GENERAL AUTONOMIC COMPONENTS OF MOTION SICKNESS

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ABSTRACT

This report refers to a body of investigations performed in support of experiments aboard the Space Shuttle, and designed to counteract the symptoms of Space Adapatation Syndrome, which resemble those of motion sickness on Earth. For these supporting studies we examined the autonomic manifestations of earth-based motion sickness. Heart rate, respiration rate, finger pulse volume and basal skin resistance were measured on 127 men and women before, during and after exposure to nauseogenic rotating chair tests. Significant changes in all autonomic responses were observed across the tests ($p<.05$). Significant differences in autonomic responses among groups divided according to motion sickness susceptibility were also observed ($p<.05$). Results suggest that the examination of autonomic responses as an objective indicator of motion sickness malaise is warranted and may contribute to the overall understanding of the syndrome on Earth and in Space.

DESCRIPTORS: heart rate, respiration rate, finger pulse volume, skin resistance, biofeedback, motion sickness.

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Motion sickness is a pervasive feature of human travel. Unfortunately, the practical understanding of this unpleasant condition is very limited. With the advent of manned space flight there is a genuinely urgent need to understand and control motion sickness, since approximately 50% of the first human space travellers have suffered from "space adaptation syndrome" (SAS)--the zero gravity analogue of ordinary terrestrial motion sickness. The SAS is a particularly troublesome problem because traditional treatments, such as anti-motion sickness drugs, have had limited value in preventing or aborting in-flight symptoms, and at present there are no reliable ground-based tests for predicting susceptibility in space. Furthermore, deleterious side-effects of various drugs have been noted which could potentially interfere with crew performance (Wood, Graybiel & Kennedy, 1966; Wood & Graybiel, 1968; Homick, Kohl, Reschke, Degioanni, & Cintron-Trevino, 1983).

We cannot, on the surface of a planet, simulate the unique stimulation to the vestibular system (inner ear) which occurs in a weightless environment (except for brief periods during parabolic flight). But using a variety of ground-based tests, we can induce the symptoms of motion sickness. Laboratory procedures that are used to study motion sickness, (e.g., rotating chairs, vertical accelerators and optokinetic stimuli) provide an excellent means of investigating those mechanisms involved in responses to unusual gravito-inertial environments. As reviewed by Reason & Brand (1975), several early investigations have established the importance of the vestibular system

as the principal sensory receptors for motion sickness. The most influential theories on the etioligy of motion sickness have been couched in terms of vestibular physiology, related central nervous system (CNS) pathways and centers, and sensory conflicts or "mismatches" between afferent channels (Kohl, 1983). While this work has clarified certain aspects of motion sickness, it does not fully describe the mechanisms involved in the etiology of this disorder, nor has it yielded a viable treatment.

Since 1974, our research group at NASA Ames Research Center has been studying terrestial motion sickness and thinking about the SAS from a different perspective. The focus of our investigations has been autonomic nervous system (ANS) responses to stimulation rather than central nervous system (CNS) mechanisms involved in the etiology of this syndrome. Our method of treatment involves training in physiological self-regulation as an alternative to pharmacological management. The treatment method, Autogenic-Feedback Training (AFT), combines two techniques that have been used widely to facilitate self-regulation of involuntary ANS responses and minimize the debilitating effects of various stressors. Autogenic Therapy (Schultz & Luthe, 1969) uses self-suggestions (e.g., "My arms are heavy.") to encourage beneficial psychophysiological changes, while biofeedback employs an exteroceptive feedback signal to facilitate voluntary control over ANS responses that are frequently dysregulated by stress. The rationale for using AFT to combat motion sickness and SAS was based on the following assumptions: (a) there are profound ANS changes associated with these disorders, and (b) learned self-regulation of the participating ANS response systems will enable a person to successfully resist the debilitating effects of nauseogenic stimulation. Consistent with these assumptions, a series of

studies have shown that AFT significantly increases the time that trained individuals can tolerate motion sickness stimulation, as compared to control subjects who received no AFT (Cowings, Billingham & Toscano, 1976; Cowings & Toscano, 1982; Cowings & Malmstrom, 1984; Toscano & Cowings 1978; Toscano & Cowings, 1982; Levey, Jones & Carlson, 1981; Stewart, Clark, Cowings & Toscano, 1978). This observed increase in motion sickness tolerance supports the notion that the treatment effect is due to learned self-regulation of ANS activity.

Necessarily, our research group has devoted a great deal of time to gaining a better clinical understanding of human autonomic manifestations of motion sickness. To apply AFT, an individual's ANS responses to motion stimuli are first documented, and emphasis is placed on training the individual to gain control of those variables that diverged the most from his or her own resting levels. The very fact that AFT does reduce the severity of symptoms experienced points out the need to examine more systematically the relationship between autonomic activity levels and motion sickness malaise.

The relative importance of ANS responses in understanding and treating motion sickness has been a matter of some controversy. There are several published articles which deny the usefulness of examining ANS activity at all. Money (1970), in his lengthy review of motion sickness research, discussed many possible ANS changes during motion sickness, but correctly noted that there was little consistency in either procedures used or results of the available research. He then rather pointedly argued against the importance of the ANS in motion sickness: "...to the extent that motion sickness is nausea and vomiting, it is not an autonomic phenomenon and it cannot be considered a development of the autonomic nervous system."

Graybiel and Lackner (1980) have also minimized the role of the ANS in motion sickness. They subjected 12 college students to an unusual "sudden-stop" vestibular/visual test designed to induce motion sickness symptoms. Measures of heart rate, body temperature and blood pressure were taken prior to and following, but not during, repeated (30 second) test exposures. They found these measures to be remarkably invariant from pretest baseline throughout the onset of nausea which was the end point for each test sequence. They concluded that "Such measures, therefore, appear to have little value in assessing or diagnosing severity of motion sickness. This lack of correlation means that use of physiological training procedures to control these variables is likely to be of little value in preventing symptoms of motion sickness" (Graybiel & Lackner, 1980, p. 214).

Those investigators who do believe that ANS activity may yield valuable information on the motion sickness syndrome are divided in their interpretation of the ANS mechanisms involved. Tang & Gernandt (1969) demonstrated various ANS responses to electrical stimulation of the vestibular apparatus in cats, and concluded that " . . . all common symptoms of motion sickness, probably have their genesis in the strong responses of the sympathetic system to vestibular stimulation." Consistent with the hypothesis of Tang and Gernandt (1978), Parker (1964, 1971), and Parker, Schaeffer & Cohen (1972) developed a practical psychophysiological test for motion sickness susceptibility. They classed individuals as susceptible or nonsusceptible to motion sickness stimulation based on the amplitude of their electrodermal responses to a film depicting a ride down rough, twisting mountain roads in a speeding, open sports car. When tested later on the open sea on a sailing vessel, all of the 10 susceptibles either vomited or reported

severe nausea, while none of the nonsusceptibles showed or reported any signs of motion sickness.

In contrast, Kohl & Homick (1983) have emphasized the contribution of cholinergic descending limbic pathways as a modulatory mechanism by which a sensory conflict may result in actual sickness. These authors stress that the beneficial results produced by anti-motion sickness drugs are due to the action of parasympatholytics and/or sympathomimetics.

There are two major purposes of this paper: First, we wish to describe general ANS changes during motion sickness stimulation, as well as before and after, since this has not been done heretofore, and to utilize a large sample of people. Second, since our motion sickness treatment method involves modifying ANS responses to motion sickness stimulation, we wish to examine ANS responding as a function of naturally-occurring differences in susceptibility to motion sickness stimulation, to determine whether there are consistent ANS response characteristics of high- and low-susceptible individuals. The general question of individual differences in ANS response during motion sickness stimulation, including individually sterotyped ANS patterns, will be considered in detail in another paper. We have used the ANS variables of heart rate, respiration rate, finger pulse volume and basal skin resistance because they are easily measured, represent different aspects of the ANS, and have been used in previous studies of motion sickness.

Method

Subjects.

The data from 127 people, (101 males and 26 females, 18 to 46 years of age) are described in this paper. On the basis of the total number of minutes

tolerated during their first motion sickness inducing test, subjects were categorized as either high (N=46), moderate (N=43) or low (N=38) susceptibles. All subjects were certified to be in good health and to have normal vestibular function on the basis of a medical examination. Subjects were paid and were assured a minimum of 2 hours pay per visit.

Apparatus.

A Stille-Werner rotating chair was used to provoke the initial symptoms of motion sickness. Padded head rests were mounted at 45 degree angles from the vertical on the left, right, front, and back of the chair, enabling subjects to execute standardized head movements in these directions.

The physiological responses measured were (a) electrocardiogram (ECG) derived from precordial placement of silver/silver chloride disposable electrodes, with heart rate (HR) computed beat-to-beat and processed with a Gould Biotachometer; (b) respiration derived through a nose clip thermistor, with respiratory rate (RR) computed breath-to-breath using a Gould Biotachometer; (c) blood volume pulse (PV) of the hand, derived from a photoplethysmograph transducer placed on the right index finger; and (d) basal skin resistance (BSR) derived from silver/silver chloride electrodes placed on the index and middle fingers of the left hand.

Biomedical amplifiers were mounted on the rear and sides of the chair, and the physiological signals were sent to recorders through slip rings. These biological data were recorded simultaneously on strip charts and on 14-track magnetic tape; they were digitized on-line using a Nicolet Med-80 signal processor and were analyzed off-line using a DEC PDP 11/34 computer.

Procedure.

Rotating chair tests. The motion sickness test was a modification of

a widely used procedure to create Coriolis stimulation by combining head movements out of the vertical axis with body rotation (Miller & Graybiel, 1970). The blindfolded participant sat in a Stille-Werner rotating chair in a sound-shielded experimental chamber. Following a resting baseline period of either 5 or 10 minutes (depending on the experiment), rotation was initiated at 6 rpm (0.628 rad/sec) and incremented by 2 rpm (0.209 rad/sec) every 5 minutes. The rotational velocity during each 5-min interval was held constant. The maximum velocity was 30 rpm (3.142 rad/sec). At 2-sec intervals throughout each 5-min interval, head movements at 45 degree angles from the vertical were executed in four directions (left, right, forward, and backward), the direction randomized and signaled via tape-recorded running voice instruction. At the end of the 5-min interval, the head movements ceased for 30 seconds, but rotation continued, while a standard diagnostic motion sickness scale was administered (Graybiel, Wood, Miller, & Cramer, 1968). This scale was also administered upon the termination of tests.

Each participant was instructed in advance to ride as long as he or she could, short of vomiting. The test was terminated when either: (a) the participant requested termination, (b) the diagnostic scale indicated sufficient symptoms so that the experimenter judged it unwise to continue, or (c) vomiting occurred (which rarely happened).

Diagnostic scale. The diagnostic scale, referred to as the Coriolis Sickness Susceptibility Index (CSSI), was developed as a means to obtain standardized reports of the level of malaise that an individual is experiencing at any given time in a motion sickness eliciting test. This instrument is based on self-report and experimenter observations with respect to vomiting, subjective body temperature, dizziness, headache, drowsiness,

sweating, pallor, salivation, and nausea. A single global motion sickness score for a given test period can be derived using a complex scoring and weighting system (see Table 1).

Table 1

The symptom of vomiting is pathognomonic of motion sickness under the conditions of the test, and as such receives the maximum number of points. On the other end of the motion sickness spectrum, very minor symptoms of motion sickness are listed in this diagnostic scale as Additional Qualifying Symptoms (AQS). Included in this symptom category are (a) increased body temperature (TMP), (b) dizziness/vertigo (DIZ), and (c) headache (HAC). The subject has the option of reporting two levels of increased temperature and dizziness (mild-moderate "I" or moderate-severe "II"). Level of headache is not differentiated with respect to point value. Remaining symptoms of motion sickness (not including nausea) are (a) drowsiness (DRZ), (b) sweating (SWT), (c) facial pallor (PAL), and (d) increased salivation (SAL). Each of these symptoms can be described as mild, moderate or severe by writing in the appropriately marked box, "I", "II" or "III", respectively. Symptoms of nausea or any sensations associated with the "gut" can be reported as five separate levels: (a) epigastric awareness (EA), which is described as increased sensations in the stomach but not considered uncomfortable; (b) epigastric discomfort (ED), which is described as NOT nausea, but becoming uncomfortable (e.g., lump in throat, knot in stomach); and (c) nausea (NSA), reported as mild, moderate or severe by entering "I", "II" or "III", respectively.

Results

The duration in minutes of motion sickness stimulation tolerated by each

participant was used as a measure of motion sickness susceptibility. Fig. 1 shows that there was a wide range of motion sickness susceptibilities. The median test length was 19.5 min., with a range of 3 to 55 min.

Fig. 1

A one-way ANOVA revealed that the final motion sickness scale scores were larger than the initial scores, $F(1, 122) = 526.02$, indicating that subjects did indeed become more motion sick across the test (all ANOVA effects, correlations and differences between means reported in this paper are statistically significant at $p < .05$). The concluding scale score for 94.5% of the participants was at or above 8 points, the criterion for severe malaise; thus, subjects did comply with our request to ride until their motion sickness was at a high level. The participants were divided into three approximately equal-sized susceptibility groups based on how many minutes of motion sickness stimulation they tolerated. The characteristics of these groups are shown in Table 2. The initial motion sickness scale scores differed between groups, $F(2,124) = 31.52$, providing objective evidence that more highly susceptible subjects became motion sick earlier in the test. There was no difference between groups on the final scale scores, indicating that the different susceptibility groups rode to similar motion sickness endpoints.

Table 2

The role of ANS responses in motion sickness was examined in two complementary sets of analyses which explored: (a) the time course of ANS changes across the motion sickness test; and (b) multiple correlations between ANS responses as predictor variables, with motion sickness scale scores and minutes of rotation tolerated as criterion variables. The results of these analyses are presented in separate sections below.

ANS changes across the motion sickness test.

For each participant, 1-min. means were computed for HR, BSR, PV, and RR across the following stages of the motion sickness test: (a) the 5 minutes immediately preceding rotation (Pretest), (b) the first 5 minutes of rotation (Start), (c) the last 5 minutes of rotation (Finish), during at least part of which the subject was assumed to be motion sick, and (d) the 5 minutes immediately following the termination of rotation (Posttest). The data for four participants who tolerated only three minutes of rotation were excluded from these analyses. The means are shown in Figures 2-5 for Stages (Pretest, Start, Finish, Posttest) X Minutes (1-5 within Stages) X Susceptibility Group (Low, Moderate, High).

Figs 2-5

For each ANS variable, a preliminary ANOVA was conducted to assess the effects of test stages. Then, three sets of ANOVAs were conducted to examine the time course of changes for each ANS variable: (a) Susceptibility (3) X Minutes (5) within each of the four stages, (b) Susceptibility (3) X Minutes (2) for the two minutes immediately before and after the onset of rotation, and (c) Susceptibility (3) X Minutes (2) for the two minutes immediately before and after rotation ceased.

Visual inspection of Figures 2-5 reveals that all four ANS measures respond to motion sickness stimulation, and that there is some ANS recovery when stimulation stops. In the preliminary ANOVA, there was an effect for Stages for every ANS variable. Tests of significance between each possible pair of Stages were conducted using the Fischer Test at $p < .05$ (Keppel, 1982, p. 157). The results of these comparisons are listed in Table 3.

Table 3

The results of the second and third sets of ANOVAs are described below for each ANS response.

Heart rate. HR drifted upward slightly across the minutes of pretest from 69.9 beats/min in the first minute to 71.1 beats/min in the fifth, $F(4, 496) = 6.06$. In pretest there was a Minutes X Susceptibility effect, $F(8, 496) = 3.22$, apparently caused by an anticipatory increase in HR for the low susceptibility group at the end of the segment. The onset of rotation was accompanied by an average increase in HR of 5.5 beats/min, $F(1, 124) = 59.21$, that did not interact with Susceptibility. Following the more or less uniform increase in HR at the onset of rotation, HR continued to rise for the high susceptible participants while it stabilized for those who were less susceptible, resulting in a Minutes X Susceptibility interaction, $F(8, 496) = 8.55$. Heart rate increased significantly across the first five minutes of rotation for the high susceptibles, but did not change significantly for the other two groups. A posthoc ANOVA showed that the three groups had different heart rate responses that occurred as early as the first two minutes of rotation, $F(2, 124) = 9.90$. There was an increase in HR of 4.1 beats/min across the final five minutes of rotation, $F(4, 496) = 16.06$, with no differences between groups on this effect. When rotation stopped, there was immediate HR recovery, reflected in an average HR decrease of 4.2 beats/min in the first minute of posttest, $F(1, 124) = 29.65$. There was a Minutes X Susceptibility effect within the posttest segment for HR, $F(8, 496) = 3.23$. The more susceptible the participant had been to motion sickness stimulation the greater the HR decrease once rotation ceased, $F(8, 496) = 3.23$. Across the five minutes of recovery, HR decreased by an average of 11.9, 10.7, and 5.3 beats/min for the high, moderate, and low susceptibles, respectively.

Since the high, moderate and low susceptiblity groups rode progressively longer, the different HR responses of these groups across rotation is minimized in Figure 3. Perhaps a more revealing analysis is to take the HR change over the total period of rotation and divide by the duration of rotation to express the HR change per minute of stimulation. HR for the high susceptibles accelerated at a rate of 1.14 beats/min across the test, the moderate susceptibles at 0.44, and the low susceptibles at 0.06. Thus, during motion sickness stimulation, the HR of high susceptibles accelerated about 19 times as fast as the HR of the low susceptibles.

In summary, HR responded vigorously to motion sickness stimulation. The overall magnitude of the HR response was related to motion sickness susceptibility, with those who were more susceptible showing greater changes in HR, even within the first two minutes. When rotation stopped HR returned quickly to pretest levels.

Basal skin resistance. The substantial mean differences in BSR between groups shown in Figure 3 are not statistically significant because of pronounced individual differences within the groups. BSR changed across minutes during the pretest, $F(4, 496) = 4.62$. After drifting upward by a mean of 14 Kohms across the first several minutes of the pretest segment, there was a decrease of about 26 Kohms, apparently in anticipation of rotation, $F(4, 496) = 4.62$. The onset of rotation resulted in an immediate mean 131 Kohm drop in BSR, $F(1, 124) = 54.98$. There was adaptation over the first five minutes of rotation, with a mean 40 Kohm increase in BSR, $F(4, 496) = 7.59$. BSR decreased across the final five minutes of rotation, $F(4, 496) = 5.98$. This effect interacted with Susceptibility, $F(8, 496) = 2.37$. There was a significant decrease in BSR only for the high susceptibles, who showed an

average decrease of of 41 Kohms, as compared to the moderate and low susceptibles, who decreased only 7 and 4 Kohms, respectively. There was no immediate change in BSR in the first minute of posttest, $F < 1$, but there was an average increase of 41 Kohms across the five minutes of posttest, $F(4, 496) = 11.67$, reflecting some BSR recovery.

In summary, BSR decreased during motion sickness stimulation, and then recovered afterward. Those who were highly susceptible to motion sickness experienced a decrease in BSR as they became sick. Basal skin resistance did not recover to pretest levels within five minutes after the termination of rotation.

Pulse volume. As in the case of BSR, there was no significant group effect on differences in PV during the pretest segment. Pulse volume decreased from 54.Ø to 49.4 arbitrary units across the pretest baseline, $F(4, 496) = 5.21$, with an apparent anticipatory decrease near the end of the segment. The onset of rotation prompted an abrupt decrease in PV of 12.5 arbitrary units, $F(1, 124) = 54.42$, that did not vary with susceptibility. Pulse volume recovered somewhat across the first five minutes of rotation, with a mean increase of 3.4 units, $F(4, 496) = 6.78$. There was a Minutes X Susceptibility interaction at the end of rotation, $F(8, 496) = 2.11$. Pulse volume decreased significantly across this time period only for the high susceptibles, who showed a mean drop in PV of 6.3 units. During the first minute after rotation stopped, PV recovered an average of 8.Ø units, $F(1, 124) = 31.1Ø$. There was a change in PV across the minutes of the posttest, $F(4, 496) = 12.83$. Pulse volume increased significantly by an average of 6.6 units from the first to third minutes of posttest, then decreased significantly by an average of 3.3 units from the third to the fifth minutes.

In summary, following an abrupt drop in PV at the onset of rotation, PV gradually increased as motion sickness stimulation continued. The most susceptible participants experienced decreased PV as motion sickness developed. There was a rebound increase in PV when rotation stopped, followed shortly by a decrease.

Respiration rate. Respiration rate did not change significantly across the pretest segment. When rotation commenced, RR increased in the first minute by 3.2 breaths/min, $F(1, 124) = 117.60$. This initial RR response to rotation was followed by partial recovery across the remainder of the first five minutes of rotation $F(4, 496) = 8.40$. Respiration rate changed across the final five minutes of rotation, $F(4, 496) = 2.73$. After decreasing an average of 0.5 breaths/min from the first to the third minutes, RR increased by 0.9 breaths/min across the remaining two minutes of the test. When rotation ended, there was an average drop in RR of 0.7 breaths/min, $F(1, 124) = 6.69$. Recovery continued across the five minutes of posttest, with a mean decrease of 1.9 breaths/min from the first to the fifth minute, $F(4, 496) = 7.79$. None of the significant effects for RR interacted with Susceptibility.

In summary, RR increased with the onset of rotation, recovered as rotation continued, and then increased across the several minutes leading to termination. Respiration rate decreased to pretest levels in the five minutes following the end of rotation.

ANS responses as predictors of motion sickness.

The results presented thus far suggest that certain ANS responses found early in the motion sickness test may herald the subsequent global psychophysiological response to motion sickness stimulation. Therefore, in order to determine which ANS variables might best predict motion sickness

susceptibility, analyses were undertaken in which all ANS variables were entered into a predictive model with the minutes of rotation tolerated, and motion sickness scale scores as the criterion variables.

Two sets of stepwise multiple regression analyses were conducted. The ANS variables were entered into the regression equations in descending order of their correlations with the dependent variable, corrected for the effects of any independent variables that had already been entered. The criterion to enter an independent variable was $F(2,124)$ of at least 4.0, which corresponded to a correlation of about .17.

In the first analysis, the 16 predictor variables were the Pretest mean, change from the last minute of Pretest to the first minute of rotation, change from the first to the fifth minute of rotation, and change across the last five minutes of rotation for HR, BSR, PV, and RR. The dependent variable was the minutes of rotation tolerated. Two ANS variables had adequate predictive value to enter the multiple regression equation: (a) HR change across the first five minutes of rotation, $r(125) = -.35$--the greater the HR increase, the fewer minutes of rotation tolerated; and (b) HR response to the first minute of rotation, $r(125) = -.23$— again, the greater the HR increase, the fewer minutes of rotation tolerated. Thus, the greater the HR response to rotation, in the first minute or across the first five minutes, the greater the susceptibility to motion sickness stimulation. The multiple r between these variables and minutes of rotation was .42.

In a second approach, the ANS measures for the final five minutes of rotation were deleted and multiple regression was conducted with the first diagnostic score as the dependent variable, since this score distinguished between susceptibility groups as mentioned earlier. The results were similar

to those for test length. The variables entering the equation, in order, were (a) HR change across the first five minutes of rotation, $\underline{r}(125) = .35$, and (b) HR response to the first minute of rotation, $\underline{r}(125) = .37$. In both cases, the greater the HR acceleration, the more motion sickness reflected in the scale score. The multiple-$\underline{r}$ between the two predictors and the initial motion sickness scale score was .49.

In summary, the initial HR response to rotation predicted eventual motion sickness as measured both by the diagnostic motion sickness scale and by how much motion sickness stimulation the participant was able to tolerate.

Discussion

It is clear from these analyses that autonomic responses do change as a function of motion sickness stimulation. The lack of autonomic disturbance observed by Graybiel & Lackner (1980) during sudden-stop motion sickness tests can only be attributed to the types of measures taken and the manner in which these measurements were obtained. As can be seen from Figures 2 - 5, physiological response levels change rapidly and dramatically at the onset of stimulation and when the test concludes. Measures of oral temperature, blood pressure and pulse rate, taken in a 30 second period immediately before and after motion sickness tests, would not necessarily reflect these changes. Pulse rate alone could, in fact, produce widely different values if two measurements were taken only seconds apart during the recovery period.

The inconsistency in ANS responses to motion sickness reviewed by Money (1970) might be explained as the result of several well-known principles of psychophysiology, relating to the sources of variability in physiological activity. As in Graybiel & Lackner's study, the data Money reviewed also

were influenced by differences in timing of the measurements relative to stimulus onset. But even if this methodological precaution were taken, the experimenters would probably still see a certain amount of inconsistency in their data. First, there is the principle of individual-response stereotypy: some individuals exhibit maximal ANS responses in one or more organ systems while showing no significant changes in the activity of other systems. Second, the principles of autonomic balance, law of initial values, and (parasympathetic) rebound may explain why some subjects exhibit increases in the activity levels of specific physiological functions while other subjects show (paradoxical) decreases in activity. Third, there is the principle of stimulus-response specificity: the response variability observed within subjects exposed to different types and durations of motion stimuli. These sources of response variability were not controlled in the studies reviewed by Money.

The final set of analyses performed in this paper describe the value of examining autonomic behavior as predictors of motion sickness susceptibility. Although early changes in heart rate were clearly the most consistent predictors, the reader is cautioned against drawing the conclusion that heart rate alone is a sufficient descriptor of ANS involvement in motion sickness. It may well be that the most valuable information on ANS components of motion sickness will be yielded by additional analyses of this data base, examining types of individual response patterns. Analyses of individual differences, however, are beyond the scope of the present paper. We have examined the data of a large sample of people, all given the same motion sickness test (Coriolis acceleration), in order to describe the general trend among all subjects. These analyses have produced

evidence which may elucidate the role of ANS activity in the motion sickness syndrome.

Our results are in agreement with the findings of Tang & Gernandt (1978) that sympathetic-like activity is characteristic of ANS responses to vestibular stimulation. All of the ANS variables described in this paper show sympathetic activation in the transition from pretest baseline to start of rotation. These findings also confirm Parker's (1964, 1971) use of amplitude of the electrodermal response to predict motion sickness susceptibility. The sympathetic activation associated with motion sickness as demonstrated in these studies and in this paper indicates that motion sickness can be categorized as a stress response. In the future, it might be valuable to compare autonomic responses to other environmental stressors (e.g., cold-pressor) to those of motion sickness.

If motion sickness is characterized by sympathetic activation of the ANS, how can we account for inconsistent, yet beneficial results that are obtained from anti-motion sickness drugs? As described by Kohl & Homick (1983), the action of one widely tested drug, Scopalomine, is to block cholinergic transmitters. They emphasize the contribution of cholinergic descending limbic pathways to motion sickness, and it may be that Scopalomine blocks these central pathways rather than affecting peripheral parasympathetic activity. Another possible answer to this puzzle might be gained by examining the time-course of ANS changes observed throughout the motion sickness tests. Initially, all responses show sympathetic activation. In most cases, this activation is exaggerated or sustained throughout the end of the test, and is followed by a profound "rebound" phenomenon during the posttest recovery. This observation of parasympathetic rebound may hold the key: it may be that

the nausea and vomiting associated with motion sickness are components of a large-magnitude parasympathetic reaction to sustained sympathetic activity. If that were the case, parasympatholytic drugs could result in diminished symptomatology by effectively reducing the magnitude of this rebound. Therefore, observations of the beneficial effects of such drugs are not really inconsistent with our own findings on autonomic responses to motion sickness. In fact, the behavioral treatment used by our laboratory (AFT) and parasympatholytic anti-motion sickness drugs may be viewed as being maximally effective during different time-courses in the development of the motion sickness syndrome. Autogenic feedback training may effectively reduce sympathetic tone, thereby preventing the occurence (or likelihood) of the parasympathetic rebound, while parasympatholytics are effective only when rebound is imminent, or on-going.

Our use of the present ANS measures does not imply that these are the best indicators of ANS involvement in motion sickness. Further research using a variety of other ANS measures, such as the electrogastrogram, impedance cardiography and blood pressure, will be necessary to ascertain which variables provide the most comprehensive information on ANS involvement, its interaction with motion sickness susceptibility, and the propensity for learned control.

To conclude, we have demonstrated profound autonomic nervous system changes in association with ground-based motion sickness. The time course and magnitude of changes in certain of the ANS variables measured differ as a function of motion sickness susceptibility, and the heart rate measure appears to predict motion sickness susceptibility within the first two minutes of the motion sickness test. These findings support our use of AFT in the treatment

of motion sickness symptomatology, and may lead to a more comprehensive understanding of physiological mechanisms underlying motion sickness.

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Table 1

Sample Motion Sickness score sheet

DIAGNOSTIC CRITERIA SCALE - MOTION SICKNESS

MALAISE LEVEL	POINTS	VMT	TMP	DIZ	HAC	DRZ	SWT	PAL	SAL	NSA	ED	EA
PATHOGNOMIC	16	I										
MAJOR	8					III	III	III	III	II,III		
MINOR	4					II	II	II	II	I		
MINIMAL	2					I	I	I	I		I	
AQS	1		I,II	I,II	I							I

Levels of Severity Identified by Total Points Scored

FRANK SICKNESS (S)	SEVERE MALAISE (MIII)	MODERATE-A (MIIA)	MODERATE-B (MIIB)	SLIGHT MALAISE (MI)
16 points	8-15 points	5-7 points	3-4 points	1-2 points

SYMPTOMS OBSERVED												TOTAL
RPM	VMT	TMP	DIZ	HAC	DRZ	SWT	PAL	SAL	NSA	ED	EA	POINTS

Table 2

Group Assignment By Initial Susceptibility To Motion Sickness

Susceptibility	N=	Mean Test Duration in Minutes	Mean Diagnostic Score	
			Start	Finish
HIGH	46	9.7	5.7	12.6
MODERATE	43	18.1	2.3	13.3
LOW	38	32.9	1.2	12.1

Table 3

Significant Differences Between Stages, All ANS Responses

	Stage			
Stage	Pretest	Start	Finish	Posttest
Pretest	—	HR,BSR,PV,RR	HR,BSR,PV,RR	HR,BSR
Start		—	HR,BSR	RR,PV
Finish			—	HR,PV
Posttest				—

Note: Significance tested by Fischer Test at $p < .05$

(Keppel, 1982, p.157)

FIGURE CAPTIONS

Figure 1. Frequency distribution of minutes of rotation tolerated by subjects during motion sickness tests.

Figure 2. Mean heart rate, for subject groups divided according to motion sickness susceptibility, across four time blocks of the motion sickness test: prestest baseline, first five minutes of the test, last five minutes of the test, post-test recovery.

Figure 3. Mean basal skin resistance, for subject groups divided according to motion sickness susceptibility, across four time blocks of the motion sickness test: pretest baseline, first five minutes of the test, last five minutes of the test, post-test recovery.

Figure 4. Mean finger pulse volume (in arbitrary units), for subjects divided according to motion sickness susceptibility, across four time blocks of the motion sickness test: pretest baseline, first five minutes of the test, last five minutes of the test, post-test recovery.

Figure 5. Mean respiratory rate, for subject groups divided according to motion sickness susceptibility, across four time blocks of the motion sickness test: pretest baseline, first five minutes of the test, last five minutes of the test, post-test recovery.

Figure 1

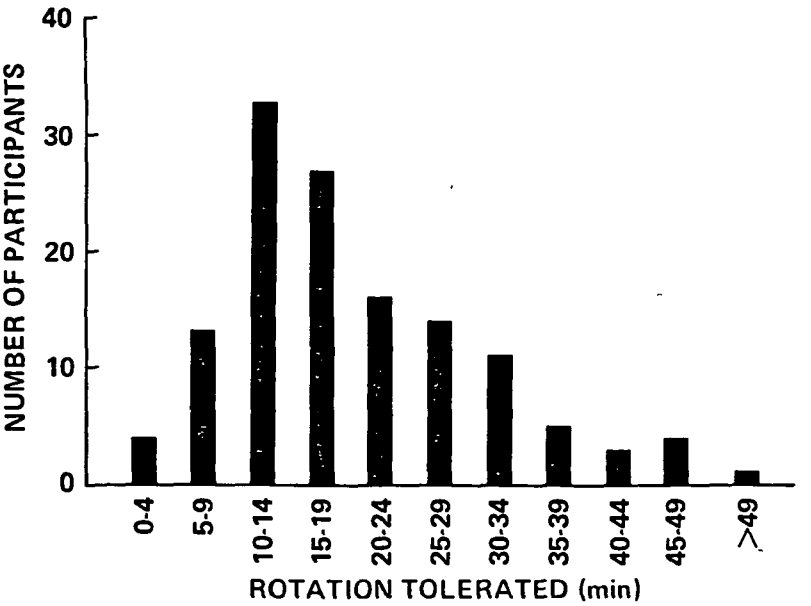

NUMBER OF PARTICIPANTS
40
30
20
10
0
0-4
5-9
10-14
15-19
20-24
25-29
30-34
35-39
40-44
45-49
>49
ROTATION TOLERATED (min)

Figure 2

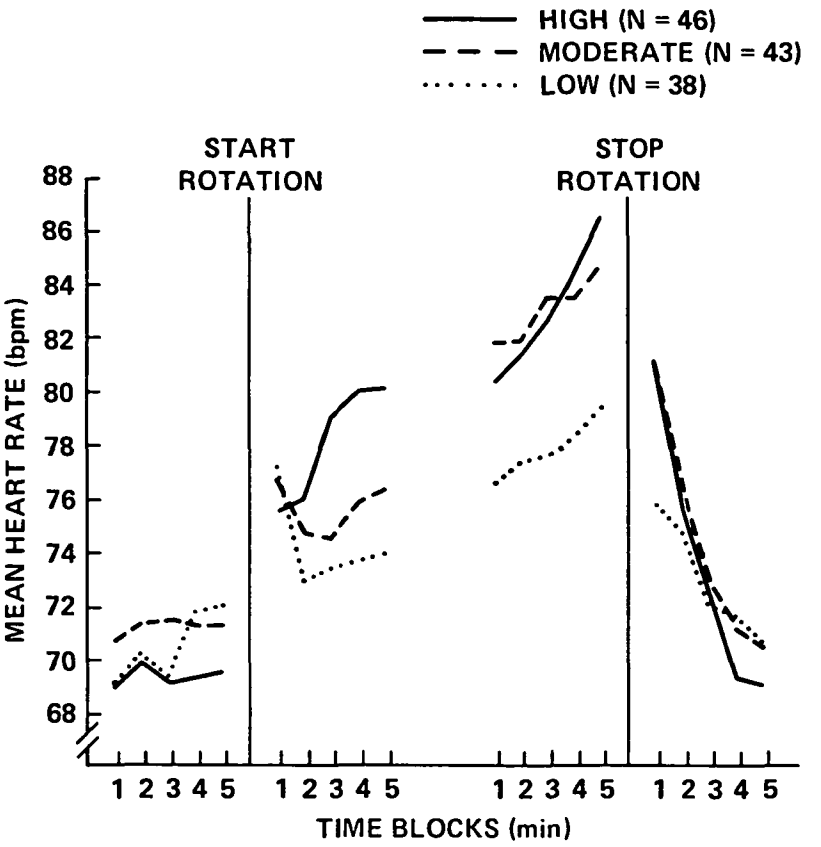

HIGH (N = 46)
MODERATE (N = 43)
LOW (N = 38)
START ROTATION
STOP ROTATION
MEAN HEART RATE (bpm)
88
86
84
82
80
78
76
74
72
70
68
1 2 3 4 5
1 2 3 4 5
1 2 3 4 5
1 2 3 4 5
TIME BLOCKS (min)

Figure 3

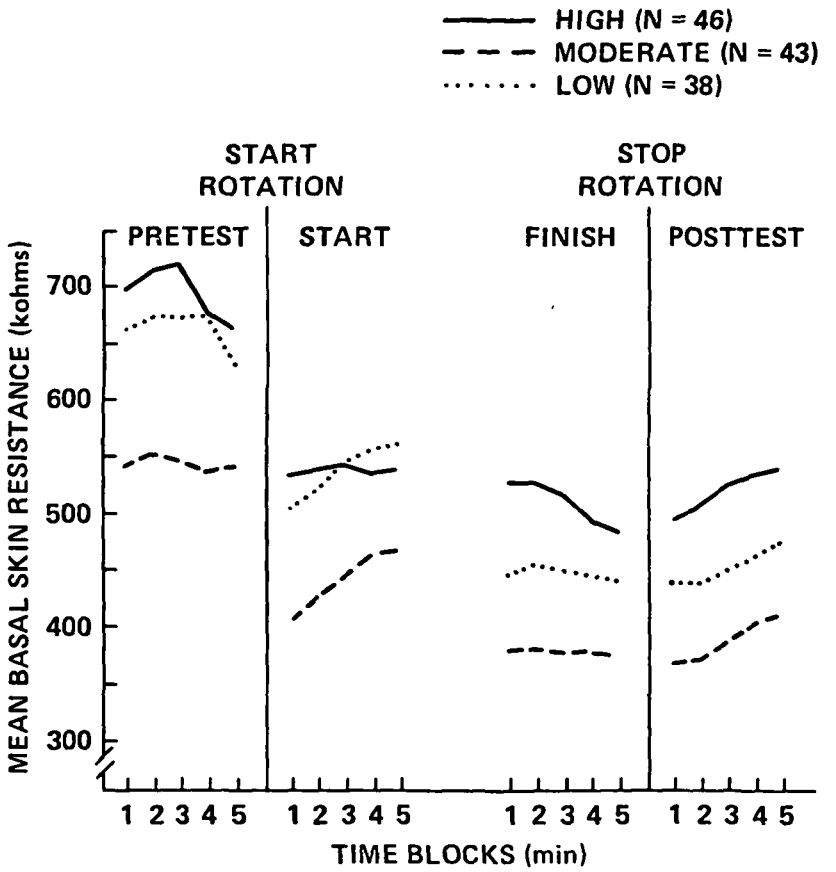

HIGH (N = 46)
MODERATE (N = 43)
LOW (N = 38)
START ROTATION
STOP ROTATION
PRETEST
START
FINISH
POSTTEST
MEAN BASAL SKIN RESISTANCE (kohms)
700
600
500
400
300
1 2 3 4 5
1 2 3 4 5
1 2 3 4 5
1 2 3 4 5
TIME BLOCKS (min)

Figure 4

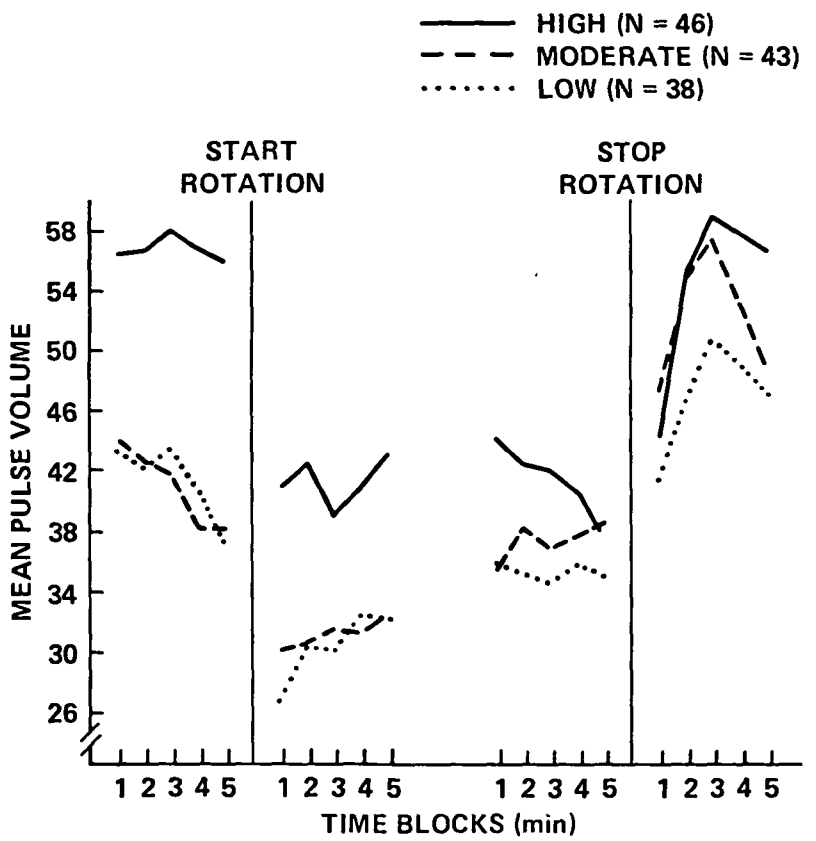
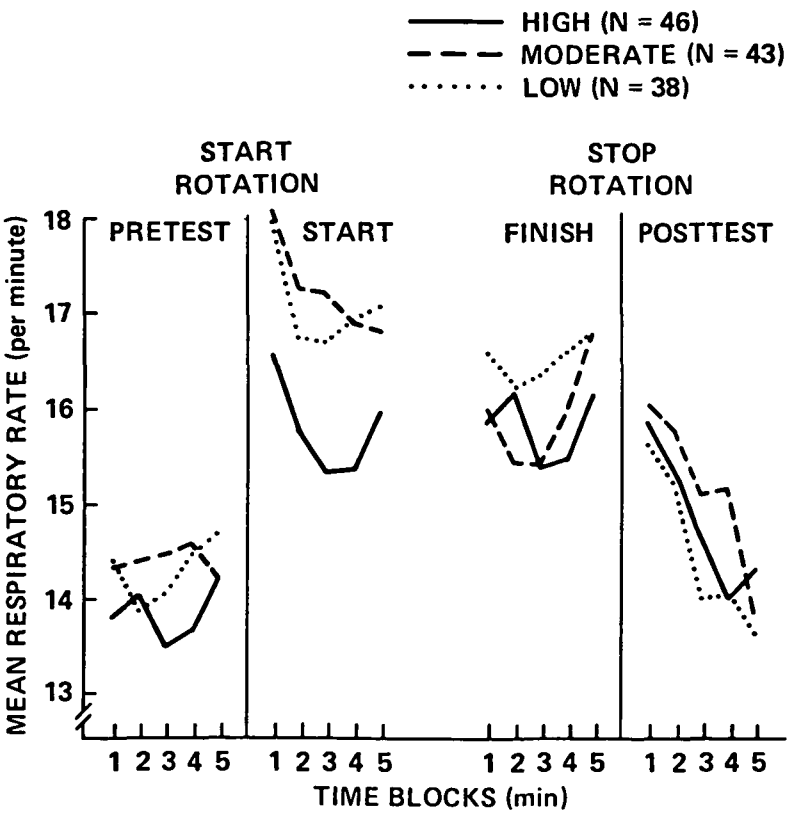

HIGH (N = 46)
MODERATE (N = 43)
LOW (N = 38)
START ROTATION
STOP ROTATION
MEAN PULSE VOLUME
58
54
50
46
42
38
34
30
26
1 2 3 4 5
1 2 3 4 5
1 2 3 4 5
1 2 3 4 5
TIME BLOCKS (min)

Figure 5


HIGH (N = 46)
MODERATE (N = 43)
LOW (N = 38)
START ROTATION
STOP ROTATION
PRETEST
START
FINISH
POSTTEST
MEAN RESPIRATORY RATE (per minute)
18
17
16
15
14
13
1 2 3 4 5
1 2 3 4 5
1 2 3 4 5
1 2 3 4 5
TIME BLOCKS (min)