STIMULUS SPECIFICITY AND INDIVIDUAL STEREOTYPY OF AUTONOMIC RESPONSES TO MOTION STRESSORS.

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STIMULUS SPECIFICITY AND INDIVIDUAL STEREOTYPY OF AUTONOMIC RESPONSES TO MOTION STRESSORS

A thesis submitted to the faculty of
San Francisco State University
in partial fulfillment of the
requirements for the
degree

Master of Arts
in
Physiological Psychology

by

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San Francisco, California

August 1985

CERTIFICATION OF APPROVAL

I certify that I have read STIMULUS SPECIFICITY AND INDIVIDUAL STEREOTYPY OF AUTONOMIC RESPONSES TO MOTION STRESSORS by Maxwell Guy Morgan, and that in my opinion this work meets the criteria for approving a thesis submitted in partial fulfillment for the Master of Arts in Physiological Psychology degree at San Francisco State University.

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1985

Motion sickness research shows a lack of agreement regarding the contribution of the autonomic nervous system (ANS). The resolution of this question is exigent for Space Adaptation Syndrome, zero gravity sickness. A case is drawn for the necessity to apply a methodological approach that incorporates: 1) standardization of parameters in relation to the individual differences in variability and prestimulus levels, 2) a concern for patterning of responses, and 3) the physiological association with subjective reports. Vasomotor, heart rate, respiration rate, skin conductance and subjective reports of malaise were collected from 22 subjects while participating in three motion stressors; vertical acceleration, Coriolis stimulation, and combined optokinetic and Coriolis stimulation. The results demonstrate that ANS response patterns can be separated into three mutually exclusive components: 1) a generalized response to motion sickness, 2) a stimulus specific response to the type of stressor being presented, and 3) individualized stereotypical response patterns that are associated with subjective reports of malaise.

I certify that the abstract above is a correct representation of the content of this thesis.

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Thesis Advisor Date

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Space Adaptation Syndrome (SAS) is considered a one of the major biomedical problems of short duration space flight. The inability to effectively predict its occurrence or counteract symptom development has compounded the problem. Similarities in symptomatology have led researchers to conclude that SAS is a result of a particular form of motion sickness inducing stimulation. Many components of motion sickness episodes have been identified, such as the etiological contribution of the vestibular system and much of the ensuing symptomatology. However, a wide range of intraindividual variability exists in rates of susceptibility and symptom development that is not adequately understood.

The literature review shows a lack of agreement regarding the importance of the autonomic nervous system's (ANS) contributions to motion sickness (Graybiel & Lackner, 1980; Money, 1970; Reason & Brand, 1975). The conflicting results of previous research can be resolved with appropriate consideration for the methodological approach used; which will be demonstrated in this paper. Application of psychophysiological methodologies that take into account the type of stimulus (stimulus specificity), the indvidual's inherent propensities of responding (stereotypical responses) and subjective interpretations of the experience (idiosyncractic responses) are necessary.

The thesis of this study is that the ANS responses to motion sickness inducing stimuli play a central role in intraindividual variability of susceptibility and symptom development. More specifically

the questions to be addressed are that ANS responses are comprised of: 1) a patterned autonomic response to motion stressors, regardless of the type of stimuli, 2) a stimulus specific response pattern that can be differentiated by types of motion stressors, and 3) a reproducible idiosyncratic response pattern of subjects that is related to intraindividual variability in susceptibility and subjective reports of malaise.

Motion Sickness and Space Adaptation Syndrome

In a most general sense motion sickness is the result of an inability to adapt to certain types of movements. The symptomatology can be elicited by certain types of physical movements and by some optokinetic stimuli (movement within the visual field) (Money, 1970; Parker et al, 1964, 1972, 1974). The predominant elicitor of motion sickness has been transportation by sea, ground, and air. With new technology, air transportation is becoming faster and more maneuverable; fators which elicit the motion sickness symptoms more frequently.

Weightlessness is another powerful elicitor of motion sickness that is a special problem facing the space shuttle era. Debilitating episodes have been reported by 17% of the cosmonauts, and even caused one premature termination of a mission. The American space program reports 15% incidences of motion sickness. The stimulus conditions were more favorable for eliciting motion sickness in the Apollo flights, where 36% of the astronauts reported some symptoms. In all other flights (Mercury and Gemini) movement was much more restrained or helmets were worn more often (preventing rapid head movements) than in the Apollo missions

(Schneider & Crosby, 1980a). In fact, about 50% of all people that have traveled to space have experienced motion sickness. However, the National Aeronautics and Space Administration no longer referes to this malady as motion sickness, the official terminology is now Space Adaptation Ayndrome. SAS comprises many of the biological problems of space travel; such as blood and interstitial fluid shifts, cardiovascular decomposition, bone mineral loss, and versibular problems.

The most easily identifiable and most frequently reported characteristics of motion sickness are pallor, sweating, nausea, and vomiting (Grabiel, Wood, Miller, & Cramer, 1968; Money, 1970). Pallor and sweating are normally caused by hyperactivity of the sympathetic division of the ANS. Nausea and vomiting are not considered to exclusively results from sympathetic hyperactivity since these can occur in gut-denervated animals. Money (1970) has reported that most physiological changes associated with motion sickness have been inconsistent. Both increases and decreases for pulse rate, blood pressure, respiration, and pupil size have been reported while consistent changes have been reported only for reductions in peripheral circulation and temperature of the extremities (Money, 1970). Consistent behavioral characteristics also have been shown to be affected by motion sickness; these include increases in drowsiness, depression (Clark & Graybiel, 1961; Graybiel & Clark, 1965), spatial disorientation, and anxiety; accompanied by decrease in muscle coordination, time estimation, and arithmetic performance (Money, 1970).

Repetitive exposure to nauseogenic stimuli will usually lead to a decrease and finally a disappearance of symptomatology. Highly susceptible individuals adapt very slowly to motion stimuli and

sometimes not at all (Reason & Brand, 1975). Habituation is also highly specific to the stimulus condition, exhibiting poor transfer to other motion environments (Graybiel & Knepton, 1978; Reason & Brand, 1975). Prehabituation to a rotation chair and to parabolic flight were apparently unsuccessful for the Skylab crew (Graybiel, Miller, & Homick, 1974). Also, individual susceptibility to SAS can not be effectively predicted at present, as there is no known relationship to motion sickness susceptibility on Earth.

THEORIES OF MOTION SICKNESS

It is known that the vestibular system must be intact for the development of symptomatology to occur. Although the etiology underlying motion sickness is not entirely understood. Brooks (1939) considers motion sickness to be an overstimulation of the inner ear equilibrium organs (the otoliths) which results in an overflow of neural activity to autonomic nervous system centers that produce the symptoms. In fact, many ANS centers that are responsible for the symptoms are in close proximity to the vestibular nuclei.

A much more widely accepted theory of motion sickness is the Sensory Conflict Theory described by Reason and Brand (1975). According to their theory, motion sickness occurs during conditions of sensory rearrangement; it occurs when the pattern of sensory inputs from the vestibular system, other proprioceptors, and vision is at variance with what is expected, based on stored patterns derived from past experience with the spatial environment. In other words, a "conflict" is thought to result between two contrasting sensory systems, and the integration of

the neurophysiological processes which support them (Mirable, Glueck, & Stroebel, 1979).

The theory states that all situations which produce motion sickness can be characterized by an influx of incompatible sensory inputs, and that the vestibular apparatus of the inner ear must be one of the sensory systems involved. It is hypothesized that these conflicting pieces of information cause the vestibular system's activity to reach non-vestibular sites in the medulla, where first order symptoms (e.g. pallor, sweating, and nausea) have their immediate origin. The vestibular nuclei project into the cerebellum where vestibular, proprioceptive, and visual impulses about one's orientation in space are integrated. Vestibular nuclei are also assumed to project to the reticular nuclei and possibly to other ANS nuclei in the medulla (Guyton, 1981). Benson (1977) proposes the vestibular nuclei as sites that may function as comparators, since they have the necessary convergence of visual, somaesthetic, and cerebellar afferents. Due to the physiological responses that have been observed with the onset of motion sickness, the implicated connections of the vestibular nuclei are the emetic, respiratory, vasomotor, and cardiac centers, although none of these connections has been positively identified (Gernandt & Gilman, 1959).

A high interindividual variability exists in susceptibility and rate of adaptation to provocative motion sickness conditions. These differences are not solely a function of the peripheral vestibular receptors (Reason & Brand, 1975); instead, it is thought that individual differences exist in the relative dominance of particular sensory mechanisms in the overall organization of the perceptual

process. The relationship of different sensory mechanisms to the total perceptual process is believed to cause the high variability in susceptibility to motion sickness. The sympathetic nervous system reactions to motion sickness have been viewed by Reason and Brand (1975) as possible defensive reactions to the specific stress of motion stimulation and not as an integral part of motion sickness. As with almost any environmental stressor, there is an increase in sympathetic activity.

Kohl, (1983) has modified the sensory conflict model to incorporate a more central integrative mechanism, rather than relative dominance of a sensory system. The "neural mismatch" delegates sensory conflict as secondary to mismatching occurring between ongoing sensory experiences and long term memory. A hypothesized candidate for the neural mismatch center is the limbic system. In support of the limbic system's role, Kohl points out the interconnections with viseral centers in the brain stem, hypothalumus, and pituitary. Other connections with the telencephalon are involved in emotions, memory, motivation, and attention. Pharmacological evidence is also sited in support of the limbic system's comparator role in motion sickness. Anticholinergic drugs like scopolamine (an antiemetic) elicit the following responses: 1) decreased acquisition of short term memory without disrupting long term memory retrieval, 2) reduction in preference for and reaction to novelty, and 3) reduction in the ability to focus attention. Therefore, anticholinergic drugs decrease the strength of association between the present sensory information and past experiences, causing novelty not to be recognized. This lack of recognition is followed by a decreased

stress response.

Schneider and Crosby (1980a) believe that current explanations for motion sickness are inadequate, especially for space motion sickness. Although space sickness shows all the signs of motion sickness, additional symptoms have been reported by astronauts and cosmonauts. These additional symptoms include a postural inversion illusion (i.e. a feeling that one is falling or standing upside down), and both formed and unformed hallucinations. Clinical studies of intercerebral lesion cases have led Schneider and Crosby to believe that vascular insufficiency of the posterior cerebral artery, that supplies the temporoparieto-occipital region may account for these additional symptoms of motion sickness that are encountered in weightless environments (Schneider & Crosby, 1980b).

ANS Studies of Motion Stressors

Various early studies have attempted to correlate susceptibility to motion sickness with characteristic alterations in blood pressure and cardiac actions, but have not been able to show consistent relationships (Tyler and Bard, 1949, Taylor, et al, 1960). In a review of the literature, Money (1970) concludes that the inconsistency of ANS responses made meaningful interpretations impossible. Graybiel and Lackner (1980) measured changes in blood pressure, heart rate, and body temperature of 12 subjects after a sudden stop centrifuge test. The results indicated no consistent physiological reactions for within-subject tests or for between-subjects tests. These results were interpreted as discounting the ANS response to motion sickness as a

decisive factor in the development of motion sickness symptoms.

Parker and collaborators have investigated the relationship of the ANS and motion sickness. Parker (1964) demonstrated that nausea could be induced in individuals by viewing a film of a high speed drive on a curved mountain road (without vestibular stimulation). The subjects also viewed the motion film run backwards and a highly graphic film of surgery. The results indicate a clear difference in patterning of ANS response for the motion and surgical films with little response to the film run backwards. The motion film resulted in increases for HR and face temperature with decreased peripheral blood volume pulse (BVP) while the surgical film yielded the opposite physiological reactions. Skin conductance increased and respiration rate decreased during the motion film while less of a response was elicited by the surgical film (although the responses were in the same direction).

A subsequent study (Parker, 1971) demonstrated that subjects could be classified as susceptible or non-susceptible based on forearm skin conductance response to a film depicting high speed travel on a curved road. Ten of the 20 subjects were classified as susceptible (greater than .1 log megaohms change). All subjects were then tested for motion sickness by an hour of sailing in 2-3 foot swells. All subjects classified as susceptibles either vomited or reported strong levels of nausea.

Skin conductance changes have been shown to be less for subjects who report little past boating experience while viewing a motion film (Parked et al, 1972). Repeated exposures of the film depicting a sailing on a rough sea was shown to reduce the magnitude of conductance

responses in susceptibles with each viewing while no change in responsiveness was seen for non-susceptibles (Parker and Howard, 1974).

Autonomic response patterns to motion films have been shown to be different for subjects classified as parasympathetic dominant (P.S.) or sympathetic dominant (S.) based on Wenger's (1941) Autonomic Factor. The P.S. dominant group decreased face and forehead temperature, BVP, and respiration rate (RR), while S. dominant group increased. Skin conductance and HR increased for both groups but less for S. dominant group (Parker & Wilsoncroft, 1978).

The Parker and collaborator studies indicate the following: 1) ANS reactions to motion sickness films can predict susceptibility to sea sickness, 2) Patterning of ANS reactions to motion films are different for other types of stress films, 3) Patterns of ANS reactions to motion films are different for subjects with P.S. dominance and S. dominance, 4) Adaptation (past experience) can be seen in ANS reactions to motion films.

Further evidence of ANS reactions to motion sickness is provided by Cowings, Suter, Toscano, Kamiya , & Naifeh (1984). Provocative tests of coriolis stimulation (rotation about ones own axis) were administered to 186 subjects. Heart rate (HR), Pulse volume (PV), basal skin resistance (BSR), and RR were monitored before, during, and after the test. The subjects were grouped by susceptibility, (high, medium, and low) based on the number of rotations withstood for analysis.

As a total group there was a stress-like ANS response followed by some recovery after rotation. Highly susceptible subjects showed an average of 1.14 beats per minute (BPM) increase in HR, moderate

susceptibles a .44 BPM increase, and low susceptibles a .Ø6 BPM increase across the test. Also, the rate of increase during the first 5 minutes of the test was highest for the high susceptibles while the rate of increase for the last 5 minutes was the same for all groups. BSR decreased during the test for all groups with the high susceptibles showing the largest decrease. PV decreased at the onset of rotation and showed the largest rebound effect for the high susceptibles. RR increased at the onset but did not differentiate the groups. The consistency in the direction of ANS responses across repeated testing of subjects was also reported. Subjects responded with more directional consistency during transition from rest to rotation and following rotation, than during the actual periods of rotation, which presumably incorporated the development of motion sickness.

Specificity and Stereotypy

The sympathetic and parasympathetic systems are thought to be in a state of dynamic homeostasis. Presentation of stressful stimuli (physical or emotional) results in a shift in the relative dominance of each of these opposing systems. The shift toward a sympathetic dominance of the autonomic nervous system, results in a generalized activation of the organism. Later studies have not focused on the general responses of activation but on the individual differences in autonomic reactivity.

Cannon (1929) demonstrated that autonomic responses were organized in the hypothalumus. He viewed the peripheral autonomic responses as reflexive and varying only in intensity and direction. This immediate physiological reaction was termed "fight-or-flight reflex". The emphasis

of the activation concept is on generalized physiological state, ranging from excitement to unconsciousness and the adaptive consequences of the adjustment.

The integration, coordination, and adaptive function of physiological responses to stressful stimuli are well documented and represent a generally accepted model (Duffy, 1962; Hebb, 1955; Malmo, 1959). The stress or activation responses are basically mediated by norepinepherine and epinepherine while conservation or relaxation is produced by acetylcholine. This model holds well for groups of individuals when responses are averaged across people. However, an individual's responses will not always fit this model. In fact, large interindividual differences are seen in physiological responses to the same stressor.

A concurrent line of research has demonstrated that an individual's autonomic responses to stimuli show not only a generalized activation of the organism but an individualized and reproducible response. An individual tends to respond with an idiosyncratic pattern. This patterning of responses for an individual is reproducible for most stimuli presented.

Eppinger and Hess (1910) proposed the ergotropic reflex (moving in the direction of work) and the trophotropic reflex (relaxation and energy conservation) as the mechanisms of physiological adjustment. Imbalances in the mutual antagonisim of the two systems were viewed as a principle that might account for many bodily disorders. Wenger (1941) refined Eppinger and Hess's hypothesis to state that autonomic imbalances may be phasic or tonic and are a result of differential

chemical reactivity of the adrenergic and cholinergic branches of the ANS. The idea of autonomic balance was, in turn, applied by Wilder (1950) to developed the Law of Initial Values (LIV). The LIV states: A higher prestimulus level results in a small increase in the response while a lower prestimulus level results in a larger response.

The work of Lacey and collaborators integrated and extended these concepts to encompass psychophysiological reactivity, and most imporantly, demonstrated that these individual differences are consistent and reproducable.

Lacey et al (1953) challenged the validity of a generalized arousal response, in which the basic argument was that simultaneous recordings of two different physiological functions yielded low correlations. Lacey proposed the principal of relative autonomic response specificity, which states: Subjects tend to respond with an idiosyncratic pattern of autonomic functions, and stereotypy (reproducability) of these patterns are exhibited across different stressors. In the study of these phenomena, Lacey developed the Autonomic Lability Score (ALS) as a general measure of autonomic reaction. In effect, the ALS is a means of standardizing and relieving a physiological response measure from the influence of the prestimulus level. The removal of the influence of prestimulus level is necessary since there is a strong negative correlation between the prestimulus level and the response to the stimulus (Lacey, 1956). Lacey's ALS yields an adjusted score for each individual and this score has a zero correlation with prestimulus levels. The standardization allows comparisons to be made across physiological responses. Using the ALS scores Lacey was able to show

that a significant number of the subjects demonstrated repeatable patterns of responses across different types of stimuli.

In a series of mathmatically sophisticated papers, Engle (1960) was able to show that autonomic response patterns are a function of both the subject's idiosyncratic response as well as the type of stimulus. Twenty subjects were monitored for blood pressure, skin resistance, skin temperature, and heart rate. Five stimulus conditions were used, a loud horn, mental arithmetic, proverbs, cold pressor, and exercise. A covariate analysis was computed to take out the effects of the prestimulus levels (actually the covariate that was used was the anticipation level). Difference scores were then standardized for each of the physiological responses. Different response patterns were evoked by the stimulus type and by the subject, as shown in the significant effects for subjects and stimuli. Coefficients of concordance and ranking of standard scores showed that only the horn, arithmetic, and cold pressor conditions yielded significant effects. The same type of analysis showed that 8 of the 20 subjects demonstrated significant individual response specificity.

In a subsequent replication study by Engle and Bickford (1961) a group of hypertensives were added. The same results were obtained for the normal group while the hypertensive group showed significantly more individual response specificity to all of the stimuli conditions. In this group 66% responded maximally and consistently with blood pressure.

PURPOSE

Strong evidence has been reported by Parker (1964, 1974), Parker et

al (1972, 1974, 1978) and by Cowings et al (1984) for consistent and stable ANS responses to motion sickness, while much of the earlier research reports inconsistent responses. The primary differences in these are that the earlier studies did not utilize any of the conceptual directions supplied by the works of Wilder (1940), Wenger (1941), Lacey et al (1953, 1956) and Engel et al (1960, 1961). The studies by Parker et al and Cowings et al, while not using all of these concepts in their analyses, still demonstrated strong ANS components of motion sickness. Both of these researchers relied on groupings of subjects that effectively seperated for differences in pre-stimulus levels (similar to the LIV) and interpreted the data in terms of patterning of responses.

Most theories of motion sickness, relying on the earlier studies, have discounted the ANS's contribution to symptom development. In turn, theoreticians have placed emphases on sensory sensitivity or central nervous system integration of motion stimuli. Further demonstration of consistent and reproducible ANS responses to motion stimuli is needed before modification of current theories are justified.

Concepts of stimulus specificity and stereotypy for ANS resposnses have been developed in the area of psychophysiology that will facilitate understanding of the ANS's relationship with motion stressors. Application of these concepts to motion sickness requires viewing ANS responses as a function of both the type of motion stimuli presented and an individual's tendency to make a particular type of response to these stimuli.

The major purposes of the thesis presented here are to :
(1) determine the general autonomic response pattern that prevails

across different motion stressors

(2) determine the type of stimulus specific response patterns associated with three types of motion stressors

(3) determine the types and occurrences of idiosyncratic patterns for motion stressors

(4) determine the relationship between idiosyncratic patterns and subjective reports of malase.

MATERIALS AND METHODS

Subjects

Twenty-two naive subjects (14 males and 8 females) ranging in age from 18 to 35 (mean age of 23.23) were used in this experiment. The basic requirements for participation were: (a) certification of physical fitness by medical examination; (b) clean shaven to observe pallor during the stressors; and (c) willingness to cooperate, as evidenced by a signed informed consent form. A minimum of two hours pay, at five dollars per hour, was assured for each visit.

Additional selection from the larger database used the following criteria: a) participation in three stress tests and two baseline conditions, b) at least ten minutes of data on each of the three stressors, and c) "good" physiological data on all channels for the duration of each condition used in this paper.

Physiological Measures

During each condition physiological measures were recorded on strip chart recorders and analog tape. Real-time signal processing and data reduction were performed using a Nicolet Med-8Ø laboratory computer. The methods used for monitoring each of the physiological responses were as follows:

Blood Volume Pulse (BVP) Amplitude - Relative changes in peripheral vasomotor activity was monitored using a photophethysmograph, an incandescent photoemitter and a transistor mounted in a clip placed on the left index finger. All BVP data was inverted so that higher values

represent vasoconstriction.

Respiration Rate (RR) - Respiratory cycles were detected by a thermistor taped to the nasal passage.

Heart Rate (HR) - Electrocardiography was monitored using three disposable silver-silver chloride electrodes. Each electrode was pre-gelled and self-adhesive. Standard precordial placement was used. Exploring electrodes were placed on the midclavicular line over the fifth intercostal space and over the fourth intercostal space to the right of the sternum. The ground electrode was placed over the midclavicular line over the fourth intercostal space.

Basal Skin Conductance (COND) - Absolute changes in the electrolytic properties of the skin was monitored from surface silver-silver chloride electrodes. Two dry electrodes, mounted on velcro, were attached to the middle and ring finger pads on the left hand. Higer COND values represent more sweating.

Motion Sickness Test

CSSI: Coriolis Suscptibility Sickness Index (CSSI), a widely used Coriolis acceleration test was conducted with a Stille-Werner rotating chair. Padded head rests are mounted on the left, right, front and back of subjects, allowing for the execution of head movements at 45 degree angles. Subjects were blindfolded during CSSI tests. The CSSI tests were conducted by initiating rotation of the chair at 6 rpm and incrementing by 2 rpm every 5-minutes. The maximum velocity is 30 rpm. During each 5-min interval at a constant rotational velocity, subjects executed 150 head movements at 45 degree angles in four quadrants.

Instructions for making head movements at 2-second intervals were delivered to subjects by a tape recorded voice. The direction of head movements were randomized. There was a 30-second pause between each 5-min period (no head movements but continued rotation) during which a diagnostic scale was administered. The maximum duration for the CSSI tests was 65 minutes.

DRUM: Combined optokinetic and Coriolis acceleration (DRUM) tests were conducted using the rotating chair described above, located in the center of a rotating drum. The drum is 1.676 m high and 1.829 m in diameter. The inside of the drum is painted with alternating black and white strips which are 17.78 cm wide. The rotation of the drum is controlled independently of the chair, and is capable of a maximum velocity of 30 rpm. The DRUM tests are conducted by initiating chair rotation at 2 rpm and drum rotation at 4 rpm in a clockwise direction. During a 5-minute period at these constant rotational velocities, subjects are instructed to perform 150 head movements at 2-sec intervals in four quadrants. Every 5-minute, the speed of the chair was incremented by 2 rpm and the velocity of the drum was incremented to twice that of the chair. The maximum velocity of the chair in these tests was 10 rpm while the drum was 20 rpm. The diagnostic scale was administered at five-minute intervals. The DRUM tests had a maximum duration of 25 minutes.

VARD: Linear acceleration tests was conducted on the Vertical Acceleration and Roll Device (VARD) located at Ames Research Center. The VARD is a light-proof enclosed cab which can achieve a maximum displacement of + or - 1.829 m during sinusoidal oscillations. The

frequencies and g-load are programmable. Subjects were monitored by closed-circuit video and by an intercommunication system throughout tests. VARD tests were conducted by initiating vertical sinusoidal oscillations at Ø.33 Hz, Ø.35 g. Subjects were instructed to perform head movements in four quadrants at 2-sec intervals. The diagnostic scale was administered at five-minute intervals. The VARD tests had a maximum of 75-min of oscillation.

Assessment of Malaise

The subjective level of malaise experienced by each subject during the stressors was determined by the Coriolis Sickness Susceptibility Index (MAL), as show in the appendix A (Graybiel, Wood, Miller & Cramer, 1968). The MAL diagnostic scale consists of a series of questions asked by the experimenter that are directed at the subject's perception of his own physiological state during rotation. These are regarding changes in temperature, dizziness, headache, drowziness, sweating, salivation, and nausea. The scale also includes a record of the experimenter's observations of facial pallor and sweating. Changes in a response are weighted differently according to their relative importance in development of the motion sickness. Each 5 minutes of stimulation was scored separately. The point value of the subject's reports fell in one of four categories of sickness: mild (malaise I), moderate (malaise II), severe (malaise III), or frank sickness (emesis).

Procedure

Optokinetic stimulation (DRUM), vertical acceleration and roll

device (VARD) and coriolis motion tests (CSSI), were conducted in experimental test chambers on consecutive days. 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, (c) vomiting occurred, which happened rarely, or (d) the maximum duration of the test was achieved.

Resting baselines were conducted in a darkened, sound-isolated chamber for 30 minutes while listening to tape recorded music. This chamber was not used for any of the stress conditions. The session was repeated on separate days for each subject after completion of the stress test.

Design and Analysis

In this design there was no systematic counterbalancing of testing order since previous studies have shown that there is no habituation transfer across various types of vestibular stimulation, (Graybiel & Knepton, 1978; Reason & Brand, 1975). In addition, unpublished results of analyses in connection with the current study indicate that there is no substantial ANS carryover effects various types of vestibular motion stimulation.

The experimental design for this study uses a completely repeated measures ANOVA. There are three stressor conditions (CSSI, DRUM, VARD), four normalized ANS variables (BVP, RR, HR, COND), and four epochs of data (minute one, five, fifth minute from termination, and the last

minute), consituting a 3 X 4 X 4 design. This type of design uses a different error term for each of the testable effects and comparisons. The error terms are comprised of the differential effect of the subjects plus the true experimental error (Keppel, 1982). The subject by condition effects are real and interesting sources of variance (non-additive model), however this ANOVA design does not permit disentanglement of the separate contributions to these terms (Wilson, 1967).

A repeated measures ANOVA was computed on the data set as a whole, followed by ANOVAs for each of the tests separately to show the types of patterns associated with each of the conditions. Trend analyses were then used to determine the relationship between the stressors for each ANS response over time. Omega squares were computed to estimate the magnitude of variability associated with the idiosyncratic responses of the subjects (Dodd and Schultz, 1973). Each ANS measure was corrected for the average responses of each cell mean, as an estimate an individuals non-specific reactivity. Cluster analyses were then computed (Anderberg, 1973), for each test separately, to determine the types and frequencies of patterns occurring in each test. Cluster analysis partitions subjects into groups that have the most similar type of response pattern. The percentage of subjects exhibiting response stereotypy was estimated by the occurrence of subjects in similarly characterized clusters across the stressors. These clusters of subjects with similar response patterns were used as a grouping factor to compare subjective reports of malaise.

This series of analyses is an attempt to adequetly account for the

previously set fourth principals of psychophysiological litterature pertaining to stimulus specificity and individual stereotypy. There are no defined statictical models to follow in this pursuit and controversy abounds ,at each step, regarding the appropriate procedure to be followed. Unfortunately simplicity is not the answer to this problem, as many reaschers seem to believe. The analyses has a logical ordering of data manipulations and statistical analyses and has attemted to remain as simplistic as possible for addressing the question at hand.

RESULTS

Data Description

The average number of minutes withstood for the CSSI condition was 21.09 minutes, 19.41 for the DRUM, and 48.23 for the VARD. The average malaise reported was 12.09 for the CSSI, 11.55 for the DRUM, and 9.55 for the VARD. Subjective self-reports of malaise (MAL) are used as an index of just how sick a subject became during the test. Severe sickness is considered eight points or more, and termed malaise level three. All subjects (22) reported malaise level three for the CSSI, while three subjects did not reach this level in the DRUM and eight did not in the VARD. No sex differences were found.

To facilitate analysis, four epochs were selected for each test that would both equalize the size of the data set and characterize each subject's responses to the stressors. The epochs selected for analysis were, the first minute of the test (M1), the fifth minute (M+5), the fifth minute before the subject terminated the test (E-5), and the last minute of the test (E).

Each subject's stress data were transformed to z-scores based on their own mean and average standard deviation of each physiologcal parameter for the two days of baseline. This transformation meets two requirements for the data set: a) normalization of values across the parameters, and b) reflection of the magnitude of the response induced by the stressor for the subject, based on his/her own resting levels and normal variability.

A preliminary analysis using the time that each data point was

collected as a covariate demonstrated that the differences in test length has very little effect on the ANS responses. Therefore, the differences in the time of collection of the last two epochs were considered negligable contributors to variance and are not statistically controlled in the remainder of the analysis.

Stimulus Specificity

The first repeated measures ANOVA contained the complete data set, three stressors, four epochs and four standardized ANS measures. The main effects and interactions that sum across the ANS variables in the ANOVA are of little interest due to problems of interpretation. Any Sum of Squares that collapses across the ANS variables will be difficult to interprete for example, what is revealed when adding HR to COND? A possible interpretation is an index of autonomic tone, however since autonomic variables rarely show strong positive correlations this interpretation would probably be incorrect.

Three interactional terms are of interest, all of which are significant. Each of the tests produced different levels of sustained activity or average responses of the ANS variables, as indicated from the Test X ANS interaction, F (6, 126) = 2.67, $p < .05$. This interactional term sums across the epochs for each response and may be viewed as an indication of sustained activity because each subject is represented by their means for each ANS variable on each test. This points to the fact that the tests differencially effect the sustained activity of the ANS responses.

There are similarities in the patterns of responses for the tests,

or a reproducibility of response patterns across the tests, which is significant as shown by the Epoch X ANS interaction, F (9, 189) = 6.45, $p < .001$. This term sums across the tests, indicating that there is a patterned response that is similar to all three of the tests.

The triple interaction of Test X Epoch X ANS is also significant, F (18, 378) = 2.44, $p < .001$, showing that there is a distinctive response pattern associated with each of the stressor conditions. The F-ratio expresses the degree to which there are specific response patterens that appear for all subjects under a particular stress condition. The error term represents the variabiltiy in response components that are unique to the individual stressors.

Identification of the response patteren associated with each of the stressors was preformed by three separate ANOVAs, each using only the data collected for each test. The analysis within the CSSI condition demonstrates a significant response pattern associated with the test as shown by the Epochs X ANS effect, F (9, 189) = 6.71, $p < .001$. Figure 1 illustrates the response pattern for the CSSI condition. The simple effects for each of the ANS variables were significant, with the exception of BVP, showing that the the responses were stable across the epochs. These effects are as follows:RR F (3, 63) = 6.28,$p< .001$; HR F (3, 63) = 7.46, $p < .001$; and COND F (3, 63) = 4.89, $p < .01$.

The ANOVA within the DRUM condition (Figure 2) also shows a significant response pattern, from the two-way interaction, F (9, 189) = 6.44, $p < .001$. The simple effects for each of the ANS responses were significant, demonstrating stability, with the exception

of BVP. The simple effects for the ANS variables are as follows: RR F (3, 63) = 6.00, $p < .001$; HR F (3, 63) = 7.70, $p < .001$; and COND F (3, 63) = 5.48, $p < .01$.

Figure 3 illustrates the response pattern induced by the VARD. The ANOVA within the VARD condition does not show a strong significant response pattern F (9, 189) = 1.79, $p = .07$. Computations of the simple effects were carried out since the overall comparison was close to a .05 level of significance to see if any of the ANS responses are

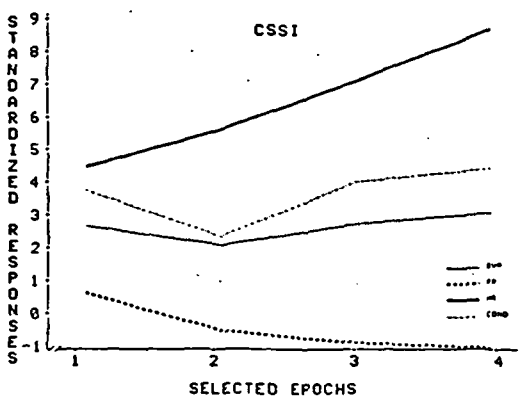

Figure 1. Standardized autonomic responses to CSSI condition for selected epochs.

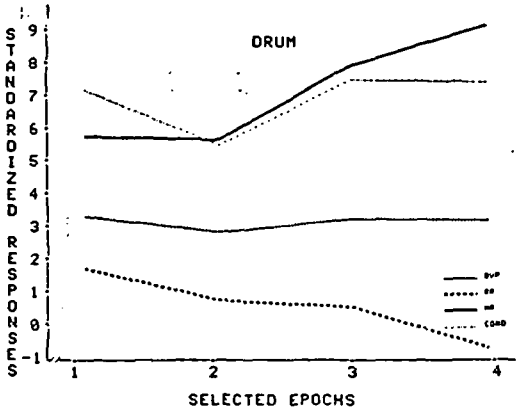

Figure 2. Standardized autonomic responses to DRUM condition for selected epochs.

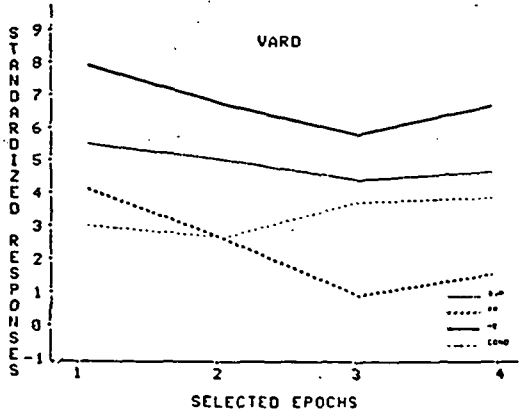

Figure 3. Standardized autonomic responses to VARD condition for selected epochs.

stable. The only effect that reached significance was RR, F (3, 63) = 5.92, $p < .001$.

Comparisons Across Tests

Having identified the stable response patterns associated with the three tests, the analysis now addresses the similarities and differences that are induced by each of the stressors on each physiological response. Due to the great number of possible and interesting comparisons to be made, trend analyses across the epochs were determined to be the most efficient procedure. The results of the trend analyses should not be viewed as characterizing the true time course of the responses, due to the small number of epochs used. However, this procedure does accurately define the probability that two responses changing in the same or different ways over time. Limited comparisons were also made between tests on the first and last epochs, since the trends in a resposne may be the same across tests and yet show different levels.

Figure 4 depicts the trends of the BVP response to the tests. The quadratic main effect for the BVP responses F (1, 21) = 3.55, $p = .07$ showed the strongest effect of any weighting scheme. This component accounts for 73% of the variation between the BVP responses across the tests, indicating that the stressors induce approximately the same (quadratic) effect on BVP. The BVP responses for the first minute of the stressors resulted in no difference for the CSSI and DRUM response, while the average of the two conditions was significantly lower (less vasoconstriction) than the VARD BVP F (1, 21) = 11.58, $p < .01$. During the last minute of the tests this distinction had disappeared and

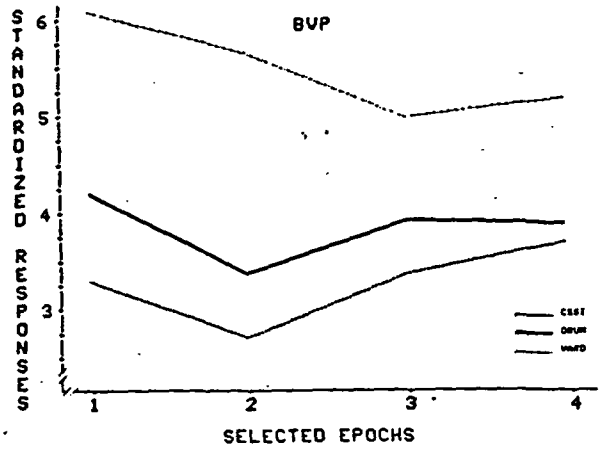

Figure 4. Standardized BVP responses on three tests for selected epochs.

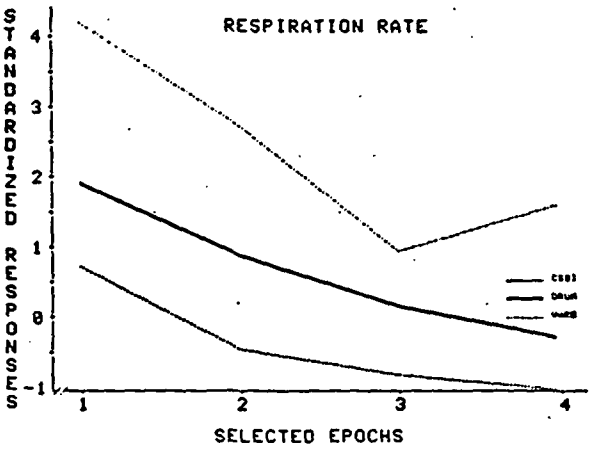

Figure 5. Standardized RR responses on three tests for selected epochs.

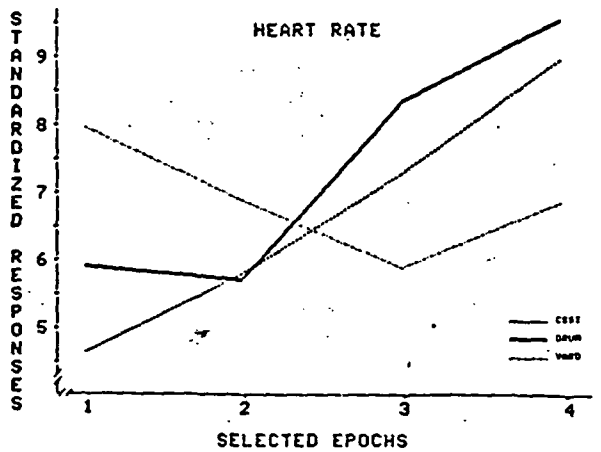

Figure 6. Standardized HR responses to three tests for selected epochs.

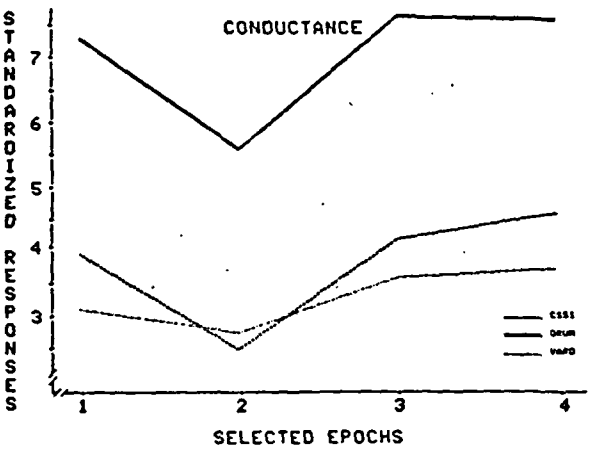

Figure 7. Standardized COND responses on three tests for selected epochs.

there were no differences in the levels of the BVP for any of the stressors.

Figure 5 depicts the trends in RR across testing conditions. There is a significant linear main effect for RR F (1, 21) = 16.95, $p < .001$, accounting for 88% of the variation between the RR responses across the tests. This finding indicates that each of the conditions result in a steady linear decrease in RR throughout the test. The RR responses for the first minute show all tests to be significantly different from each other. The DRUM shows a higher RR than the CSSI F (1, 21) = 9.88, $p < .01$, and these conditions combined have a lower RR than the VARD F (1, 21) = 12.17, $p < .01$. During the last minute of the tests the differences in RR for the CSSI and DRUM had disappeared while the average of the two was significantly lower than the VARD F (1, 21) = 5.96, $p < .05$.

There is a significant interaction for the effect of Tests X Linear HR F (2, 42) = 13.28, $p < .001$, as depicted in Figure 6. This linear interaction of HR X Test accounts for approximately 89% of the overall variation between the interaction. Heart rate shows a steady increase for both the CSSI and DRUM conditions and a decrease for the VARD. Ther are no differences, during the first minute, for the CSSI and DRUM test, while the combination of these were significantly lower than the VARD condition F (1, 21) = 4.76, $p < .05$. The last minute of the stressors shows no difference in the CSSI and DRUM, while there was a complete reversal (interaction) in the difference seen in the VARD. On the last epoch, the VARD condition produced a significantly lower HR than the other two tests combined F (1, 21) = 4.597, $p < .05$.

Figure 7 shows the COND response to the three tests. There is a similarity of responses with a quadratic trend for COND across the stressors as demonstrated by a significant quadratic main effect F (1, 21) = 12.02, $p < .01$. This accounted for 24% of the variance between the tests. However, there is also a significant cubic main effect that accounts for an additional 48% of the variance in the COND responses to the tests F (1, 21) = 7.21, $p = .01$. The first epoch of COND show no difference between the CSSI and VARD, while the combination of these are significantly lower than the DRUM condition $F(1, 21) = 6.61$, $p < .05$. The differences in the last epoch of the tests were very similar to the first epoch. The CSSI and VARD are not significantly different, while the DRUM induces a higher response F (1, 21) = 6.46, $p < .05$.

Idiosyncratic and Stereotypic Responses

Omega squares were computed for all the testable effects for each of the stressors. The controllable effects in CSSI condition accounted for 55.1% of the variability associated with the test, leaving 44.9% of the total variability due to subject variability plus true experimental error. The variability associated with subjects and error in the DRUM condition was 88.4%, while in the VARD it was 92.9%.

The subject variability in these analyses is a very large part of the overall variability of the data. Furthermore, it should be noted that the subject variability increases for the tests as the average subjective reports of malaise decrease. To analyze this uncontrolled contributor of variability the difference in each data point and its cell mean was

generated for each subject as an indicator of non-specific reactivity. This procedure also removes the effect of the test with regard to time course of the tests, resulting in relatively flat response curves for each subject. Therefore, averaging across epochs for a response results in a more stable estimate of a subject's non-specific reactivity.

The average adjusted scores for each subject on a stressor were submitted to a cluster analysis. This technique partitioned the subjects into groups, or clusters, that have the most similar type of idiosyncratic response pattern. A similar type of pattern is defined as the Euclidian distance (the square root of the sum of the squares of the differences between the values of the variables for two subjects). This was the measure used to assign subjects to clusters. This procedure empirically derives clusters of subjects with a minimum geometric distance between the values of all variables for the subjects within a cluster, while maximizing this distance between the clusters. Values close to zero have little determination on cluster development while larger absolute values of a variable carry a heavier weight. For example, a subject who responded with only an extremely large HR response will tend to be grouped with subjects displaying a similar response pattern. The optimal number of clusters was defined as the fewest number of clusters that resulted in significant separation of the variable between the groups. However, RR never resulted in significant differences between the groups since the values were close to zero and did not weight heavily in group assignment.

Five distinct clusters were derived from the CSSI condition, as shown in Table 1. The largest cluster contained nine subjects and was

characterised by a low COND response. The second largest grouping contained five subjects that exhibited a very high COND response. The other two groups were each comprised of three subjects, one group underreacted with BVP and COND, while the other overreacted with HR. The smallest group, with only two subjects, overreacted primarily with BVP.

TABLE 1
SUMMARY OF CLUSTER ANALYSES

CLUSTERS	CSSI	DRUM	VARD
LOW COND	1, 2, 6, 7, 10, 13, 19, 20, 32	1, 3, 6, 7, 10, 11, 16, 19, 20, 21	2, 3, 6, 10, 11, 12, 16, 18, 19, 20, 21, 22
HIGH COND	2, 4, 14, 18, 22	4, 13, 14, 17, 18	7, 13, 14, 17
HIGH HR	3, 8, 9	2, 5, 8, 9, 12, 15, 22	4, 8, 9, 15
HIGH BVP	15, 17	none	none
LOW BVP & LOW COND	11, 12, 16	none	5, 1

Table 1. Subject numbers are entries, showing the frequency of pattern type in each cluster and where each subject was located for the three stressors.

Three different groups were found for the DRUM condition, see Table 1. The largest cluster contained ten subjects with a very low COND response. The next largest group with seven subjects primarily underresponded with HR. The last group, containing five subjects, overresponded with COND.

In the VARD condition subjects clustered into four distinct groups, as shown in Table 1. The largest contained 12 subjects and was characterised by small underreactiveness of COND. Two groups were

comprised of four subjects each. One group exhibited a large overreactivness of COND, the other group was characterized by a high HR response. The smallest group showed a moderately low BVP and COND response.

Stereotypy of response was demonstrated by a subject's response pattern occurring in a similarly characterized cluster across stressors. Eight subject's response patterns (36.36%) occurred in the same cluster in all three test. Five of these were in the low COND clusters, two in the high HR groups, and one in the high COND groups, see Table 1. Of the remaining subjects, an additional 45.45% were in similar clusters on two of the tests, six subjects responded similarly in the DRUM and CSSI, and four subjects responded similarly in the CSSI and VARD. The remaining four subjects (18.18%) exhibited no similarity of responses across the testing conditions and therefor moved to a different group on each test.

Comparisons were made using t-tests with separate variances to address how the patterns of non-specific reactivity are related to the subjective reports of discomfort. In the CSSI condition the high HR group reported more malaise (12.67) than the low BVP low COND group (9.0), $p < .05$. Also the low BVP low COND group reported less malaise (9.0) than the low COND group (13.0) $p < .01$. In the DRUM the high COND group reported higher malaise levels (14.0) compared to the low COND group (10.1), $p < .05$. In the VARD condition it was found that the high HR group reported a higher malaise level (13.25) than the low COND group (6.83), $p < .01$. Also, the high COND group withstood less of the stressor (38.25 minutes) than the low COND group (59.25 minutes), $p < .05$ during the VARD condition.

Since some of these comparisons involve very small number of samples, similar groups across the tests were combined, to increases the size of each group. Furthermore, by combining the groups, any differences seen in malaise levels demonstrates that the association holds across the testing conditions. The high HR groups (12.43) reported a higher malaise than the low COND groups (9.61), $\underline{p} < .01$. Also the high COND groups reported more malaise (11.93) than the low COND group (9.68), $\underline{p} < .05$.

DISCUSSION

The results demonstrate that the ANS responds vigorously to motion stressors. There is a consistently reproducable response patteren associated with all three of the stressors. In addition to the general components, each of the stressors produce a distinctive ANS response pattern (stimulus specific). Furthermore, the majority of subjects tend to exhibit reproducable patterns of over or under reactivity of responses across the different stressors (individual stereotypy). The particular pattern of reactivity that the subject displays is associated with his/her subjective report of malaise and to a small extent susceptibility.

The predominant similarity in the tests is that all ANS responses are initially higher than resting levels; and that throughout the test, they continue to be higher than baseline levels, with the exception of RR. The CSSI and DRUM both produce similar levels of BVP, below baseline levels for RR toward the end of the tests, and steady increases in HR. The Vard and the CSSI both produce similar COND levels throughout the tests. Furthermore, there are similarities in the time course of responding to the stressors for BVP, RR, and COND.

These results are supported by Parker (1964) and by Cowings et al (1984); in that there is a characteristic ANS response to the development of motion sickness. They both report a general stress-like response at onset of motion stimulation; which includes increases in heart rate, sweating, respiration rate, and peripheral vasoconstriction. In addition, Parker also reports a time course decrease in respiration rate during the

stimulus which coincides with the findings of this study.

The major distinctions between the tests are that: 1) the VARD condition results in a decreasing trend in HR until the last epoch of the stimulus, while the other tests produce increasing trends, 2) the VARD also produces higher levels of BVP and RR than the other tests, 3) the DRUM results in a higher COND level than the other tests, and 4) the CSSI yields a lower RR at the beginning than the other tests and decreases below resting levels by the second epoch.

These results demonstrate that different motion stressors induce a different type of effect on the ANS pattern. Linear acceleration results in a steady decrease in HR during the first half of stimulation while the rotational stressors produce a steady increase in HR. Also, higher RR and more vasoconstriction are associated with linear motion. However, there are many differences in the level of a response that are not associated with the directional movement of the stimuli. The closest comparisons to be made from the literature are studies that use completely different types of stressors. Parker (1964) reports opposite ANS responses to a motion sickness inducing film and a surgical film, and Engle (1960) reported different ANS responses to a loud horn, arithmetic, and cold pressor stimuli.

In general, the onset of motion stress produces a large sympathetic shift in autonomic physiology, which is seen in all the conditions used in this study. Furthermore, the time course of this sympathetic shift is similar across the stressors, particularly in the final stages of the tests. Convincing as this evidence appears, still it is not a complete view. Major differences are seen between types of motion stressors in

their influence on this sympathetic shift and time coure of responding. Most pronounced are the differences between linear and rotational stimulation in both sympathetic shift and time course of responses. Differences are also seen in the sympathetic influences by adding visual stimulation to the rotational condition.

The increase of sympathetic tone, as a general description of the physiological reaction to motion stress, is complicated by consideration of the responses over the time course of the stimulus. Characteristicly, stress responses that are directly related to sympathetic stimulation involve copious sweating via hypothalamic areas; and within the lower brain stem, cardiac acceleration and vasoconstriction, via the vasomotor center of the pons. Increases in respiration are not considered to be a direct autonomic function, however almost any factor that increases vasomotor activity has at least a moderate effect of increasing respiration via the closely related center of the medulla. Furthermore, there is a moderate degree of neural signal spillover mutually occurring between the vasomotor and respiratory centers (Guyton, 1981).

In the case of motion stress, there is a steady decrease of respiration rate throughout the tests, even to the extent of falling below baseline levels; while being accompanied by increasingly high levels of HR and vasoconstriction. However, increases in respiration rate do not necessarily translate into an increase in respiratory ventilation. This uncoupling of vasomotor activity and respiration is seen only in the two rotational conditions and not during linear motion. Although this may be a function, not of directionality of the stressor, but, of the extent of symptom development. This alternative view is based

on the fact that the linear stress had the lowest malaise levels and the most subjects reporting no symptoms at all. Disentanglement of these two hypotheses are not possible with the design used in this study, or from the number of subjects used. To fully address this idea of vasomotor-respiratory uncoupling, be it a function of directionality of stimulus or extent of symptom development, it is necessary to obtain a measure of tidal volume. However, tidal volume measures were not taken in this study since it was not realized until this analyses that the uncoupling of vasomotor activity and respiration were of possible importance.

The percentages reported from the Omega squares analyses must be taken as approximations since the uncontrolled variability is overestimated by the number of controlled conditions (in this case eight) times the true experimental error. Conversely, the controlled variability is underestimated by the same amount. Quantitatively there is no means of disentangling the true error term from subject variability. However, by implication the extent of idiosyncratic responding of the subjects increased from the CSSI to the DRUM with the greatest amount in the VARD, given that true experimental error does not greatly differ across the tests. This increase in subject variability for the DRUM and VARD coincides with a decrease in the levels of subjective reports of malaise. In fact, this is to be expected since lower malaise levels indicate less of a stimulus effect; thereby allowing for more individualized patterns of responding to emerge.

The results of the cluster analyses show that 36.36% of the subjects make similar responses on all three stressors and 82% respond similarly on at least two of the tests. There are no previous studies in the

literature that use cluster analysis to determine stereotypical responses. Engle (1960) used concordance analysis and reported that 40% of the subjects show directional consistency across widely different stimulus conditions. Cowings et al (1984) reported that a large percentage of subjects show directional consistency during the recovery period on repeated exposures to the same type of motion stressor.

Cluster analysis is one of the only methods for directly examining patterned ANS responses. Furthermore, it does not require rank-ordering of responses. Most analytic techniques in the literature, however, determine consistency of response by ranking of responses; and in the process, negating potentially important information. Other approaches analyze each response separately and draw inferences regarding the patterns. These approaches have yielded definitive results; however, the clustering approach seems to be a superior method with promise of greater utilization by researchers.

The superiority of cluster analysis is exemplified by the fact that the clusters are associated with the subjective reports of malaise, both within a stressor and across the stressors. In general, the response patterns that are more sympathetic-like in their characterization resulted in higher subjective reports of malaise. Parker (1971) reported that higher levels of skin conductance were associated with higher reports of malaise. This supports the results of the present study that show high conductance responders report higher malaise and withstand less of the stress than low conductance responders.

It must be noted that the original transformation of the data to z-scores will significantly effect the results of the cluster analysis.

In all the conditions, COND responses were the largest contributor to cluster development, and the majority of subjects were therefore characterized as COND responders. There are two possible explanations for this; either the transformation biased the data, or in fact COND responding is a major contributor to symptom development. The transformation (z-scores based on the subject's own levels and variability without stress) directly implies the author's belief that a subject's ability to percieve a change in his/her physiology is a function of how far it has changed from what "feels" normal. Weighting the data in this way brings COND to the forefront; furthermore, it is associated with subjective reports.

The amount of time that a subject was able to withstand a stressor was differentiated by response patterns only for the VARD condition. Cowings et al (1984) report HR (untransformed) to be strongly associated with susceptibility to a rotational condition. This is not supported by the present study, however, it is likely that the transformation used in this analysis is more appropriate for identifying malaise than differences in susceptibility. However, the propensity for a subject to respond similarly across stressors was not associated with time to the end of the test, which is similar to Cowing's results. They report that the direction of change is consistent and occurred primarily during transition to, or from, rotation and not during the stress itself.

Considering the large effect of response pattern on malaise levels, and the minimal effect on the duration of the stressor, it seems reasonable to hypothesize that development of symptomatology hinges, not on intensity of the stressor, but on subjective interpretation of the

intensity; which, in turn is intimately linked to the autonomic response pattern. Furthermore, since a high percentage of subjects consistently produced the same autonomic pattern across the tests, subjective interpretation of the intensity of the stressor is fairly consistent. This is the case, at least, when the physiological data are in terms of his/her own normal state. In general terms, this approach to the question of patterning of ANS responses reveals that a subject tends to consistenly produce the same pattern and consistently interpret the intensity of the stimulus (duration of the stress).

In conclusion, transformation of ANS data to reflect divergence from a normal level can enhance interpretation of the results. The results constitute a strong demonstration that the concepts of stimulus specificity and individual stereotypy can guide analysis and interpretation of ANS responses to motion sickness. Particularly, this study has shown that there is a general ANS response pattern representing motion stress, and in addition there are different patterns for each type of stressor. Removing the effects of the test and clustering the non-specific responses of subjects is a new approach to determining individual patterns of responding. This method yields strong relationships to subjective reports, which is necessary for complete validation of a statistical procedure. Furthermore, the clustering technique demonstrated a strong consistent of responses patterns across the tests for most subjects, in fact larger than other studies reviewed by the author.

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APPENDIX

SUBJECT _____ DATE _____ TIME _____

Disc ID#: Tape ID#:

PRETEST: BP / Temp: Pulse:

POSTTEST:BP / Temp: Pulse:

Cal time: Zero cal time: Data collect time:

DIAGNOSTIC CRITERIA SCALE - MOTION SICKNESS TEST

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

CHAIR RPM'S CW/CCW	SYMPTOMS OBSERVED											TOTAL POINTS
	VMT	TMP	DIZ	HAC	DRZ	SWT	PAL	SAL	NSA	ED	EA	
6												
8												
10												
12												
14												
16												
18												
20												
22												
24												
26												
28												
30												