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The Effects of Autogenic-Feedback Training on Motion Sickness Severity and Heart Rate Variability in Astronauts

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Summary

Space motion sickness affects 50 percent of all people during early days of spaceflight. The present study describes preliminary results of a Shuttle flight experiment in which Autogenic-Feedback Training (AFT) was tested as an alternative to pharmacological management of this disorder. AFT is a physiological conditioning method which has been used to train people to voluntarily control several of their own physiological responses and thereby suppress motion sickness symptoms. Thirteen subjects participated in this study (four women and nine men) of whom six later flew aboard the Space Shuttle. Of the 13 subjects, 10 were given AFT. Of the six who were designated as flight subjects, three were given treatment and three served as control subjects (i.e., did not receive AFT). All subjects participated in baseline data collection sessions. These included both rotating chair and vertical motion sickness inducing tests, and 12 hour mission simulations. Treatment subjects were given rotating chair motion sickness tests after 2, 4, and 6 hours of AFT.

Preflight results showed that AFT produced a significant increase in tolerance to rotating chair motion sickness tests. Further, this increased tolerance was associated with changes in specific physiological responses and reports of reduced malaise. Spectral analyses of heart rate variability revealed that power in the low-frequency band decreased after AFT, while power in the mid-frequency band increased. Further, coherence between heart rate and respiration was significantly higher after training.

The flight results showed that two of the three control subjects experienced multiple vomiting episodes on the first mission day, while one control subject experienced only moderate malaise. All control subjects took medication for symptom suppression and/or sedation. Of the three treatment subjects, one experienced only mild discomfort, one experienced moderate discomfort (one vomiting episode on mission day 2), and one experienced severe motion sickness on the first day. The latter subject took a laxative on mission day 4 for symptoms unrelated to motion sickness. None of the other treatment subjects took any medication throughout the flight. Measures of cardiac function reflective of vagal control were shown to be affected especially strongly on the first day of spaceflight. AFT given for control of heart rate, respiration, and other autonomic activity influenced both the vagal control measures and the space motion sickness symptoms experienced. Comparisons of flight to ground-based simulation data revealed significant differences between physiological responses on Earth and in space.

These data suggest that AFT may be an effective treatment for space motion sickness, but this is not demonstrated conclusively with the small number of subjects described in this paper. It was concluded that continuous physiological monitoring combined with self-reports of symptoms provides an objective method for examining individual differences in adaptation to spaceflight and the time course of this adaptation. Further, it was possible to clinically predict from the preflight training performance which of the flight treatment subjects would be most resistant and least resistant to symptoms in space.

Introduction

The Problem of Space Motion Sickness

Since the Space Shuttle accident in 1986 the United States space program has undergone an extensive restructuring. Additionally, the United States has renewed its commitment to an American presence in space. This commitment is evident by the eagerness of NASA, the military, and private industry to return to space. All three groups are planning manned missions into space. These new plans include extended duration Shuttle missions (30 days), the Space Station, a mission back to the moon, and a mission to Mars.

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In space, the absence of gravity alone causes unique physiological stress. Significant biomedical problems such as loss of body fluids, diminished musculoskeletal strength, cardiovascular deconditioning, and reduced sensorimotor control have been reported (Sandler and Vernikos, 1986). The time course of development of these disorders and the severity of symptoms experienced by individuals vary widely. A major biomedical concern which occurs early in the mission is a form of motion sickness known as space motion sickness (SMS).

Motion sickness is a generic term which includes sea sickness, air sickness, car sickness, simulator sickness, cinerama sickness, space sickness, etc. Each condition is a form of the malady and is named after the environment or vehicle. Generally, motion sickness is induced by actual motion; however, motion sickness can also be induced by perceived motion. Although motion sickness can be considered to be a disease it is also a normal response to an abnormal environment. In fact, the absence of symptoms during a motion stimulus may indicate a deficient vestibular system (Dhenin, 1978; Reason and Brand, 1975).

Motion sickness is a physiological dysfunction induced by a real or perceived motion stimulus and characterized primarily by nausea, pallor, cold sweating, and vomiting (Dhenin, 1978; Reason and Brand, 1975; Homick, Reschke, and Vanderploeg, 1984). Other possible symptoms include salivation, feeling of warmth, lightheadedness, depression or apathy, yawning and drowsiness, headache, and occasionally hyperventilation.

The currently accepted explanation for motion sickness is the sensory conflict theory (Dhenin, 1978; Reason and Brand, 1975). The theory suggests that the brain is constantly receiving information from the visual system and from the vestibular system on the position and movement of the body. Sensors in muscles of the neck, arms, legs, and other parts of the body also provide the brain with positioning data known as proprioceptive information. Motion sickness can occur when the brain perceives these various signals to be in conflict with normal motion cues (Gillingham and Wolfe, 1985).

Space motion sickness (SMS) is "characterized by increased sensitivity to motion and head movements, headache, malaise, lethargy, stomach awareness, loss of appetite, nausea, and episodic vomiting" (Jenning, Davis, and Santy, 1988). However, unlike terrestrial motion sickness, space motion sickness rarely induces pallor or sweat (Reschke, 1990). In 1983, Graybiel and Lackner studied the effect of motion sickness in microgravity and macrogravity. Their data suggest that SMS is a result of the brain receiving conflicting information from the visual system and the gravity receptors (otoliths) of the vestibular system. However, their data also point out that motion sickness is enhanced when the eyes are opened and the sight of the surroundings is permitted (Graybiel and Lackner, 1983). These results agree with the actual occurrences of space motion sickness during both American and Russian spaceflight missions. Data from these missions suggest that space motion sickness occurs more frequently when astronauts and cosmonauts have increased movement capability, greater exterior vision and/or fewer internal visual orientation cues to rely on (Homick, Reschke, and Vanderploeg, 1984).

Approximately 50 percent of all astronauts and cosmonauts have suffered symptoms of SMS, ranging from mild discomfort to repeated vomiting. There are currently no ground-based methods for predicting susceptibility to motion sickness in space. Data from previous spaceflights indicate that some individuals who have had wide exposure to motion devices and accelera-tory forces on Earth or in aircraft, and who have never previously shown any tendency to develop motion sickness symptoms, were severely debilitated in space (Bungo, Bagian, Bowman, and Levitan, 1987). Conversely, some individuals who had a history of susceptibility to motion sickness on Earth were unaffected by symptoms in space. The earliest reported episode began within only 7 minutes of orbital insertion, and malaise has been reported to last from 1 to 5 days. Finding a solution to this biomedical problem has become a high-priority goal of NASA because of its potential impact on crew safety, comfort, and operational efficiency. Planned crew activities are disrupted when space motion sickness threatens crew safety, crew operations, and crew comfort. To date, SMS has not claimed the lives of any astronauts, but it has affected crew operations since the Apollo missions (Homick, and Miller, 1975; Homick, Reschke, and Vanderploeg, 1984).

Space motion sickness is a potential danger to susceptible astronauts. Astronauts suffering from symptoms are prohibited from performing extravehicular activities (EVAs). An EVA is a very complex and dangerous activity that requires 100 percent of the astronaut's mental and physical abilities. A degradation in health, such as headaches, malaise, or nausea, increases the danger of an already dangerous situation. Further, astronauts would probably asphyxiate from their own vomit if emesis occurred in their space suits. Even crewmembers suffering from mild symptoms could be in danger during an EVA because emesis can occur suddenly and without any warning (Homick, Reschke, and Vanderploeg, 1984). NASA flight planners postponed planned EVAs for both Apollo 9 and STS-5 Shuttle missions because crewmembers were suffering from space motion sickness (Homick, Reschke, and Vanderploeg, 1984). Flight

controllers can reschedule or cancel planned EVAs if astronauts become sick; however, there is no contingency plan to cover a mission scenario in which a contingency EVA for either the orbiter or payload must be performed and the crew is symptomatic.

Space motion sickness is an operationally relevant biomedical problem for crewed spaceflight. Davis and his colleagues (Davis, Vanderpleog, et al., 1988) reported that 71 percent of the crewmembers of the first 24 Space Shuttle missions reported symptoms of space sickness, which included 26 mild cases (30 percent), 20 moderate (24 percent), and 11 severe (13 percent). According to the NASA space motion sickness grading criteria (table 1), almost half of the 71 percent of space sickness cases impacted operations. Also, according to the symptom grading criteria, even a mild case of space motion sickness may produce retching or vomiting, and symptoms may last as long as 48 hours. It is clear that such a disorder could potentially jeopardize the success of future NASA and DOD missions.

Table 1. Symptom grading criteria used in space

None	No signs or symptoms reported with exception of mild transient headache or mild decreased appetite.
Mild	One to several symptoms of a mild nature; may be transient and brought on only as the result of head movements; no operational impact; may include a single episode of retching or vomiting; all symptoms resolved in 36 to 48 hours.
Moderate	Several symptoms of a relatively persistent nature which wax and wane; loss of appetite; general malaise, lethargy, and epigastric discomfort may be the most dominant symptoms; includes no more than two vomiting episodes; minimal operational impact; all symptoms resolved within 72 hours.
Severe	Several symptoms of a relatively persistent nature that may wax and wane; in addition to loss of appetite and stomach discomfort, malaise and/or lethargy are pronounced; strong desire not to move head; includes more than two episodes of vomiting; significant performance decrement may be apparent; symptoms may persist beyond 72 hours.

The operational problem posed by SMS is of significance during the initial phases of long duration missions, and particularly during the critical, short duration (2–7 days), high-activity missions planned for the Shuttle. Because the symptoms are present frequently enough in the first few days of flight there is a significant constraint on the level of routine activities that can be accomplished and, therefore, planned during that time. The impact of space sickness on crew efficiency has led to the development of several different approaches attempting to prevent and control this malady. Currently, there are two treatments for space motion sickness: pharmocological agents (e.g., scopolamine and promethazine) and behavioral techniques (e.g., autogenic feedback training and biofeedback).

Pharmacological and Behavioral Countermeasures

Current NASA policy recommends treatment of crewmembers with moderate to severe symptoms of SMS with intramuscular (IM) promethazine 25–50 mg (Davis, Jennings, Beck, and Bagian, 1993). To reduce the possibility of drowsiness, this medication is given in the presleep period on flight day 1. If symptoms develop earlier than presleep and require treatment, IM promethazine can be given and oral dextroamphetamine can be added, to counter the effects of sedation (Schroeder, Collins, and Elam, 1985).

Observations that intramuscular injections of promethazine are effective in attenuating motion sickness have been evaluated during both ground-based and space studies. Intramuscular promethazine was first used during a Shuttle flight in March 1989 and has been used on 14 other occasions since (Davis, Jennings, Beck, and Bajian, 1993). Intramuscular promethazine and its efficacy in the treatment of space motion sickness were evaluated using standardized questions administered during postflight debriefings. Results showed that 25 percent of crewmembers treated with IM promethazine were "sick" on flight day 2, compared to 50 percent of crewmembers who did not receive promethazine (Davis, Jennings, Beck, and Bagian, 1993). In addition, symptom relief occurred within 1–2 hours in 90 percent of individuals treated with promethazine.

The efficacy of promethazine was also evaluated on subjects during zero gravity maneuvers in a KC-135 aircraft. Subjects were given a 50 mg dose of promethazine only if they experienced severe nausea or vomiting and requested an injection. Within 10 minutes of IM injection, 78 percent of individuals experienced symptom relief, whereas 25 mg of promethazine was not effective (Graybiel and Lackner, 1987). Intramuscular injections (25 mg) of promethazine increased motion sickness

tolerance by 78 percent during cross-coupled angular accelerations; injections were given 30 minutes prior to testing (Wood, Stewart, Wood, and Mims, 1992). Intramuscular injections of promethazine given to eight subjects 2 hours prior to testing in the slow rotation room also resulted in beneficial effects (Graybiel, Wood, et al., 1975).

The Physician's Desk Reference cautions, under Information for Patients, that ". . . promethazine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a vehicle or operating machinery" (Physician's Desk Reference, 1993). Ground-based studies have shown that significant decrements in performance scores, psychomotor function, information processing, and alertness may occur with both oral and IM injections of promethazine. For an oral dose of promethazine (12.5 mg, 25 mg), maximal effects may be seen on information processing and psychomotor performance (tested at 2 hour intervals), 3–4 hours after ingestion, with a return to baseline after 8–9 hours (Parrot and Wesnes, 1987). Impaired dynamic tracking performance and reduced ability to maintain visual fixation were observed following oral ingestion of 25 mg of promethazine (Wood, Manno, et al., 1985). Decrements in a computerized pursuit motor task following both oral and IM 25 mg promethazine were significant. Measurements were made 2, 3, and 4.5 hours following administration of medications (Wood, Manno, et al., 1984).

Several studies demonstrate maximal impairments 5 and 6 hours after drug administration, while performance remained below baseline 1.5 and 7 hours postdrug oral (10 mg) (Clarke and Nicholson, 1978). Large, Wayte, and Turner (1971) noted no decrements in hand–eye coordination 1.5 hours postdrug (25 mg), with maximal impairment at 3 hours postdrug, following oral administration. A similar time course for 50 mg oral dose also has been found for hand–eye coordination (Molson, Mackay, Smart, and Turner, 1966). Flight simulator performance of subjects on 25 mg IM promethazine decreased as compared to performance of subjects on placebo (Taylor, Dellinger, Hyman, and Richardson, 1984). Decrements in tracking performance were found 1, 2, and 4 hours following administration of IM 25 and 50 mg promethazine (Schroeder, Collins, and Elam, 1985). Tracking decrements may be attributed to reduced optokinetic nystagmus which makes less accurate the following ability of the eye (Collins, Schroeder, and Elam, 1982). Impairment of information processing, memory, reaction time, and spatial processing following IM injection have not been assessed.

Motion sickness research has primarily focused on the study of vestibular physiology, perceptual phenomena, or pharmacological interventions in man and animals (Reason and Brand, 1975). In contrast, Cowings and her colleagues at Ames Research Center are using psychophysiological methods for studying motion sickness and are developing a treatment for training people to control their own motion sickness symptoms (Blizzard, Cowings, and Miller, 1975; Cowings, 1990; Cowings, Billingham, and Toscano, 1977; Cowings and Toscano, 1977, 1982; Cowings, Toscano, et al., 1988; Cowings, Toscano, Sekiguchi, and Ishii, 1993; Toscano and Cowings, 1982). The method of treatment is Autogenic-Feedback Training (AFT), a combination of biofeedback and Autogenic Therapy (Schultz and Luthe, 1969), which involves training physiological self-regulation as an alternative to pharmacological management. The rationale for using AFT to treat motion sickness was based on the observation that there were profound autonomic nervous system (ANS) changes associated with this disorder (Cowings, Suter, et al., 1986), and, although these responses are highly idiosyncratic, they are repeatable over time (Cowings, Naifeh, and Toscano, 1990; Stout, Toscano, and Cowings, 1993).

Because certain ANS responses were correlated with, and indeed predictors of (i.e., consistently preceded), reports of motion sickness distress, it was hypothesized that training subjects to control these responses might prevent or reduce symptoms. The observed individual differences in responding suggested that, to be effective, such training would have to be directed at the different ANS responses for different people. In other words, training would have to be tailored for each individual.

AFT is a combination of several physiological and perceptual training techniques, principal among these are Autogenic Therapy (Schultz and Luthe, 1969) and biofeedback (Miller, 1969). These two techniques have been used widely to facilitate self-regulation of involuntary autonomic responses and minimize the debilitating effects of various stressors. Biofeedback consists of providing the subject with augmented sensory information about the ongoing activity levels of some physiological response (e.g., heart rate on a digital panel meter), and rewarding him whenever such levels fluctuate in a direction selected by the trainer (whenever heart rate fluctuates above baseline). The result is an enhanced ability by the subject to maintain the changed level for increasing periods of time. Only repetition and practice are required before physiological control is achieved.

Autogenic Therapy is an alternative self-regulatory technique that has been shown to have wide effects on autonomic reactivity (Schultz and Luthe, 1969). This

training method involves the use of self-suggestion exercises that are designed to induce bodily sensations (e.g., warmth in the hands) that are highly correlated with specific physiological responses such as peripheral vasodilatation (Harano, Ogawa, and Naruse, 1973). When these exercises are practiced in series, the result is a relaxed (i.e., parasympathetic-like) physiological profile within the subject which prevents the emergence of behavioral and physiological reactions to stress. Cowings (Blizzard, Cowings, and Miller, 1975; Cowings and Toscano, 1977) found that the combined techniques, AFT, produces larger magnitude physiological changes that are more reliable over time.

Ground Studies of Motion Sickness

Money (1970), in his 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. The relative importance of autonomic nervous system (ANS) responses in understanding and treating motion sickness has been a matter of some controversy (Graybiel, and Lackner, 1980). In a recent paper, Cowings and colleagues (Cowings, Suter, et al., 1986) examined the data of 127 people, all given the same motion sickness test in order to describe the general trend of ANS responses in all subjects. Individual differences in initial motion sickness susceptibility were also examined as a possible source of variability in ANS responding reported by others (Parker, 1974; Parker and Wilsoncroft, 1978). Results clearly showed sympathetic-like activation of four ANS responses during motion sickness stimulation. These included significant changes in heart rate acceleration, peripheral vasoconstriction, and increases in skin conductance. Physiological response levels changed rapidly and dramatically at the onset of stimulation and when the test concluded. ANS response differences were also found among motion sickness susceptibility groups, with highly susceptible subjects producing, in general, larger magnitude changes than the moderate or low susceptible subjects.

In another study, comparisons were made of two separate motion sickness tests on each of 58 subjects (Cowings, Naifeh, and Toscano, 1990). Again, the same four physiological responses (heart rate, finger pulse volume, respiration rate, and skin resistance) were measured during both motion tests. The objective of this study was to examine individual differences in physiological responding (i.e., response patterns) to motion stimuli and determine how these data were related to self-reports of motion sickness malaise experienced.

The results revealed eleven separate patterns of physiological responding in which all or some combination of the four physiological measures clearly reflected motion sickness malaise levels of each of the 58 subjects. Individual response patterns produced on the first tests were not significantly different than those of the second test. Analyses showed that of the 58 subjects, 27 showed the same response patterns on both tests for all four physiological measures, 14 were stable for three variables, 6 were stable for two, and 11 were stable responders for at least one variable.

Cowings (1990) reviewed a number of studies conducted by her research group that examined AFT as a treatment for motion sickness. In one study, differences in motion sickness tolerance were compared in subjects given AFT, an alternative cognitive task (computer Blackjack), or no treatment (Toscano and Cowings, 1982). Two hours of AFT were administered to treatment group subjects before the third, fourth, and fifth rotating chair motion sickness tests (6 hours total). Results showed that subjects who received AFT had significantly greater motion sickness tolerance (rode longer) than subjects performing an alternative cognitive task or those performing no task. Although the cognitive task group had slightly greater mean tolerance than the no-task control group, the difference was not statistically significant.

In another experiment, the objective was to determine if an individual's initial susceptibility to motion sickness was related to the ability to learn to control one's own symptoms (Cowings and Toscano, 1982). Subjects were assigned to groups based on their initial tolerance to motion sickness in a rotating chair. Two AFT treatment groups (highly and moderately susceptible to motion sickness) were compared to two control groups who were matched to the AFT groups for initial susceptibility, but were given no treatment. Results showed that both AFT treatment groups significantly improved their motion sickness tolerance while neither control group improved significantly. During the last two tests, after 6 hours of AFT, the highly and moderately susceptible treatment groups were no longer significantly different in their motion sickness tolerance, while the high and moderate control groups remained significantly different across all tests.

The results of other studies (Cowings, 1990; Cowings, Toscano, Sekiguchi, and Ishii, 1993) showed that (1) the effects of AFT for symptom control are equal for both men and women, (2) symptom control with AFT can be retained for up to 2 years after training, and (3) the primary component of the treatment effect in each of these studies was attributed to learned control of physiological responses. Subjects who increased their

tolerance to motion sickness consistently showed a significant reduction in the magnitude of changes in their autonomic responses after training.

Experiments in the literature (Reason and Brand, 1975) and clinical experience show that habituation to a specific nauseogenic situation does not transfer to new situations. Repeated exposure apparently affects primarily the sensory side (or "input" side) of the response system. Autogenic-Feedback Training is aimed at controlling the "output" side, i.e., the various symptoms or autonomic manifestations of motion sickness. To the extent that such control can be learned, it is much more likely to transfer to different situations that induce nausea, including the unique condition of spaceflight.

An extensive examination of transfer of training was made in another study which involved several different types of stimuli that induce motion sickness (Cowings and Toscano, 1993). Twenty-four men and women were assigned to two equal groups, matched for gender and initial susceptibility to motion sickness in a rotating chair. A second type of motion sickness test combined the rotating chair with optokinetic stimulation in a rotating drum that surrounded the chair. The subject's perception of the combined stimulus was rotation in the opposite direction of actual chair rotation. A final motion sickness test was given to subjects using a vertical simulator that produced slow up–down motion.

The two groups of subjects, an AFT treatment group and a no-treatment control group were given the three types of motion sickness inducing tests at the start of the study. Treatment subjects were then given 6 hours of AFT over 5 days, while the control subject received no training. Both groups of subjects were given their second exposure to the three motion sickness tests at the end of the experiment. Results showed that subjects given AFT significantly improved their tolerance to the different types of motion sickness tests, whereas the control subjects did not.

The U.S. Air Force had adopted a similar form of AFT to treat crewmembers for whom other methods had proved unsuccessful in combating persistent air sickness in high-performance military planes (Levy, Jones, and Carlson, 1981; Jones, Levy, et al., 1985). They have found that such training transfers from the rotating chair on the ground to the variety of maneuvers in military flight well enough to return air crew that otherwise would have been permanently grounded, to active flying duty.

Research on Heart Rate Variability

Studies of heart rate during motion sickness have focused exclusively on changes in mean heart rate or changes in heart rate variance (Money, 1970; Graybiel and Lackner, 1980; Cowings, Suter, et al., 1986; Igarashi, Himi, et al., 1987). For example, Igarishi, Himi, et al. (1987) reported that an increase in r–r interval variance correlated with susceptibility to sensory sickness in adult squirrel monkeys. These reports implicate the autonomic nervous system in the etiology of motion sickness, but have limited practical application to spaceflight for two reasons. First, the use of terrestrial motion sickness as a model for space motion sickness is of questionable validity. Second, conventional analysis of heart rate changes only on the basis of changes in mean or changes in variance gives only a limited representation of the complete dynamics. This type of analysis will not, for example, detect the presence of oscillations and is of limited use in characterizing the sudden changes in heart rate dynamics that may occur during spaceflight (Goldberger, Thornton, et al., 1987). A more complete understanding of cardiovascular dynamics during spaceflight and their relation to space sickness requires analysis of beat-to-beat heart rate fluctuations using time series and spectral analysis techniques (Sayers, 1973; Kitney and Rompelman, 1980; Akselrod, Gordon, et al., 1981; Kobayashi and Musha, 1982).

Several reports in the medical literature demonstrate the usefulness of heart rate spectral analysis (Goldberger, Goldwater, and Bhargava, 1986; Pangani, Lombardi, et al., 1986; Jarisch, Ferguson, et al., 1987). Whereas sinus rhythm in healthy individuals is characterized by considerable beat-to-beat variability and a broad bandwidth spectrum, a variety of disorders are associated with increased heart rate periodicity, and sometimes distinct oscillations can be seen (Goldberger, Findley, Blackburn, and Mandell, 1984). Examples of pathologic heart rate oscillations have been described in fetal distress (Karinemi and Ammala, 1981) and in congestive heart failure (Goldberger, Findley, Blackburn, and Mandell, 1984). Goldberger (Goldberger and Rigney, 1987) described low-frequency oscillations in sinus rhythm prior to the onset of potentially fatal ventricular tachyarrhythmias (sudden death syndrome). The mechanism of the low-frequency oscillations (usually 0.03 Hz) in these conditions is not known, but probably is related to an instability in neuroautonomic control.

Another feature of note is that heart rate oscillations are observed to start and stop abruptly, a feature that indicates a nonlinear type of system (Goldberger, West, and Bhargava, 1985). In a preliminary study, Goldberger (Goldberger, Thornton, et al., 1987) reported on low-frequency (<0.03 Hz) heart rate oscillations in two astronauts. Prominent oscillations were observed for both subjects during periods of severe space motion sickness and were characterized by marked sensitivity to rotational

movement, malaise, and anorexia. The frequency spectrum of one subject, after recovery from sickness was reported, did not contain the usual low-frequency oscillations. However, in another subject, prominent oscillations were noted intermittently in the apparent absence of overt symptoms.

Respiratory sinus arrhythmia (RSA) is one of many oscillations which are manifested in the heart rate pattern. It is, however, one of the few physiological oscillations which may be directly linked to a specific physiological mechanism. Research by Katona and Jih (1975) suggested that measurement of RSA amplitude could be used as a sensitive index of parasympathetic control of the heart (i.e., cardiac vagal tone). Other experiments (McCabe, Younge, Porges, and Ackles, 1984; Dellinger and Porges, 1984), in which vagal activity was manipulated with pharmacological treatments (atropine and propranol) and electrical stimulation, have also shown that RSA magnitude is a sensitive index of vagal tone.

In an experiment by Cowings, Suter, et al. (1986), it was reported that among several autonomic measures heart rate was the best predictor of symptoms of motion sickness. These authors concluded that motion sickness was characterized by sympathetic activation. However, this conclusion was somewhat premature, because not all measures were exclusively indices of the sympathetic nervous system. An increase of heart rate during motion sickness could be completely or partially explained by vagal withdrawal. In fact, in a later study (Uijtdehaage, Stern, and Koch, 1992) on vection-induced motion sickness, RSA, exclusively reflecting vagal chronotropic control, was found to be predictive of symptom levels. That is, high levels of vagal tone were inversely related to motion sickness scores. Furthermore, increases in cardiac vagal tone were positively associated with normal gastric activity (3 cpm) and negatively associated with dysrhythmic activity of the stomach (tachyarrhythmia). Tachyarrhythmia is believed to be a physiological marker of nausea in general and motion sickness in particular (Grashuis, van der Schee, and Geldhof, 1985; Koch, Stern, Vasey, and Dwyer, 1990). Support for this finding of a relationship between RSA and motion sickness was shown in the work by Vybiral, Bryg, et al. (1990). These researchers measured RSA and heart rate before and after administration of scopolamine, a potent (anticholinergic) anti-motion sickness drug. A strong increase in vagal tone and a heart rate decrease were observed compared with predrug levels. The paradoxical parasympathomimetic action of scopolamine was attributed to central stimulation of vagal motor centers, which overruled the weaker parasympatholytic action in the periphery. The combined results of this study and data reported by Uijtdehaage (Uijtdehaage , Stern, and Koch, 1992) suggest that a general state of increased peripheral parasympathetic activity can alleviate motion sickness symptoms by suppressing, in part, its gastric dysrhythmic underpinnings.

In light of the above results, the studies by Cowings (Cowings, Toscano, Sekiguchi, and Ishii, 1993; Cowings and Toscano, 1993) demonstrate that the primary physiological effect of AFT in alleviating motion sickness was a reduction in the magnitude of autonomic response changes observed in subjects after training. These authors also speculate that a general state of increased parasympathetic tone, seen with AFT subjects, can alleviate motion sickness symptoms. Further studies are needed that address the physiological mechanism by which AFT alters motion sickness susceptibility.

The Current Study

This study investigated the use of AFT for alleviating symptoms of SMS in astronauts, as an alternative treatment to pharmacological management. The hypotheses of the experiment were (1) AFT administered before flight will reduce or eliminate the symptoms of space motion sickness; (2) data recorded in space will objectively reveal effects of early exposure to microgravity (when compared to ground-based data) on human physiological responses and can be used to evaluate the course of adaptation to that environment; and (3) individual susceptibility to space motion sickness can be predicted on the basis of each crewmember's demonstrated ability to learn control of his or her own physiological responses during preflight AFT.

The specific objectives of the study were, first, to determine the effects of preflight AFT on susceptibility to motion sickness and symptom levels induced by stressful Coriolis stimulation in a rotating chair. All subjects were given a baseline motion sickness test in a rotating chair to measure their initial tolerance (number of rotations achieved), and symptom levels to this stimulus. Treatment subjects were retested in the rotating chair at one week intervals after 2, 4, and 6 hours of AFT and changes in motion sickness tolerance and symptom levels were compared to their pretreatment scores. Control subjects were given only one exposure to the rotating chair motion sickness test. Cowings, Billingham, and Toscano (1977) showed that repeated motion sickness testing at 1 week intervals in untreated subjects does not influence their susceptibility. Second, to investigate changes in heart rate variability, vagal tone, and coherence between heart rate and respiration during motion sickness in a rotating chair and a vertical motion stimulus. Although preliminary data from space suggest that low-frequency heart rate oscillations may provide a sensitive diagnostic marker of

motion sickness, it is not known whether terrestrial motion sickness is associated with these oscillations and if so whether they occur at the same frequencies characteristic of SMS. The effects of AFT on these measurements during motion sickness testing were also examined. Third, to determine if AFT is effective for preventing or alleviating motion sickness symptoms during actual spaceflight. To evaluate AFT treatment effects, self-reported symptoms of motion sickness were compared for treatment and control subjects on mission days 1, 2, 3, and 4. And last, to examine changes in physiological responses to spaceflight. These psychophysiological data were then compared to ground-based simulations of specific mission days. During flight, continuous physiological recordings were collected from subjects during wakeful periods on mission days 1, 2, 3, and 4.

Methods

Overview

This experiment represents part of a larger study that was approved by NASA as a life sciences flight experiment to be flown on several Space Shuttle missions and required obtaining data on a total of sixteen subjects (eight treatment and eight controls). This experiment was first flown on the Space Shuttle in 1985, and during that mission data were collected on four crewmembers (two treatment and two controls). The experiment was reflown on another Shuttle mission in 1992, in which two crewmembers served as subjects (one treatment and one control). The current study included the six flight subjects (three treatment and three controls) from two previous Shuttle missions. The assignment of treatment and control subjects was not random. Because mission training schedules were different for each astronaut, only those who were available to participate in 3 weeks of preflight AFT served as treatment subjects.

Subjects

Thirteen people (four women and nine men) participated in the preflight activities for this experiment. Ages ranged from 32 to 59 years. Six of these subjects subsequently flew in space, while six other subjects served as their alternates and did not fly. One additional subject was an active duty military pilot who received AFT in parallel with the astronauts. This pilot was attempting to overcome air sickness in the F-18 tactical fighter aircraft. There were three treatment and three control (four men and two women) flight subjects. All subjects were medically and otherwise qualified to serve as crewmembers on scheduled Space Shuttle missions. Informed voluntary consent was obtained and all procedures were approved by NASA's human research review boards at Ames Research Center, Johnson Spaceflight Center, and the University of California at San Francisco.

Apparatus

Motion sickness stimuli– A Stille-Werner motor powered rotating chair was used to induce the initial symptoms of motion sickness. The chair was located within a sound attenuated room which was temperature controlled (70 ± 2°F). Subjects were seated in the rotating chair and the center of rotation was through their own vertical axis (spine). Padded headrests were mounted on the sides, front, and back of the chair, which allowed the blind-folded subject to execute head movements in randomized directions at 45 degree angles from the upright position. Preamplifiers for physiological signals were mounted on the rear of the chair and a belt-worn physiological monitoring system (see Physiological Measures) was secured around the waist of the subject. The amplified signals were sent through slip rings in the base of the chair to laboratory recorders.

Subjects were given a second motion sickness test using the Vertical Acceleration and Roll Device (VARD) at Ames Research Center. The VARD is a light-proof enclosed cab which can achieve a maximum vertical displacement of ±6 feet (±1.829 m), without roll or pitch. The frequency and gravity load are programmable. Preamplifiers for physiological measures were mounted in the cab. These signals were sent through a cable in the rear wall of the cab to laboratory recorders. All physiological data were recorded on two 8 channel strip chart recorders and a 14 track FM analog tape recorder, and were processed in real time and stored on a Masscomp 6600 computer.

Symptom diagnostic scale– During each 5 minute interval throughout the motion sickness tests, subjects were asked to report their symptoms to the experimenter. The symptoms were graded using a standardized diagnostic scoring procedure (Graybiel, Wood, Miller, and Cramer, 1968). Table 2 is an outline of the diagnostic scale used. Frank vomiting (VMT) was indicated as either present (I) or absent (no entry). The array of symptoms included increased temperature (TMP), dizziness (DIZ), headache (HAC), drowsiness (DRZ), sweating (SWT), pallor (PAL), and salivation (SAL). The presence or absence and/or strength of most symptoms were assessed subjectively by the subject as mild "I," moderate "II," or severe "III." Nausea was evaluated as epigastric awareness (EA) epigastric discomfort (ED), and nausea (NSA).

Table 2. Motion sickness diagnostic scale

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

For example, a subject may report headache (1 point), moderate drowsiness (4 points), and severe sweating (8 points), summing to 13 points.

Motion sickness scores between 1 and 4 points represented mild malaise, scores between 5 and 7 represented moderate malaise, scores of 8 or higher represent severe malaise with 16 points scored for vomiting (i.e., frank sickness).

Physiological measures– The Autogenic-Feedback System-2 (AFS-2) is a portable belt-worn physiological monitoring system (fig. 1) developed by NASA to support the flight experiment flown in 1992. An earlier version of this system, the AFS-1, was used to support the experiment flown in 1985 and redesigned to improve signal quality and crew mobility. Physiological measures recorded with both systems were identical. The AFS-2 includes a garment, transducers, signal conditioning amplifiers, a digital wrist-worn feedback display, and a cassette tape recorder. The wrist display provided treatment subjects with continuous numeric feedback of heart rate, respiration rate, blood volume pulse, skin conductance, and skin temperature. Hardware malfunction indicators and time were also provided to subjects via display. The entire instrument is powered by a self-contained battery pack.

The physiological measures recorded with the AFS-2 were:

Electrocardiography: Three pregelled Ag/AgCl disposable electrodes were placed on the chest just below the left and right clavicles (distally), and on the left midclavicular line over the fourth intercostal space.

Respiration: A piezoelectric transducer was attached to the garment with snaps over the chest to measure the respiratory waveforms.

Finger pulse volume: An infrared photo transistor and detector was mounted in a ring positioned on the volar surface of the small finger on the left hand for measuring relative changes in peripheral vasomotor activity.

Skin temperature: A solid state temperature transducer was also mounted in the same ring for measuring skin surface temperature at the same location.

Skin conductance level: Two pregelled Ag/AgCl disposable electrodes were mounted on the volar surface of the left wrist and spaced 1 inch apart to measure the conductivity of the skin produced by moisture from the sweat glands.

Additionally, the AFS-2 recorded head motions in the X, Y, and Z planes from a triaxial accelerometer mounted on a headband.

Other physiological measures recorded with standard laboratory equipment included:

Electromyography: Three pregelled Ag/AgCl disposable electrodes were attached to the forearm extensor muscles of the arms and the gastrocnemius muscles of the legs to measure the muscle activity at these locations.

Skin conductance: An electrolyte paste (skin saline concentration) was applied to two Ag/Ag Cl electrodes and these were attached to the tips of the right index and middle fingers with velcro.

Finger pulse volume: A photoplethysmograph transducer was attached with a finger clip to the little finger of the right hand to measure relative changes in peripheral vasomotor activity.

Skin temperature: A thermistor was taped to the small finger on the right hand to measure the skin temperature at this site.

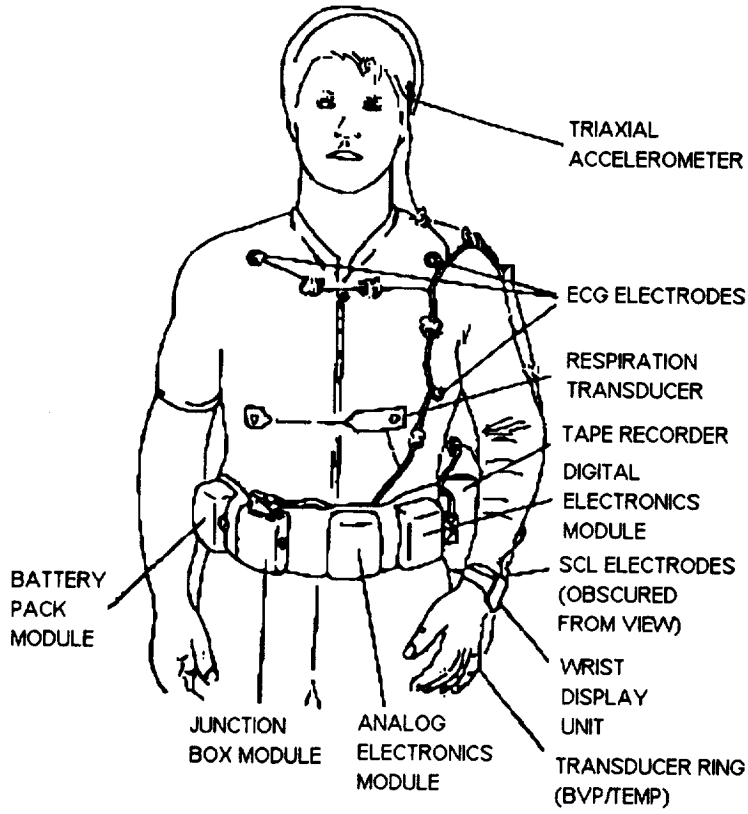

Figure 1. The Autogenic-Feedback System-2 (AFS-2).

Procedures

Preflight– All subjects were initially given two baseline motion sickness tests on separate days, one in the rotating chair and the other in the vertical motion simulator. Each subject also participated in two resting baselines given on consecutive days, and one 12 hour mission simulation. Additionally, treatment subjects were given twelve training sessions and three rotating chair motion sickness tests over 3 weeks.

On each day of motion sickness testing subjects were given a brief orientation on the test procedures. They were then seated in the rotating chair or vertical motion simulator, and their physiological sensors were attached. Following a 10 minute baseline condition (no rotation), the chair was rotated to 6 rpm (0.628 rad/s) and maintained at this speed for 5 minutes. During the 5 minute period of rotation, subjects were instructed by a tape recorded voice to make head movements (front, back, left, and right). The order of the head movements was randomized and the duration of each movement was 1 second. At the end of each 5 minute period the subject held his head in the upright position for 30 seconds (while rotation continued) and reported his symptoms to the experimenter. The rotation continued at increasingly higher speeds, incrementing by 2 rpm (0.209 rad/s) every 5 minutes, until the subject reached severe motion sickness or was unwilling to continue or had reached a maximum velocity of 30 rpm (3.142 rad/s). When the rotating chair was finally stopped, another 10 minute baseline period was taken.

During vertical motion tests, the frequency and gravity load were held constant at 0.33 Hz, 0.35 g. Again, subjects were instructed by the same tape recorded voice to make head movements and report their symptoms every 5 minutes throughout the test. Vertical motion tests were terminated after 75 minutes or when the subjects reached severe malaise or they were unwilling to continue. Pre- and posttest baseline periods (10 minutes) without motion were also included in these tests.

Resting baselines of physiological responses were recorded from subjects while they were seated in a chair in a sound attenuated room and listened to tape recorded music for 30 minutes. This procedure was repeated the next day.

Flight subjects also participated in a mission simulation. Physiological responses were recorded continuously for

12 hours with the AFS-2 while subjects performed procedures related to this experiment and other scheduled flight experiments.

Treatment subjects were to be given 12 AFT sessions that were distributed over 3 weeks. Training sessions (30 minutes each) were conducted in a stationary chair within a sound-attenuated room over four consecutive days each week. A rotating chair motion sickness test was given on day 5 of each week. The purpose of these motion sickness tests was to examine AFT effects on changes in motion sickness tolerance, symptom levels, and physiology.

Training sessions were divided into ten 3 minute trials in which subjects were instructed to increase and decrease, on alternate trials, their autonomic response levels. In an earlier study (Cowings and Toscano, 1982) it was shown that bidirectional training was more effective for symptom control than training in only one direction. Physiological feedback was displayed to subjects as a raw analog waveform (e.g., respiration) on a CRT, a numeric display, and/or an auditory tone. Autogenic self-suggestion exercises were used by subjects to help them produce response changes in the desired direction. Training sessions were preceded and followed by 6 minutes of baseline.

Flight– Continuous physiological recordings were collected with the AFS-2 during waking hours (approximately 12 hours) on the first four mission days from both treatment and control subjects.

An 11 item diagnostic log book (fig. 2) was used by the subjects to report the type and severity of their symptoms (Graybiel, Wood, Miller, and Cramer, 1968; Cowings, Suter, et al., 1986). This diagnostic scale was identical to that used in preflight motion sickness testing, except that subjects were trained to self-report their own symptoms in space.

Timelined symptom reporting was performed by subjects immediately after awakening in the morning and before retiring at night. If space motion sickness occurred at any other time during the day, those symptoms were also reported. Subjects also made written comments describing their symptoms (e.g., if different from symptoms on Earth) and evaluated the effects of AFT on symptom control.

SUBJECT'S ID#_____ PRE AFT____ POST AFT____

TIME (GMT)_____

TIME	SYMP			SYMPTOMS OBSERVED								
LINE	CONT	VMT	TMP	DIZ	HAC	DRZ	SWT	PAL	SAL	NSA	ED	EA
		I	I,II	I,II	I	I,II, III	I,II, III	I,II, III	I,II, III	I,II, III	I	I

COMMENTS: _____

Figure 2. Illustration of the diagnostic log book.

Treatment subjects also performed daily 15 minute AFT sessions in which they practiced control of their physiological responses using feedback from their wrist-worn display. If space sickness symptoms occurred, the subject attempted to alleviate the symptoms with AFT. These symptom-contingent sessions were 30 minutes in duration.

Postflight– On the day of landing, each subject attended a private 15 minute briefing with the experimenter, and specific details pertaining to this experiment were noted. Flight hardware, data tapes, and diagnostic log books were removed from the Shuttle and returned to the experimenter for data processing. Two weeks later each subject attended another private 2 hour meeting with the experimenter to discuss his or her data and to help explain unusual data anomalies possibly due to changes or delays in conducting flight procedures.

Data analysis plan– Analyses of physiological data included the heart rate and respiration measures obtained during preflight motion sickness tests, during a mission simulation, and during spaceflight. Other data analyses included the motion sickness symptom scores collected during the preflight motion sickness tests and self-reports of symptoms during flight. Preflight motion sickness tolerance, measured as the number of rotations, was also analyzed to examine AFT effects on this variable.

Although recordings of other physiological measures were obtained during this experiment, only analyses of heart rate and respiration data are included here. The other physiological variables and data obtained during preflight training (AFT) will be discussed in a future paper.

Analog electrocardiographic and respiration data were digitized at 100 samples/s via a 12 bit A/D converter using the Lab Workbench data acquisition program on a Concurrent 6600 computer. The stored ECG and respiration records for each subject were displayed on the monitor in successive 8 minute screens and subjected to a program for artifact removal and interpolation of missing beats. Inter-beat intervals (r-to-r peaks) were computed from the ECG records of all subjects using a custom peak detection program. Heart period data were then converted to a weighted heart rate (4 samples/s) to establish equal time intervals, and the respiration data were subjected to a smoothed average (4 samples/s). Before analysis the entire heart rate and respiration series for each subject was high-pass filtered (0.005 Hz) and the mean was centered to zero.

The heart rate and respiration data from preflight motion sickness tests were then divided into contiguous 5 minute time blocks (epochs) that included (1) Two prerotation baselines. (2) Five minutes at each rotational speed (e.g., 6, 8, 10 rpm, etc.). Time of rotation was based on individual tolerance to the stimulus; therefore, the number of epochs of rotation for each subject varied. (3) Two postrotation baselines. Spaceflight data, consisting of approximately 12 hours on each day (mission days 1, 2, 3, and 4), were divided into contiguous 8.5 minute time epochs.

A spectral analysis program (BMDP1T) on the PC was used to analyze the heart rate and respiration data. Applying a cosine window, the first and last 5 percent of data in each epoch were tapered to zero to reduce artifactual end effects, and then Fourier transformed. Spectral density estimates were formed from an average of adjacent periodograms within a bandwidth of 0.015 Hz. To quantify the power in different frequency bands, each heart rate spectrum was divided into three bands: a low-frequency band that encompasses the oscillations reported by Goldberger (Goldberger, Thornton, et al., 1987) in a preliminary study of space motion sickness (<0.05 Hz), a mid-frequency band (>0.05, <0.1 Hz), and a high-frequency band (>0.1, <0.4 Hz) that encompasses the usual respiratory frequency. The power in each band was calculated using the root-mean-square (rms) measurement, and the rms value for the entire spectrum was also computed. The rms spectral power in each band was expressed in two ways: as an absolute value and also as a percent of total power in the entire spectrum.

Two auxiliary analyses were performed (1) the weighted coherence (Porges, Bohrer, et al., 1980) between heart rate and respiration, and (2) estimates of cardiac vagal tone (Porges, 1985) from the amplitude of respiratory sinus arrythmia (RSA) as seen in heart rate data. Cross spectral analyses (BMDP1T) were used to generate a coherence function, a measure of covariation between heart rate and respiration. Then a weighted coherence (Cw) was derived by weighting the coherence function across a band of frequencies (>0.1, <0.4 Hz) by the spectral densities. Cw may provide a quantitative estimate of stretch receptor influence on heart rate activity (Porges, Bohrer, et al., 1980). Estimates of cardiac vagal tone were derived by spectral analysis of heart rate epochs. First, a third order moving polynomial window (10 seconds) was applied to the heart rate series to remove aperiodic trends, then the series was bandpass filtered (>0.1, <0.4 Hz) to allow nominal respiratory frequencies to pass, and finally the new heart rate series was subjected to spectral analysis to obtain estimates of vagal tone.

Group comparisons of the physiological data were performed on selected time epochs of the preflight motion sickness tests, a mission simulation, and flight data using multivariate analysis of variance (MANOVA) with

repeated measures (BMDP4V). The Greenhouse-Giesser method was used to reduce the degrees of freedom for the repeated measures analyses, and a type I error rate of 0.05 was used. For example, a comparison was made of data obtained during a mission simulation to data collected in space on the same mission day. Because environmental conditions were the same (e.g., work load, crew activity schedule), any differences observed in these measures could be attributed to the effects of microgravity. Other analyses were performed to determine possible differences in physiological responses over mission days as crewmembers adapted to microgravity.

The nonparametric Friedman analysis of variance (ANOVA) for related samples was used to compare symptom scores (ordinal data) across motion sickness tests for treatment group subjects. It was expected that their scores on tests 2, 3, and 4 (2 hours of training preceded each test) would be lower than symptom scores on test 1 (no training). Preflight motion sickness tolerance scores of treatment subjects, measured as the number of rotations achieved, were compared across rotating chair tests using a repeated measures ANOVA. A Kruskal-Wallis ANOVA for independent samples was used to compare the symptom scores of treatment and control subjects obtained during flight on four mission days.

Results

Preflight Motion Sickness Tolerance Results

To examine AFT effects on changes in motion sickness tolerance, the number of accumulative rotations for each rotating chair test was computed for each subject and used as the dependent measure for the analyses described below. Figure 3 shows the distribution of motion sickness tolerance scores for each treatment subject before AFT (test 1) and after AFT (test 4). Test 1 tolerance scores for the three control group subjects (I.D.s 8, 12, and 13) are also plotted on this graph. Asterisks are shown above the bars for those subjects who flew in space. The center bar graph represents treatment group (N = 10) means and standard errors for tolerance scores on tests 1–4. Five of these subjects repeated the training a year later because of a delay in the mission. In the lower bar graph the means and standard errors for this group (N = 5) are plotted for tests 1–4 (first year) and tests 5–7 (second year).

The first analysis examined changes in motion sickness tolerance of 10 treatment subjects during four rotating chair motion sickness tests (test 1, no treatment; test 2, after 2 hours of AFT; test 3, after 4 hours of AFT; and test 4, after 6 hours of AFT). The ANOVA revealed a significant effect for tests, $F(1.48, 13.29) = 8.37$, $p < 0.007$, indicating that tolerance increased over tests. Separate contrasts showed that motion sickness tolerance significantly increased after 2 and 4 hours of AFT (test 1 versus test 2, $F(1,9) = 7.79$, $p < 0.02$, and test 2 versus test 3, $F(1,9) = 8.72$, $p < 0.01$), but did not significantly increase after 6 hours of training (test 3 versus test 4). A second analysis was performed using only the data of subjects (N = 5) for whom the training was repeated. Tolerance scores from test 1 (no treatment), test 4 (after 6 hours of AFT (first year)), and test 7 (after 6 hours of AFT (second year)) were analyzed. Again, there was a significant effect for tests, $F(1.27, 5.08) = 7.04$, $p < 0.04$, suggesting that AFT increases tolerance to motion sickness.

Specific contrasts show that during the first year after 6 hours of AFT, motion sickness tolerance did significantly increase (test 1 versus test 4, $F(1, 4) = 8.12$, $p < 0.04$), but during the second year after 6 hours of AFT tolerance did not significantly increase (test 1 versus test 7, $F(1, 4) = 6.66$, $p < 0.06$).

Preflight Symptom Score Results

Appendix A includes the graphs of each individual's diagnostic scores plotted over 5 minute epochs of the rotating chair motion sickness tests (figs. A-1–A-13). The number of symptom scores per test varied as individual tolerance to the motion sickness stimulus changed. Symptom scores were derived by totaling the point values for each symptom reported during each test epoch. The symptom scores of each subject were averaged over the epochs of each test and were analyzed to investigate AFT effects on changes in motion sickness malaise. Table 3 lists the mean symptom scores of each subject on four (N = 10) and seven (N = 5) rotating chair tests. The first analysis compared the symptom scores of 10 treatment subjects across the four motion sickness tests. The Friedman test statistic was significant, $X(3) = 12.36$, $p < 0.006$, and specific comparisons revealed that test 1 versus test 3 and test 1 versus test 4 were significant, $Z(3) = 2.94$, $p < 0.05$, and $Z(3) = 3.12$, $p < 0.05$, respectively. Motion sickness discomfort was significantly decreased following 4 and 6 hours of training for this group. Two additional analyses were conducted to compare the symptom scores of five treatment subjects across four motion sickness tests in their first and second year of training. Table 4 shows the mean symptom scores of each subject on seven motion sickness tests. No significant change in symptom scores was found over the four tests for the first year, although the comparison of test 1 versus test 4 did approach significance.

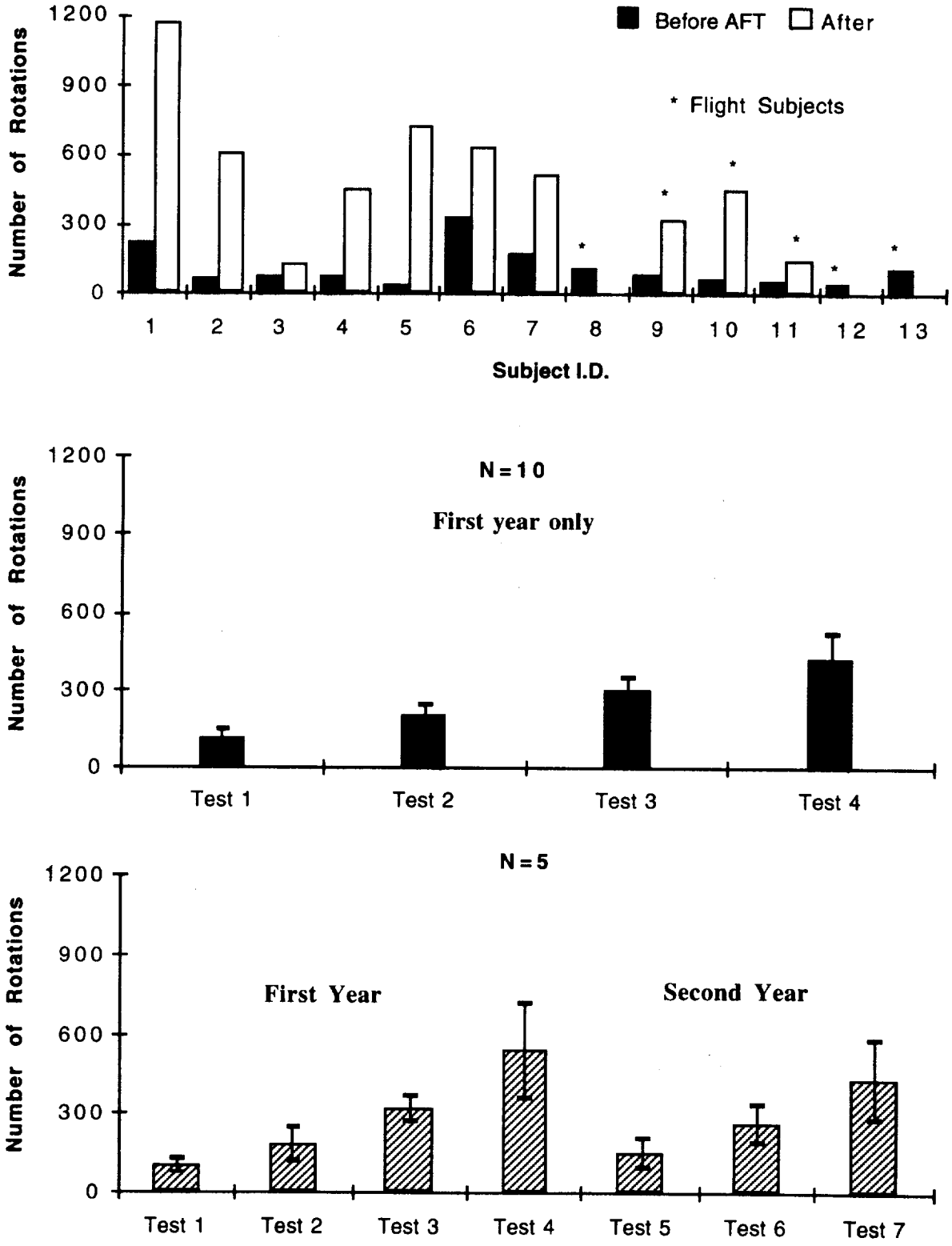

Figure 3. Individual and group changes in motion sickness tolerance before and after training.

Table 3. Mean symptom scores for treatment subjects across four rotating chair motion sickness tests (N = 10)

Subject I.D.	Test 1	Test 2	Test 3	Test 4
1	2.6	3.8	3.0	2.08
2	8.0	6.5	2.4	3.71
3	9.0	5.5	4.0	3.33
4	6.67	5.67	5.75	3.6
5	7.0	4.6	3.67	3.3
6	5.5	3.57	2.14	3.22
7	4.75	2.4	0.38	5.67
9[a]	5.33	2.66	3.0	3.16
10[a]	6.0	1.83	2.14	1.75
11[a]	10.1	1.33	4.0	4.5

[a]Flight treatment subjects.

During the second year, there was a significant change in symptom scores over tests, $X(3) = 7.8$, $p < 0.05$, and only the comparison of test 1 (no treatment) versus test 7 was significant, $Z(3) = 2.69$, $p < 0.05$. Malaise was significantly reduced for this group, but only after the training was completed in their second year.

Preflight Physiological Results

Another set of analyses was conducted on the physiological data collected during the preflight motion sickness tests to explore (1) the time course of physiological responses to motion sickness stimuli, and (2) the effects of AFT on physiological responses to motion sickness. Ten physiological measures were used as variates in a repeated measures MANOVA. The variates were (1) heart rate (HR), (2) respiration rate (RR), 3() vagal tone (VT), (4) coherence of heart rate and respiration (COHER), (5–7) root-mean-square values computed from the spectral estimates of heart rate in the low-, mid-, and high-frequency bands (RMSLOW, RMSMID, RMSHIGH), and (8–10) the ratio of the power in each heart rate frequency band to the total power in the spectrum (RATIOLOW, RATIOMID, RATIOHIGH). Five minute time epochs that were common to all subjects during motion sickness tests were selected for analyses. These epochs were two prerotation baselines, 6 RPM, 8 RPM, End (before chair rotation stopped), and two postrotation baselines. Identical epochs were selected for the vertical motion test.

The first analysis examined the physiological responses of 13 subjects (10 treatment, 3 controls) during their initial exposure to the rotating chair. Figures 4 and 5 show the means (N = 13) for each of the physiological measures plotted over time epochs of the first rotating chair motion sickness test. A significant effect for epochs was observed for heart rate, $F(2.36, 28.33) = 12.38$, $p < 0.0001$; respiration rate, $F(3.10, 37.26) = 3.84$, $p < 0.02$; vagal tone, $F(2.89, 34.7) = 3.83$, $p < 0.02$; and coherence of heart rate and respiration, $F(3.58, 42.96) = 5.0$, $p < 0.003$. Heart rate was low in the prerotation baselines, increased abruptly and remained high over rotation epochs, and decreased in the postrotation baseline. Respiration rate was relatively stable during the prerotation baselines and during rotation, but decreased in the postrotation baselines. Vagal tone was low in the initial prerotation baseline and increased before rotation. A gradual decrease in vagal tone was observed over rotation epochs and the decrease continued in the postrotation baselines. Coherence was stable in the prerotation baseline epochs, decreased over epochs of rotation, and increased in the final postrotation baseline.

Table 4. Mean symptom scores for treatment subjects across seven rotating chair motion sickness tests (N = 5)

	First year				Second year		
I.D.	Test 1	Test 2	Test 3	Test 4	Test 5	Test 6	Test 7
1	2.6	3.8	3.0	2.08	4.2	1.28	0.31
2	8.0	6.5	2.4	3.71	3.33	7.0	3.66
3	9.0	5.5	4.0	3.33	7.0	2.67	4.0
4	6.67	5.67	5.75	3.6	3.0	3.71	3.63
9	5.33	2.66	3.0	3.16	3.86	4.16	3.0

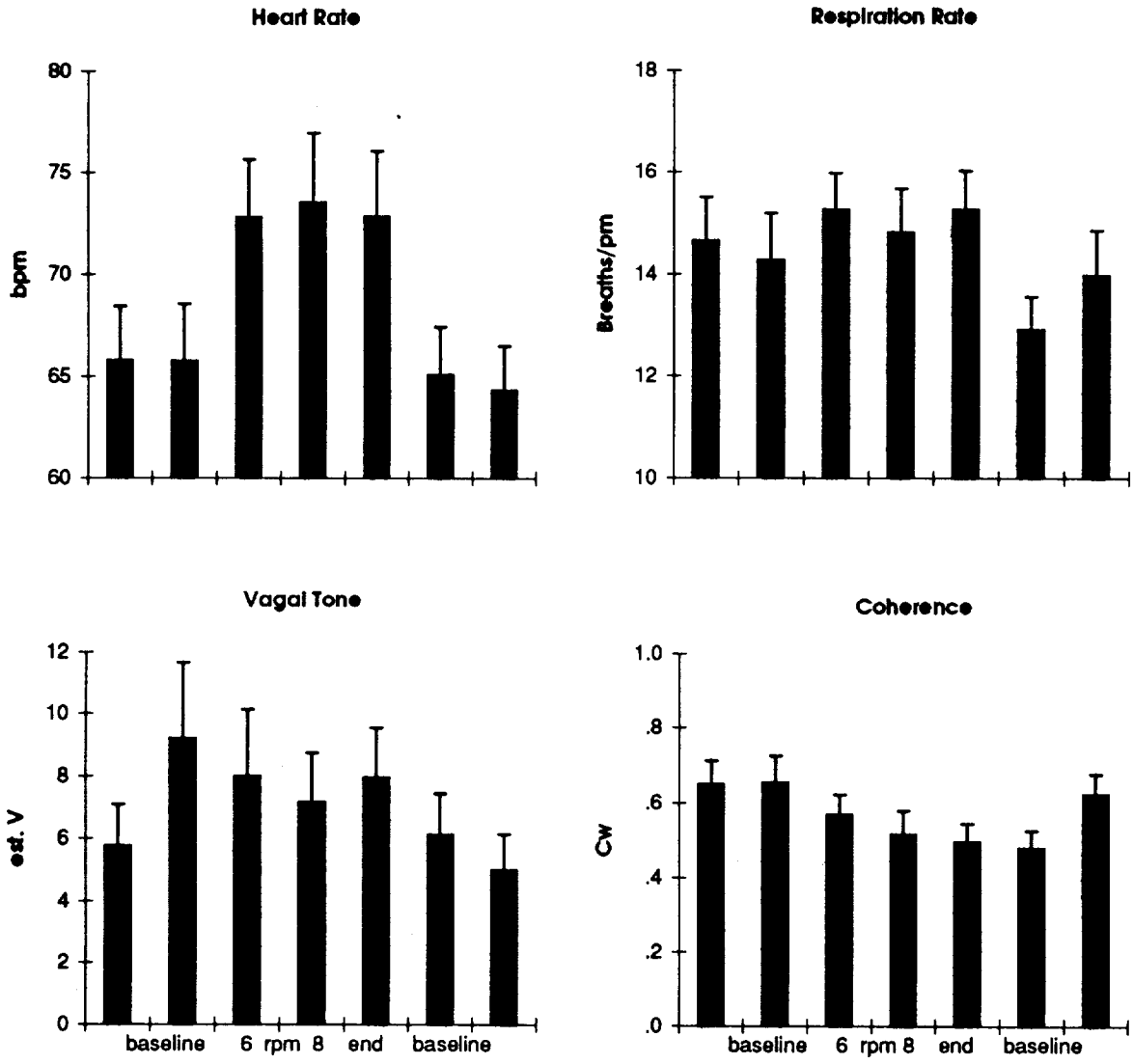

Figure 4. Group means of physiological responses to the first rotating chair motion sickness test (N = 13).

A second analysis examined changes in the physiological responses of six subjects (four subjects were excluded from the analysis because their data were incomplete) to two types of motion sickness stimuli, a rotating chair and a vertical motion simulator. A significant two-way interaction (tests × epochs) was found for RMSMID, $F(3.66, 18.28) = 3.15$, $p < 0.04$, and for RATIOMID, $F(2.78, 13.88) = 3.56$, $p < 0.04$. During the rotating chair test, heart rate variability in the mid-frequency band showed a gradual increase from the prerotation baseline to the end of rotation and then a decrease in the post-rotation baseline. This trend was not as apparent during the vertical motion test. In fact, RMSMID was initially higher and appeared more stable during this test than during the rotating chair stimulus. A similar effect was observed for RATIOMID. Heart rate variability in the mid-frequency band appeared to be more sensitive to the rotating stimulus than to vertical motion.

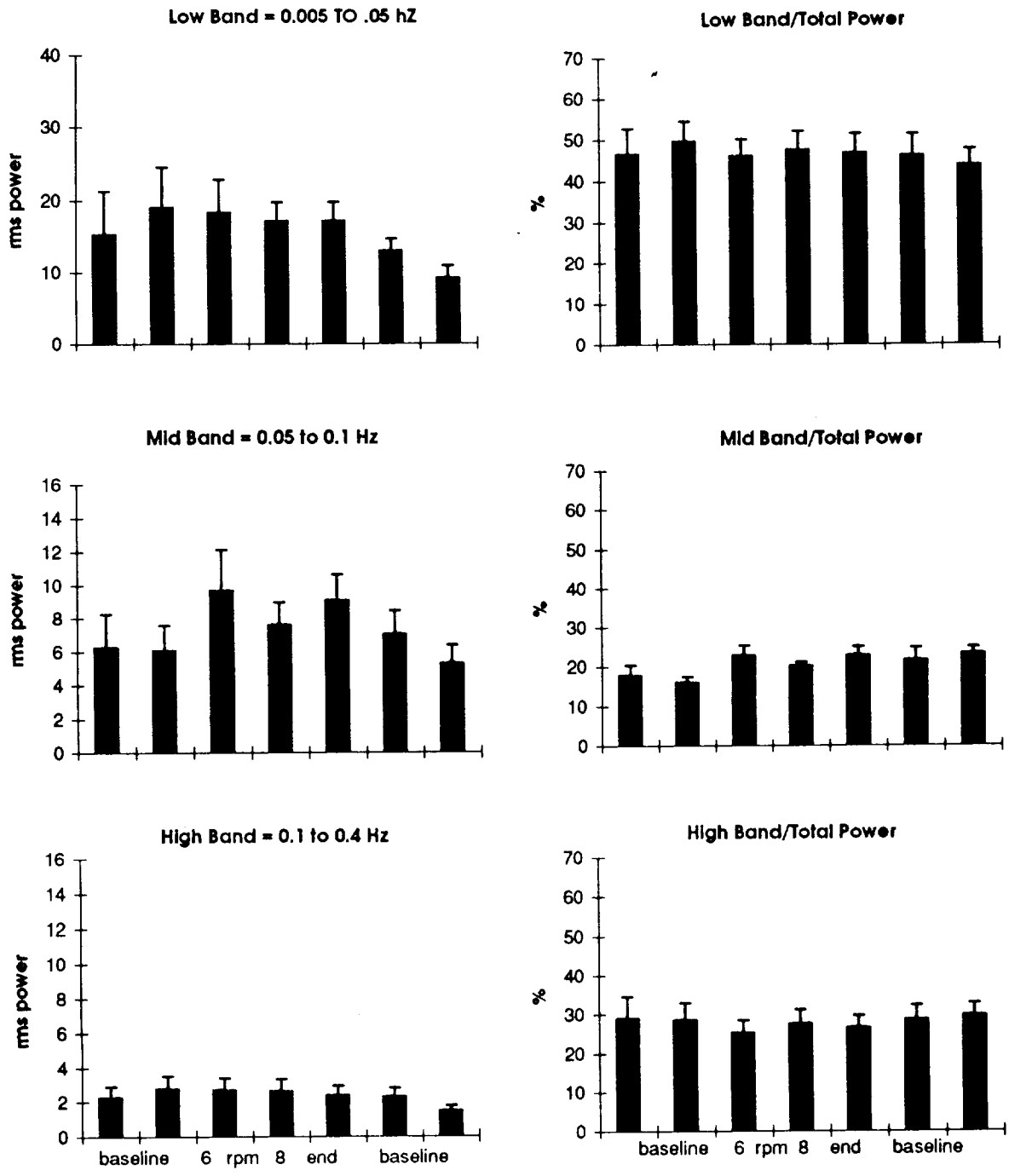

Figure 5. Group means of heart rate variability (expressed as rms power) and percent of total power in three frequency bands during specific epochs of the first rotating chair motion sickness test (N = 13).

A third analysis was conducted to investigate AFT effects on physiological responses during motion sickness stimulation in a rotating chair. Ten physiological variables of treatment subjects (N = 10) were examined before training (test 1) and after 6 hours of AFT (test 4). Again, epochs were included as a factor in the analysis to assess the time course of physiological responses to the stimulus. The test × epochs interaction was significant for COHER, $F(3.49, 31.37) = 3.71$, $p < 0.01$, and RATIOMID, $F(3.75, 33.77) = 2.61$, $p < 0.05$. Only one other physiological variable approached significance, RATIOLOW, $F(2.88, 25.94) = 2.74$, $p < 0.06$. Visual inspection of figures 6 and 7 reveals that coherence between heart rate and respiration over epochs of the motion sickness test was higher after training (test 4) than before (test 1). Figure 7 shows that before AFT (test 1) percent power in the mid-frequency band of heart rate variability gradually increases over epochs, and after AFT (test 4) the ratio is initially higher and then decreases over epochs. The inverse can be seen for percent power in the low-frequency band. Before training (test 1) the ratio is initially higher and slightly decreases over the epochs, but after training the ratio is initially lower and then increases over epochs of the test.

Two final analyses of the physiological measures from the preflight motion sickness tests were conducted using

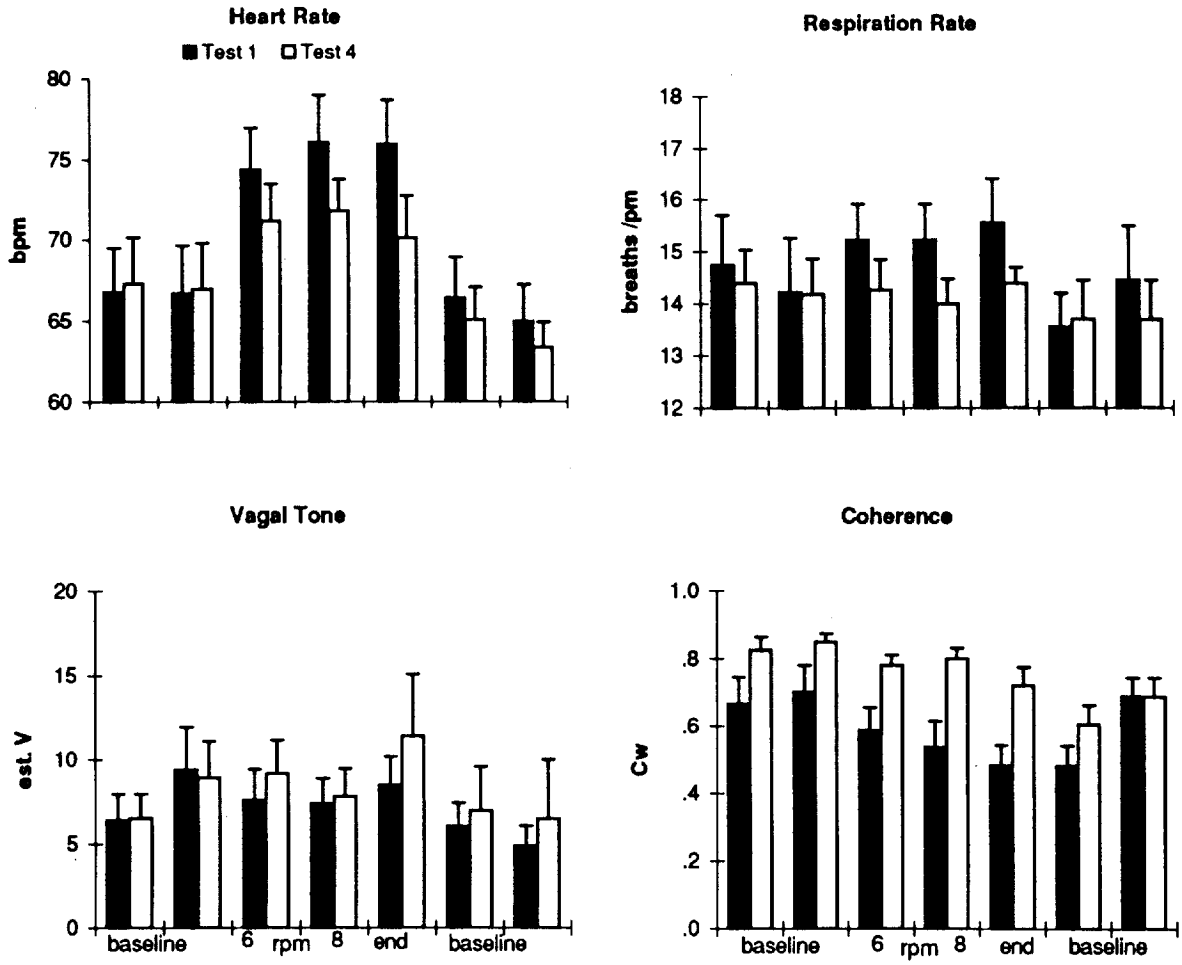

Figure 6. Group means of physiological responses to rotating chair motion sickness tests before and after training (N = 10).

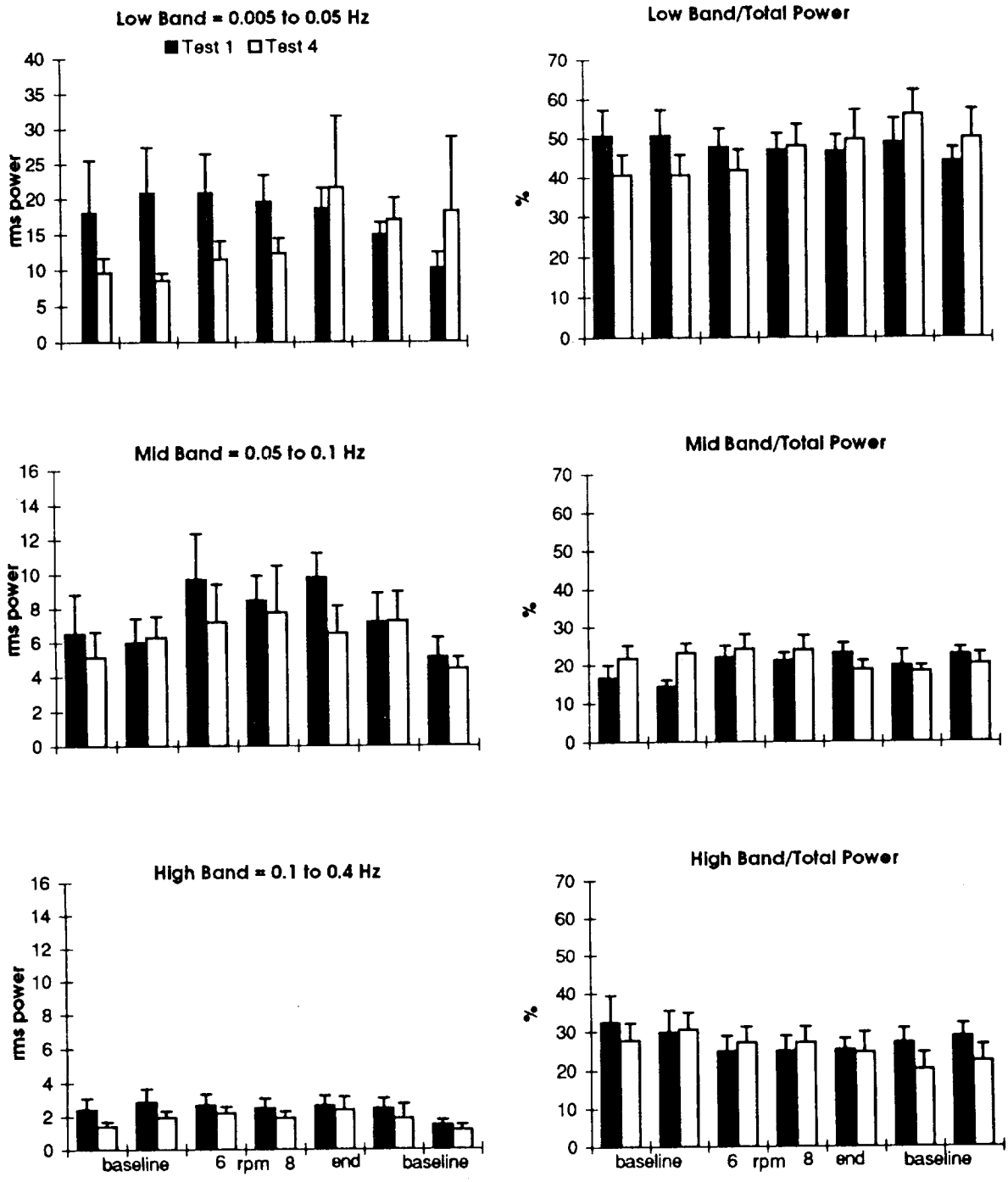

Figure 7. Group means of heart rate variability (expressed as rms power) and percent of total power in three frequency bands during specific epochs of rotating chair motion sickness tests before and after training (N = 10).

the data of treatment subjects (N = 5) who were given AFT over 2 years. In the first analysis, 10 physiological variables from test 1 (no treatment) and test 4 (6 hours of AFT (first year)) were compared over the epochs of each test. The tests × epochs interaction was not significant for any of the physiological variables. Figure 8 shows the group means for heart rate, respiration, vagal tone, and coherence over specific epochs of motion sickness tests. A second analysis compared the physiological measures of test 1 (no treatment) and test 7 (after 6 hours of AFT (second year)) for the same time epochs. A significant tests × epochs interaction was found only for RMSMID,

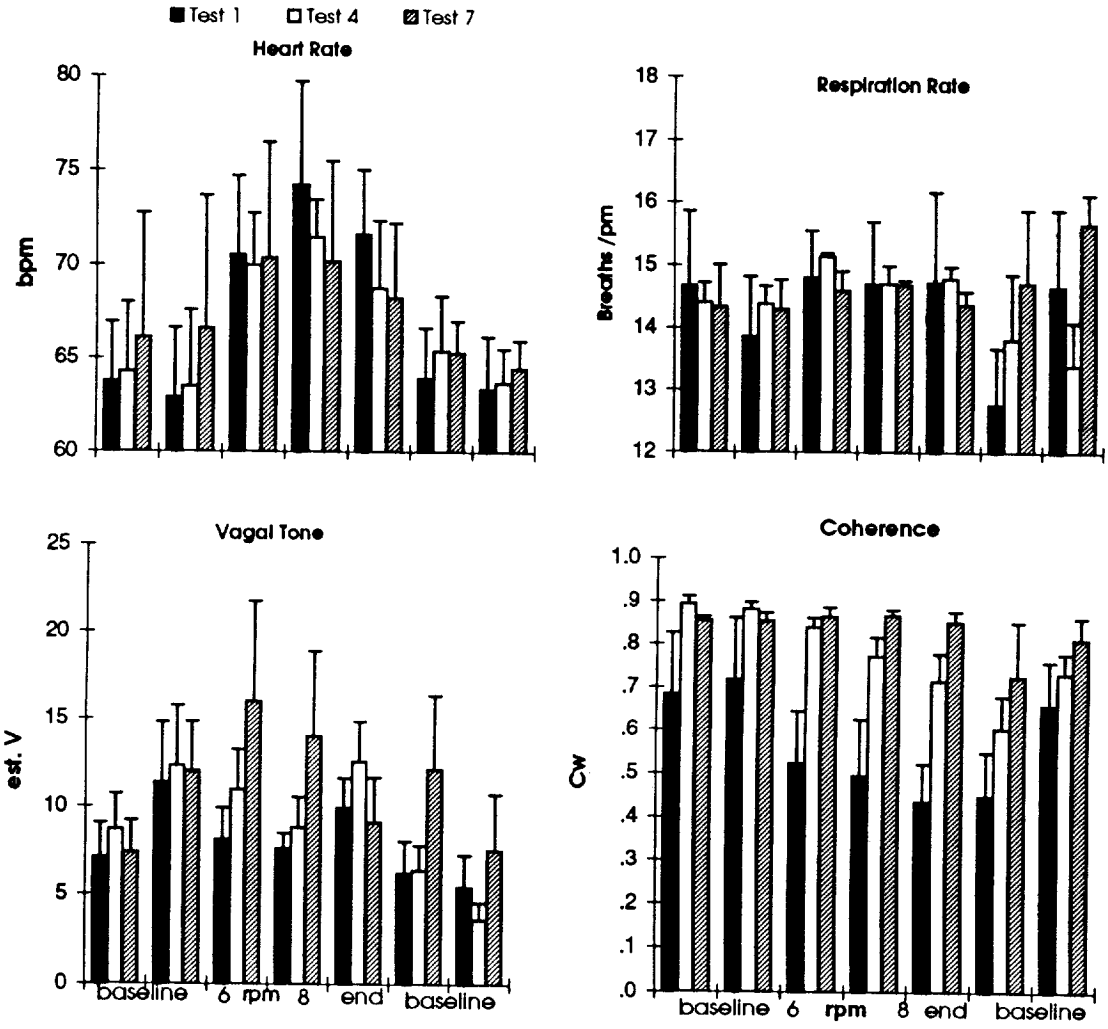

Figure 8. Group means of physiological responses to rotating chair motion sickness tests before and after training during the first and second year (N = 5). First year = tests 1 and 4, second year = test 7.

$F(2.49, 9.97) = 3.22$, $p < 0.05$. Figure 9 shows that on test 1 heart rate variability in the mid-frequency band sharply increases over epochs until the end of rotation and then decreases during the postrotation baseline. During test 7, after 6 hours of AFT, the power in this frequency band is initially higher in the prerotation baseline and then decreases over epochs until the postrotation baseline.

Flight Symptom Score Results

Table 5 lists the type and frequency of symptoms reported by each subject during 4 days of spaceflight. The three treatment subjects who were given preflight AFT for control of their motion sickness symptoms did not take antimotion sickness medications during the flight. However, the two of the three control subjects, who were given no preflight treatment, took antimotion sickness medications for symptoms experienced during flight. Note that this was the second spaceflight for subject 13.

Table 6 represents the symptom score totals of each subject on each mission day. The scores were derived by summing the point values for the individual symptoms reported on each day. These data were analyzed to examine group effects and changes in motion sickness malaise over days. No significant group difference was found. But, there was a significant difference over the 4 days ($N = 6$), indicated by the Friedman test statistic, $X(3) = 13.55$, $p < 0.003$. Comparisons showed that day 1 versus day 3 and day 1 versus day 4 were significant, $Z(3) = 2.80$, $p < 0.05$, and $Z(3) = 3.24$, $p < 0.05$, respectively. Motion sickness malaise was significantly reduced by mission day 3.

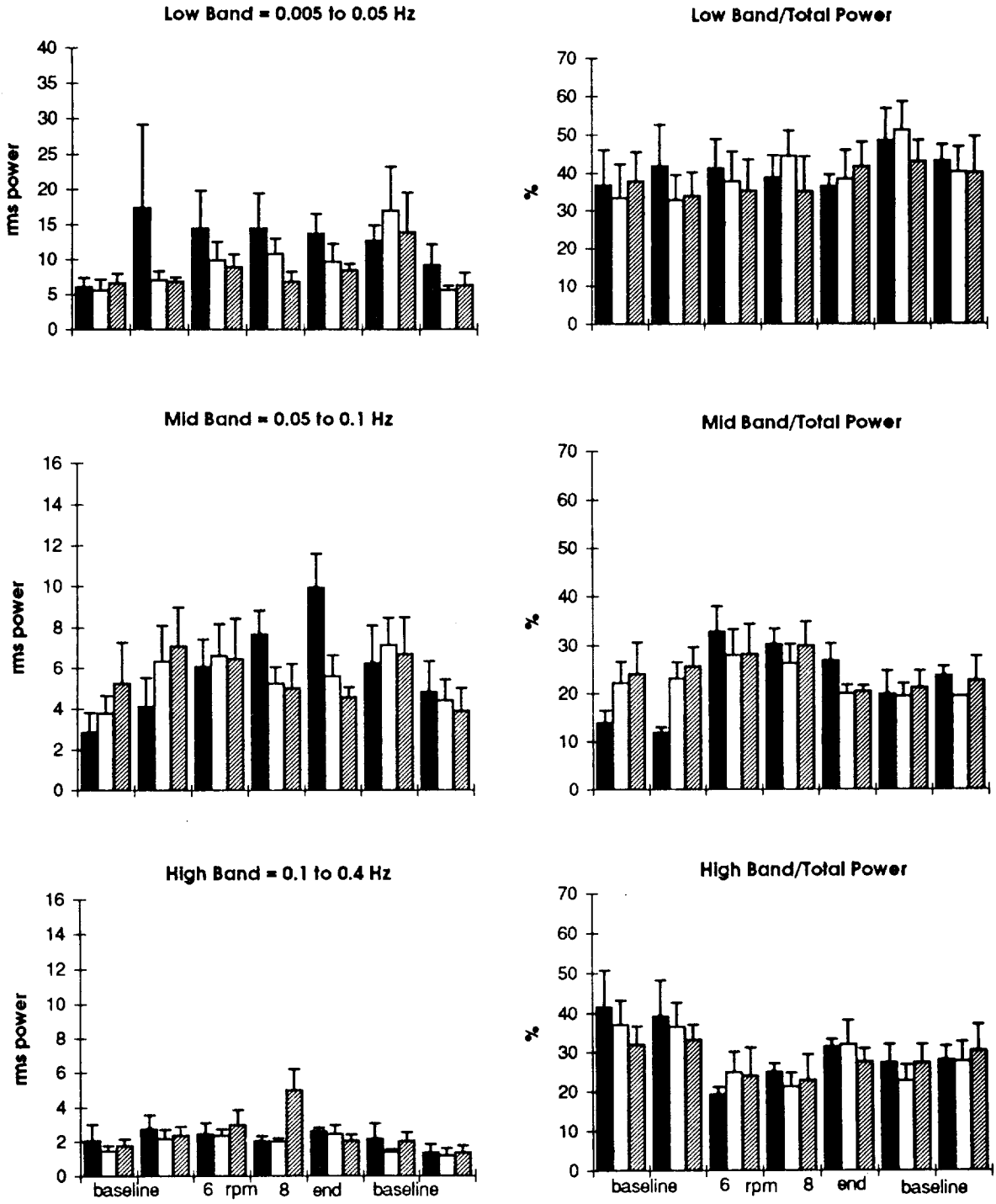

Figure 9. Group means of heart rate variability (expressed as rms power) and percent of total power in three frequency bands during specific epochs of rotating chair motion sickness tests before and after training during the first and second year (N = 5). First year = tests 1 and 4, second year = test 7.

Table 5. Motion sickness symptoms reported over mission days

I.D	Day 1	Day 2	Day 3	Day 4	Medication
			Treatment group		
9	Vomiting (5 times)	Vomiting (1 time)	Mild nausea	Gastric awareness	Ducolax
	Mild drowsiness	Mild drowsiness	Mild drowsiness		
	Mild salivation	Moderate nausea			
	Moderate nausea				
10	Mild nausea	Headache	None	None	None
	Headache				
	Dizziness				
11	Moderate nausea	Vomiting (1 time)	None	Facial pallor	None
	Headache	Mild nausea			
			Control group		
8	Moderate nausea	None	None	None	Restoril
	Headache				
12	Vomiting (6 times)	Gastric discomfort	Gastric awareness	None	Compazine
	Severe nausea	Moderate nausea			Valium
	Profuse sweating				
	Hyper salivation				
13[a]	Vomiting (2 times)	Moderate nausea	Gastric discomfort	None	Reglan
	Mild drowsiness		Increased warmth		Valium

[a]Second spaceflight.

Table 6. Symptom score totals for each subject over mission days

I.D.	Day 1	Day 2	Day 3	Day 4
Treatment group				
9	28	26	6	1
10	6	1	0	0
11	9	20	0	2
Control group				
8	9	0	0	0
12	40	10	1	0
13	26	8	3	0

Flight Physiological Results

Physiological data of the six flight subjects were analyzed to examine (1) the effects of early adaptation to microgravity (the first 4 days in space), (2) physiological responses on day 2 in space compared to a ground-based simulation of the same mission day, and (3) physiological differences between treatment and control subjects over mission days. Appendix B includes the graphs of each individual's physiological data during spaceflight. The data are plotted as contiguous 8.5 minute epochs over each mission day. Missing data are indicated on the graphs. The respiration data of four subjects could not be processed because of poor signal quality. Group means for each physiological measure, excluding RR and COHER, were computed from the 8.5 minute epochs of each flight day and a ground-based mission simulation. Figures 10–13 represent the physiological means of all subjects (N = 6), treatment subjects (N = 3), and control subjects (N = 3) plotted over flight days (left bar graph) and means comparing mission day 2 to a ground simulation of the same flight day (right bar graph). Standard errors are also plotted on the graphs. The first analysis examined differences in the physiological responses of treatment and control subjects over the 4 days of flight. No group differences over days were observed; however, the mid-frequency band for heart rate variability approached significance, $F(2, 7.99) = 3.33$, $p < 0.06$. The treatment group showed a gradual increase over days in the mid-band frequency for heart rate variability, while this response for the control group was the inverse. A significant day's effect was found only for heart rate, $F(2.77, 11.07) = 7.88$, $p < 0.004$. Comparisons of day 1 with each of the other flight days were all significant, (day 1 versus day 2), $F(1, 4) = 14.69$, $p < 0.01$; (day 1 versus day 3), $F(1, 4) = 14.21$, $p < 0.01$; and (day 1 versus day 4), $F(1, 4) = 17.69$, $p < 0.01$. Heart rate is initially high on the first day of flight, significantly decreases by the second day, and remains low over days 3 and 4.

The final analysis compared the physiological responses of treatment and control subjects on day 2 of flight with their data collected during a ground simulation of the same mission day. Results of the MANOVA indicated a significant day's effect (flight versus simulation) for HR, $F(1, 4) = 90.08$, $p < 0.007$; RATIOLOW, $F(1, 4) = 8.39$, $p < 0.04$; RATIOHIGH, $F(1, 4) = 7.90$, $p < 0.04$. Heart rate and percent power in the low-frequency band for heart rate variability were both significantly lower in space than on Earth. Percent power in the high-frequency band for heart rate variability, which primarily reflects respiratory sinus arrhythmia, was higher in space than during the ground simulation. A significant group's × day's effect was observed for heart rate only, $F(1, 4) = 13.73$, $p < 0.02$. The heart rate responses of both groups were comparable in space, but during the ground simulation heart rate was significantly higher for treatment subjects (86 bpm) than controls (75 bpm).

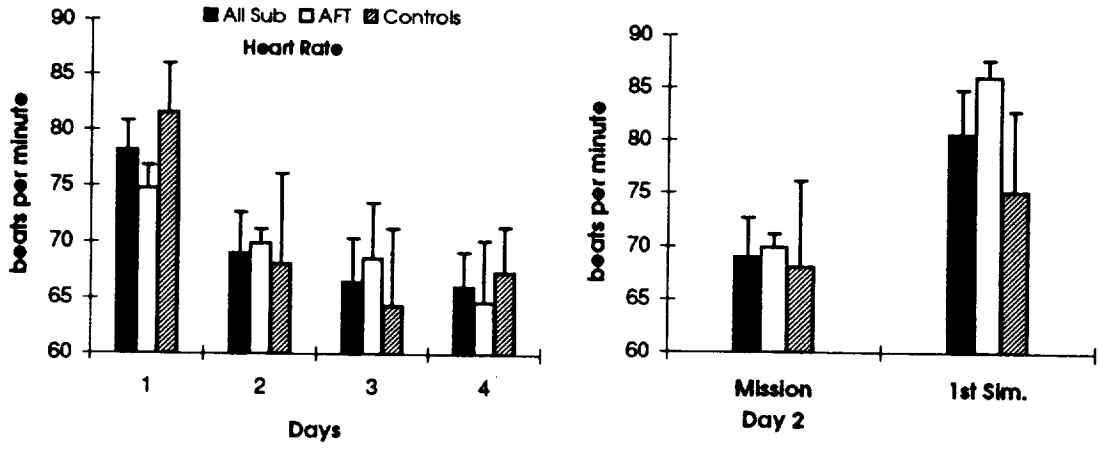

Figure 10. Group means of heart rate across days in space and compared to an Earth-based simulation.

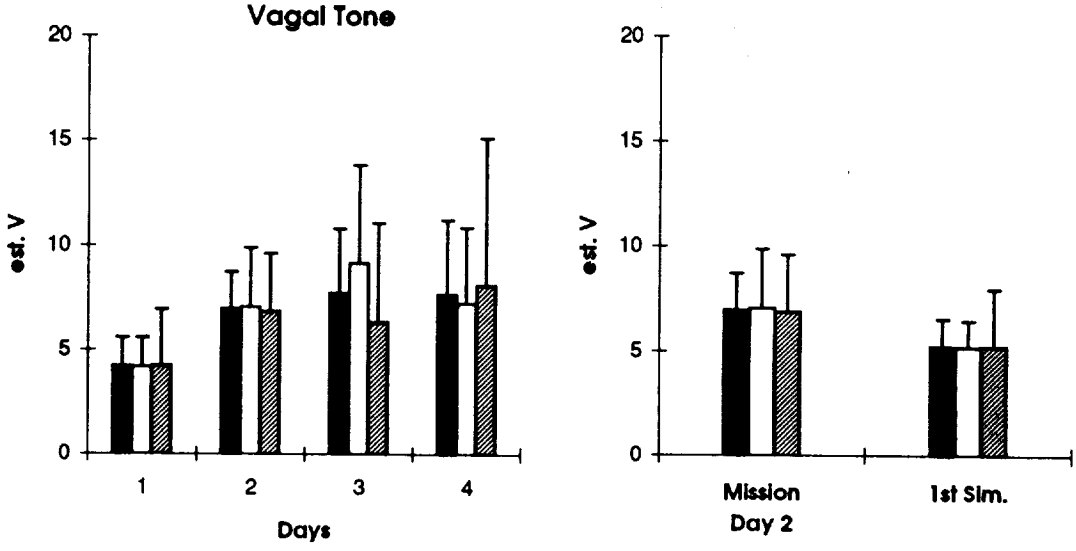

Figure 11. Group means of vagal tone across days in space and compared to an Earth-based simulation.

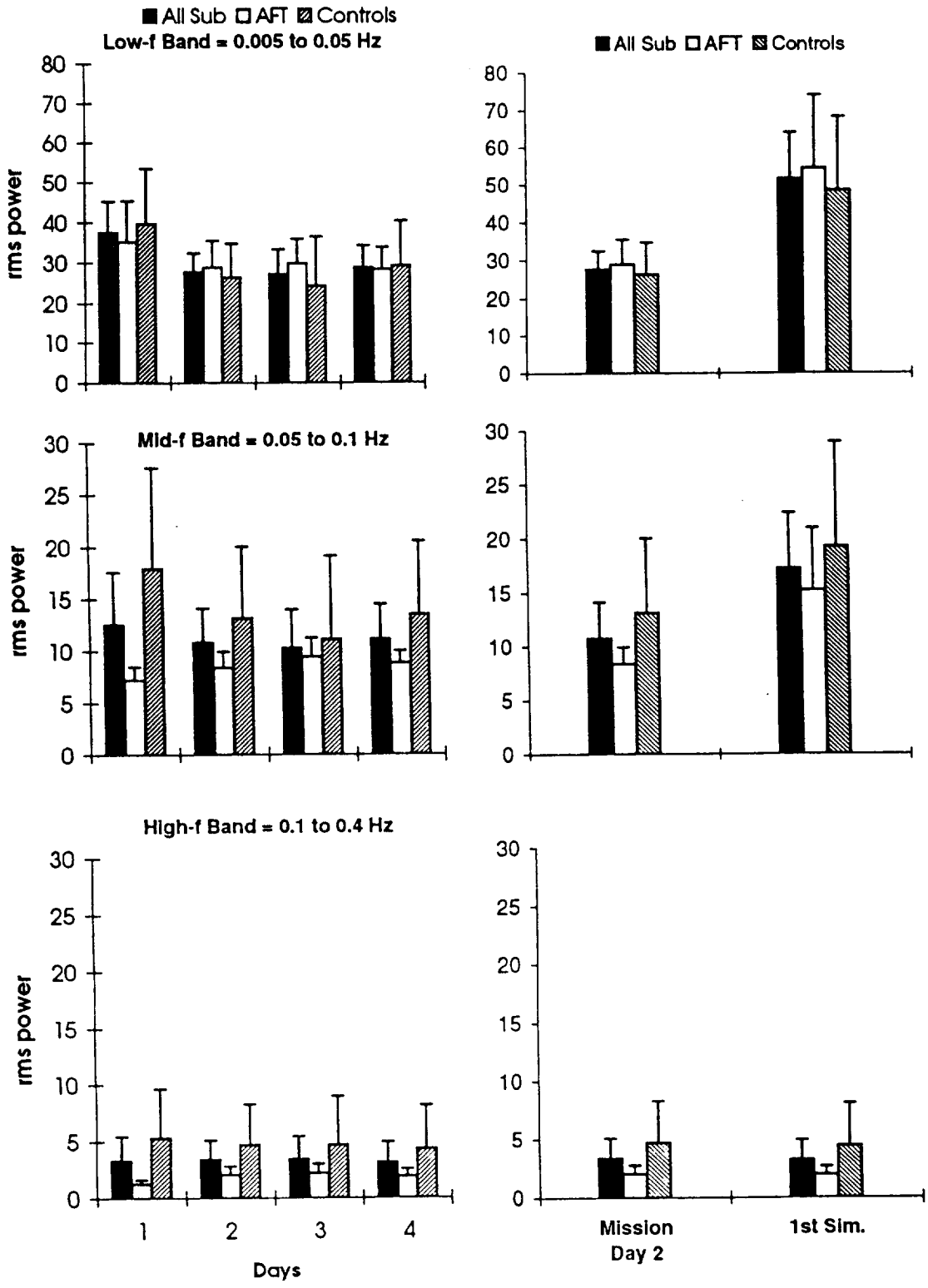

Figure 12. Group means of heart rate variability (expressed as rms power) in three frequency bands across days in space.

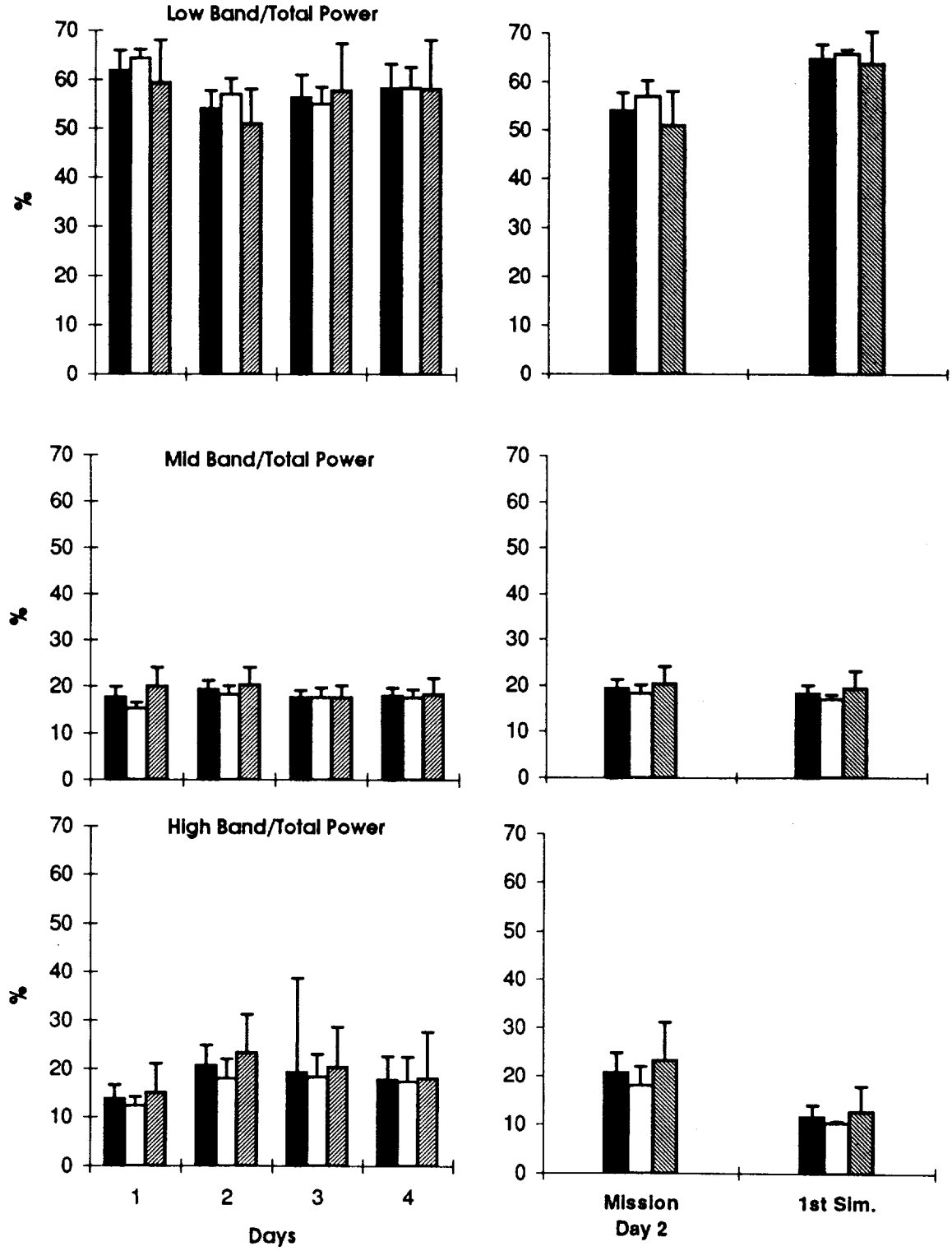

Figure 13. Group means of heart rate variability as a percent of total power in three frequency bands across days in space and compared to an Earth-based simulation.

Discussion

The results of this experiment are in agreement with previous studies (Cowings, 1990) demonstrating that AFT significantly increases tolerance to rotating chair motion sickness tests. Further, it was shown that this increased tolerance was associated with changes in specific physiological responses and reports of reduced malaise. However, subjects in this study varied widely in their ability to learn control of motion sickness symptoms as can be seen in the individual symptom reports (appendix A). A possible explanation for this variability in performance was the different AFT schedules used for some subjects. Four treatment subjects (two flight and two alternates) were given rotating chair tests over intervals ranging between 30 and 300 days. Because of delays in the mission launch date, mission management rescheduled these subjects for AFT over a 17 month period.

For the other treatment subjects, we succeeded in maintaining an AFT schedule more closely aligned to that used in laboratory studies, which produced the most effective learning of symptom control (Cowings, 1990). These subjects received twelve 30 minute sessions (6 hours total), administered in blocks of four consecutive days over 3 weeks. Rotating chair motion sickness tests were separated by 7 day intervals. In both cases, AFT was initiated approximately 1 year prior to the scheduled launch date. When delays in the launch date for this mission were extended 1 year, these subjects received an additional 6 hours of AFT. Despite the scheduling changes for some of the astronauts participating in preflight AFT, the results indicated that the training method was an effective treatment for ground-based motion sickness. It was concluded that the latter schedule with AFT sessions on consecutive days produced better learning.

The primary hypothesis of this research was that preflight AFT would reduce or eliminate the symptoms of space motion sickness. Previous research (Cowings, 1990) showed that the AFT treatment effect transfers from motion sickness stimulation in a rotating chair to vertical up and down motion and to a combination of optokinetic stimulation with rotation in a chair. AFT apparently operates on the final common path in the development of motion sickness symptoms. The demonstrated ability to transfer training effects to a variety of Earth-based motion environments led us to hypothesize that AFT would transfer to space as well.

The flight results showed that two of the three control subjects experienced multiple vomiting episodes on the first mission day, while one control subject experienced only moderate malaise. All control subjects took medication for symptom suppression and/or sedation. Of the three treatment subjects, one experienced only mild discomfort, one experienced moderate discomfort (one vomiting episode on mission day 2), and one experienced severe motion sickness on the first day. The latter subject took a laxative on mission day 4 for symptoms unrelated to motion sickness. None of the other treatment subjects took any medication throughout the flight. These data suggest that AFT may be an effective treatment for space motion sickness; however, this cannot be demonstrated conclusively with the small number of subjects described in this paper.

Physiological data obtained in space clearly demonstrate changes over days as subjects adapted to that environment. Analyses of all six crewmembers showed a significant decrease in heart rate over days. Vagal tone increased whereas low-frequency heart rate oscillations decreased during spaceflight, although these trends were not significant. However, review of the individual physiological data obtained during spaceflight (appendix B) shows that for any given crewmember, within-subject changes in physiological responses across days in space are associated with reports of reduced malaise, i.e., adaptation to microgravity. For example, subject 8, who was least affected by space motion sickness of all the flight subjects, showed an increase in vagal tone during the mission, and also had the highest initial vagal tone on day 1. Further, coherence between heart rate and respiration for this subject also increased over flight days.

In contrast, subject 9, who was highly susceptible to motion sickness, showed lower vagal tone when symptoms were severe on early mission days and a significant increase in vagal tone on those days when symptoms were mild. Heart rate and respiration coherence also followed the same pattern of change for this individual. Further, an examination of heart rate variability for this subject revealed dominant low-frequency oscillations on the first day in space when severe motion sickness was reported. However, these dominant low-frequency oscillations were not as apparent for other subjects for whom motion sickness was severe and were also seen later in the mission when subjects were asymptomatic.

The observations of increased vagal tone and higher coherence between heart rate and respiration across mission days suggest that cardiorespiratory regulatory mechanisms may play a significant role in adaptation to spaceflight. Although the basic anatomy for cardio-respiratory coupling exists and has been demonstrated under different conditions on Earth (Katona, Poitras,

Barnett, and Terry, 1970; Dellinger and Porges, 1984), the findings of the current study provide new information for further examinations of the dynamic behavior of these two systems as an index of stress. The results may also have implications for developing and testing antimotion sickness medications and for the optimization of non-pharmacologic modes of therapy (e.g., AFT) for motion sickness. For example, simply pacing breathing at optimal rates may entrain heart rate in a way that both systems are sufficiently coupled, which may then provide substantial relief from symptoms. Another approach might include training individuals to modify specific patterns of heart rate variance. In particular, training people to increase the amplitude or quality of respiratory sinus arrhythmia (RSA), which correlates with vagal tone (Porges, 1985), may help to prevent symptom onset.

Comparisons of flight to ground-based simulation data revealed marked differences between physiological responses on Earth and in space. For all crewmembers, regardless of group or symptom level in space, heart rate variability in the low-frequency band (0.005 to 0.05 Hz) was greatly reduced in space, being nearly half the normal magnitude. Significant differences were also found for heart rate (lower in space) and for heart rate variability in the high-frequency band (0.1 to 0.4 Hz). The latter measure, which reflects RSA, was higher in space than on Earth.

Physiological results of laboratory motion tests showed that heart rate, respiration rate, vagal tone, and coherence between heart rate and respiration do change during the initial exposure to motion sickness stimulation. These findings are in general agreement with other reports (Cowings, Suter, et al., 1986; Crampton, 1955) which showed increases in heart rate and respiration rate to a rotating chair stimulus, and decreases in vagal tone to a rotating optokinetic drum (Uijtdehaage, Stern, and Koch, 1992). In the latter study it was also shown that initially high levels of vagal tone were inversely related to malaise scores and were predictive of motion sickness. The large increases in heart rate during motion sickness observed in the present study may partially be explained by vagal withdrawal.

The comparison of physiological responses to a rotating chair and a vertical motion stimulus showed that heart rate variability in the mid-frequency band (0.05 to 0.1 Hz) increased during motion sickness in the chair only. This frequency band reflects changes in blood pressure control mechanisms which typically appear as 8 to 10 second cycles in the heart rate spectrum. Although blood pressure recordings were not taken in this study, such information would be useful to help explain the changes seen in heart rate variability.

The effects of AFT on physiological responses during rotating chair motion sickness tests showed that coherence between heart rate and respiration was higher after training than before. Heart rate variability in the low-frequency band was initially reduced following AFT, while the inverse was seen for the mid-frequency band. Further, these effects were related to increases in motion sickness tolerance and decreases in malaise. The paced breathing given with AFT may partially explain the changes in the pattern of heart rate variance. However, the data also suggest that AFT indirectly modulates changes in blood pressure, as was seen in the pattern for heart rate variability.

Finally, it was concluded that ambulatory physiological monitoring is an appropriate way of studying individual differences in adaptation to spaceflight and the time course of this adaptation. And, by examining the physiological profiles of treatment subjects during preflight motion sickness tests it was possible to accurately predict which of the flight treatment subjects would be most resistant and least resistant to symptoms in space. Treatment subjects who were most resistant to space motion sickness showed reduced autonomic variability during preflight motion sickness testing, which strongly suggested that they had learned better autonomic control.

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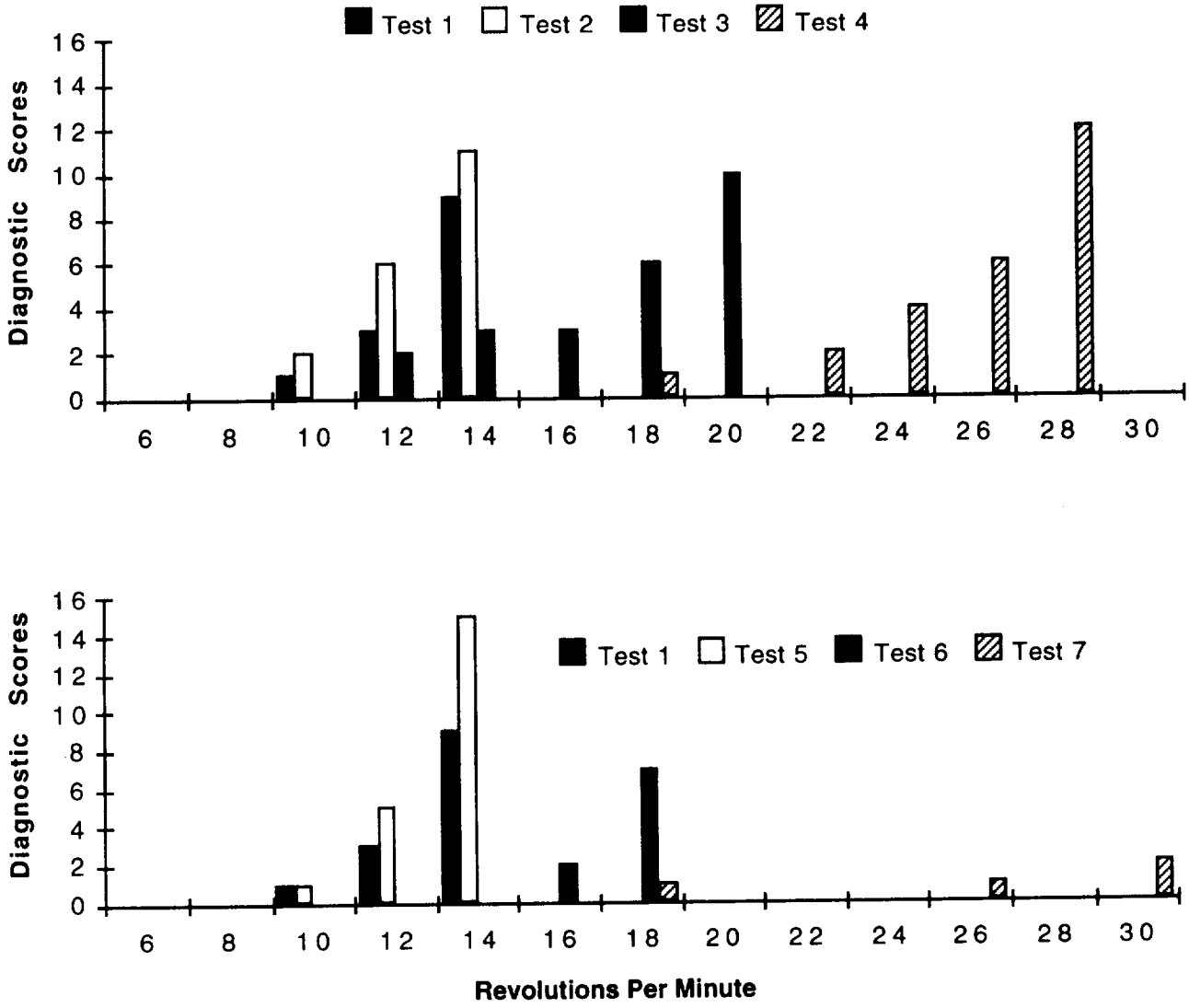
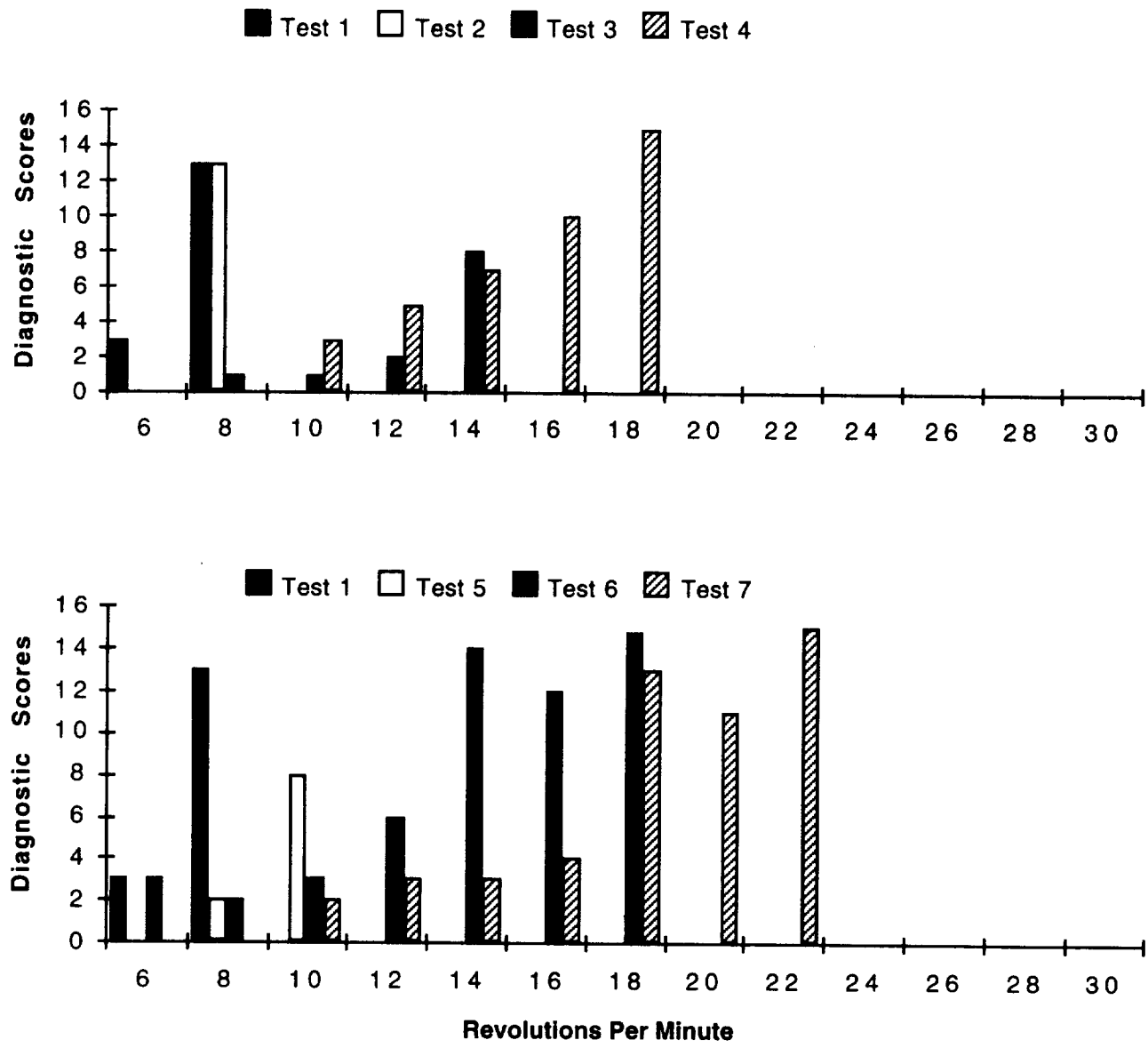
Appendix A

Individual Preflight Symptom Scores and Physiological Data



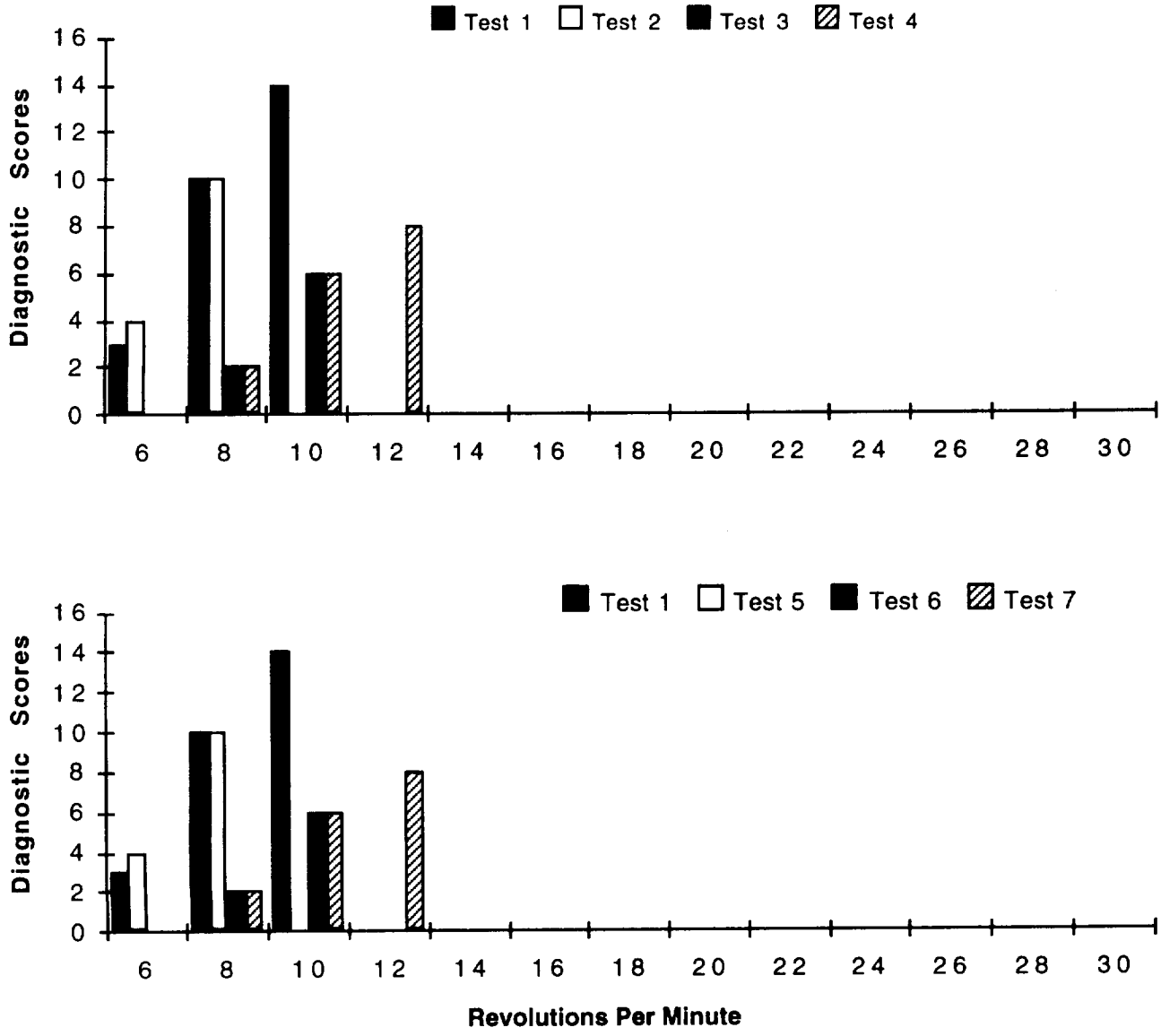
Note: Tests 1–4 were at 1 week intervals. Test 5 was administered approximately 1 year later with tests 5–7, also at 1 week intervals.

Figure A-1. Reports of motion sickness malaise across tests—subject 1.



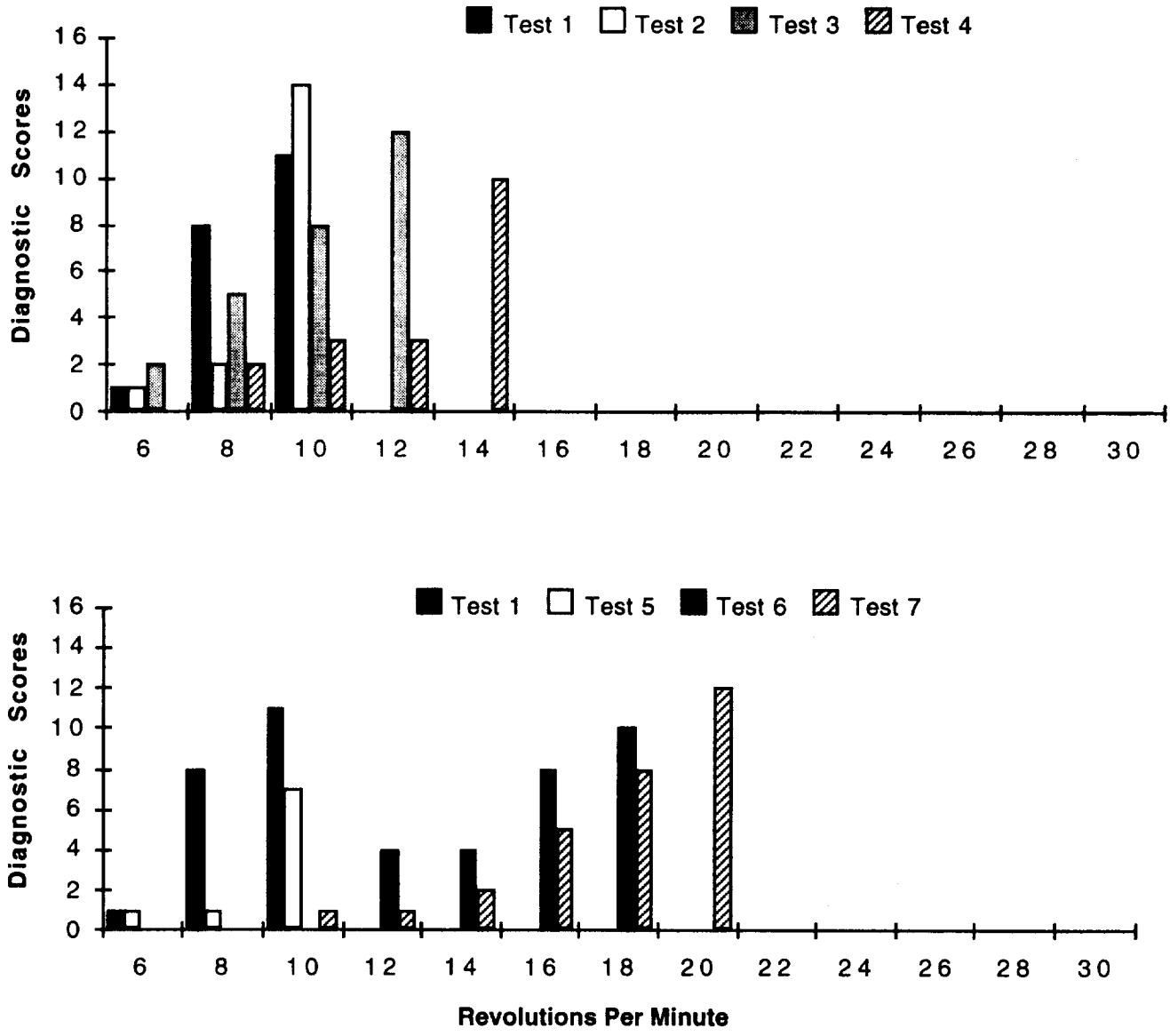
Note: Tests 1–4 were at 1 week intervals. Test 5 was administered approximately 1 year later with tests 5–7, also at 1 week intervals.

Figure A-2. Reports of motion sickness malaise across tests—subject 2.



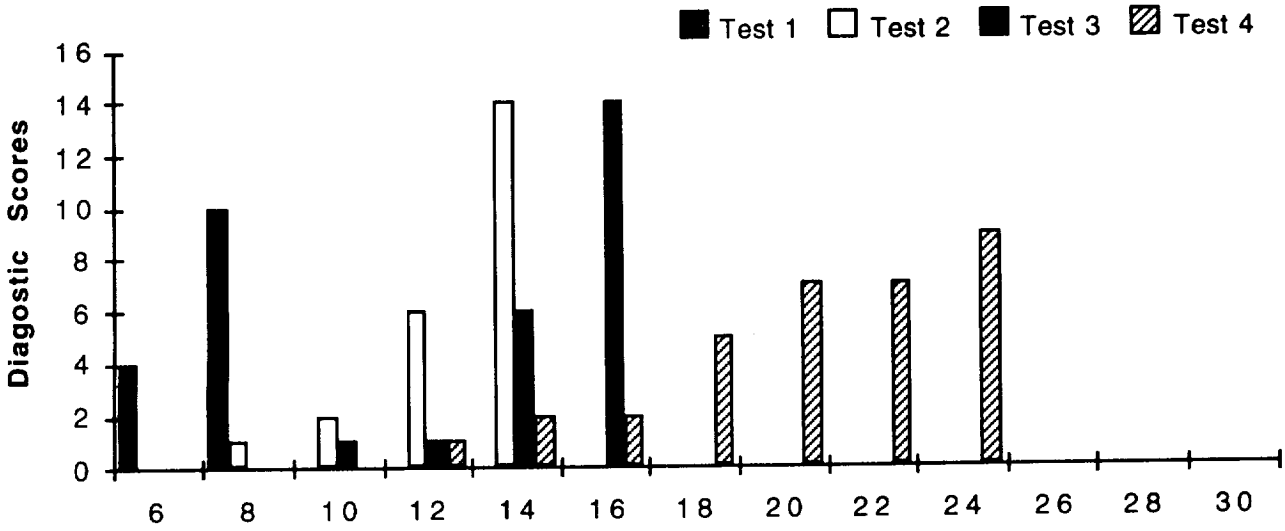
Note: Tests 1–4 were at 1 week intervals. Test 5 was administered approximately 1 year later with tests 5–7, also at 1 week intervals.

Figure A-3. Reports of motion sickness malaise across tests—subject 3.



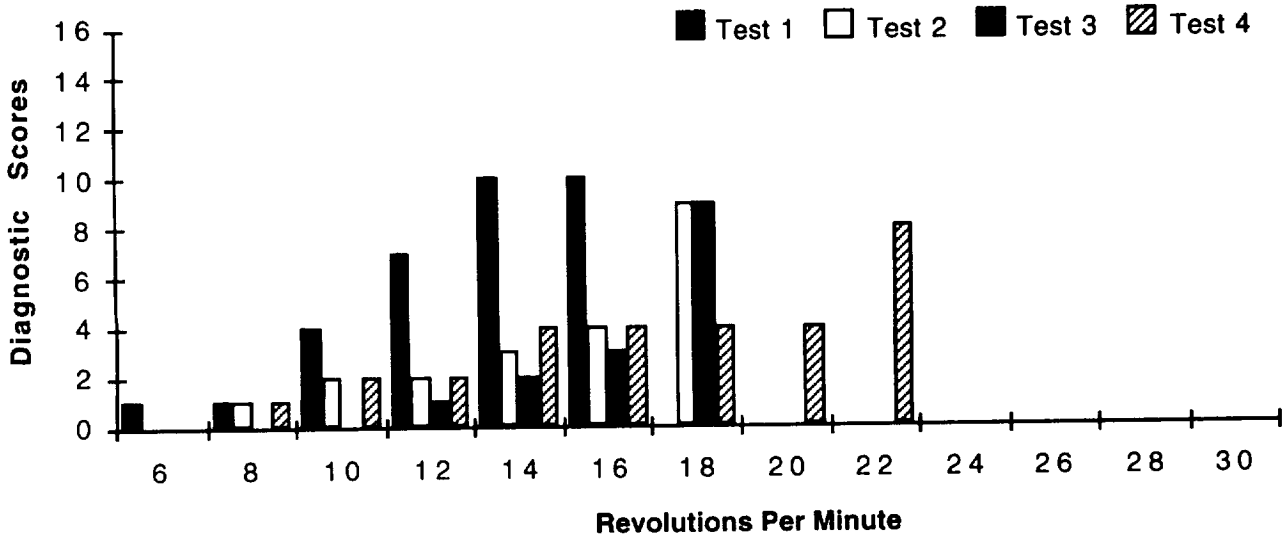
Note: Tests 1–4 were at 1 week intervals. Test 5 was administered approximately 1 year later with tests 5–7, also at 1 week intervals.

Figure A-4. Reports of motion sickness malaise across tests—subject 4.



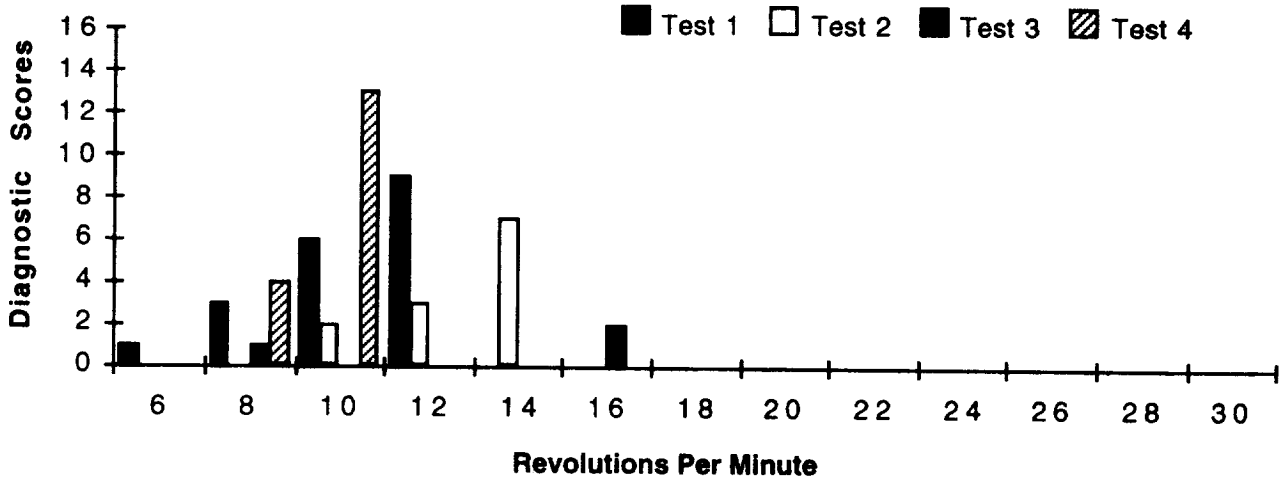
Note: Tests were conducted at weekly intervals.

Figure A-5. Reports of motion sickness malaise across tests—subject 5.



Note: Number of days between tests 1 and 2 = 234; 2 and 3 = 74; 3 and 4 = 54.

Figure A-6. Reports of motion sickness malaise across tests—subject 6.



Note: Number of days between tests 1 and 2 = 201; 2 and 3 = 102; 3 and 4 = 57.

Figure A-7. Reports of motion sickness malaise across tests—subject 7.

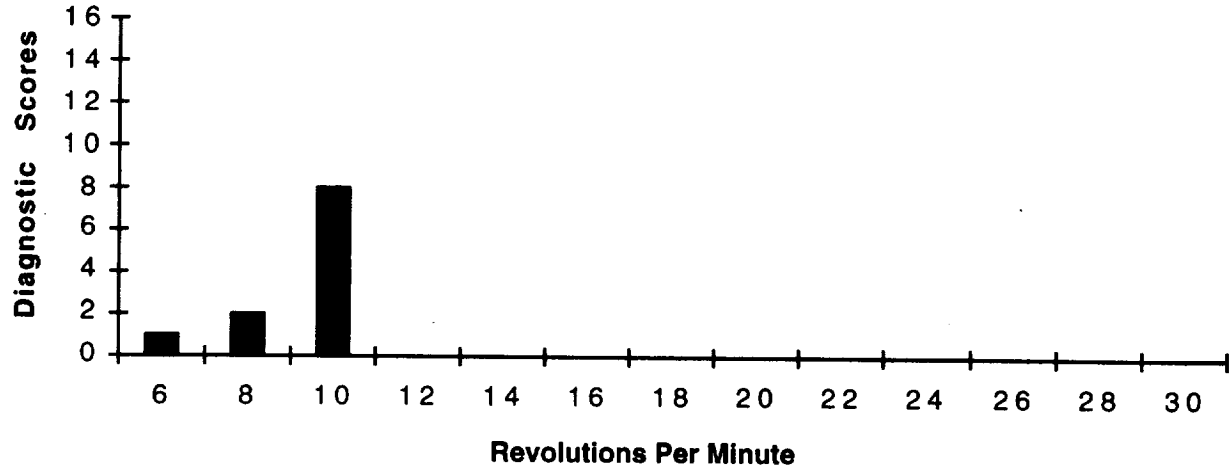
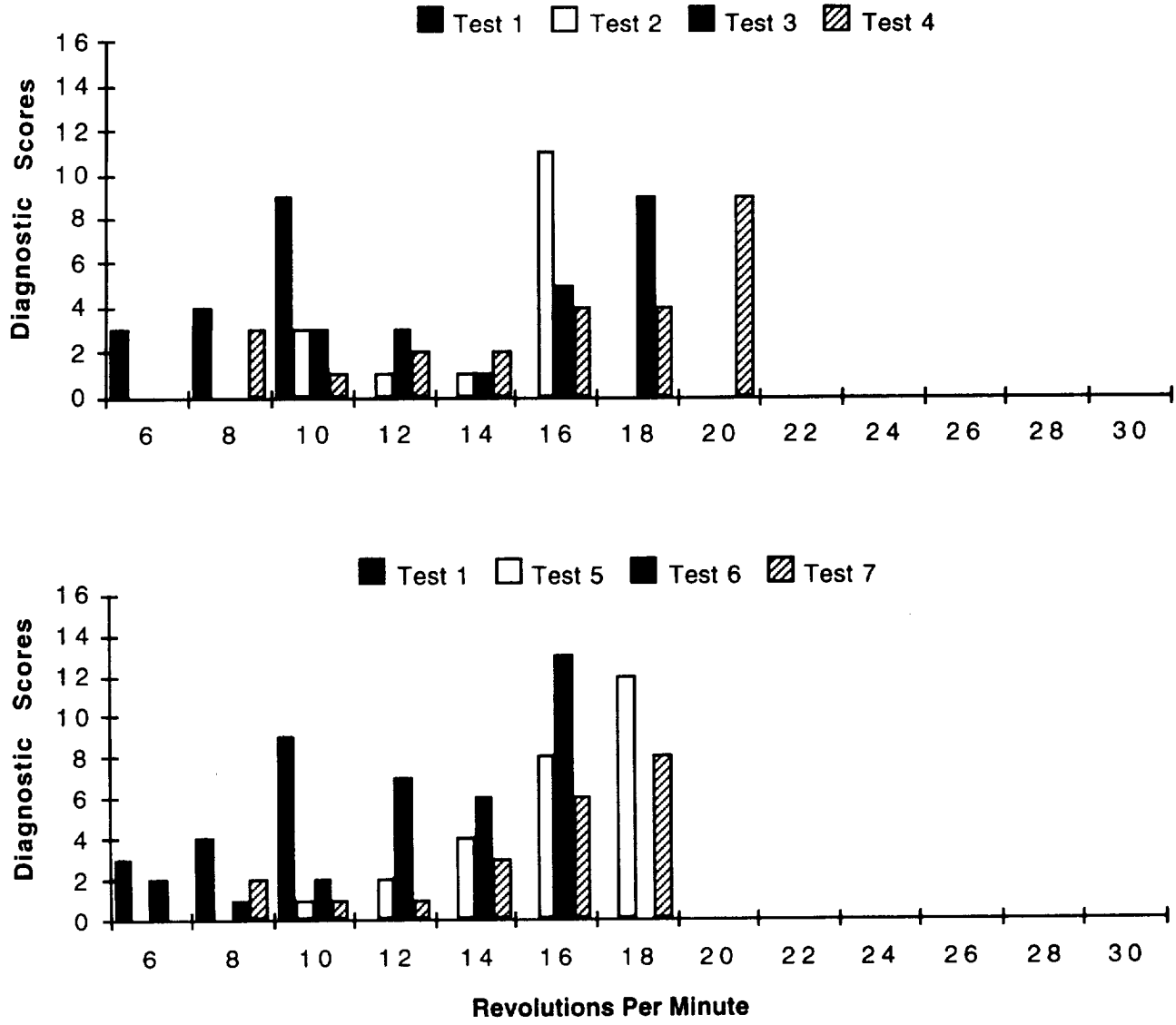
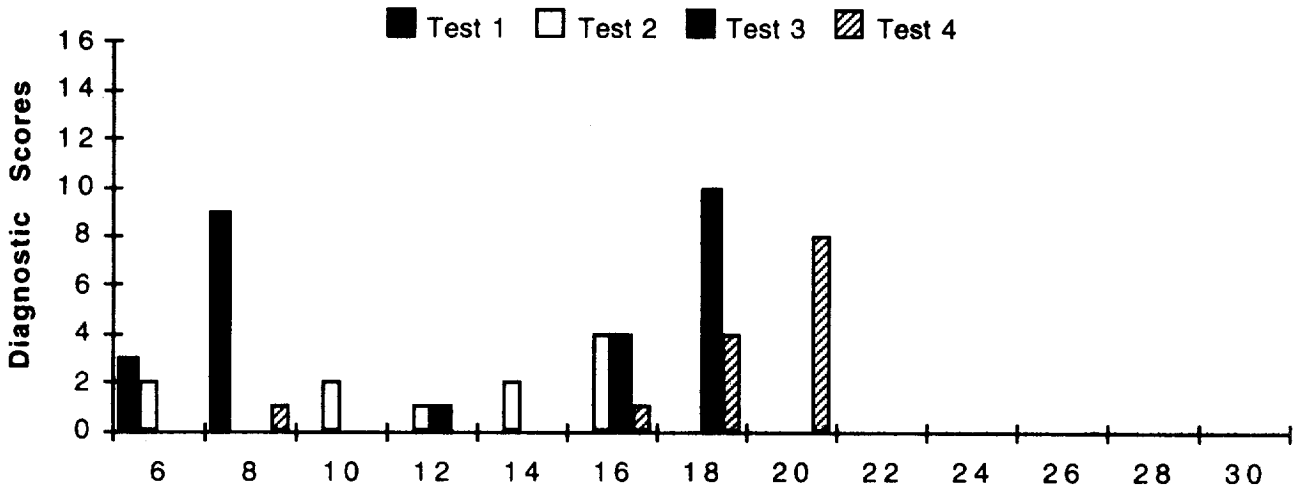


Figure A-8. Reports of malaise during initial motion sickness test—subject 8.



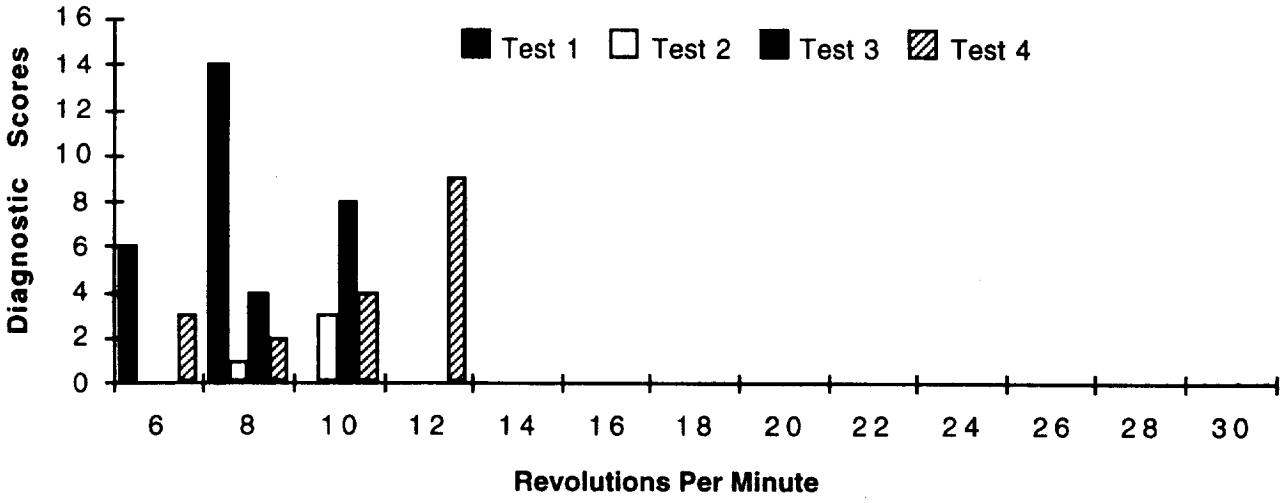
Note: Tests 1–4 were at 1 week intervals. Test 5 was administered approximately 1 year later with tests 5–7, also at 1 week intervals.

Figure A-9. Reports of motion sickness malaise across tests—subject 9.



Note: Number of days between tests 1 and 2 = 189; 2 and 3 = 105; 3 and 4 = 34.

Figure A-10. Reports of motion sickness malaise across tests—subject 10.



Note: Number of days between tests 1 and 2 = 173; 2 and 3 = 125; 3 and 4 = 31.

Figure A-11. Reports of motion sickness malaise across tests—subject 11.

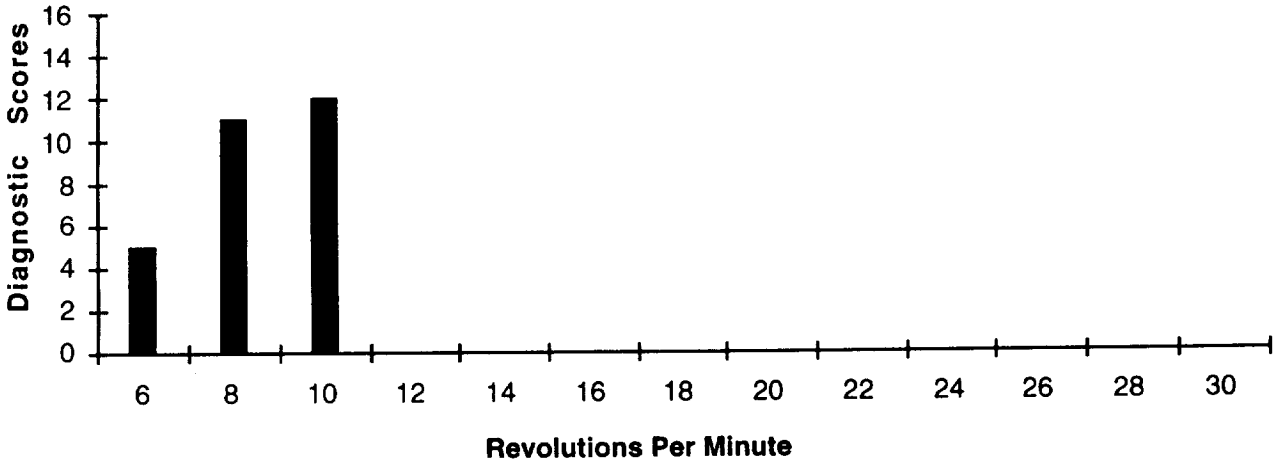


Figure A-12. Reports of malaise during initial motion sickness test—subject 12.

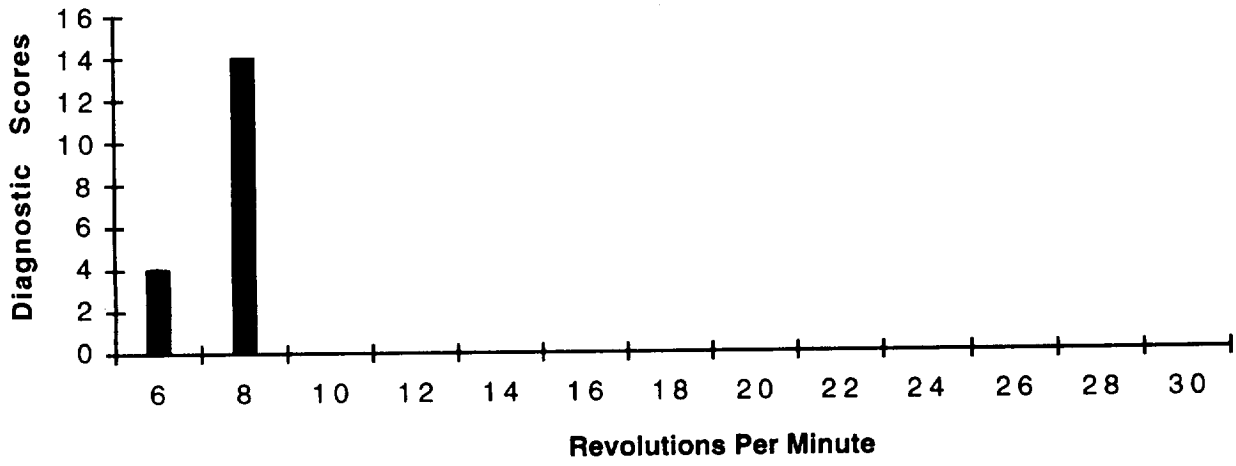
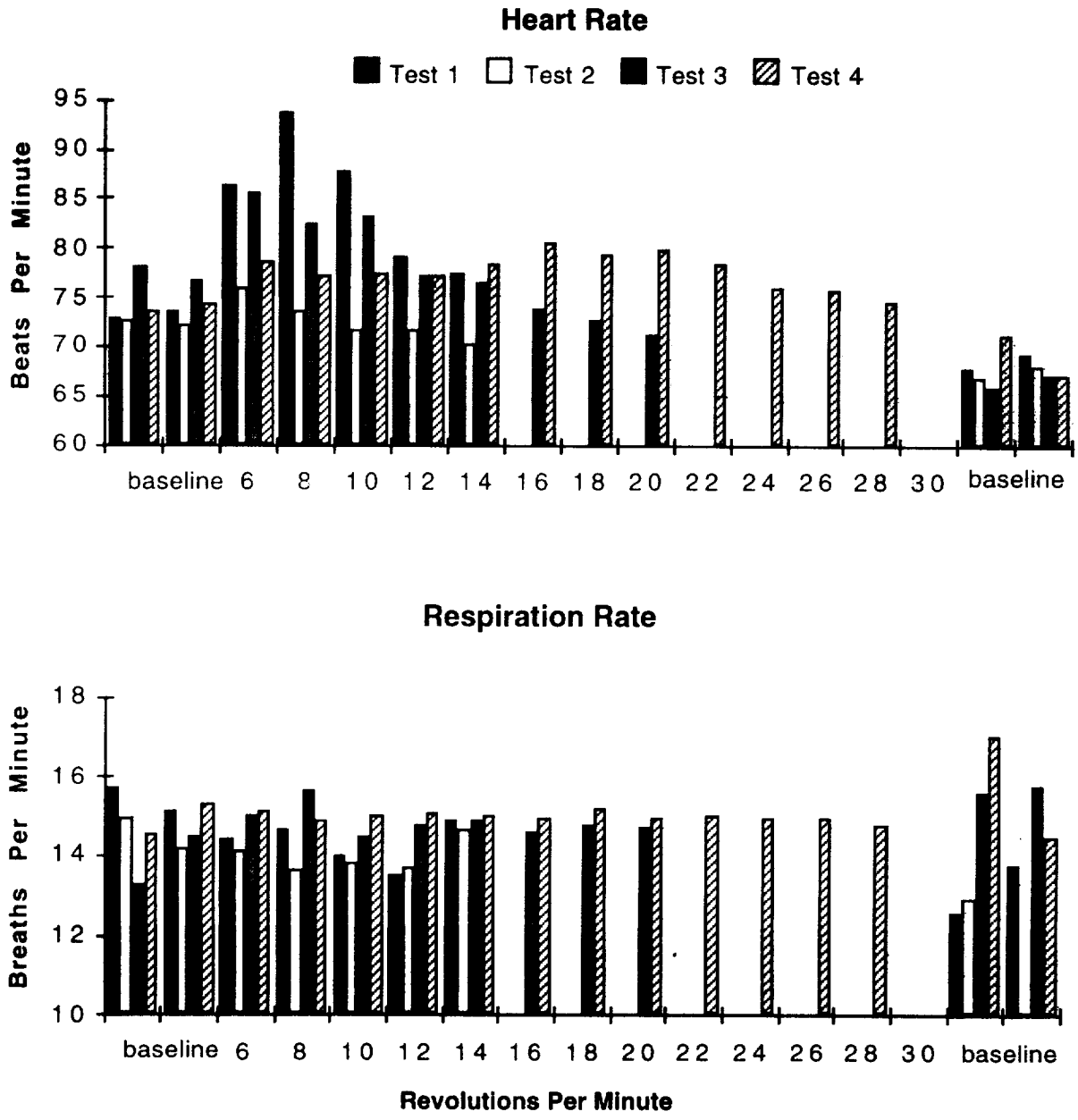
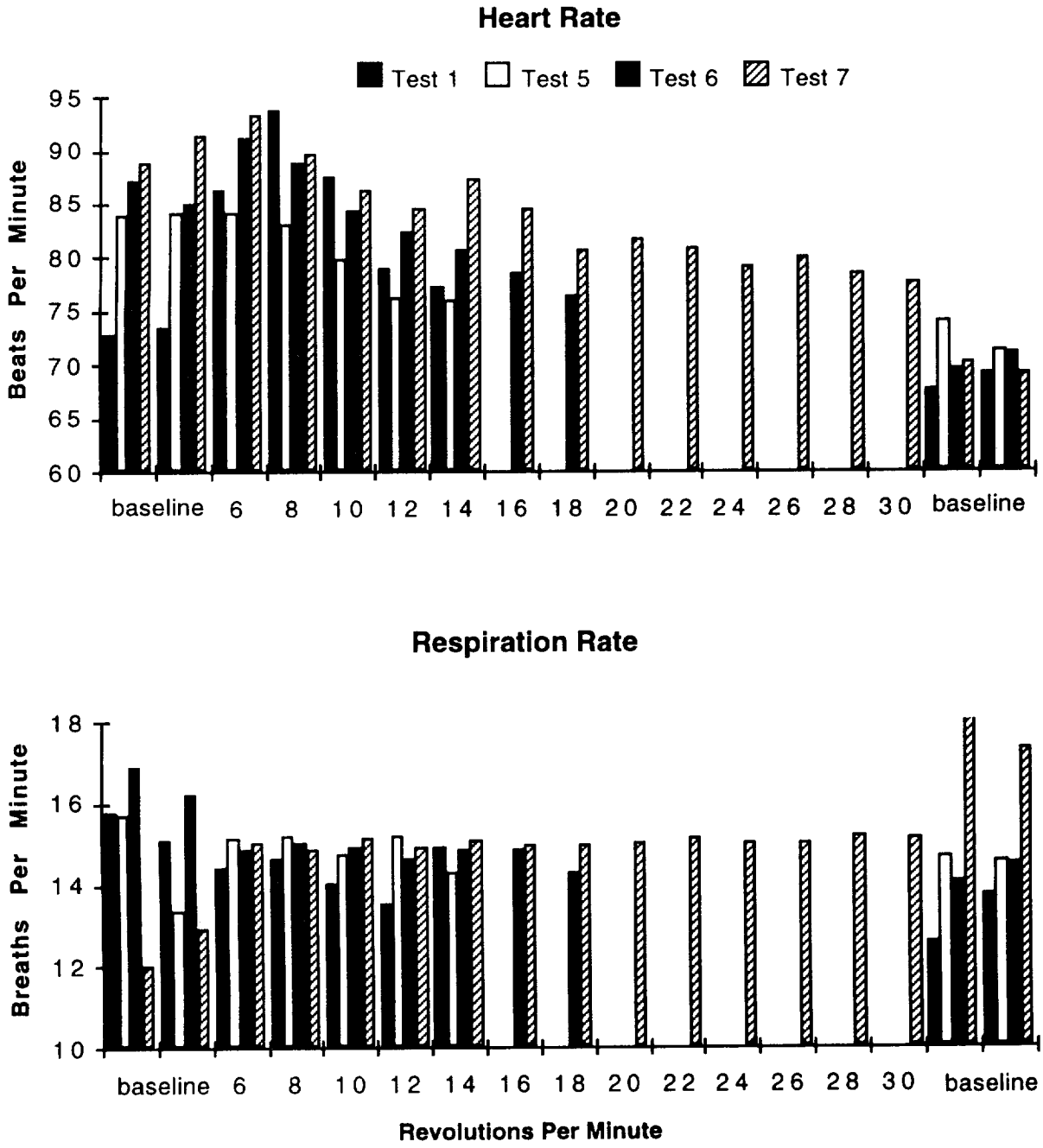


Figure A-13. Reports of malaise during initial motion sickness test—subject 13.



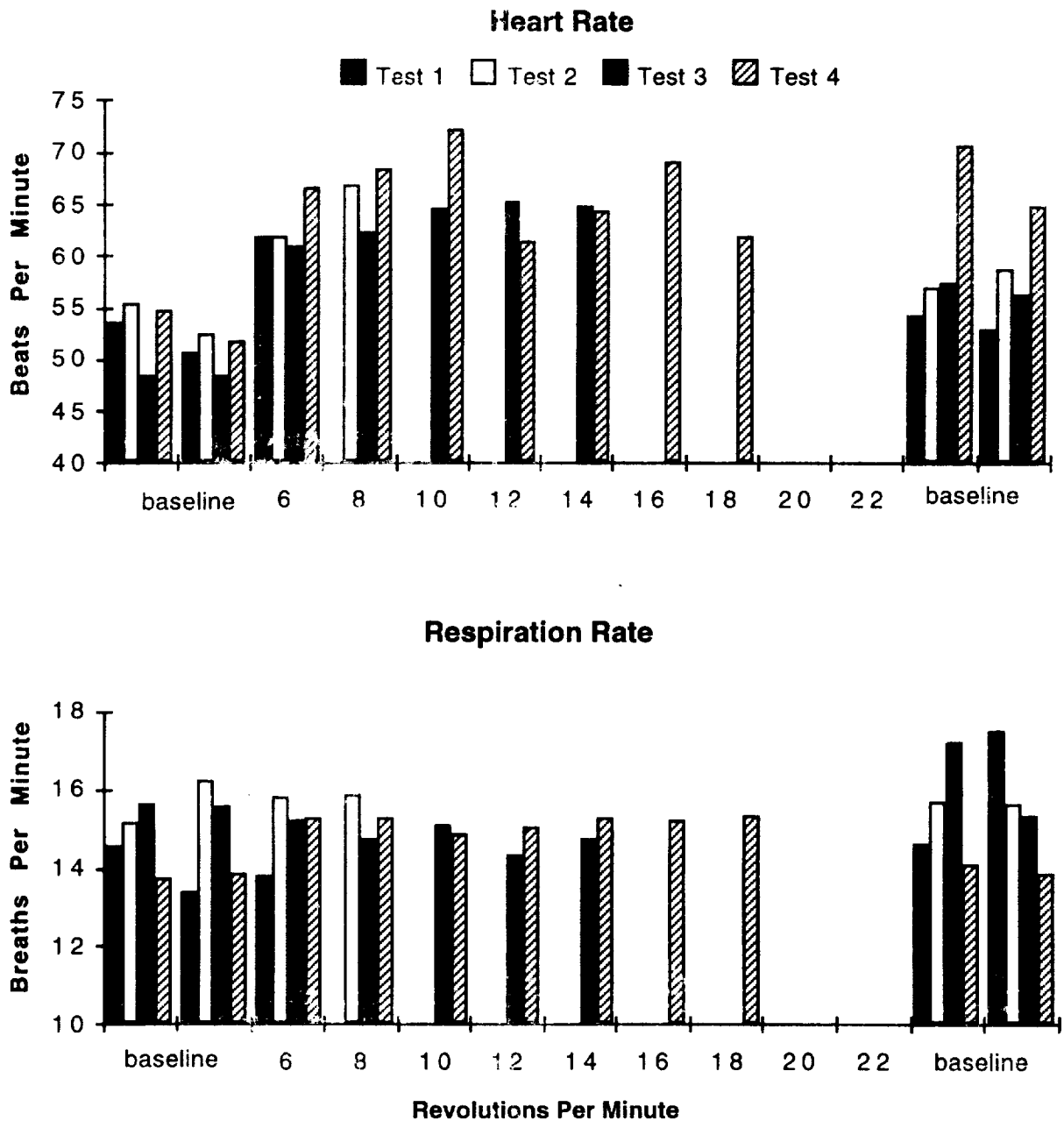
Note: Tests 1–4 were at 1 week intervals.

Figure A-14(a). Heart rate and respiration rate across motion sickness tests (year 1)—subject 1.



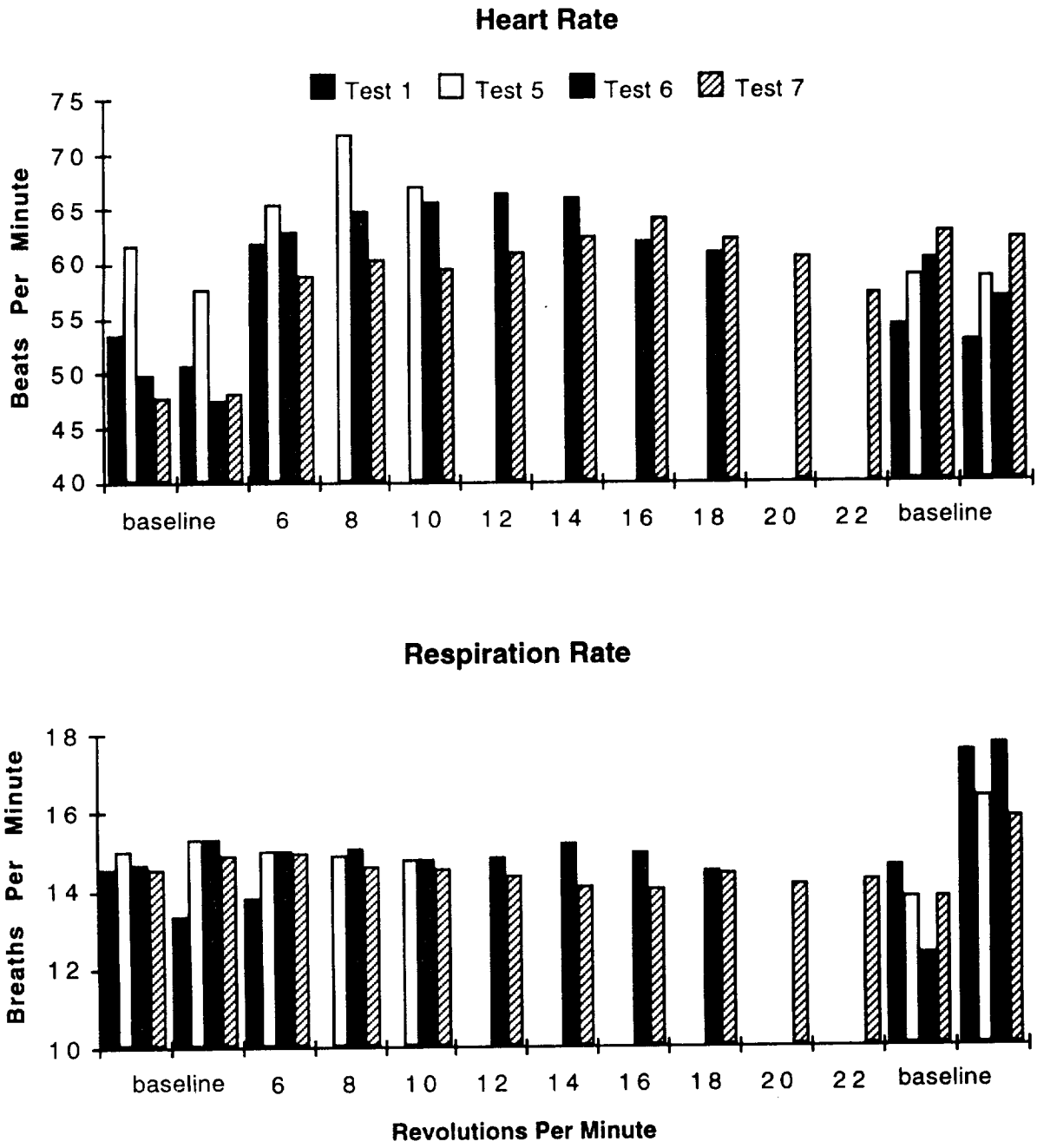
Note: Tests 5–7 were at 1 week intervals.

Figure A-14(b). Heart rate and respiration rate across motion sickness tests (year 2)—subject 1.



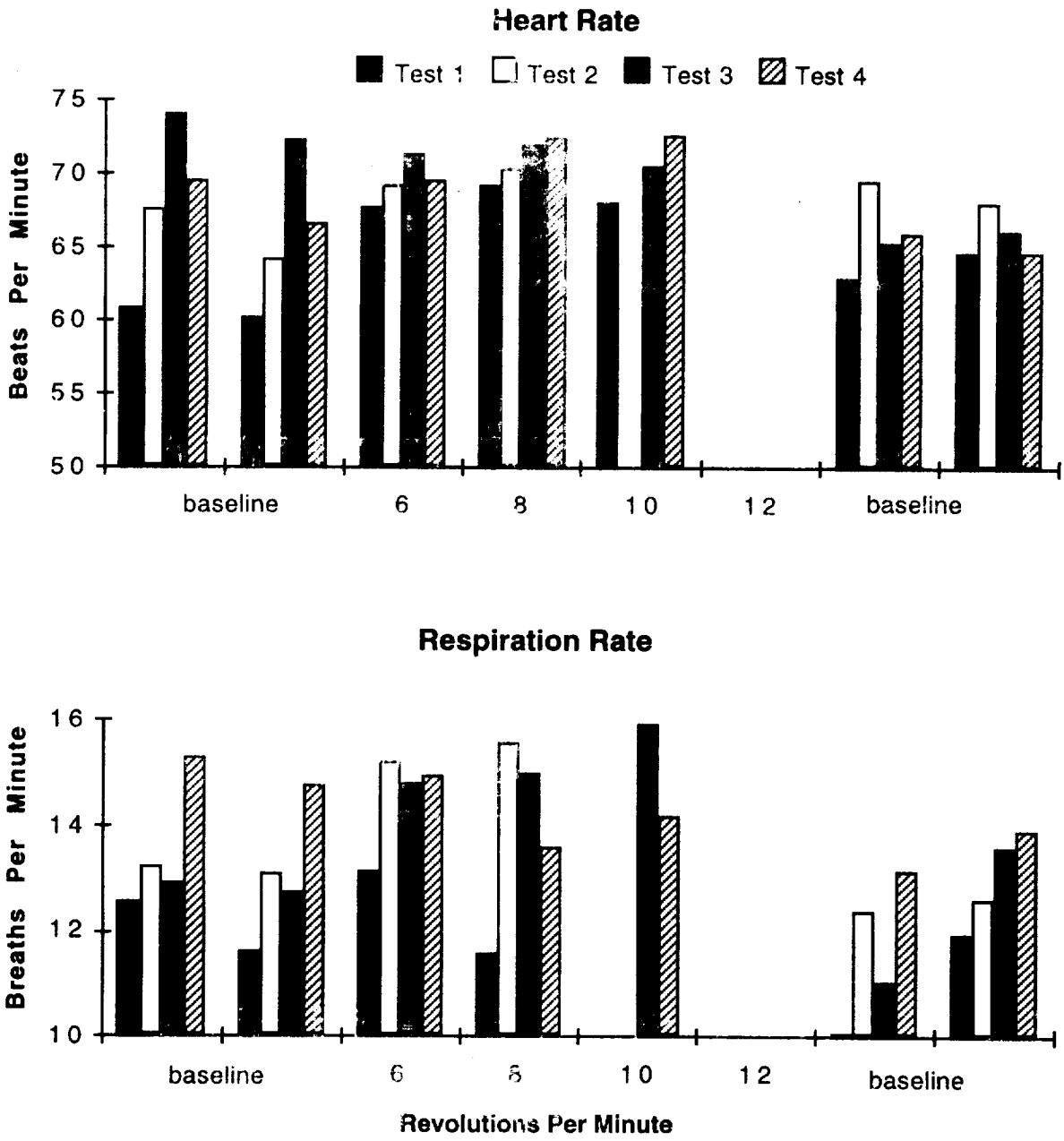
Note: Tests 1–4 were at 1 week intervals.

Figure A-15(a). Heart rate and respiration rate across motion sickness tests (year 1)—subject 2.



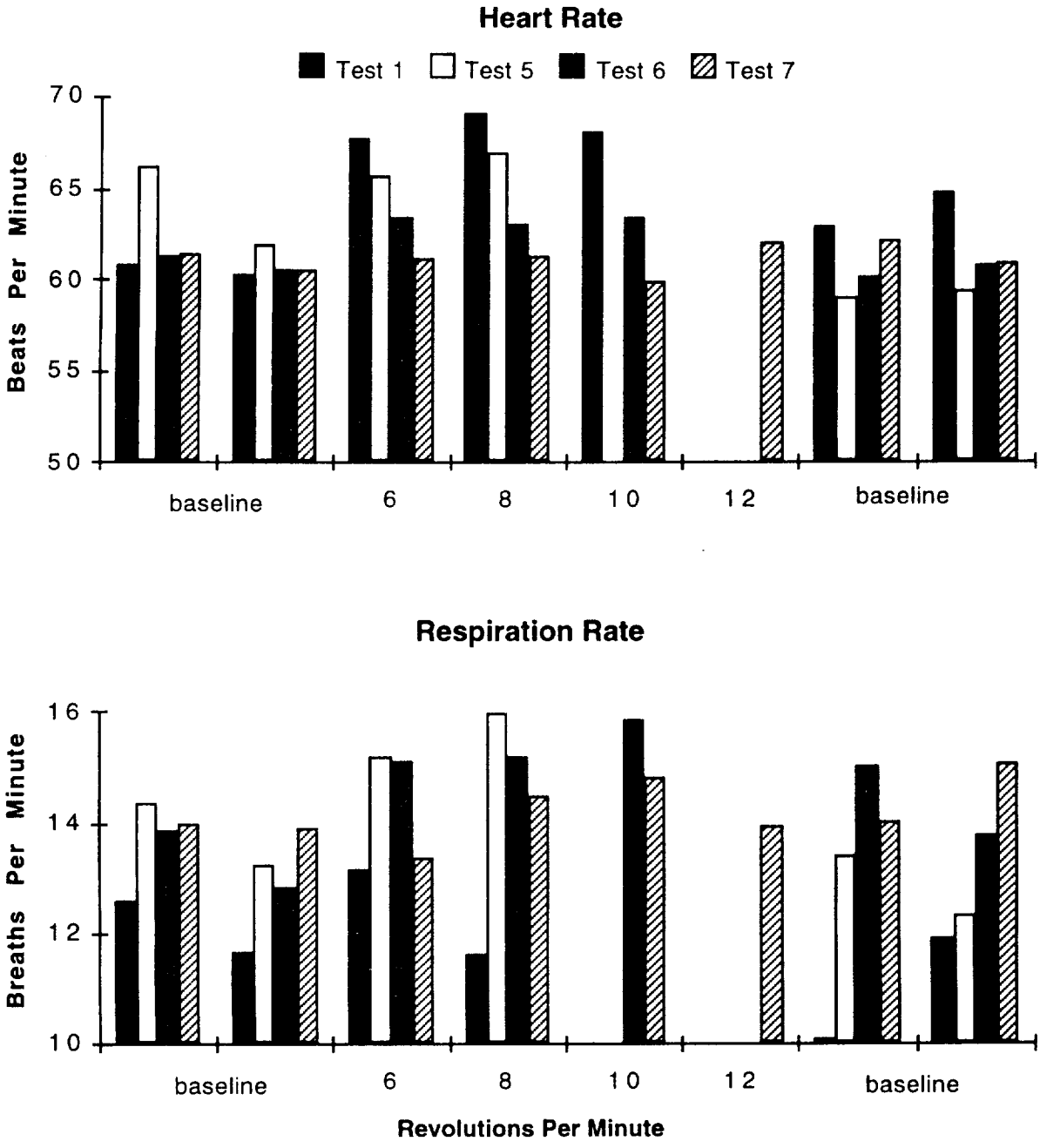
Note: Tests 5–7 were at 1 week intervals.

Figure A-15(b). Heart rate and respiration rate across motion sickness tests (year 2)—subject 2.



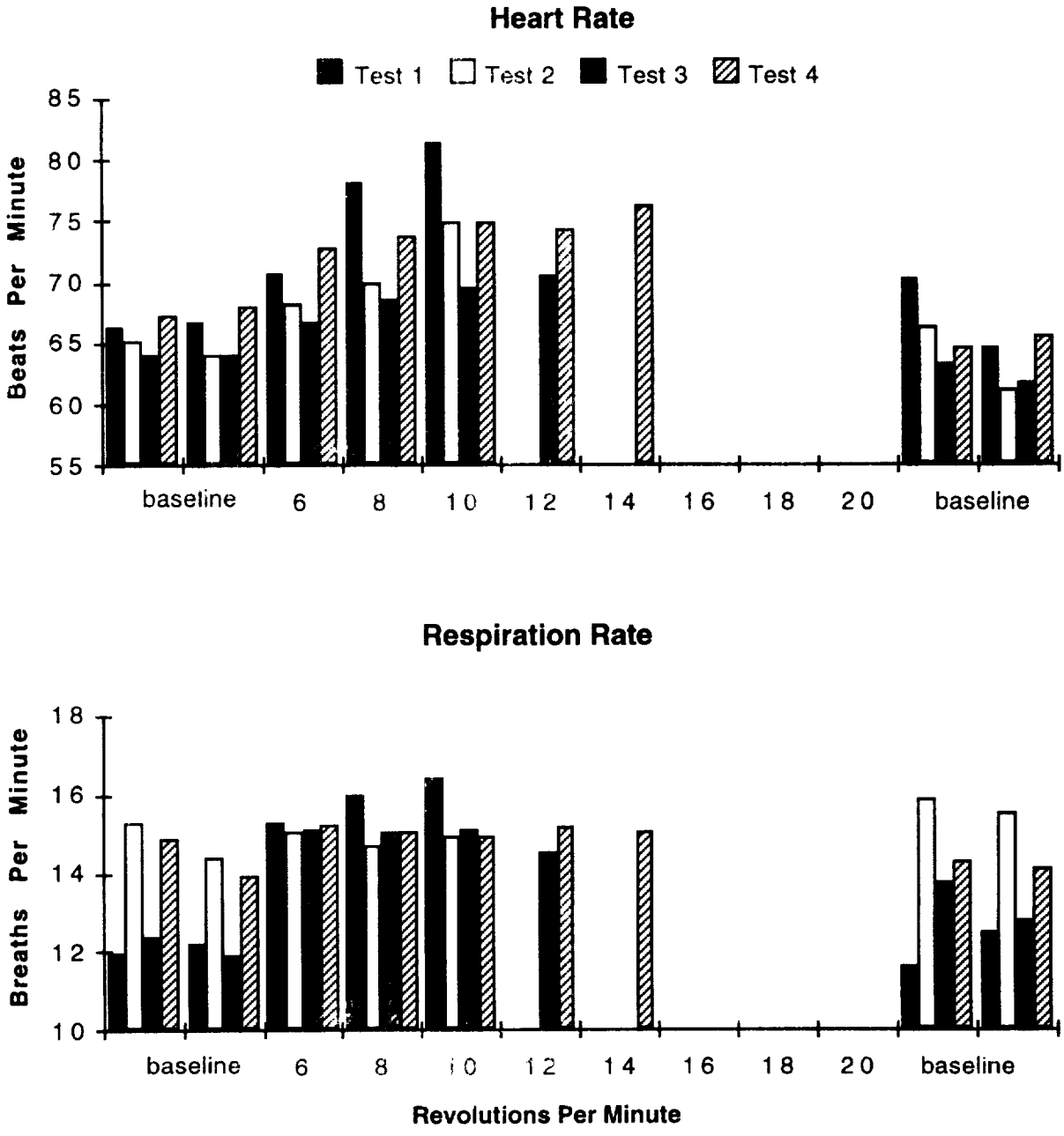
Note: Tests 1–4 were at 1 week intervals.

Figure A-15(a). Heart rate and respiration rate across motion sickness tests (year 1)--subject 3.



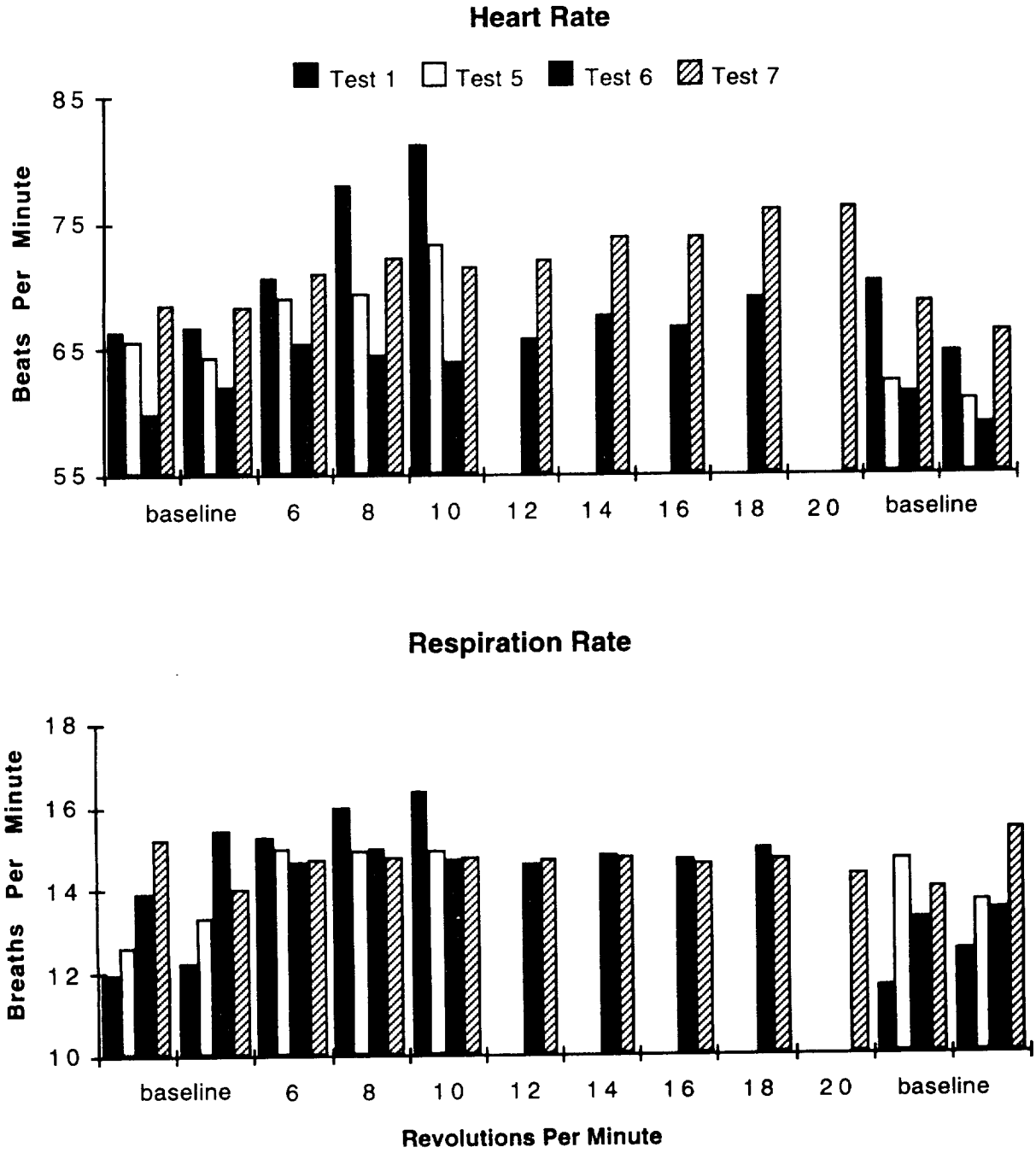
Note: Tests 5–7 were at 1 week intervals.

Figure A-16(b). Heart rate and respiration rate across motion sickness tests (year 2)—subject 3.



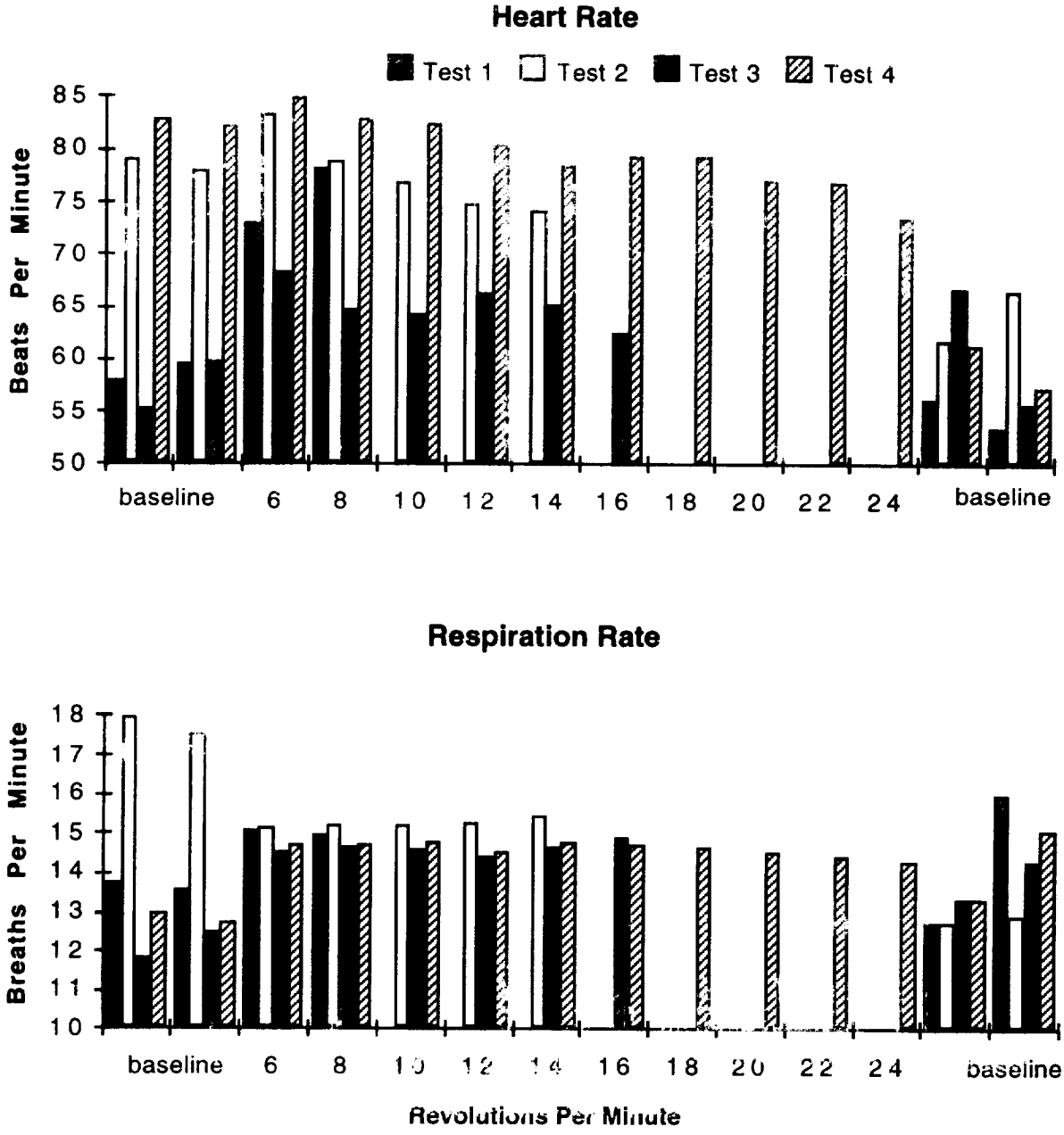
Note: Tests 1–4 were at 1 week intervals.

Figure A-17(a). Heart rate and respiration rate across motion sickness tests (year 1)—subject 4.



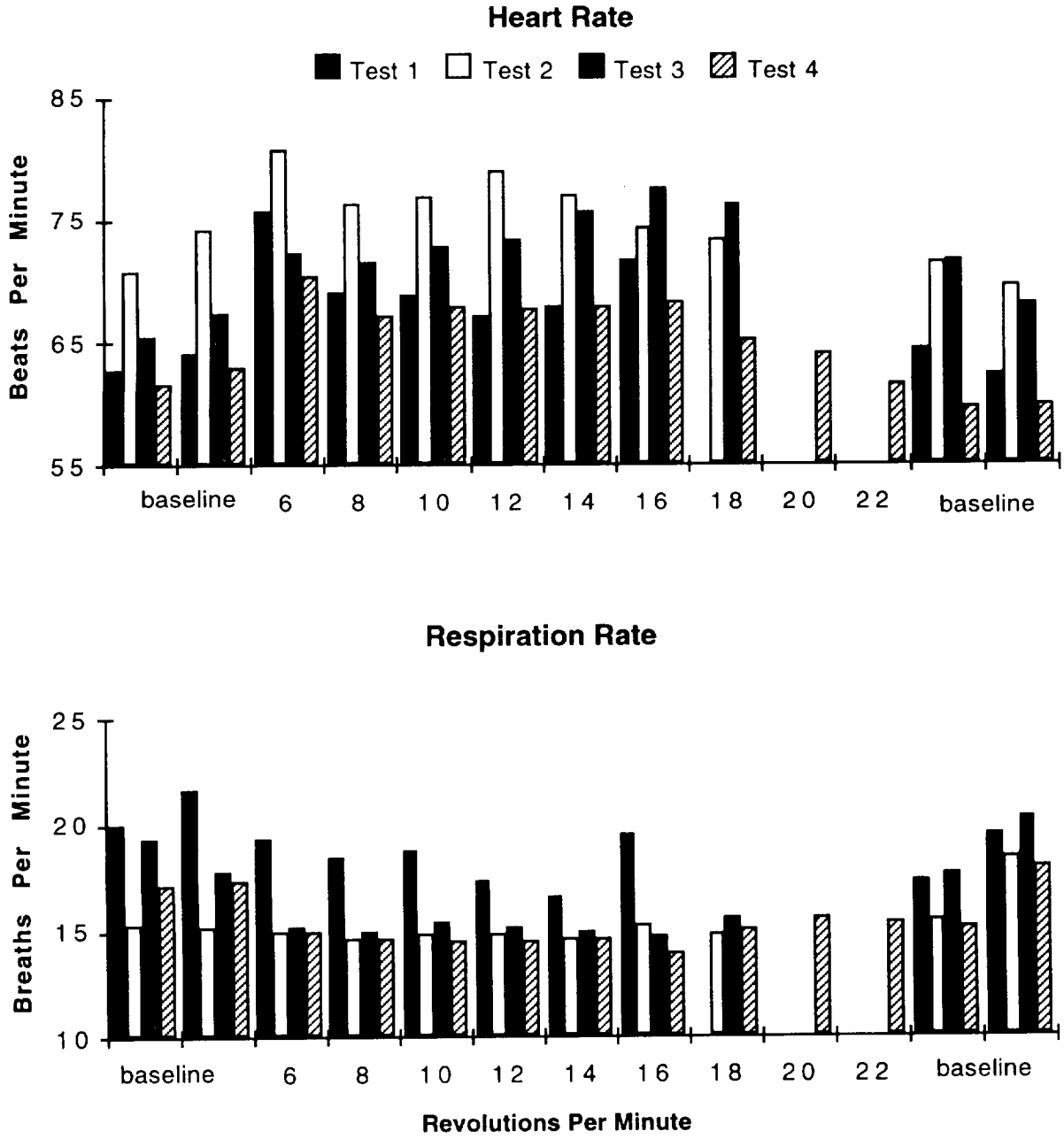
Note: Tests 5–7 were at 1 week intervals.

Figure A-17(b). Heart rate and respiration rate across motion sickness tests (year 2)—subject 4.



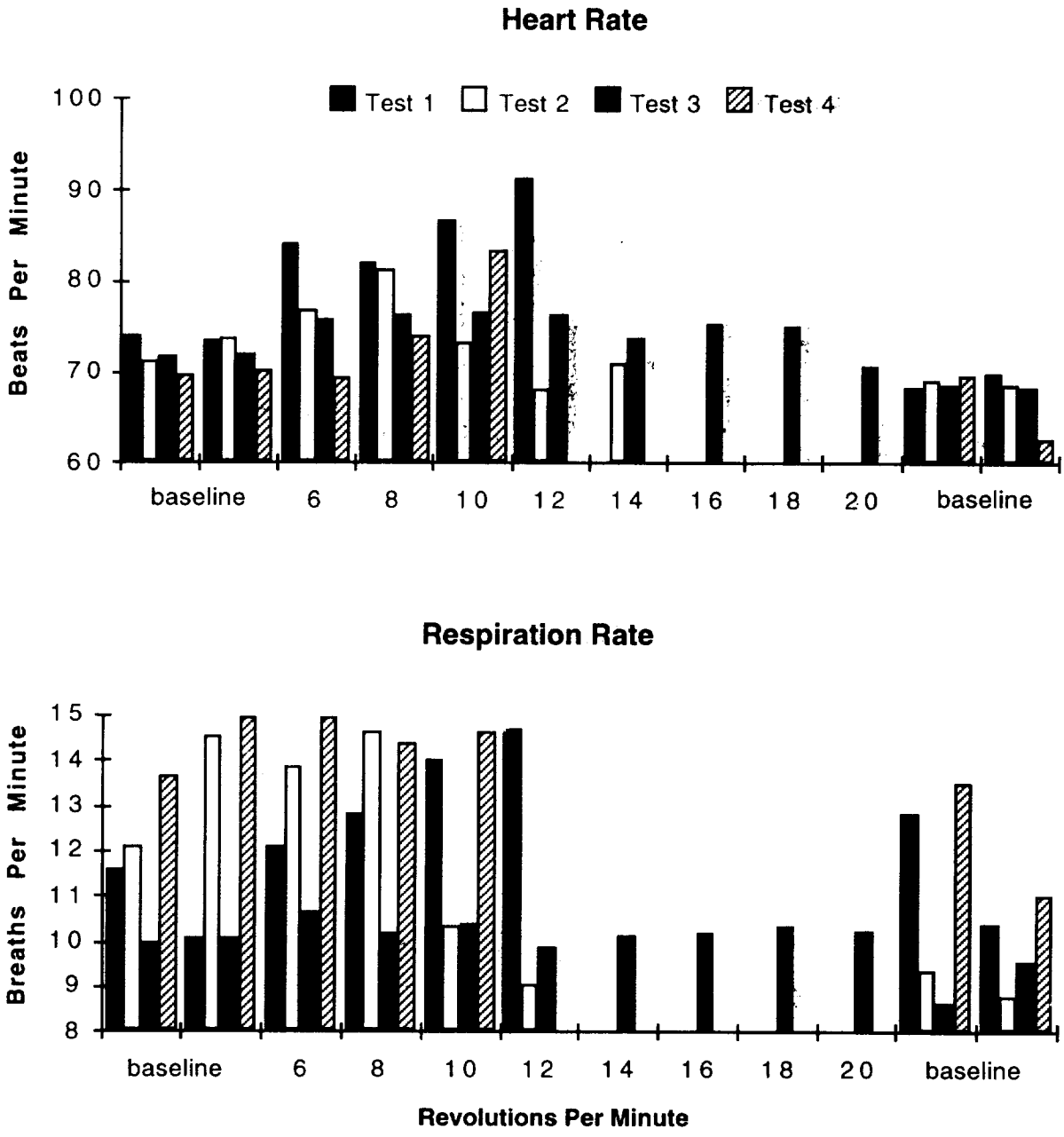
Note: Tests were conducted at weekly intervals.

Figure A-18. Heart rate and respiration rate changes across motion sickness tests—subject 5.



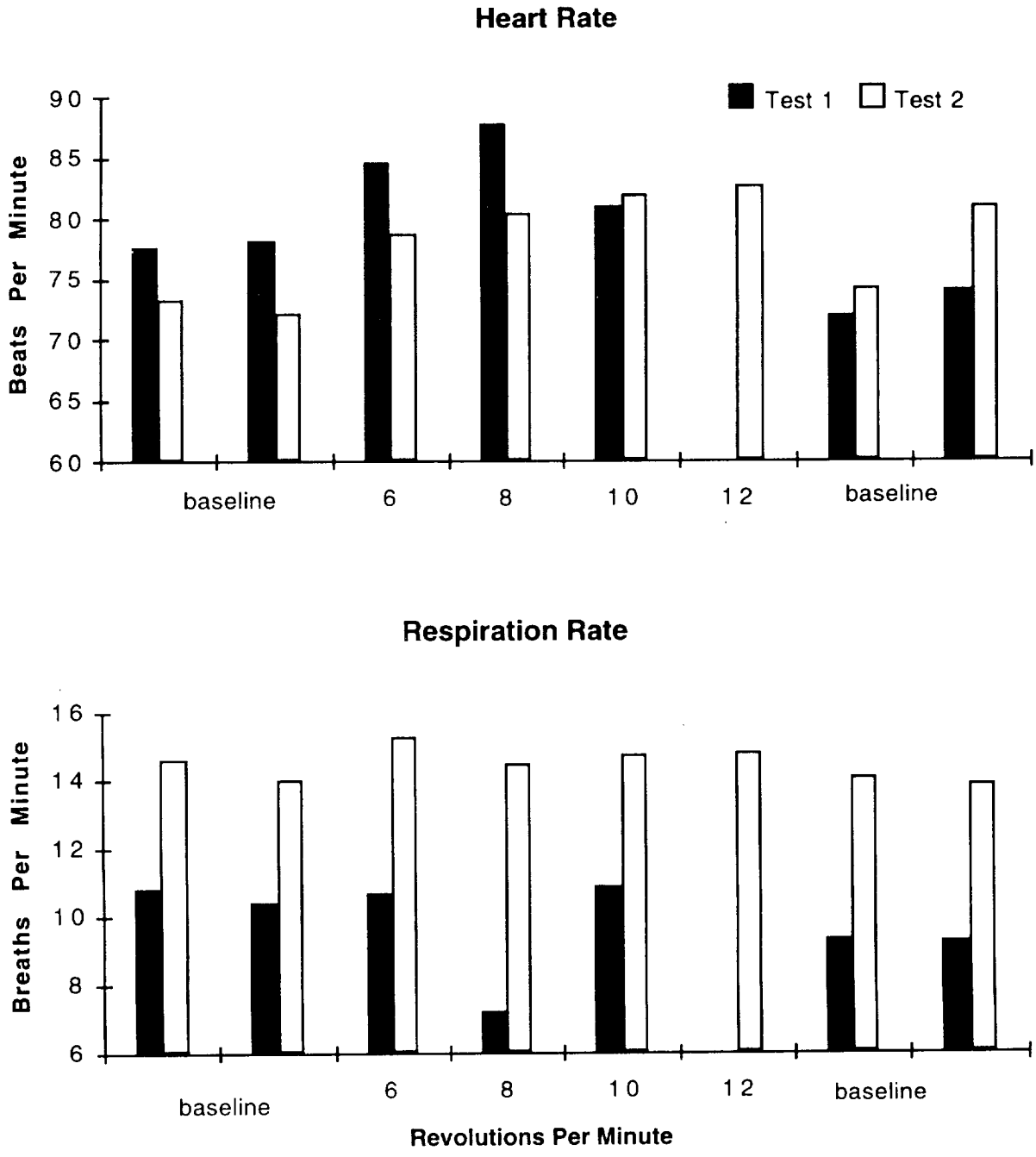
Note: Number of days between tests 1 and 2 = 234; 2 and 3 = 74; 3 and 4 = 54.

Figure A-19. Heart rate and respiration rate changes across motion sickness tests—subject 6.



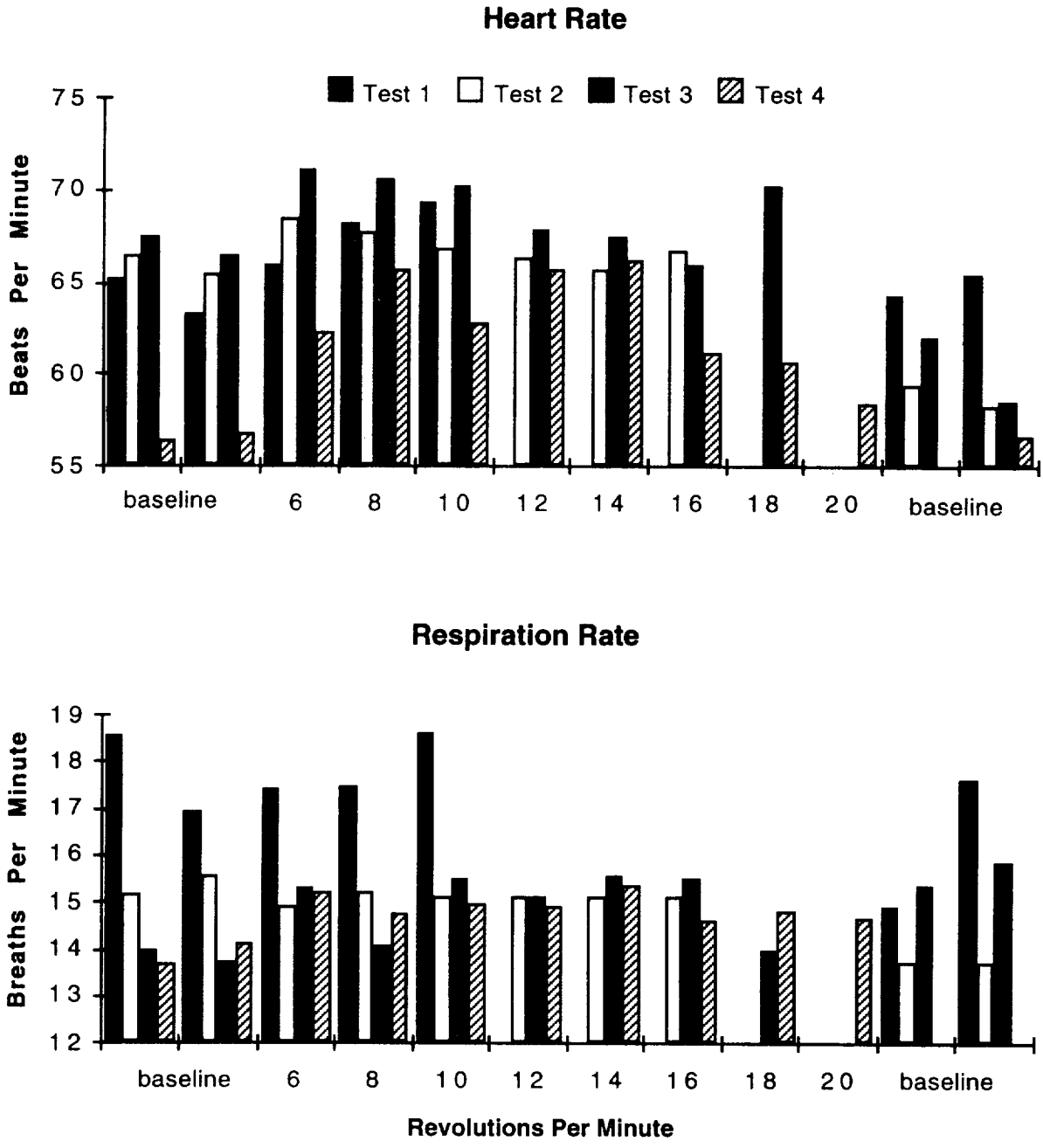
Note: Number of days between tests 1 and 2 = 201; 2 and 3 = 102; 3 and 4 = 57.

Figure A-20. Heart rate and respiration rate changes across motion sickness tests—subject 7.



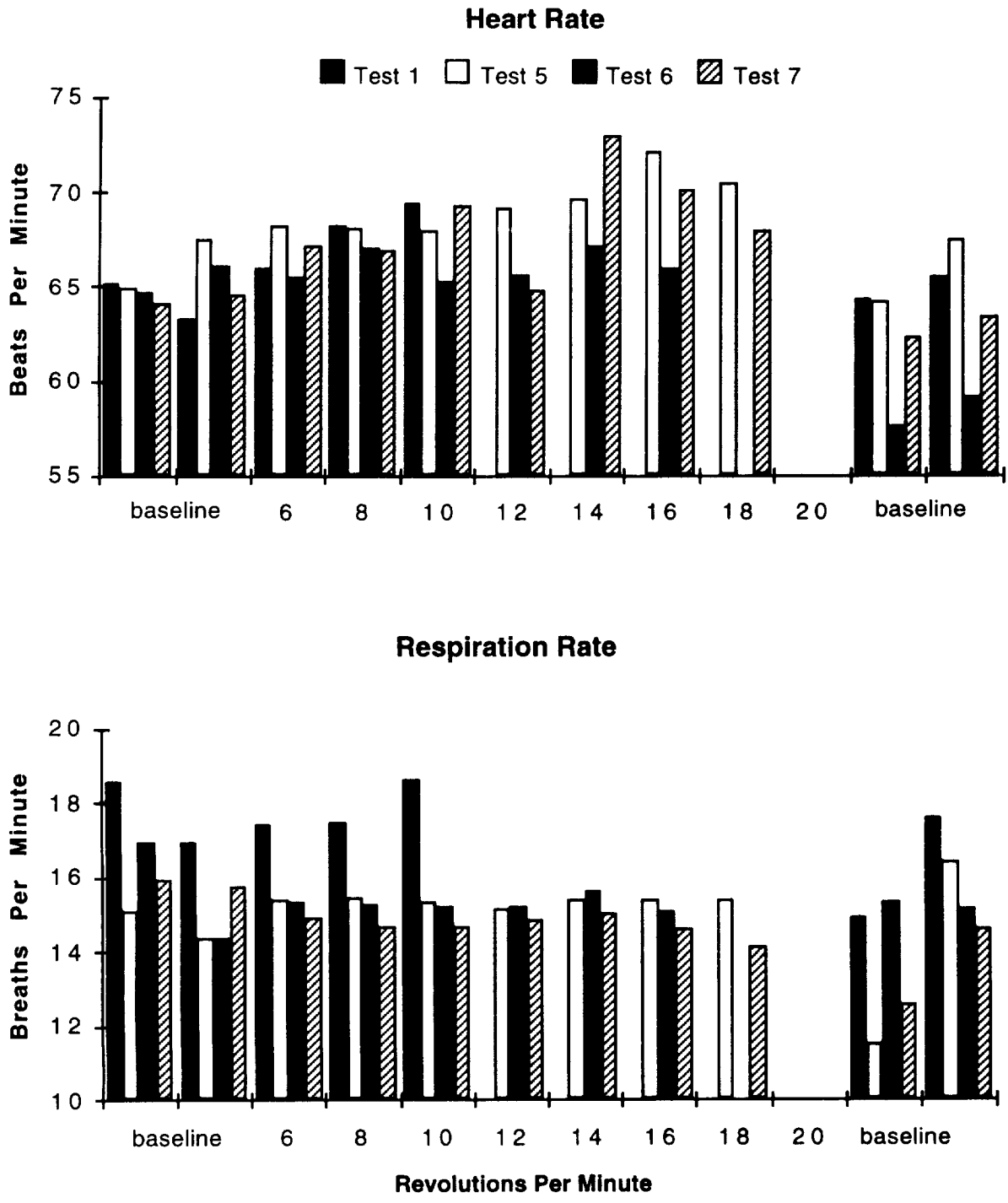
Note: There was a 1 year interval between these "baseline" rotating chair tests.

Figure A-21. Heart rate and respiration rate during initial motion sickness tests—subject 8.



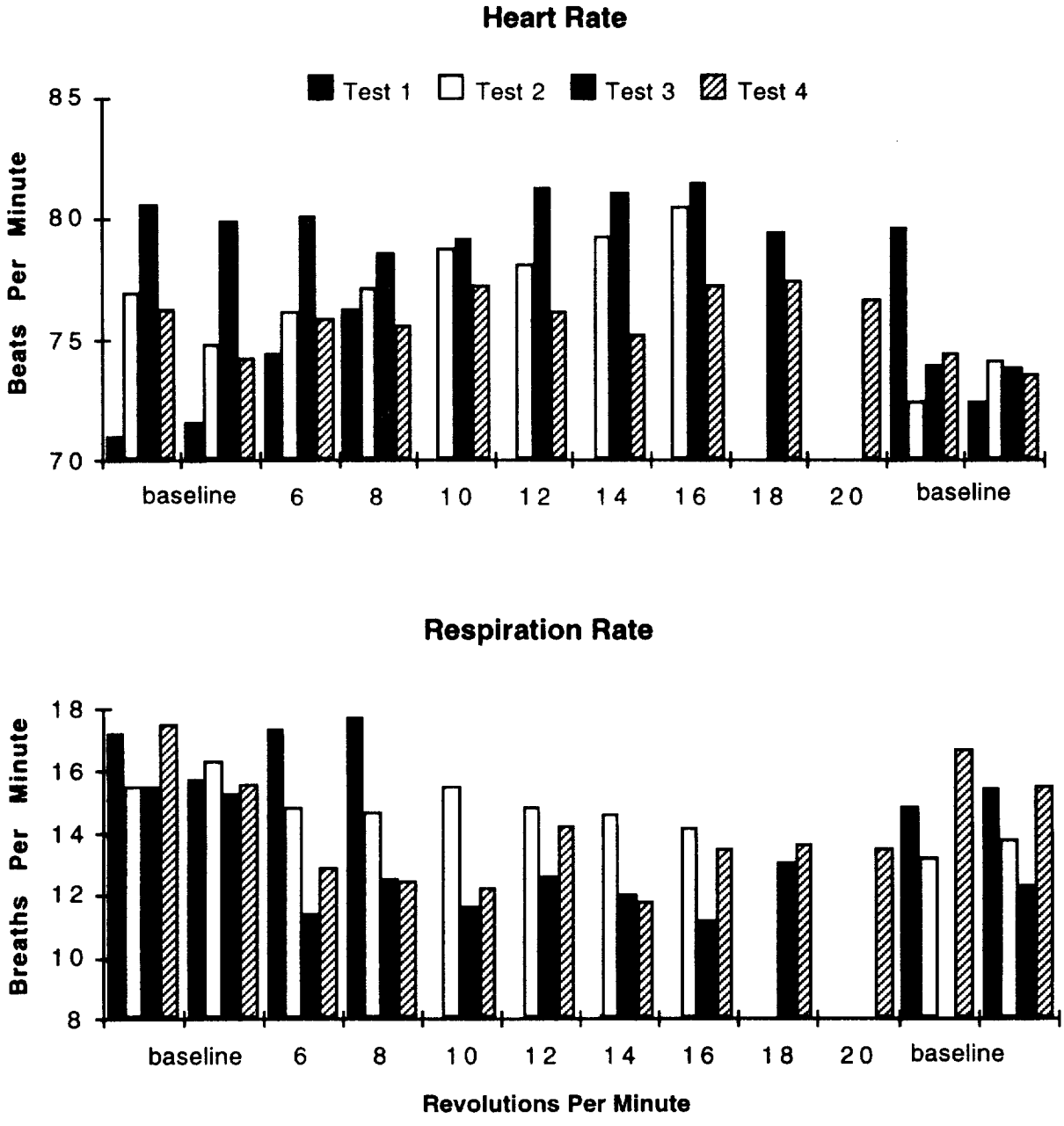
Note: Tests 1–4 were at 1 week intervals.

Figure A-22(a). Heart rate and respiration rate across motion sickness tests (year 1)—subject 9.



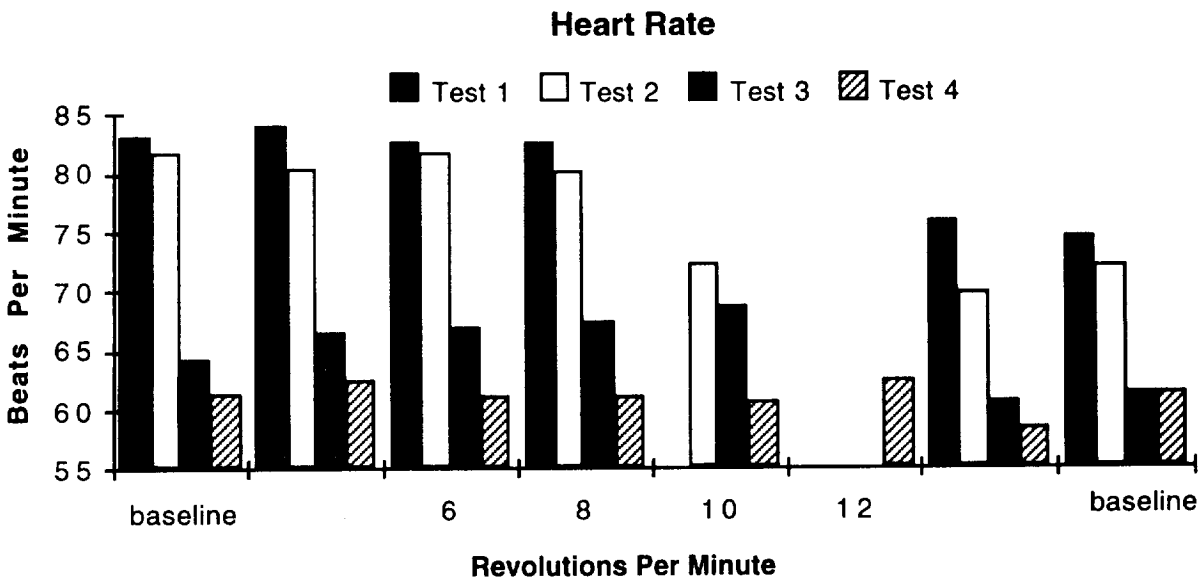
Note: Tests 5–7 were at 1 week intervals.

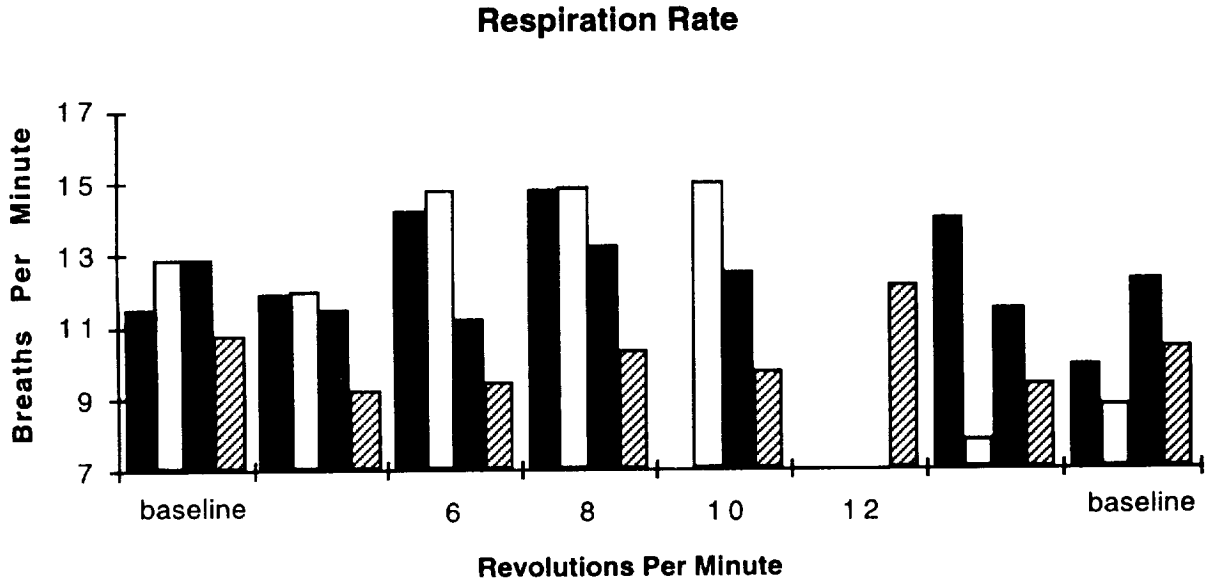
Figure A-22(b). Heart rate and respiration rate across motion sickness tests (year 2)—subject 9.



Note: Number of days between tests 1 and 2 = 189; 2 and 3 = 105; 3 and 4 = 34.

Figure A-23. Heart rate and respiration rate changes across motion sickness tests—subject 10.





Note: Number of days between tests 1 and 2 = 173; 2 and 3 = 125; 3 and 4 = 31.

Figure A-24. Heart rate and respiration rate changes across motion sickness tests—subject 11.

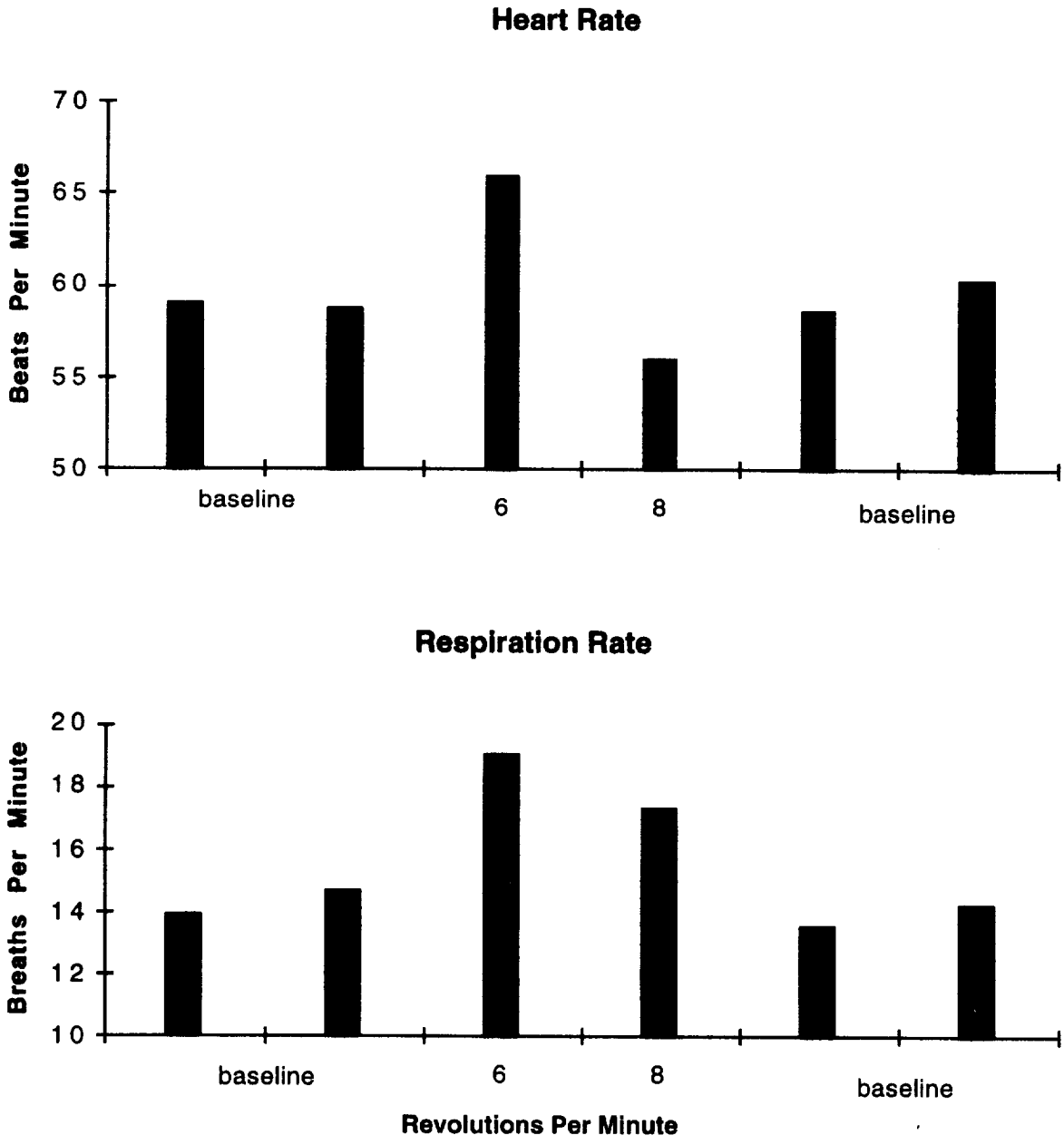


Figure A-25. Heart rate and respiration rate during initial motion sickness test—subject 12.

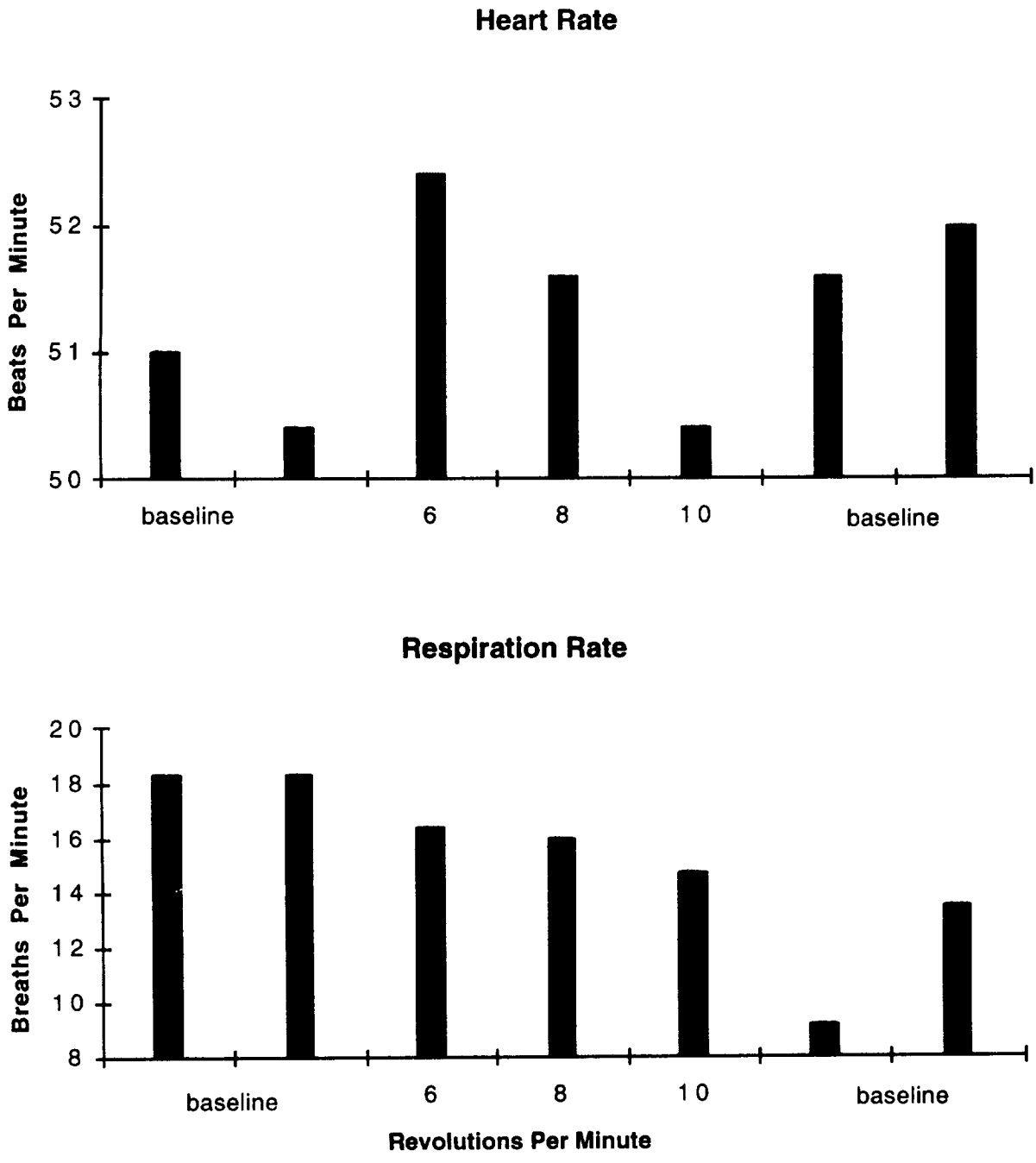
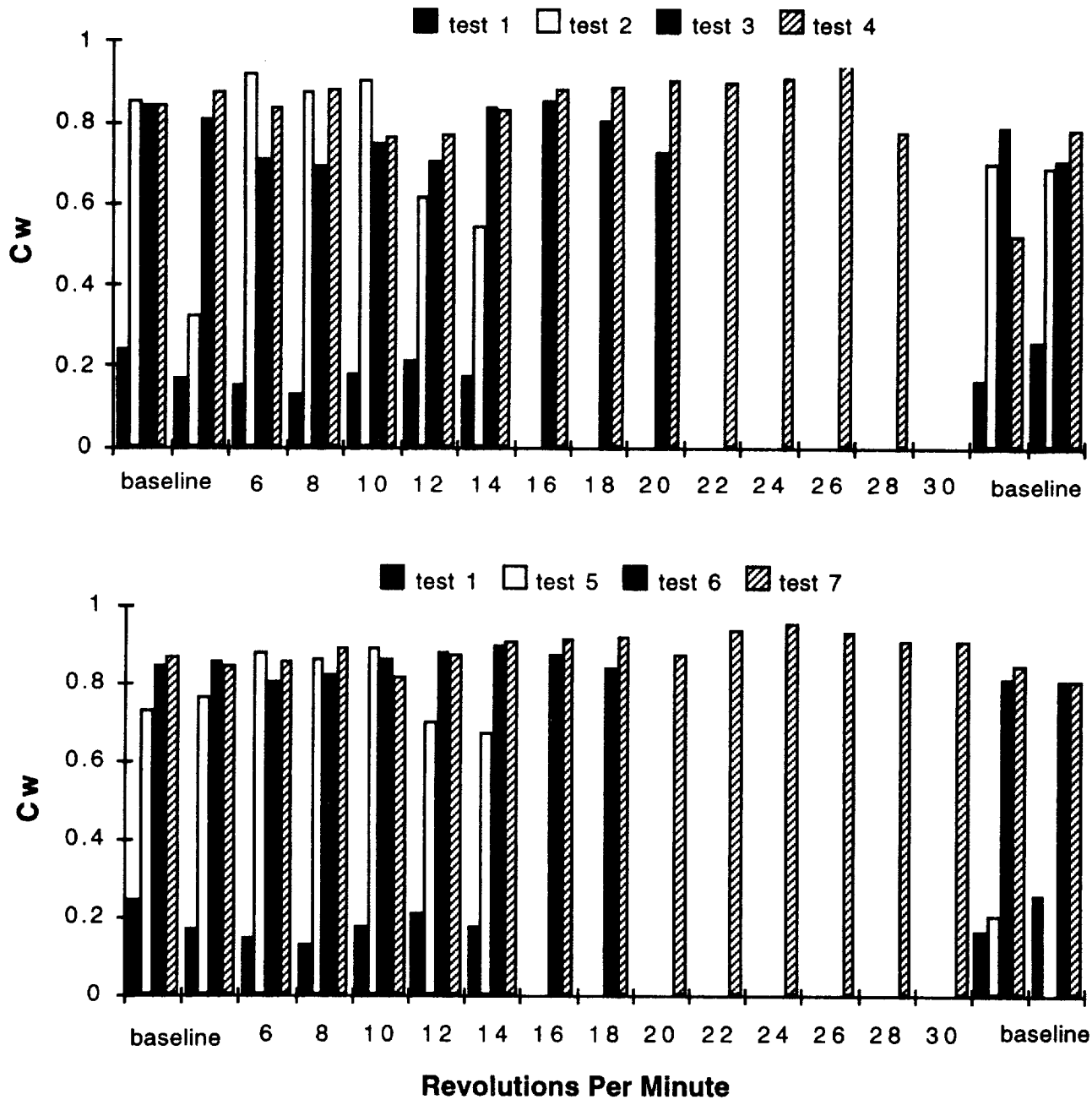
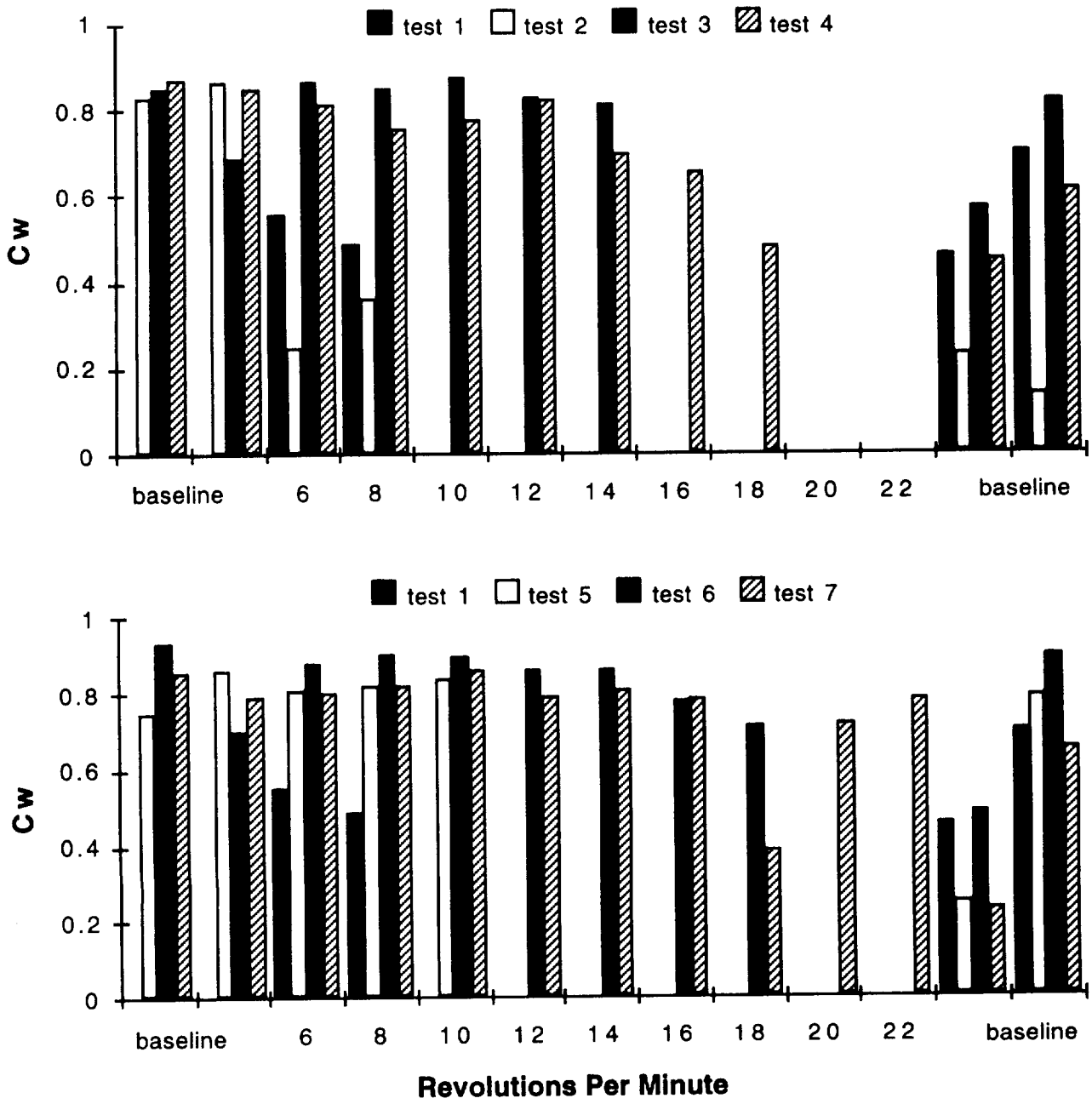


Figure A-26. Heart rate and respiration rate during initial motion sickness test—subject 13.



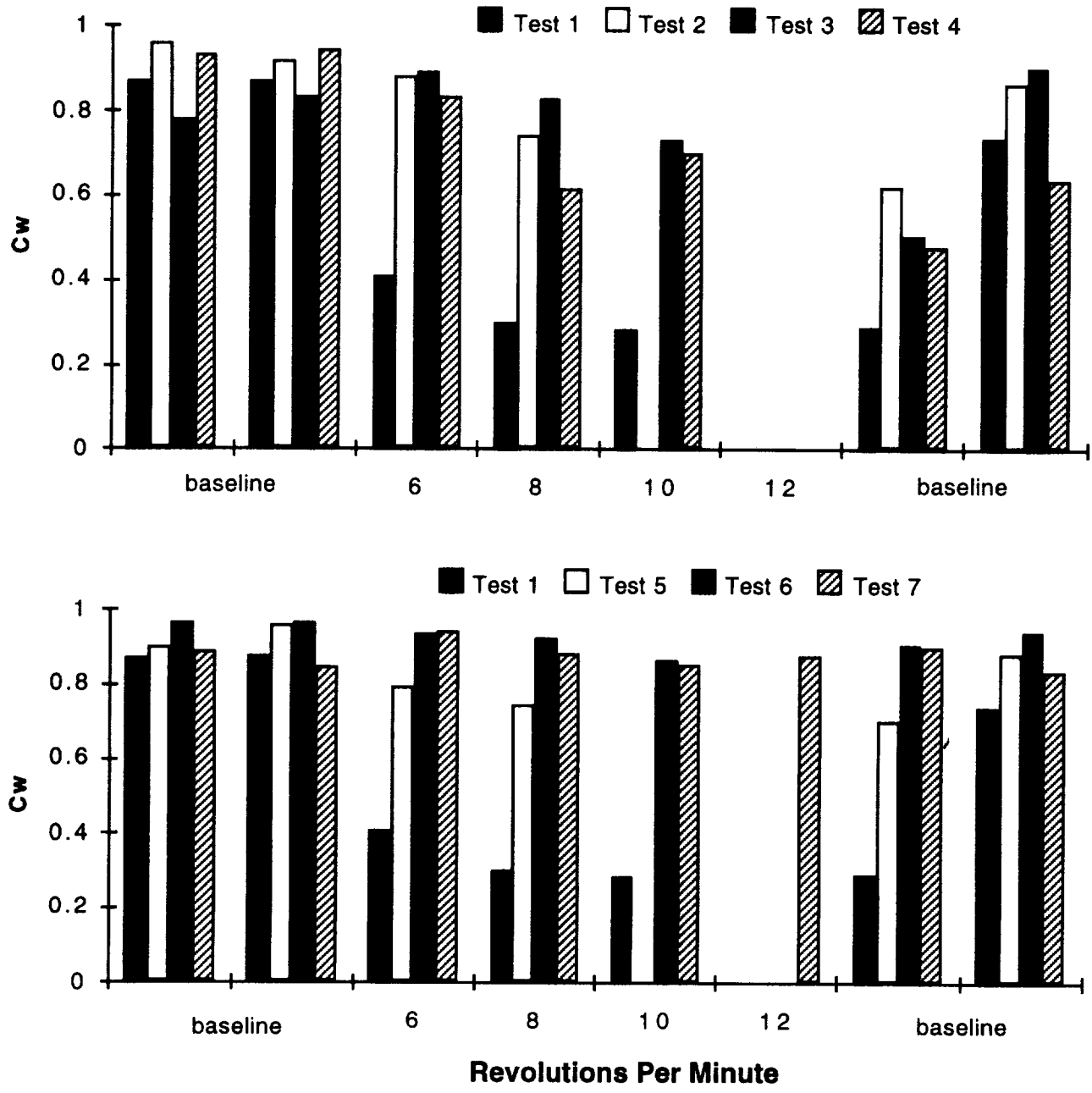
Note: Tests 1–4 were at 1 week intervals. Test 5 was administered approximately 1 year later with tests 5–7, also at 1 week intervals.

Figure A-27. Changes in coherence between heart rate and respiration across tests—subject 1.



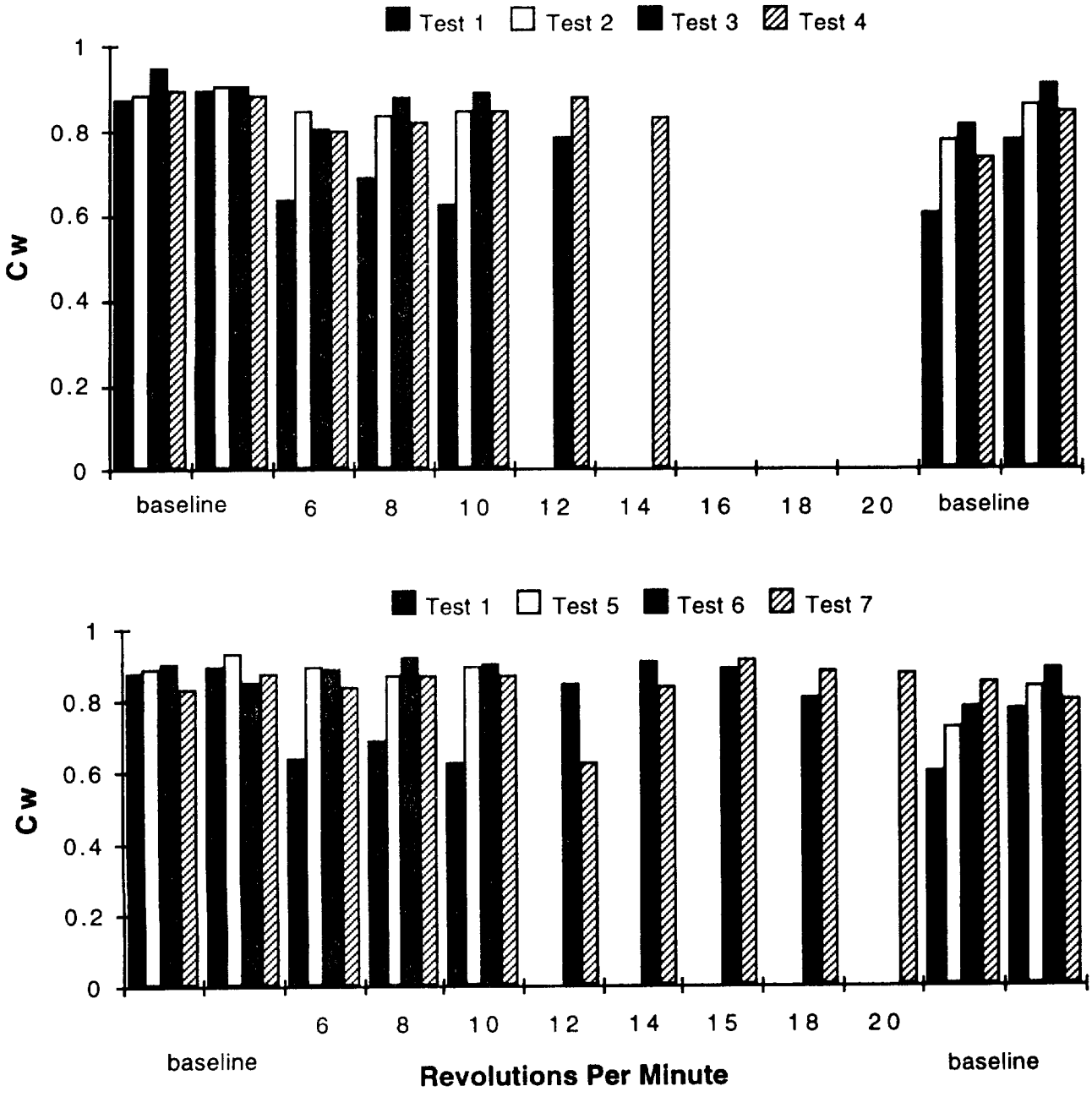
Note: Tests 1–4 were at 1 week intervals. Test 5 was administered approximately 1 year later with tests 5–7, also at 1 week intervals.

Figure A-28. Changes in coherence between heart rate and respiration across tests—subject 2.



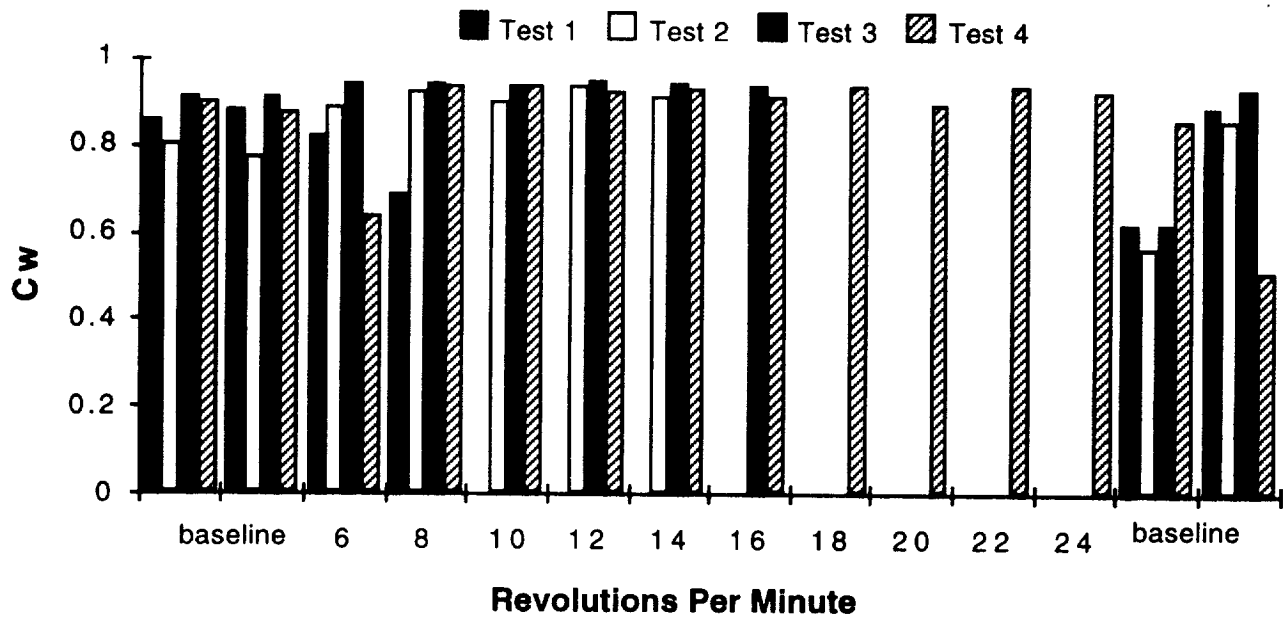
Note: Tests 1–4 were at 1 week intervals. Test 5 was administered approximately 1 year later with tests 5–7, also at 1 week intervals.

Figure A-29. Changes in coherence between heart rate and respiration across tests—subject 3.



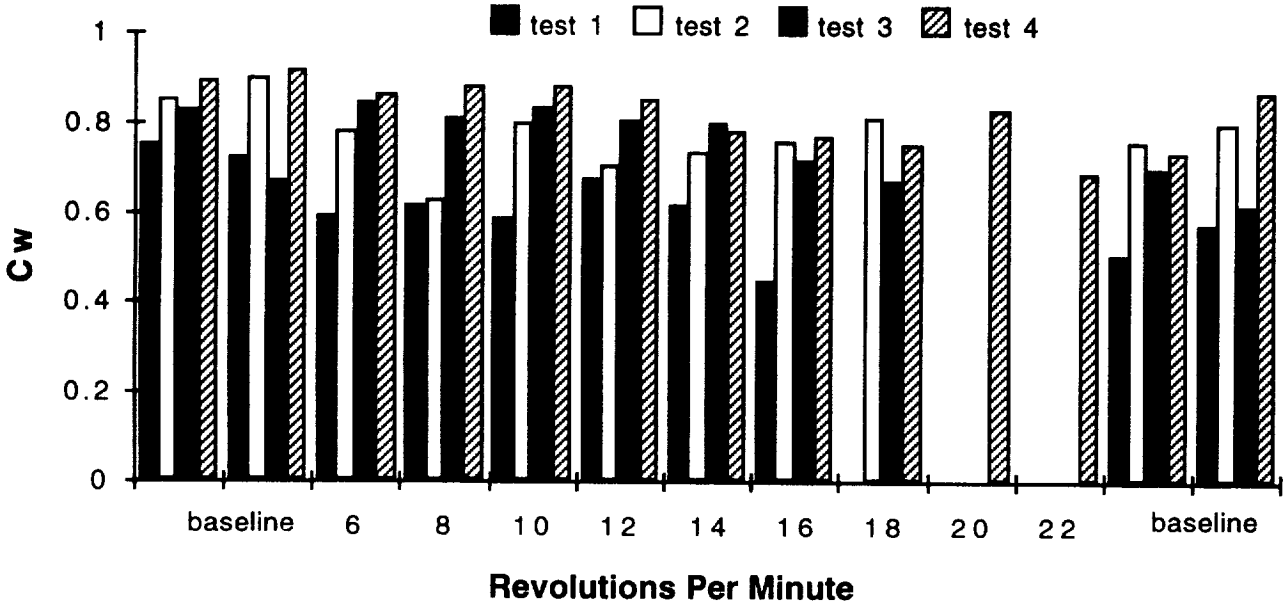
Note: Tests 1–4 were at 1 week intervals. Test 5 was administered approximately 1 year later with tests 5–7, also at 1 week intervals.

Figure A-30. Changes in coherence between heart rate and respiration across tests—subject 4.



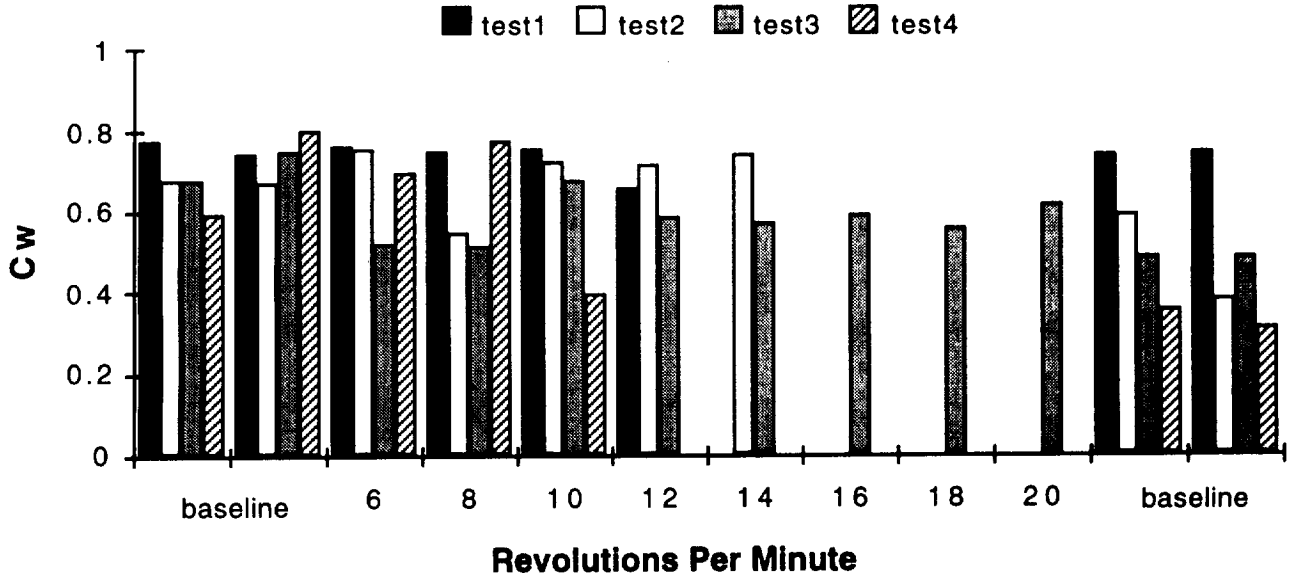
Note: Tests were conducted at weekly intervals.

Figure A-31. Changes in coherence between heart rate and respiration across tests—subject 5.



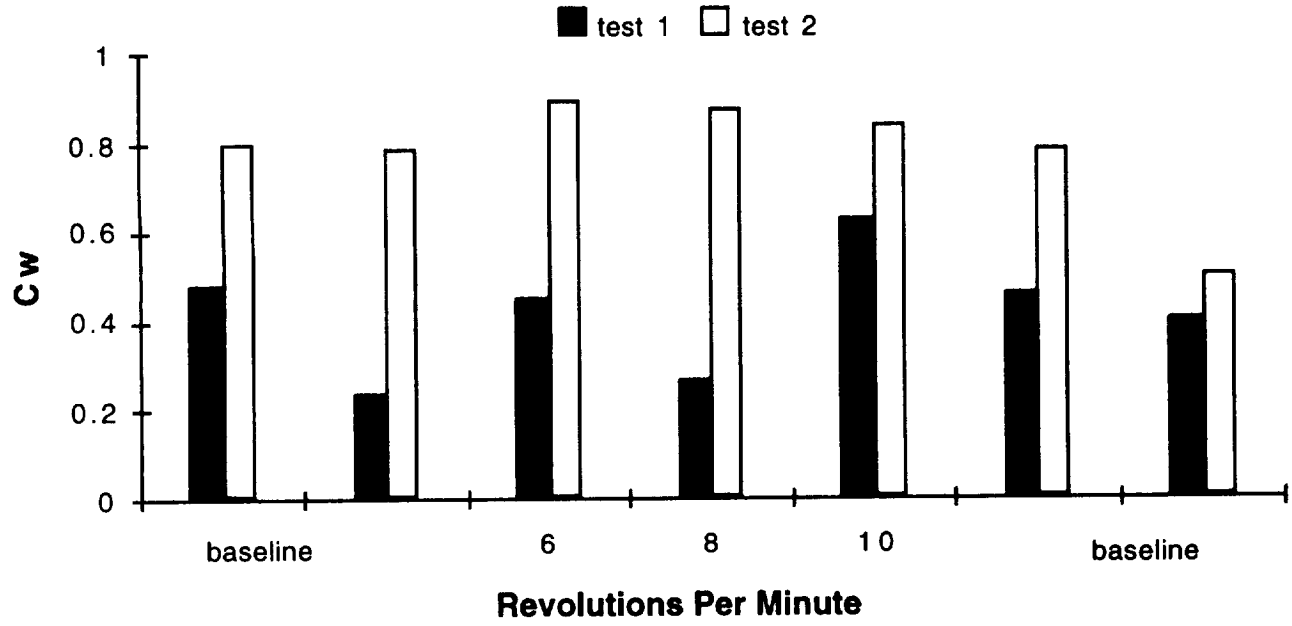
Note: Number of days between tests 1 and 2 = 234; 2 and 3 = 74; 3 and 4 = 54.

Figure A-32. Changes in coherence between heart rate and respiration across tests—subject 6.



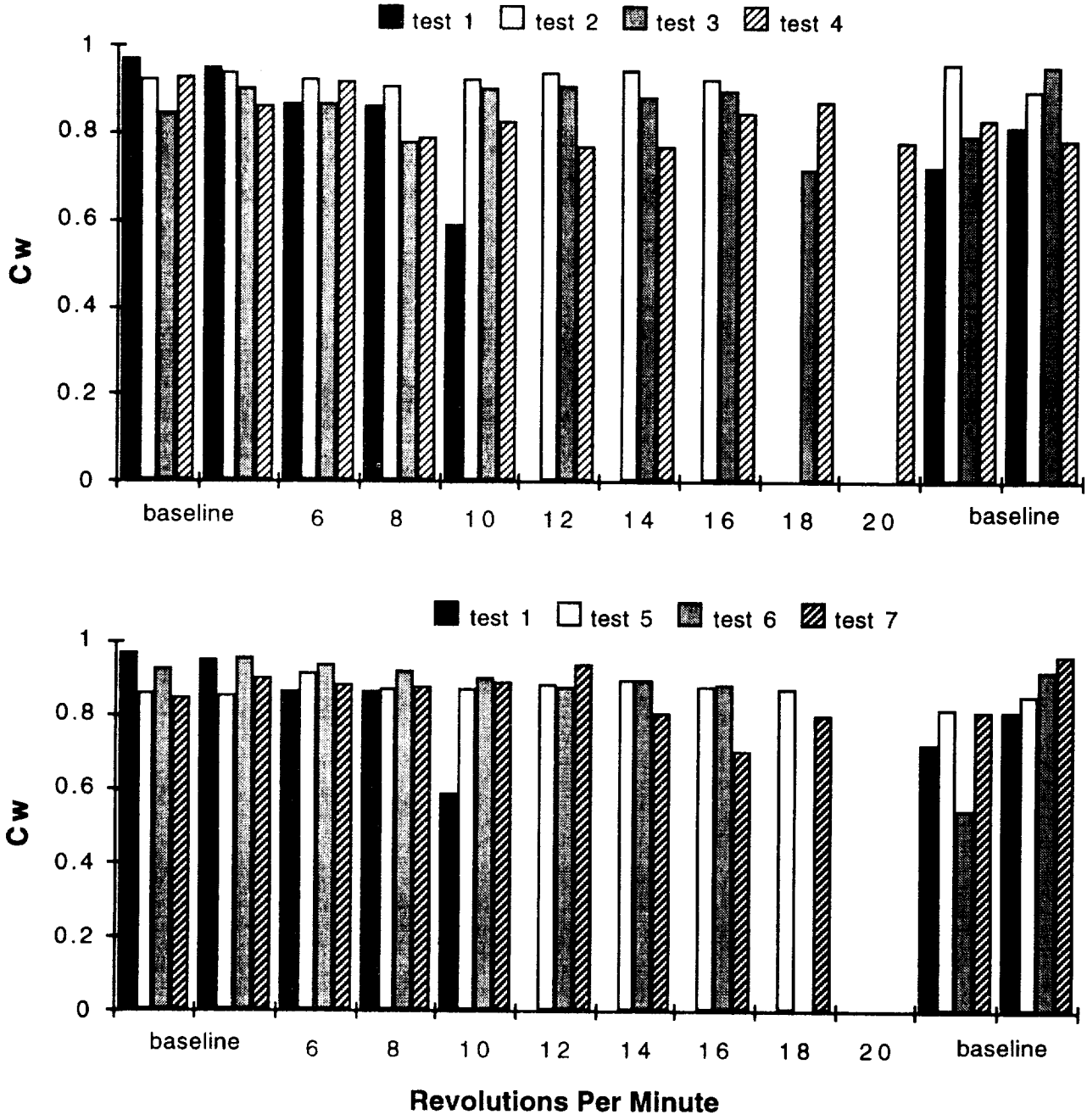
Note: Number of days between tests 1 and 2 = 201; 2 and 3 = 102; 3 and 4 = 57.

Figure A-33. Changes in coherence between heart rate and respiration across tests—subject 7.



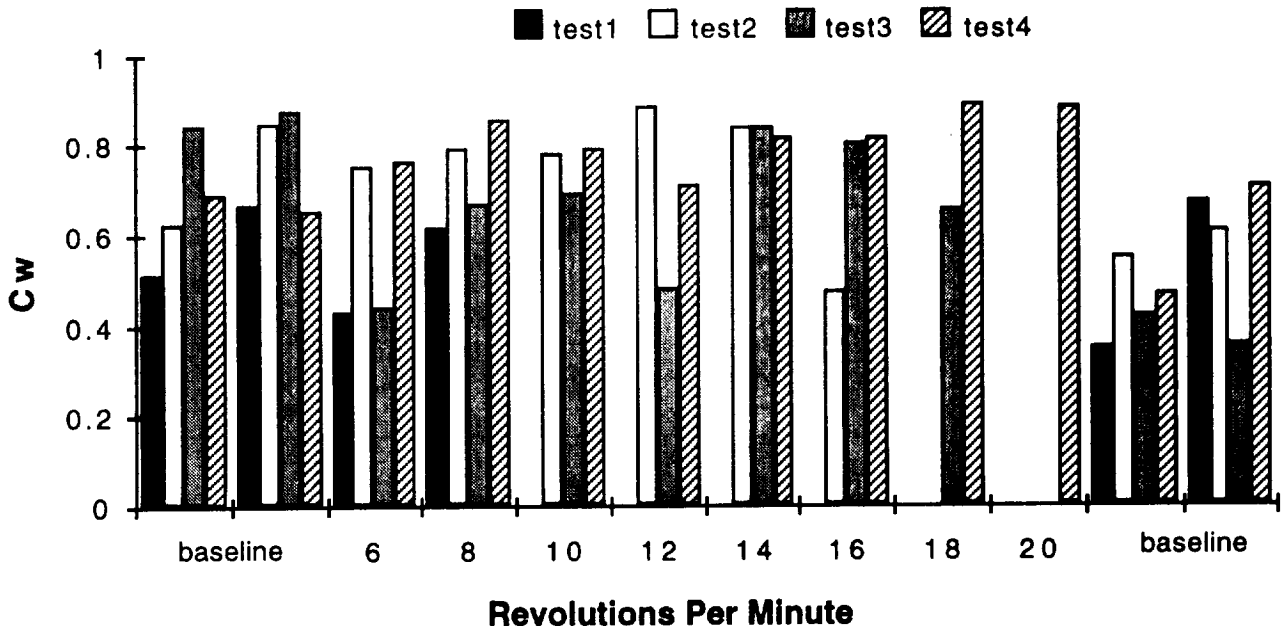
Note: There was a 1-year interval between these "baseline" rotating chair tests.

Figure A-34. Coherence between heart rate and respiration rate during two baseline motion sickness tests—subject 8.



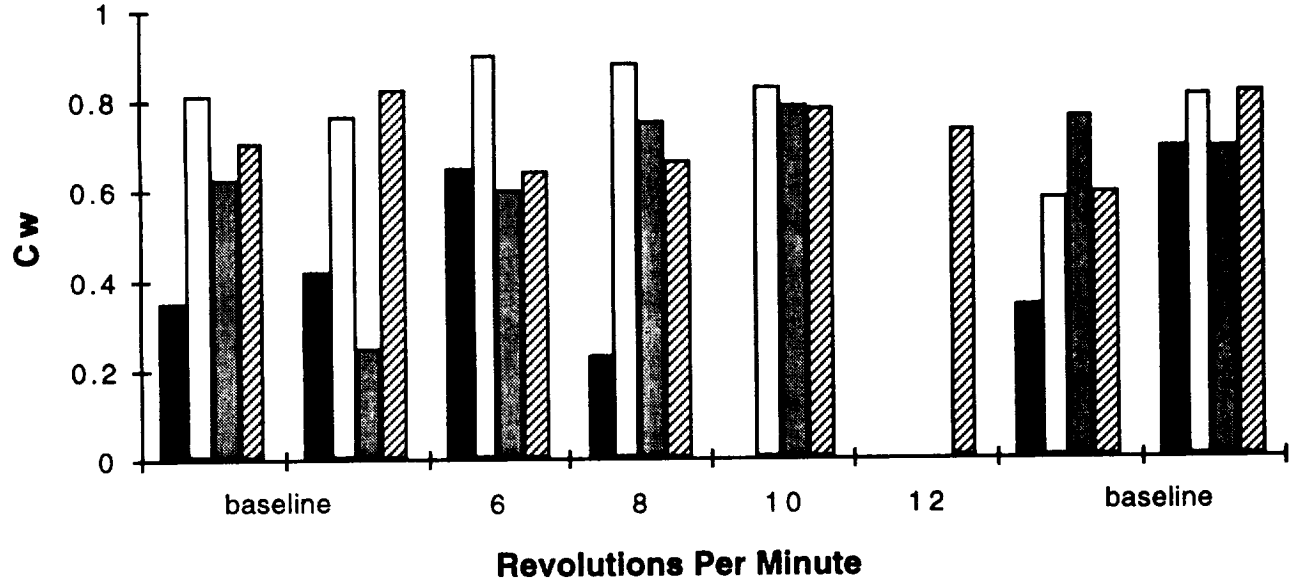
Note: Tests 1–4 were at 1 week intervals. Test 5 was administered approximately 1 year later with tests 5–7, also at 1 week intervals.

Figure A-35. Changes in coherence between heart rate and respiration across tests—subject 9.



Note: Number of days between tests 1 and 2 = 189; 2 and 3 = 105; 3 and 4 = 34.

Figure A-36. Changes in coherence between heart rate and respiration across tests—subject 10.



Note: Number of days between tests 1 and 2 = 173; 2 and 3 = 125; 3 and 4 = 31.

Figure A-37. Changes in coherence between heart rate and respiration across tests—subject 11.

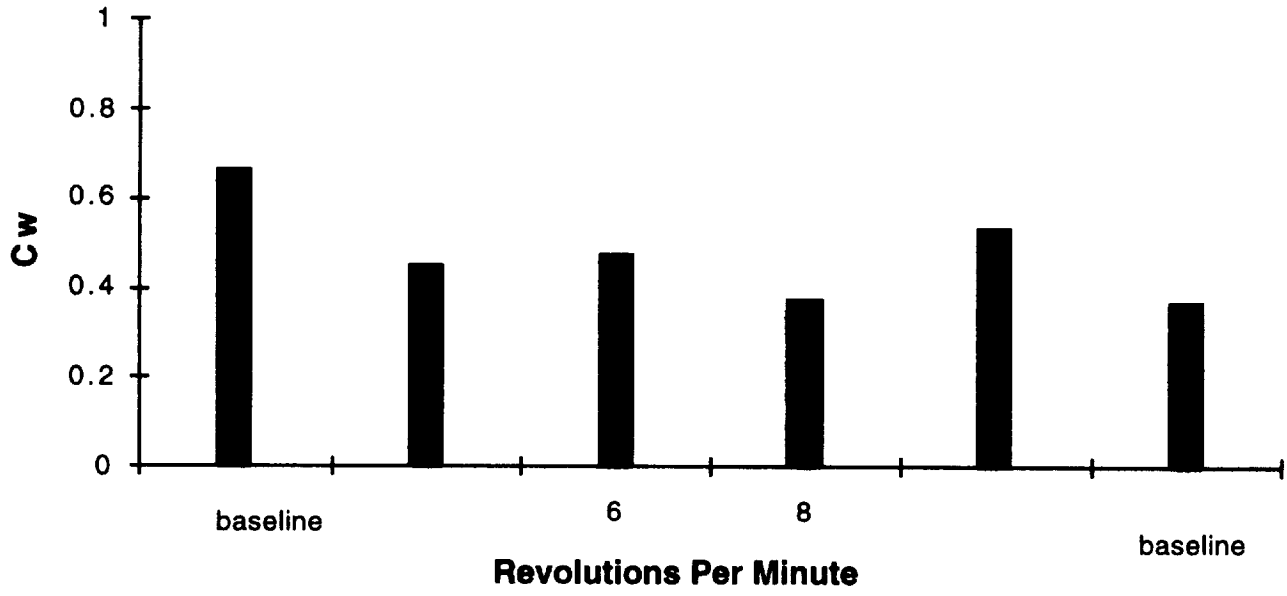


Figure A-38. Coherence between heart rate and respiration during baseline motion sickness test—subject 12.

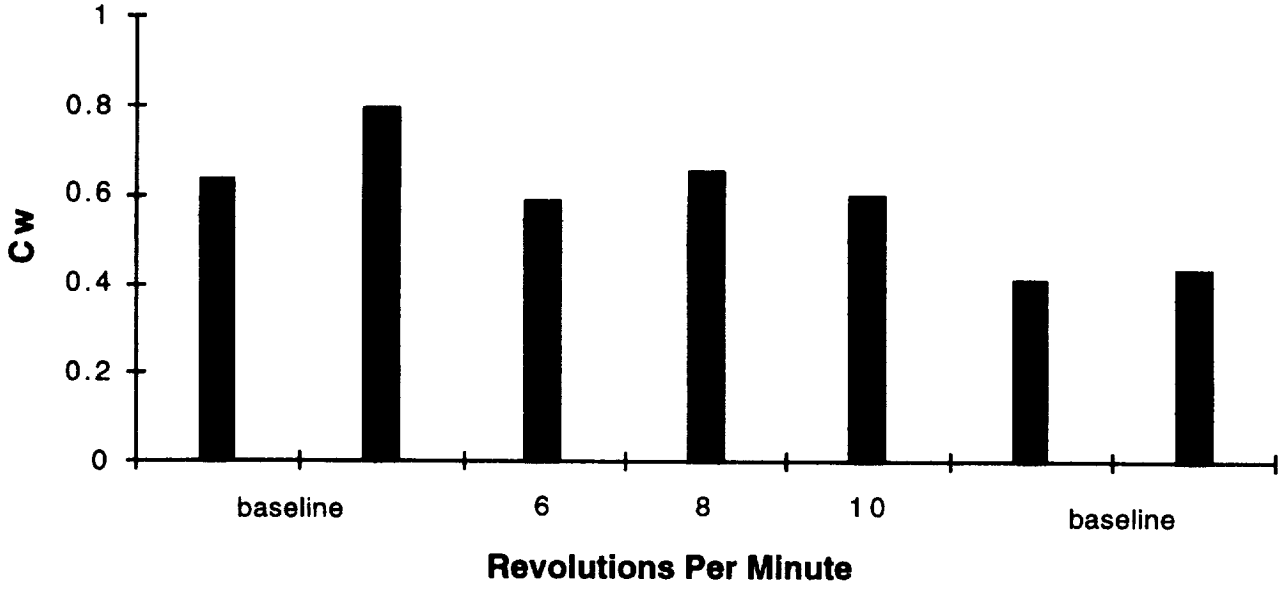
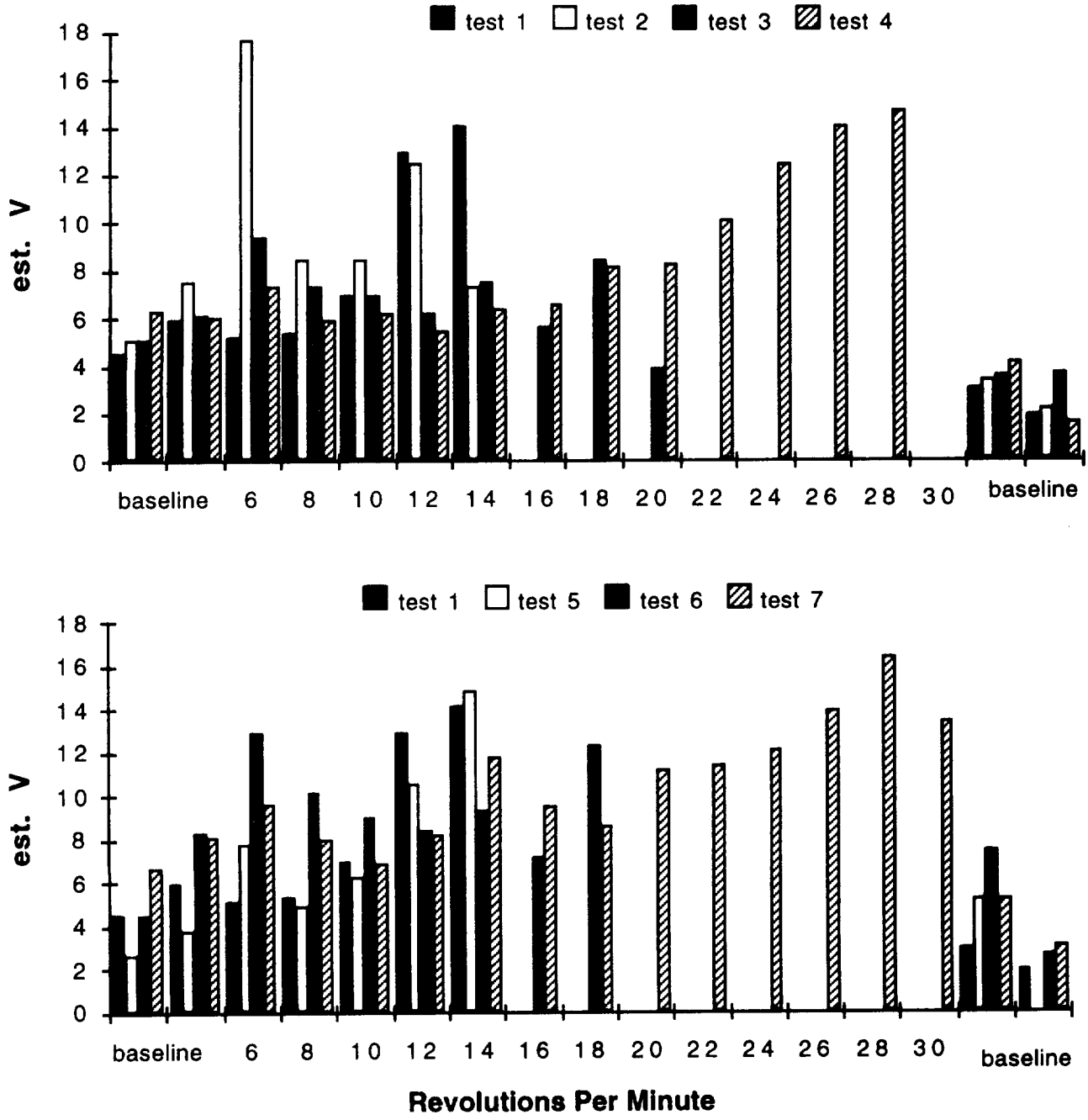
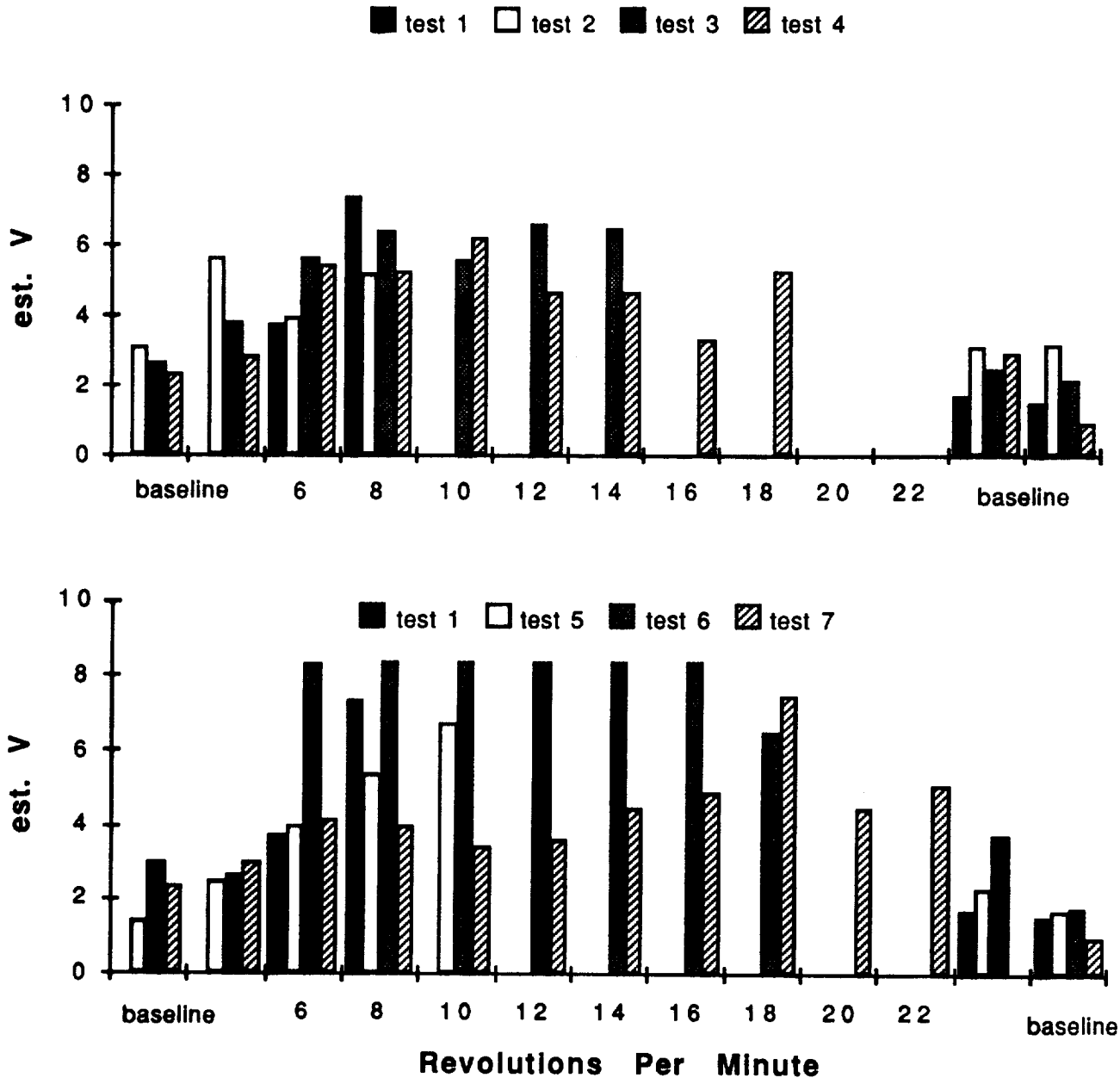


Figure A-39. Coherence between heart rate and respiration during baseline motion sickness test—subject 13.



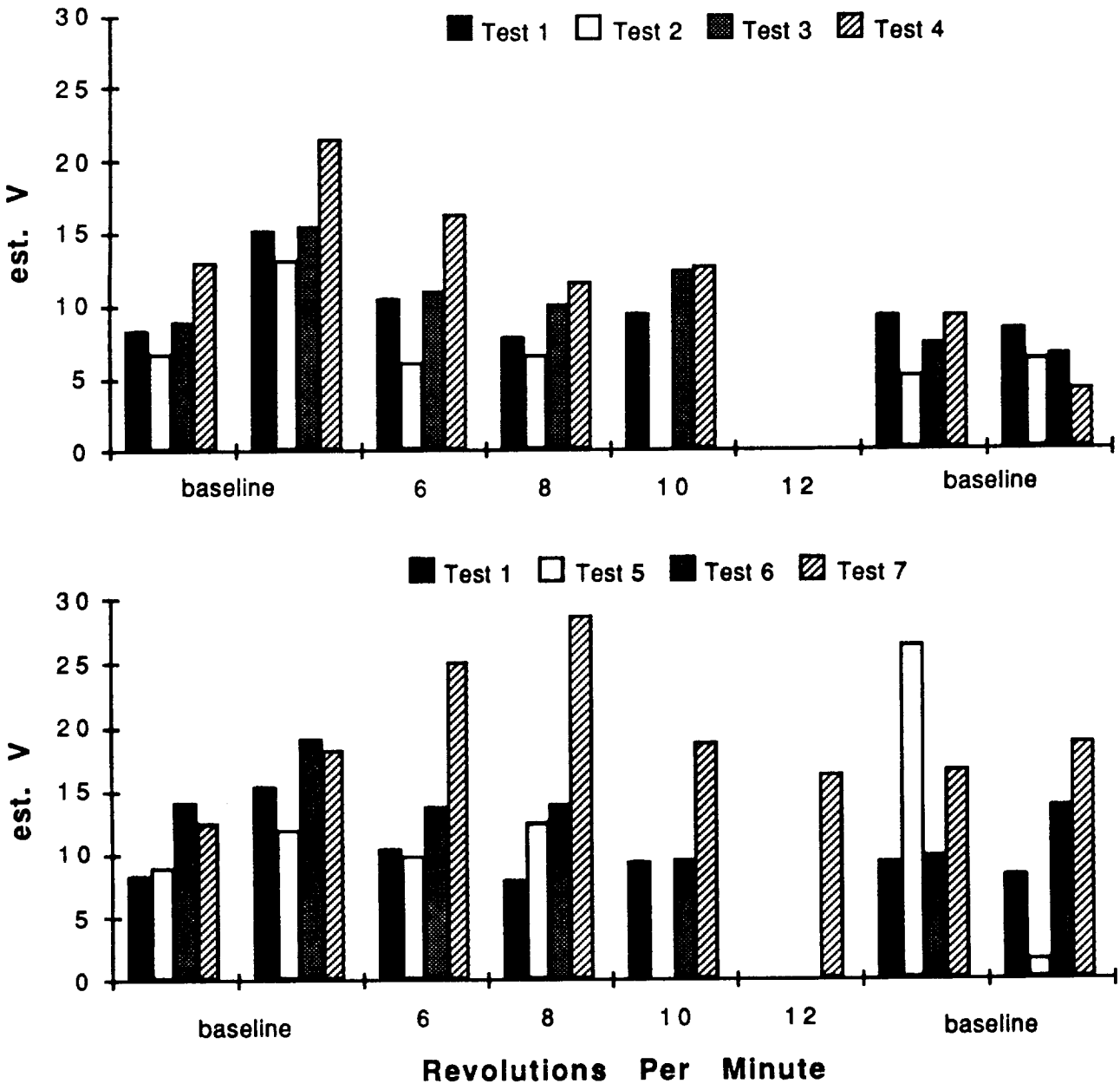
Note: Tests 1–4 were at 1 week intervals. Test 5 was administered approximately 1 year later with tests 5–7, also at 1 week intervals.

Figure A-40. Changes in estimate of vagal tone across motion sickness tests—subject 1.



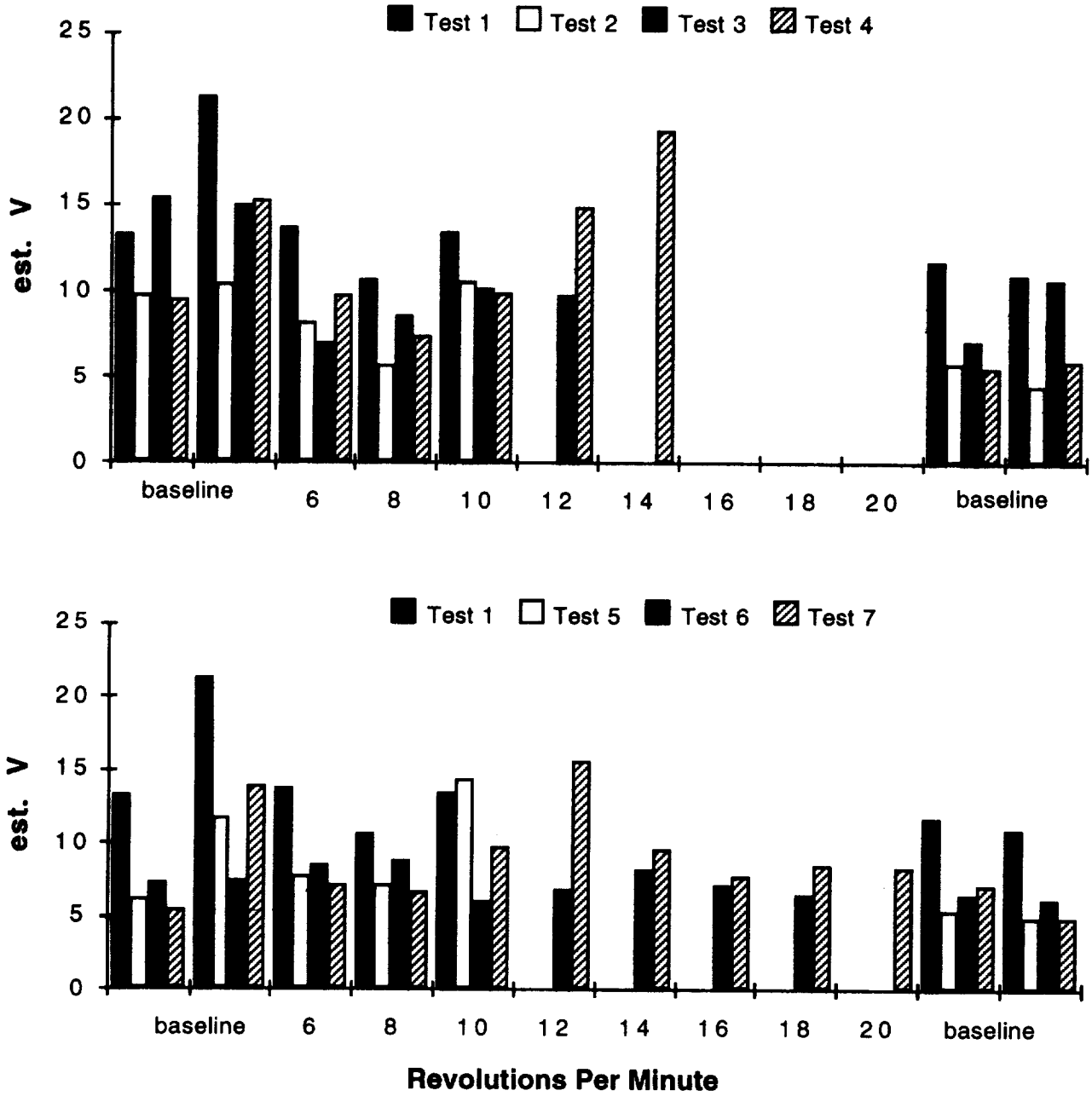
Note: Tests 1–4 were at 1 week intervals. Test 5 was administered approximately 1 year later with tests 5–7, also at 1 week intervals.

Figure A-41. Changes in estimate of vagal tone across motion sickness tests—subject 2.



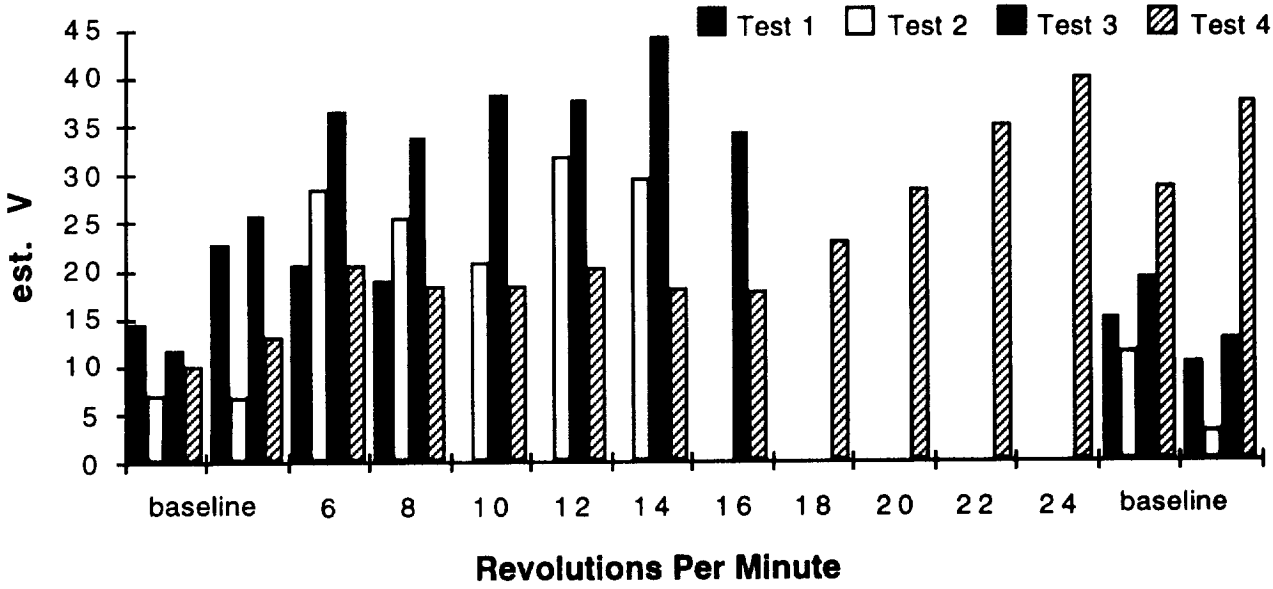
Note: Tests 1–4 were at 1 week intervals. Test 5 was administered approximately 1 year later with tests 5–7, also at 1 week intervals.

Figure A-42. Changes in estimate of vagal tone across motion sickness tests—subject 3.



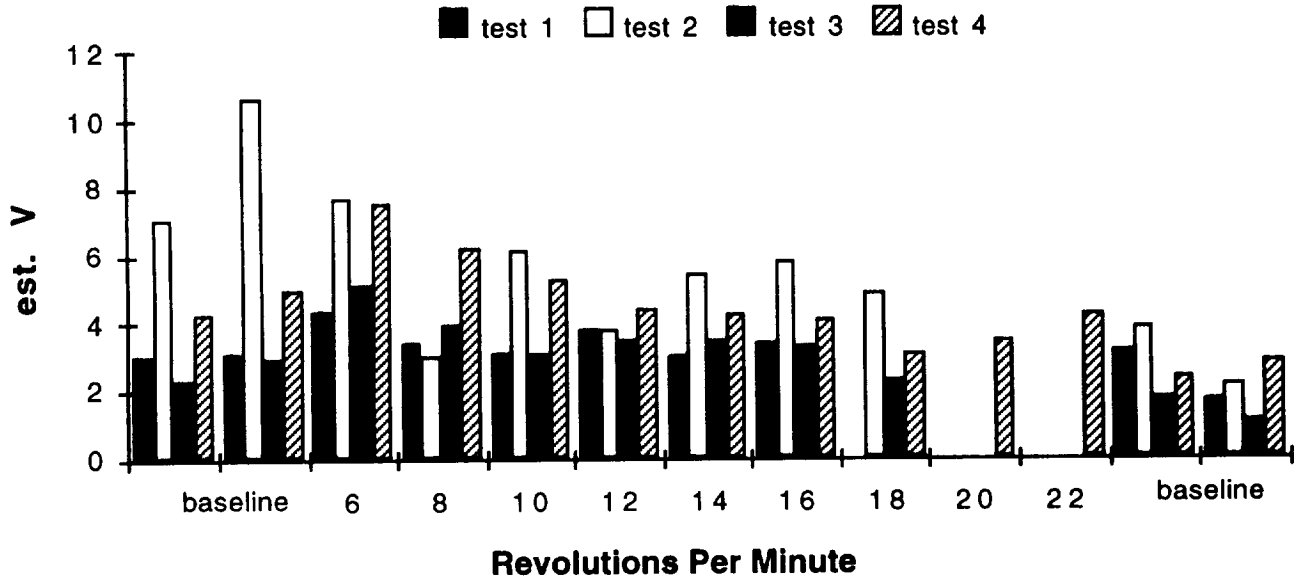
Note: Tests 1–4 were at 1 week intervals. Test 5 was administered approximately 1 year later with tests 5–7, also at 1 week intervals.

Figure A-43. Changes in estimate of vagal tone across motion sickness tests—subject 4.



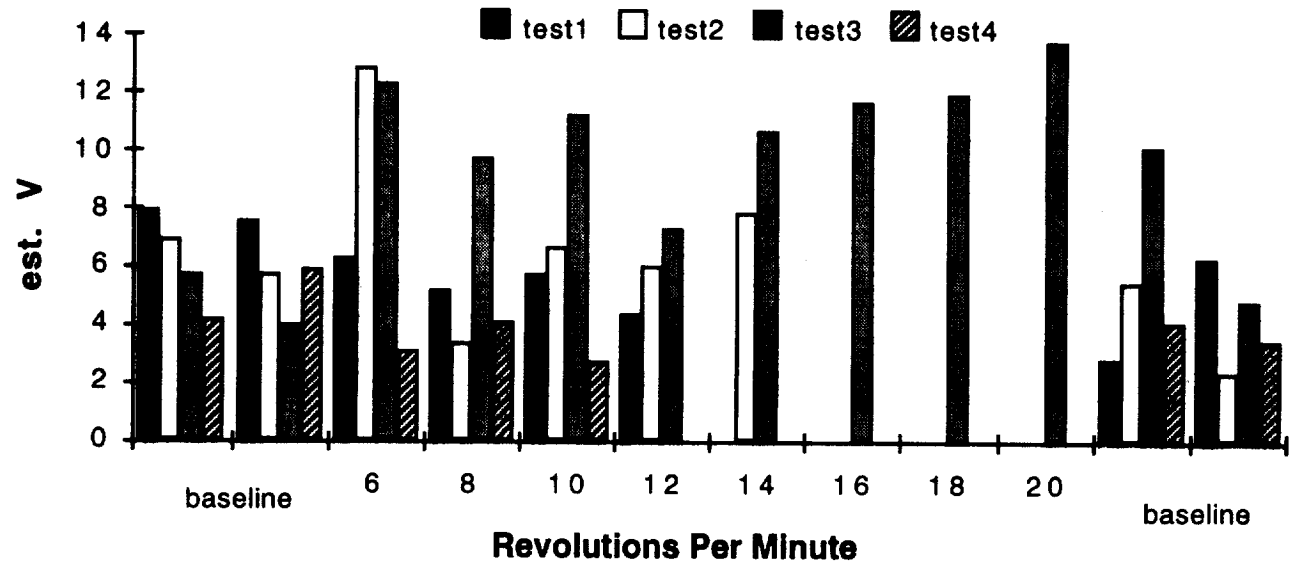
Note: Tests were conducted at weekly intervals.

Figure A-44. Changes in estimate of vagal tone across motion sickness tests—subject 5.



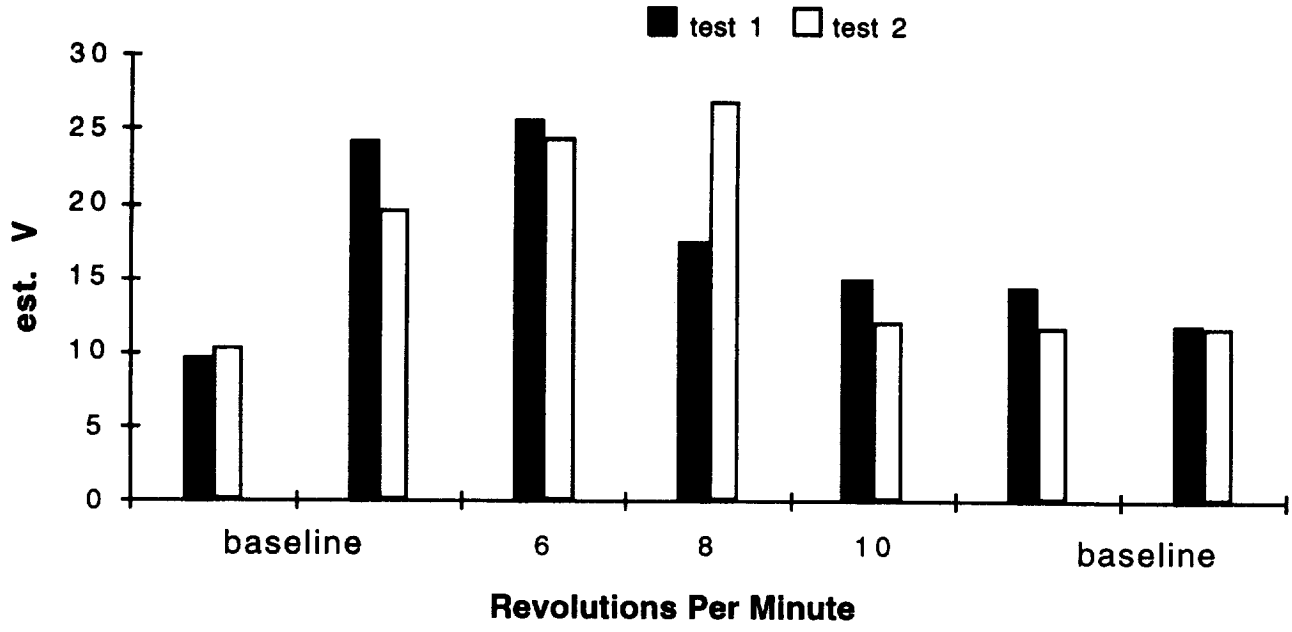
Note: Number of days between tests 1 and 2 = 234; 2 and 3 = 74; 3 and 4 = 54.

Figure A-45. Changes in estimate of vagal tone across motion sickness tests—subject 6.



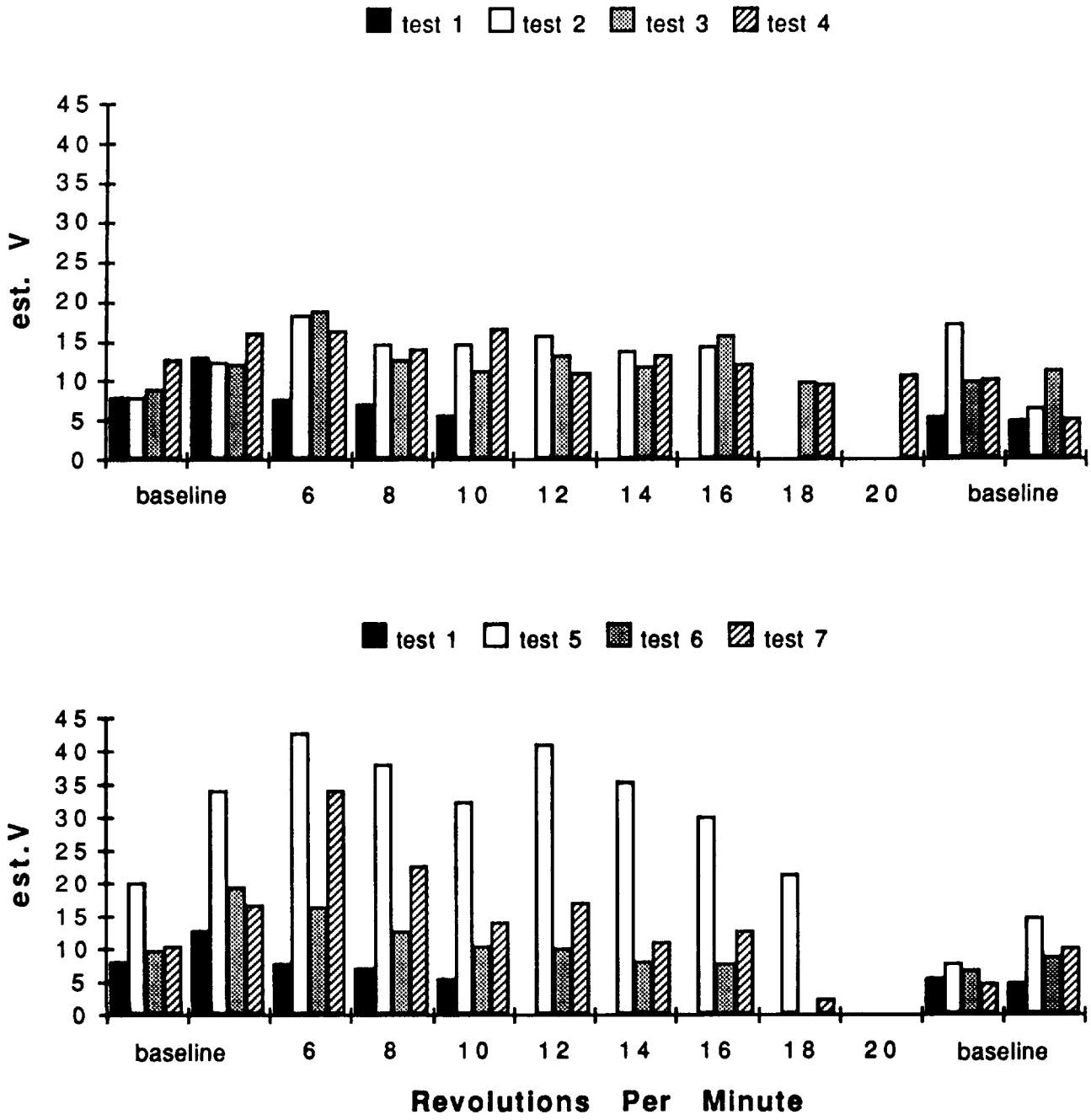
Note: Number of days between tests 1 and 2 = 201; 2 and 3 = 102; 3 and 4 = 57.

Figure A-46. Changes in estimate of vagal tone across motion sickness tests—subject 7.



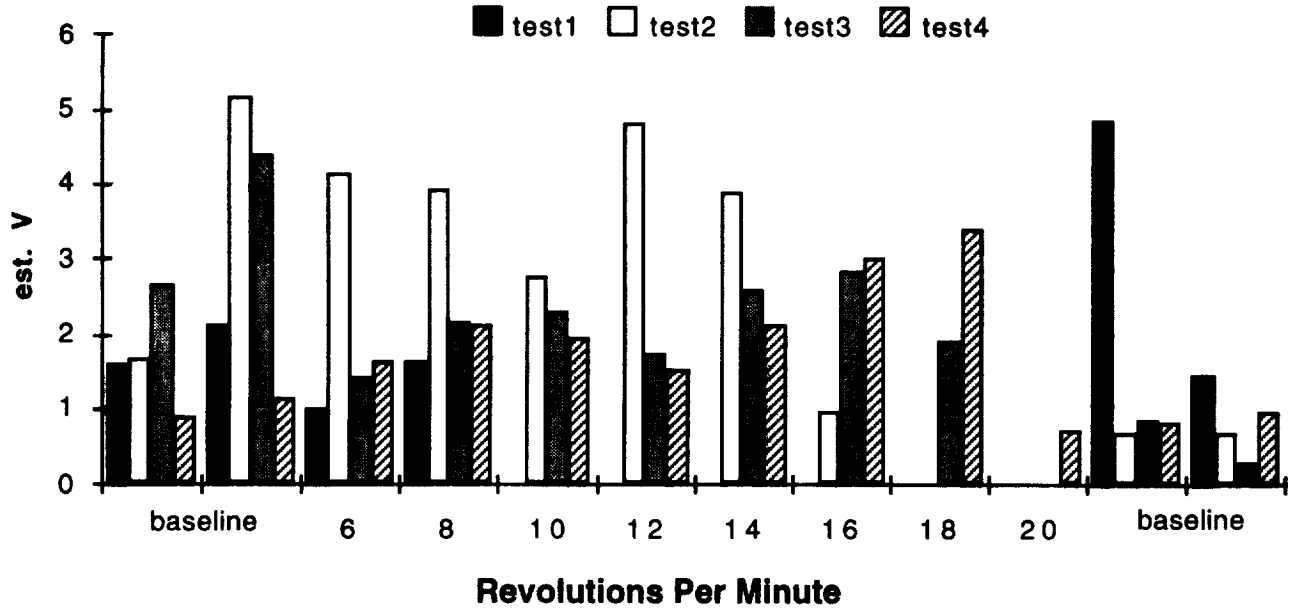
Note: There was a 1-year interval between these "baseline" rotating chair tests.

Figure A-47. Changes in estimate of vagal tone during two baseline motion sickness tests—subject 8.



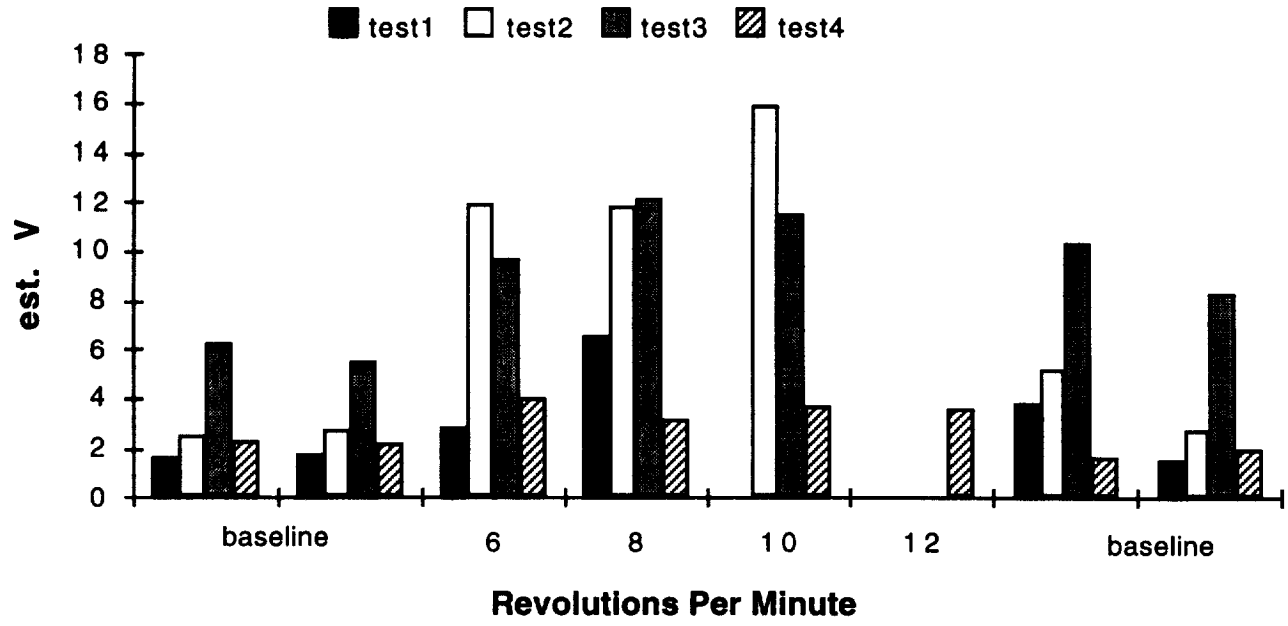
Note: Tests 1–4 were at 1 week intervals. Test 5 was administered approximately 1 year later with tests 5–7, also at 1 week intervals.

Figure A-48. Changes in estimate of vagal tone across motion sickness tests—subject 9.



Note: Number of days between tests 1 and 2 = 189; 2 and 3 = 105; 3 and 4 = 34.

Figure A-49. Changes in estimate of vagal tone across motion sickness tests—subject 10.



Note: Number of days between tests 1 and 2 = 173; 2 and 3 = 125; 3 and 4 = 31.

Figure A-50. Changes in estimate of vagal tone across motion sickness tests—subject 11.

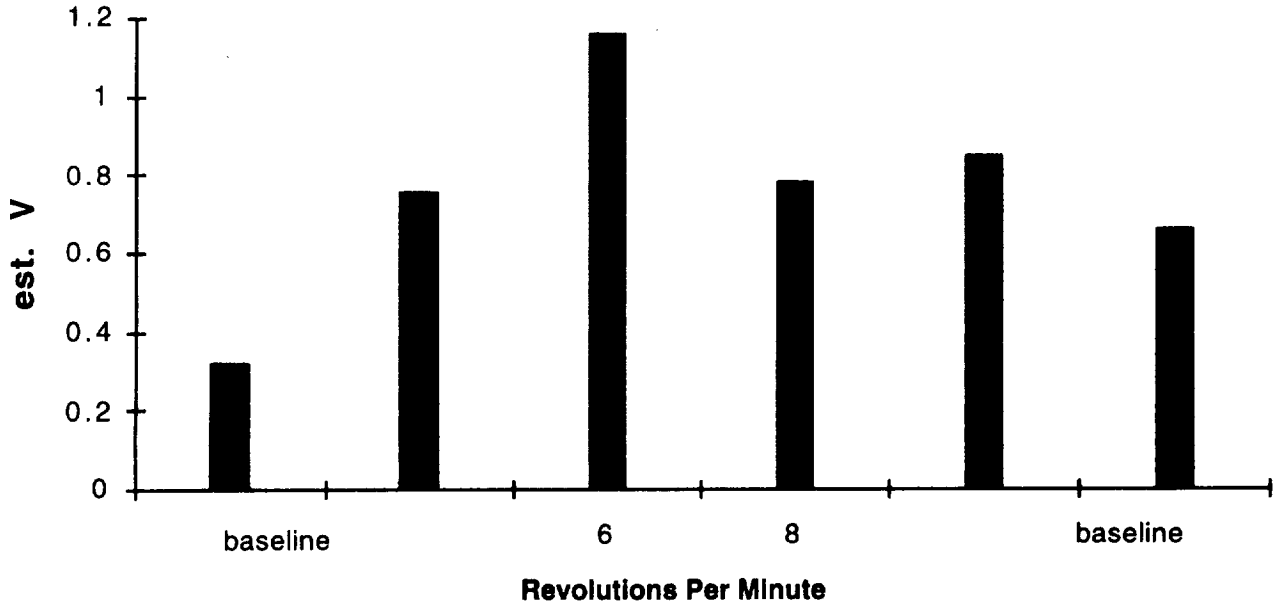


Figure A-51. Estimate of vagal tone during baseline motion sickness test—subject 12.

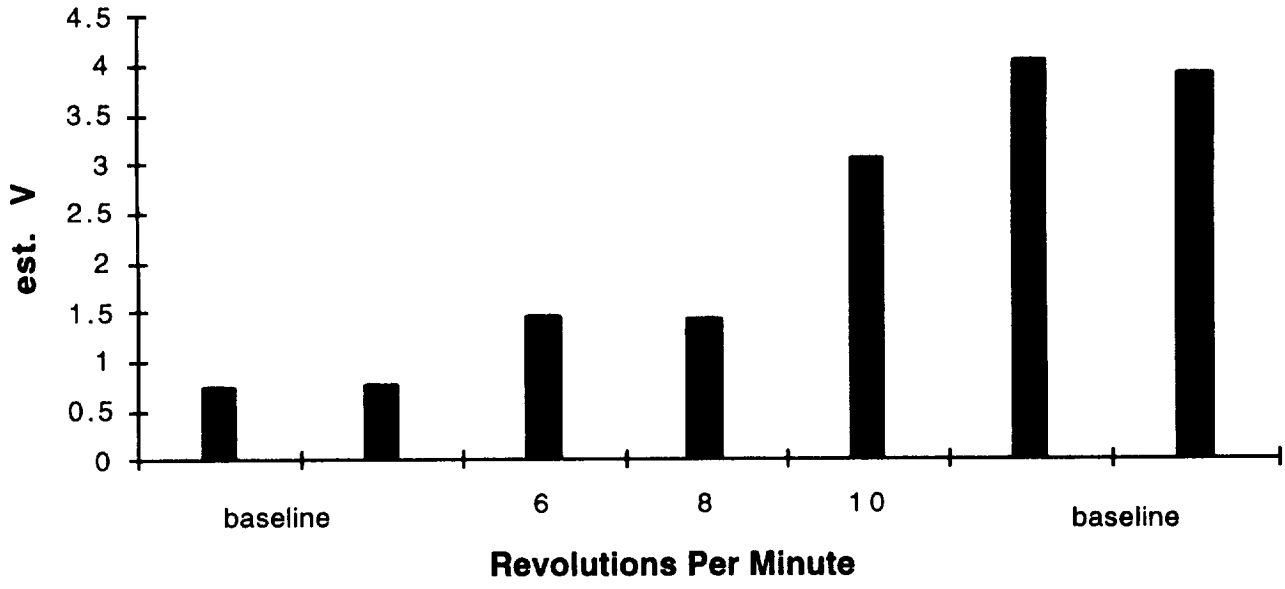
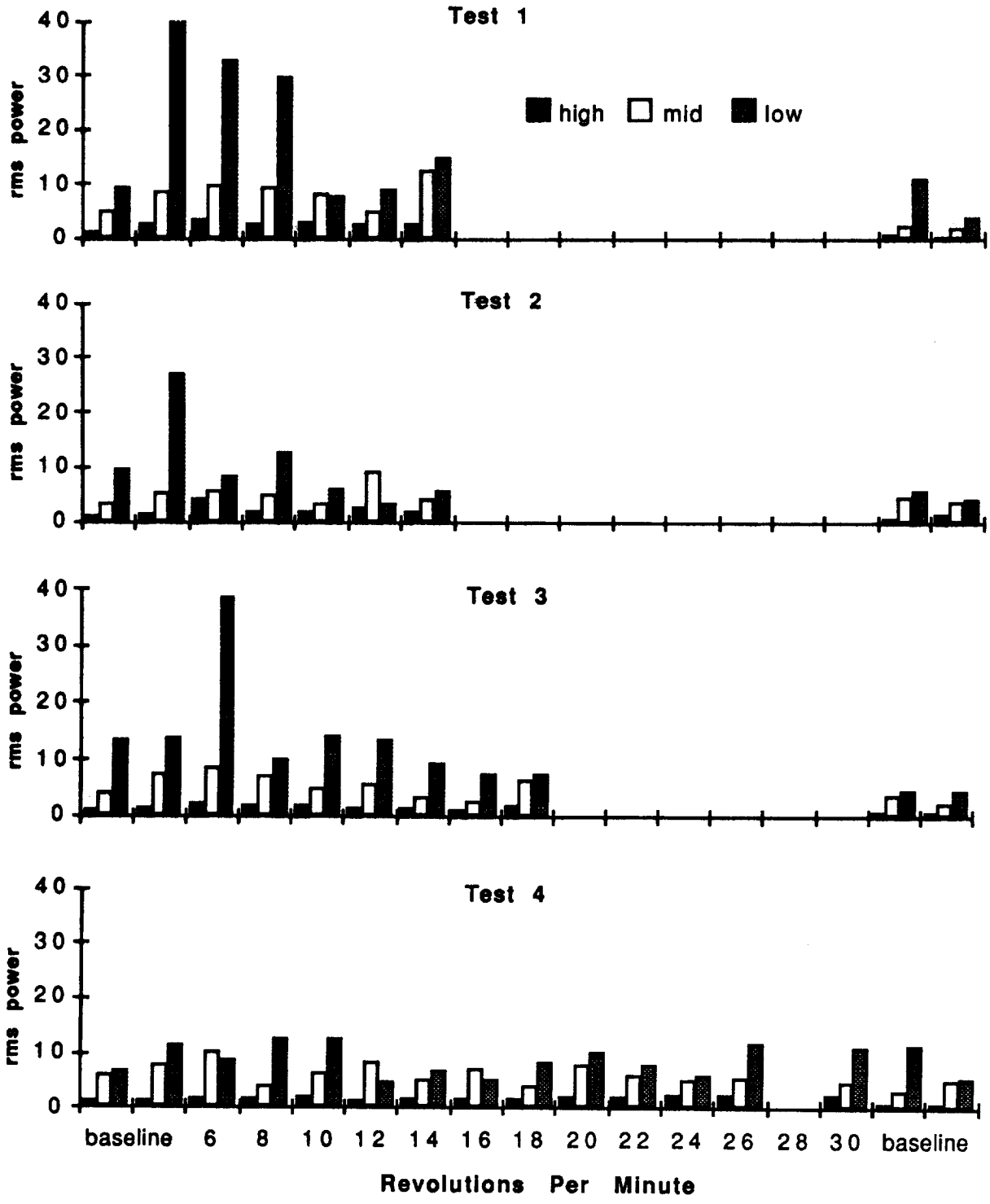
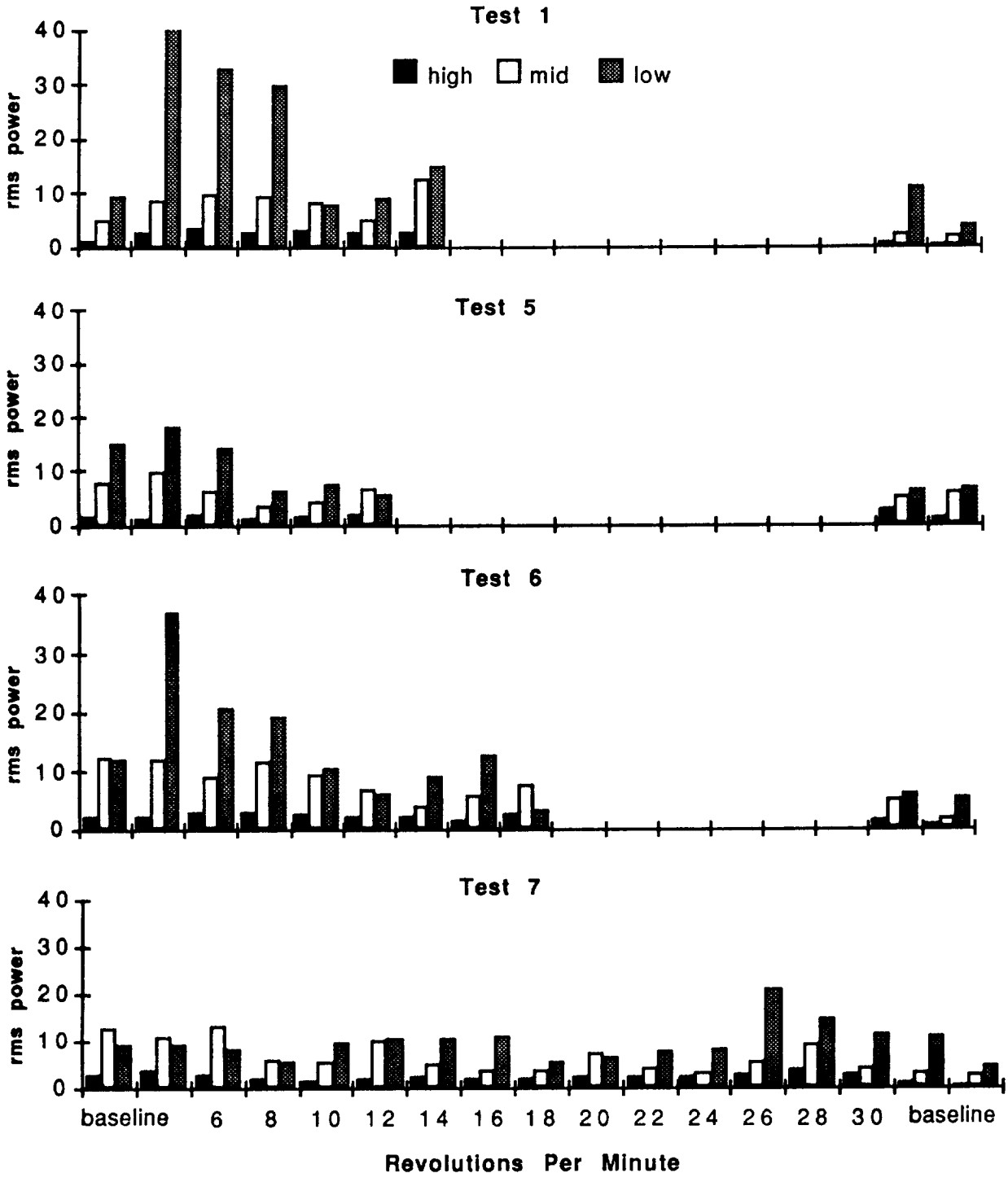


Figure A-52. Estimate of vagal tone during baseline motion sickness test—subject 13.



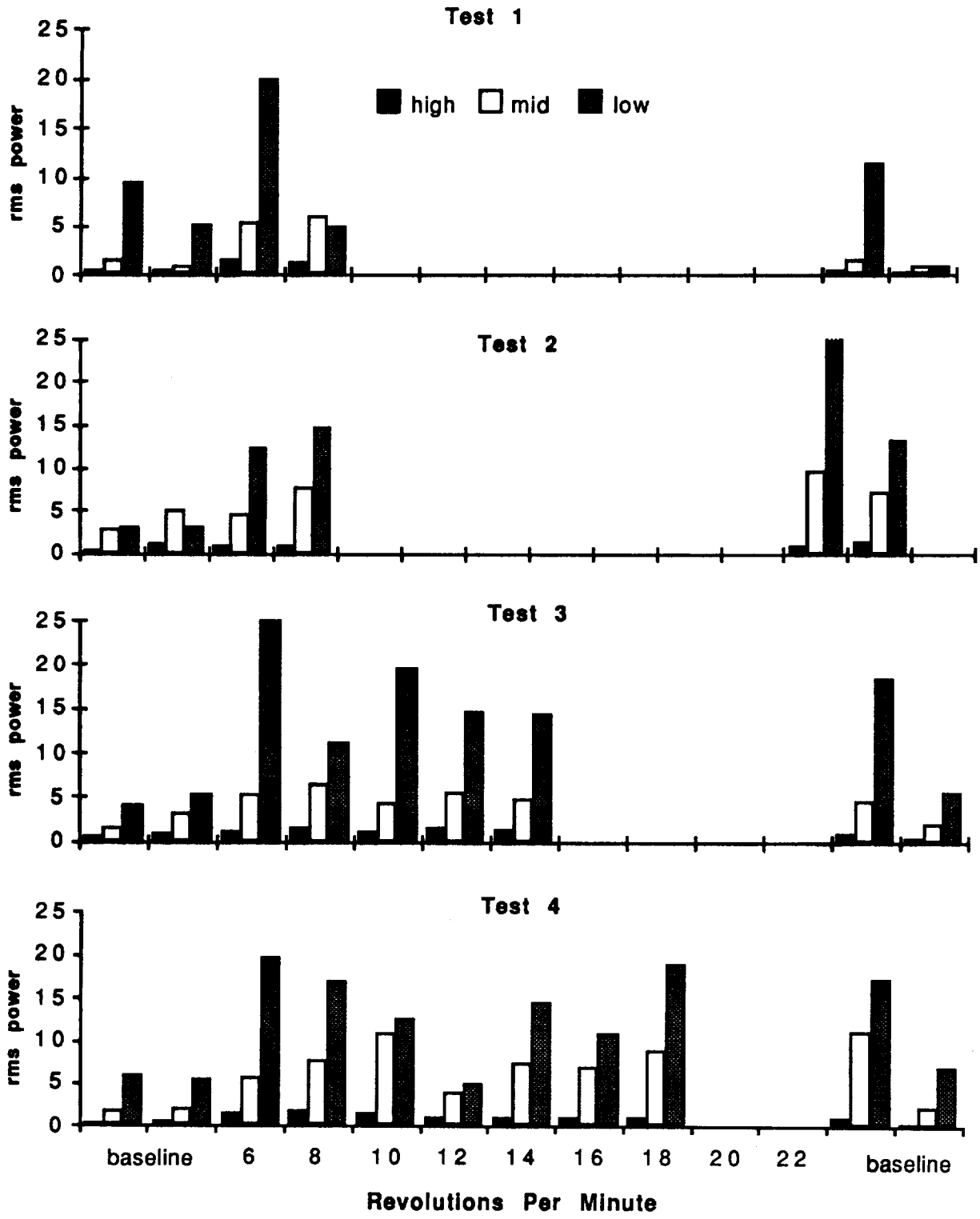
Note: Tests 1–4 were at 1 week intervals. High = 0 to 0.05 Hz, mid = 0.05 to 0.1 Hz, low = 0.1 to 0.4 Hz.

Figure A-53(a). Heart rate variability across motion sickness tests (year 1)—subject 1.



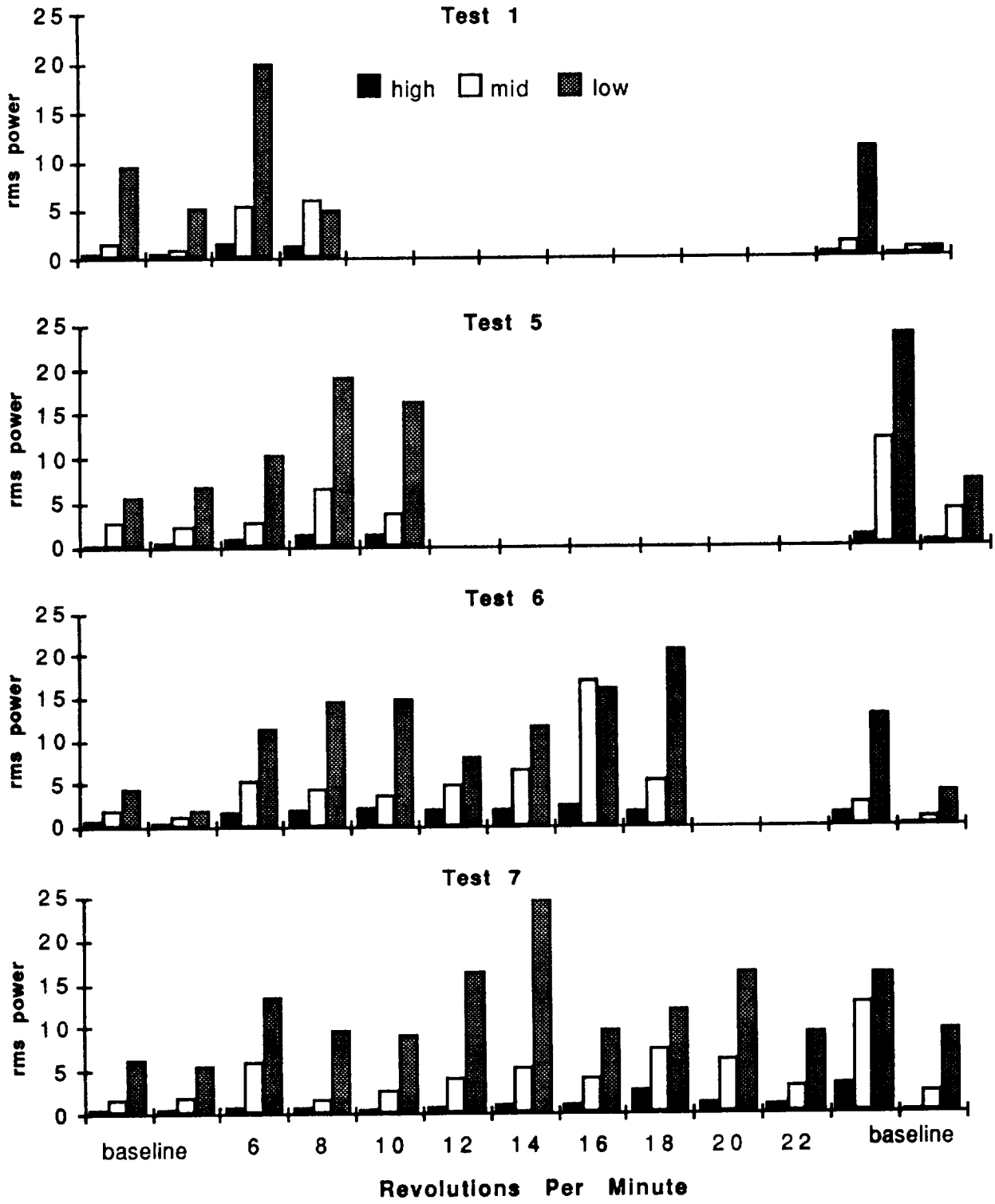
Note: Tests 5–7 were at 1 week intervals. High = 0 to 0.05 Hz, mid = 0.05 to 0.1 Hz, low = 0.1 to 0.4 Hz.

Figure A-53(b). Heart rate variability across motion sickness tests (year 2)—subject 1.



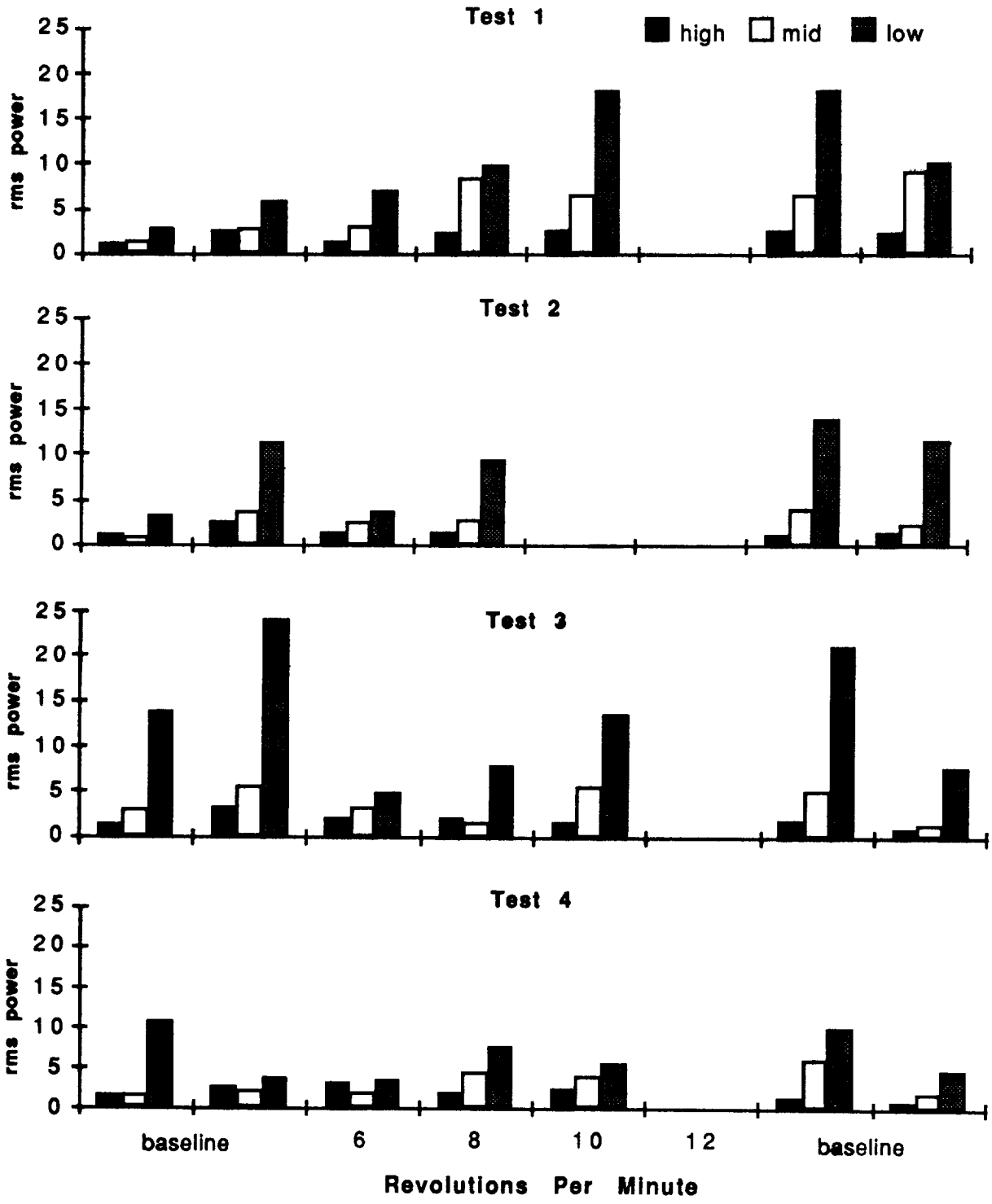
Note: Tests 1–4 were at 1 week intervals. High = 0 to 0.05 Hz, mid = 0.05 to 0.1 Hz, low = 0.1 to 0.4 Hz.

Figure A-54(a). Heart rate variability across motion sickness tests (year 1)—subject 2.



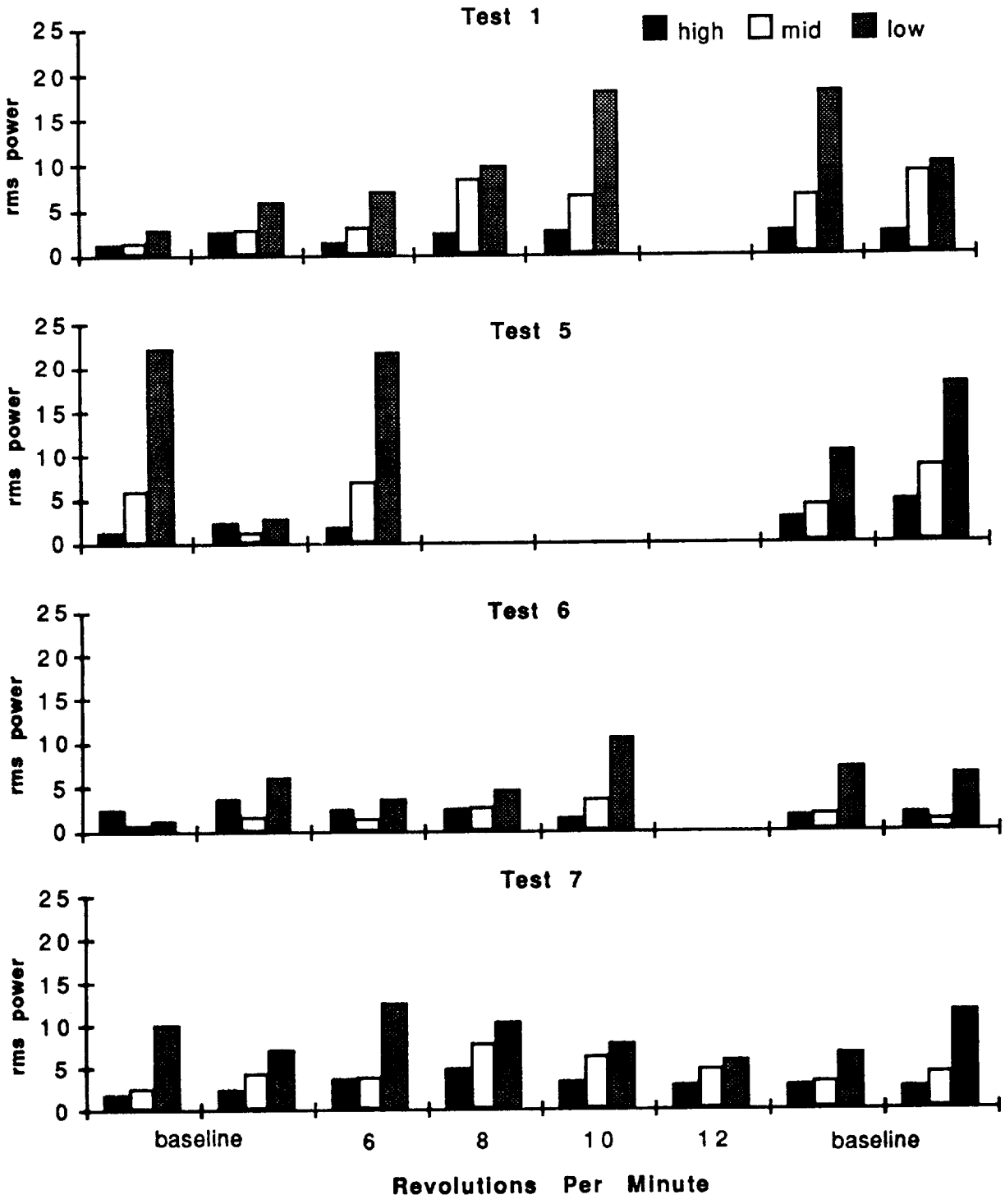
Note: Tests 5–7 were at 1 week intervals. High = 0 to 0.05 Hz, mid = 0.05 to 0.1 Hz, low = 0.1 to 0.4 Hz.

Figure A-54(b). Heart rate variability across motion sickness tests (year 2)—subject 2.



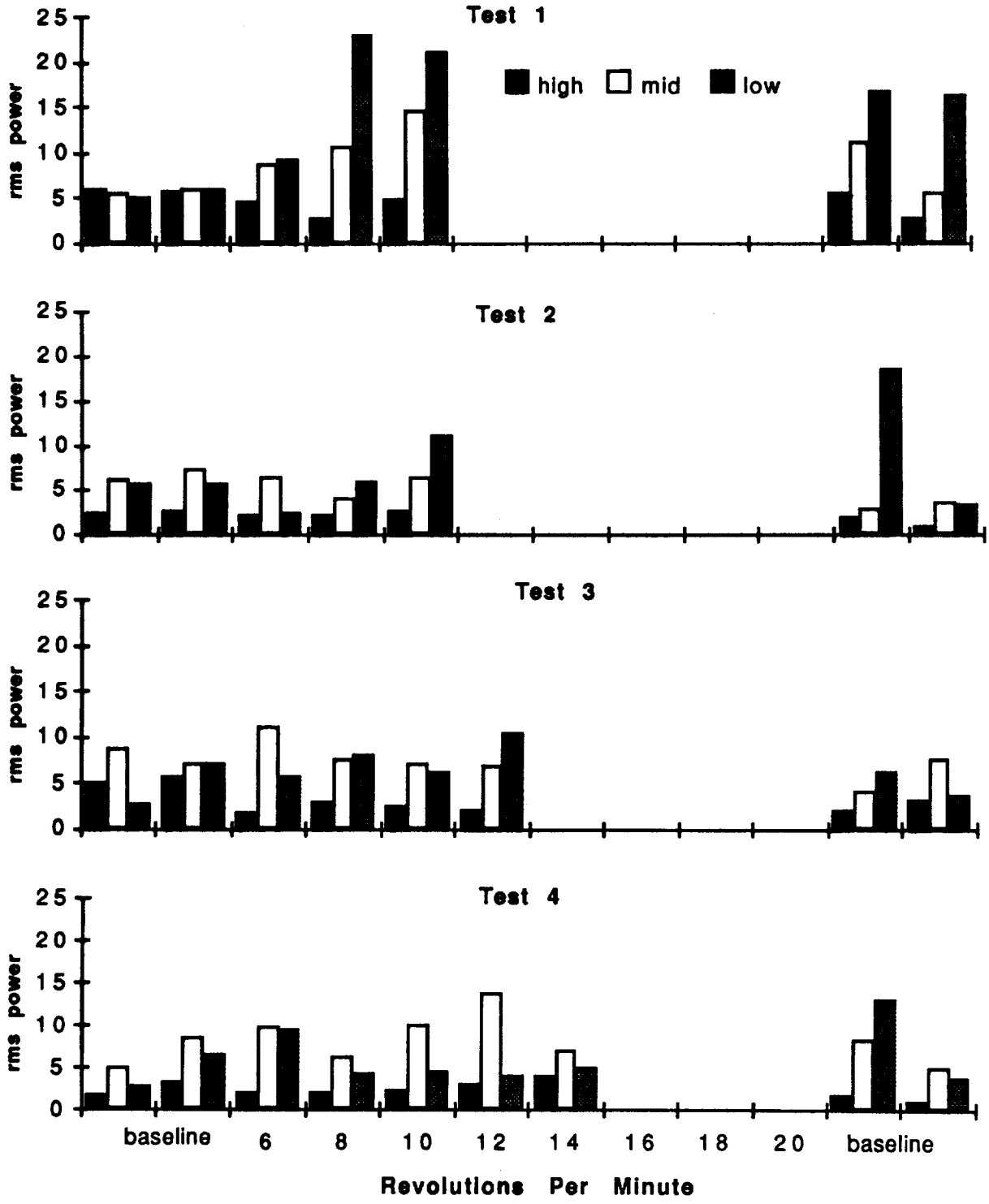
Note: Tests 1–4 were at 1 week intervals. High = 0 to 0.05 Hz, mid = 0.05 to 0.1 Hz, low = 0.1 to 0.4 Hz.

Figure A-55(a). Heart rate variability across motion sickness tests (year 1)—subject 3.



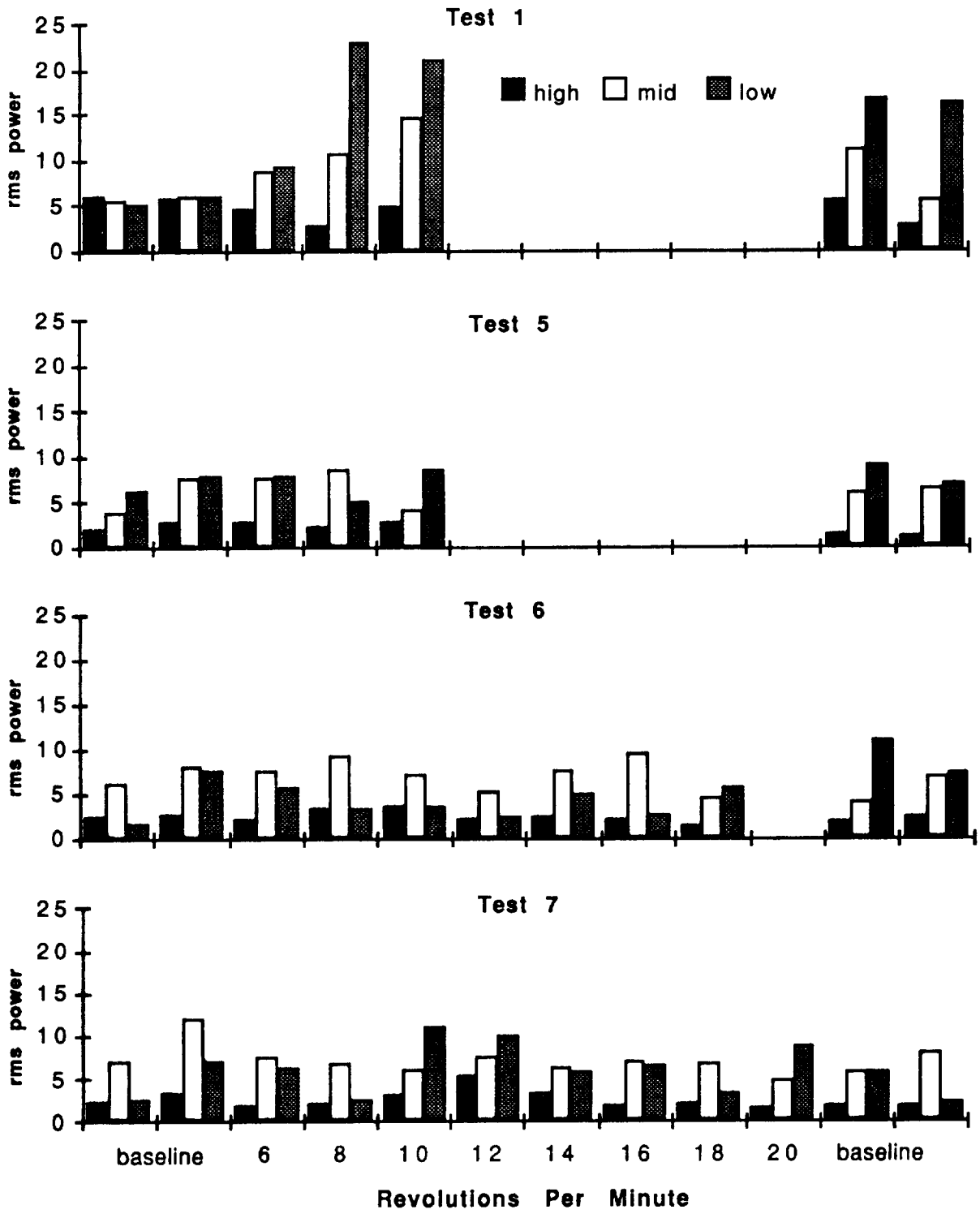
Note: Tests 5–7 were at 1 week intervals. High = 0 to 0.05 Hz, mid = 0.05 to 0.1 Hz, low = 0.1 to 0.4 Hz.

Figure A-55(b). Heart rate variability across motion sickness tests (year 2)—subject 3.



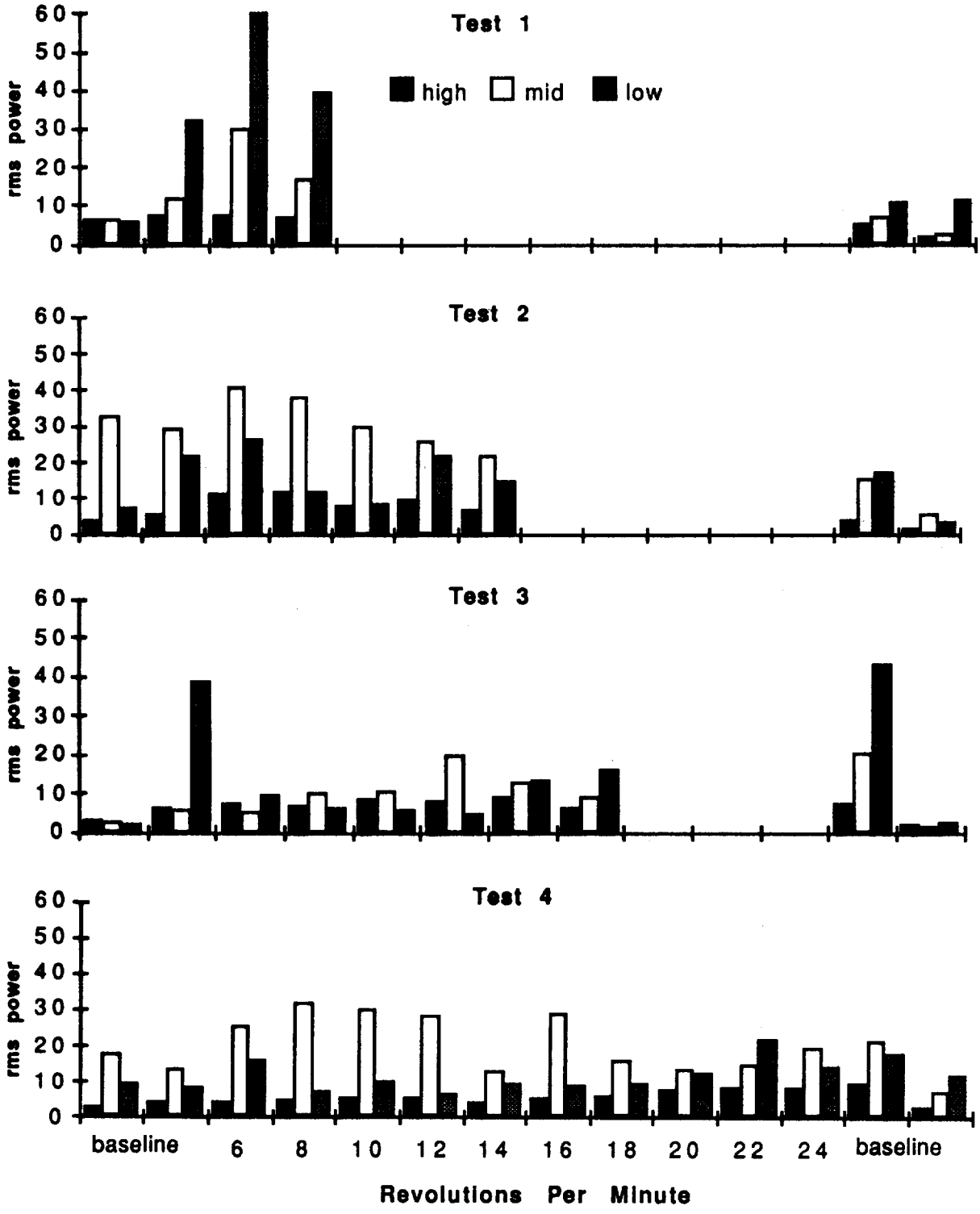
Note: Tests 1–4 were at 1 week intervals. High = 0 to 0.05 Hz, mid = 0.05 to 0.1 Hz, low = 0.1 to 0.4 Hz.

Figure A-56(a). Heart rate variability across motion sickness tests (year 1)—subject 4.



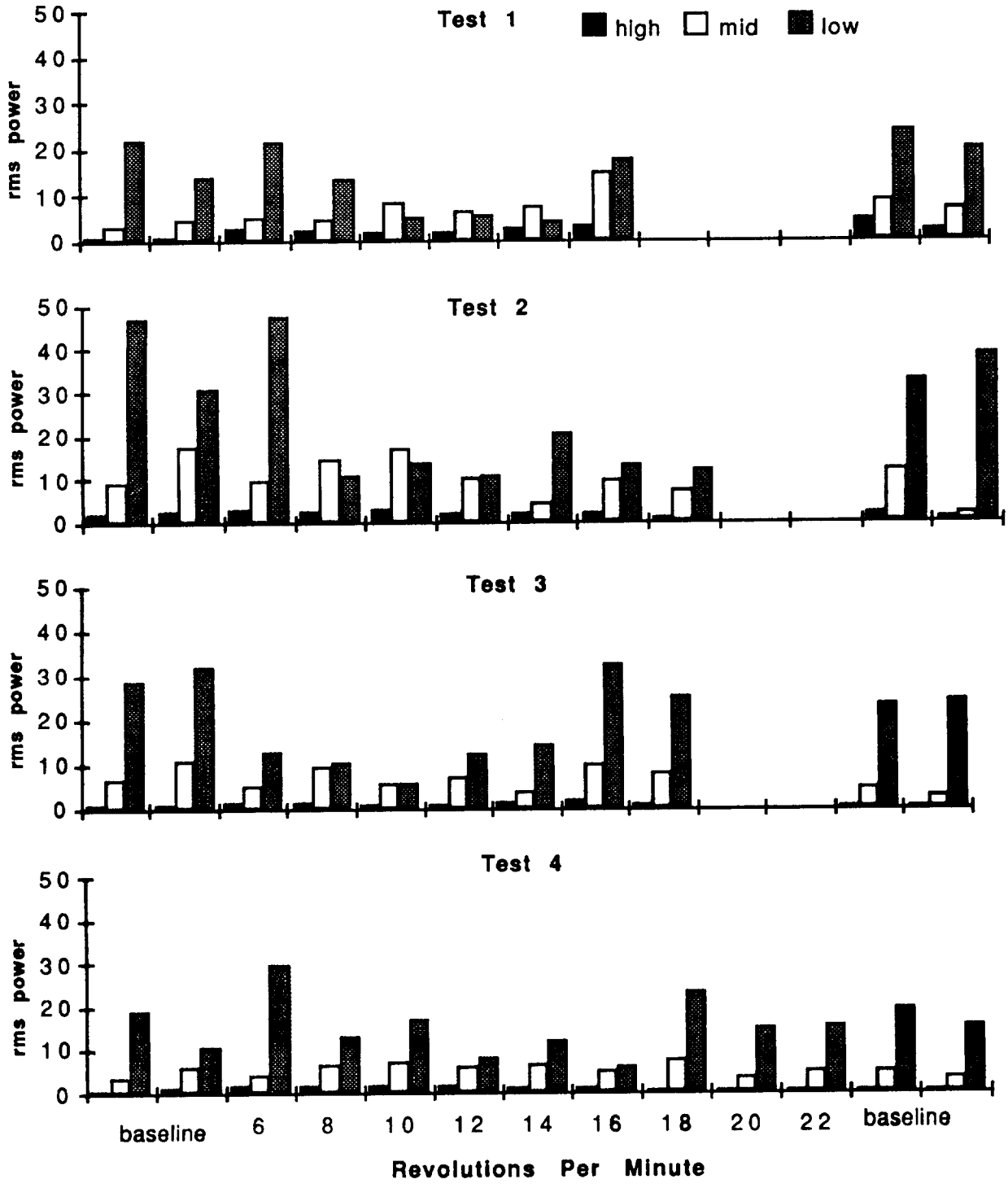
Note: Tests 5–7 were at 1 week intervals. High = 0 to 0.05 Hz, mid = 0.05 to 0.1 Hz, low = 0.1 to 0.4 Hz.

Figure A-56(b). Heart rate variability across motion sickness tests (year 2)—subject 4.



Note: Tests were conducted at weekly intervals. High = 0 to 0.05 Hz, mid = 0.05 to 0.1 Hz, low = 0.1 to 0.4 Hz.

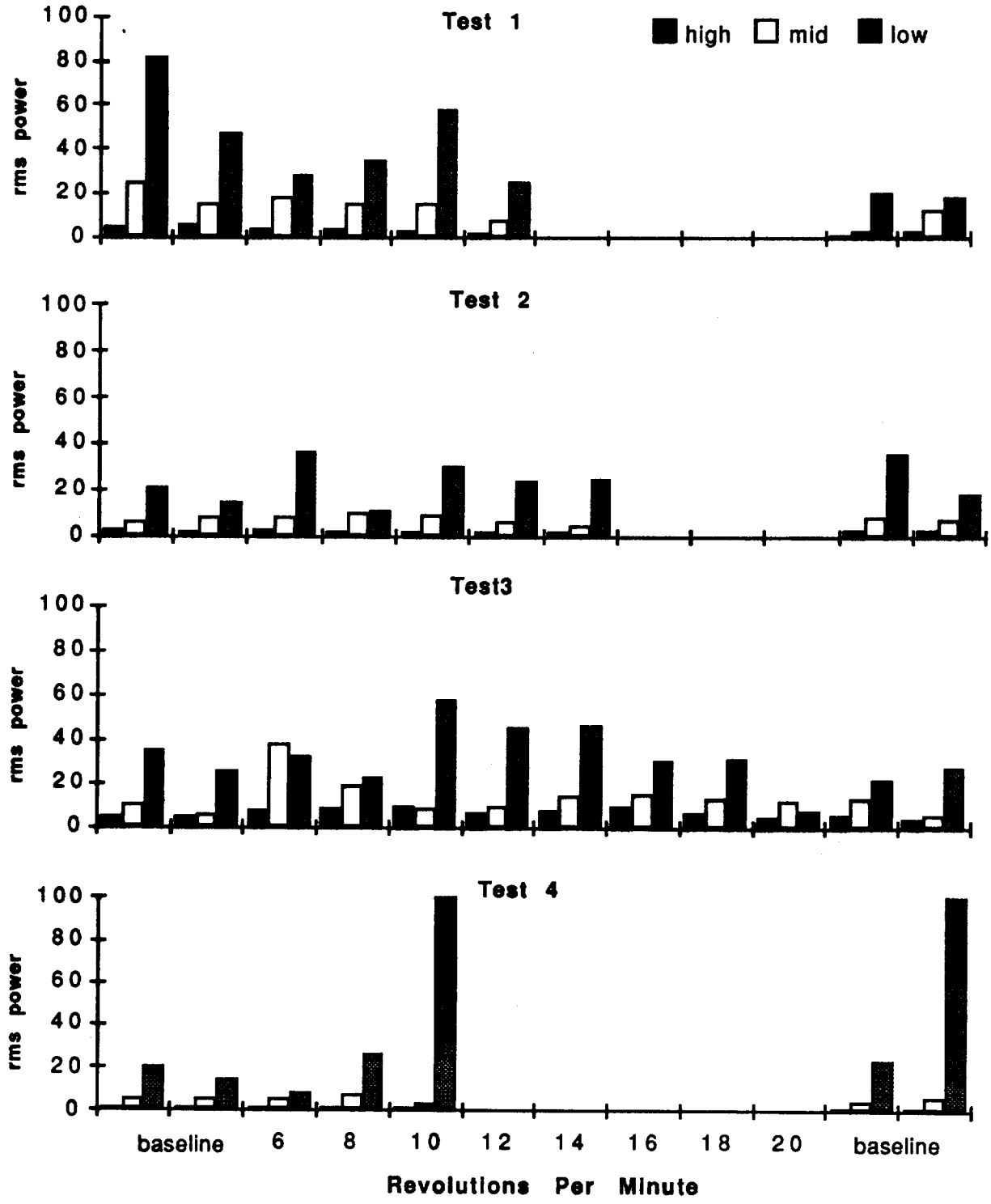
Figure A-57. Heart rate variability across motion sickness tests—subject 5.



Note: Number of days between tests 1 and 2 = 234; 2 and 3 = 74; 3 and 4 = 54. High = 0 to 0.05 Hz, mid = 0.05 to 0.1 Hz, low = 0.1 to 0.4 Hz.

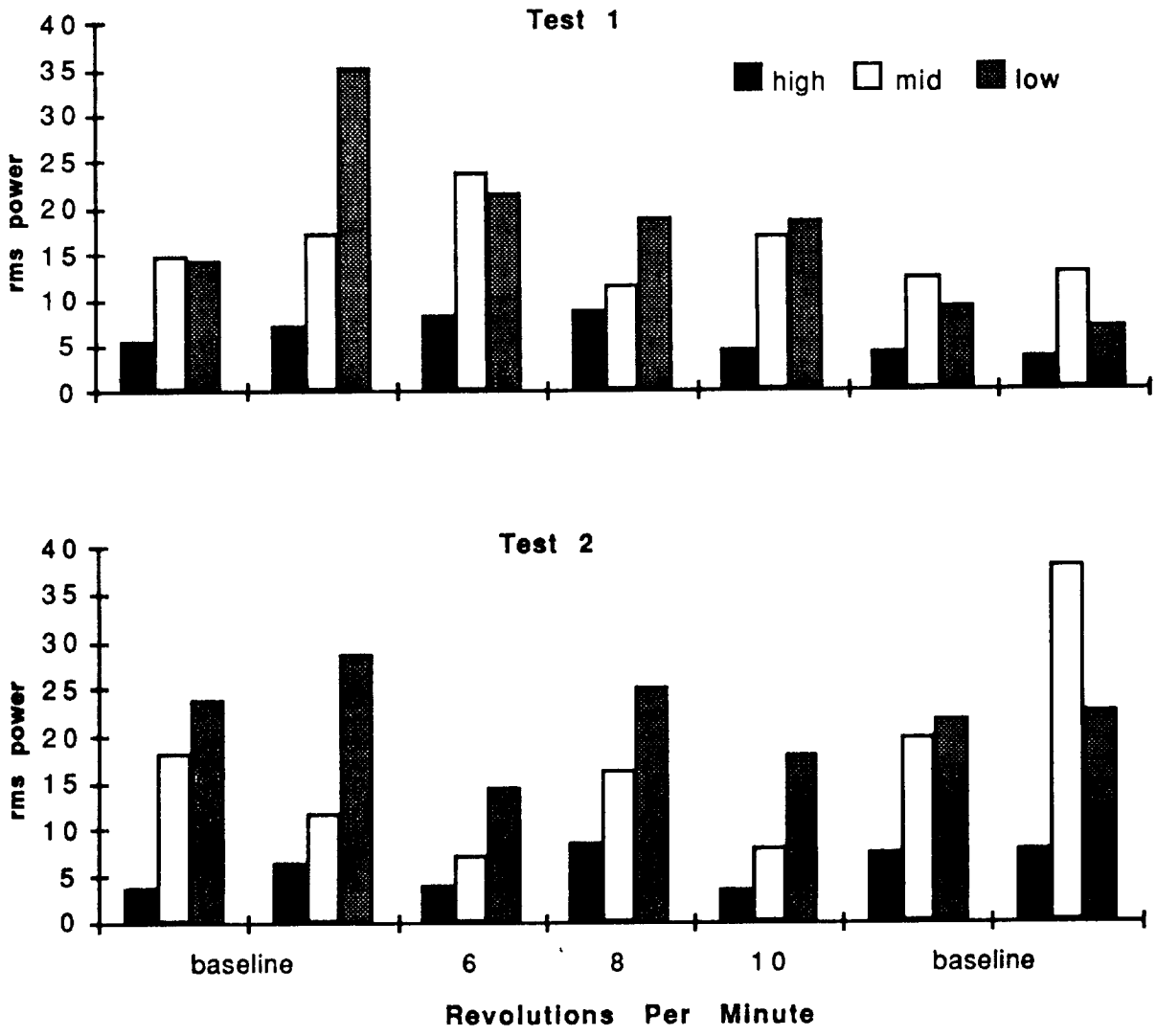
Figure A-58. Heart rate variability across motion sickness tests—subject 6.

C-2



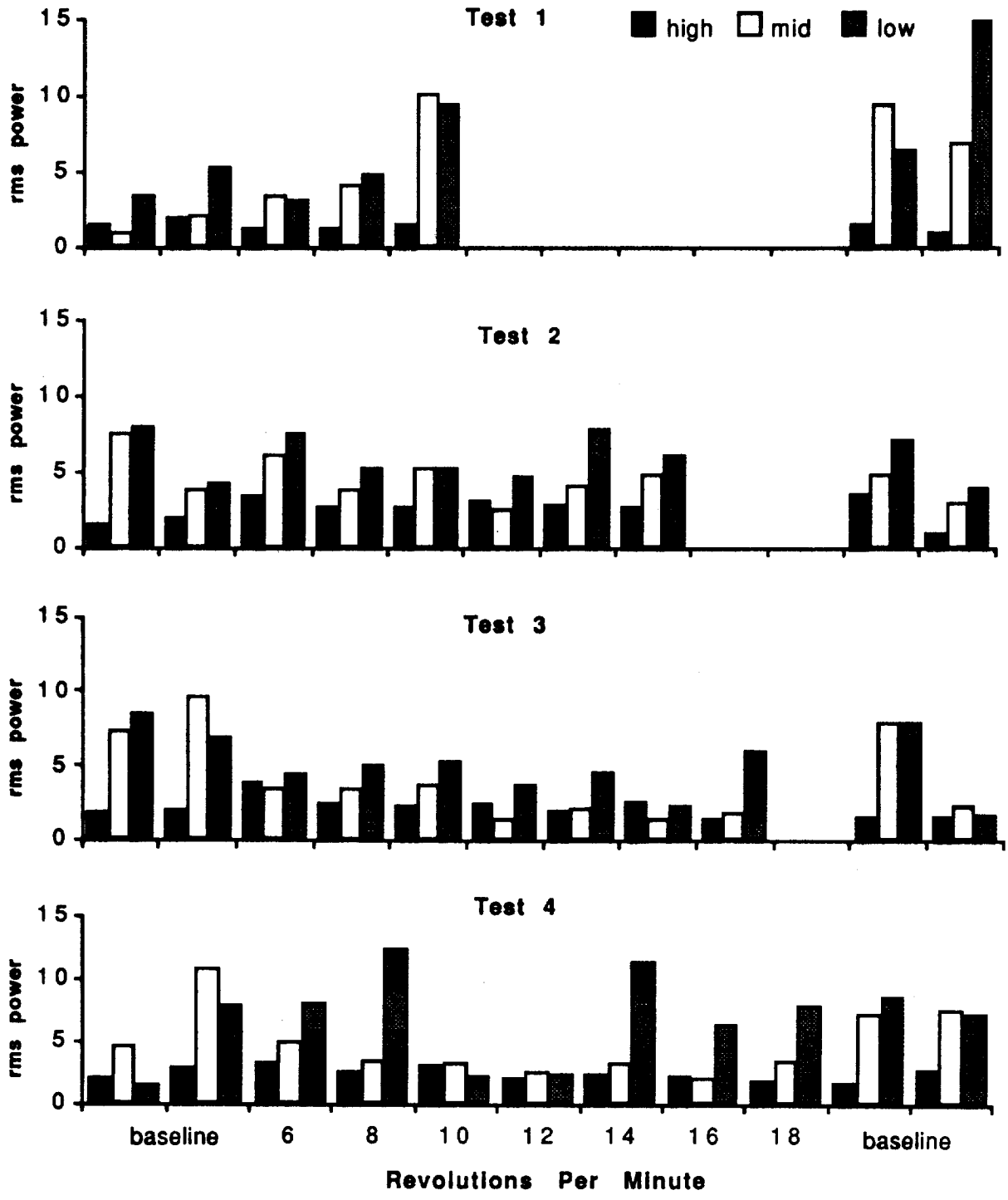
Note: Number of days between tests 1 and 2 = 201; 2 and 3 = 102; 3 and 4 = 57. High = 0 to 0.05 Hz, mid = 0.05 to 0.1 Hz, low = 0.1 to 0.4 Hz.

Figure A-59. Heart rate variability across motion sickness tests—subject 7.



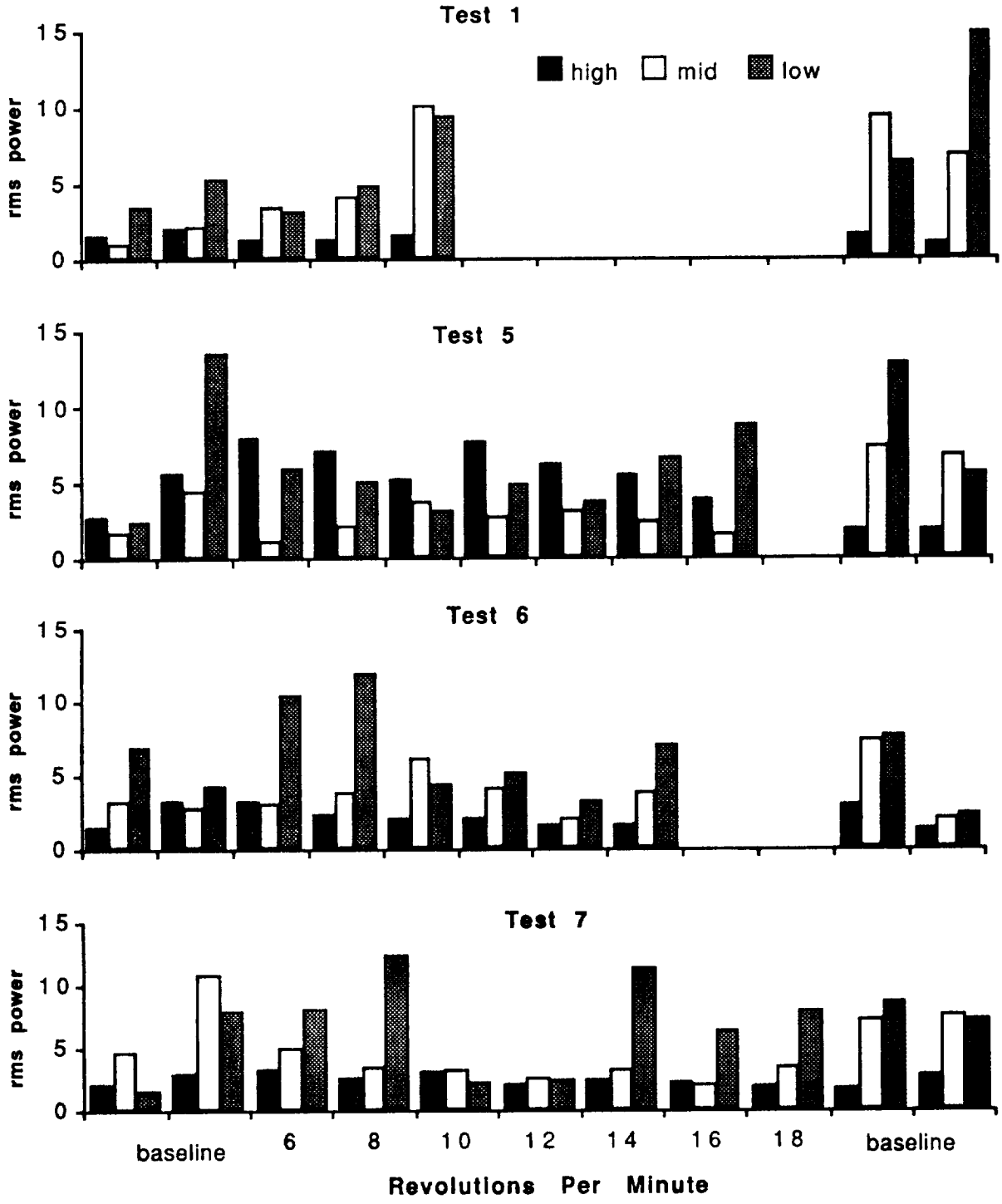
Note: There was a 1-year interval between these "baseline" rotating chair tests. High = 0 to 0.05 Hz, mid = 0.05 to 0.1 Hz, low = 0.1 to 0.4 Hz.

Figure A-60. Heart rate variability during initial motion sickness test—subject 8.



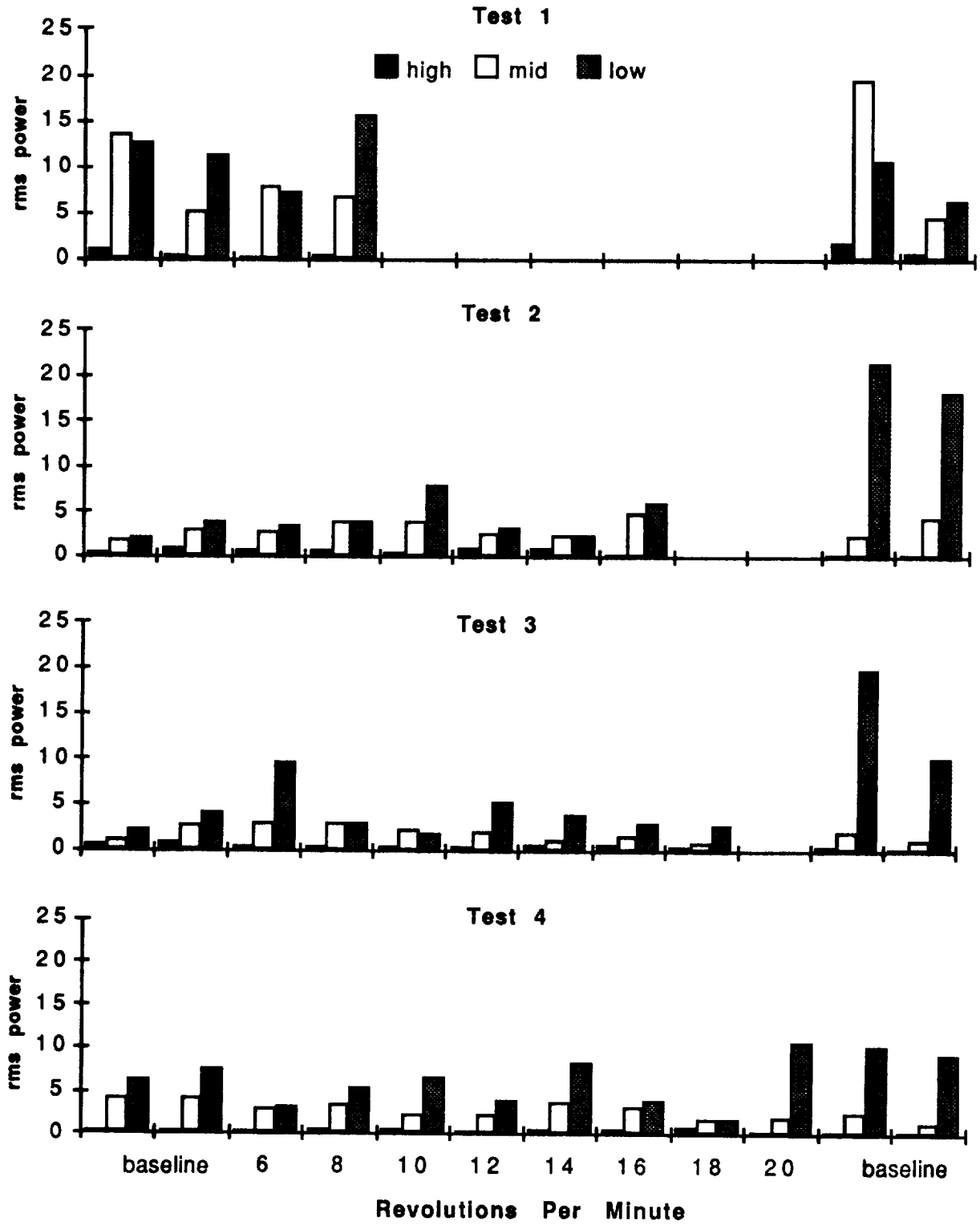
Note: Tests 1–4 were at 1 week intervals. High = 0 to 0.05 Hz, mid = 0.05 to 0.1 Hz, low = 0.1 to 0.4 Hz.

Figure A-61(a). Changes in heart rate variability across motion sickness tests (year 1)—subject 9.



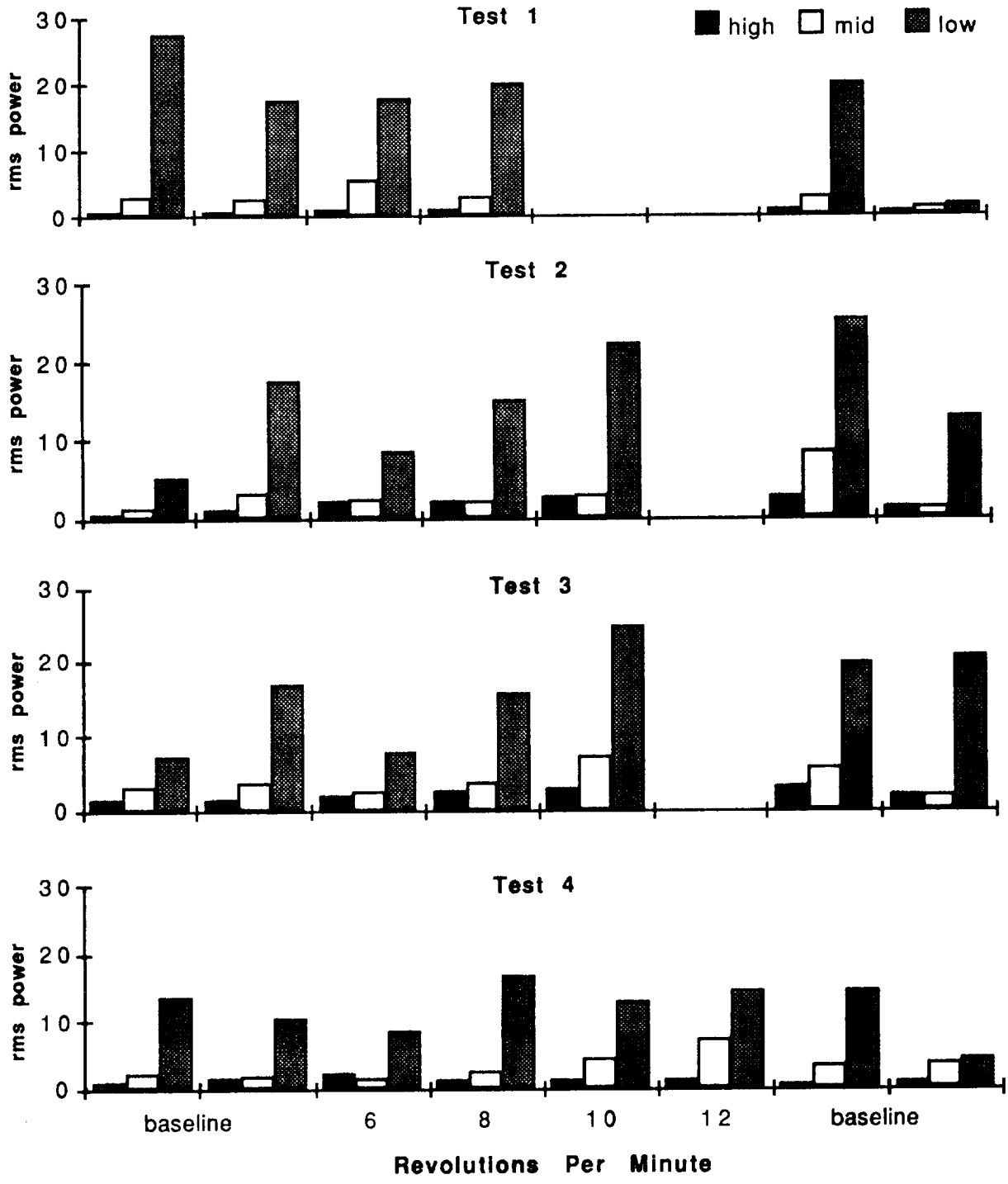
Note: Tests 5–7 were at 1 week intervals. High = 0 to 0.05 Hz, mid = 0.05 to 0.1 Hz, low = 0.1 to 0.4 Hz.

Figure A-61(b). Changes in heart rate variability across motion sickness tests (year 2)—subject 9.



Note: Number of days between tests 1 and 2 = 189; 2 and 3 = 105; 3 and 4 = 34. High = 0 to 0.05 Hz, mid = 0.05 to 0.1 Hz, low = 0.1 to 0.4 Hz.

Figure A-62. Changes in heart rate variability across motion sickness tests—subject 10.



Note: Number of days between tests 1 and 2 = 173; 2 and 3 = 125; 3 and 4 = 31. High = 0 to 0.05 Hz, mid = 0.05 to 0.1 Hz, low = 0.1 to 0.4 Hz.

Figure A-63. Changes in heart rate variability across motion sickness tests—subject 11.

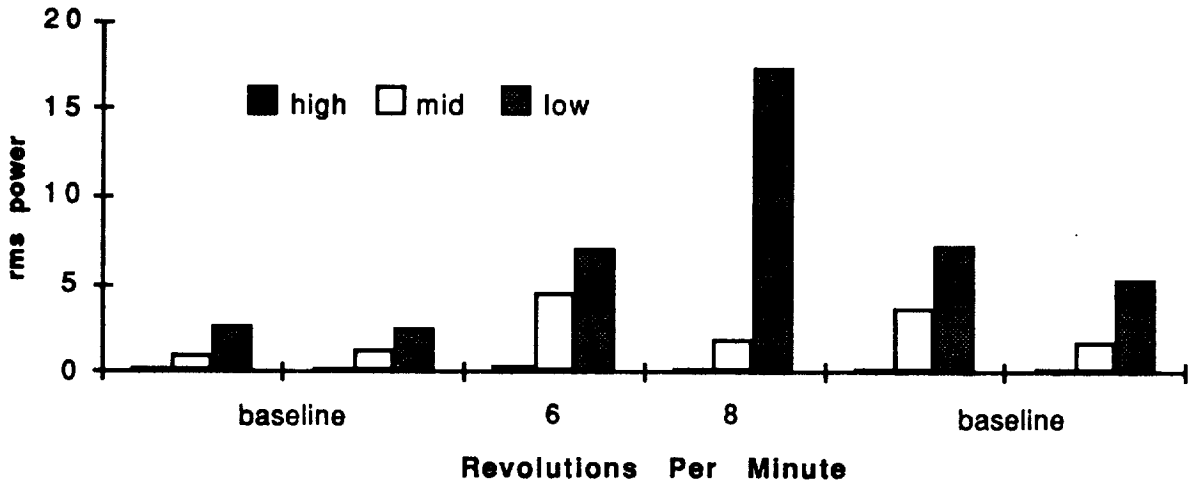
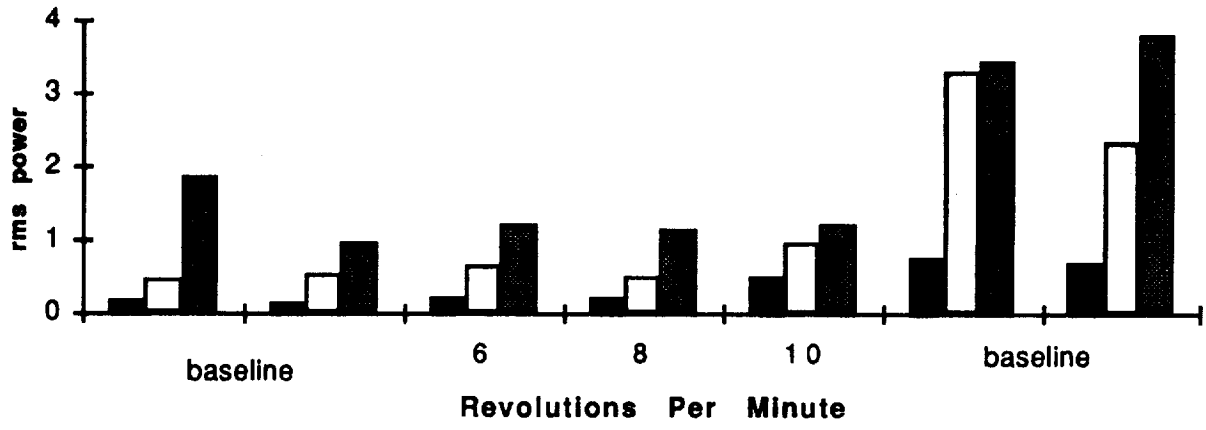


Figure A-64. Heart rate variability during initial motion sickness test—subject 12.

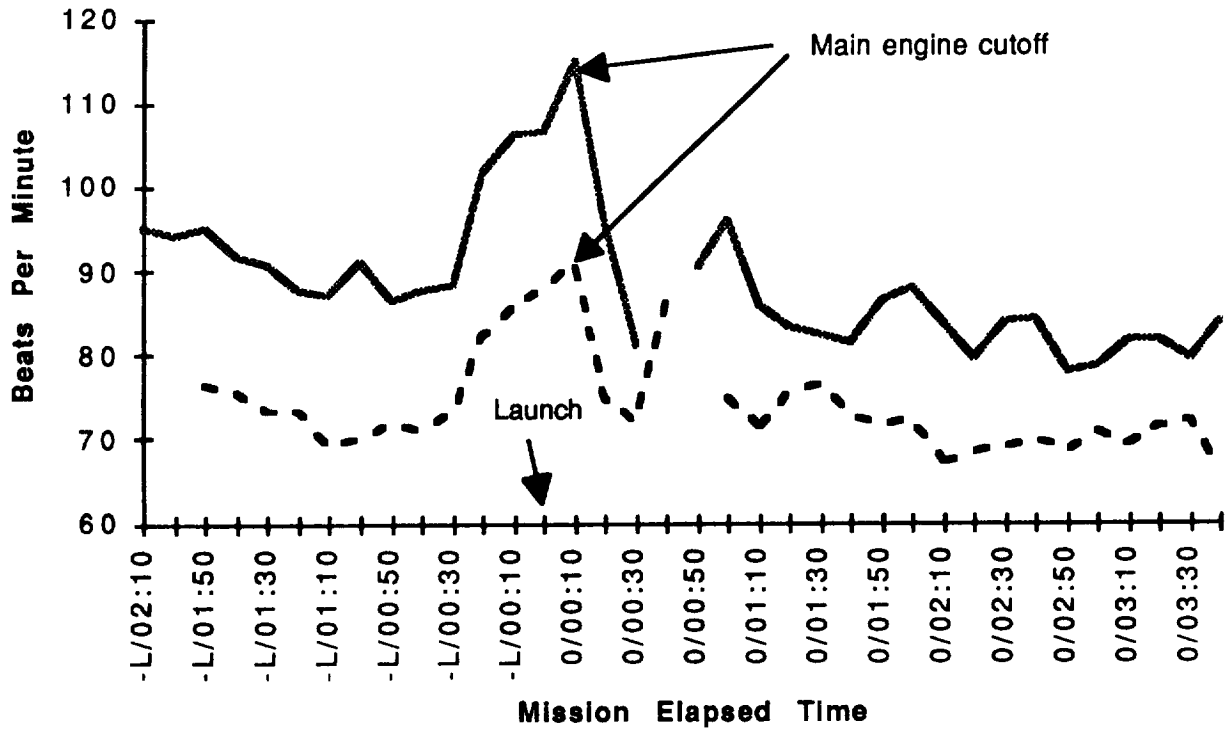


Note: High = 0 to 0.05 Hz, mid = 0.05 to 0.1 Hz, low = 0.1 to 0.4 Hz.

Figure A-65. Heart rate variability during initial motion sickness test—subject 13.

Appendix B

Individual Physiological Data During Spaceflight and in Earth-Based Mission Simulations



Note: Physiological recordings of crewmembers were initiated within the Shuttle 2 hours and 10 minutes before the launch (–L/02:10) and continued for the first 3 hours and 30 minutes in space (0/03:30), when these crewmembers retired for the evening. The brief (10-minute) interruption in data occurred as subjects disconnected recording instrumentation to remove their launch-entry suits.

Figure B-1. Heart rate data of two crewmembers: launch day.

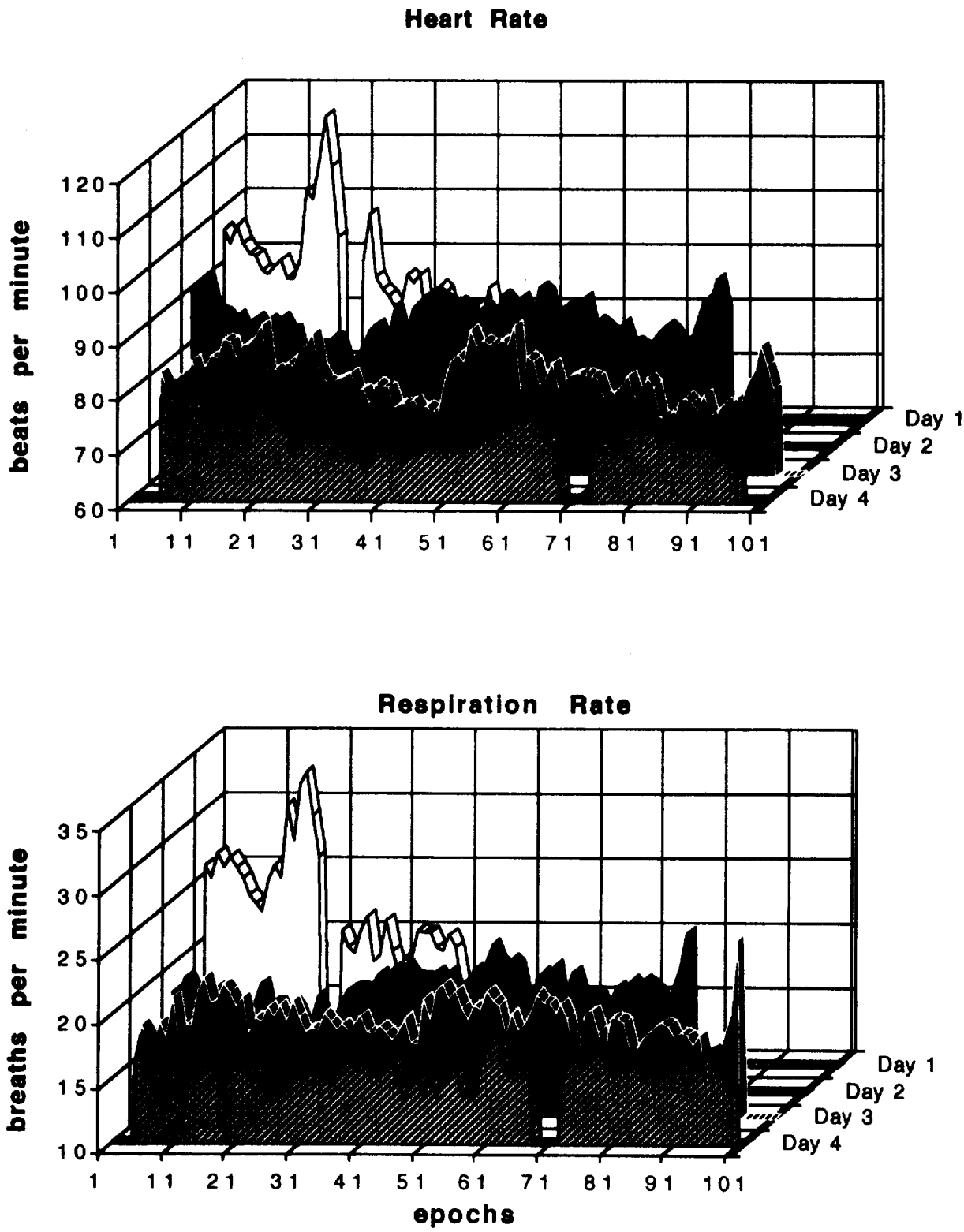


Figure B-2. Heart rate and respiration rate changes during early adaptation to microgravity—subject 8.

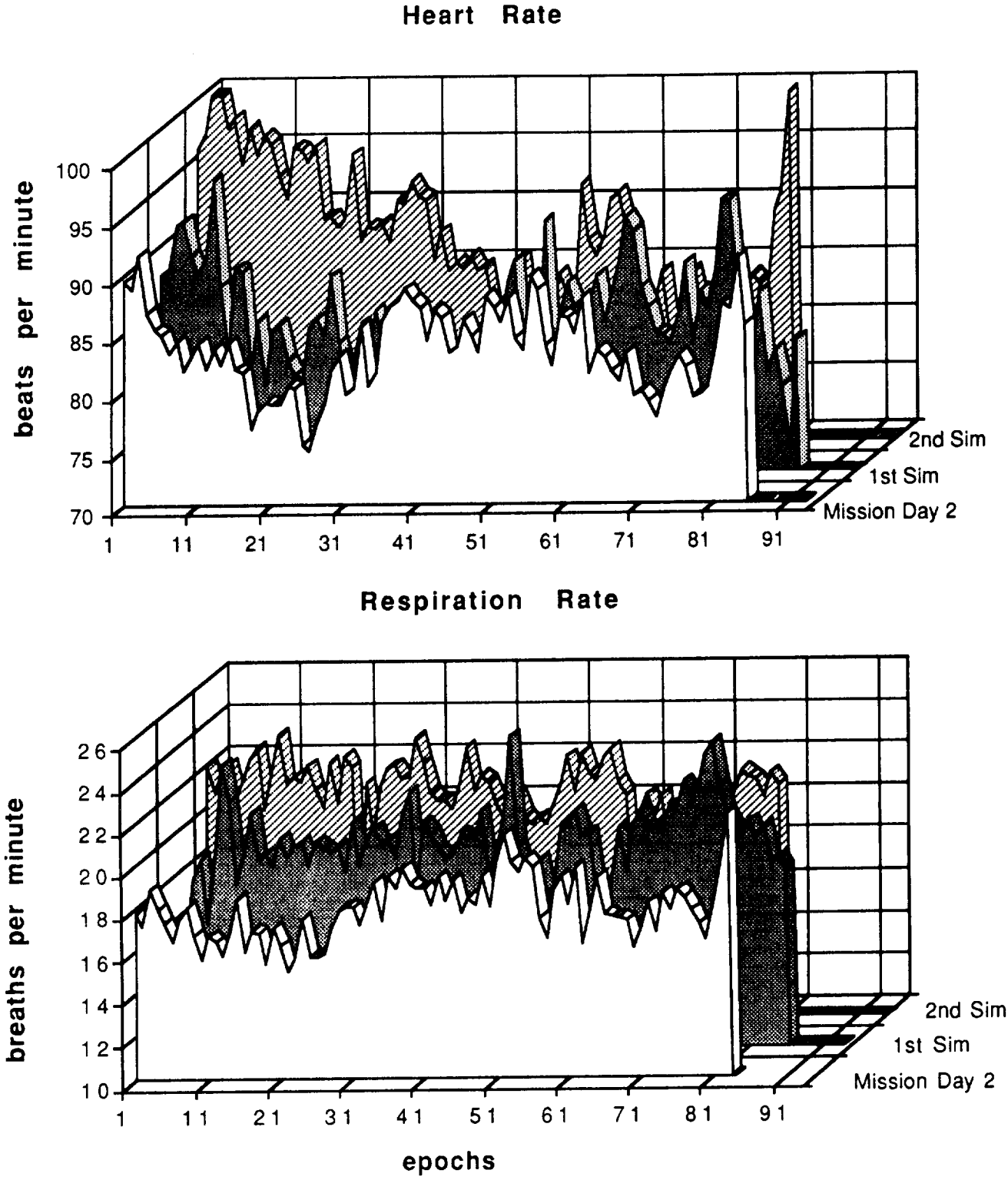


Figure B-3. Heart rate and respiration rate in space vs. Earth-based simulations—subject 8.

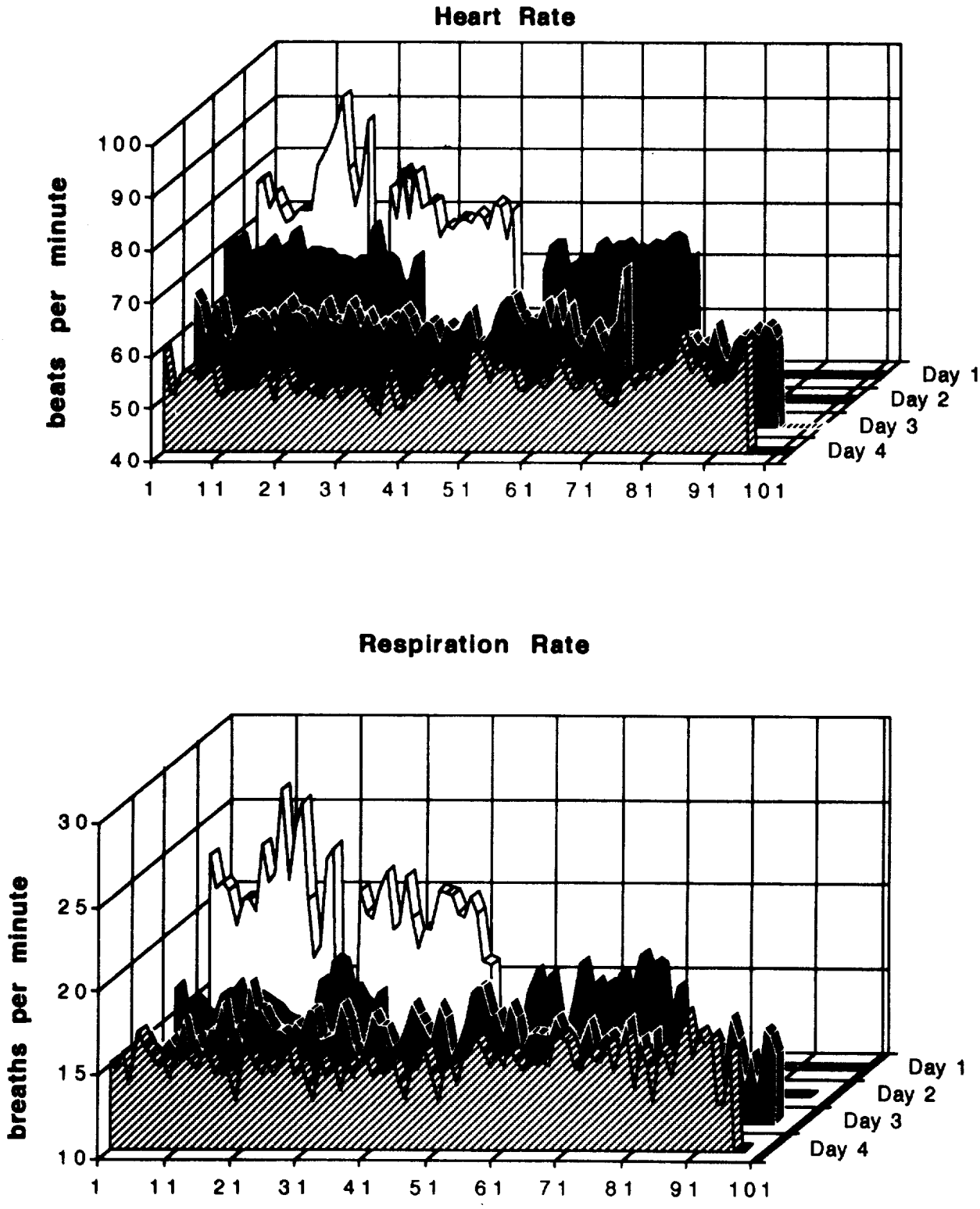


Figure B-4. Heart rate and respiration rate changes during early adaptation to microgravity—subject 9.

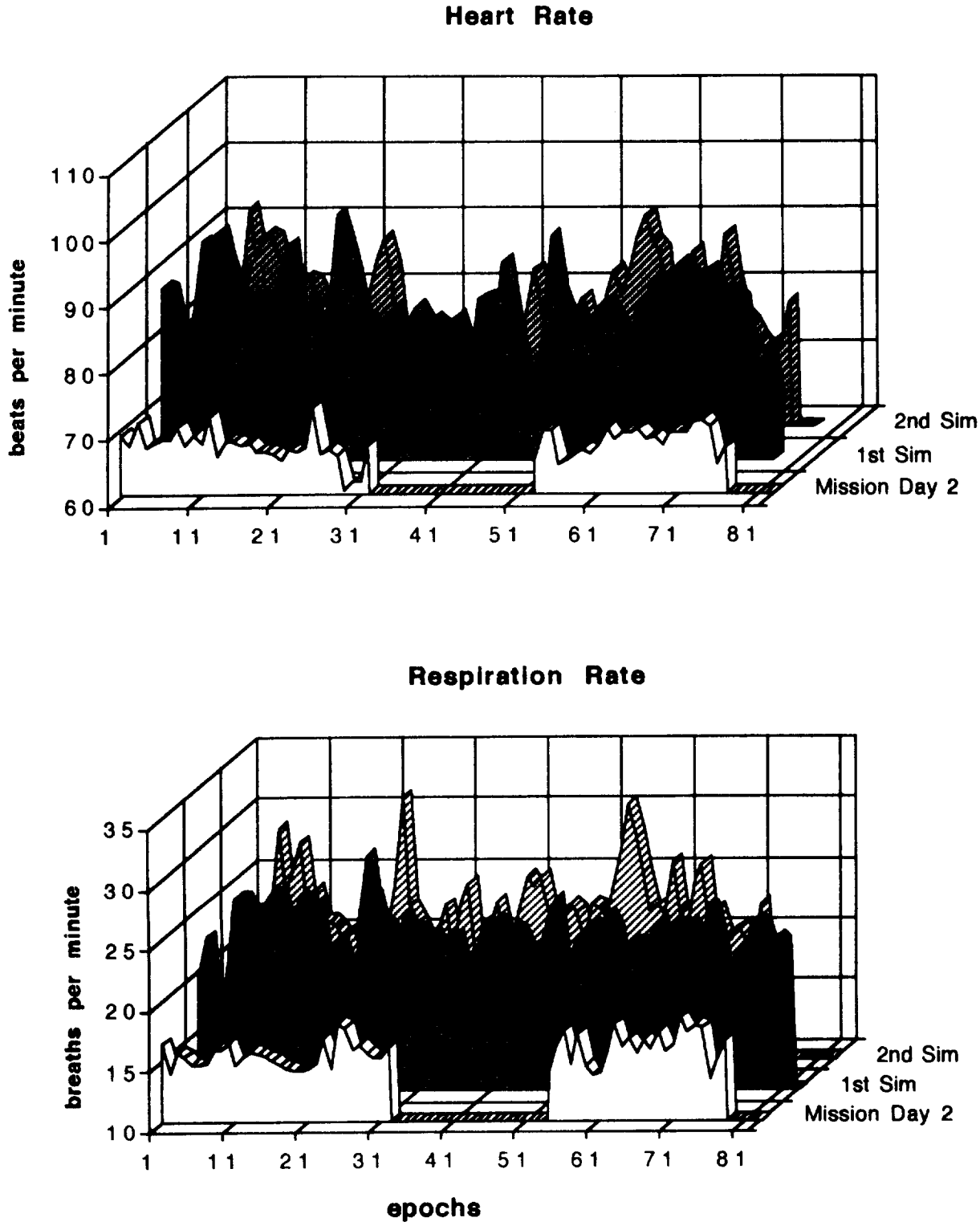


Figure B-5. Heart rate and respiration rate in space vs. Earth-based simulations—subject 9.

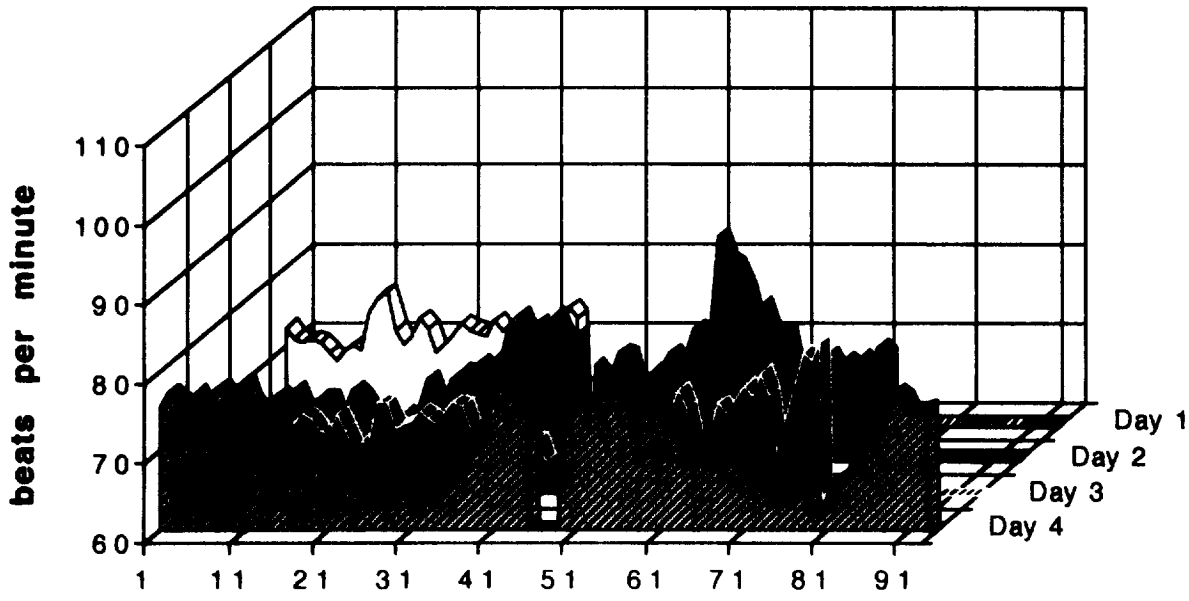


Figure B-6. Heart rate changes during early adaptation to microgravity—subject 10.

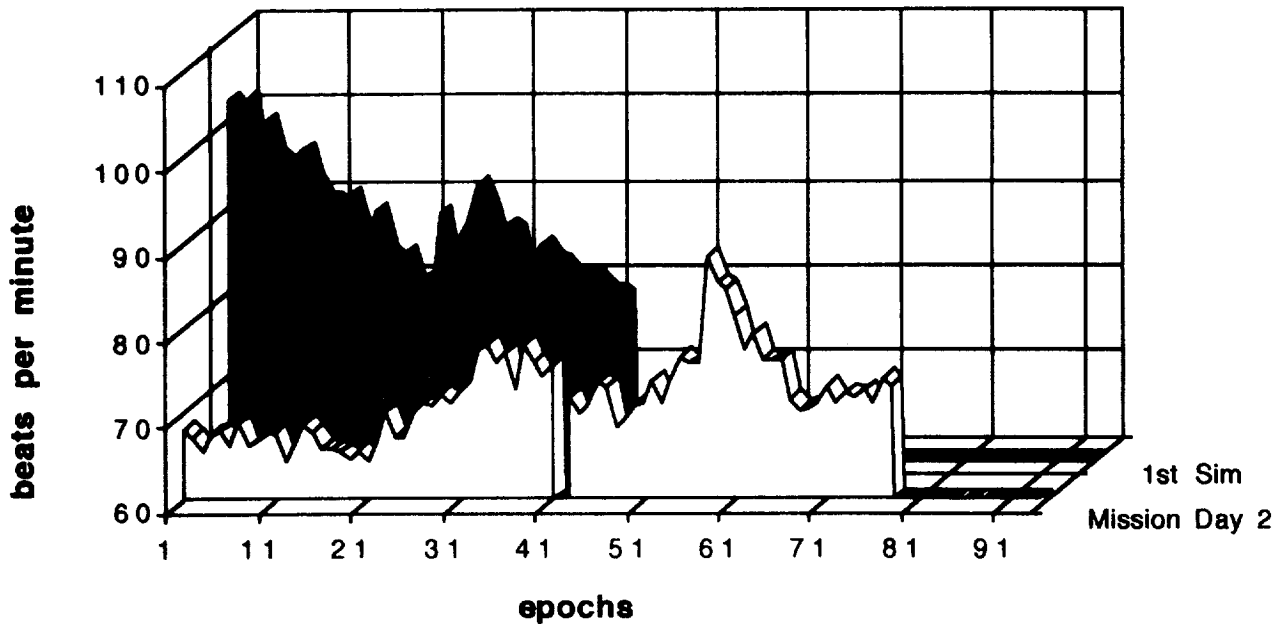


Figure B-7. Heart rate in space vs. Earth-based simulations—subject 10.

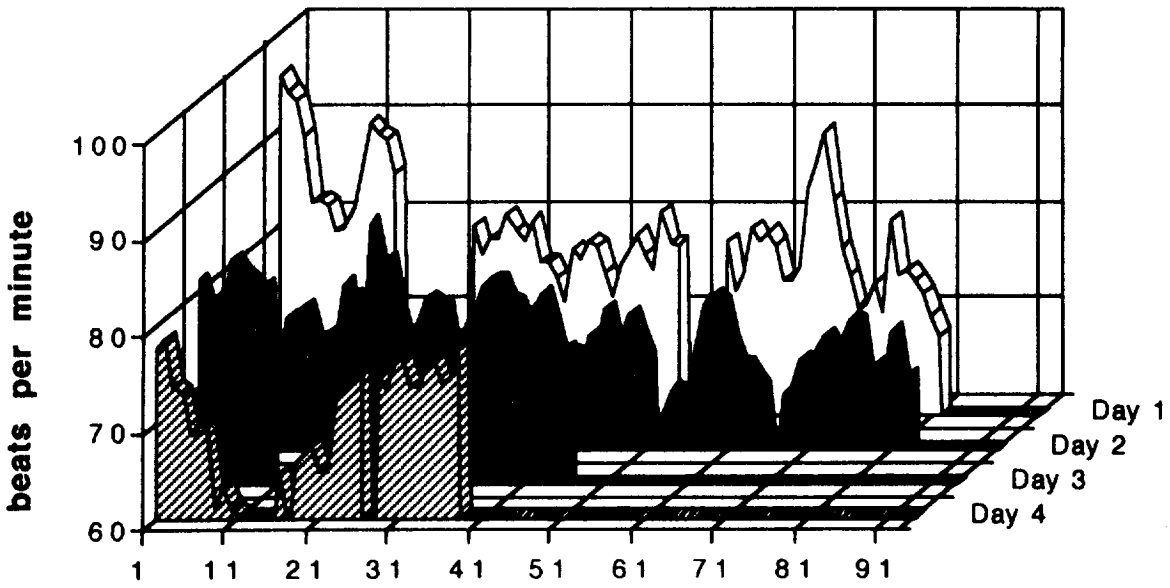


Figure B-8. Heart rate changes during early adaptation to microgravity—subject 11.

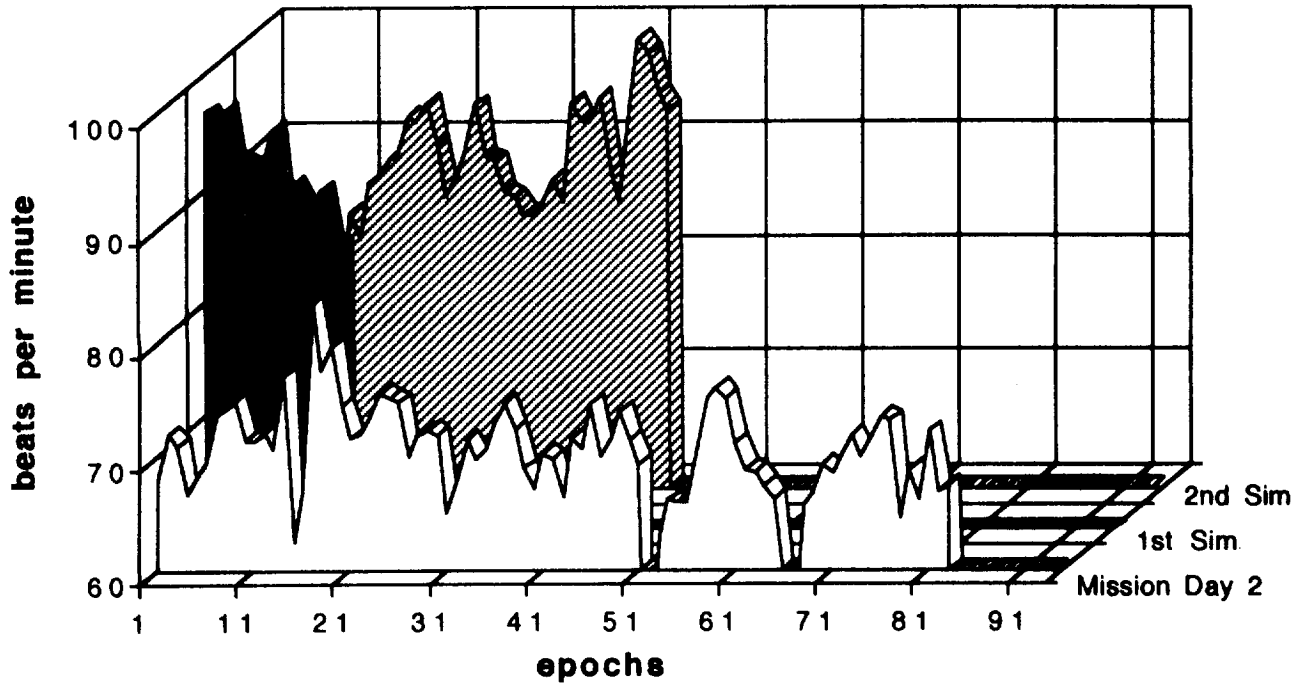


Figure B-9. Heart rate in space vs. Earth-based simulations—subject 11.

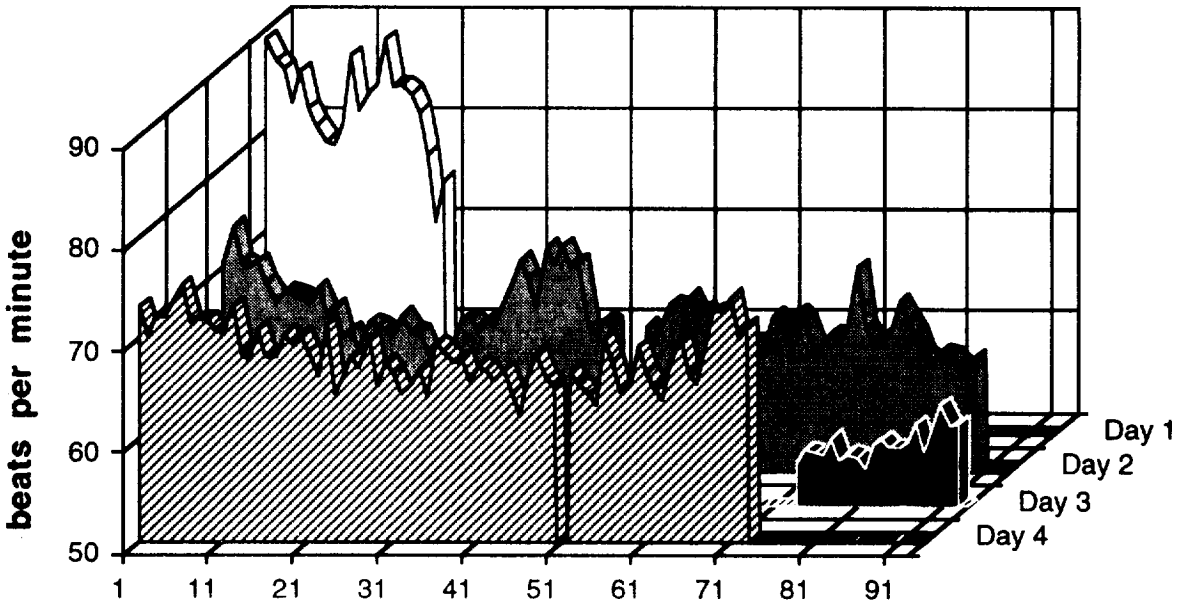


Figure B-10. Heart rate changes during early adaptation to microgravity—subject 12.

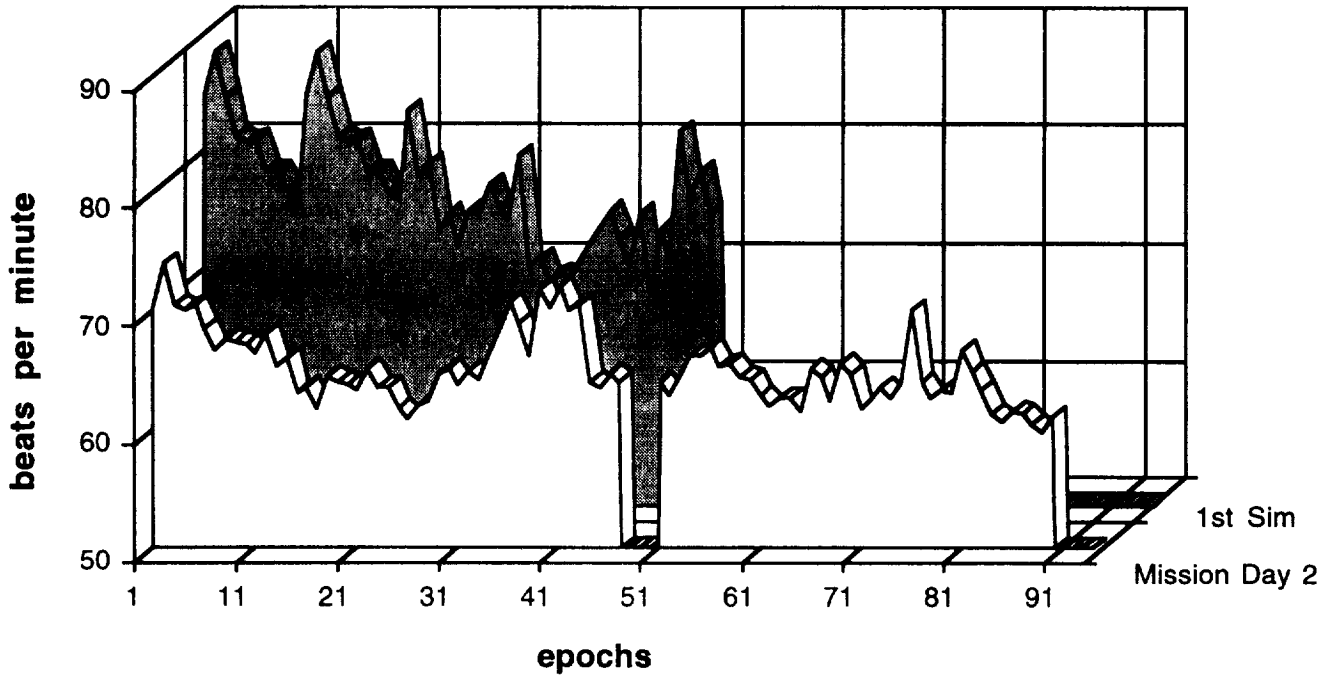


Figure B-11. Heart rate in space vs. Earth-based simulations—subject 12.

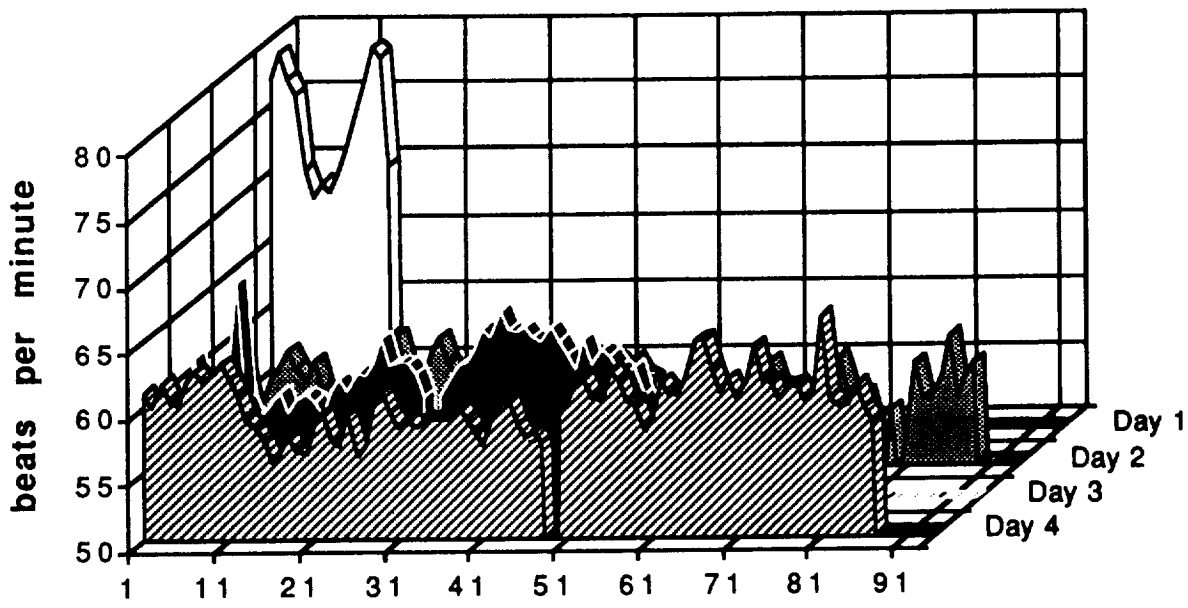


Figure B-12. Heart rate changes during early adaptation to microgravity—subject 13.

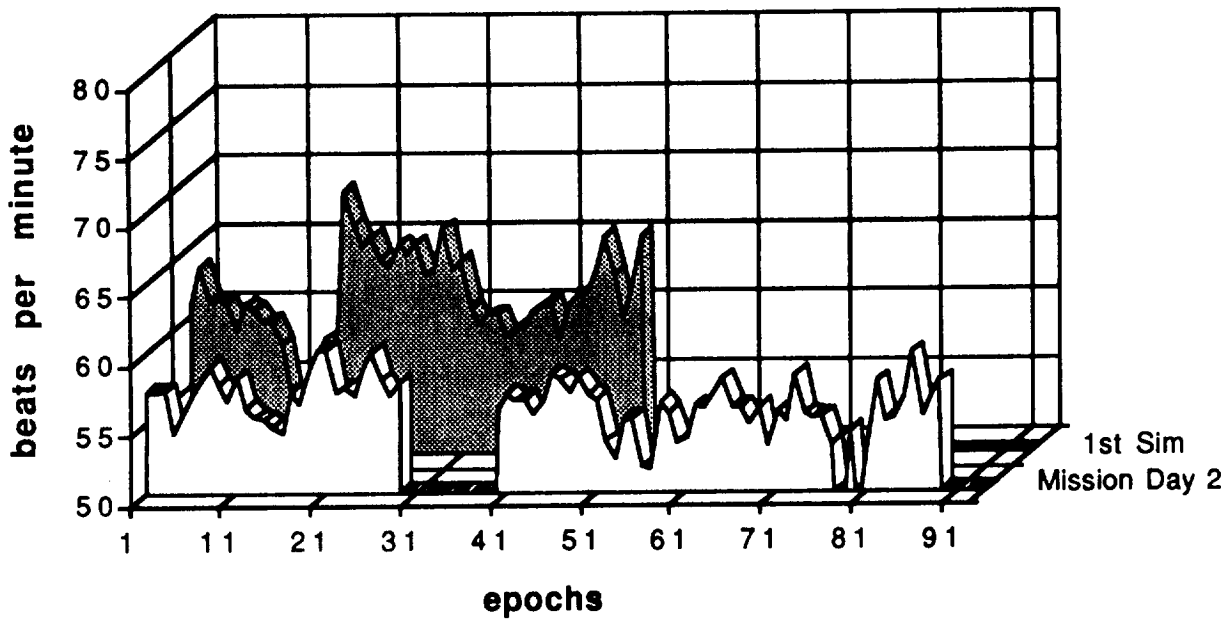


Figure B-13. Heart rate in space vs. Earth-based simulations—subject 13.

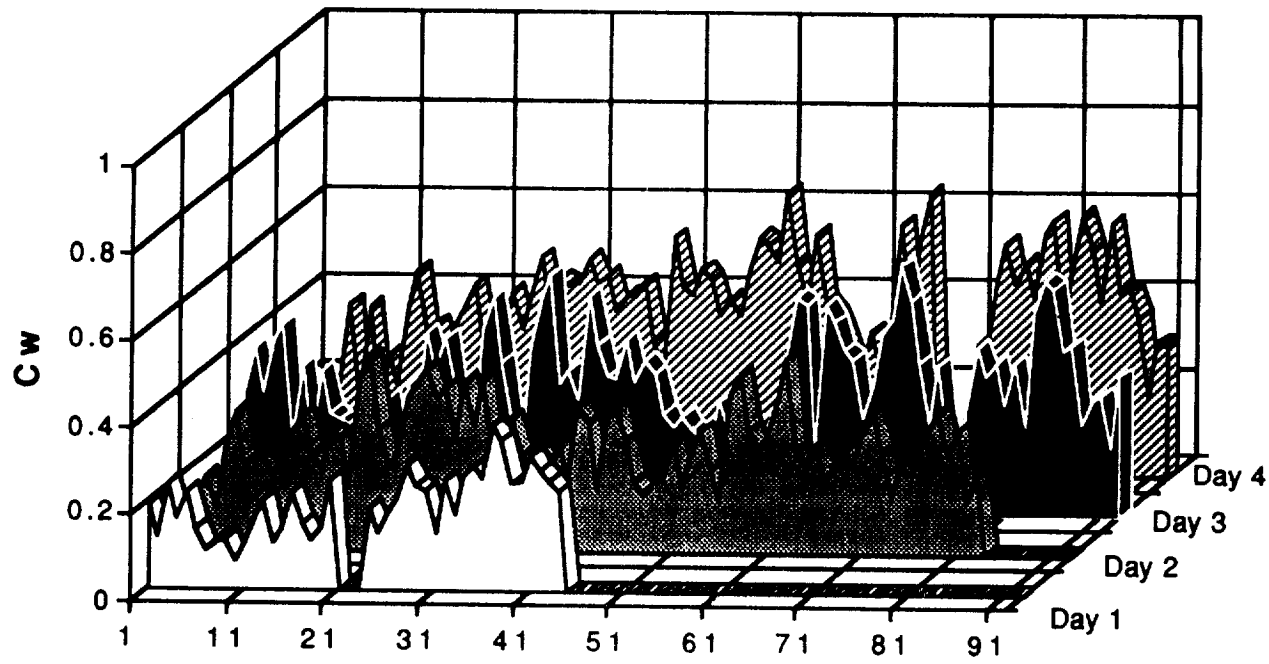


Figure B-14. Coherence between heart rate and respiration during early adaptation to microgravity—subject 8.

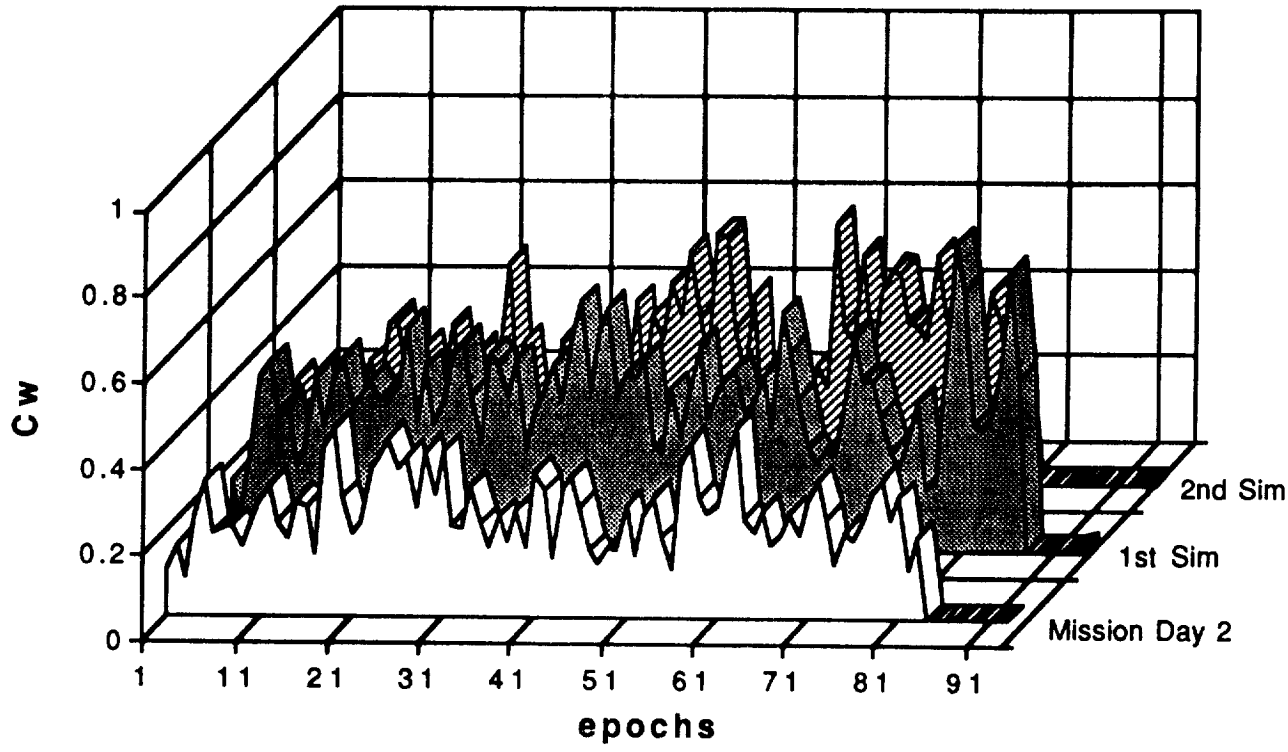


Figure B-15. Coherence between heart rate and respiration in space vs. Earth-based simulation—subject 8.

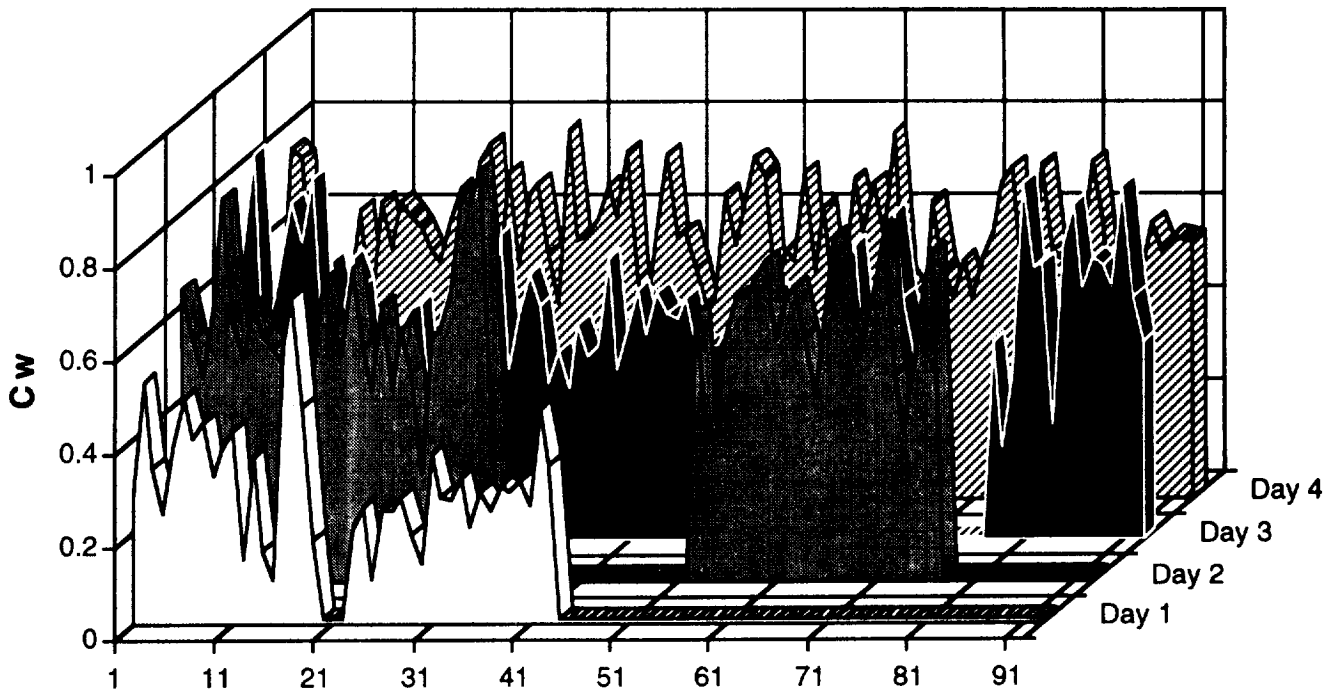


Figure B-16. Coherence between heart rate and respiration during early adaptation to microgravity—subject 9.

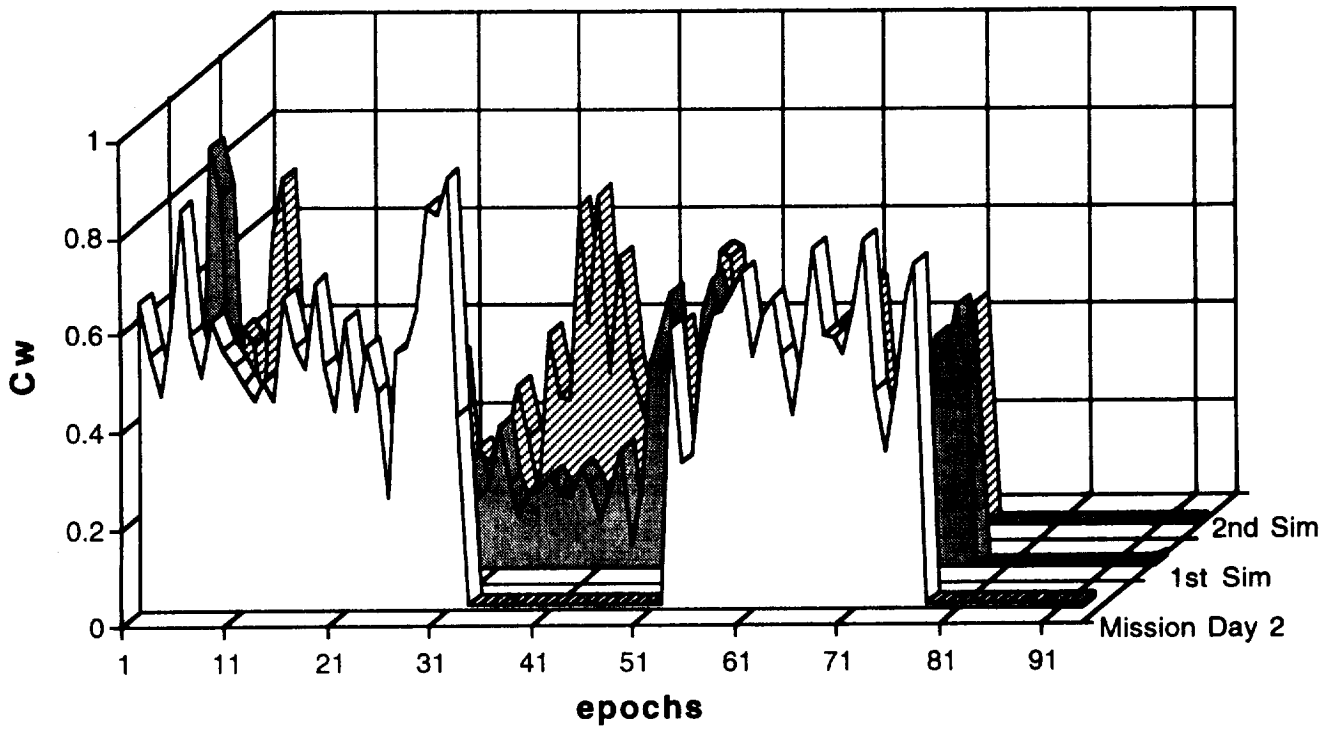


Figure B-17. Coherence between heart rate and respiration in space vs. Earth-based simulation—subject 9.

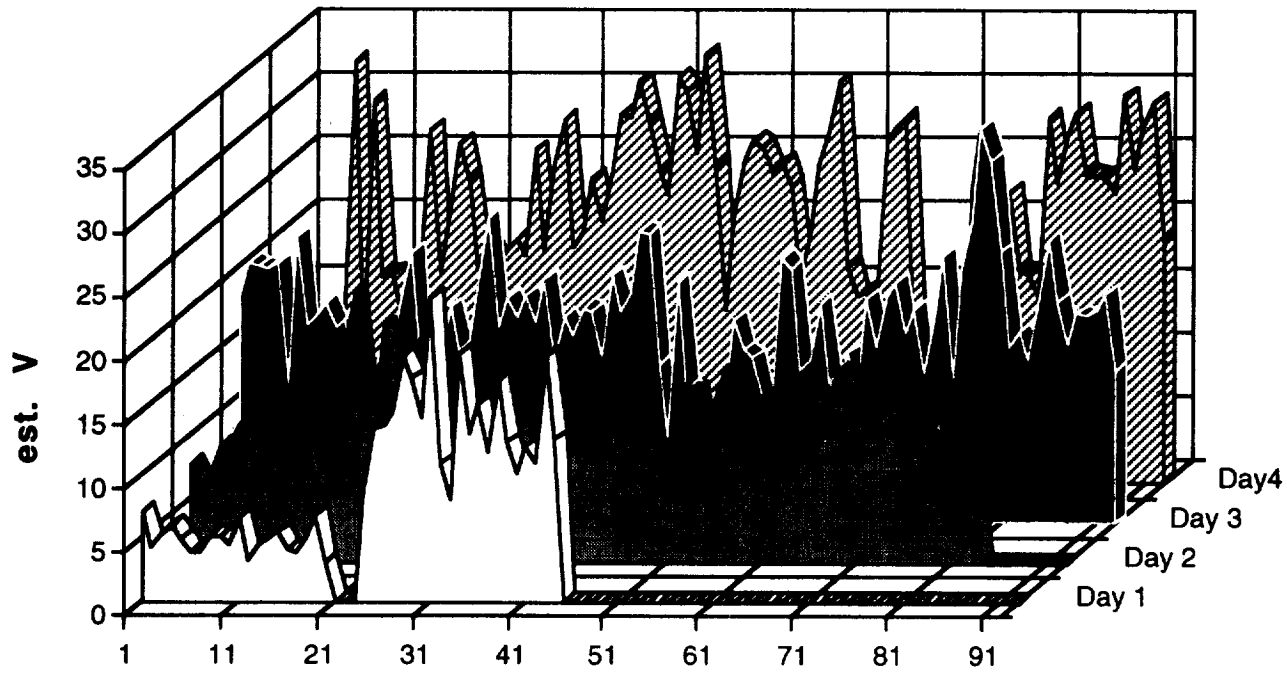
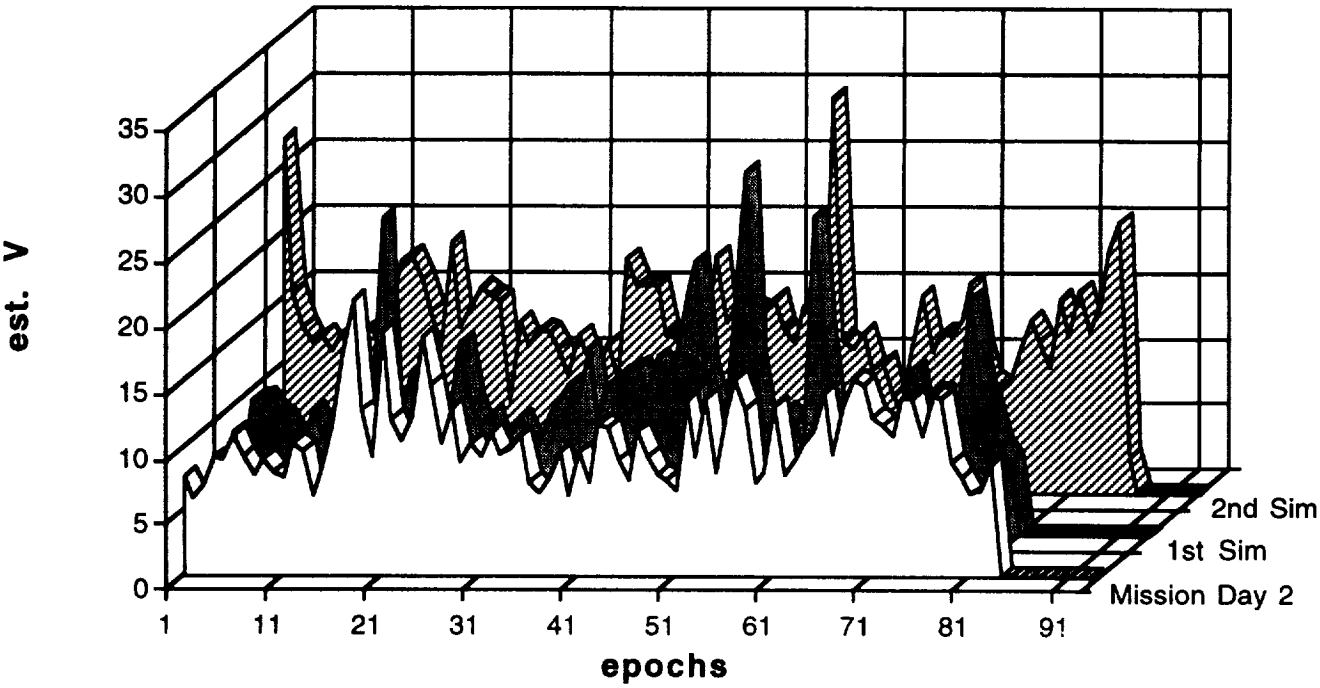


Figure B-18. Estimate of vagal tone in space—subject 8.



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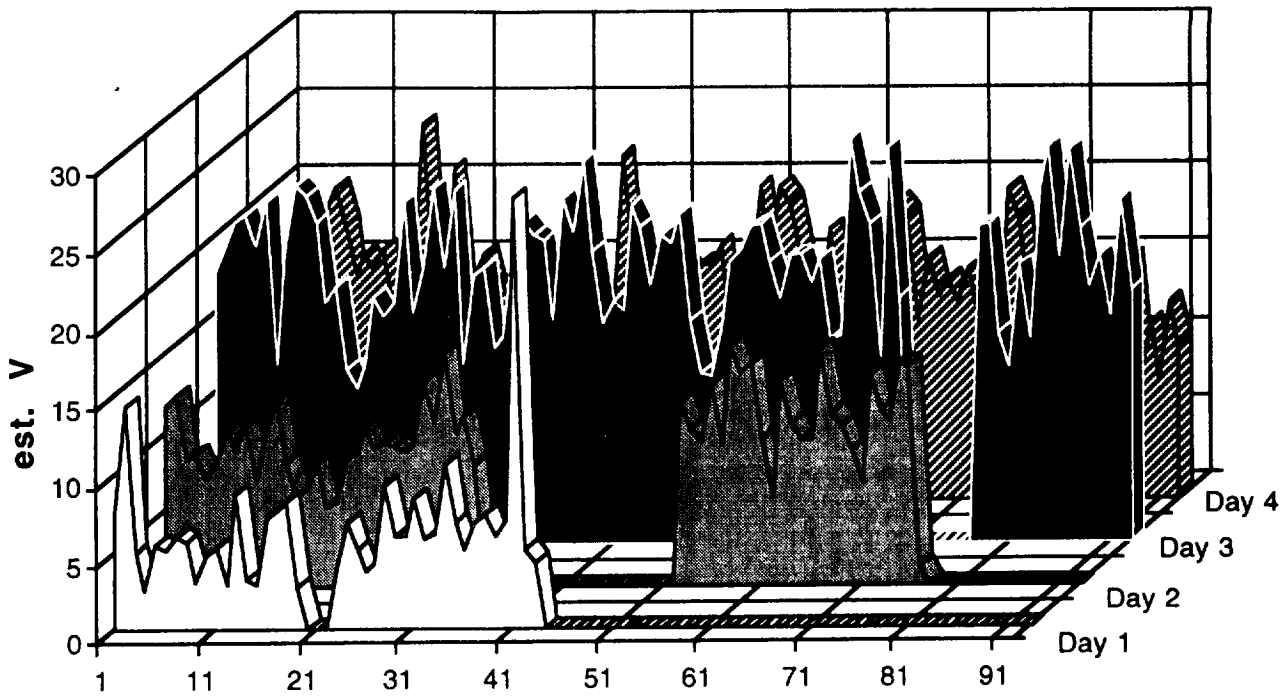

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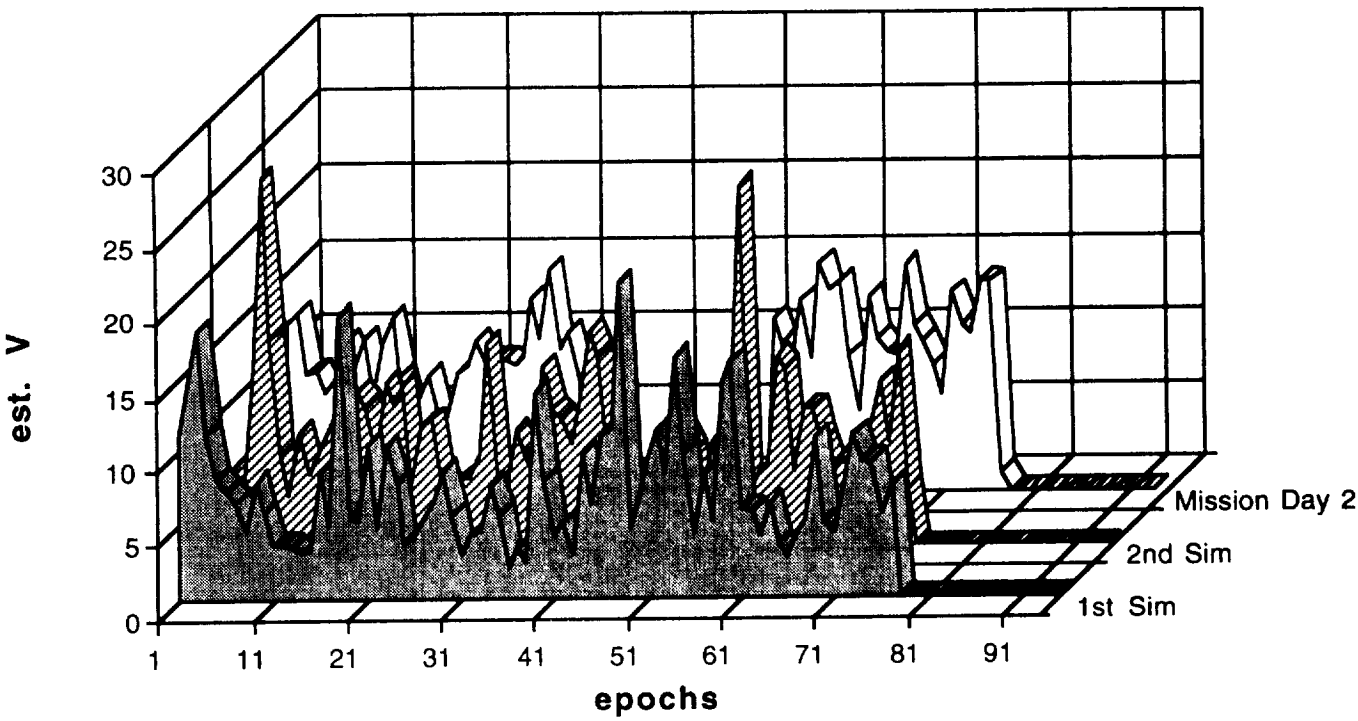

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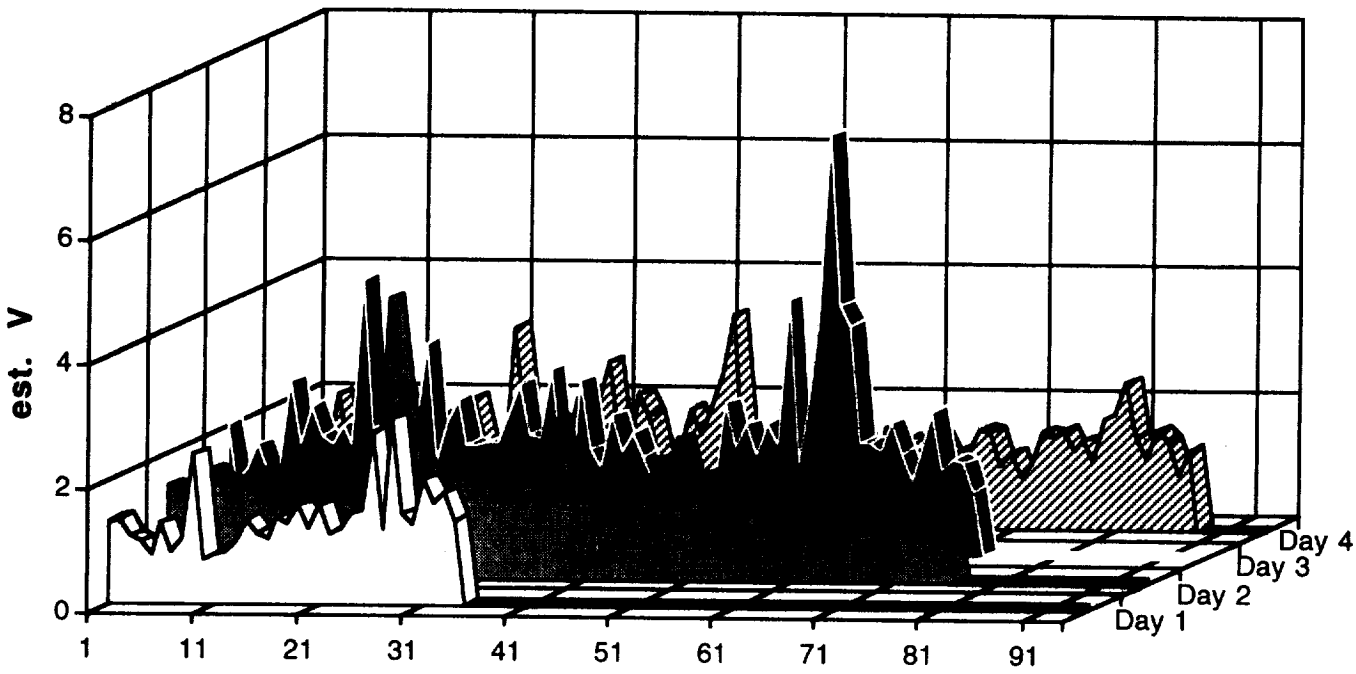
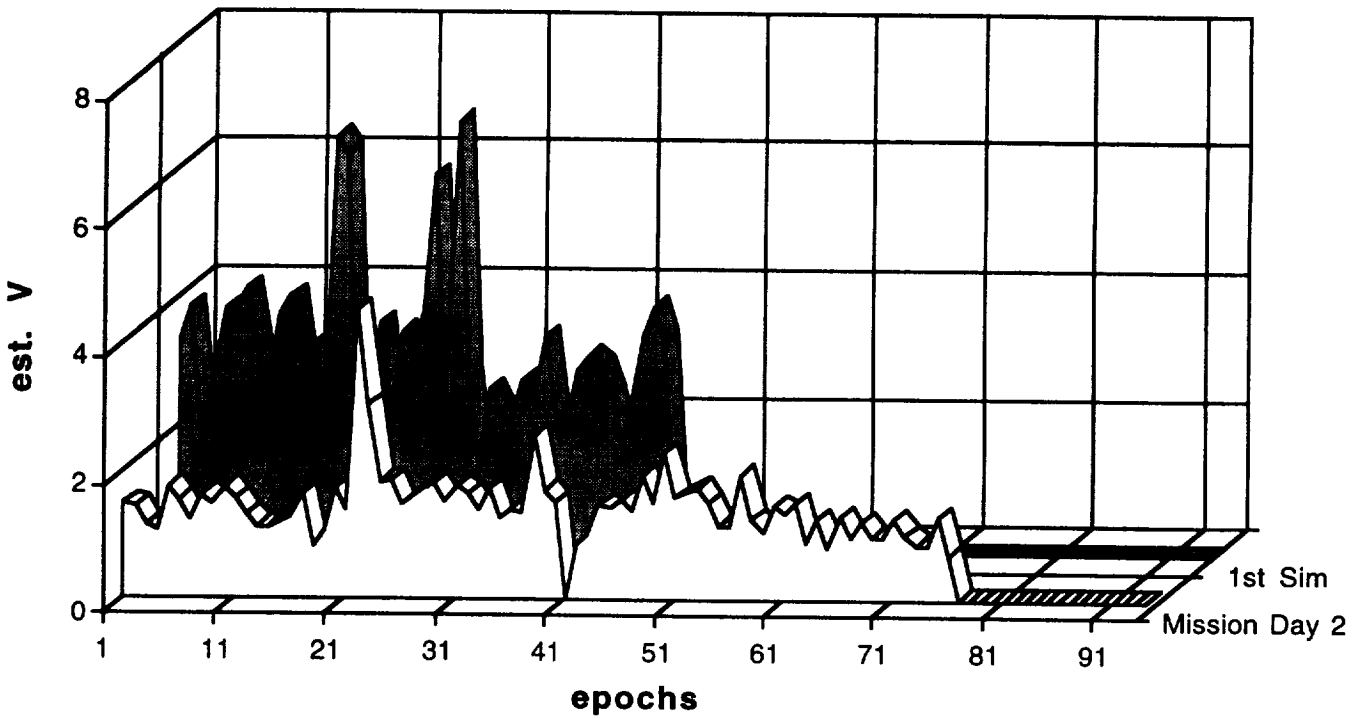


Figure B-22. Estimate of vagal tone in space—subject 10.



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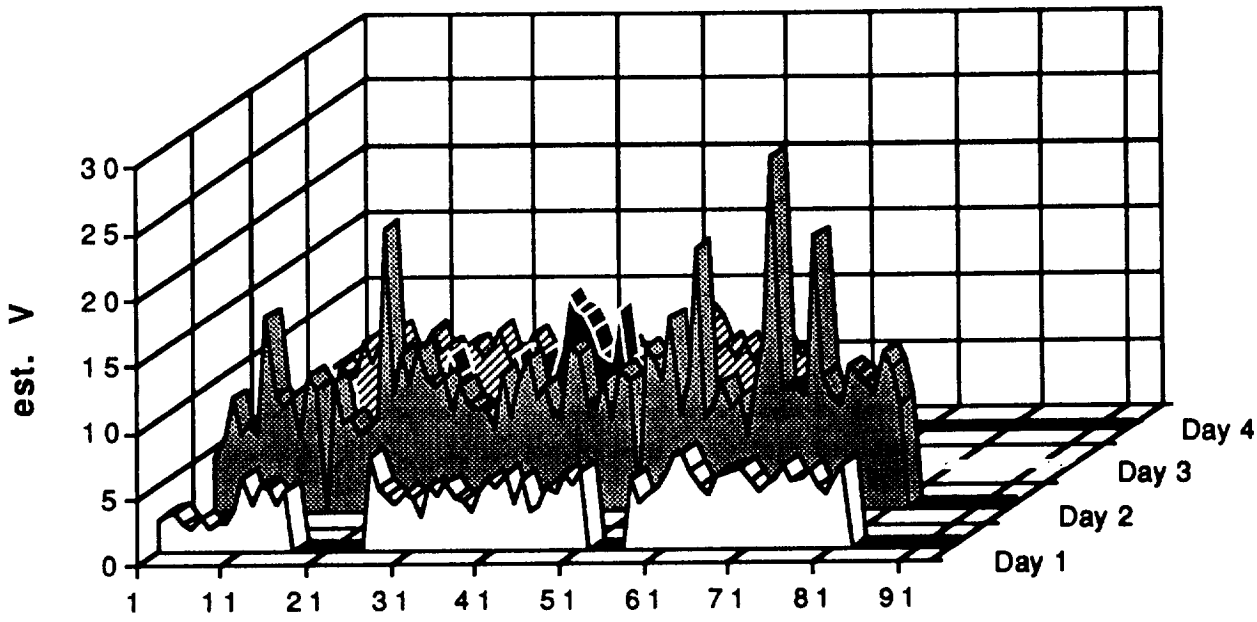

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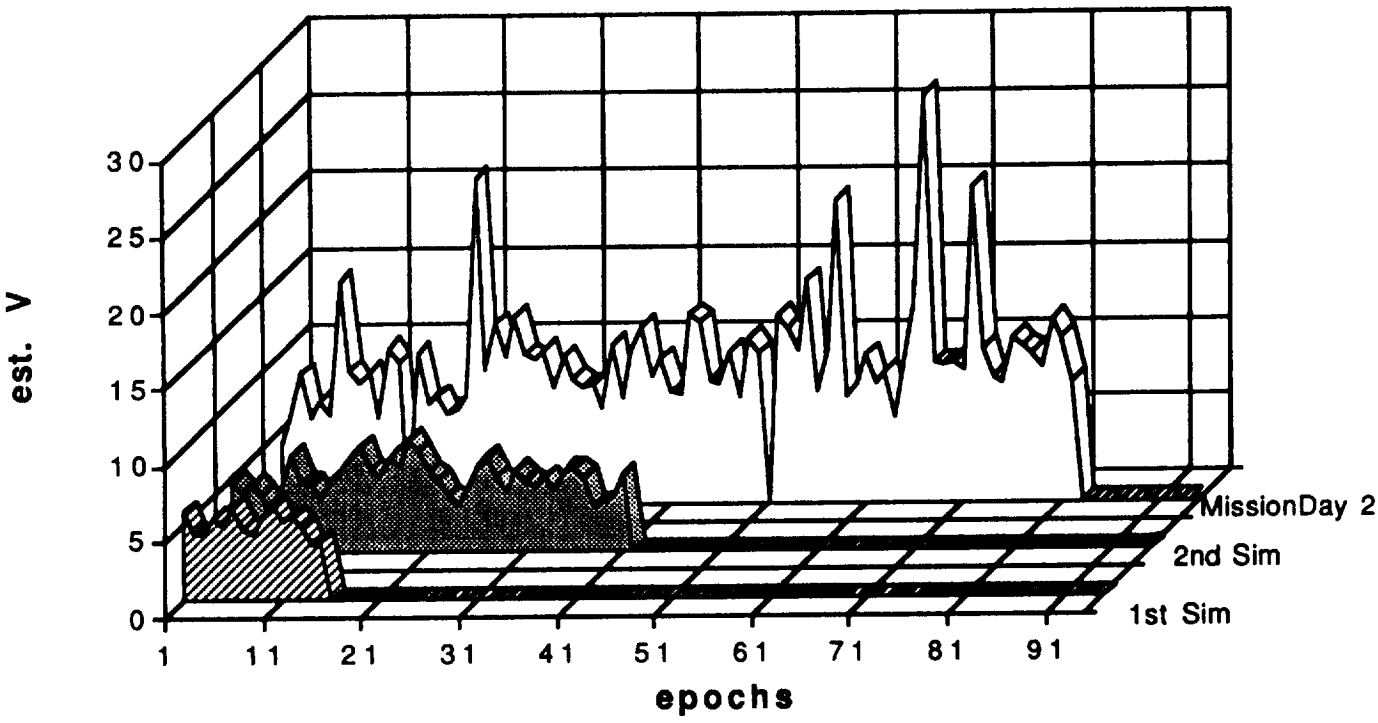

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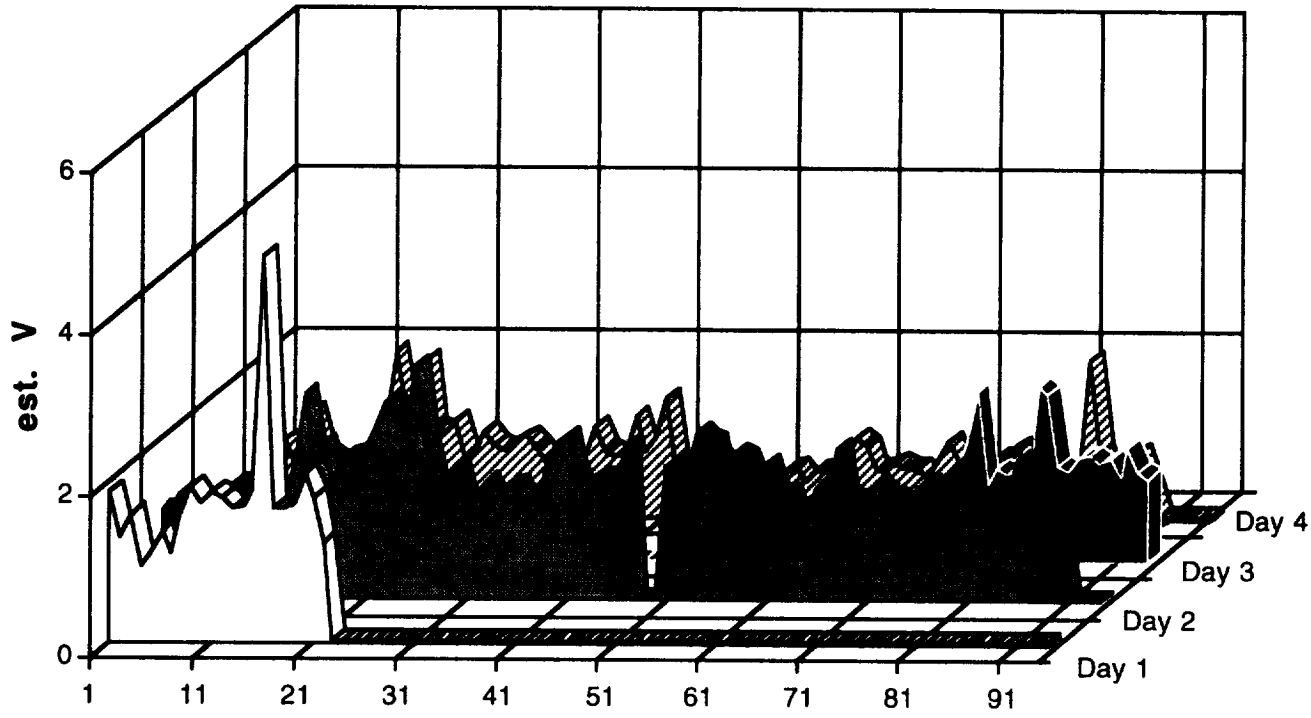
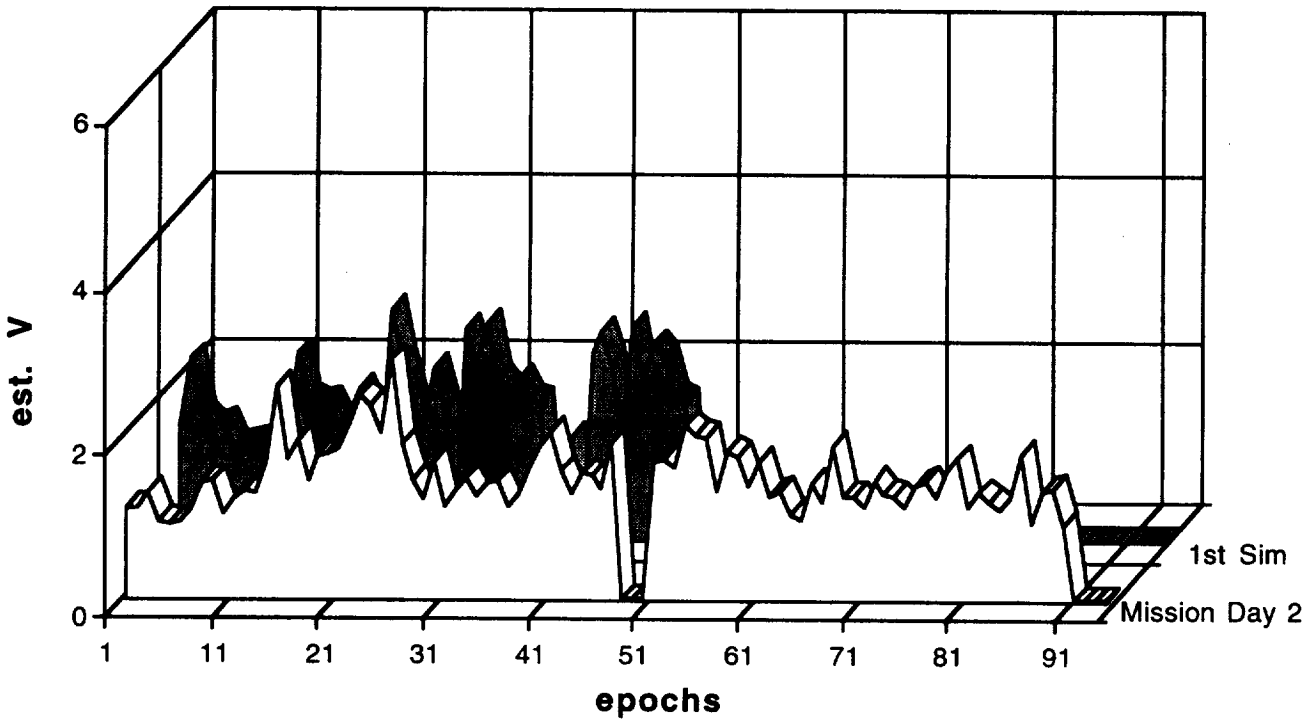


Figure B-26. Estimate of vagal tone in space—subject 12.



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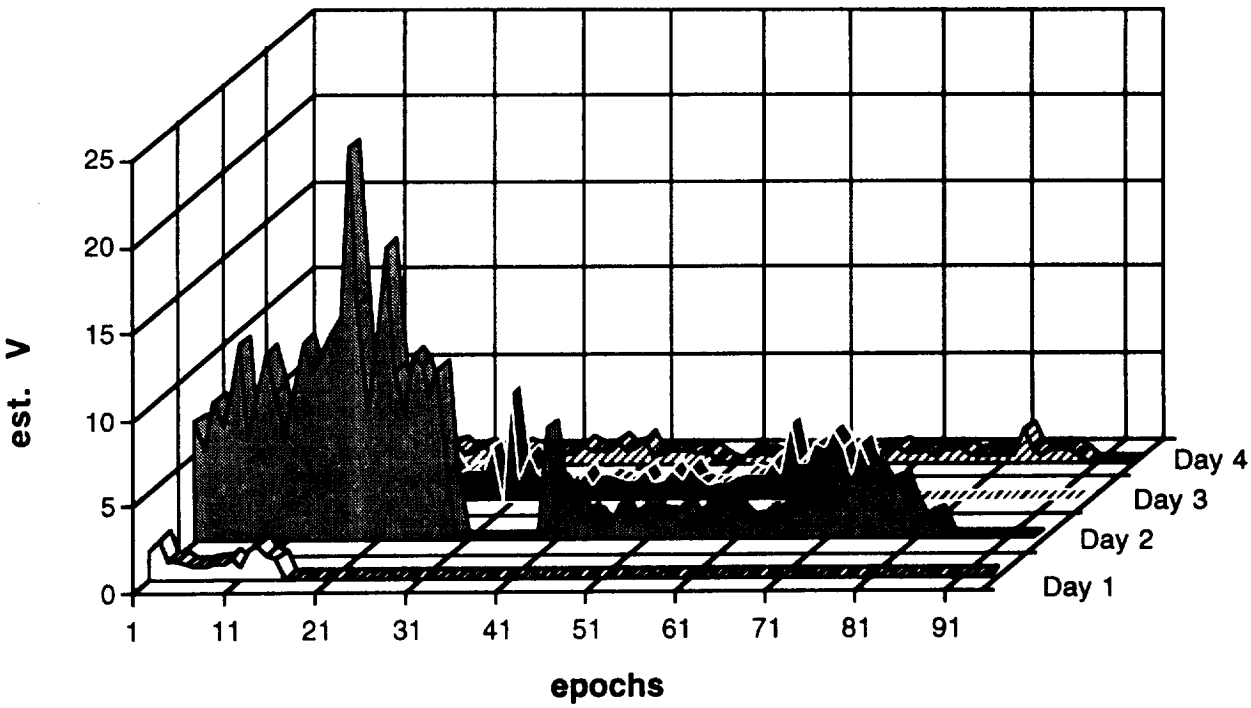

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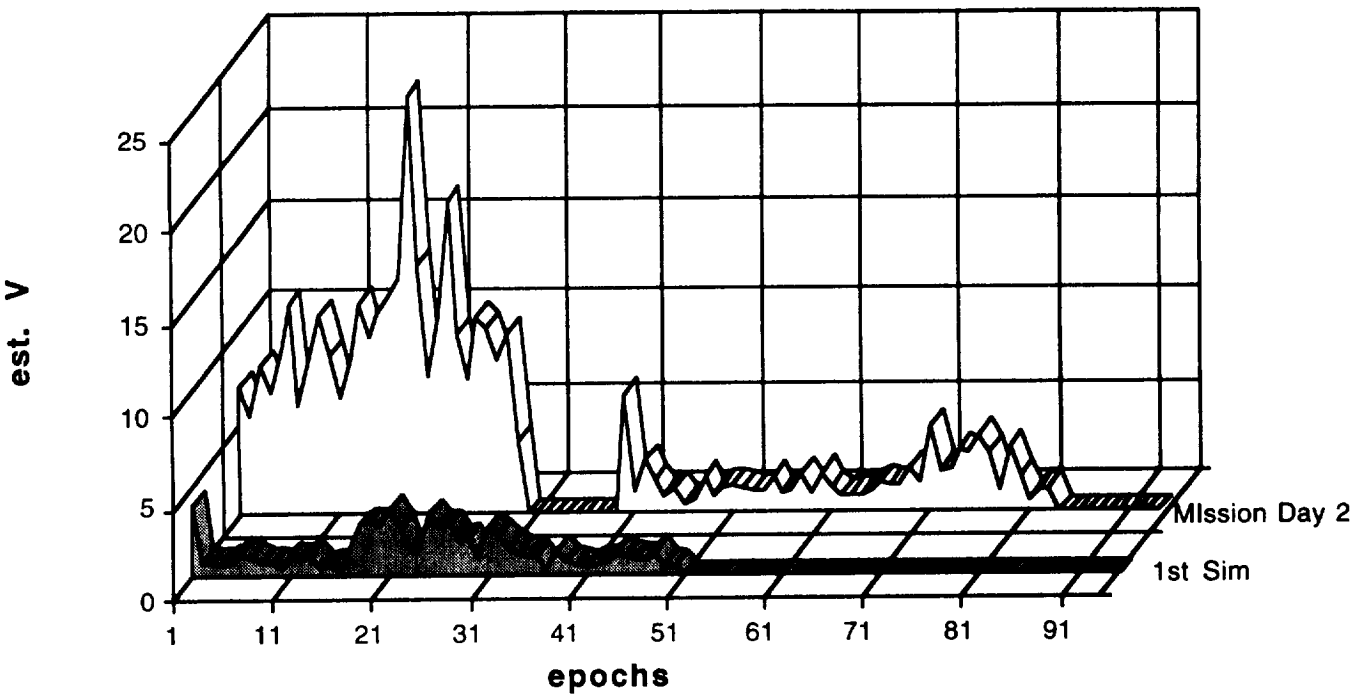

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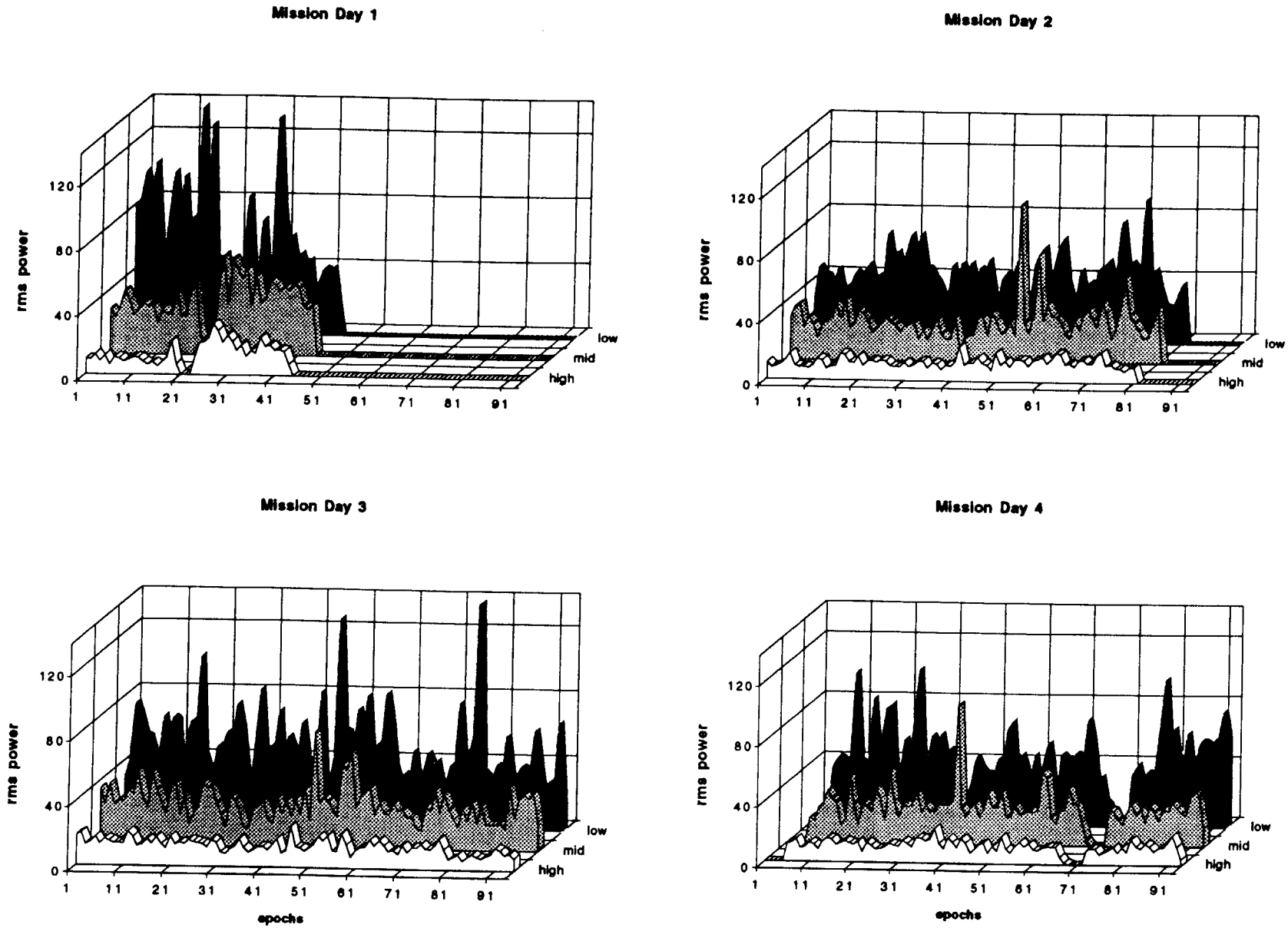


Figure B-30. Heart rate variability during spaceflight—subject 8.

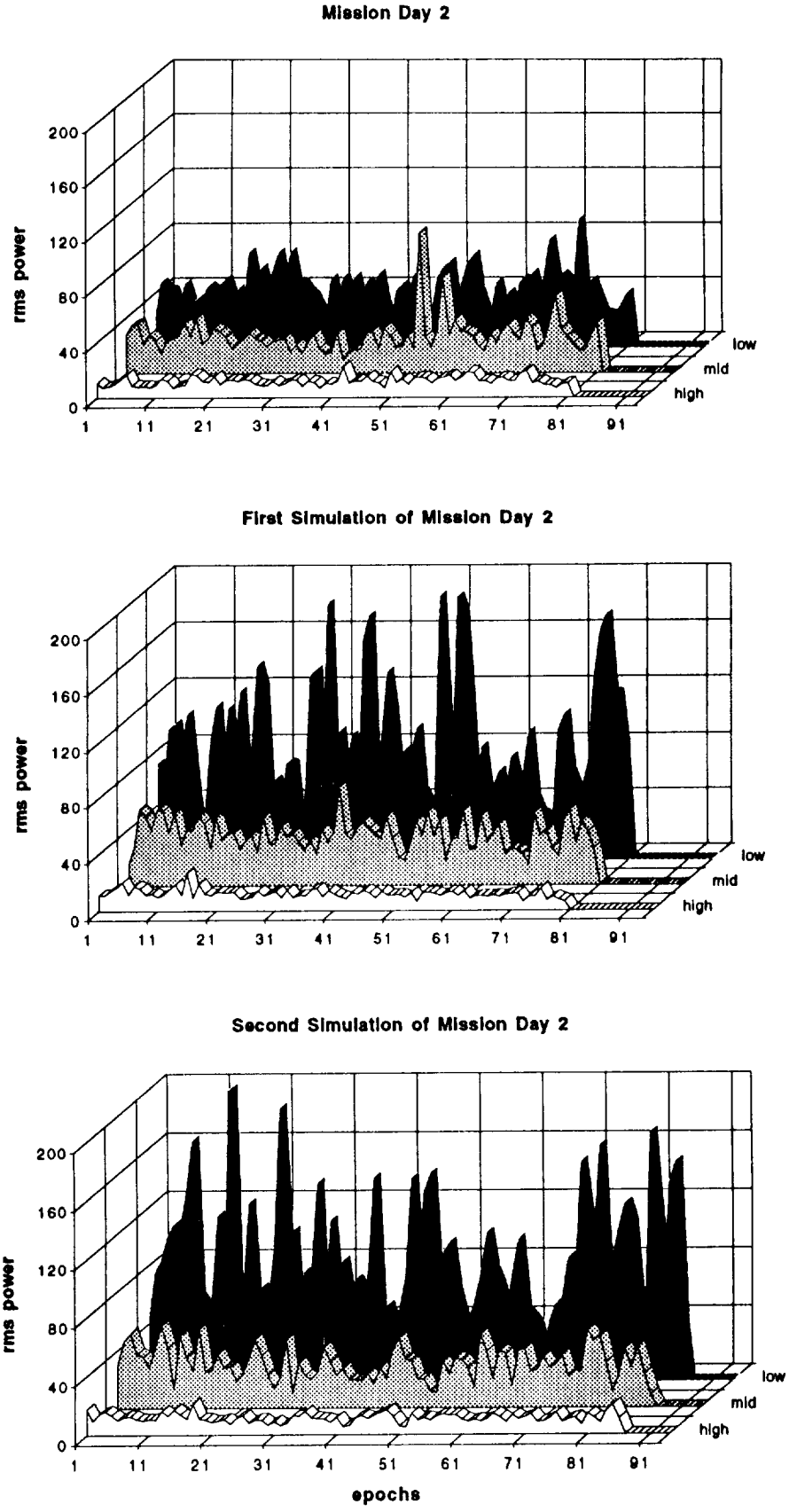
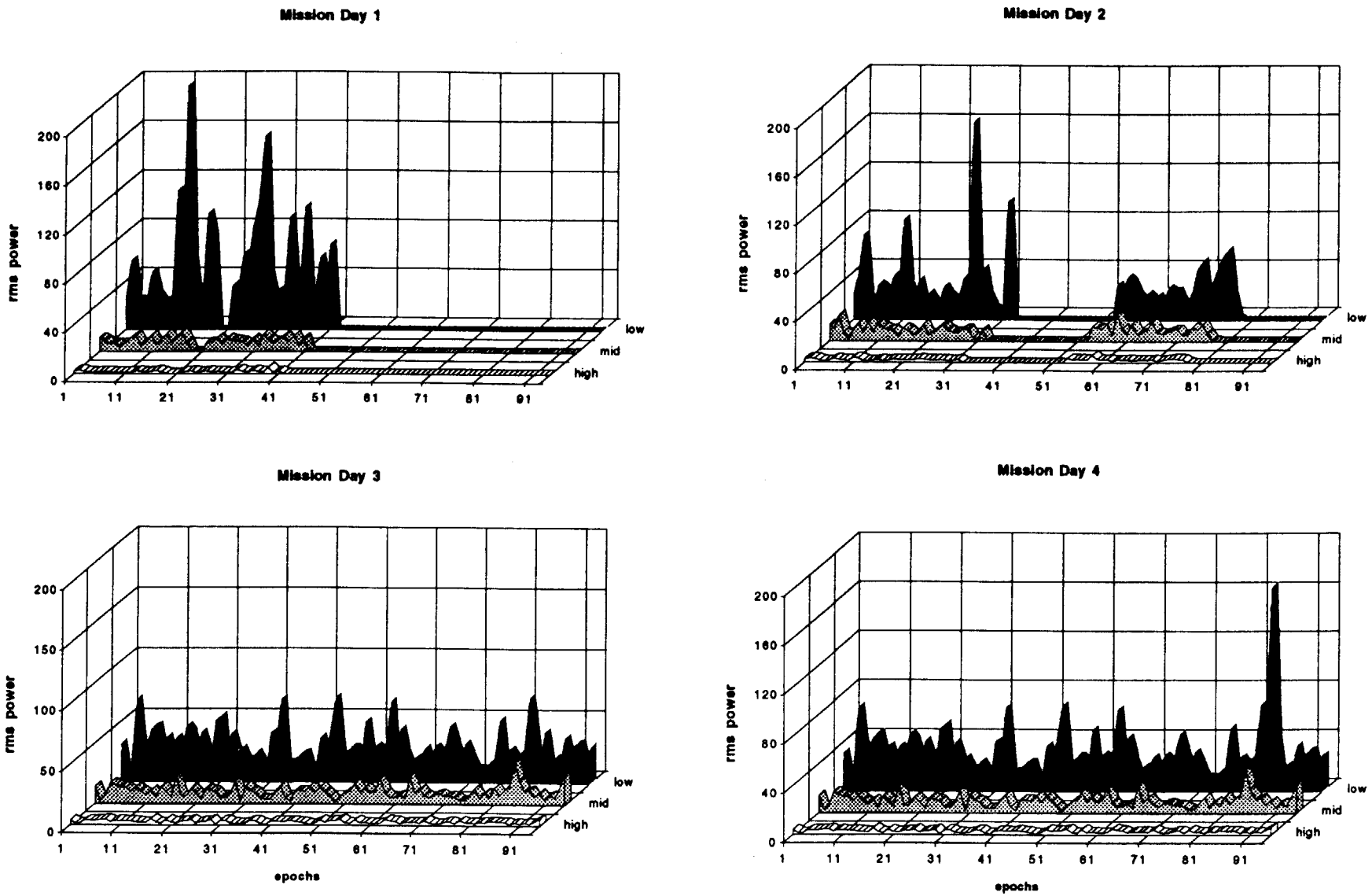


Figure B-31. Heart rate variability in space vs. Earth-based simulations—subject 8.



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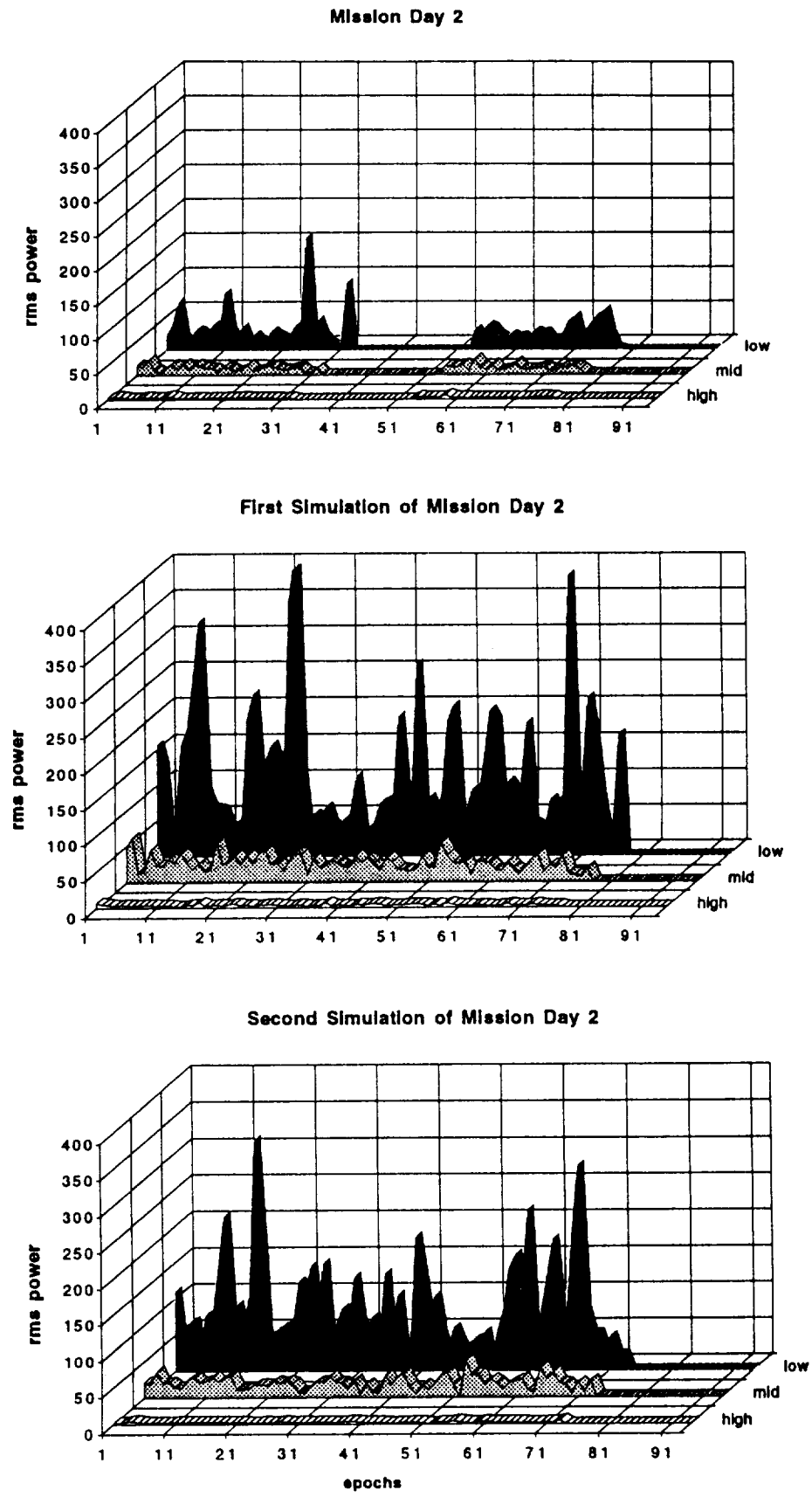


Figure B-33. Heart rate variability in space vs. Earth-based simulations—subject 9.

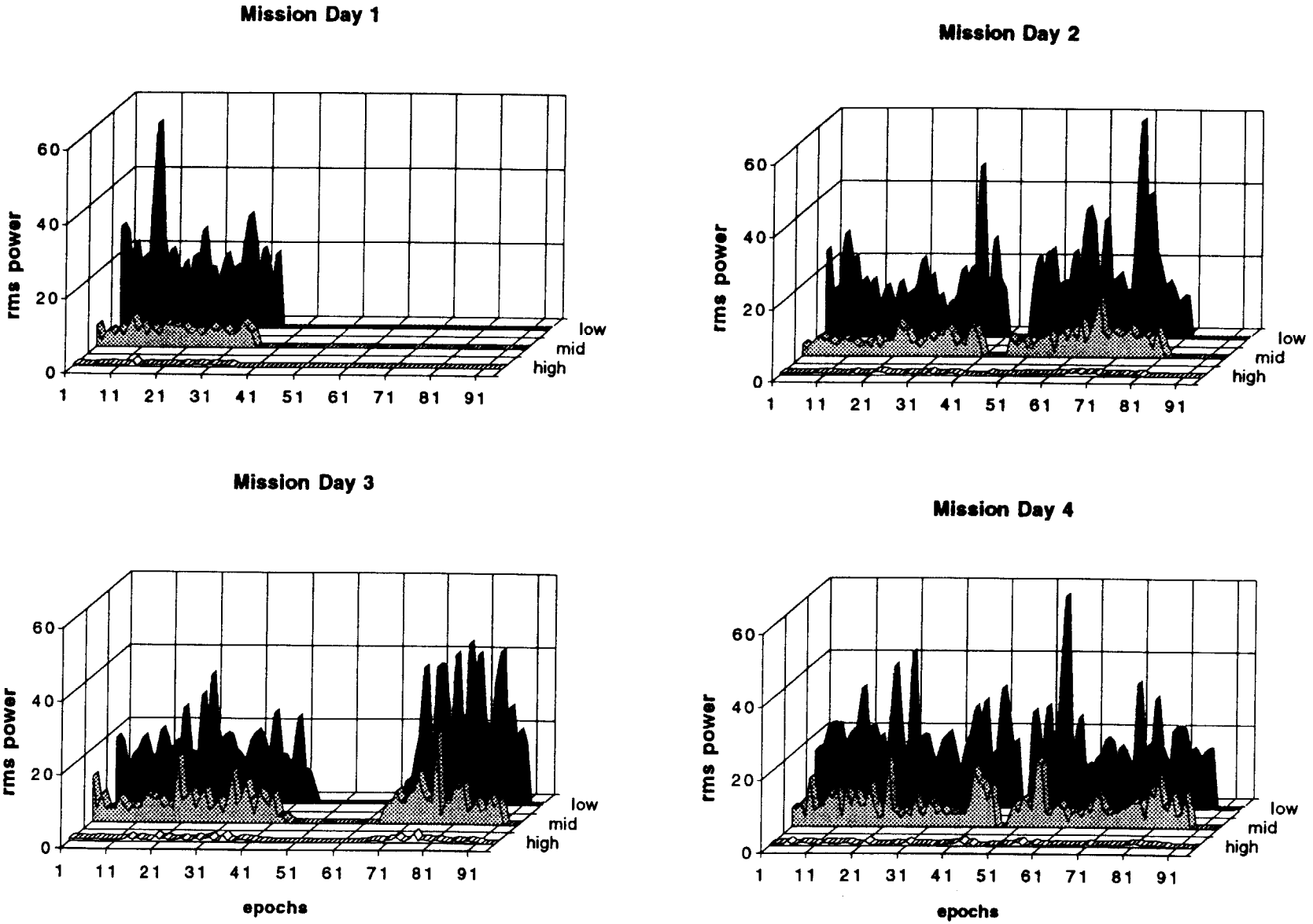
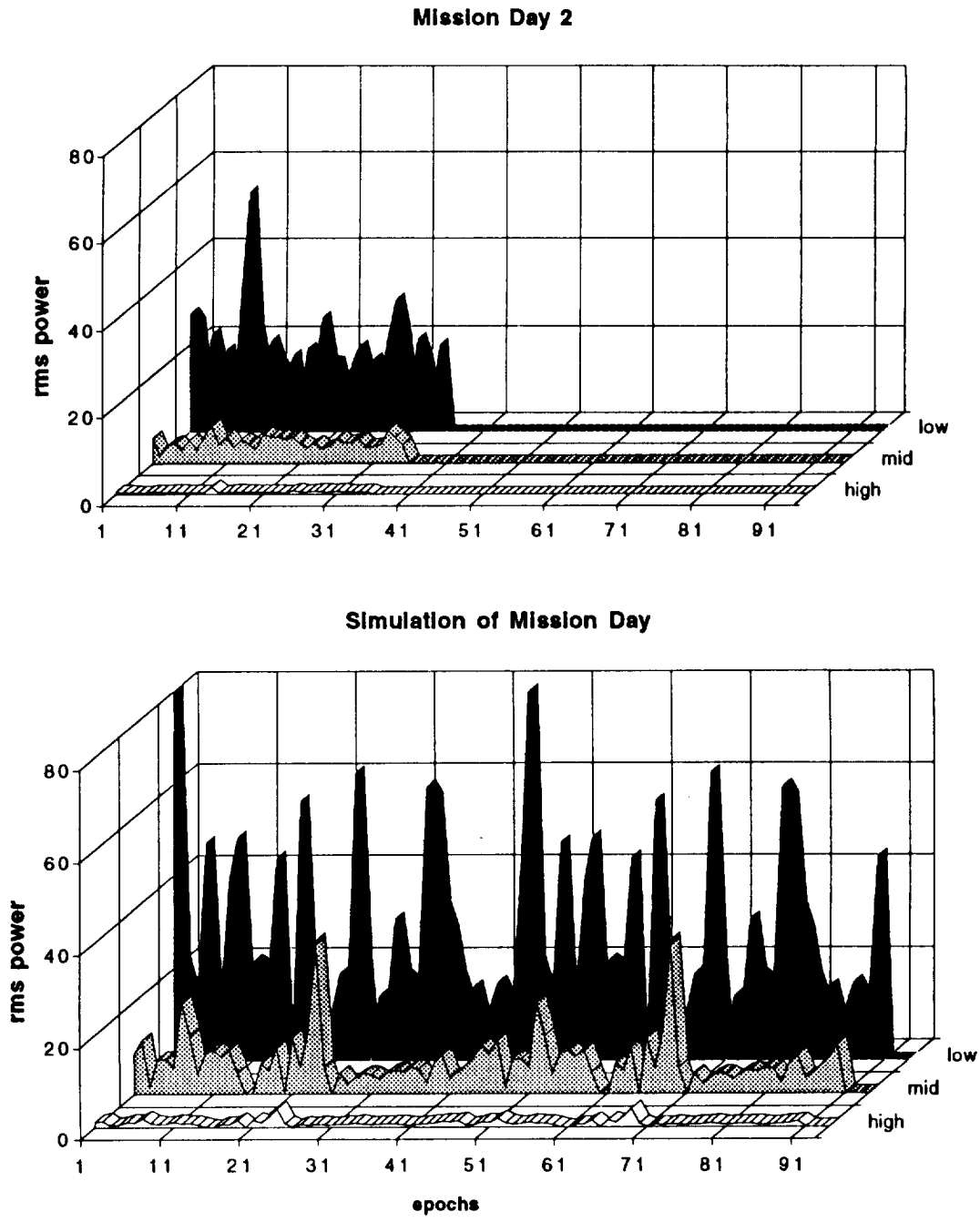


Figure B-34. Heart rate variability during spaceflight—subject 10.



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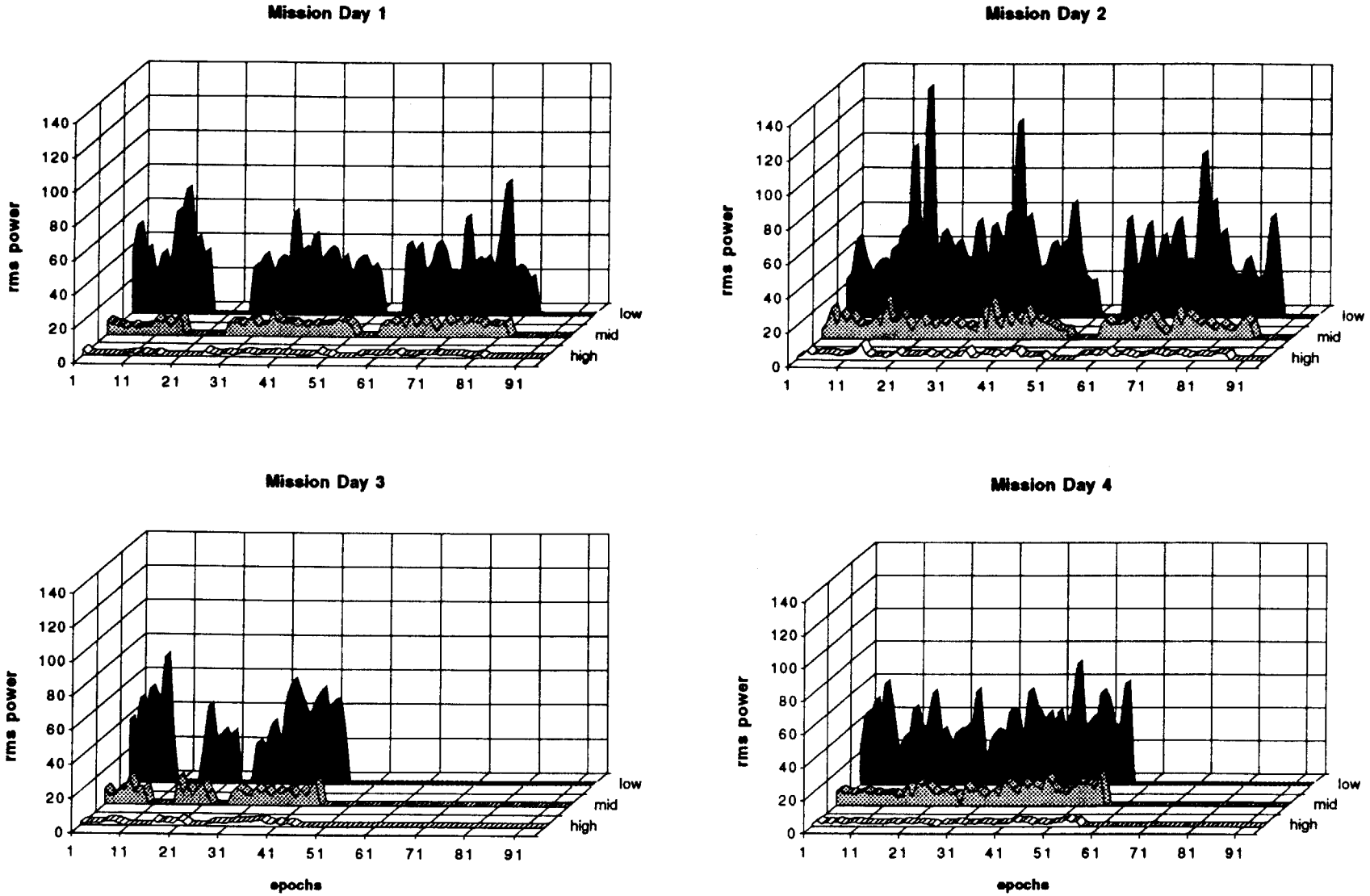

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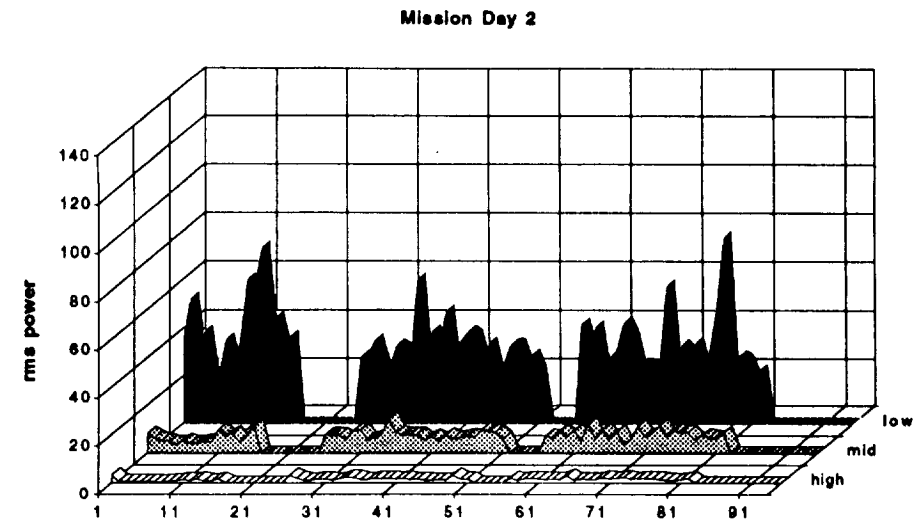

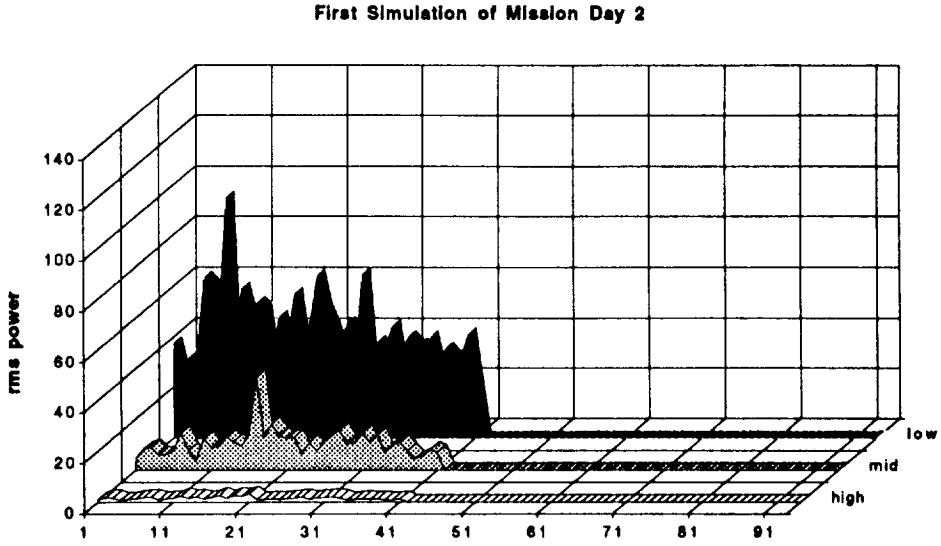

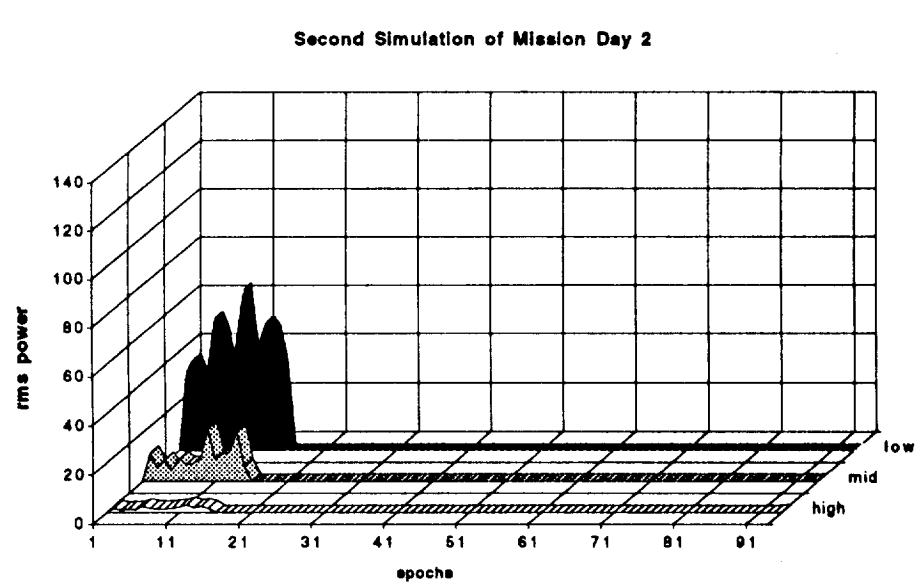

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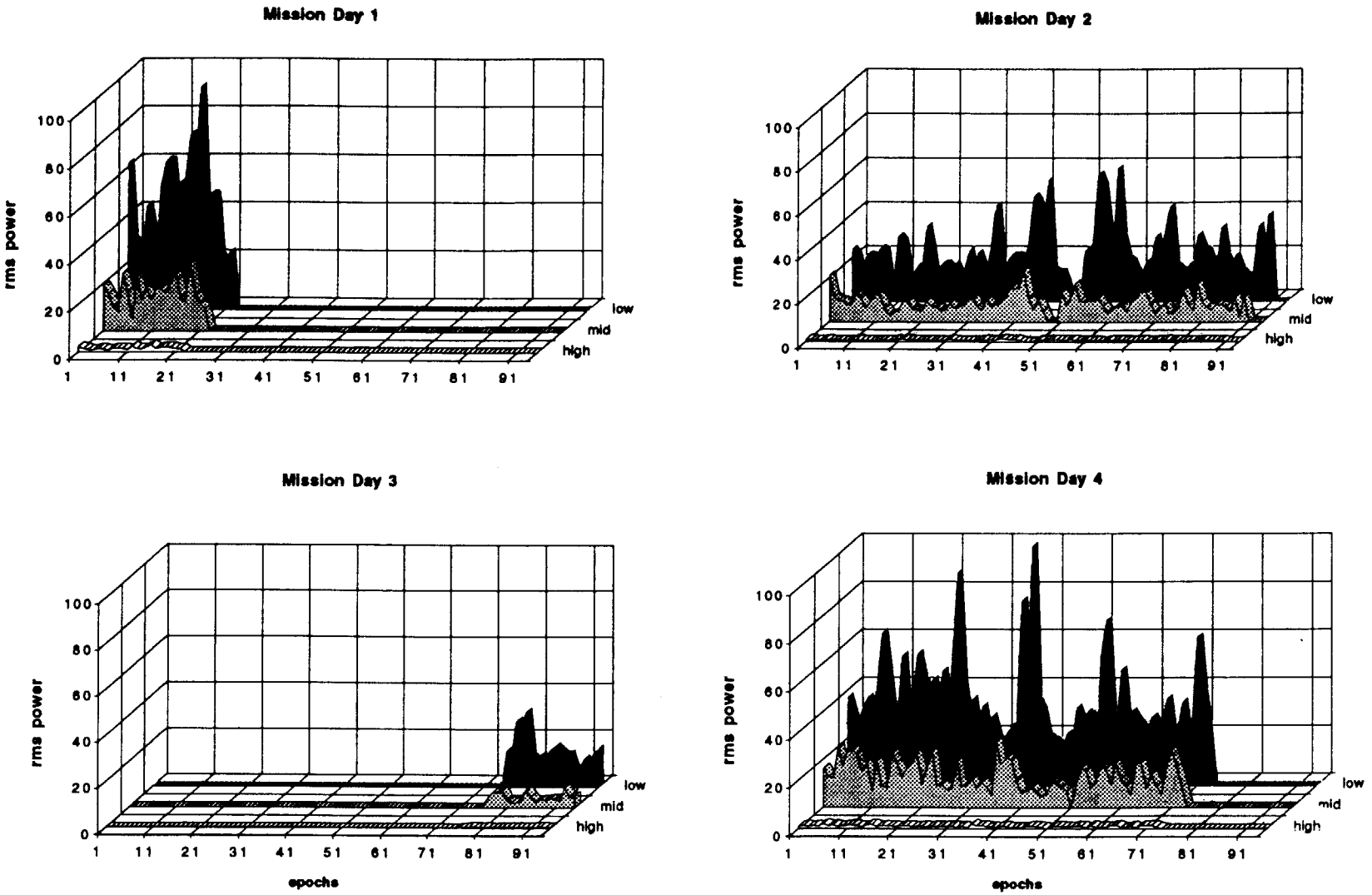
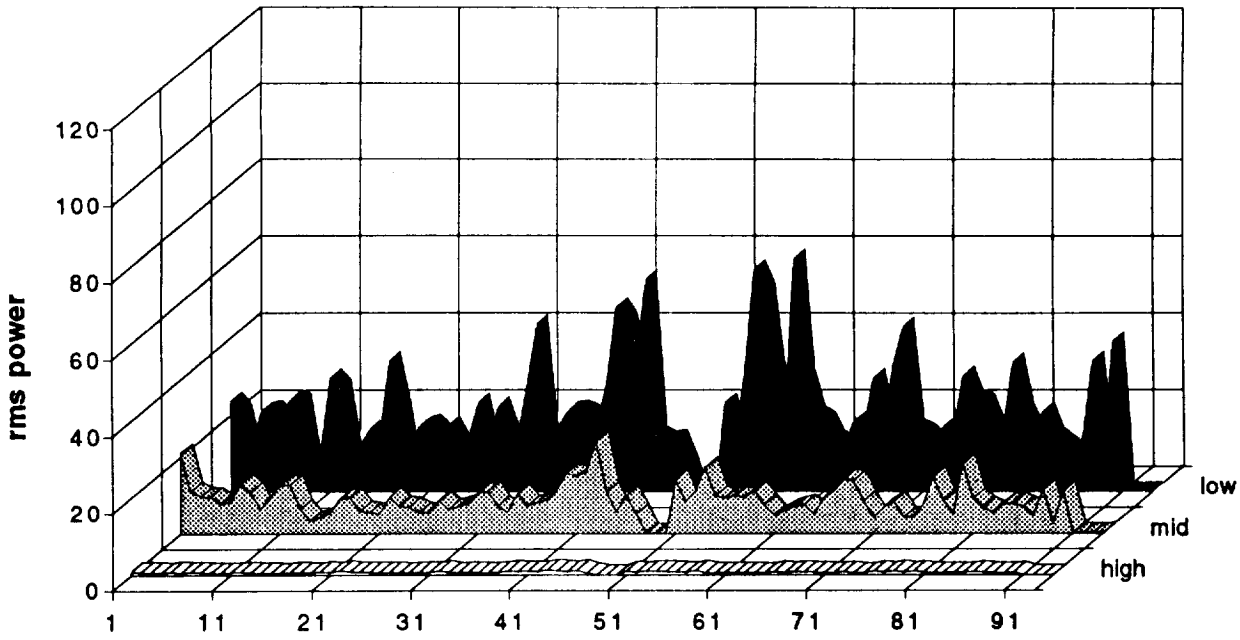


Figure B-38. Heart rate variability during spaceflight—subject 12.



Simulation of Mission Day 2

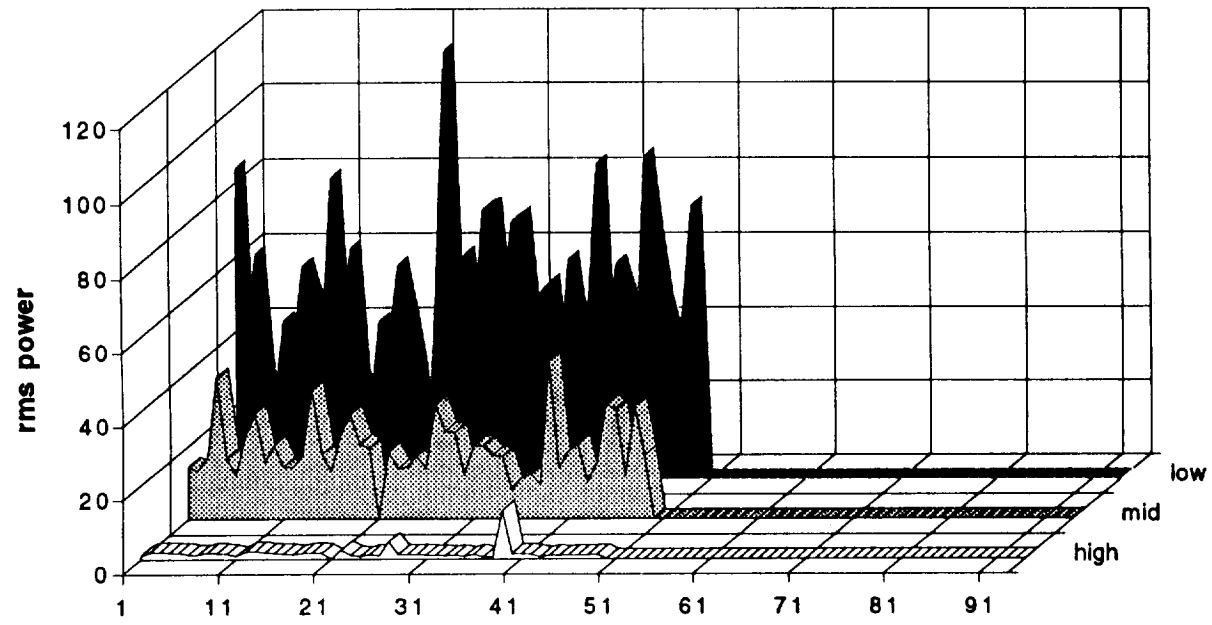


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Figure B-39. Heart rate variability in space vs. Earth-based simulations—subject 12.

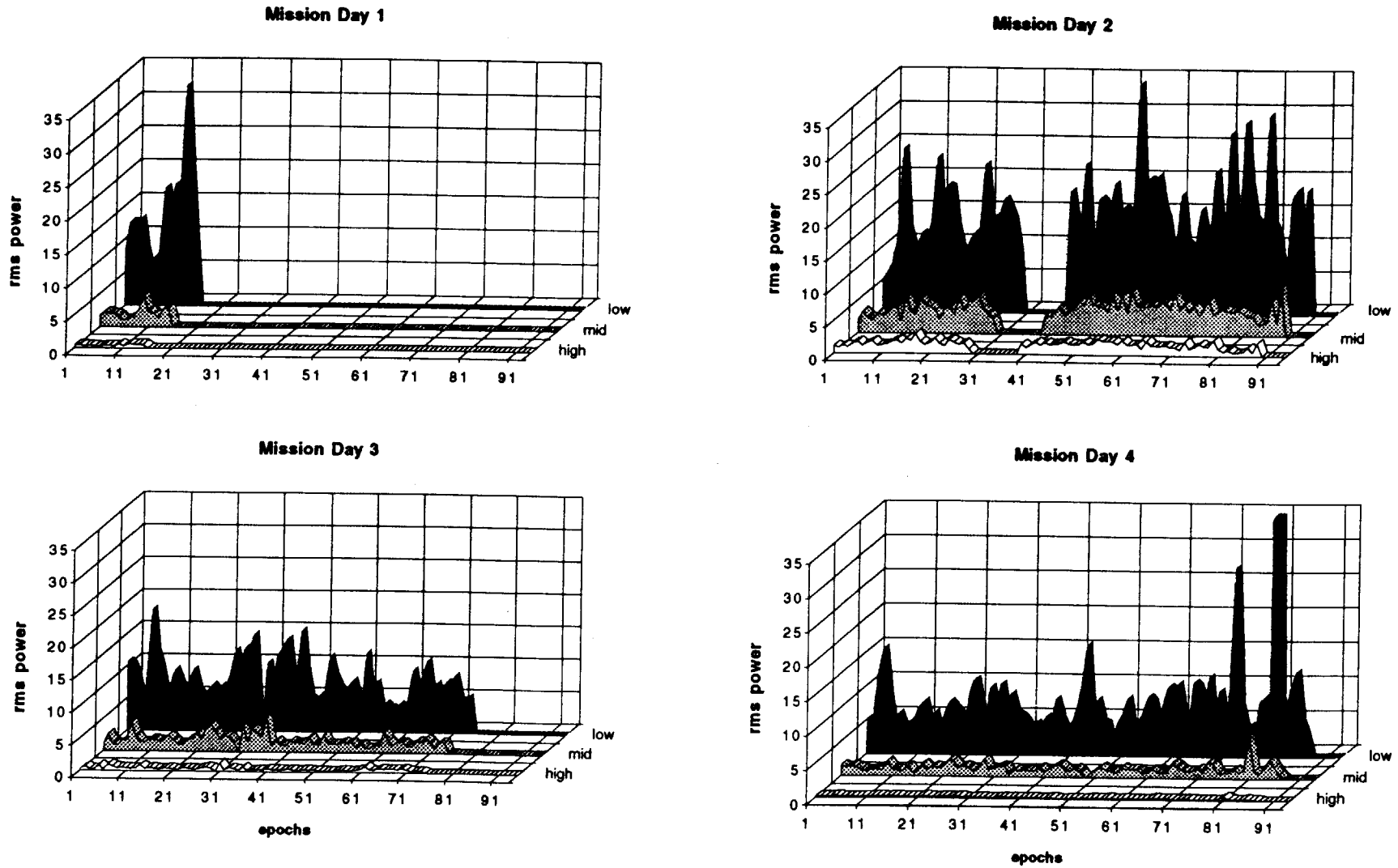

Figure B-40. Heart rate variability during spaceflight—subject 13.

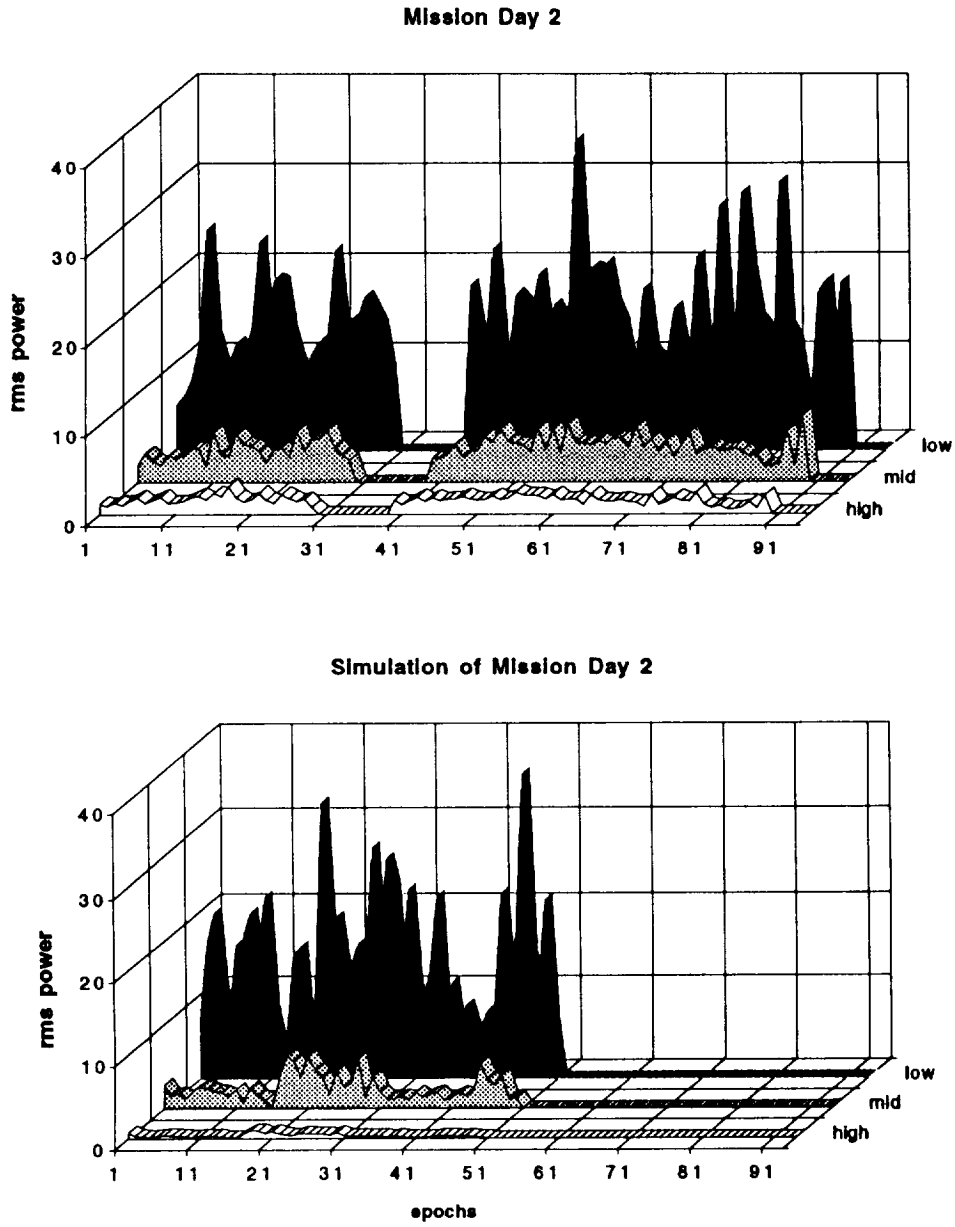

Figure B-41. Heart rate variability in space vs. Earth-based simulations—subject 13.

Appendix C

Subject Consent Form

NASA HUMAN RESEARCH
MINIMAL RISK
INFORMED CONSENT FORM

1. I, the undersigned, do voluntarily give my informed consent for my participation as a test subject to the following test, experiment, or other evaluative procedure.

NAME OF EXPERIMENT: _____

TRAINING TOUR NUMBER: _____

FLIGHT TO WHICH ASSIGNED: _____

NAME OF DESIGNATED PRINCIPAL INVESTIGATOR: _____

NAME OF RESPONSIBLE NASA PROJECT SCIENTIST: _____

I understand that:

(a) This procedure is part of an experiment approved by NASA.

(b) I am performing these duties as part of my employment, with _____.

(c) This procedure has been reviewed and approved by the JSC Human Research Policy and Procedures Committee (HRPPC) and determined that the procedure involves no more than minimal risk to the subject.

(d) "Minimal risk" means that the harm or discomfort anticipated in the proposed research is not greater, considering probability and magnitude, than those encountered in the daily lives of healthy individuals, including the recognized risks inherent in a chosen occupation.

(e) I am medically qualified to participate in the procedure.

(f) I may withdraw from the procedure at any time unless, as recommended by the Principal Investigator, or his/her designee, the withdrawal is dangerous or impossible.

(g) In the event of physical injury resulting from the procedure and calling for immediate action or attention that NASA will provide, or cause to be provided, the necessary treatment. I also understand that NASA will pay for any claims of injury, loss of life or property damage to the extent required by the Federal Employees' Compensation Act or the Federal Tort Claims Act. My agreement to participate shall not be construed as a release of NASA or any third party from any future liability which may arise from, or in connection with, the above procedures.

(i) My identity will remain confidential, and no raw medical data or information will be released to *any* group or individual without my prior written consent. This includes NASA and its representatives (eg., NASA flight surgeons, etc.). I will have an opportunity to review the mission report before its release. Under no circumstances will data release cause compromise of my identity confidentiality without my prior written consent.

2. I, the undersigned, the Principal investigator of the experiment designated above, certify that:

(a) I have accurately described the procedure to the test subject.

(b) The test set-up involves minimal risk to the test subject. All equipment used has been inspected and certified for safe and proper operation.

(c) The test subject is medically qualified to participate.

(d) The test protocol has not been changed from that approved by the JSC Human Research Policy and Procedures Committee (HRPPC).

APPROVED:

Test Subject	Date
Principal Investigator	Date
Project Scientist	Date

This consent form is valid for a 60-day period from the date of signature by the subject and the Principal Investigator (which dates would be identical). A signed, dated copy of the form should be forwarded to the JSC Human Research Policy and Procedures Committee, Mail Code SA, Lyndon B. Johnson Space Center, Houston, Texas 77058.

REPORT DOCUMENTATION PAGE

Form Approved
OMB No. 0704-0188

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6. AUTHOR(S)

William B. Toscano* and Patricia S. Cowings

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Moffett Field, CA 94035-1000

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Point of Contact: Patricia S. Cowings, Ames Research Center, MS 239A-2, Moffett Field, CA 94035-1000; (415) 604-5724. Funded in part by NASA/Univ. Calif., San Francisco Cooperative Agreement (NCC2-115)
*Neuropsychiatric Institute, University of California at Los Angeles, Los Angeles, California.

12a. DISTRIBUTION/AVAILABILITY STATEMENT

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12b. DISTRIBUTION CODE

13. ABSTRACT *(Maximum 200 words)*

Space motion sickness (SMS) affects 50% of all people during early days of spaceflight. This study describes the results of two Shuttle flight experiments in which Autogenic-Feedback Training (AFT), a physiological conditioning method, was tested as a treatment for this disorder. Of the six who were designated as flight subjects (two women and four men), three were given treatment and three served as controls (i.e., no AFT). Treatment subjects were given 6 hours of preflight AFT.

Preflight results showed that AFT produced a significant increase in tolerance to rotating chair motion sickness tests. Further, this increased tolerance was associated with changes in specific physiological responses and reports of reduced malaise. Flight results showed that two of the three control subjects experienced repeated vomiting on the first mission day, while one subject experienced only moderate malaise. Of the three treatment subjects, one experienced mild discomfort, one moderate discomfort, and one severe motion sickness. Only the three control subjects took medication for symptom suppression. Measures of cardiac function reflective of vagal control were shown to be affected especially strongly on the first day of space flight. AFT given for control of heart rate, respiration, and other autonomic activity influenced both the vagal control measures and SMS. These data suggest that AFT may be an effective treatment for space motion sickness; however, this cannot be demonstrated conclusively with the small number of subjects described in this paper.

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Heart rate variability, Microgravity, Motion sickness, Autogenic-Feedback Training	146
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Unclassified	Unclassified		