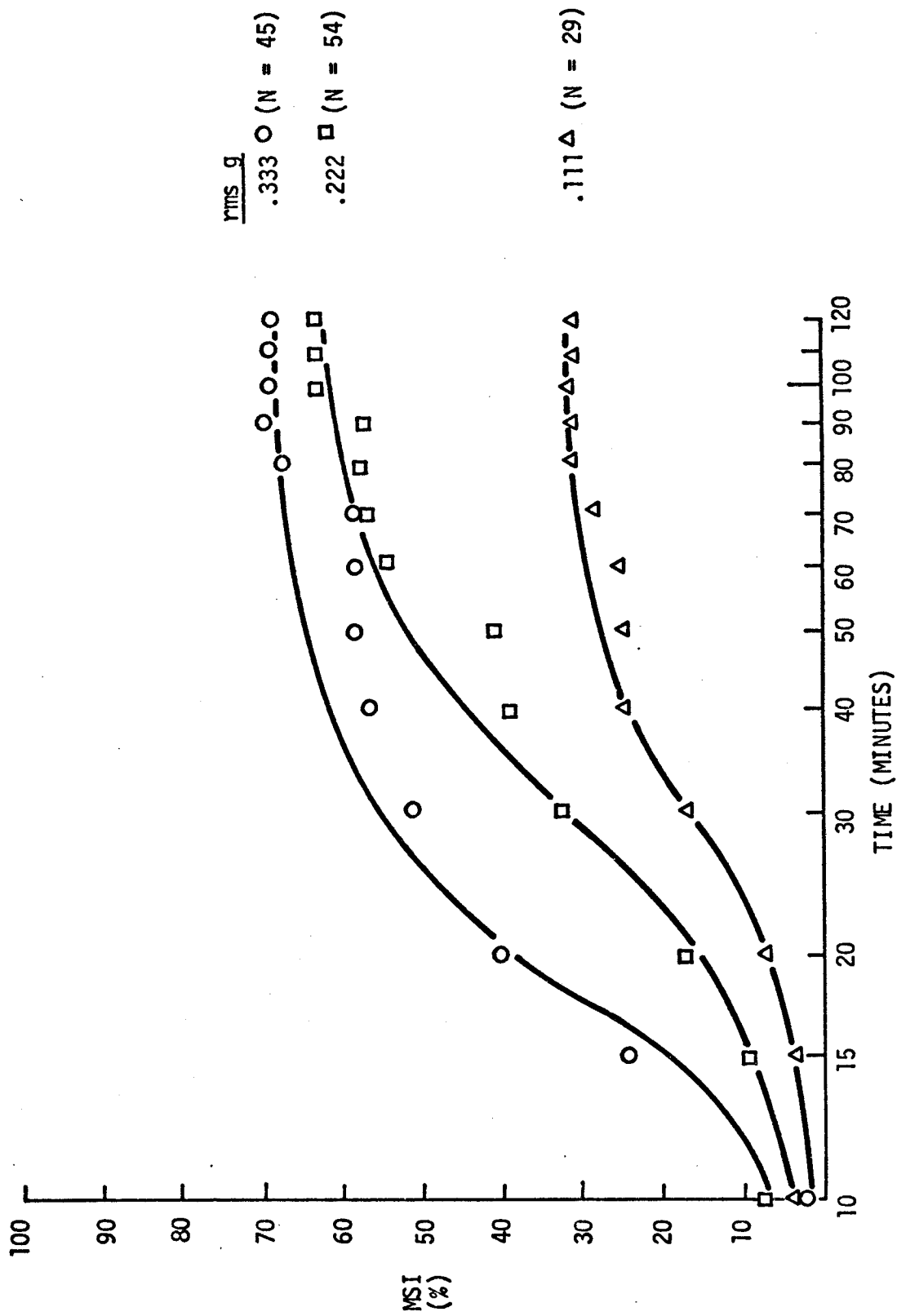
Figure 7. Motion sickness incidence (%) as a function of time for three independent groups at one frequency (0.25 Hz) and three levels of rms acceleration (g).

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where

$$\mu_a(f) = k_1 + k_2 \log_{10} f + k_3 (\log_{10} f)^2$$

It follows from this model that when we consider MSI as a function of log acceleration while holding frequency and time constant, we do indeed get a distribution proportional to the normal distribution function; and likewise when we consider the log time dependence of MSI while holding frequency and acceleration constant. Furthermore, the expected ("average") value of the log acceleration threshold is a linear function of the log time threshold, and vice versa. The slopes of these linear relationships are dependent upon the correlation coefficient (ρ) and the respective standard deviations $(\sigma_a$ and $\sigma_t)$, and the intercepts are dependent upon the mean values $(\mu_a$ and $\mu_t)$.

The bivariate normal distribution model for MSI may sound qualitatively appealing on the basis of the above considerations but, of course, it must be tested quantitatively before one can fully evaluate it. Toward this end, we must be able to evaluate the bivariate normal distribution function itself. Tables and algorithms for doing so are not generally available, but the *univariate* normal distribution function *is* widely tabulated, and algorithms for evaluating it are widely available for use on digital computers. Therefore, it is convenient to convert the two-dimensional normal distribution given in equation 1 into the products of two univariate normal distributions in order to simplify our quantitative determination of the parameters of the model, and to facilitate the use of the model for the reader's own purposes. First, let us express the bivariate normal density in terms of standardized normal variables z_a and z_t (i.e., variables with mean = 0 and standard deviation = 1) so that we have the following three equations by simple change of variables:

$$\phi(a,t)da\ dt = \frac{1}{2\pi\sqrt{1-\rho^2}}\ \exp\left[\frac{-1}{2(1-\rho^2)}\ (z_a^2 - 2\rho z_a z_t + z_t^2)\right]\ dz_a dz_t \qquad (2)$$

where

$$z_a = \frac{\log_{10}a - \mu_a(f)}{\sigma_a} \qquad (3)$$

$$z_t = \frac{\log_{10}t - \mu_t}{\sigma_t} \qquad (4)$$

Now, we may express the joint density function shown above as the product of the density functions of two statistically independent standardized normal variables by converting the normal variate z_t to z_t' as shown below (Korn & Korn, 1968):

$$z_t' = \frac{z_t - \rho z_a}{\sqrt{1-\rho^2}} \qquad (5)$$

Since this linear transformation (equation 5) leads to uncorrelated, statistically independent standardized normal variables, their joint distribution function is given by the product of the univariate distribution functions of the respective variables. Therefore we may restate equation 1:

$$\text{MSI} = 100\ \Phi(a,t) = 100\ \Phi_z(z_a)\ \Phi_z(z_t') \qquad (6)$$

where

$\Phi(z)$ is the familiar standardized cumulative normal distribution function:

$$\Phi_z(z) = \frac{1}{\sqrt{2\pi}}\ \int_{-\infty}^{z}\ \exp\left[-\frac{1}{2}\ x^2\right]\ dx \qquad (7)$$

The joint distribution function was thus reduced to the product of two univariate distribution functions which are readily evaluated, but the seven parameters of this model

remained to be determined in some optimum way. We wished to
adjust the parameters of the model to minimize the mean square
of the deviation between the data and the model's predictions.
Most algorithms for "adjusting" parameters to perform least-
squares curve fitting, however, involve analytic techniques and
require functions which are linear in the parameters. The
response of the model we have posed to the variation of its
parameters is, to say the least, nonlinear, and no analytic
approach to least-squares estimation of these parameters is
known to us. Most algorithms for the least-squares deter-
mination of nonlinear parameters involve numerical iterative
processes which use either Taylor's series expansions of the
model or some method of steepest descent. These methods have
complementary strength, as Marquardt has pointed out, and we
have used his algorithm for least-squares estimation of non-
linear parameters, which combines both methods (Marquardt,
1963).

The version of Marquardt's algorithm available to us for
this analysis was limited to 100 observations of the dependent
variable (i.e., MSI), a limitation we could not control be-
cause the source code was not accessible to us. We could have
easily exceeded 100 observations of our empirical independent
variable, for example, by representing the cumulative MSI
function at 1 minute intervals. However, we believe there
would have been little to be gained by doing so. We repre-
sented the data in the following manner: for each frequency
and acceleration, the experimental cumulative MSI was evalu-
ated at 10-minute intervals, and was considered as a data
point for use in the estimation of the parameters of the model
*only if the empirical cumulative MSI function had changed
from the previous 10-minute interval.* In addition, the cumu-
lative MSI at the end of each experimental session was included
as a data point, regardless of whether it represented a change
from the preceding 10-minute interval or not. This procedure
for representing the empirical MSI data yielded 99 observations

(by coincidence, one less than the maximum accepted by the computer program), each of which is listed in Appendix B and characterized by its frequency (Hz), acceleration (rms g), time (minutes) at the midpoint of the 10-minute interval, and the observed cumulative MSI (% emesis). A Fortran representation of the MSI model was prepared, and together with the 99 data points was used in conjunction with Marquardt's algorithm to determine least-squares estimates of the model's parameters. These estimates are given below:

$$\mu_a(f) = 0.87 + 4.36 \log f + 2.73 (\log f)^2$$

$$\sigma_a = 0.47 \qquad \mu_t = 1.46$$

$$\sigma_t = 0.76 \qquad \rho = -0.75$$

With these parameter values, the model yields the predicted MSIs tabulated in Appendix B, and the root-mean-square deviation between the predicted and observed MSIs for all 99 data points was 6.1% MSI, representing, in our judgment, a remarkably good fit to the data. This (time dependent) bivariate normal model of MSI is quite consistent in its predictions with the time *independent* model previously reported, where the two models are comparable (i.e., at 120 minutes), including the result that the most pathogenic frequency of vertical sinusoidal acceleration is approximately .16 Hz. It is noteworthy, we think, that the inclusion of time dependent data in the present version of the model has hardly changed the rms error (previously, 4% MSI), despite a considerable expansion in the number of data points, in terms of both time dependent points and total number of subjects. This suggests to us that the bivariate normal model is representative of an underlying lawful relationship among the various variables.

A three-dimensional representation of the model is given in Figure 8, including a cross-sectional depiction of the 25th, 50th, and 75th percentile "iso-emesis" curves.
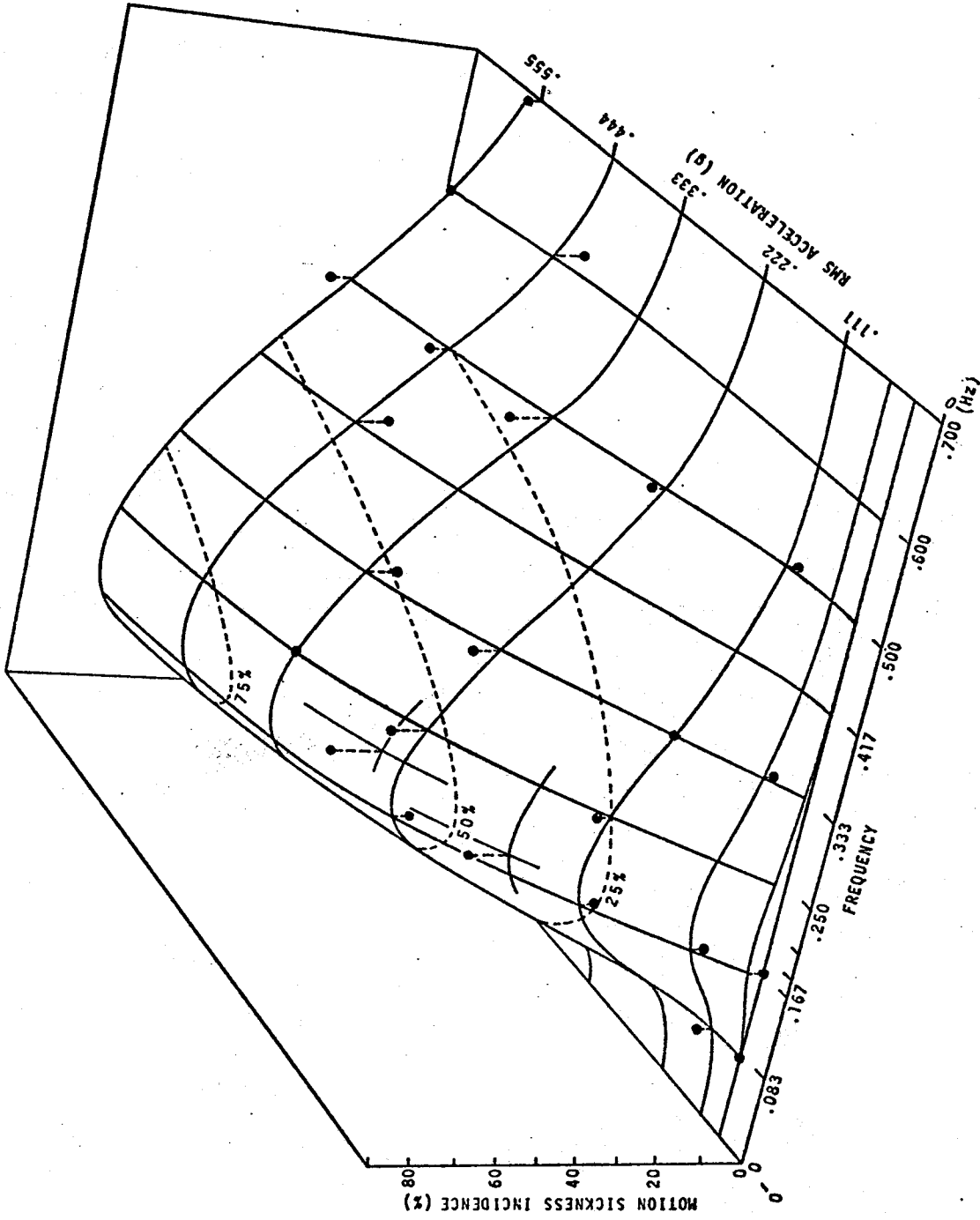
Figure 8. Three-dimensional representation of the current model of Motion Sickness Incidence as a function of wave frequency and acceleration for 2-hour exposures to vertical sinusoidal motion.

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Example Calculation of an MSI

To provide a direct example of the utilization of the mathematical model to predict an MSI using the commonly tabulated normal probability distribution function, let us say that it is desired to predict MSI at 60 minutes resulting from exposure to vertical sinusoidal accelerations of .21 rms g at a frequency of .25 Hz. First it is necessary to calculate the normal deviate z_a. From equation (3) and the parameters on p. 38:

$$z_a = \frac{\log 0.21 - (0.87 + 4.36 \log 0.25 + 2.73 (\log 0.25)^2)}{0.47} = \frac{(-0.68)-(-0.76)}{0.47} = 0.17$$

Next, it is necessary to calculate the value of the normal deviate z_t. From equation (4) and the parameters on p. 38:

$$z_t = \frac{\log 60 - 1.46}{0.76} = \frac{1.78 - 1.46}{0.76} = 0.42$$

And, finally, it is necessary to calculate the transformed (statistically independent) normal deviate z'_t. From equation (5):

$$z'_t = \frac{z_t + 0.75 z_a}{\sqrt{1 - 0.75^2}} = \frac{0.42 + (0.75)(0.17)}{0.66} = 0.83$$

Now, we enter the tables to evaluate the normal distribution function at the value z_a and at the value z'_t. The product of these two values times the factor 100 is the predicted MSI:

$$MSI = (100)(0.57)(0.80) = 46\%$$

See Appendix C for an example of a FORTRAN subprogram to evaluate the model.

Range of Predictive Validity

Figure 9 indicates the frequency/acceleration points represented in the data from which the parameters of the model were determined. We feel quite confident that any calculations of predicted MSIs for frequency/acceleration points interior to the region we have investigated (indicated approximately by the dashed line) would represent quite valid interpolations of the data. For the experimental situation in these studies, we would be most surprised by any markedly irregular
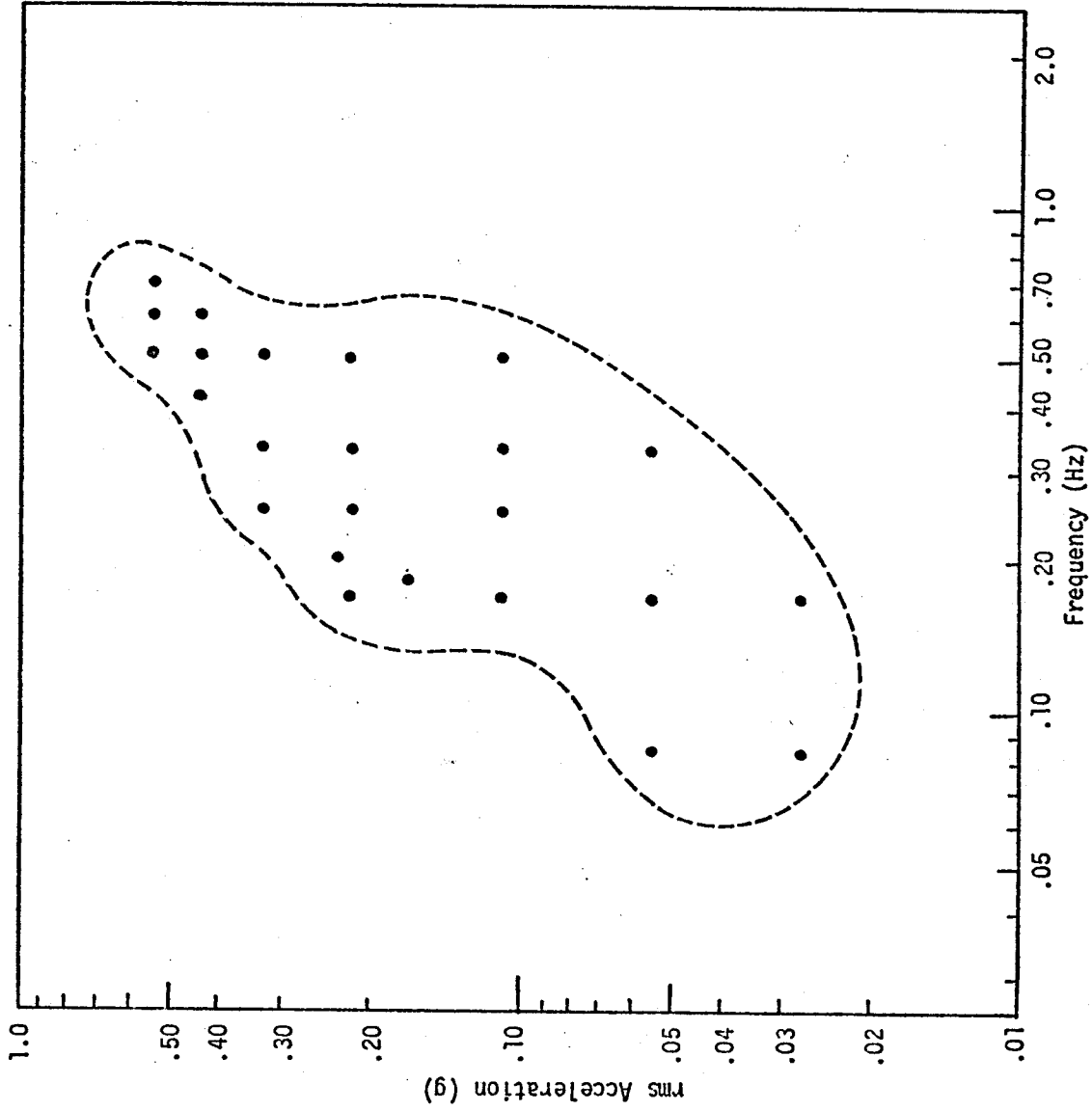
Figure 9. Data points representing the 24 motion conditions on which the mathematical model was based. (Predictions for the area outside the dotted line would be an extrapolation of the model.

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behavior (i.e., substantial deviations from the model) in this region. Extrapolation outside this region is to be taken with increasing caution the further one proceeds away from the experimentally examined regions. A quick analysis of the model in its limits suffices to indicate certain important considerations. First of all, as frequency goes to zero, MSI also goes to zero; this seems reasonable. As frequency increases without bound, MSI again goes to zero, but beyond some point (say, 1.0 Hz) we move into a region where vibration has effects other than that of producing motion sickness (see Guignard & King, 1972; Hornick, 1972).

As acceleration goes to zero, so does the predicted MSI; and this also seems reasonable. As acceleration increases, without bound, the model asymptotically increases; but it must be remembered that for acceleration peaks beyond approximately 1 g, motion sickness can become quite secondary to other considerations (e.g., performance of tasks, bodily injury, etc.). In no practical sense does it serve to analyze the behavior of the model as acceleration increases without limits.

With respect to the time variable, it will be noted that as the duration of the exposure decreases, so does the predicted motion sickness incidence; a reasonable result. But as the duration of exposure increases, MSI approaches asymptotic values which are only slightly greater than those observed at 2 hours. This is consistent with the data, but it must be remembered that for extrapolations far beyond the region that has been experimentally investigated for the purpose of developing this model, it is virtually certain that habituation effects will occur; these are not accounted for in the model at the present time.

If the model is used with some consideration of the points discussed above, we have considerable confidence in its validity for the purpose of predicting motion sickness incidence as a function of vertical sinusoidal accelerations, and perhaps as

a stimulus for a better understanding of the physiological mechanisms responsible for motion sickness. The following discussion of errors may serve to further substantiate this confidence.

Error Analysis

The genesis of this model has no doubt been influenced by the fondness of one of the authors for the concept of logarithmic responses to physical stimuli, and by his trust in the Central Limit Theorem to cause normal distributions to appear conveniently when needed. But the values of the parameters of the model, and its overall validation, have been soundly empirical. Because it does rest on an empirical foundation, however, we bring into question the matter of measurement errors and their propagation through the model. In particular, the independent variables of the model (frequency, acceleration, and time) must have associated with them some measurement errors (σ_f, σ_a, and σ_t). Do measurement errors of the magnitude we experienced in conducting these studies propagate through the model to cause large errors in predicted MSI? We may approach the question by making a Taylor's series expansion about a point f*, a*, t* to show that the error variance propagating into the predicted MSI value as a result of independent variable error variances σ_f^2, σ_a^2, and σ_t^2 is given by:

$$\text{VAR (MSI*)} = \frac{\partial \Phi^*}{\partial f} \sigma_f^2 + \frac{\partial \Phi^*}{\partial a} \sigma_a^2 + \frac{\partial \Phi^*}{\partial t} \sigma_t^2$$

By analysis of our methods and procedures, we have estimated that our measurement errors were $\sigma_f \approx 0.01$ Hz, $\sigma_a \approx 0.01$ g, and $\sigma_t \approx 3.0$ minutes.

However, the evaluation of the partial derivatives is tedious, the result complicated, and there are questions regarding the goodness of the first-order approximation. An alternate method, which we prefer for being empirical in nature and intuitively meaningful, consists of a Monte Carlo

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approach to the question of error propagation. We have evaluated the model 500 times at approximately each one of the 24 frequency/acceleration combinations explored in the experimental studies. By "approximately" we mean that for each particular evaluation we have added independent random variables normally distributed with mean zero and sigma as appropriate, to each of the independent variables frequency, acceleration, and time, to represent measurement errors typical of those found in the present studies. We then calculated the standard deviation of the MSIs resulting from the 500 trials at each of the 24 frequency/acceleration points involved in our experiments, for a total of 12,000 evaluations of the model incorporating simulated measurement errors. The results of those 12,000 evaluations indicate conclusively that the estimated errors of measurement in these studies propagate through the model and result in an overall standard deviation of 2.4% MSI; and in none of the 24 cases was a standard deviation observed to exceed 3.8% MSI. The conclusion we draw from this is simple: Errors in the measurement of frequency, acceleration, and time result in small errors in predicted MSI as compared with sampling variability.

Sampling variability is something that must be taken into account in making predictions regarding the response of small groups of subjects to actual motion. The MSI model (divided by the factor 100) may be viewed as estimating a probability, p, that a given individual will experience motion sickness under the specified conditions of frequency, acceleration, and time. Testing a group of, say, 20 subjects constitutes a series of Bernoulli trials, and the total number of persons who will actually experience emesis is subject to considerable variability from group to group (see, for example, Guilford, 1950). For groups of subjects of approximately size 20, and for typical motion sickness incidence levels, one can expect a sampling variability of approximately 10% MSI. Thus, deviations of 10% or so from MSIs predicted by the model are

quite to be expected. And it can be seen that, compared to expected sample errors, errors owing to lack of accuracy in the measurement of independent variables is secondary. Indeed, the size of the sampling error would be sufficient for us to doubt the validity of the model itself, had we very few data points. However, we have now collected sufficient data to have a reasonable estimate of the "true" probabilities underlying motion sickness incidence. These data have been collected at a variety of frequency/acceleration points, but have been unified by the model, and the 6.1% rms error between the predicted and observed data points testifies to convergence upon a rather accurate description of motion sickness in response to very low frequency vertical sinusoidal motion.

GENERAL DISCUSSION, POTENTIAL APPLICATIONS, AND AREAS FOR FURTHER RESEARCH

Several practical applications of this research can be suggested, although the limitations of the present data must not be overlooked when attempting to utilize the results for such applications. The use of single sine waves precludes confident prediction of MSI in seacraft or other types of transportation vehicles that are characterized by broadband motion. Current research under this contract is addressing that issue by observing MSI in response to more complex waveforms. An associated problem is the physical description of the intensity of the stimulus. The rms acceleration is not necessarily the appropriate measure, because it does not reflect the peak accelerations that occur in broadband motion in the same way that it does for sinusoidal motions. Jex and Allen (1974) have discussed this issue and state that "the practice of equating sinusoidal effects with random effects on an rms basis per one-third octave band may have a number of built-in errors."

The frequency dependence of MSI which is described by the mathematical model may have implications for the design of ships, and possibly for aircraft and other vehicles as well. The frequency of maximum motion sickness is estimated to be .16 Hz. Perhaps engineers concerned with ship's design and habitability could consider minimizing the vertical acceleration at approximately .16 Hz when they are attempting to reduce problems of motion sickness.

The data from this research has the potential for being transformed into a format compatible with current vibration standards, for the purpose of extending the standards into the frequency range below 1.0 Hz. The criterion for a vibration standard in this very low frequency range could be defined as motion sickness rather than comfort or fatigue decreased

proficiency, the criteria for vibration standards above 1.0 Hz.
Allen (1974) has extensively reviewed this issue, and McCauley
and Kennedy (in press) have given an example of such a standard.
However, the tenuous nature of making predictions about broadband
motion from data on sinusoids is an important consideration.

Another area of potential application is the identifica-
tion and screening of personnel who will be exposed to dynamic
environments. Concern for this problem is reflected in the
proceedings of a conference on the prediction of motion sick-
ness in the selection of pilots (Lansberg, 1973). An MSI
calculated from the present mathematical model can be con-
sidered the normative response for healthy young males. An
individual could be assessed for motion sickness susceptibility
by exposing him to a given motion and comparing his response
to the norm. Development of a metric in terms of exposure
time or symptom development, or both, would be required to
implement this procedure. More research is needed on the
specificity of susceptibility to particular motions and the
combined effects of different axes of motion before this tech-
nique of personnel selection can be seriously considered. When
more data become available regarding habituation and its
specificity, the utility of vestibular training, rather than
selection, can be evaluated as well.

The present research has provided basic data regarding
the relationship between motion sickness and the frequency and
acceleration of vertical oscillation, but a large number of
issues require further research. Some of them are: exposure-
time variables that affect habituation and dishabituation;
the specificity of habituation/adaptation to motions of
different frequencies, accelerations, and axes; the relation-
ship between visual processes and the physical parameters of
motion in situations inducing motion sickness; and MSI in com-
plex waveforms including single and multiple axes.

From a historical viewpoint, it seems that the incidence
of motion sickness will grow with increasing technology. An

excellent description of the problem is given by Reason and .
Brand (1975):

> This wretched and debilitating condition has
> always been intimately linked with man's
> technological efforts to improve and extend
> his natural powers of locomotion. As both
> the variety and availability of means of
> transport are increasing at an ever accele-
> rating rate, so the magnitude of the motion
> sickness problem grows accordingly. (p. v)

Man will continue to propel his body through water, air,
and space with dynamics that are increasingly different from
his normal body propulsion. Motion sickness research can con-
tribute to the success of these ventures, as prediction leads
to understanding, and understanding to control of the ill
effects of new dynamic environments.

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APPENDIX A
ORIGINAL AND MODIFIED ONR/HFR MOTION GENERATOR
SPECIFICATIONS AND PERFORMANCE CRITERIA

Studies I and II were performed before 1975 on the
original ONR/HFR Motion Generator. Study III was performed
in 1976 after modification of the heave system increased its
frequency range and acceleration capability. Summary data
regarding the performance of the original and the modified
Motion Generator are given in Table A-1. These data were
obtained from engineering studies by Hogge and Jex (1975) and
O'Hanlon, Seltzer, and Sanderson (1975). Those sources should
be consulted for a detailed description of the dynamics of
the upgraded system.

Also in Table A-1 is the result of a digital Fourier
series analysis of the displacement waveforms from two motion
conditions representative of Studies I and II with the pre-
modified heave system (.4 Hz/.25 rms g, and .5 Hz/.33 rms g).
The harmonic distortion was determined to be less than 10%.

TABLE A-1
ORIGINAL AND MODIFIED ONR/HFR MOTION GENERATOR
SPECIFICATIONS AND PERFORMANCE CRITERIA

Heave	Original System (Pre 1975)	Modified System
Amplitude	±11 ft	±10 ft
Velocity	± 8 ft/sec	>±18 ft/sec
Acceleration	±.5 g	>+ 1.2 g - .9 g
Minimal Incremental Acceleration Command	+.10 g	±.04 g @ .1-5 Hz
Harmonic Distortion at .4 Hz, .25 rms g and .5 Hz, .33 rms g	<10%	<10%
Pitch & Roll		Capability un-changed, performance restricted to maxi-mum required for achieving specific test requirements
Amplitude	±15	
Velocity	±25°/sec	
Acceleration	±180°/sec²	

APPENDIX B
DATA USED IN FORMULATING MATHEMATICAL MODEL OF MOTION SICKNESS INCIDENCE

Datum	Frequency (Hz)	Acceleration (rms g)	Time (min)	Observed MSI	Predicted MSI	Difference
1	.083	.055	115	5	4	+ 1
2	.167	.055	95	5	11	− 6
3	.167	.055	115	10	12	− 2
4	.167	.111	35	5	21	−16
5	.167	.111	45	10	24	−14
6	.167	.111	55	15	27	−12
7	.167	.111	75	20	31	−11
8	.167	.111	85	25	33	− 8
9	.167	.111	115	30	36	− 6
10	.167	.222	5	10	11	− 1
11	.167	.222	15	40	32	+ 8
12	.167	.222	25	45	44	+ 1
13	.167	.222	45	50	55	− 5
14	.167	.222	55	55	58	− 3
15	.167	.222	115	60	65	− 5
*16	.180	.170	5	8	6	+ 2
17	.180	.170	15	28	21	+ 6
18	.180	.170	25	40	31	+ 9
*19	.180	.170	35	47	37	+10
20	.180	.170	45	55	42	+13
21	.180	.170	85	60	50	+10
22	.200	.234	5	11	11	+ 1
23	.200	.234	15	37	32	+ 5
24	.200	.234	25	43	44	− 1
**25	.200	.234	35	57	50	+ 7
26	.200	.234	45	60	55	+ 5
27	.200	.234	55	69	58	+11
28	.200	.234	85	71	62	+ 9
29	.250	.111	5	3	1	+ 3
30	.250	.111	15	7	5	+ 2
31	.250	.111	25	17	9	+ 8
32	.250	.111	55	24	18	+ 6
33	.250	.111	65	28	20	+ 7
34	.250	.111	115	31	26	+ 5
35	.250	.222	5	6	6	− 1
36	.250	.222	15	17	23	− 6
37	.250	.222	25	32	33	− 1
38	.250	.222	35	39	39	− 1
39	.250	.222	45	41	44	− 3
40	.250	.222	55	54	47	+ 7
41	.250	.222	65	56	49	+ 6
42	.250	.222	85	57	52	+ 5
43	.250	.222	115	63	55	+ 8

Datum	Frequency (Hz)	Acceleration (rms g)	Time (min)	Observed MSI	Predicted MSI	Difference
44	.250	.333	5	2	15	-13
45	.250	.333	15	40	40	0
46	.250	.333	25	51	52	- 1
47	.250	.333	35	56	59	- 3
48	.250	.333	65	58	67	- 9
49	.250	.333	75	67	68	- 1
50	.250	.333	115	69	71	- 2
51	.333	.055	115	5	3	+ 2
52	.333	.111	35	4	5	- 1
53	.333	.111	45	12	7	+ 5
54	.333	.111	115	15	13	+ 2
55	.333	.222	5	4	2	+ 2
56	.333	.222	15	11	10	+ 1
57	.333	.222	55	15	29	-14
58	.333	.222	65	27	31	- 4
59	.333	.222	75	31	33	- 2
60	.333	.222	95	38	36	+ 2
61	.333	.222	105	42	37	+ 5
62	.333	.222	115	46	38	+ 8
63	.333	.333	5	9	6	+ 3
64	.333	.333	15	19	23	- 4
65	.333	.333	35	31	39	- 8
66	.333	.333	45	37	44	- 7
67	.333	.333	55	41	47	- 6
68	.333	.333	65	44	49	- 5
69	.333	.333	85	47	52	- 5
70	.333	.333	115	50	55	- 5
71	.417	.444	15	20	17	+ 3
72	.417	.444	65	33	42	- 9
73	.417	.444	115	40	48	- 8
74	.500	.222	65	5	5	0
75	.500	.222	95	10	8	+ 2
76	.500	.222	115	14	9	+ 5
77	.500	.333	25	5	6	- 1
78	.500	.333	55	10	12	- 2
79	.500	.333	95	15	17	- 2
80	.500	.333	105	20	18	+ 2
81	.500	.333	115	25	19	+ 6
82	.500	.444	5	5	1	+ 4
83	.500	.444	25	19	11	+ 8
84	.500	.444	45	29	18	+11
85	.500	.444	115	33	29	+ 4
86	.500	.555	15	8	11	- 2
87	.500	.555	25	13	17	- 5
88	.500	.555	35	21	22	- 2
89	.500	.555	45	33	26	+ 7
90	.500	.555	95	38	36	+ 1

Datum	Frequency (Hz)	Acceleration (rms g)	Time (min)	Observed MSI	Predicted MSI	Difference
91	.500	.555	115	42	38	+ 4
92	.600	.444	65	4	8	- 4
93	.600	.444	115	8	12	- 4
94	.600	.555	15	9	3	+ 6
95	.600	.555	115	18	18	0
96	.700	.555	115	4	6	- 2
97	.083	.0275	115	0	0	0
98	.167	.0275	115	0	2	- 2
99	.500	.111	115	0	1	- 1

* Data from research for ALZA Corporation (McCauley & O'Hanlon, 1975).
** Data from research for ALZA Corporation (Royal et al., 1976).

APPENDIX C
AN EXAMPLE OF A FORTRAN SUBPROGRAM

EMSI   05/20/76

```
100*  THIS SUBPROGRAM EVALUATES THE MATHEMATICAL MODEL PRESENTED
110*  IN HUMAN FACTORS RESEARCH, INC. TECH RPT 1733-2 FOR THE
120*  PURPOSE OF PREDICTING MOTION SICKNESS INCIDENCE (%) AS A
130*  FUNCTION OF FREQUENCY (HZ), ACCELERATION (RMSG), AND TIME (MIN).
140*
150*
160*
170        FUNCTION EMSI(F,A,T)
180*
190        FLOG=ALOG10(F)
200        AMU=0.87+4.36*FLOG+2.73*FLOG*FLOG
210        ACCELOG=ALOG10(A)
220        ZA=(ACCELOG-AMU)/0.47
230*
240        TLOG=ALOG10(T)
250        ZT=(TLOG-1.46)/0.76
260        DENOM=SQRT(1.00-(-0.75)**2.)
270        ZTPRIME=(ZT+0.75*ZA)/DENOM
280*
290        EMSI=100.*STDPHI(ZA)*STDPHI(ZTPRIME)
300        RETURN
310        END
320*
330*
340*  STDPHI, BELOW, EVALUATES THE STANDARDIZED NORMAL DISTRIBUTION
350*  FUNCTION. THE APPROXIMATION IS BASED ON S/R NDTR, IBM SSP VERSION III.
360*
370*
380        FUNCTION STDPHI(Z)
390        AZ=ABS(Z)
400        W=1.0/(1.0+0.2316419*AZ)
410        D=0.3989423*EXP(-Z*Z/2.)
420        STDPHI=1.0-D*((((1.330274*W-1.821256)*W+1.781478)*W
430      &        -0.3565638)*W+0.3193815)*W
440        IF(Z)1,2,2
450     1  STDPHI=1.0-STDPHI
460     2  RETURN
470        END
```

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