NASA Technical Memorandum 84400

Physiological Effects of Simultaneous Exposures to Heat and Vibration

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September 1983

National Aeronautics and
Space Administration

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Moffett Field, California 94035

DEDICATED TO

My wife, Patricia

for continued patience, faith, tolerance, and encouragement

ACKNOWLEDGMENTS

The author wisnes to express sincere appreciation to Dr. R. C. Spear, Professor of Environmental Health Sciences, School of Public Health, University of California, Berkeley, and Chairman of his guidance committee, for untiring assistance and guidance throughout this research project. Sincere appreciation is also given to Dr. I. Fatt and Dr. A. Smith for their guidance and assistance in reviewing this manuscript, and for serving on the author's Ph.D. committee.

Special acknowledgment is expressed to Dr. J. E. Greenleaf, Human Environmental Physiology Laboratory, NASA Ames Research Center, Moffett Field, California, for making it possible through numerous opportunities, valuable training, and material and personnel support to develop and advance the author's desires and interest in multistressor physiology. Without his gracious assistance it is unlikely this experiment would have been conducted with the breadth and depth in which it was performed.

Acknowledgments are also extended to Chris Elder for acting as my primary assistant during the start-up and data collection phases. She was also instrumental in the reduction of the blood impedance data. Individual thanks are expressed to Joan Silver, David Datu, Jon Starr, Paula Katz, and Nancy Wong for their able technical assistance. Joan Silver was especially helpful with the blood drawing and most of the blood component analyses. Drs. S. Kravik and R. Pelligra, and Mr. D. Peeler are acknowledged for their assistance and medical monitoring of the subjects during the experiments.

Sincere appreciation is given to Mr. H. Menche and Mr. H. Garrison, of Syscon Corporation, for the use and monitoring of their Ling shaker table. These men were extremely helpful in suggesting ways to better

instrument this experiment. Mr. Menche was very helpful in writing the computer programs used to arrange the data and to plot the graphs.

Acknowledgments are given to Mr. B. Loya and Mr. T. Sereno, of TRW, Inc. of Redondo Beach, for their professional recommendations of outlining the safety systems used in man-rating this type of shaker table, and in earlier attempts to conduct this experiment at the TRW Space Systems' Group facility.

This investigation was financially supported by the Northern California Occupational Health Center, for equipment and some living expenses; NASA Ames Research Center, for laboratory and computer support, use of the shaker table, and construction of the portable heat booth, and recruitment and payment of the subjects; and by the Office of Naval Research contract No. N00014-82-K-0634, for living and travel expenses during the last year of this project.

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BACKGROUND

Occupational heat exposures can be produced either by intrinsically hot processes and equipment, or extrinsically. Intrinsically hot processes or equipment are those which generate the heat locally. Extrinsic heat exposures refer to climatic conditions. For example, workers exposed to intrinsically hot processes and equipment might include bakers, boiler heaters, coke-oven operators, cooks, foundry workers, deep-mine workers, ship propulsion engineers, smelters, forgers, and tire manufacturers. Extrinsic heat exposures are those tasks in which workers are exposed periodically to climatically stressful conditions such as farming, harvesting, heavy equipment operating, and military operations in desert or tropical environments.

The basic thermodynamic processes involved in the exchange of heat between the environment and man have been described by the equation:

$$\Delta S = M - E \pm R \pm C \pm K$$

where ΔS equals the change in the rate of body heat content; M is the rate of metabolic heat production; E is the rate of heat loss through evaporation; R is the rate of heat loss or gain through radiation; C is the rate of heat loss or gain through convection; K is the rate of heat loss or gain through conduction (ref. 1). In most occupational health settings, heat loss by conduction has a minor role.

The mechanisms for responding to imbalances in the above equation due to increased body-heat content involve (1) vasomotor adjustments (primarily vasodilation) which determine the rate of heat exchange between the core and peripheral tissues, and subsequently the heat exchange between the body's surface and the environment, and (2) evaporative heat loss mechanisms such as sweating. Changes in circulation

(vasomotor responses) directly control the heat-exchange rates of convection, radiation, and conduction. Several authors have also reported that changes in skin blood flow indirectly affect sweat rates (refs. 2-4). Estimates of normal skin-blood flow for a nude resting male in a normal indoor environment range from 197 to 490 ml/min, with maximum cutaneous blood flow during heat stress ranging from 2.1 to 3.5 liters/min (ref. 5). The blood vessels that dilate during heat stress conditions penetrate subcutaneous insulator tissues and are distributed profusely along the subpapillary portions of the skin, thus providing the only functional pathway for heat to pass rapidly from the body core to the peripheral areas. Therefore, heat transfer to the skin is controlled primarily by the degree of vasodilation of the arterioles and arteriovenous anastomoses that supply the venous plexus of the skin.

Several factors that determine the rate of heat transfer to the surface of the body may interfere with the degree of vasodilation or vasoconstriction of the vessels that supply the venous plexus of the skin, and therefore can interrupt the body's homeostatic thermoregulatory mechanisms. These factors may produce either localized or whole-body responses, and may occur by local control or may be transmitted through the body via neural reflexes.

The idea of localized interference is important since the body does not cool itself uniformly with vasodilation nor with sweating. Several studies have indicated different rates of heat loss for localized regions of the body: head (refs. 6, 7); hands and arms (refs. 8, 9); neck (ref. 10); chest (refs. 10, 11); and feet (ref. 12).

According to National Institute for Occupational Safety and Health (NIOSH), an estimated 8 million workers are exposed to occupational

vibration. Of this 8 million, 6.8 million are estimated to be exposed to "whole-body" vibration, and about 1.2 million are exposed to "segmental" vibration. Frequently, vibration exposures occur simultaneously with elevated heat exposures. The source of the elevated heat exposures can be either intrinsic to the vibrating equipment (e.g., engines, boilers), or extrinsic to the vibrating equipment (e.g., earth-moving equipment, automated farm equipment, or military armored personnel carriers, when used in warm climates). Thus, almost all vibrating equipment could potentially be associated with heat-stress exposures at some time.

Much of the research done on the physiological effects of vibration has been done by the Russians, which frequently presents some ambiguities due to the complexities of the translated versions of Soviet medical parlance. The Russians frequently refer to a "vibration syndrome," which, by Western standards of defining a species of disability, is somewhat obscure. According to the Russians, the physiological effects of vibration are focused in the peripheral vascular, nervous, muscular, osseous-articular, and cardiopulmonary systems, and produce within these systems complicated alterations manifested for the most part in polymorphous symptoms. The symptoms (1) may occur in a single system, (2) may be presented from the most discrete, hardly noticeable ones up to massive symptoms, and (3) may range from functional alterations to organic changes (ref. 13). Persons exposed to local vibration generally complain of disturbances of a vegetative nature, which are predominantly manifested by spastic states in the distal parts of the vascular system. In the initial stages of vibration disease syndrome, contraction of peripheral vessels is the first sign. The actual mechanisms for the

peripheral vessels' contractions are not well understood; however, the contractions appear to be due to a very complicated syndrome of disturbances of the neurodynamic reflexes responsible for vasomotoricity. Also, the spasms produced by vibration have been shown to become more pronounced when the intensity of the vibration was increased.

The effects of vibration on human performance have received considerable attention and will not be further mentioned in this report except where performance may interfere with the cooling of the body. Specific areas of other investigations on the effects of vibration on performance have included tracking efficiency (refs. 14, 15), motor performance and body configuration (refs. 16, 17), and visual activity and reaction time (refs. 15, 18, 19).

There have been no previous investigations aimed at determining the effect of vibration on the thermal homeostatic mechanisms of humans; however, there have been a few investigations involving human psychological responses to heat and vibration (refs. 14, 20). The conclusion in these reports was the combination of heat, noise, and vibration produced antagonistic rather than additive effects on performance. In addition Megel et al. (refs. 21, 22) performed two investigations on the effects of sublethal intensities of heat, vibration, and the combination of heat and vibration on adult male Sprague-Dawley strain rats. They reported that the sublethal intensities of vibration and heat act synergistically to produce an incidence of mortality greater than would be statistically expected, based on the incidence of mortality resulting from exposure to each stress alone. Changes induced in the kidneys and adrenals also suggested the synergistic action of heat and vibration (ref. 21). Exposure to heat and sinusoidal vibration caused an

elevation in rectal temperature of rats greater than that produced by heat or vibration alone (ref. 22).

Since the primary responses to hot environments and vibration are vasodilation and vasoconstriction, respectively, it is interesting to speculate on the possibility that the body's cooling mechanisms may be impaired in a simultaneous occupational exposure with results being similar to those reported in the above animal studies. Such an impairment would seriously limit the thermal exposure to which workers may be subjected who are also exposed to vibration. The objective of this study is to investigate this possibility and, if confirmed, to obtain information which may be very useful in reducing the rapidity of onset and the severity of heat-related casualties by suggesting a more applicable work-rest regime than is currently recommended and by providing detailed information for better engineering controls.

The possibility of a synergistic effect of heat and vibration has important implications. The currently recommended heat-stress exposure limitations are based on a hot environmental exposure without any potentiating factors and assume that the workers have normally operating physiological compensatory cooling mechanisms. If vibration induces vasoconstriction, either locally or generalized, in a hot environment, then the standards defining the exposure limitations should be more conservative for this situation. It is intended that the nature of the information generated from this study be utilized in either of these casualty-abating endeavors. With a reduction in the rapidity of onset and severity of heat-related casualties, individual worker health, industrial productivity, and potential future military response may be

enhanced. This study is oriented toward these more pragmatic, occupational health issues than it is toward more basic physiological research.

SPECIFIC AIMS

The specific aims of this project are to determine if exposure to vibration has an effect on the body's ability to handle heat stress and, if so, to determine the specific levels of the vibration parameters (frequency and intensity) for both whole-body and segmental vibrations which have the most detrimental effect on thermal regulation.

EQUIPMENT AND METHODS SELECTION RATIONALE

This experiment involves the observation of human physiological responses to heat as a function of different conditions of vibration. The physiological responses to be measured are cutaneous circulation and various indices of heat stress levels. There are several methods available to measure cutaneous circulation. If the direct, invasive (traumatic) methods are omitted, then the remaining indirect techniques are based on the measurement of some physical property of the skin that can be related to vascular content or blood flow. The cutaneous circulation can be measured indirectly by skin temperatures and body-segment blood flow rates. When there is a change in degree of peripheral vasoconstriction, there is a change in cutaneous circulation and a time-lagged change in skin temperature. Regional skin-temperature changes can be measured by thermistors, thermocouples, or by comparing infrared scans. The degree of vasoconstriction and rate of blood flow can be measured by the transcutaneous Doppler flow method (ref. 23), or by various plethysmography methods (refs. 24, 25). The body heat-stress levels can be

indirectly measured by core temperature and heart rate. If the heat loading of the body exceeds the cooling mechanisms then there is an increase in the body heat stress level and in the deep body temperature. The latter is usually measured in one of the body's orifices — rectal, esophageal, or tympanic.

Localized sweat rates are measured in this study since sweating is the primary route of heat loss in warm environments. A reduction in sweating would result in heat storage and a subsequent rise in rectal temperature. The rate of sweating appears to be linearly related to internal temperature (specifically hypothalamic temperature), with the skin temperature affecting the central temperature threshold at which sweating is initiated. Sweat secretion is not a continuous process but is characterized by a cyclic discharge of sweat on the body surface. Also, the sweating activity of humans is synchronous over the entire body surface. Three variables used for comparing rates of sweat involve changes in the overall intensity of the sweat rates at each site, and changes in the amplitude and periodicity of the sweat cycle. The resistance hygrometry method (refs. 26-28) for measuring sweat rates is continuous, rapidly sensitive to small changes, and is used in this investigation.

Because of the scale and complexity of these experiments, various parameters were measured which were not of direct relevance to the objectives of the project but which may be of interest to other investigators. For example, blood samples were taken at various times and analyzed for differential blood cell counts, hematocrit, hemoglobin, potassium, sodium, and osmotic concentrations.

The independent stress-related variables in this study include vibrational frequency and intensity, environmental temperature, and environmental humidity. Vibration exposures can be variously described depending on the particular wave form. In this study only sinusoidal vibration is employed. In simple harmonic or sinusoidal motion at a specific frequency, the acceleration is sufficient to also determine velocity and displacement amplitudes.

It should be noted that the vibration parameters are measured at the base of the chair. The common practice in health-related vibration research has been to measure frequency and acceleration. (Displacement measurements are significant in the study of deformation and bending of structures, and are easily measured when the frequencies are low.) Following this practice, acceleration and frequency are monitored in this study. An additional reason for choosing acceleration is that it is directly related to those forces which cause stretch in the body receptors and thus initiate the body reflex reactions.

Vibration Exposure Specification

A wide range of frequencies was selected in order to "range-find" over the part of the spectrum where the detrimental effects may occur. The specific frequencies were selected from documented vibration spectra of various types of equipment. The intensity levels, measured by acceleration (g-rms), were determined by selecting the "exposure limits" and "fatigue-decreased proficiency limits" for each frequency from the International Standards Organization (ISO) recommended standards for whole-body vibration exposure (ref. 29).

Below are examples of frequency and acceleration ranges for various types of equipment.

Equipment	Frequency range, Hz	Acceleration range
Crawler loader	0.12 - 20	0.01 - 0.25 g (peak)
Scrapers, graders	0.10 - 5.25	0.04 - 0.13 g (peak)
Wheel-type compactors	0.13 - 14.95	0.02 - 0.06 g (peak)
Dump truck	0.12 - 12.12	0.01 - 0.05 g (peak)
Tractor	0.5 - 11	--
Pneumatic hammers	10 - 1200; max 83	--
Chain saws	31.5 - 1000; max 125	80 ms^{-2} (rms)

From studies of whole-body vibration, the tolerance for a seated man is lowest in the frequencies between 3 and 14 Hz. The reason for the lack of tolerance in this range is that the torso resonates at approximately 3-6 Hz and 10-14 Hz. In addition to this whole-body resonance, different segments of the body have different resonant frequencies. For example, the head-shoulder area resonates at 20-30 Hz, the eyeball at 60-90 Hz, and the lower jaw and skull area at 100-200 Hz (refs. 30-32).

Thermal Exposures

There exist many methods which attempt to describe the total thermal environment by a single number and then to use this number to predict the level of human heat stress. Actually, there probably is not a single index which can adequately account for every environmental and physiological characteristic in all situations. The rationale in this study was to select an index based on physiological body stress levels, since the focus of this study is the correlation of circulatory system responses to vibration and heat-stress exposures. The WBGT (wet bulb globe temperature) index is such an index and it was originally developed to provide a quick and convenient method to assess the heat conditions

which would pose threats to thermal casualties among military personnel. Due primarily to its simplicity, this method has also been adopted as the principal index for a tentative threshold limit value (TLV) for heat stress by the American Conference of Governmental Industrial Hygienists (ref. 33). The WBGT index is computed by appropriate weighting of a black globe temperature (t_g), dry-bulb temperature (t_a), and natural wet-bulb temperature (t_{nwb}). The natural wet bulb is depressed below air temperature by evaporation resulting from the natural motion of the ambient air, in contrast to the thermodynamic wet bulb, which is cooled by an artificially produced fast air stream and eliminates the air movement as a variable. The weighting formula for indoor use (without a solar radiation load) is (ref. 33)

$$WBGT = 0.7(t_{nwb}) \pm 0.3(t_g)$$

Although the range of application for the WBGT index has been expanded beyond the original intent, it is one of the most common indices, and most people working with hot environments are familiar with it. The WBGT index was used in this study because of the ease of evaluation and its wide familiarity among other researchers and industrial personnel. However, additional information on the test conditions is presented in order to allow other researchers to calculate other heat-stress indices. In this study, the specific numbers for this index or any other heat measuring index are used principally to demonstrate that the test environment is definitely heat stressful. In summary, the dose-related variables for heat in this study include environmental temperatures and humidity, which are held constant over all vibration exposures and during recovery periods.

PROCEDURES

General Overview

This experiment was conducted at NASA Ames Research Center, Moffett Field, California. Six healthy males, 22-33 years, volunteered as subjects. Each subject passed a comprehensive medical examination and signed informed consent documents (see appendix I). The six subjects were divided into three pairs, with each pair tested over a 5-week period. Within each pair, one subject was tested in the morning and the other in the afternoon.

Each subject spent 3 hr per day at rest in the heat (t_a = 43.5°C, RH = 25%) Monday through Friday for the first 2 weeks to insure similar moderate levels of heat acclimation (Phase I). Three hours per day of heat exposure at rest is the minimum required to insure heat acclimation over a 2-week period (ref. 34). During the 2-week acclimation period, three 20-ml antecubital venous blood samples were taken (Monday, Friday, Friday). After the 2-week acclimation period, the subject entered into the second part of the experiment — one of three 1-week combined heat and vibration exposure phases. The three combined heat and vibration exposure phases were termed heat and high intensity whole-body vibration (Phase II, sec. A); heat and low intensity whole-body vibration (Phase II, sec. B); and heat and segmental-body vibration (Phase III). These three exposure phases were rotated to minimize the effects that may occur with vibration conditioning and possible continued heat acclimation. The general format for each combined heat and vibration phase was as follows: The subject arrived at the laboratory and inserted the rectal temperature probe; he was then transported to the shaker table area where monitoring electrodes were attached, and arm dimensions and body weight were measured. He then

sat in the heat booth and was strapped onto the metal vibration seat (for the whole-body vibration exposures) or stood (for the segmental-body vibration exposures), while additional sensors were applied. After approximately 20 min of monitoring the subject at room temperature (t_a 23.5°C, 41% RH, WBGT = 18°C), booth heating was initiated and it remained heated until the termination of the experiment. After 1 hr of booth heating, vibration was started and lasted 25, 60, or 150 min, depending on the vibration phase of the experiment. After the vibration exposure, the subject spent an additional 50 min in the heated booth so that postvibration recovery data could be collected. After recovery, the subject was disconnected from the monitors, removed from the booth, and arm dimensions and body weight were measured again. (See fig. 1 for a diagram of the study design for a pair of subjects.) The subject was then transported back to the main laboratory for a shower, general observation, and release.

Throughout the entire study the subjects wore only gym shorts and tennis shoes with no socks, and were exposed to only one frequency and one intensity per day. During the heat and vibration phases, the subject was monitored for mean skin temperature, rectal temperature, localized sweat rate, weight loss, oxygen uptake, arm blood flow, and heart rate. Blood samples were taken on three different days during the week (Monday, Wednesday, Friday), just prior to and at the end of the vibration exposure. All laboratory analyses were performed in the Laboratory for Human Environmental Physiology at NASA Ames Research Center. The blood samples were analyzed for differential blood cell counts, hemoglobin, hematocrit, potassium, sodium, and osmotic concentrations. Hematocrit, sodium ion, and osmotic concentrations were determined to check on dehydration level, or fluid shifting from the blood to the interstitial

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Figure 1. Diagram of the study design for a pair of subjects

spaces. Potassium ion concentrations and urine were determined to check on lysis of red blood cells due to the vibration.

The total time involved in this study by each subject was 100 hr over a 5-days-per-week, 5-week schedule. Each subject had 22 venipunctures; total blood volume withdrawn was 440 ml.

Environmental Test Conditions, Equipment, and Schedules

The 2-week heat acclimation phase (Phase I) was performed within the Laboratory of Human Environmental Physiology's Environmental Chamber. The chamber temperature was maintained at 43.5 ± 0.5°C (mean ± S.E.M.) and a relative humidity of 25 ± 2% (WBGT = 31°C). Each subject spent 30 hr in the booth (3 hr/day for 10 days). During this time the subjects read, played cards, or watched television. Water was provided ad libitum and the subject was asked to record the number of cups of water consumed. Subjects who underwent heat acclimation in the morning were also exposed to combined heat and vibration in the morning, while those subjects who were heat acclimated in the afternoon were vibrated in the afternoon.

After the 2-week heat acclimation period, each subject was exposed to heat and vibration over the last 3 weeks of the experiment in a heat booth which was constructed over the shaker table. During the 2-week heat acclimation period, the subjects were not monitored since the only set of biomedical monitoring equipment was at the shaker table area. The air temperature in this booth was maintained at 43.5 ± 1.0°C. Relative humidity was not controlled but averaged 20 ± 4% (WBGT = 31°C). The temperature and relative humidity were measured with a Thunder Scientific Corporation Digital Humidity and Temperature Measurement System (model HS-1CHDT-2A). A black globe temperature was determined

from a thermometer inserted to the center of a 15-cm-diam matte-black painted copper sphere.

These heat conditions are about 7°F wet bulb globe temperature index (WBGT) above the recommended 90°F WBGT threshold limit value (TLV) of the American Conference of Governmental Industrial Hygienists (ACGIH) for the work place (ref. 33). The ACGIH exposure limits assume a set of working conditions which do not apply to this study — a fully clothed person working a minimum of 15 min/hr at a light workload (200 kcal/hr) for an 8-hr work day. Also, since these levels have been developed for the general worker population, a safety factor has been incorporated to allow for a wide range of ages and levels of general health. The ACGIH do make provision in their recommended guidelines for higher temperature exposures if medical surveillance is available or if the particular work group is more tolerant to heat than the average worker. Our subjects are younger and in better health than the general worker population, and were lightly clothed in gym shorts and tennis shoes (thus allowing for greater sweat evaporation). Also, our subjects were at rest for 100% of the heat test period, which was never more than 4.5 hr/day throughout the entire study. In addition, the subjects were monitored for core temperature and heart rate throughout the time they were in the test chambers. These thermal conditions were the same throughout all three vibration exposures.

The vibration source was a Ling Electronics General Shaker (series 300B) and was constructed of an electric oscillator, and amplifier, and motor, which is the power drive. The amplifier drives the shaker table platform. The primary purpose of the shaker table has been to vibrate electrical components for quality assurance; however, primates

and other animals have been vibrated occasionally. The table was not originally "man rated," so safety engineering checks and procedures had to be initiated to make the machine safe for human use. The safety equipment and operating procedures which were implemented with this shaker are discussed below in a separate section on safety procedures.

During the second phase of the study, in addition to the heat conditions of Phase I, the subject experienced whole-body sinusoidal vibration in the vertical Z-axis (seat to head). The majority of these test frequencies were less than 30 Hz, and included frequencies where the whole body resonates, as shown below. To insure good contact between the subject and the seat, the subject was strapped securely to the vibrating chair with an automobile-styled lap seat belt. This second phase was divided into two sections of 5 days each in which an identical spectrum of frequencies was tested at two different accelerations and for two different periods of time (fig. 1). Exposure levels in Phase II, Section A were those termed as "exposure limitations" by the International Standards Organization (ISO) document 2631-1974, ISO, Geneva, 1974 (ref. 29). The exposure limitations presented in the ISO standards for vibration were developed from observations in industry, as well as some laboratory studies, and have a considerable safety margin. As in the TLVs on heat stress, these standards were developed for the general working population. In addition to the safety margins built into these ISO standards for vibration exposure, our subjects were much younger and were in better physical health than the general population. Exposure conditions for both sections of Phase II are listed below.

Phase II, Section A

Heat: t_a = 43.5 ± 0.5°C; RH = 20 ± 4%; WBGT = 31.0°C;

duration = 145 min/day

Vibration:

Day	1	2	3	4	5
Frequency, Hz	5	10	16	30	80
Acceleration, g-rms	0.37	0.46	0.72	1.40	3.70

Duration: 25 min/day

Activity level: seated, at rest

Phase II, Section B

Heat: t_a = 43.5 ± 0.5°C; RH = 20 ± 4%; WBGT = 31.0°C;

duration = 3.5 hr/day

Vibration:

Day	1	2	3	4	5
Frequency, Hz	5	10	16	30	80
Acceleration, g-rms	0.14	0.18	0.28	0.55	1.44

Duration: 2.5 hr/day

Activity level: seated, at rest

Phase III

Heat: Same as Phase II; duration = 3 hr/day

Vibration:

Day	1	2	3	4	5
Frequency, Hz	10	25	60	125	250
Acceleration, g-rms	0.57	0.90	2.10	4.40	8.70

Duration: 1 hr/day

Activity level: standing, at rest

In Phase III of the study, the temperature and humidity remained the same as in Phase II, but the subject was exposed to "segmental-body" vibration. This involved the subject standing on a nonvibrating platform while grasping a vibrating hand grip.

The six subjects were divided into three pairs and subjected to exposures in the sequence given in the following table.

Pair Group	Weeks 1 and 2	Week 3	Week 4	Week 5
1	Heat only	Phase II, Sec. A	Phase II, Sec. B	Phase III
2	Heat only	Phase II, Sec. B	Phase III	Phase II, Sec. A
3	Heat only	Phase III	Phase II, Sec. A	Phase II, Sec. B

A daily time schedule for all data collecting and sampling for each phase is located in appendixes II-IV. These schedules were used by the monitor and all technicians on every experimental run and were rarely more than 2 min off the actual time.

Physiological Monitoring Equipment and Methods

Temperatures — Skin and rectal temperatures were measured with Yellow Springs Instrument Company series 700 thermistors (model 709A and model 701, respectively) and were recorded on a Digitek Datalogger (model 2000) thermometry system. The rectal thermistor was inserted 15 cm beyond the anal sphincter. The six skin thermistors were placed on the lateral surface of the forearm and upper arm, center of the calf muscle, the middle lateral side of the thigh, and above the nipples of the right and left chest. The skin thermistors were held to the skin by specially designed skin thermistor holders (see appendix V). Skin and rectal temperatures were recorded at 5-min intervals throughout the

entire testing periods of Phase II and Phase III. The mean skin temperature was calculated according to the formula based on surface area (ref. 35):

$$\text{Mean Tsk} = 0.13\text{Tsk}_{\text{forearm}} \pm 0.06\text{Tsk}_{\text{upperarm}} \pm 0.21\text{Tsk}_{\text{calf}} \pm 0.21\text{Tsk}_{\text{thigh}} \pm 0.20\text{Tsk}_{\text{left chest}} \pm 0.19\text{Tsk}_{\text{right chest}}$$

These constants for each segment represent the fraction of the surface area of the whole body accounted for by that body segment. The mean body temperature was calculated (ref. 36)

$$\text{Mean } T_{\text{body}} = 0.20 \text{ Mean Tsk} \pm 0.80T_{\text{rectal}}$$

Sweat rates — Localized sweat rates were determined by passing dry air through a capsule attached with Velcro straps to the skin. The air temperature and water vapor in the airstream exiting the capsule were measured by resistance hygrometry, and compared to the capsule supply side air humidity and temperature. The sweat capsules designed by the author used a tangentially spiraled airflow to capture the sweat (see appendix VI). The inlet air enters the side of the capsule tangentially, spirals around the inside of the capsule, and exits through the center top of the capsule. The airstreams were measured for relative humidity and temperature by Thunder Scientific Corporation Resistance Hygrometers (model 2300H1T21) and were recorded with a Digetek Data Logger (model 1100 Data Scan Sentinel). Airflow throughout the capsules was calibrated weekly and monitored continuously by in-line rotameters and a vacuum pressure gauge. The sweat capsules were attached to the upper arm, lower arm, and calf, and were placed adjacent to the skin thermistor at those sites. See appendix VII for calculations used to compute sweat rate.

Weight changes were determined by weighing the subject prior to entering the booth and within 5 min after exiting the booth at the end of the daily test. No fluid intake was allowed during Phase II or III testing.

Oxygen uptake – Oxygen uptake measurements were measured with a standard technique utilizing an Applied Electrochemistry S-3A oxygen analyzer and a Godard Capnograph Mark II carbon dioxide analyzer (ref. 37). Both gas analyzers were calibrated prior to each determination and at the end of the day with two reference gases using the Scholander technique (ref. 38). Five oxygen-uptake determinations were performed daily on each subject: prior to booth heating; 35 min after the start of booth heating; 5 min after the beginning and before the end of the vibration and in the middle of the 2.5-hr vibration period; and 15 min after vibration during the heated recovery period. The respiration rate and volume were determined with a Parkinson-Cowan spirometer and recorded on a three-channel Gould model 2400 recorder.

Heart rate – Heart rate was monitored with a Hewlett Packard (model 78203C) ECG unit attached to three Quinton Quik-Prep electrodes. The ECG electrodes were attached in the area of the sternal notch, xiphoid process, and fifth intercostal space. The heart rate obtained with the Hewlett Packard unit was also routinely compared to the heart rate obtained with the Beckman Impedance Rheograph unit, which was used in the arm blood flow measurements (refs. 39, 40).

Arm blood flow – Upper and lower arm blood flows were measured by changes in limb segment impedance with a Beckman Bilateral Impedance Rheograph (model BR-100) and Quinton Quik-Prep electrodes (refs. 39, 40). The unit was calibrated daily, prior to and at the end of each subject's

test period. During the segmental-body vibration phase (Phase III), upperarm blood flow was not calculated since the electrical signal was not discernible. Heart rate (beats per minute), pulse blood flow (milliliters per pulse), minute blood flow (milliliters per minute), and volume perfused (milliliters per minute per 100 ml of tissue) were calculated. Each of these values was calculated at t_{-70} min (preheat, previbration); t_{-55} min (preheat, previbration); t_{-30} min (heat, previbration); t_{-20} min (heat, previbration); t_{-5} min (heat, previbration); t_{+5} min (heat, postvibration); t_{+30} min (heat, postvibration); t_{+55} min (heat, postvibration). Due to difficulties in getting clear readings during vibration, no blood perfusion data were collected during vibration test periods.

Blood analyses — Blood samples were analyzed for sodium and potassium ion concentrations, osmolality, hemoglobin, hematocrit, and white blood cell differential count.

The sodium and potassium ion concentrations were determined from 4-ml blood samples which were clotted and spun at 3000 rpm for 10 min. The serum was removed and triplicate determinations were made with an Instrumentation Laboratory flame photometer (model 643). The unit was calibrated at the beginning and end of each set of analyses with two of the manufacturer's reference solutions. The high reference solution contained 140 mmol Na^+/liter and 5 mmol K^+/liter. The low reference solution contained 120 mmol Na^+/liter and 2 mmol K^+/liter.

Serum osmolality was analyzed in triplicate with an Advanced Instruments Digimatic Osmometer (model 3D11). The osmometer was calibrated prior to and at the end of each analyses period with the manufacturer's 290 mosm/kg water reference solution.

Hemoglobin determinations were performed by placing 3 ml of blood in a chilled EDTA anticoagulent tube and analyzed with a Coulter Electronics Hemoglobinemometer (model HGBR) in quadruplicate. Hematocrit was determined by filling four capillary tubes, spinning them in an International Electronics Company microcapillary centrifuge (model MB), and then reading the percent hematocrit with a modified International hematocrit reader.

White blood cell counts were performed from glass slide smears which had been stained with a Wright-Giemsa stain (Camco Quik Stain II, Cambridge Chemical Product). The differential count was determined by counting 100 white blood cells found under randomly chosen fields.

Venous blood samples were drawn from an antecubital vein by venipuncture. The largest daily blood samples were 40 ml: a 20-ml previbration and a 20-ml postvibration exposure sample. The 40-ml sample days occurred only on Mondays, Wednesdays, and Fridays of weeks 3, 4, and 5. The postvibration exposure sample was drawn from the arm opposite the previbration exposure sample. Also, a 20-ml blood sample was taken during the week following the last exposure phase of the experiment. See table 1 for the blood sampling regimen.

Blood samples were never taken on consecutive days, and the samples were collected alternately from the arms when possible. There is no significant difference in these blood constituent concentrations in samples taken from the right and left arms (ref. 41). This blood sampling plan allowed the arm to heal somewhat between punctures and reduced the discomfort.

TABLE 1.- BLOOD SAMPLING REGIMEN

Week 1 (40 ml withdrawn/2 punctures)

Monday, 0900: arm A, withdraw 20 ml venous blood

Friday, 0900: arm B, withdraw 20 ml venous blood

Week 2 (20 ml withdrawn/1 puncture)

Friday, 0900: arm A, withdraw 20 ml venous blood

Week 3 (120 ml withdrawn/6 punctures)

Monday, 0855 (previbration): arm A, withdraw 20 ml venous blood

Monday, 0930 (postvibration): arm B, withdraw 20 ml venous blood

Wednesday, 0855 (previbration): arm A, withdraw 20 ml venous blood

Wednesday, 0930 (postvibration): arm B, withdraw 20 ml venous blood

Friday, 0855 (previbration): arm A, withdraw 20 ml venous blood

Friday, 0930 (postvibration): arm B, withdraw 20 ml venous blood

Week 4 (120 ml withdrawn/6 punctures)

Monday, 0825 (previbration): arm A, withdraw 20 ml venous blood

Monday, 1110 (postvibration): arm B, withdraw 20 ml venous blood

Wednesday, 0825 (previbration): arm A, withdraw 20 ml venous blood

Wednesday, 1100 (postvibration): arm B, withdraw 20 ml venous blood

Friday, 0825 (previbration): arm A, withdraw 20 ml venous blood

Friday, 1100 (previbration): arm B, withdraw 20 ml venous blood

Week 5 (120 ml withdrawn/6 punctures)

Monday, 0855 (previbration): arm A, withdraw 20 ml venous blood

Monday, 1015 (postvibration): arm B, withdraw 20 ml venous blood

Wednesday, 0855 (previbration): arm A, withdraw 20 ml venous blood

Wednesday, 1015 (postvibration): arm B, withdraw 20 ml venous blood

Friday, 0855 (previbration): arm A, withdraw 20 ml venous blood

Friday, 1015 (postvibration): arm B, withdraw 20 ml venous blood

Week 6 (20 ml withdrawn/1 puncture)

Monday, 0855: arm A, withdraw 20 ml venous blood

The blood sampling is summarized in the table below.

Week	Blood withdrawn, ml	Number of punctures
1	40	2
2	20	1
3	120	6
4	120	6
5	120	6
6	20	1
Total	440	22

Safety Considerations, Equipment and Procedures

Subject selection — The five subjects were chosen from a male population between the ages of 22 and 33 years old, and were paid ($4.90/hr). They were volunteers who answered a newspaper advertisement placed in the sports section of a San Jose newspaper. The sixth subject was the principal investigator. All but one of the five paid subjects were college students at San Jose State University. Each subject underwent a complete medical history, physical examination, and evaluation by a non-NASA medical clinic. The medical examination included chest and back X-rays, blood and urine analyses, spirometry testing, stress ECG testing, and a general physical examination. The subject selection was then made, based upon their meeting the following requirements:

1. No apparent physical or mental problems
2. No recent history of major or minor illnesses
3. No surgical operations within the past 12 months
4. Not taking any drugs — prescription or nonprescription
5. Nonsmokers
6. Neither obese nor very thin

7. No personal history of cardiovascular, kidney, liver, neurological, or spinal problems

8. No known or expected problems associated with work in a hot or vibratory environment, such as congenital absence of sweat glands or large areas of scar tissue

After passing the medical examination, the subjects met at the NASA Human Environmental Physiology Laboratory and were informed of the medical subject bill of rights, possible discomforts and inconveniences, and their right to terminate their participation in the experiment. Informed consent forms were passed out to each of the subjects, and discussed in detail (appendix I). At the conclusion of this meeting all the subjects signed the informed consent forms.

Man-rating the shaker table — Since the shaker table was not man-rated, Ames Research Center's Human Research Experiments Review Board (HRERB) (see appendix VIII for HRERB roster) required that a separate man-rating board be assembled. The man-rating board divided its task into three components: procedural and engineering safety requirements for the table alone; stress and fatigue design analysis, load testing, and weld inspection of the chair; fire, health, and safety procedures and equipment with the seat, shaker, and heat booth assembled and evaluated with respect to the biomedical technicians and subjects.

The safety equipment used to control a "runaway" shaker table included seven separate checks divided into four categories. The four categories include mechanical checks; internal feed-back loops; a subject control switch; and experiment monitoring control switches.

The mechanical checks included "overtravel stops" built into the instrument to limit the vertical displacement range of the shaker table.

Also within the mechanical check group, sufficient weight was added to the shaker platform to force the drive unit to work near the maximum potential, thus also insuring the shaker unit could not "run away."

Two types of internal monitoring and control feedback loops were used to monitor the shaker table's performance, and to shut off the shaker within milliseconds. There was an independent monitor (accelerometer) at the shaker table which measures "g" forces. When this accelerometer detected a change outside the "g" range prescribed by the experiment, an electrical trigger was activated which terminated the output signal. The other internal control was a servo control unit which regulated the oscillator signal.

A subject control switch was also used. The "dead man" style of switch was used by the subject, and must be depressed by the subject during the experiment. Any time the subject decided to terminate the experiment, he simply released the depressed button which in turn shut off the electrical power to the unit.

At the experiment monitor's desk a "quick kill" switch was installed so the monitor could terminate the experiment immediately by shutting off the power to the shaker assembly. Also within about 5 ft of the monitor was the main breaker switch for the entire shaker assembly.

In addition to the above seven separate checks on the shaker, prior to any subject being seated on the shaker platform, a test at the experimental frequency and acceleration was performed for each control device. This safety procedure served as a monitoring test that each safety control device was working.

The seat was purchased from a used farm equipment dealer and was an uncushioned metal tractor seat with a metal back 33 cm high. The seat

had five vertical and four diagonal airplane tubing (4130) metal legs welded to the base of the seat and to a metal base plate. The metal base plate (1/4 in. thick) was bolted to the shaker table with twelve 3/8 in. 16 threads/in. bolts, each 1 in. long. A quick release seat belt was bolted to brackets which were welded to the chair. Prior to testing the chair on the shaker table, a detailed stress and fatigue analysis was performed by the Ames Research Center structural engineering department. After their analysis, the chair was bolted to the shaker, loaded with 180 lb of lead, and vibrated at 150% the intensity of the high "g" exposure value for 1 hr, which was 240% of the actual exposure time to be used for the experimental test. This test was performed for each test frequency. The chair was then unbolted and sent to a non-NASA welding analysis firm for weld crack analysis. No cracks were detected.

After the safety features had been installed on the shaker, the seat had been thoroughly tested, and the heat booth was installed, the man-rating board performed an on-site inspection. After this on-site inspection, a list of requirements was submitted which dealt with fire safety, electrical shock analysis, biotechnician safety (including OSHA approved steps up to the shaker table), and medical emergency procedures and standby medical equipment. These requirements were completed, and the machine received a "man-rating" statement for this experiment.

In addition to the subject being able to stop the experiment at any time, three other people could stop the vibration exposure — the principal investigator, the medical monitor, and the computer operator for the shaker table. During all vibration test periods, these three people were always present. The medical monitor or the principal investigator could stop the experiment at any time. At the end of the whole-body vibration

and heat test periods, a urine sample was collected daily by the medical monitor and evaluated for possible internal problems, such as bleeding. No aberrant findings were detected from the urine samples.

RESULTS

Due to the voluminous amount of data collected during this experiment, only data trends will be discussed in this chapter. The subjects' arithmetic means for the physiological variables measured in this study are tabulated in appendixes IX-XVII, and are displayed in the graphs in this section. The data for the heart rate, and skin and rectal temperatures are found in appendixes IX and X, respectively. The sweat rate data are tabulated in appendix XI. The limb blood perfusion rate data are in appendix XII.

Segmental-Body Vibration

The evolution of a heat-stress condition involves a gradient of systemic physiological changes which begins with an increase in core temperature followed by an increase in heart rate. The sequencing of these changes is important in determining whether one is in a marginal heat-stress condition or in the region of a well-defined heat-stress problem. When a person is put into a hot environment, the body begins to absorb the heat, which gradually raises the deep body or core temperature. This increase in internal heat, which is reflected in an increased blood temperature to the brain, increases vasodilation which decreases peripheral resistance (increases limb segment blood perfusion rates), and results in decreased blood pressure. This decrease in blood pressure is the cause of the fainting often associated with heat exposures (heat syncope). With a decrease in blood pressure, the baroreceptors decrease

neural firing rates to the cardioinhibitory centers, and the heart rate is increased. Thus, increased core temperature occurs first, and is followed by an increase in the heart rate.

With continued exposure to heat-stress conditions, changes in other secondary localized responses can also be detected. Sweating increases, peripheral blood perfusion rates increase, and metabolic rate, respiration rate, and oxygen uptake rate increase slightly. The basis for the slight increases in metabolic rates, respiration rates, and oxygen uptakes is probably related to the increase in core temperature, which affects the rate of reaction of various biochemical reactions that make up the metabolic activities of the body. This temperature effect is called the Q_{10} effect and results from the temperature dependence of the chemical reaction rates which provide metabolic energy.

Systemic responses to segmental-body vibration — Attention was first directed to the effect of segmented-body vibration (sbv) on the systemic response as measured by rectal temperature, heart rate, respiratory variables, and blood components. The first hypothesis tested was that these responses were independent of the frequency of sbv. The Friedman two-way analysis of variance was chosen to test this hypothesis since it is a nonparametric test which is suitable for use with matched data; that is, it takes account of the fact each subject was tested at each frequency. The test was applied to the data at 30, 60, and 90 min after the start of vibration. The latter time is 30 min into the recovery period so that both the onset of effects and recovery therefrom were investigated. Table 2 contains the results of the three tests which indicate that there was no difference in rectal temperature, heart rate, respiration rate, and respiratory exchange ratio as a function of frequency. This result

TABLE 2.- RESULTS OF FRIEDMAN TESTS FOR SIGNIFICANT DIFFERENCES BETWEEN FREQUENCIES FOR RECTAL TEMPERATURE, HEART RATE, AND RESPIRATORY VARIABLES AT 30, 60, AND 90 min

	30 min	60 min	90 min
Rectal temperature			
X^2r	2.40	3.65	7.80
p-value	0.60	0.55	0.10
Heart rate			
X^2r	2.25	2.75	3.05
p-value	0.68	0.60	0.55
Respiration rate			
X^2r	3.18	5.40	4.25
p-value	0.60	0.25	0.36
Respiratory exchange ratio			
X^2r	3.00	4.88	3.92
p-value	0.54	0.30	0.45

indicates that these data can be pooled to allow a more powerful test of the next issue, whether or not these systemic responses differed from what would be expected under conditions of heat without vibration.

The original intent for the control period was the 1-hr test heat period prior to vibration. Due to problems in heating the booth quickly and coming to an early equilibrium, it was decided this heat period would be inadequate to establish baseline rates of change for the physiological variables. Although the booth did come to equilibrium about 20-30 min

prior to the vibration exposure, it was thought at least an hour at a constant environmental condition was necessary to provide a good baseline for a resting subject. As a consequence it was decided to use published data to establish the expected response under heat without vibration. As will be seen, the response curves for both rectal temperature and heart rate follow a shallow S-shaped curve when plotted against time. That is, there is a lag period before the variables begin to respond, followed by a relatively linear increase, which then makes a transition to an equilibrium value. The first description of this temporal pattern of change that was investigated was the slope of the linear portion of the curve. The mean rectal temperature (± S.E.M.) for the pooled sbv exposures in this study was 0.0033 ± 0.0005°C/min for the entire test period of 90 min. From a study conducted by the Navy (ref. 42), the rate of rise in resting rectal temperature over a 150-min test period at 36°C was also 0.003°C/min. This study involved three heat-acclimated men dressed similarly to the subjects in this study. Additionally, the mean rectal temperatures from that study actually match my data for the 60 min of sbv exposure. Rectal temperatures at each 20-min interval from the Navy study and my study are presented below.

	Time, min						
	0	20	40	60	80	90	100
Navy	37.3	37.3	37.4	37.5	37.5	--	37.6
sbv	37.3	37.3	37.4	37.5	37.5	37.6	--
S.E.M.	0.05	0.05	0.05	0.04	0.04	0.04	--

Henane and Bittel (ref. 43) performed a similar study with 12 men, 24.5 years, dressed similarly to our subjects, and exposed them for

90 min at 45°C, and 24% RH. They compared the rates of change in mean rectal temperatures prior to heat acclimation and after heat acclimation. The rate of increase in mean rectal temperature for the resting heat-acclimated group was 0.0033°C/min, which is the same rate as was observed in our sbv exposures over the same time period. The mean rectal temperatures for their study were a few tenths of a degree lower than the mean rectal temperatures observed in this study. These lower temperatures could be due to population differences, such as degree of conditioning, and also to the fact that my subjects were preheated about 15 min. The important fact here is not so much the difference in actual temperatures, as it is the rate of change. Additionally, from the formulas used by Givoni and Goldman (refs. 44, 45) to predict rectal temperature response to work, environment, and clothing, the rate of increase in rectal temperature observed in this study also matches the predicted slope for a 60-min period at these conditions, 0.0033°C/min. Their reported mean rectal temperature data also match the sbv rectal temperature data in this study. These equations were based on a sample of 24 young, healthy, reasonably fit and heat-acclimated men. The sbv exposure mean rectal temperature I observed (37.50 ± 0.04°C) at 60 min is also very close to the value reported by Macpherson (ref. 46) for heat-acclimated men standing in 120°F (48.9°C) dry air for 1 hr (37.51 ± 0.09°C).

If the pooled data for the sbv exposures are corrected to the data presented by Henane and Bittel (ref. 43), by subtracting the difference in baseline rectal temperatures from all the sbv exposure data points, the sbv exposure curve lies almost directly on top of their curve, and well within the S.E.M. limits of their data (fig. 2). As is also seen in figure 2, there is an initial lag in rectal temperatures for both

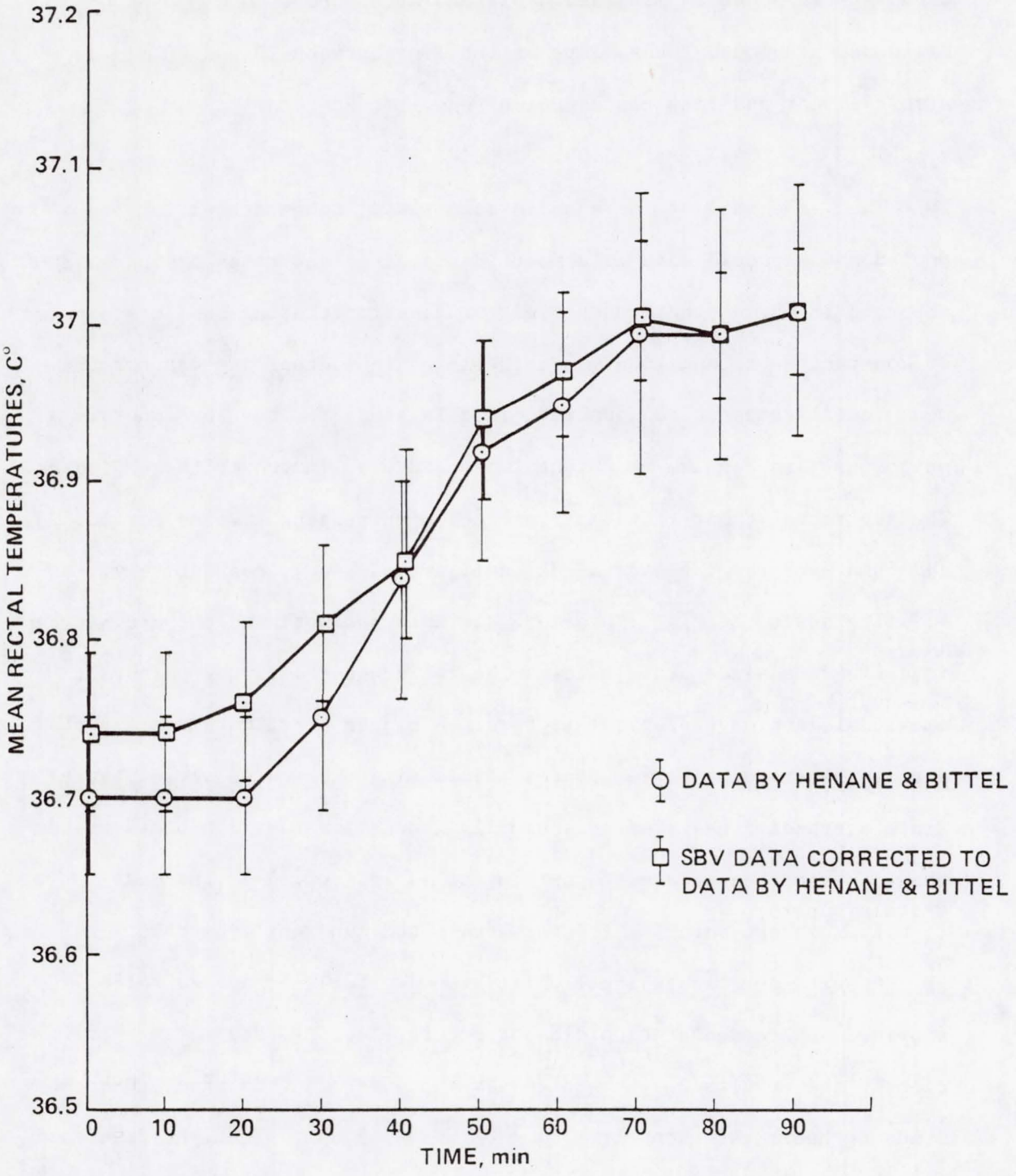

FIGURE 2. MEAN (± sem) CORRECTED RECTAL TEMPERATURES FOR THE SBV EXPOSURE, AND DATA PRESENTED BY HENANE AND BITTEL (43).

data sets of about 20 min, and a plateau after about 70 min. As was discussed previously, the slope of the line between 20 and 70 min is almost linear and fits the equation (uncorrected data):

$y = 37.23 \pm 0.0042X$, $R^2 = 0.99$.

Thus, the rate of increase in mean rectal temperatures for the pooled sbv exposure data is almost identical to the rates of increase reported in three other independent studies for resting subjects exposed to constant heat conditions. Furthermore, the actual numbers for the mean rectal temperatures observed in this study for the sbv exposure match the data for the mean rectal temperatures in two of these studies. The sbv rectal temperatures are plotted with respect to time for 10, 25, and 60 Hz and for 125 and 250 Hz in figures 3 and 4, respectively.

The heart rate is a much more variable parameter to compare between investigators since heart rate can be influenced easily by postural, emotional, and physiological factors. A higher heart rate was expected during the standing sbv exposures than during the seated wbv exposures since a standing heart rate is usually about five to eight beats higher than a seated heart rate. According to Adolf (ref. 47), the mean (± S.E.M.) heart rate for 11 heat-acclimated men went from 86 ± 4 to 99 ± 4 beats per minute (bpm) while standing for 1 hr at 120°F (48.9°C), provided severe dehydration did not occur. Adolf's data match fairly closely the results of the pooled sbv exposures in this study, where the standing heart rate went from 86 ± 3 to 95 ± 3 bpm. The 1-hr predicted heart rate (99 ± 6 bpm) from the formulas by Givoni and Goldman (ref. 45) is slightly higher than the observed heart rate (95 ± 3 bpm), but still within the range of error for their formulas. Macpherson (ref. 46) also reported a standing heart rate of 99 ± 3 bpm at the end of 3 hr at 120°F

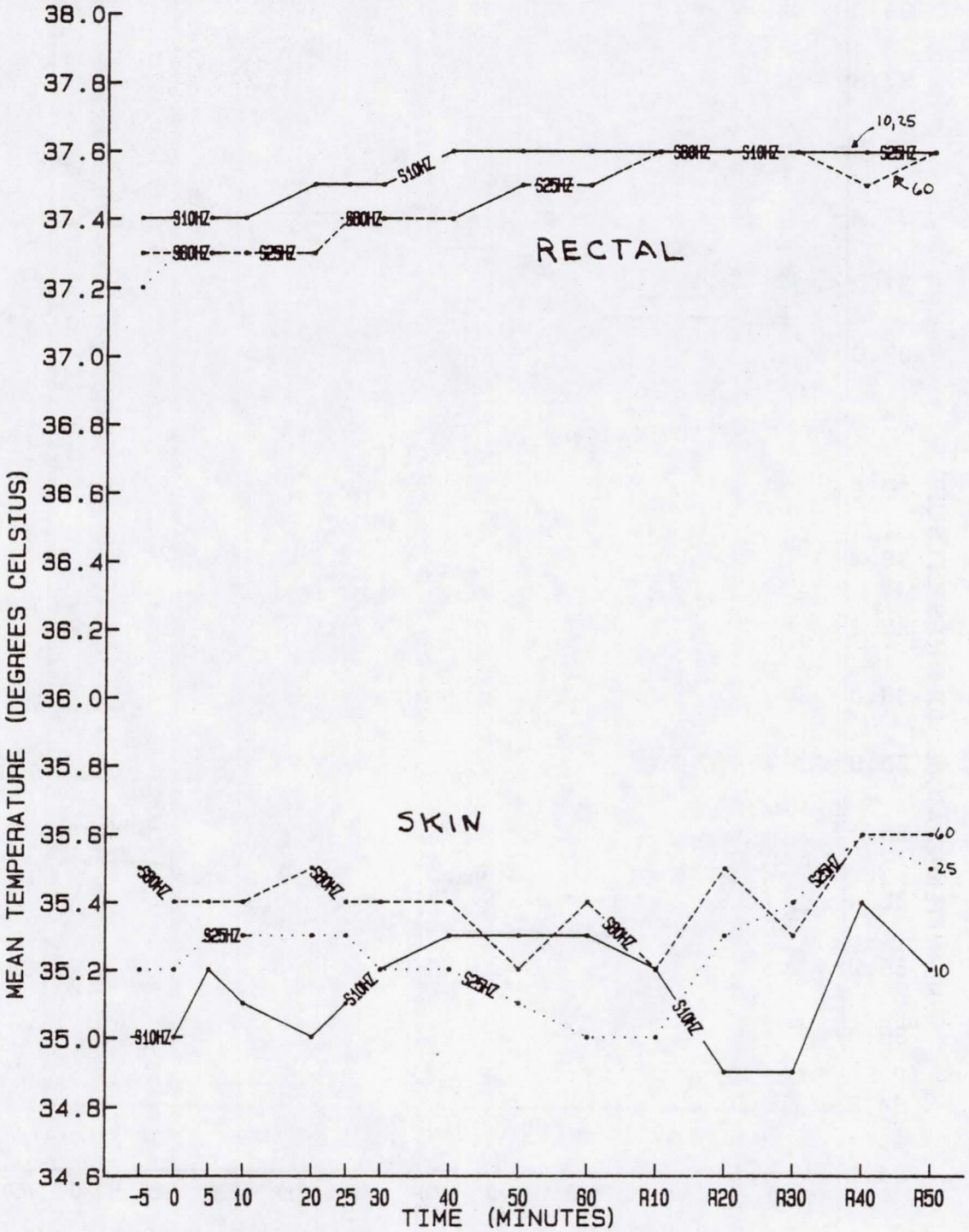

FIGURE 3. MEAN SKIN AND RECTAL TEMPERATURES FOR 10, 25, AND 60 HZ SBV EXPOSURES.

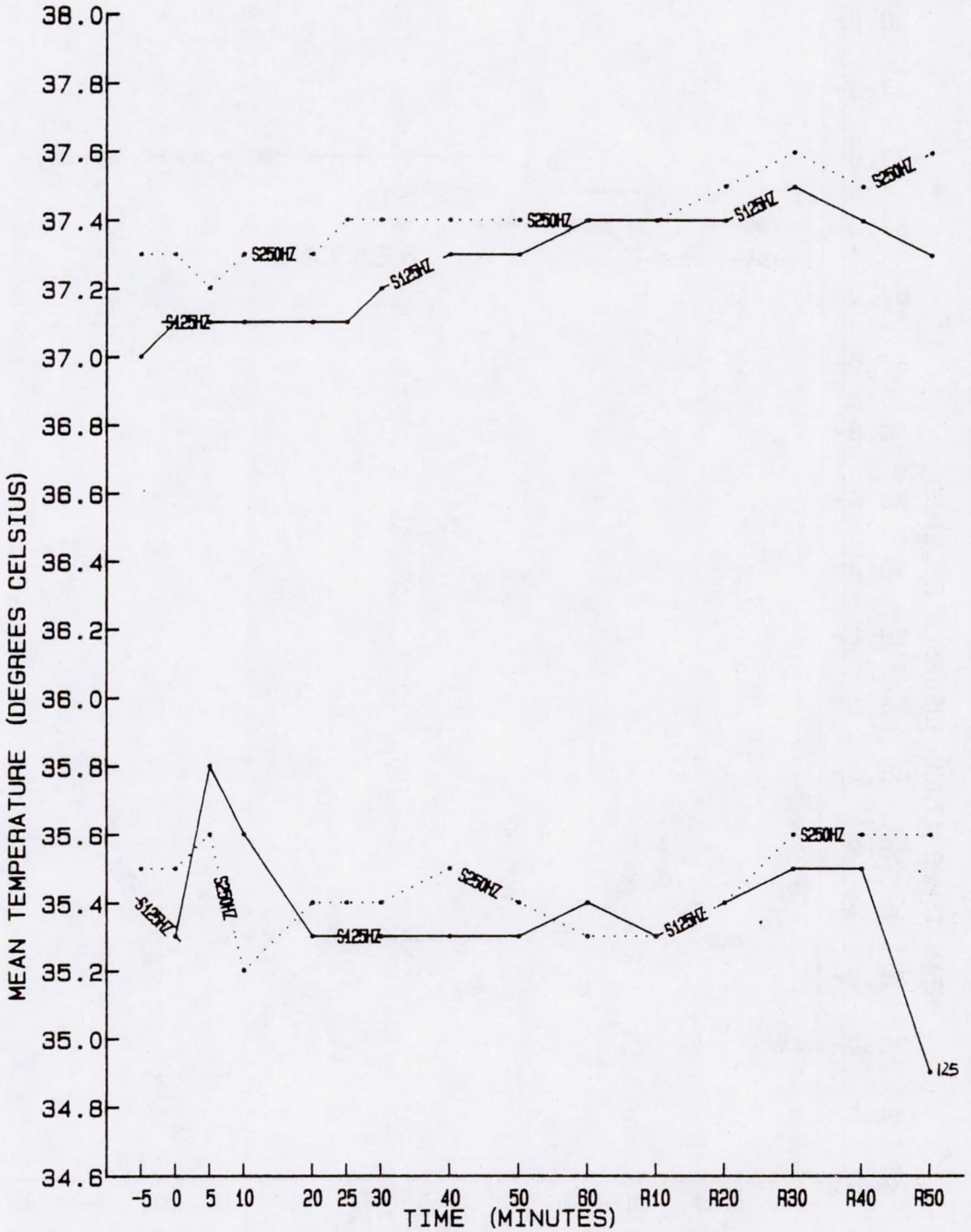

FIGURE 4. MEAN SKIN AND RECTAL TEMPERATURES FOR 125 AND 250 HZ SBV EXPOSURES.

(48.9°C), and at an air velocity of 33 m/min. Unfortunately, he did not report the heart rate at any other time during the 3 hr, so it is impossible to determine if this was a heart rate which had reached an equilibrium value.

Adolf (ref. 47) also reported the heart rate tended to stabilize after 1 hr in the heat, provided severe dehydration (6% body weight loss) was not allowed. This stabilization in heart rate was also observed in the pooled sbv exposure data but after only 30 min (tested with the paired t-test and at a significance level of 0.05). The pooled sbv exposure mean (± S.E.M.) heart rates are as follows.

	Time, min				
	0	10	30	60	90
Mean bpm	85.9	88.4	93.6	94.6	91.5
S.E.M.	3.4	3.3	3.6	3.2	3.4

The rate of increase in the pooled mean heart rate for sbv during the first 30 min (prior to equilibrium) is 0.26 bpm, with the equation fitting the line $y = 85.87 \pm 0.26X$, $R^2 = 1.00$. The rate of increase in the mean heart rate from Adolf's data was 0.22 bpm, with the equation $y = 86.00 \pm 0.22X$, $R^2 = 0.99$ (see fig. 5 for graphic representation of the mean pooled sbv heart rate). Figure 6 provides the change in heart rate with time for each frequency.

Respiration — There was no significant difference in any of the respiratory variables — rates, corrected volume of oxygen uptakes, respiratory exchange ratio (RER) — with respect to frequency (table 2). The respiration rate increased temporarily during the first 5 min of vibration at 10 and 250 Hz (fig. 7). This temporary increase in respiration rate is

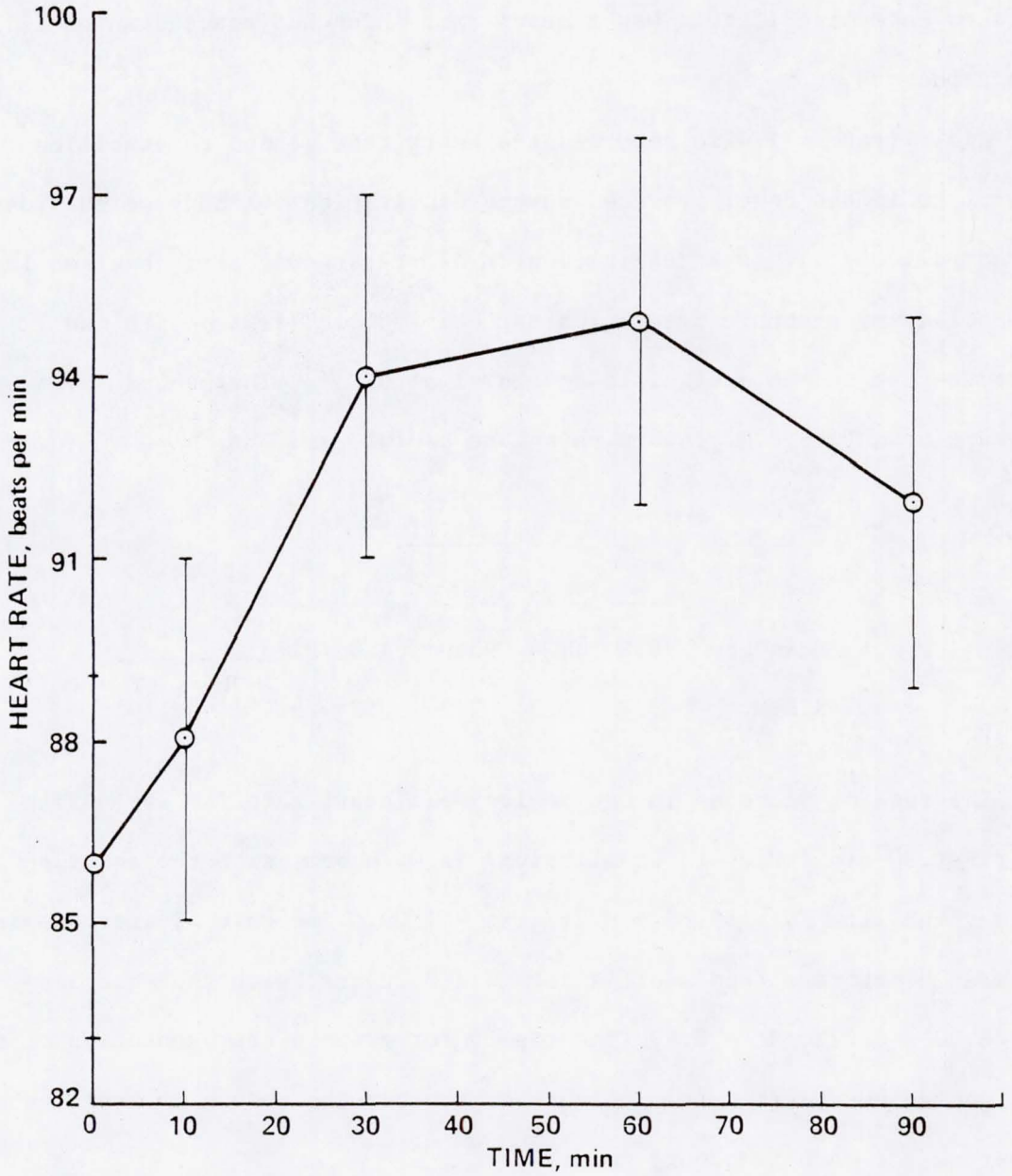

FIGURE 5. THE POOLED SBV MEAN (± sem) HEART RATE WITH RESPECT TO TIME

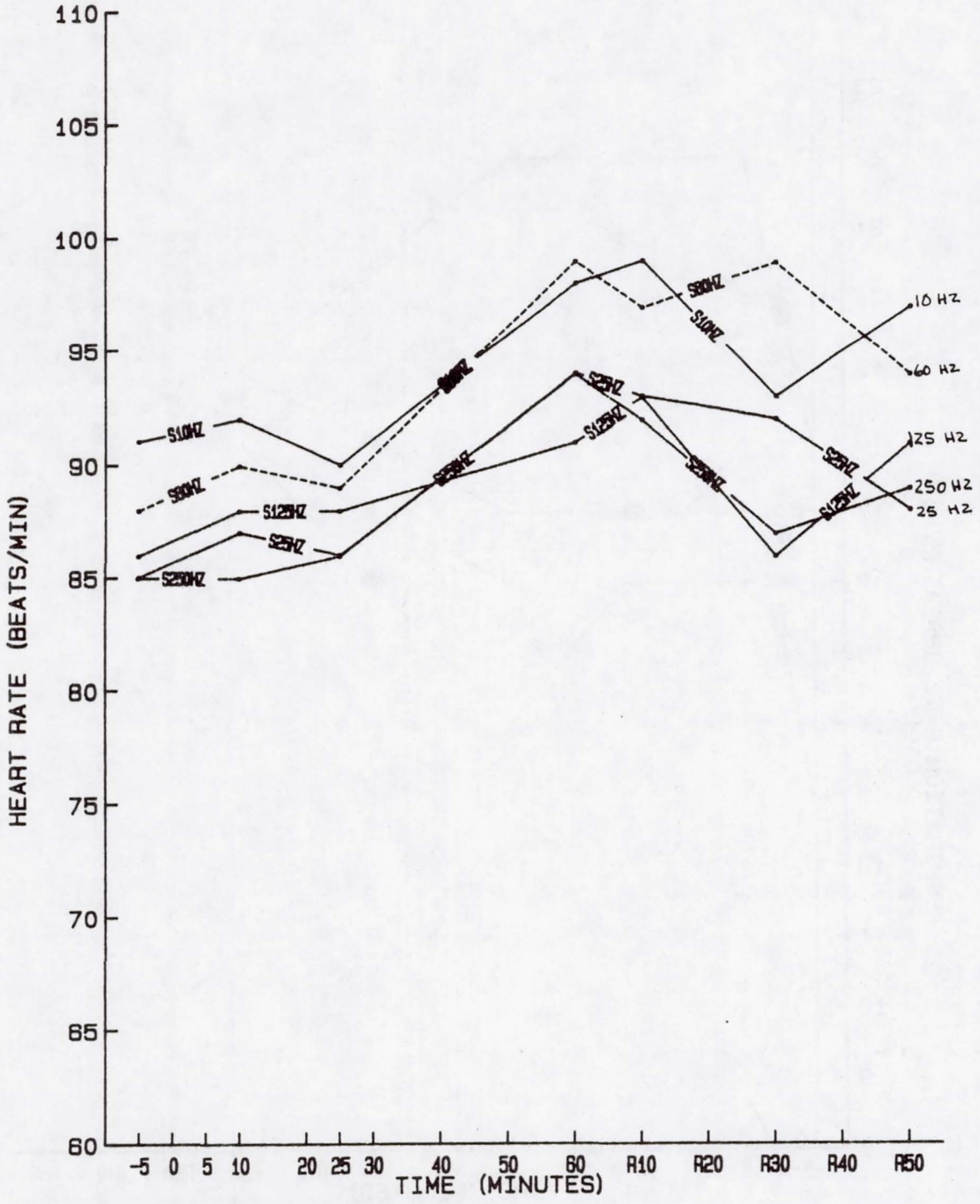

FIGURE 6. MEAN HEART RATES FOR 10, 25, 60, 125 AND 250HZ SBV EXPOSURES.

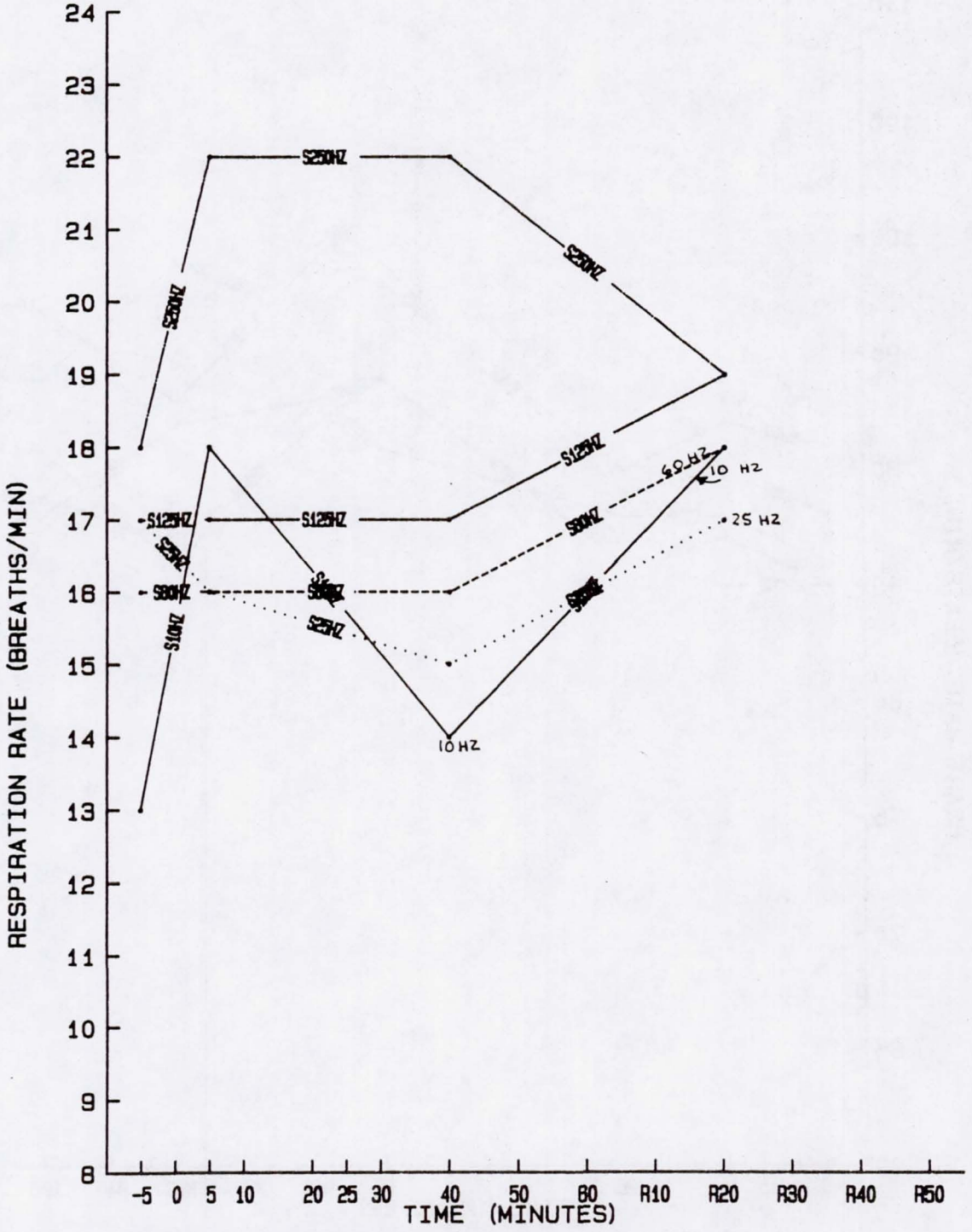

FIGURE 7. MEAN RESPIRATION RATES FOR 10, 25, 60, 125 AND 250 HZ SBV EXPOSURES.

frequently seen with whole-body vibration exposures and is not usually associated with sbv exposures. The data for the respiration rates are tabulated in appendix XIII. The mean (corrected to NTP) volume of oxygen consumed (oxygen uptake) increased slightly (not significant) from 0.23 ± 0.01 to 0.27 ± 0.01 liters/min at the beginning of sbv to the 40-min sbv test period reading. Between 40 and 80 min, the oxygen uptake rate remained constant (figs. 8, 9). The mean respiratory exchange ratio, which is the ratio of the rate of carbon dioxide output to the rate of oxygen uptake and which is indicative of metabolic rate, did not change significantly during the sbv exposure testing period (fig. 10). The data for oxygen uptakes are located in appendix XIV. The RER data are in appendix XV. Table 3 lists the data for the mean ± S.E.M. pooled sbv data on respiratory rates, oxygen uptakes, and respiratory exchange ratio for 0, 40, and 80 min.

Blood component concentrations — Serum osmolality and sodium ion concentration increased after the 1-hr sbv exposure. The increase in these concentrations probably reflects an increased water loss and implies the overall sweat rate was inhibited very little. No significant changes in potassium ion concentrations (meq/liter), hemoglobin (g), hematocrit (%), or white blood cell counts occurred after any sbv exposure. The white blood cells were counted for polymorphoneutrophils (PMN), lymphocytes, monocytes, basophils, and eosinophils. The data for all blood analyses are in appendix XVI.

Thus, from the preceding comparison of the pooled sbv exposure mean rates of increase for the rectal temperatures and heart rates with the data reported in the literature for subjects at rest or standing, it appears the results of these sbv exposures closely match the data expected in a situation with heat and without vibration.

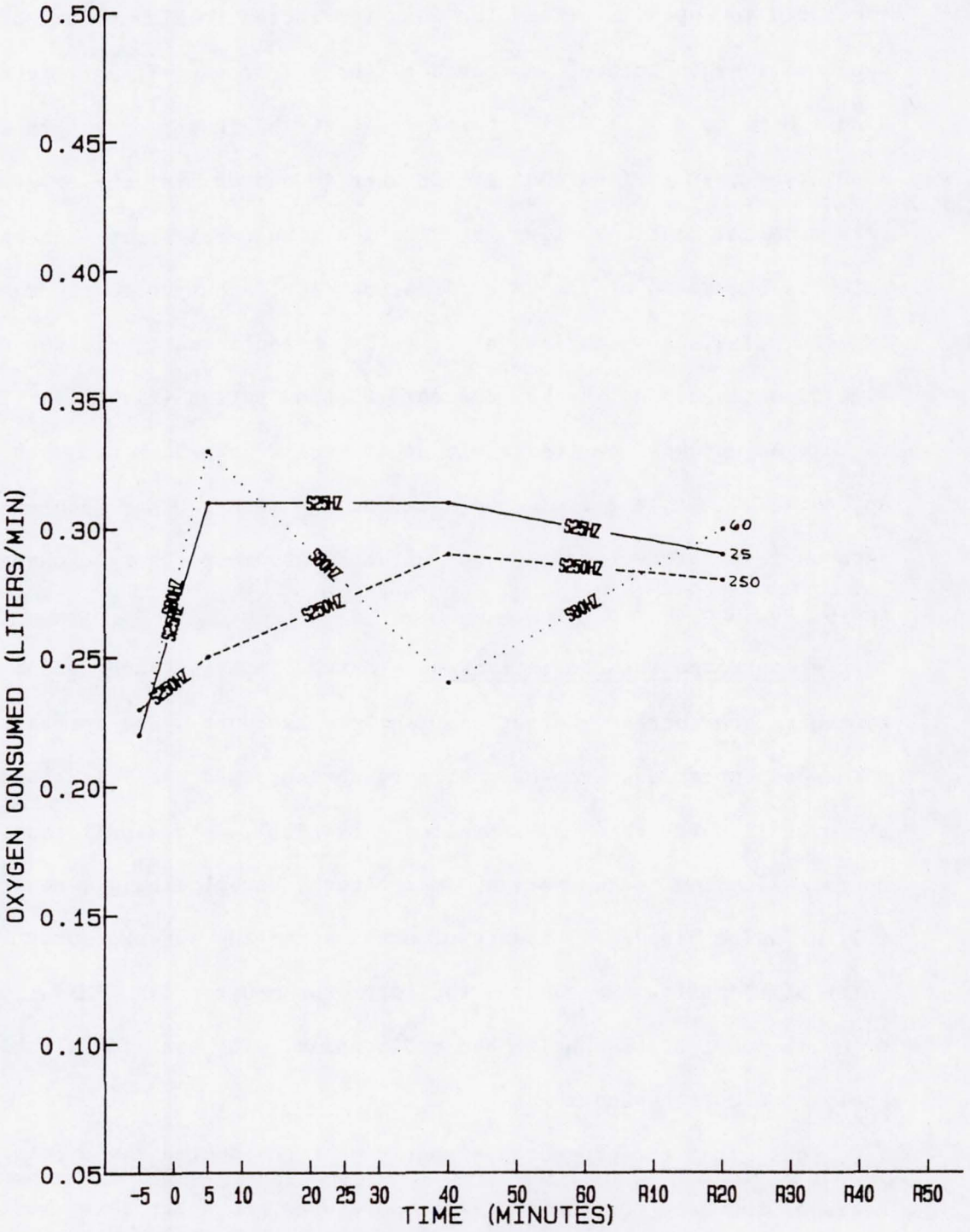

FIGURE 8. MEAN OXYGEN UPTAKES FOR 25, 60, AND 250 HZ SBV EXPOSURES.

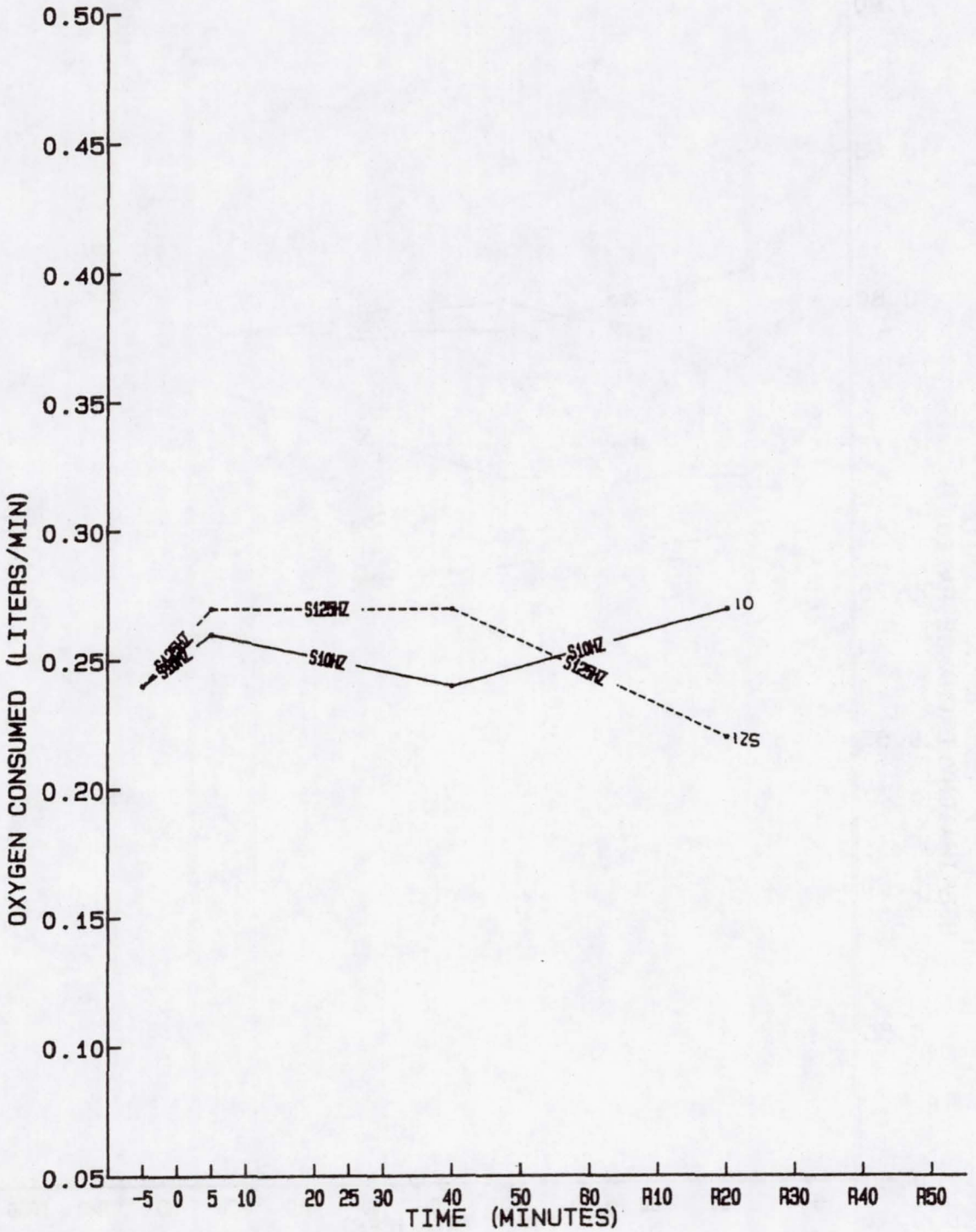

FIGURE 9. MEAN OXYGEN UPTAKES
FOR 10 AND 125 HZ SBV EXPOSURES.

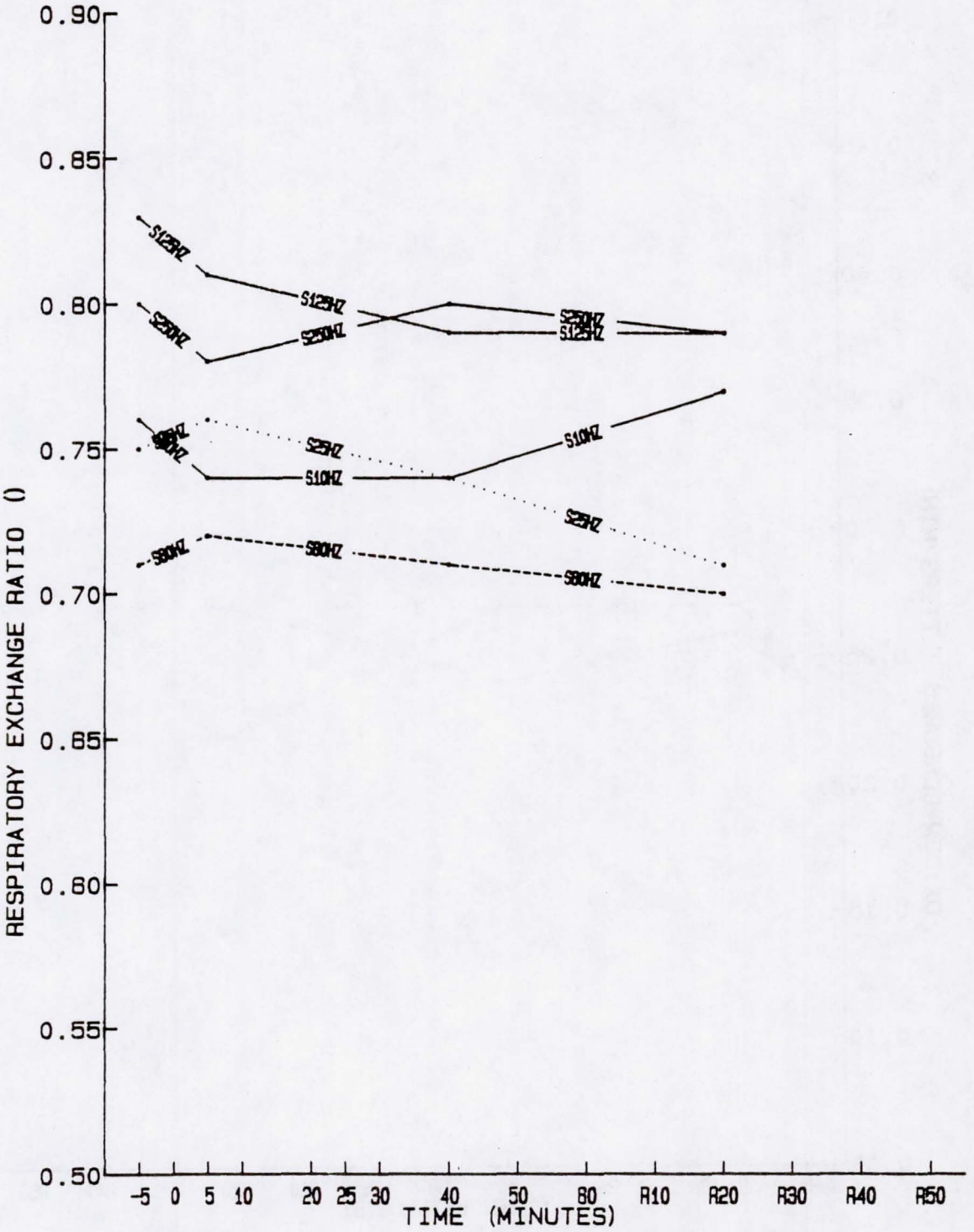

FIGURE 10. MEAN RESPIRATORY EXCHANGE RATIOS FOR 10, 25, 60, 125, and 250 HZ SBV EXPOSURES.

TABLE 3.- MEAN (± S.E.M.) RESPIRATORY RATES, OXYGEN UPTAKES AND RESPIRATORY EXCHANGE RATIO FOR POOLED sbv DATA AT 0, 40, and 80 min

	0 min	40 min	80 min
Respiratory rate, breaths/min			
Mean	16	17	18
S.E.M.	1	1	1
Oxygen uptakes, liters/min			
Mean	0.23	0.27	0.27
S.E.M.	0.01	0.01	0.01
RER values			
Mean	0.77	0.76	0.75
S.E.M.	0.02	0.02	0.02

All of these data lead to the conclusion that the sbv exposure sustained by the subjects in this study did not affect the basic systemic thermoregulatory process which control heart rate and rectal temperature in a hot environment.

Localized responses during sbv exposure — The localized responses include limb-segment blood perfusion rates, localized sweat rates, and localized skin temperatures. The localized responses are reviewed in light of being consistent with the systemic effects, that is, in this case, a mild heat-stress condition.

Limb segment blood perfusion rates — No upperarm (UA) blood perfusion rate data were collected during the sbv phase due to difficulties in getting stable UA readings when the subject was standing. The forearm

(FA) blood perfusion data during vibration appears to be consistent with the expected response to hand-arm vibration exposure in that the perfusion rate decreased as indicated in table 4.

TABLE 4.- MEAN (± S.E.M.) BLOOD PERFUSION RATES FOR THE FOREARM (FA) FOR PREVIBRATION, POSTVIBRATION, AND AFTER 50 min OF RECOVERY FOR THE sbv EXPOSURES

Frequency, Hz	Previbration	Postvibration	After 50-min recovery
10	6.7 ± 0.6	4.2 ± 0.5[a]	5.8 ± 1.1
25	5.6 ± 1.1	5.9 ± 1.1	4.9 ± 0.6
60	5.0 ± 1.2	5.4 ± 0.7	5.1 ± 1.0
125	5.8 ± 0.7	4.5 ± 0.7[a]	5.4 ± 0.5
250	5.5 ± 0.5	4.4 ± 0.4[a]	5.3 ± 0.8
Mean	5.7 ± 0.3	4.9 ± 0.3[a]	5.3 ± 0.2

[a]Significantly different from previbration level at $p < 0.05$ by paired t-test.

The mean ± S.E.M. FA blood perfusion rate prior to vibration was 5.7 ± 0.3 ml of blood per 100 ml of tissue per minute. After vibration, the mean blood perfusion rate decreased to 4.9 ± 0.3 ml per 100 ml of tissue per minute ($p < 0.05$, paired t-test) and even as late as 40 min after the vibration, had returned only to 5.3 ± 0.2 ml blood per 100 ml of tissue per minute, which indicates incomplete recovery even 40 min later. As is seen in table 4 and figure 11 significant decreases ($p < 0.05$, paired t-test) in the FA blood perfusion rates occurred at 10, 25, 60, 125, and 250 Hz, which suggests a frequency-dependent effect for this localized response.

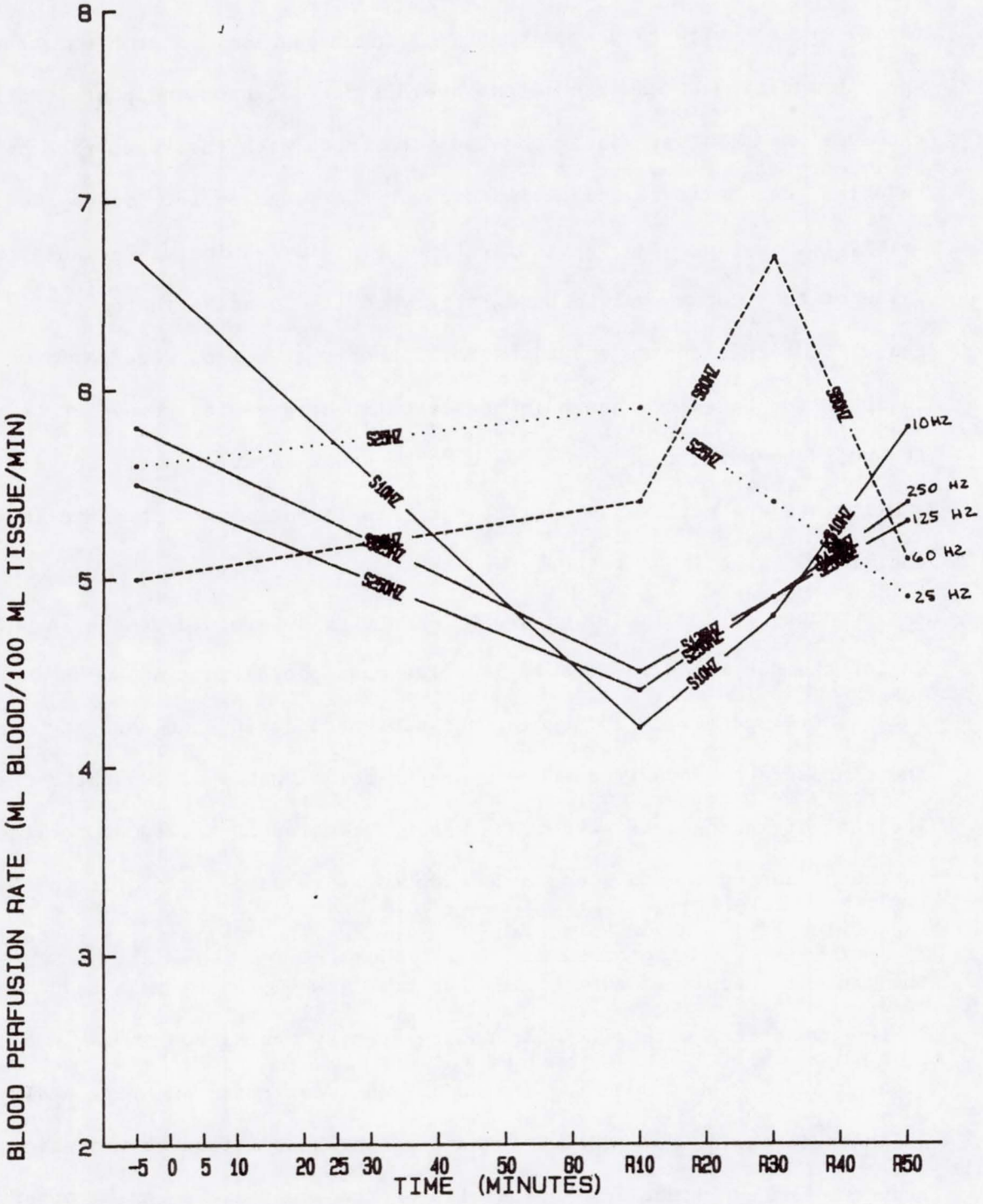

FIGURE 11. MEAN FA BLOOD PERFUSION RATES FOR 10, 25, 60, 125, AND 250 HZ SBV EXPOSURES.

Localized sweat rates — If sweat rates are significantly elevated after vibration exposure then one can state that vibration is not interfering greatly with this particular cooling mechanism. A problem occurs when in addition to changes in the overall previbration and postvibration exposure sweat rates, vibration also interferes with the sweat rate by reducing the amplitude and periodicity of the sweat cycle. Since sweating is cyclic, testing at a particular time point is meaningless because the value of that datum would depend largely on its location in the cycle phase. For this reason equations for linear regressions were performed to check for rates of change in the overall pattern with respect to time. It should be noted that the r-values for these equations will be very small since the pattern is cyclic, but this is not important since it is the slope of the lines which is sought.

Changes in the localized sweat rates were determined on the FA and UA for the sbv exposures (table 5). The mean pooled sbv FA and UA sweat rates increased from 0 to 60 min, and decreased during the recovery period. There appears to be a frequency-dependent relationship with sweat rates. The rate of increase in sweat rate was greatest at 25 and 60 Hz for the FA and UA during the sbv, and most reduced at 125 and 250 Hz for the FA and at 10, 125, and 250 Hz for the UA. During the 50-min recovery period the trend in localized sweat rates for both the UA and FA is a reduction in the sweat rate with time. As can be seen by the slopes in table 5 and figures 12-15, the slopes for the localized sweat rates are very small, which indicates a near equilibrium situation. The effect of vibration on reducing the amplitude and periodicity of the sweat cycle can be observed in figure 13 for 125 Hz.

TABLE 5.- RATES OF CHANGE (SLOPE) IN SWEAT RATES FOR THE UA AND FA FOR EACH TEST FREQUENCY OF sbv

Frequency, Hz	0-60 min	60-110 min
FA		
10	0.00065	-0.00094
25	0.00087	0.00069
60	0.00228	-0.00106
125	0.00017	-0.00037
250	-0.00012	-0.00146
Mean	0.00042	-0.00063
UA		
10	-0.00056	-0.00260
25	0.001360	-0.00103
60	0.001360	-0.00006
125	0.000831	0.00026
250	0.000824	-0.00206
Mean	0.000864	-0.00109

Localized skin temperatures — Although localized skin temperatures are usually reflective of ambient conditions, at constant conditions the skin temperature can indicate changes in cutaneous blood flows or sweat rates. A decrease in skin temperature could be associated with a reduced skin blood flow or an increase in sweat evaporation. The problem with statistically comparing a specific mean skin temperature with another point is the same problem which occurred with the sweat rates — the data

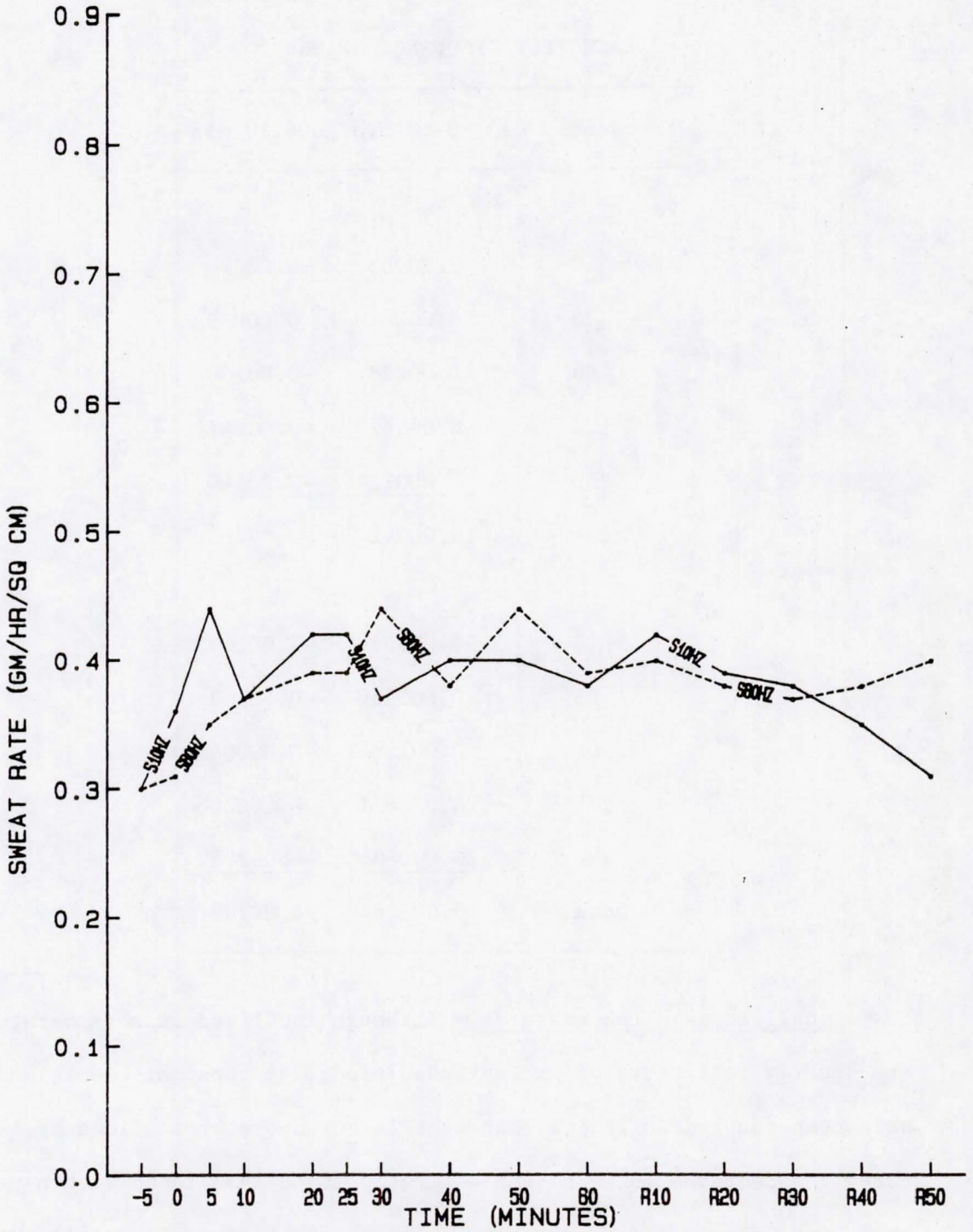

FIGURE 12. MEAN UA SWEAT RATES FOR 10 AND 60 HZ SBV EXPOSURES.

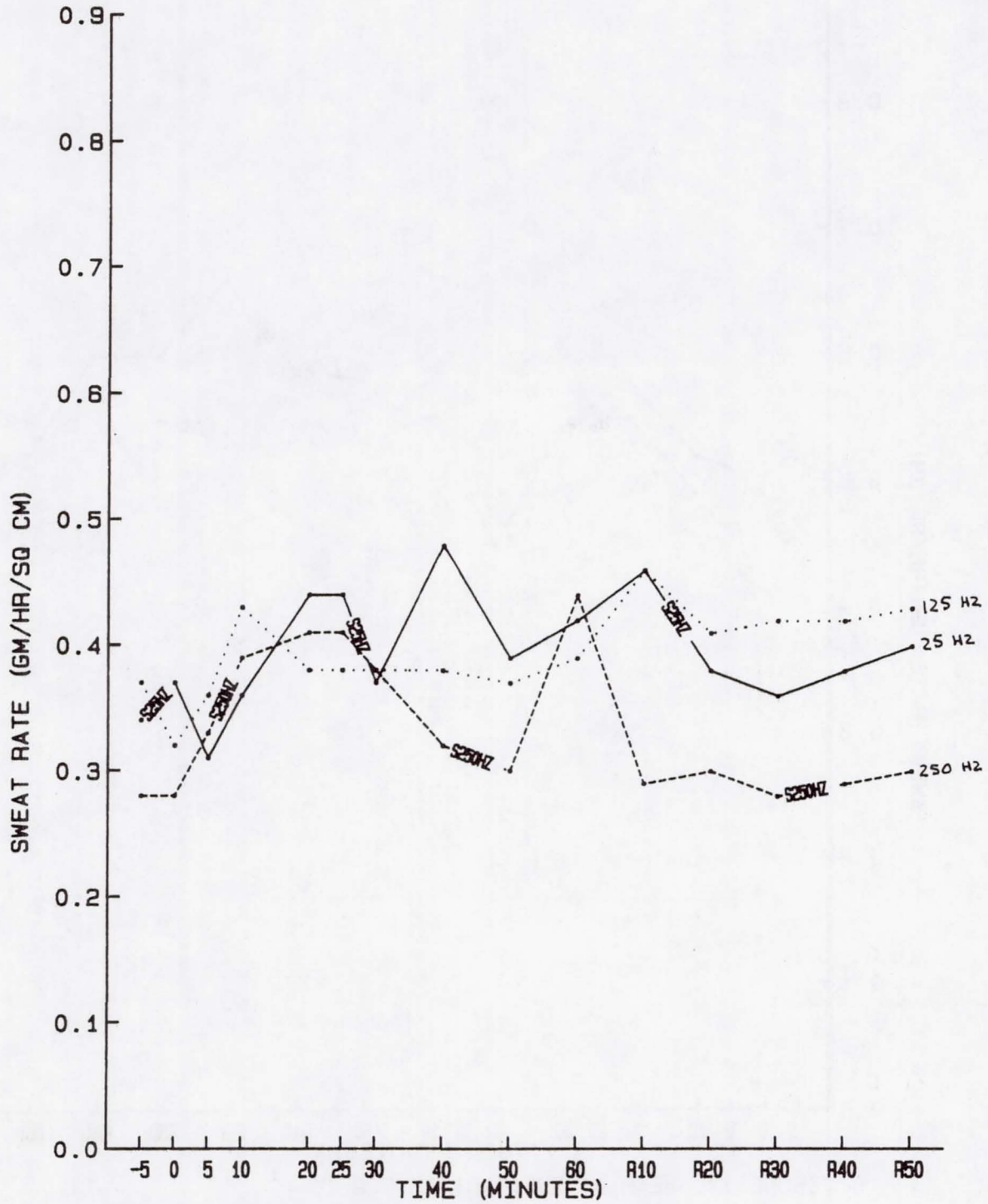

FIGURE 13. MEAN UA SWEAT RATES FOR 25, 125, AND 250 HZ FOR SBV EXPOSURES.

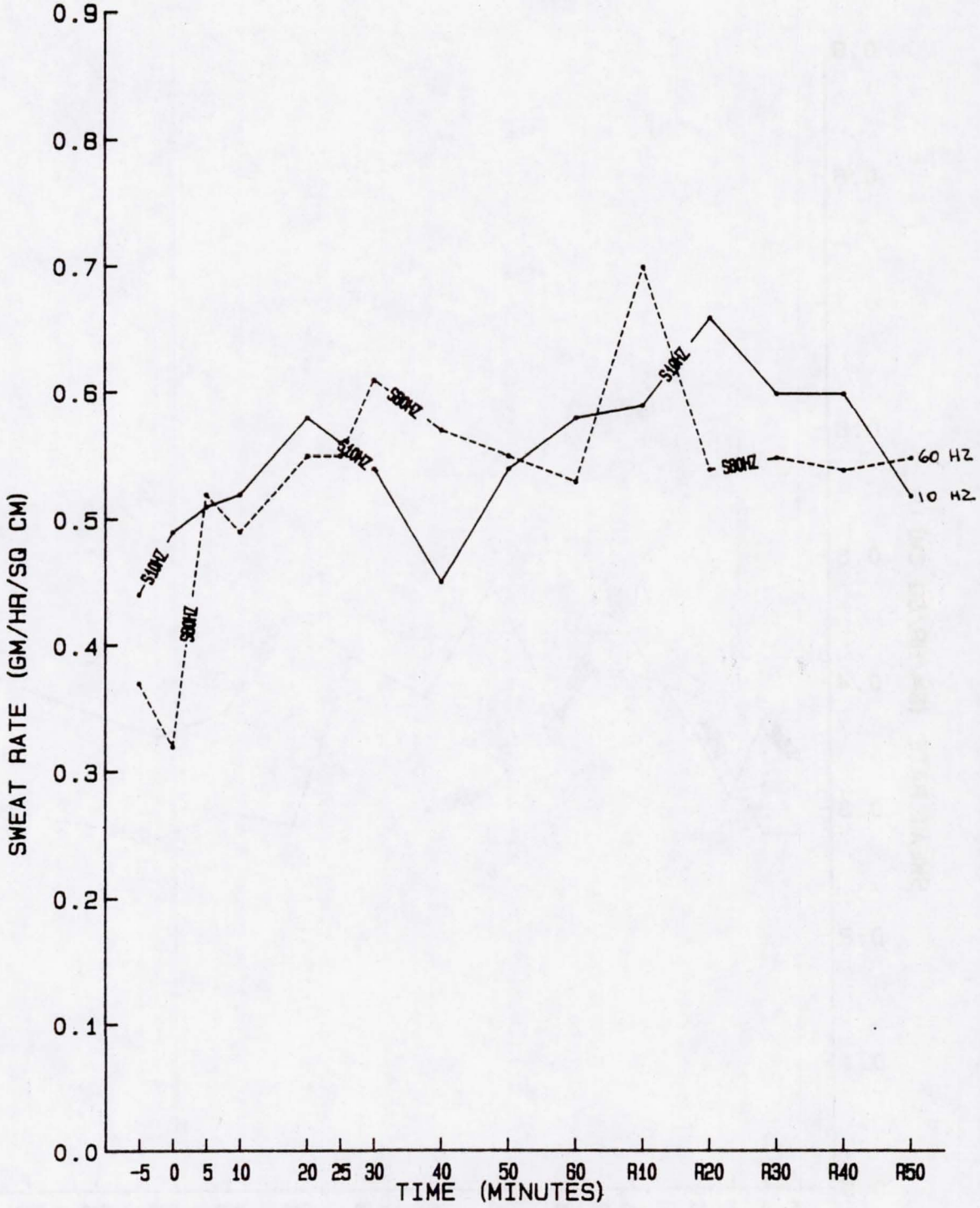

FIGURE 14. MEAN FA SWEAT RATES FOR 10 AND 60 HZ SBV EXPOSURES.

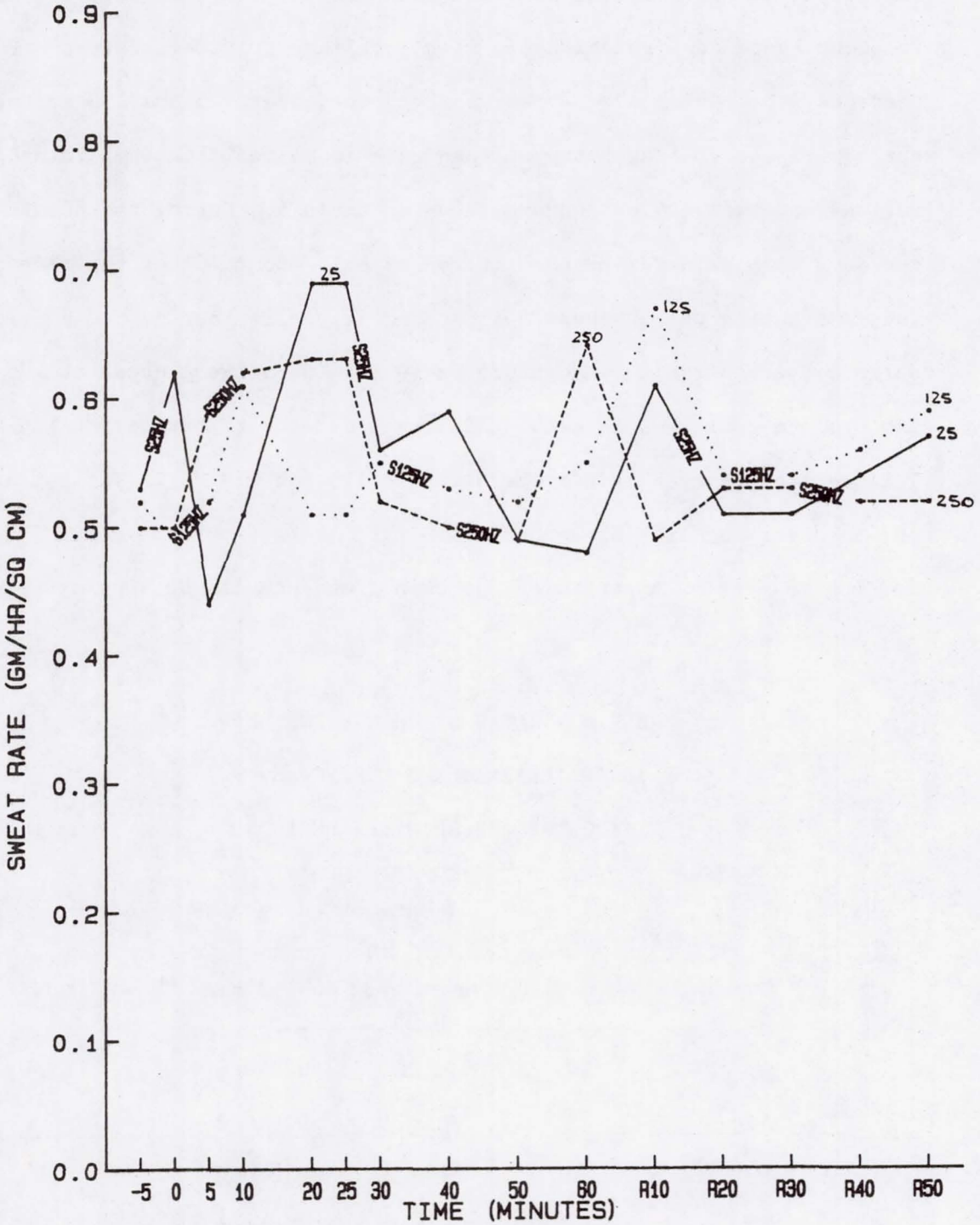

FIGURE 15. MEAN FA SWEAT RATES FOR 25, 125 AND 250 HZ SBV EXPOSURES.

cycle sinusoidally. As seen in figures 3 and 4, the cycles appear to take about 20 min. Again, like the sweat rates, there appears to be a frequency-dependent relationship with localized skin temperature. Furthermore, the cycles also appear to be dampened at 125 and 250 Hz for the sbv exposure. The rates of change in localized skin temperatures for sbv and recovery period are shown in table 6. During the 60 min of the sbv, mean skin temperature increased only about 75% of the rate of increase during the recovery period. Additionally, during the 60-min sbv period, the mean skin temperature decreased (negative slopes) at 125 and 250 Hz, and continued to decline during the recovery period for 125 Hz. This decline in mean skin temperature at 125 and 250 Hz is consistent with the data for the limb blood perfusion rate and sweat rate data. The trends for the mean skin temperatures for each frequency can be seen in figures 3 and 4.

TABLE 6.- RATES OF CHANGE FOR MEAN SKIN TEMPERATURES DURING sbv EXPOSURE AND RECOVERY PERIODS

Frequency, Hz	Rate of change	
	0-60 min	60-110 min
10	0.0100	0.00029
25	0.0039	0.01000
60	0.0026	0.01000
125	-0.0003	-0.01000
250	-0.0004	0.01000
Mean	0.0034	0.0041

Summary of localized responses during sbv — The results of the localized responses during the sbv exposure are consistent with the conclusion from the systemic response section that a mild heat-stress condition developed. Although the systemic responses during the sbv exposure reflected no frequency-dependent relationship, the localized responses did exhibit different rates of change at various frequencies; for example, the blood perfusion rates decreased at 10, 125, and 250 Hz. The FA sweat rates decreased significantly ($p < 0.05$, paired t-test) at 125 and 250 Hz, while the UA sweat decreased at 10 Hz. At 125 and 250 Hz the FA sweat rate increased only about half the rate measured at 25 and 60 Hz. Also, the amplitude of the UA sweat rate cycle became very dampened at 125 Hz (fig. 13). The mean skin temperatures decreased only at 125 Hz and 250 Hz during the 60-min vibration period. Nothing in the respiration rates, oxygen uptakes, or respiratory quotients suggests the core temperature was elevated enough to increase these variables by a Q_{10} effect. Actually, the slight increase in postexposure serum osmolality and sodium ion concentration would lead one to suggest that sweating is operating normally, and from the size of the slopes in the regression equations, the sweat rate appears to be near equilibrium.

Thus, from the results of the systemic and localized physiological responses, it appears that combined exposure to sbv and heat produces a heat-stress condition similar to one which would be produced by the heat alone.

Segmental-Body Vibration Recovery Period

The recovery period is an important aspect of exposure to many physical agents. Knowledge about the recovery period is necessary to

determine the severity of the effects and the length of time required before another exposure should be allowed. Information about the recovery period is also useful in determining possible exacerbating effects and possible increased susceptibility to other types of health problems. For example, if a vibration exposure in the heat greatly reduced one's thermoregulatory responses for an hour after the vibration exposure, then either repeated vibration exposures in the heat or activities which required higher metabolic loads could more readily initiate or exacerbate some form of heat casualty situation.

Little of the vibration biodynamic and heat-stress research has focused on the recovery aspects of the postexposure period. For this reason alone, an analysis of these data is useful.

Two data comparisons were performed for the recovery period data — a comparison between the end of vibration and the end of the heated recovery period, and a comparison of the previbration data with that for the end of recovery period. The first comparison identifies changes during the recovery period, while the second comparison provides an overview of the resulting effects from both the vibration and recovery phases. Systemic and local responses during the sbv recovery period are discussed below.

Rectal temperatures — At the end of the recovery period, rectal temperatures for all sbv test frequencies were significantly ($p < 0.05$, paired t-test) more elevated than the previbration exposure levels, and at about the same level as would be expected from a heat exposure without vibration. As also seen in figure 2 there was no change in rate of increase at the end of sbv.

Heart rates — The heart rates decreased during the first 30 min of the recovery period, except at 60 Hz (NS) (fig. 6). The mean pooled heart

rate at the beginning of the recovery period decreased from 95 ± 3 bpm to 92 ± 3 bpm (NS), 30 min later. During the last half of the recovery period, the heart rates again increased at 10, 125, and 250 Hz, and decreased at 25 and 60 Hz for a mean of 92 ± 2 bpm (fig. 6).

Limb segment blood perfusion rates — There were no significant changes in the FA blood perfusion rates during the 50-min recovery period (table 4). Although the FA blood perfusion rates decreased during the sbv exposure at 10, 125, and 250 Hz, by the end of the recovery period all the FA blood perfusion rates returned to levels not significantly different from the previbration exposure rates.

Sweat rates — During the recovery period, the rate of change in FA and UA sweat rates decreased at all frequencies except 25 Hz and 125 Hz for the FA and UA, respectively. This decrease in sweat rate is probably a result of the equilibrium reached with the rectal temperature, and a reduction in the drive to compensate for an increasing rectal temperature.

Skin temperatures — The end of recovery period UA, FA, and LC skin temperatures were not different significantly from the previbration exposure levels at any frequency. During the recovery period, the UA and left chest (LC) increased slightly (NS) over the end of vibration measurements at 250 Hz and 60 Hz, respectively. Only at 125 Hz did the mean skin temperature continue to decrease throughout the 50-min recovery period.

Respiration — The end of recovery respiration rates did not change significantly during the recovery period, nor were there significant differences from the previbration exposure level (table 3). The corrected volume of oxygen uptakes and RER did not change significantly during the recovery period.

Summary of simultaneous heat and sbv exposures — The combination of heat and sbv exposures did not appear to be any more stressful than being exposed to similar heat conditions without sbv regardless of frequency. Although both the systemic and local responses are indicative of a minor heat-stress condition, the responses were normal, expected, and compensatory reactions, and appeared to maintain thermal homeostasis. Attainment of thermal homeostasis is exemplified by the equilibrium attained for both rectal temperature (after 70 min) and heart rate (after 30 min). There was no frequency dependence in the systemic responses, yet there appeared to be frequency dependence for some of the localized responses. The frequency-dependent localized responses were observed at the skin-resonating and energy-absorbing frequencies of 125 and 250 Hz, respectively. Although the local responses displayed some degree of frequency dependency, apparently other heat-dissipating and compensatory mechanisms intervened or else the reduction in the heat-dissipating responses at those frequencies were not of sufficient magnitude to produce a problem systemically.

Whole-Body Vibration

Attention was first directed to the effects of whole-body vibration (wbv) on the primary systemic responses as measured by rectal temperature and heart rate. Although the respiratory variables (rate, oxygen uptake, and respiratory exchange ratio) and the blood components should also be considered systemic responses, they will be discussed with the localized responses since they tend to be regarded as secondary responses which occur as a result of elevated temperature or water loss.

Figures 16 and 17 contain the mean skin (lower group of lines) and rectal (upper group of lines) temperatures for each frequency at the

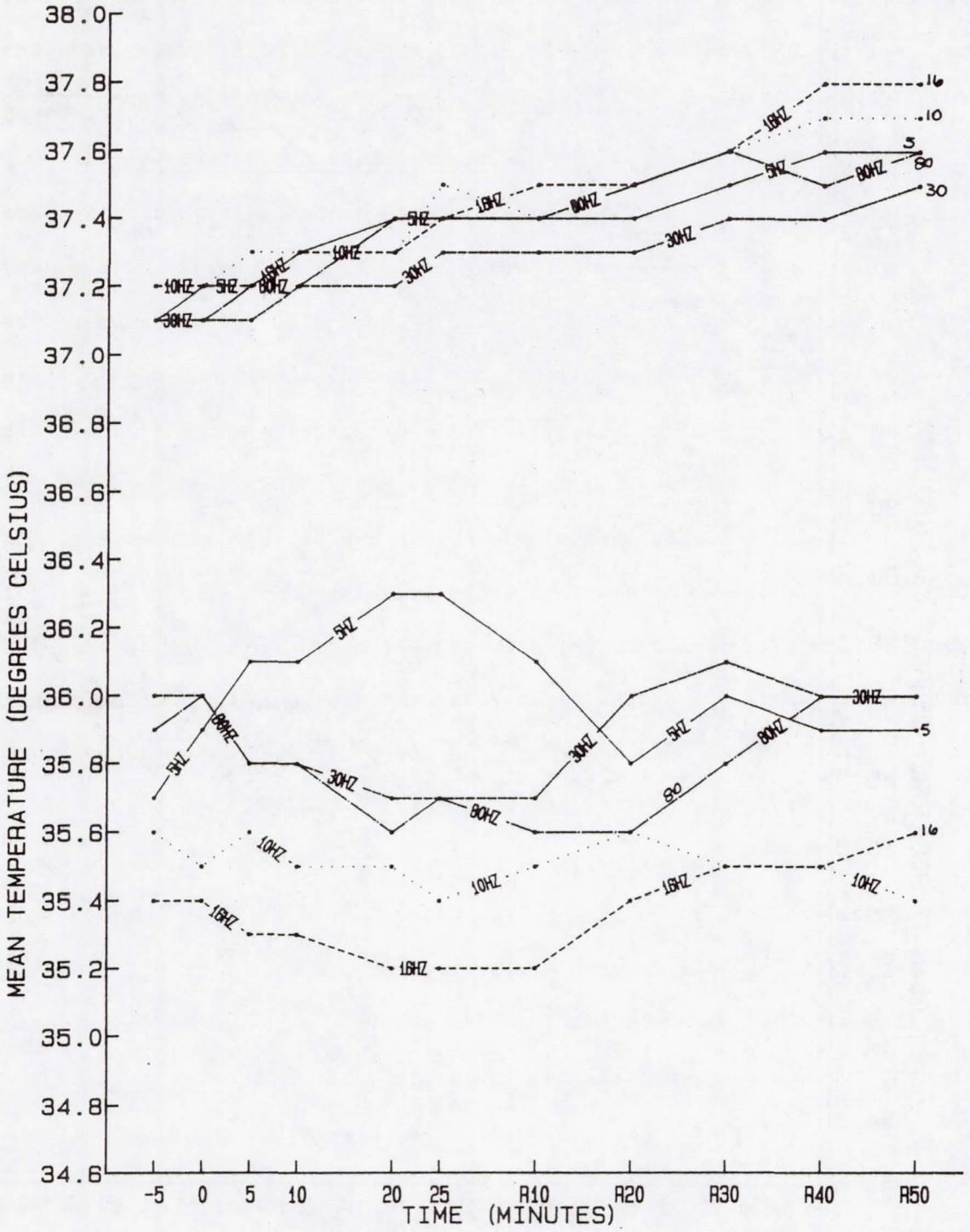

FIGURE 16. MEAN SKIN AND RECTAL TEMPERATURES FOR 5, 10, 16, 30, AND 80 HZ HI WBV EXPOSURES.

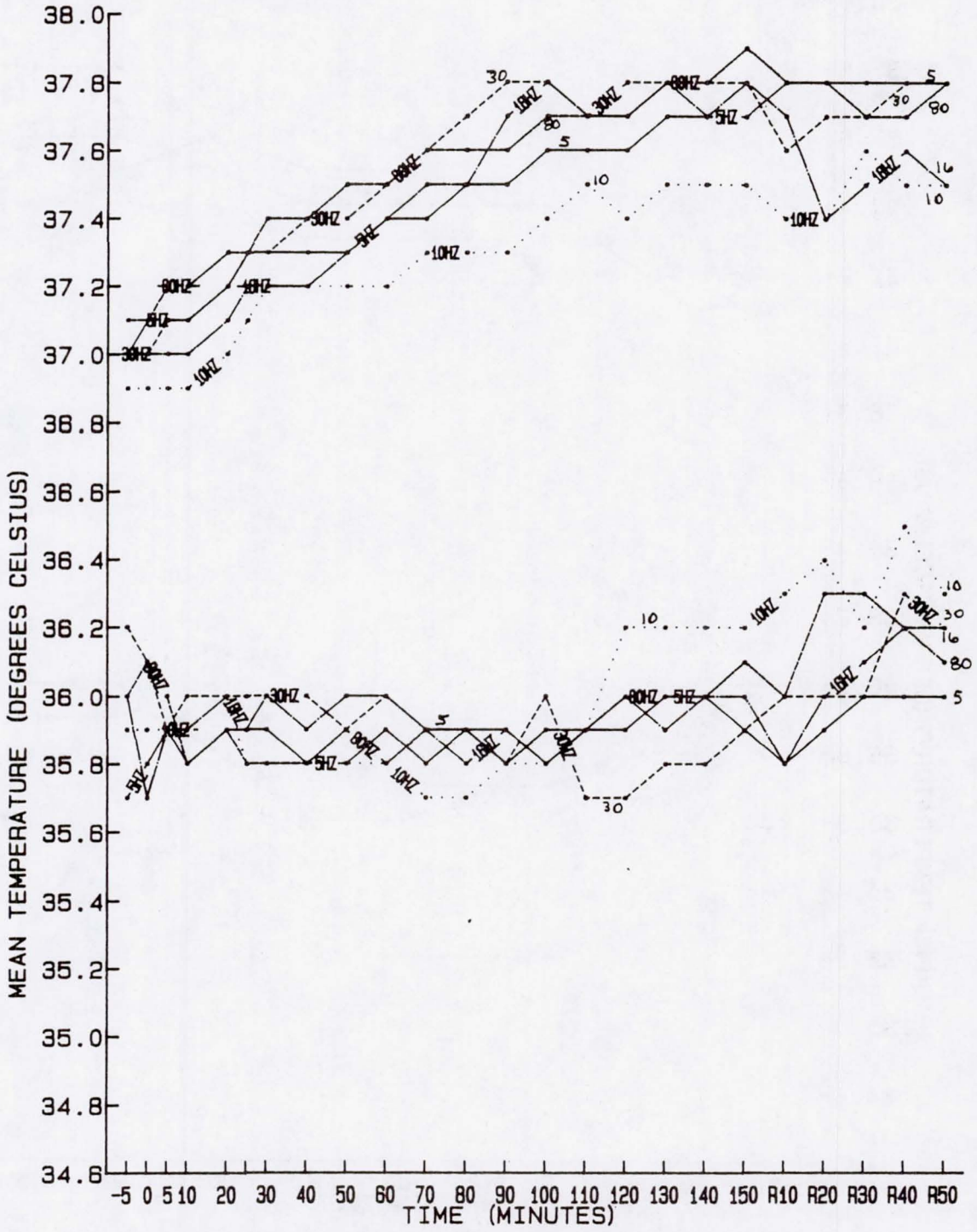

FIGURE 17. MEAN SKIN AND RECTAL TEMPERATURES FOR 5, 10, 16, 30, AND 80 HZ LI WBV EXPOSURES.

HIwbv and LIwbv exposures, respectively. As can be seen in figure 17, the LIwbv rectal temperature appears to reach an equilibrium after about 90 to 100 min.

The heart rate data for the wbv exposures are given in appendix IX. The trends for the mean heart rates for each frequency are displayed in figures 18 and 19 for the HIwbv and LIwbv exposures, respectively. Previous work has shown the expected trend with heart rate during vibration is a transient increase in heart rate during vibration and usually persists for the first 15-20 min of vibration. After the transient increase, the heart rate usually decreases, but remains slightly more elevated than the previbration exposure level. As is seen in figures 18 and 19, the heart rate for each frequency generally follows the expected trend.

As a consequence of the apparent lack of frequency dependence in the foregoing data, the first statistical hypothesis tested was that the primary systemic responses were independent of the frequency of wbv. As was mentioned previously, the Friedman two-way analysis of variance was chosen to test this hypothesis since it is a nonparametric test which is suitable for use with matched data; that is, it takes account of the fact that each subject was tested at each frequency. As in the control phase (sbv), the test was applied to the data at 30, 60, and 90 min after the start of vibration. Recall that the high-intensity wbv (HIwbv) exposure was for 25 min, while the low-intensity wbv (LIwbv) exposure was for 150 min. Table 7 contains the results of the Friedman tests for the three test periods, and, as can be seen from the results, there was no significant difference ($p < 0.05$) in the difference in mean rectal temperatures or mean heart rate as a function of frequency.

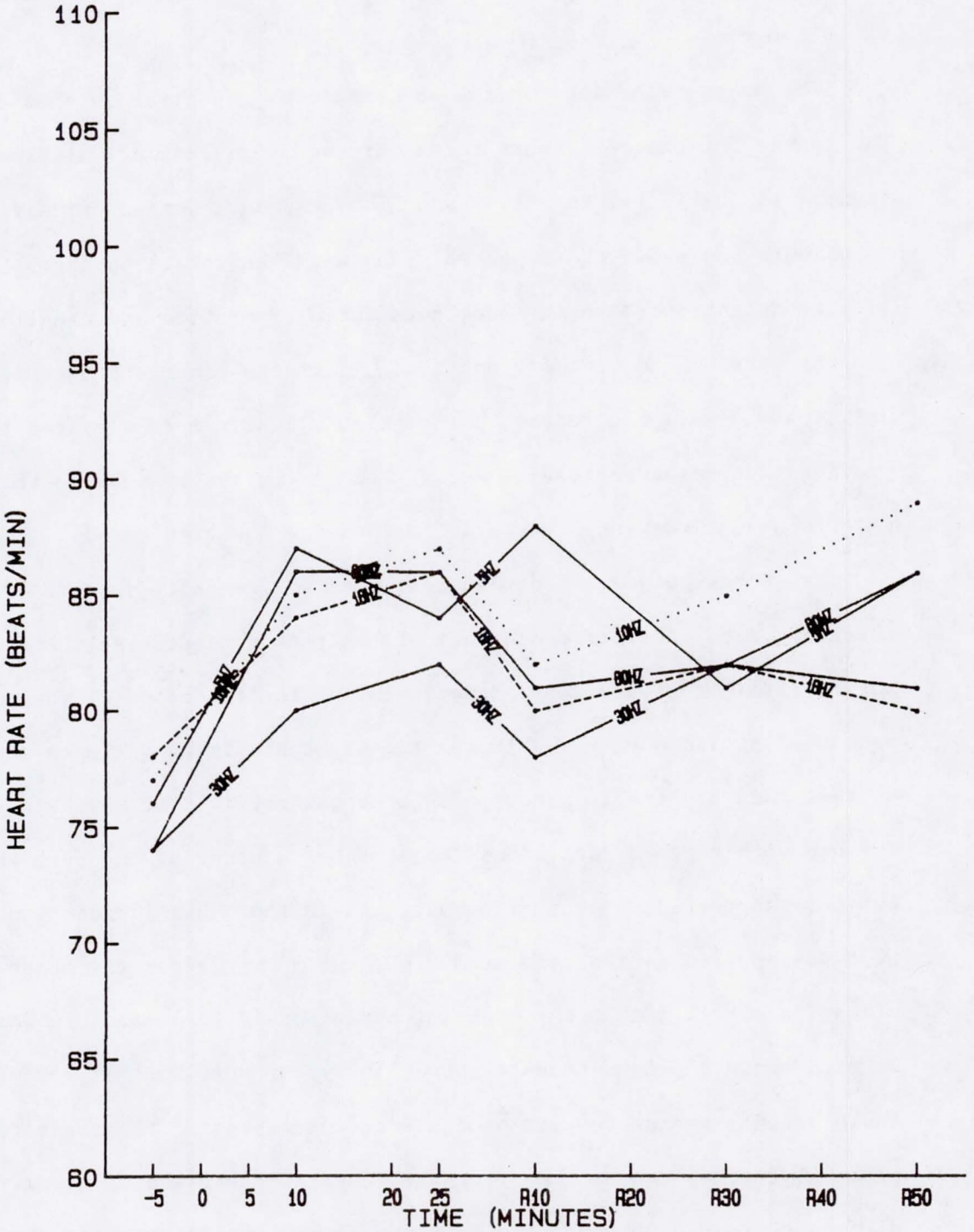

FIGURE 18. MEAN HEART RATES FOR 5, 10, 16, 30, AND 80 HZ HIGH INTENSITY WBV EXPOSURES.

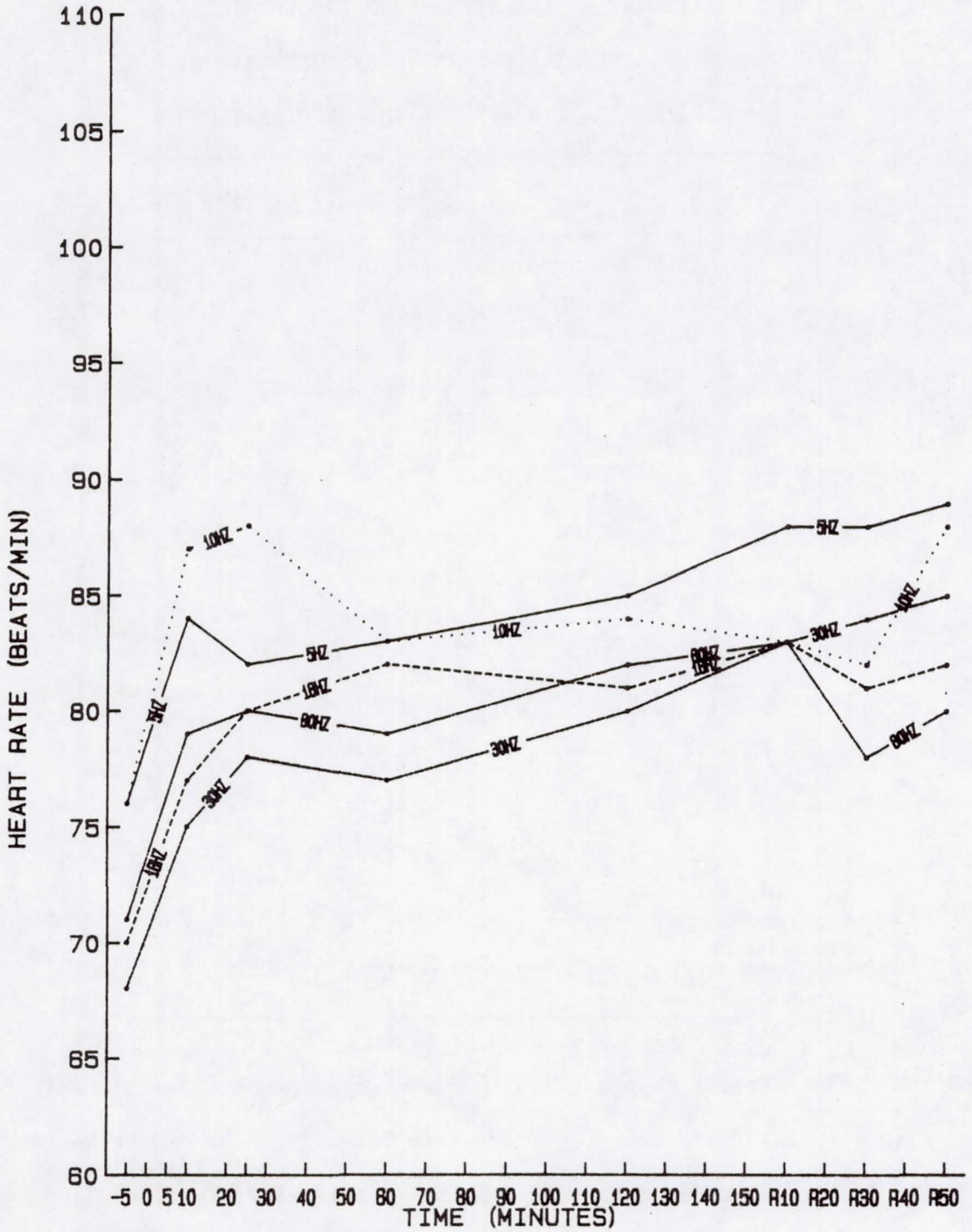

FIGURE 19. MEAN HEART RATES FOR 5, 10, 16, 30 AND 80 HZ LI WBV EXPOSURES.

TABLE 7.- RESULTS OF FRIEDMAN TESTS FOR SIGNIFICANT DIFFERENCES AMONG FREQUENCIES FOR RECTAL TEMPERATURE AND HEART RATE AT 30, 60, AND 90 min AFTER START OF VIBRATION

Variable	30 min	60 min	90 min
HIwbv			
Rectal temperature			
Xr^2	1.96	2.80	3.60
p-value	0.75	0.60	0.40
Heart rate			
Xr^2	3.80	3.16	3.57
p-value	0.40	0.60	0.40
LIwbv			
Rectal temperature			
Xr^2	2.60	3.20	1.90
p-value	0.72	0.52	0.75
Heart rate			
Xr^2	3.10	3.28	2.60
p-value	0.53	0.52	0.62

Since there appeared to be no frequency-dependent relationship for either rectal temperature or heart rate for either one of the wbv intensity levels, it was decided to pool all the rectal-temperature data and heart-rate data for the test frequencies at each intensity level of wbv in order to address the next hypothesis, namely, that the responses observed under wbv were different from the responses seen under control conditions, that

is, with heat only. In the previous section, it was shown there were no differences in the primary systemic responses between those exposed to sbv and control data obtained from the literature. It was therefore decided to use these sbv data as a control for the purposes of this section since the same individuals were involved in both sbv and wbv exposures, and statistical tests appropriate to matched pairs can be used and expected to provide some increase in power. For comparison of rates of increase between the wbv exposure and the control, the pooled rectal-temperature data and the pooled heart-rate data were corrected to the same baseline value as in the control. The correction factor was obtained by determining the difference in the mean previbration values for the control and the wbv exposure, and then subtracting that difference from each value of the wbv data. Figure 20 shows the corrected data versus time. Table 8 contains the same data at 10-min intervals. As can be seen, the pooled mean (± S.E.M.) rectal temperature for the wbv exposure increased faster than the rate of increase for the control, and appears different from the previbration level by the 10-min reading. By comparison, it took 20 min for the control to differ markedly from the initial reading. During the first 40 min, the rate of increase in rectal temperature was the same for both intensity levels of wbv (0.01°C/min), while the rate of increase for the control was considerably less (0.003°C/min). After 40 min, which was 15 min after the end of the HIwbv exposure, the rate of increase for the HIwbv decreased to the rate of the control, while the LIwbv mean rectal temperature continued to increase at the same rate. Although the rate of increase in rectal temperature was different after 40 min for the two wbv intensity levels, the actual values were not significantly different from

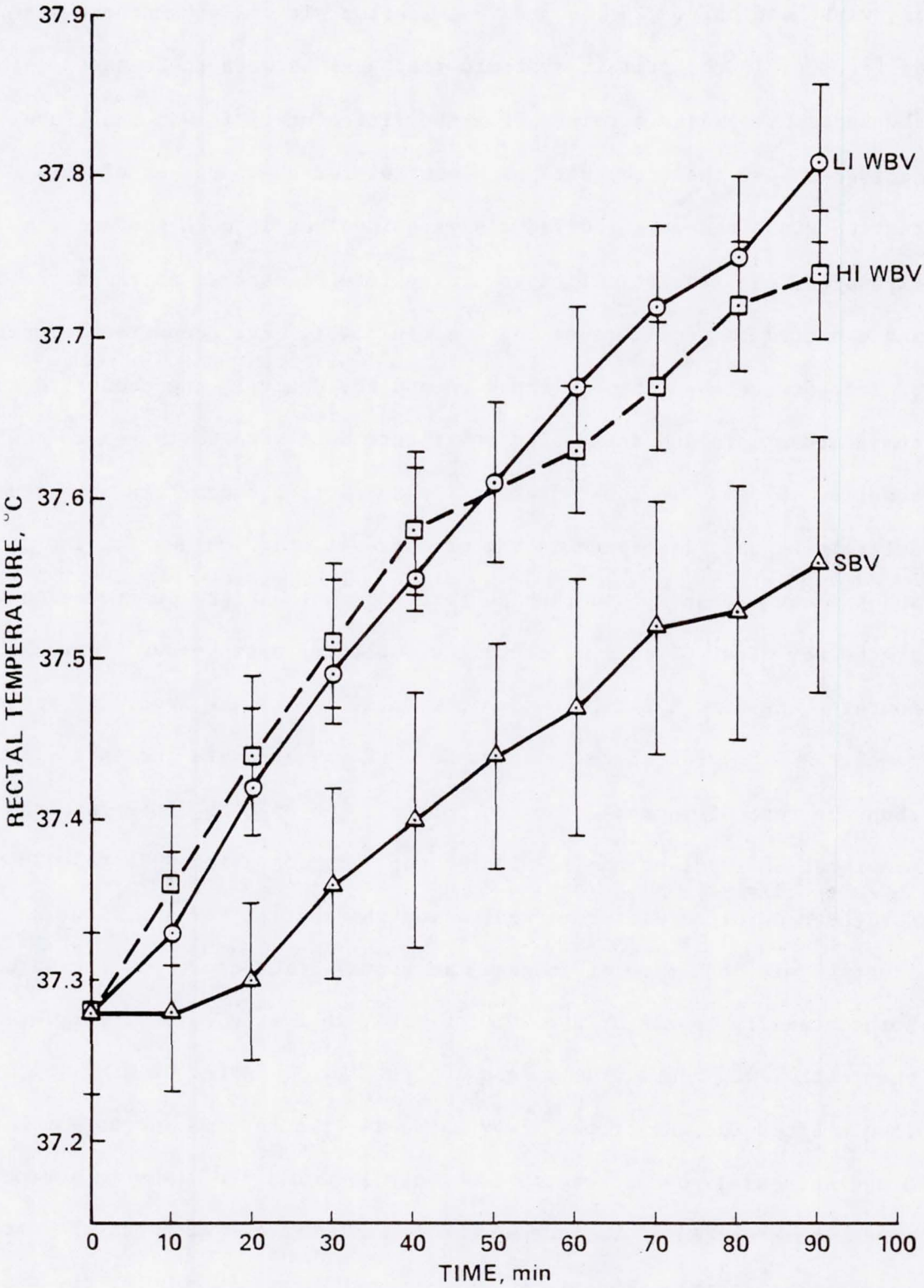

Figure 26. Mean corrected rectal temperatures for SBV, LI WBV, and HI WBV exposures.

TABLE 8.- THE MEAN (± S.E.M.) POOLED RECTAL TEMPERATURES FOR THE wbv EXPOSURES CORRECTED BACK TO THE CONTROL GROUP

	t_0	t_{10}	t_{20}	t_{30}	t_{40}	t_{50}	t_{60}	t_{70}	t_{80}	t_{90}
HIwbv										
Mean	37.28	37.36[a]	37.44[a]	37.51[a]	37.58[a]	--	37.63[a]	37.67[a]	37.72[a]	37.73[a]
S.E.M.	0.05	0.05	0.04	0.04	0.04	--	0.04	0.04	0.04	0.04
LIwbv										
Mean	37.28	37.33[a]	37.42[a]	37.49[a]	37.55[a]	37.61[a]	37.67[a]	37.72[a]	37.75[a]	37.81[a]
S.E.M.	0.04	0.05	0.05	0.05	0.05	0.05	0.05	0.06	0.06	0.04
Control										
Mean	37.28	37.28	37.30	37.36	37.40	37.44	37.47	37.52	37.53	37.56
S.E.M.	0.05	0.05	0.05	0.06	0.08	0.07	0.08	0.08	0.08	0.08

[a]Significantly ($p < 0.05$) different from the control by the paired t-test.

each other during the 90-min test period. The LIwbv and HIwbv mean rectal temperatures differ from the control, but not from each other.

Pooled mean heart rates — Since there appeared to be no frequency-dependent relationship for heart rates, the heart-rate data were also pooled for each wbv intensity level and plotted against time (fig. 21). The pooled mean (± S.E.M.) heart rate for each intensity level of wbv increased markedly during the first 10 min of wbv, as was expected. The pattern displayed in figure 21 for the HIwbv mean heart rate confirms the expected trend. The paired t-test was used to determine if significant changes ($p < 0.05$) from the initial reading occurred during the test exposure. In figure 21, the HIwbv mean heart rate was significantly elevated at 10 min, and then decreased by the 30-min reading, yet was still significantly elevated over the previbration heart rate. There were no significant differences in the mean heart rates for the HIwbv exposures for the 60 min between 30 and 90 min. The LIwbv heart increased slightly faster (NS) than the HIwbv heart rate during the first 10 min, and remained at about that level for the duration of the 90 min. After the 30-min reading, there was no significant difference in heart rate among the control, HIwbv, and LIwbv exposures. Table 9 lists the pooled mean heart rates for the HIwbv and LIwbv exposures corrected to the control baseline heart rate. After the 10-min and 30-min readings, all pooled mean heart rates were significantly elevated ($p < 0.05$) over the initial rating for the wbv and control exposures, respectively.

Summary of systemic responses during wbv — The most noticeable effect on the primary systemic responses between the wbv exposures and the control is with the rectal temperature. Even though all rectal temperatures were standardized to the same baseline temperature shortly

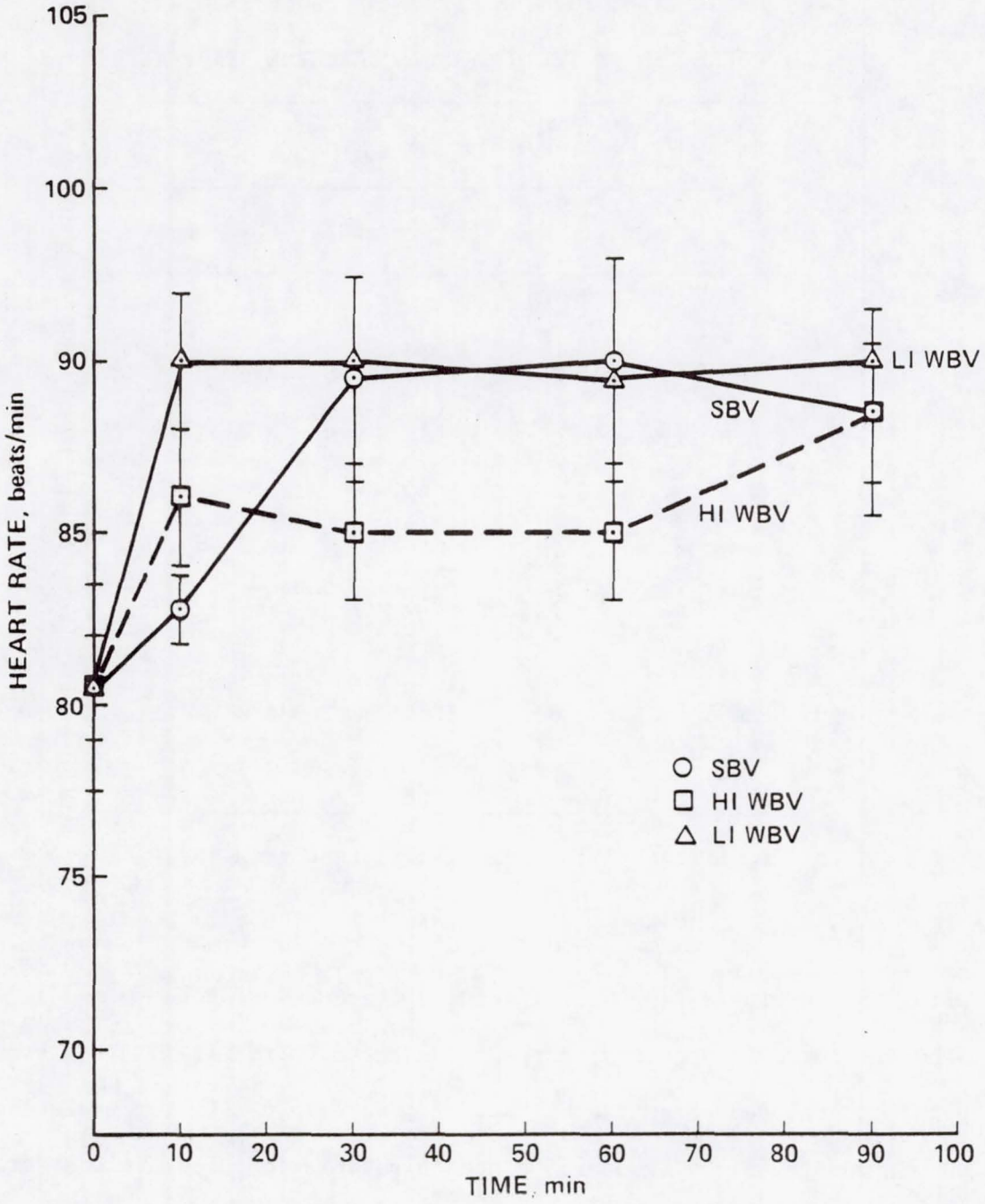

Figure 21. Mean heart rates for SBV, LI WBV, and HI WBV exposures.

TABLE 9.- HEART RATES FOR POOLED MEAN (± S.E.M.) HEART RATES FOR THE HIwbv AND LIwbv EXPOSURES CORRECTED TO THE CONTROL'S BASELINE HEART RATE

	Time, min					
	t_{-5}	t_0	t_{10}	t_{30}	t_{60}	t_{90}
Control						
Mean	80.9	80.9	83.4	88.6	89.6	86.5
S.E.M.	3.2	3.4	1.2	3.6	3.2	3.4
HIwbv						
Mean	78.3	80.9	87.3[a]	84.6	84.9	86.7
S.E.M.	2.0	2.2	1.8	2.3	2.0	1.8
LIwbv						
Mean	78.3	80.9	89.8[a]	90.2	88.8	90.1
S.E.M.	2.4	2.5	2.7	2.6	2.7	3.2

[a]Significantly ($p < 0.05$) different from the control.

after the 10-min reading, significant differences started to appear between the wbv and control exposures. Furthermore, it is noteworthy to point out the reduction in the rate of change in rectal temperature at 40 min for the HIwbv exposure, since this vibration exposure was for 25 min. After 40 min the slope of the mean HIwbv rectal temperature was reduced and became the same as the slope for the control. This change further strengthens the hypothesis that vibration exposure during a heat exposure results in a higher rate of increase in rectal temperature than in a heat exposure without vibration.

By comparison to the slopes for the rectal temperatures during vibration for the HIwbv and LIwbv, it appears the effects on the cooling mechanisms are very similar in that the rate of increase in rectal temperature was the same (slope = 0.01). As was mentioned above, after 40 min of HIwbv exposure, the rate of increase in rectal temperature decreased to that of the control (0.003), which leads one to suggest that the effect on the body's cooling mechanisms is not continued very long after vibration ends; however, there remains a marked residual effect. This lack of an observed dose-response relationship between the two intensity levels during the first 40 min could be accounted for in several ways. First, as is seen in figure 20, the mean rectal temperatures for the HIwbv were consistently, though not significantly, higher than the mean rectal temperatures for the LIwbv. Since changes in mean rectal temperature are reflective of many other changes within the body, possibly a longer exposure time would have demonstrated a difference between the two wbv intensity levels. On the other hand, there may be no intensity-dependent differences for the two wbv exposures tested. If this latter is true, then one could hypothesize that there is a rate-saturation effect of heat-conserving responses at low-intensity stimulation levels of vibration; that is, vibration exposure at the intensity levels tested produce a similar degree of localized responses. It could be that both of these test intensity levels are in the "flat" part of the response curves, and if we desired to see a dose-response relationship due to intensity levels, a much lower level than was used in this study would be necessary.

As was mentioned earlier, heart rates vary considerably and therefore tend to be less reliable as a heat-stress indicator than is rectal

temperature. The observed trend in the heart-rate pattern for the wbv exposures was fairly consistent with the expected trend for a wbv exposure. At 10 min, the heart rates for the HIwbv and LIwbv are not significantly different [$p(t) < 0.05$], and the rates of increase for the two intensity levels during the first 10 min are fairly close with respect to the control's rate of increase. The significant difference in the heart rates for the HIwbv and LIwbv after 30 min is probably due to the termination of the vibration at 25 min for the HIwbv. After 30 min, the heart rates for all conditions tended to stabilize, and were not different for the three test conditions at 90 min after the start of the vibration exposure.

Localized responses during wbv exposures — As was done in the case of segmental vibration, the localized responses will be reviewed to determine if they are generally consistent with the picture presented by principal systemic responses, namely, elevated rectal temperatures and heart rates.

Limb-segment blood-perfusion rates — During exposure to elevated ambient temperatures in which there is an increase in rectal temperature, the local blood-perfusion rate is expected to increase. If wbv exposure induces a general vasoconstrictive effect, then one would expect to see a reduction in blood-perfusion rates, whereas in a combined heat and wbv exposure, the blood-perfusion rates should probably increase but be less than the levels seen in a heat exposure without vibration. From a published literature review of seven articles which were in the temperature range for my data (ref. 40), the forearm (FA) blood-perfusion rate ranged from about 3 to 6 ml of blood per 100 ml of tissue per minute, which is also the range for FA perfusion rates in this study, as shown in appendix XII. There were no similar published data for the upperarm.

Due to the wide variability in the few subjects used in this study, the Wilcoxon's signed rank test was used to compare the central tendency of the paired samples (ref. 48). The Wilcoxon statistic may be regarded as a test intermediate between the signed test and the direct treatment of the observation through the paired t-test. The sign test does not utilize the size of the measurements, while the Wilcoxon test takes account of size through their rank order. The hypothesis tested with the Wilcoxon statistic was that vibration reduces the cooling mechanisms through a reduction in blood-perfusion rate. As was mentioned in the sbv section, blood-perfusion rates were not determined during the vibration exposures, but were measured just prior to and immediately after the vibration exposure. Due to the small number of subjects a 90% significance level was chosen.

There appeared to be a frequency-dependent relationship in localized blood-perfusion rates, particularly for the forearm (FA). As is seen in table 10, the FA blood-perfusion rates increased significantly at 30 and 80 Hz, and at 5, 30, and 80 Hz for the HIwbv and LIwbv exposures, respectively. Figures 22 and 23 show these data for the frequencies where significant differences were seen for the FA.. As seen in figure 22 for the HIwbv, the blood-perfusion rates decreased at 10 and 60 Hz. For the LIwbv, the blood-perfusion rate decreased at 10 Hz only (fig. 23).

The upperarm (UA) blood perfusion rate increased (table 10) at 10, 16, and 30 Hz, and decreased at 5 Hz for the HIwbv (fig. 24). For the LIwbv the UA blood-perfusion rate increased at all test frequencies except at 10 Hz, where it did not change significantly (fig. 25).

As is pointed out above, there appears to be a frequency-dependent relationship for the FA blood-perfusion rates, with an increase in

TABLE 10.- MEAN (± S.E.M.) BLOOD-PERFUSION RATES FOR THE UPPERARM (UA) AND FOREARM (FA) FOR PREVIBRATION, AFTER VIBRATION EXPOSURE, AND AFTER 50-min RECOVERY PERIOD

	UA			FA		
	Pre-vibration	End vibration	End recovery	Pre-vibration	End vibration	End recovery
HIwbv						
5 Hz	4.1 ± 0.5	4.0 ± 0.4	4.9 ± 0.8	4.0 ± 0.9	4.6 ± 0.5	4.4 ± 0.3
10 Hz	3.4 ± 0.2	4.4 ± 1.0	4.7 ± 0.4	4.6 ± 0.8	4.4 ± 0.7	5.1 ± 0.8
16 Hz	3.4 ± 0.8	3.6 ± 1.0	4.8 ± 0.7	4.2 ± 0.8	3.9 ± 1.1	6.1 ± 1.1
30 Hz	3.6 ± 0.8	4.6 ± 0.4	4.7 ± 0.8	3.2 ± 0.2	4.4 ± 0.4	4.0 ± 0.6
80 Hz	4.8 ± 1.0	4.9 ± 0.9	5.5 ± 0.7	3.7 ± 0.6	4.7 ± 0.9	5.5 ± 0.9
LIwbv						
5 Hz	5.1 ± 1.2	6.0 ± 0.6	6.2 ± 0.6	3.9 ± 0.6	5.7 ± 1.0	6.0 ± 0.2
10 Hz	4.9 ± 0.6	5.0 ± 0.6	5.8 ± 0.4	5.0 ± 0.6	4.2 ± 0.3	6.0 ± 0.3
16 Hz	4.2 ± 0.4	4.6 ± 0.6	5.2 ± 1.0	4.2 ± 0.8	4.7 ± 0.5	5.2 ± 0.6
30 Hz	4.4 ± 0.4	4.7 ± 0.3	4.8 ± 0.1	4.1 ± 0.3	5.6 ± 0.5	5.6 ± 0.6
80 Hz	4.3 ± 0.6	4.8 ± 0.6	4.5 ± 0.5	3.8 ± 0.5	5.1 ± 0.6	5.3 ± 0.7
sbv						
10 Hz	No data	No data	No data	6.7 ± 0.6	4.2 ± 0.5	5.8 ± 1.1
25 Hz	No data	No data	No data	5.6 ± 1.1	5.9 ± 1.1	4.9 ± 0.6
60 Hz	No data	No data	No data	5.0 ± 1.2	5.4 ± 0.7	5.1 ± 1.0
125 Hz	No data	No data	No data	5.8 ± 0.7	4.5 ± 0.7	5.4 ± 0.5
250 Hz	No data	No data	No data	5.5 ± 0.5	4.4 ± 0.4	5.3 ± 0.8

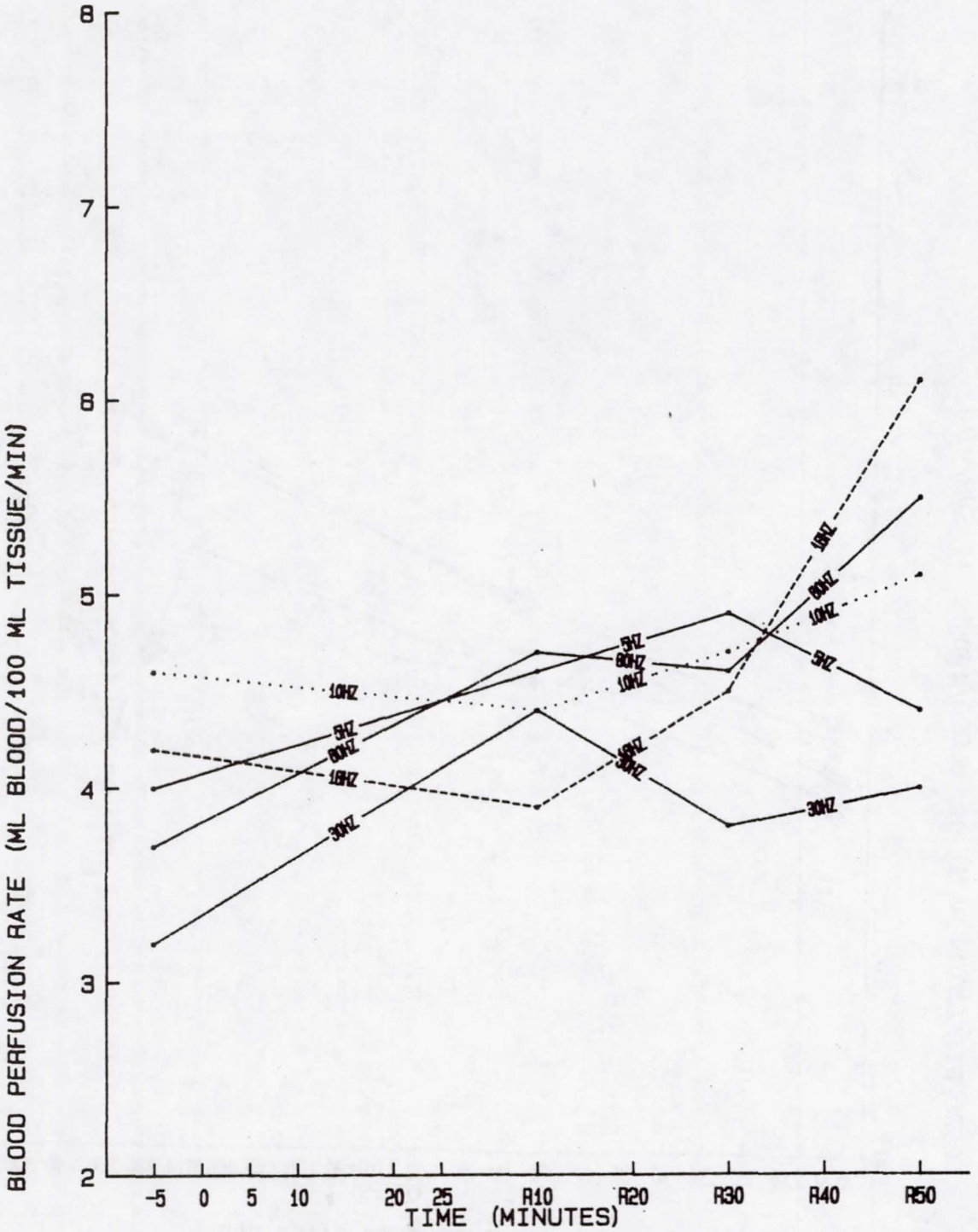

FIGURE 22. MEAN FA BLOOD PERFUSION RATES FOR 5, 10, 16, 30 AND 80 HZ HI WBV EXPOSURES.

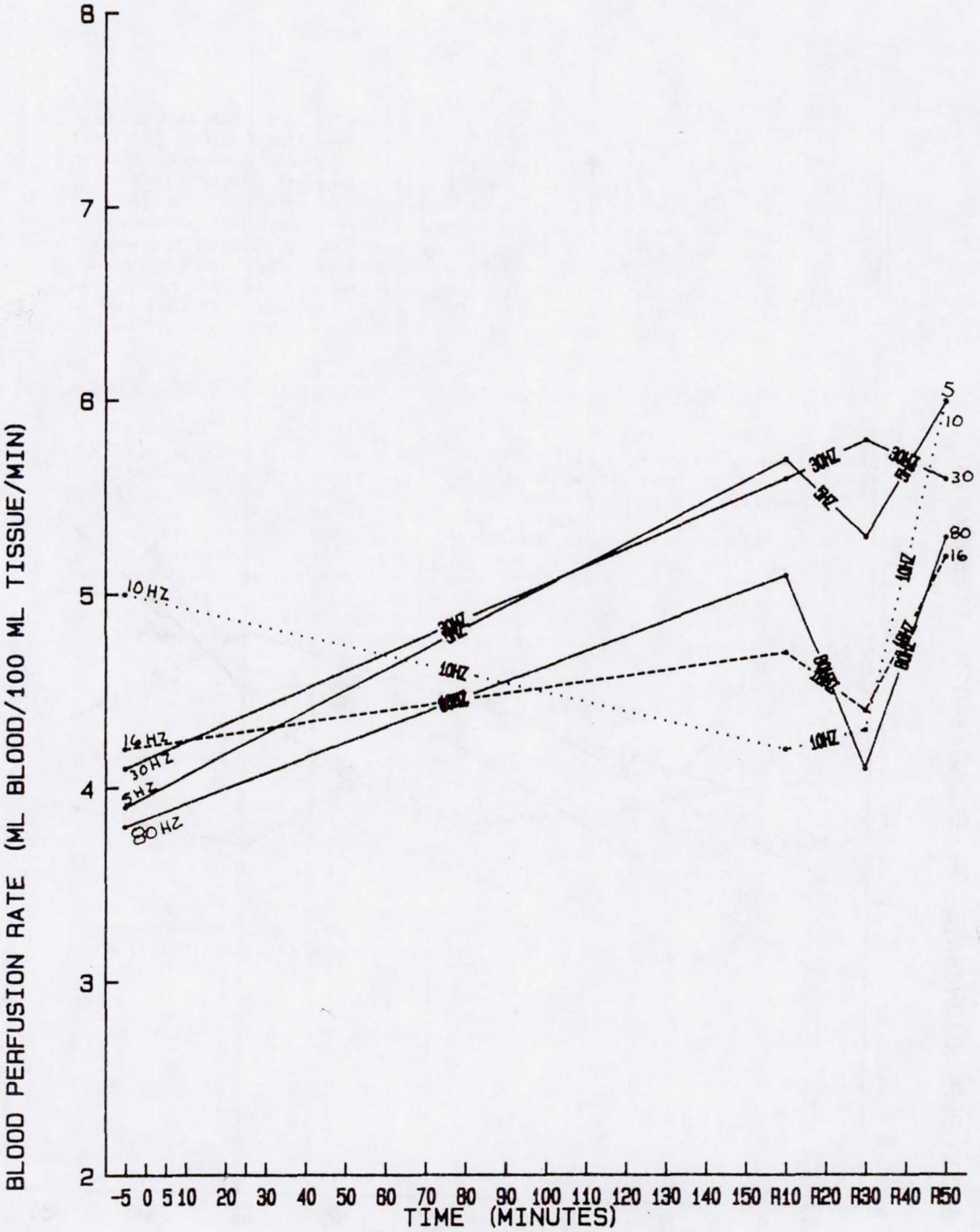

FIGURE 23. MEAN FA BLOOD PERFUSION RATES FOR 5, 10, 16, 30 AND 80 HZ LI WBV EXPOSURES.

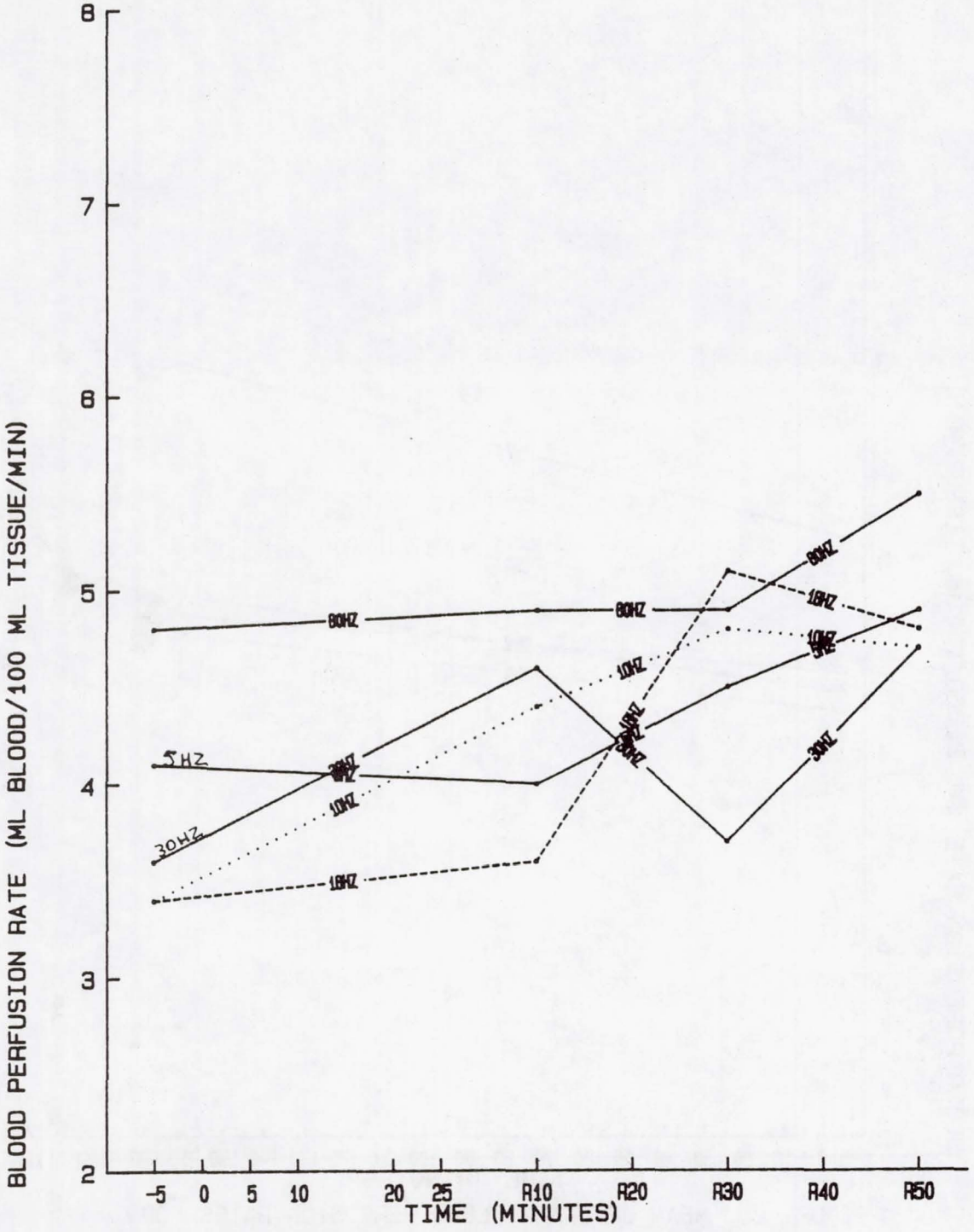

FIGURE 24. MEAN UPPERARM BLOOD PERFUSION RATES FOR 5, 10, 16, 30 AND 80 HZ HIGH INTENSITY WBV EXPOSURES.

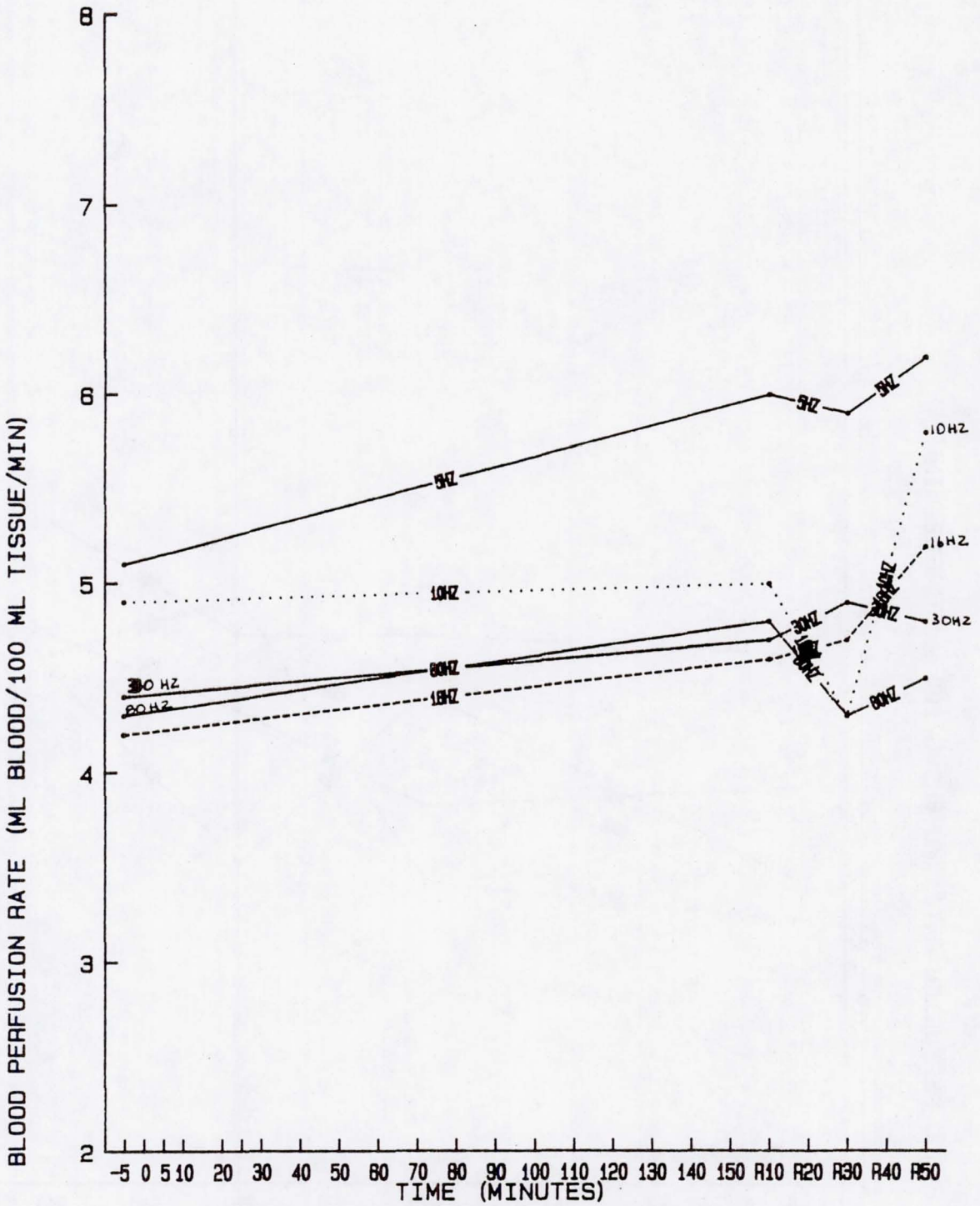

FIGURE 25. MEAN UPPERARM BLOOD PERFUSION RATES FOR 5, 10, 16, 30 AND 80 HZ LOW INTENSITY WBV EXPOSURES.

blood-perfusion rates more likely to occur at higher frequencies. Thus, the lower frequencies (10 and 16 Hz) are associated with reduced FA blood-perfusion rates. There does not appear to be as strong a frequency-dependent relationship for the UA as there is for the FA.

The next question is whether there is an intensity-level-dependent relationship with blood-perfusion rates. To obtain an overview of the effect of intensity level, the blood-perfusion rates were pooled for each body segment at each intensity level for all frequencies. The pooled mean FA blood-perfusion rate increased from 3.9 ± 0.2 to 4.4 ± 0.1 ml of blood per 100 ml of tissue per minute for the LIwbv exposure. The greater difference between the previbration and postvibration exposure blood-perfusion rate for the LIwbv (0.9) versus the HIwbv perfusion rate (0.5) may be due to a vibration-intensity-level difference, or more likely, is a result of the larger difference in rectal temperatures for the LIwbv exposure. The time between measurements and the difference in rectal temperatures are markedly different for the two intensity levels. The time between measurements for the HIwbv and the LIwbv is about 30 versus 155 min, respectively. The difference in the mean rectal temperature between the measurements for the HIwbv and LIwbv is 0.3 and 0.7°C, respectively. With a greater increase in rectal temperature one would expect to see a larger increase in the blood-perfusion rate, as was seen in the LIwbv exposure. If the difference in rectal temperature is compared for the same time periods, a comparison between the rate of change in blood-perfusion rate with respect to rectal temperature can be determined for each intensity level. For the HIwbv and LIwbv exposures, the rate of change in blood-perfusion rate with respect to rectal temperature is 1.7 ml of blood/100 ml of tissue per minute/°C,

respectively. Thus, when the blood-perfusion rate is corrected for changes in rectal temperature and in view of the variability in the data there appeared to be no marked intensity-dependent relationship between the two wbv exposures. Although there are no known published data on the rate of change in UA blood-perfusion data with respect to various rectal temperatures, the range for changes in blood-perfusion rates for the forearm can be up to 300%.

Localized sweat rates — Localized sweat rates were determined on the UA, FA, and calf (C). Since the sweat rate appears to be sinusoidal with a periodicity of about 20 to 25 min, the sweat rates were evaluated by comparison of slopes from linear regression lines and by comparing the mean of three values over a 20-min cycle — at 0, 10, and 20 min and at 130, 140, and 150 min for the LIwbv exposure. Since the HIwbv exposure is about that of one sweat cycle, linear regression slopes and graphic presentation were used instead of the mean of three values.

If vibration affects localized sweat rates, then one would expect either a decrease or no significant change in the overall trend in the sweat rate over the vibration exposure test period.

The UA sweat rates increased at 16, 30, and 80 Hz, and decreased at 5 and 10 Hz during the HIwbv exposure (fig. 26). For the LIwbv, the UA sweat rate increased slightly at all frequencies (fig. 27 and table 11). Also for the LIwbv exposure data, table 11 lists the means (± S.E.M.) for the first and last 20-min sweat cycle for the vibration period. The increase in the mean sweat rate between the last 20-min cycle and the first 20-min cycle was significantly higher [$p(t) < 0.05$] (paired t-test) for each frequency for the UA. The results of the linear regression analyses for the LIwbv sweat rates are in table 12. The last 20-min

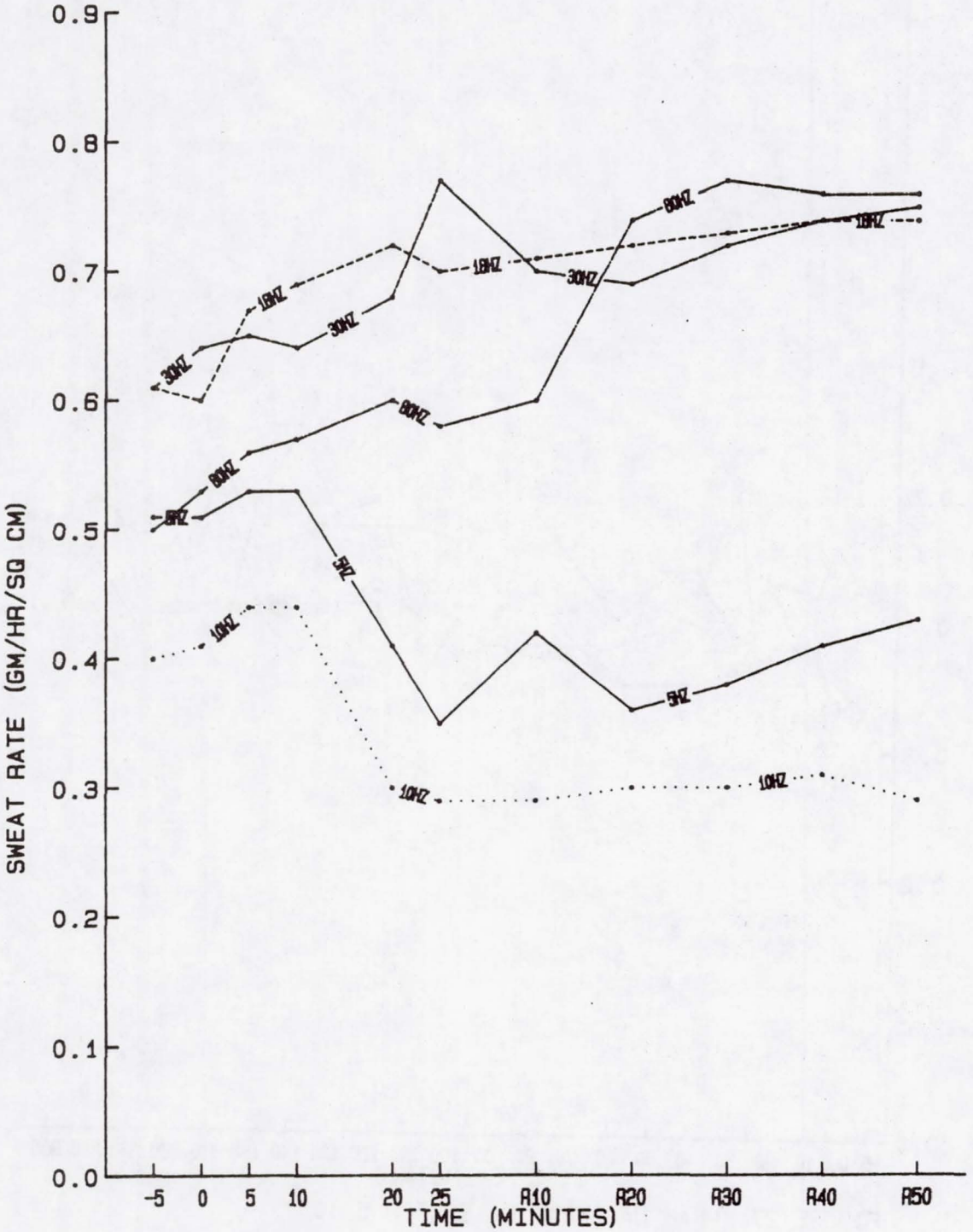

FIGURE 26. MEAN UA SWEAT RATES FOR 5, 10, 16, 30 AND 80 HZ HI WBV EXPOSURES.

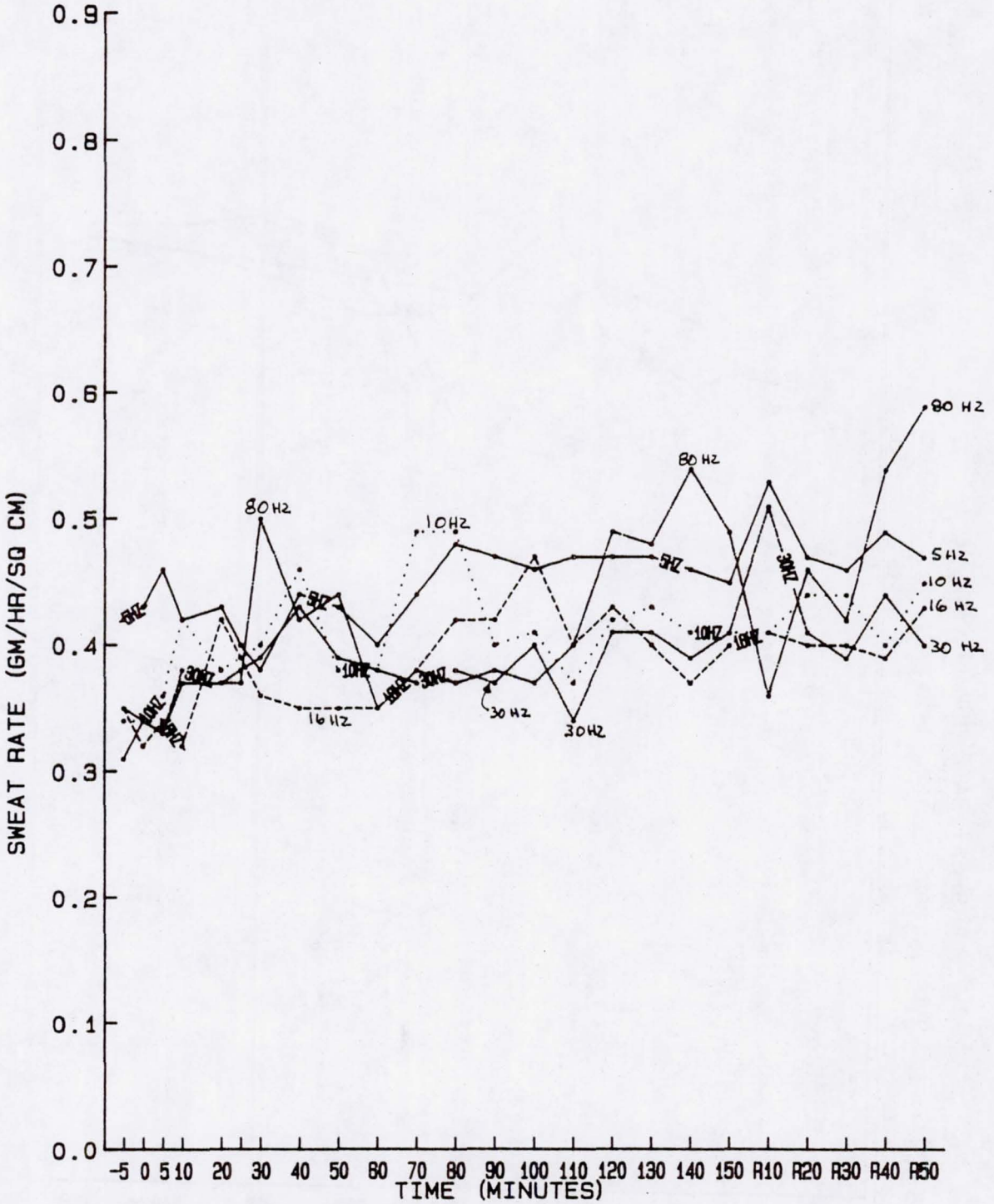

FIGURE 27. MEAN UA SWEAT RATES FOR 5, 10, 16, 30 AND 80 HZ LI WBV EXPOSURES.

TABLE 11.- MEAN SWEAT RATES ($g/hr/cm^2$) FOR THE FA AND UA DURING THE LIwbv EXPOSURE FOR THE FIRST AND LAST 20-min PERIOD

Condition	Mean sweat rate (± S.E.M.)		Difference
	First 20-min cycle	Last 20-min cycle	
FA			
5 Hz	0.52 ± 0.02	0.50 ± 0.01	-0.02
10 Hz	0.58 ± 0.02	0.56 ± 0.01	-0.02
16 Hz	0.54 ± 0.05	0.55 ± 0.02	0.01
30 Hz	0.57 ± 0.01	0.65 ± 0.01	0.08
80 Hz	0.58 ± 0.02	0.86 ± 0.05	0.16
UA			
5 Hz	0.43 ± 0.01	0.46 ± 0.01	0.03
10 Hz	0.38 ± 0.02	0.42 ± 0.01	0.04
16 Hz	0.35 ± 0.03	0.39 ± 0.01	0.04
30 Hz	0.36 ± 0.01	0.40 ± 0.01	0.04
80 Hz	0.36 ± 0.01	0.50 ± 0.01	0.14

cycle and the first 20-min cycle were significantly higher [$p(t) < 0.05$] for each frequency for the UA.

The FA HIwbv sweat rate did not change as dramatically as did the UA HIwbv sweat rate for the low frequencies (fig. 28). Since the sweat cycle was about the same duration as the vibration exposure for the high intensity, analyses of particular data points or changes within the cycle are difficult if not impossible. By comparison to the LIwbv, the UA HIwbv

TABLE 12.- RESULTS OF LINEAR REGRESSION ANALYSES FOR THE LIwbv AND RECOVERY PERIOD UA AND FA SWEAT RATES

Test condition	LIwbv		Recovery period	
	Slope	R^2	Slope	R^2
FA				
5 Hz	1.1E-4	0.01	-8.6E-5	0.001
10 Hz	-2.6E-4	0.06	2.9E-4	0.71
16 Hz	2.2E-4	0.05	1.6E-4	0.38
30 Hz	3.9E-4	0.20	-4.0E-4	0.03
80 Hz	1.6E-3	0.50	1.2E-3	0.05
UA				
5 Hz	3.9E-4	0.45	-8.6E-5	0.003
10 Hz	1.8E-4	0.04	4.9E-4	0.19
16 Hz	5.1E-4	0.33	2.6E-4	0.12
30 Hz	1.8E-4	0.12	-8.0E-4	0.12
80 Hz	7.6E-4	0.33	2.9E-3	0.42

sweat rates did not have the large amplitude of the lower-intensity vibration exposure. A frequency-dependent relationship can be seen in table 12 for the LIwbv FA (fig. 29). Thus there appears to be a frequency-dependent relationship with wbv and localized sweat rates, with the lower frequencies (5, 10, and 16 Hz) more likely to hinder an increase in sweat rate, even though the rectal temperature is increasing. Additional support to the hypothesis that vibration is inhibiting the

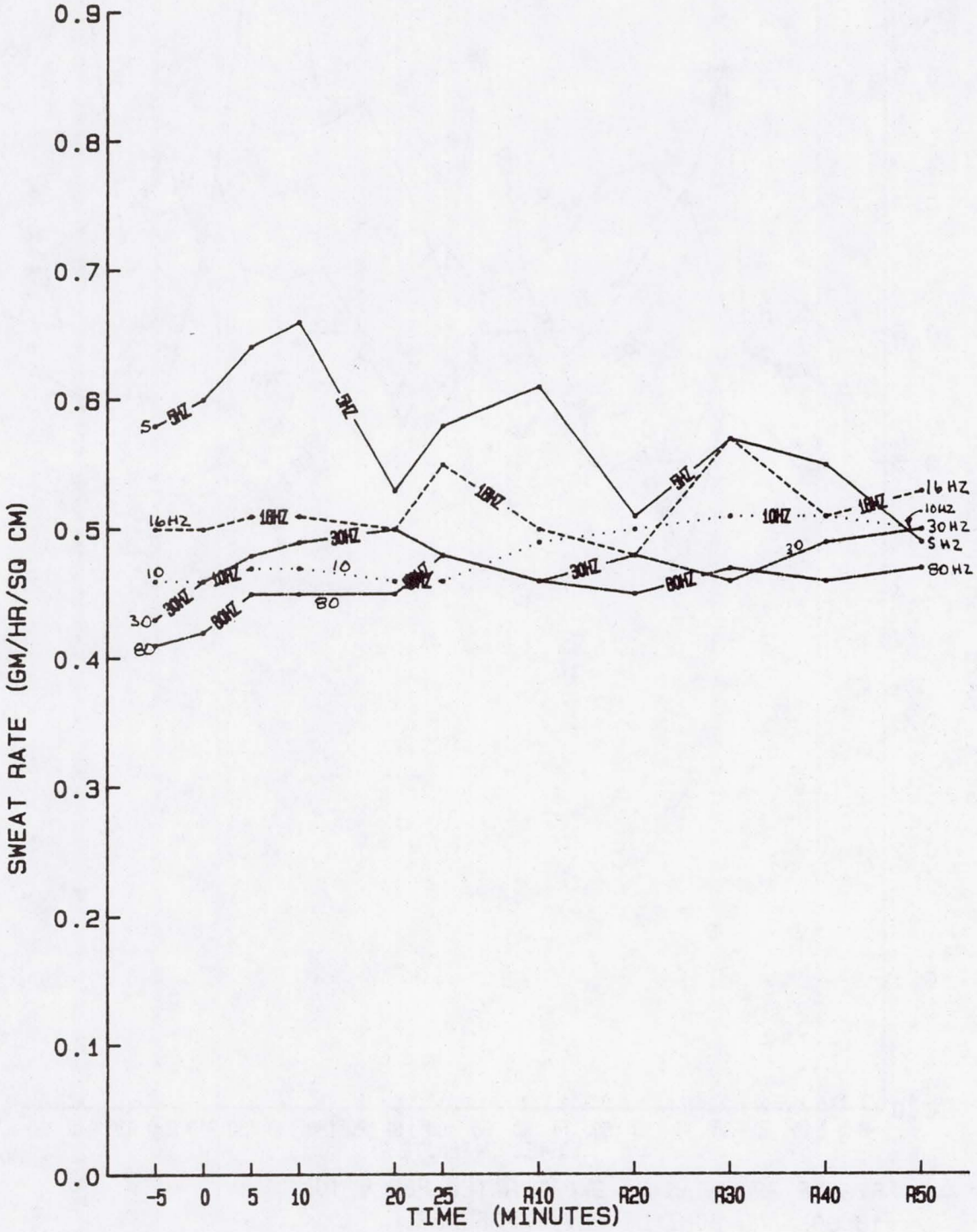

FIGURE 28. MEAN FA SWEAT RATES FOR 5, 10, 16, 30 AND 80 HZ HI WBV EXPOSURES.

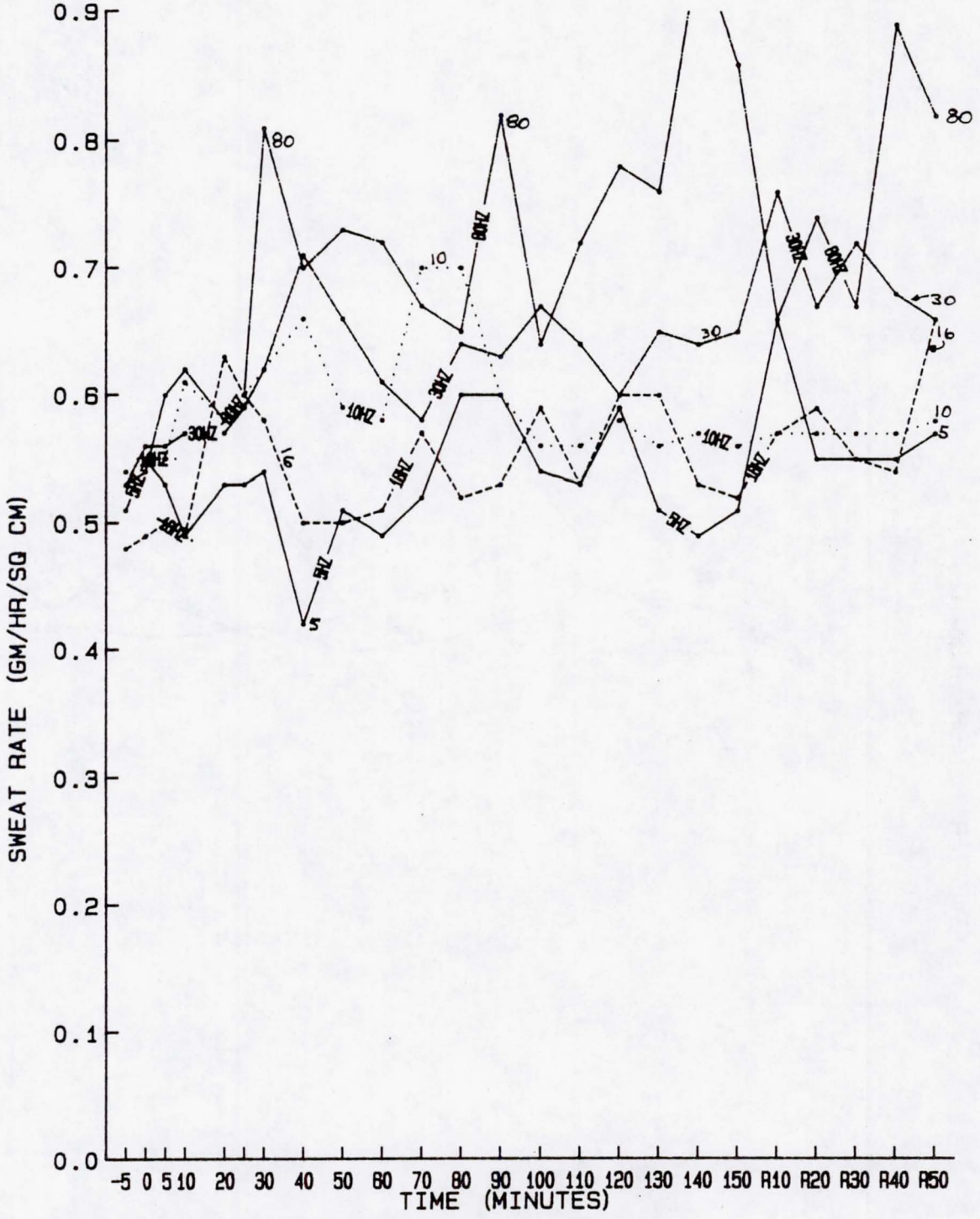

FIGURE 29. MEAN FA SWEAT RATES FOR 5, 10, 16, 30, AND 80HZ LI WBV EXPOSURES.

sweat rate at low frequencies can be seen in table 13. When the sweat rate (grams per hour per square centimeter) is converted to a water mass unit (grams) by multiplying the sweat rate for 1 hr by the surface area of that limb segment according to the Dubois formula (ref. 35), the mass of water available for evaporation can be compared between the previbration and end of vibration periods. As is seen in table 13 there was a decrease in the quantity of sweat available for evaporation for the forearm and upperarm at 5 Hz. The low frequencies, 5 and 10 Hz, appear to inhibit the sweat rate, while the higher frequencies (16, 30, and 80 Hz) do not appear to inhibit the sweat rate.

TABLE 13.- CHANGES IN THE MASS (grams) OF SWEAT AVAILABLE FOR EVAPORATION DURING wbv

Test condition	Frequency, Hz				
	5	10	16	30	80
HIwbv					
UA	-176	-132	110	143	55
FA	-47	0	117	47	140
LIwbv					
UA	-55	66	44	55	66
FA	-23	164	211	140	187

In order to investigate the hypothesis that there is an intensity-dependent relationship, the sweat-rate data for each intensity level was pooled. The mean UA sweat rate for the HIwbv did not change

(0.54 ± 0.04 g/hr-cm^2) during the first 30 min of exposure, while the LIwbv increased from 0.35 ± 0.02 to 0.39 ± 0.01 g/hr-cm^2 for the same time period. As can be seen in figures 26 and 27, the amplitude and periodicity of the sweat cycle appear to be more reduced in the high-intensity wbv exposure.

The FA pooled sweat rate for the first 30 min of HIwbv went from 0.48 ± 0.03 to 0.51 ± 0.02 g/hr-cm^2, while the LIwbv increased from 0.54 ± 0.01 to 0.58 ± 0.01 g/hr-cm^2, that is, a nonsignificant increase for the HIwbv and a significant, $p < 0.05$, increase for the LIwbv. Although the amplitude of the sweat cycle also appears qualitatively to be greatly reduced at 10 and 16 Hz for the HIwbv, the amplitude of the sweat cycles for the LIwbv exposures appears greater than those observed in the graphs of the HIwbv exposure.

Thus, for sweat rates, there appear to be frequency- and intensity-dependent relationships. The vibration appears to reduce the sweat rate the greatest at high-intensity, low-frequency (whole-body resonating) frequencies, and to have the least effect at the low-intensity, high-frequency exposures, which is also consistent with the blood-perfusion data.

Localized skin temperatures — As was mentioned in the section on segmental vibration, the localized skin temperature is usually reflective of ambient environmental conditions. If the environmental conditions are kept fairly constant, as was done in this study, then the skin temperature can be indicative of changes in sweat rates or local blood-perfusion rates.

The upperarm (UA) skin temperature did not change significantly at any frequency for the HIwbv after 25 min of exposure (table 14). For the

TABLE 14
MEAN (± SEM) SKIN TEMPERATURES FOR UPPERARM (UA), FOREARM (FA), AND LEFT CHEST (LC) FOR PREVIBRATION, AT 25 MIN VIBRATION EXPOSURE, AND AFTER 50 MIN RECOVERY PERIOD

	UA			FA			LC		
	PRE VIBRATION	25 MIN VIBRATION	END RECOVERY	PRE VIBRATION	25 MIN VIBRATION	END RECOVERY	PRE VIBRATION	25 MIN VIBRATION	END RECOVERY
Hlwbv									
5 Hz	36.7 ± 0.2	37.0 ± 0.3	37.1 ± 0.3	36.3 ± 0.2	36.6 ± 0.4	36.5 ± 0.3	35.7 ± 0.4	35.8 ± 0.5	35.5 ± 0.5
10 Hz	36.7 ± 0.2	36.6 ± 0.3	36.6 ± 0.2	36.4 ± 0.3	36.2 ± 0.4	36.2 ± 0.1	35.8 ± 0.2	35.3 ± 0.3	35.8 ± 0.4
16 Hz	36.7 ± 0.2	36.6 ± 0.2	36.6 ± 0.2	36.1 ± 0.2	36.2 ± 0.1	36.1 ± 0.1	35.8 ± 0.5	35.4 ± 0.6	36.0 ± 0.4
30 Hz	36.8 ± 0.2	36.8 ± 0.2	36.9 ± 0.2	36.7 ± 0.3	36.5 ± 0.2	36.3 ± 0.2	36.3 ± 0.3	36.0 ± 0.4	36.1 ± 0.4
80 Hz	37.1 ± 0.2	36.9 ± 0.1	36.9 ± 0.2	36.8 ± 0.2	36.7 ± 0.2	36.5 ± 0.2	36.2 ± 0.3	35.9 ± 0.4	36.3 ± 0.5
Llwbv									
5 Hz	36.9 ± 0.2	37.1 ± 0.2	36.8 ± 0.2	36.7 ± 0.2	36.6 ± 0.2	36.4 ± 0.3	36.0 ± 0.3	36.1 ± 0.3	36.1 ± 0.4
10 Hz	36.6 ± 0.1	36.8 ± 0.2	36.6 ± 0.3	36.4 ± 0.2	36.6 ± 0.2	36.3 ± 0.3	35.9 ± 0.4	36.1 ± 0.3	36.3 ± 0.3
16 Hz	36.9 ± 0.1	37.2 ± 0.2	37.0 ± 0.2	36.5 ± 0.3	36.8 ± 0.3	36.5 ± 0.3	36.0 ± 0.4	36.2 ± 0.3	35.9 ± 0.2
30 Hz	37.0 ± 0.2	37.1 ± 0.2	37.1 ± 0.2	36.7 ± 0.1	36.7 ± 0.2	36.6 ± 0.2	36.4 ± 0.3	36.5 ± 0.4	36.4 ± 0.4
80 Hz	36.9 ± 0.1	36.9 ± 0.1	36.9 ± 0.3	36.6 ± 0.2	36.6 ± 0.2	36.5 ± 0.2	36.3 ± 0.4	36.1 ± 0.2	36.7 ± 0.3
SBV									
10 Hz	36.0 ± 0.3	37.0 ± 0.3	36.5 ± 0.4	35.4 ± 0.5	36.0 ± 0.3	35.7 ± 0.2	35.7 ± 0.2	35.9 ± 0.3	35.9 ± 0.4
25 Hz	36.8 ± 0.4	37.6 ± 0.4	37.3 ± 0.3	35.8 ± 0.5	36.2 ± 0.2	36.3 ± 0.2	36.2 ± 0.2	36.1 ± 0.3	36.6 ± 0.1
60 Hz	36.7 ± 0.3	37.5 ± 0.2	37.0 ± 0.3	36.8 ± 0.4	36.4 ± 0.5	36.9 ± 0.3	36.2 ± 0.1	36.0 ± 0.3	35.9 ± 0.2
125 Hz	36.6 ± 0.4	37.4 ± 0.1	37.0 ± 0.2	36.6 ± 0.3	36.5 ± 0.5	36.8 ± 0.2	36.0 ± 0.2	36.0 ± 0.4	35.9 ± 0.2
250 Hz	37.0 ± 0.2	37.4 ± 0.2	37.5 ± 0.4	36.8 ± 0.3	36.6 ± 0.2	36.7 ± 0.2	36.2 ± 0.3	35.9 ± 0.3	36.2 ± 0.4

LIwbv, there were significant increases in UA skin temperatures at 10 and 30 Hz (table 14).

The forearm (FA) skin temperatures followed the same pattern as the UA for both the HIwbv and LIwbv exposures, except there was no significant increase at 30 Hz for the LIwbv. The skin temperature for the left chest (LC) did not increase during either intensity level of the wbv exposures, and actually decreased significantly at 10, 16, 30, and 80 Hz for the HIwbv.

In addition to the localized skin temperatures, a mean skin temperature was calculated using the formula which was included in the methods section. This mean skin temperature provides a weighted (according to body surface area) average for the entire body. See the lower group of lines in figures 16 and 17 for the mean skin temperature for each frequency. The data are also tabulated for each 5-min interval in appendix X. The pattern of change in mean skin temperature with respect to frequency is identical for the two intensity levels of wbv, although the amplitude of the change is different. For both intensity levels, the mean skin temperature increased only at 5 Hz, and decreased at 10, 16, 30, and 80 Hz. The decrease in mean skin temperature was most noticeable at 30 and 80 Hz for both intensity levels, with a greater reduction in the mean skin temperatures seen in the HIwbv than in the LIwbv. The mean skin temperatures for each frequency is tabulated below:

Time, min	V-5	V25	Difference
HIwbv			
5 Hz	35.7	36.3	+0.6
10 Hz	35.6	35.4	-0.2
16 Hz	35.4	35.3	-0.1
30 Hz	36.0	35.7	-0.3
80 Hz	35.9	35.6	-0.3
Mean	35.7	35.5	-0.2
S.E.M.	0.01	0.2	
LIwbv			
5 Hz	35.7	35.9	+0.2
10 Hz	35.9	35.8	-0.1
16 Hz	36.0	35.9	-0.1
30 Hz	36.2	36.0	-0.2
80 Hz	36.0	35.8	-0.2
Mean	36.0	35.9	-0.1
S.E.M.	0.1	0.1	

Thus, the localized skin temperatures and mean skin temperatures appear to be frequency- and intensity-dependent, although not so clearly demonstrated as in the blood-perfusion or sweat-rate section. Again, it is very difficult to get very much good information from the skin temperatures since it responds so quickly to changes in the heat booth temperature, air currents, blood-perfusion rates, and the phase and intensity of the sweat cycle. It is consistent with the patterns observed in the sweat rates in that low frequencies appear to affect the cooling mechanisms.

Respiratory variables — Although the respiratory variables — rates, corrected volume of oxygen consumed, respiratory exchange ratio (RER) — are actually more of a systemic response than a local response, they were included in the localized response section because these variables are secondary responses to heat exposures, and are reflective of increases in the rectal temperatures.

For the HIwbv exposure, the respiratory variables were tested prior to vibration, at 5 and 20 min into the vibration exposure, and at 20 min into the recovery period. For the LIwbv exposure, the respiratory variables were measured at 5 min prior to vibration, at 5, 70, and 140 min into the vibration exposure, and at 20 min into the recovery period. The respiration-rate data are tabulated in appendix XIII. The oxygen uptake and RER data are tabulated in appendixes XIV and XV, respectively. As was mentioned previously, the respiratory rate and heart rate usually increase for the first 15-20 min during a vibration exposure, and then return to almost previbration levels for the duration of the exposure. The corrected volume of oxygen consumed (oxygen uptake) would increase and the RER would decrease slightly if there were an increase in metabolic rate. An increase in metabolic rate could be the result of either the vibration or the Q_{10} effect — which was discussed earlier in this paper. If the change in these respiratory variables is due to vibration, then the expected change would tend to reach an equilibrium fairly early during the exposure. On the other hand, if the change was due to the Q_{10} effect of elevated rectal temperatures, the effect would probably not be seen until near the end of the vibration exposure, and would probably continue to increase at least for the first 90 min of vibration exposure, and then reach an equilibrium after about 110 min for the LIwbv (the same as the trend observed with the rectal temperatures).

The Wilcoxon test was used to test for changes in the respiratory variables. From the results of the Wilcoxon tests, the respiratory rate was significantly elevated at 30 Hz for the HIwbv exposures by the 20-min reading. For the LIwbv exposures, the respiratory rate increased significantly only at 10 Hz, and then only after 140 min of vibration. Other than these two frequencies, there were no significant changes in the respiratory rate during wbv. As is seen in figure 30, the respiration rates increased for all frequencies except 80 Hz during the first 5 min of HIwbv. The increase at 5 Hz for the HIwbv exposure is noteworthy (fig. 30). For the LIwbv exposures, the respiration rates at 10 and 16 Hz increased after the first 5 min, but by the next reading at 70 min into the vibration exposure, the respiration rates for these two frequencies had returned to the previbration levels (fig. 31). The corrected volume of oxygen consumed (oxygen uptake) increased significantly during the first 5 min of vibration at 5 Hz, and at 20 min at 10 Hz for the HIwbv exposures (fig. 32). For the LIwbv exposures, the oxygen uptake increased significantly during the first 5 min at 80 Hz. By the end of the LIwbv exposure, the oxygen uptake had increased significantly at 5, 10, and 30 Hz (fig. 33). By comparison, at the end of the HIwbv, the LIwbv oxygen uptake was also significantly elevated at 5 and 10 Hz even though the time between readings was considerably shorter for the HIwbv exposure. Thus, it appears there may be a weak association between frequency and oxygen uptake, with 5 and 10 Hz being associated with significant increases in oxygen uptake. There did not appear to be an association between oxygen uptake and intensity level of wbv. The respiratory exchanges ratio (RER), which as was mentioned earlier is the ratio of the rate of carbon dioxide output to the rate of oxygen uptake,

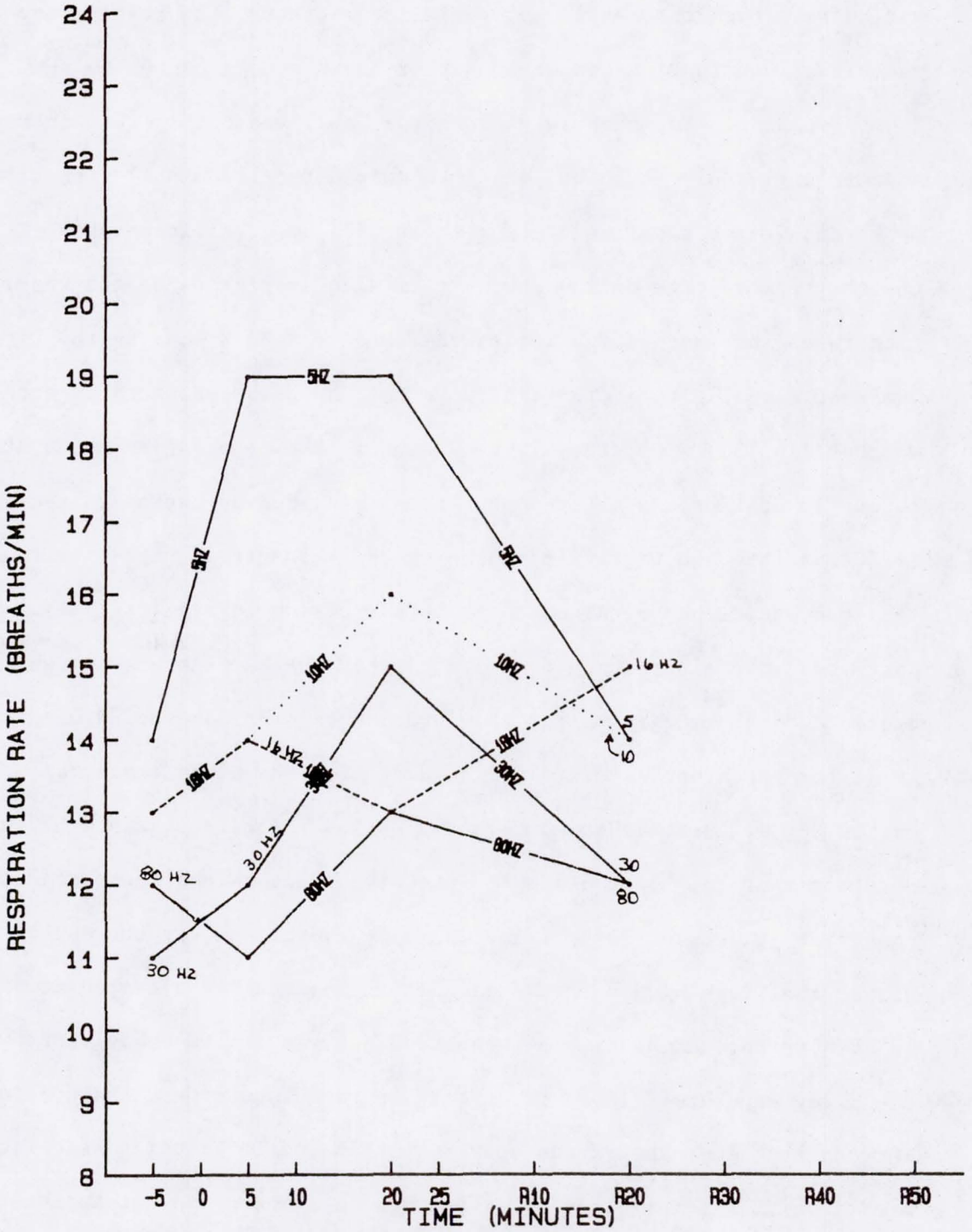

FIGURE 30. MEAN RESPIRATION RATES FOR 5, 10, 16, 30 AND 80 HZ HI WBV EXPOSURES.

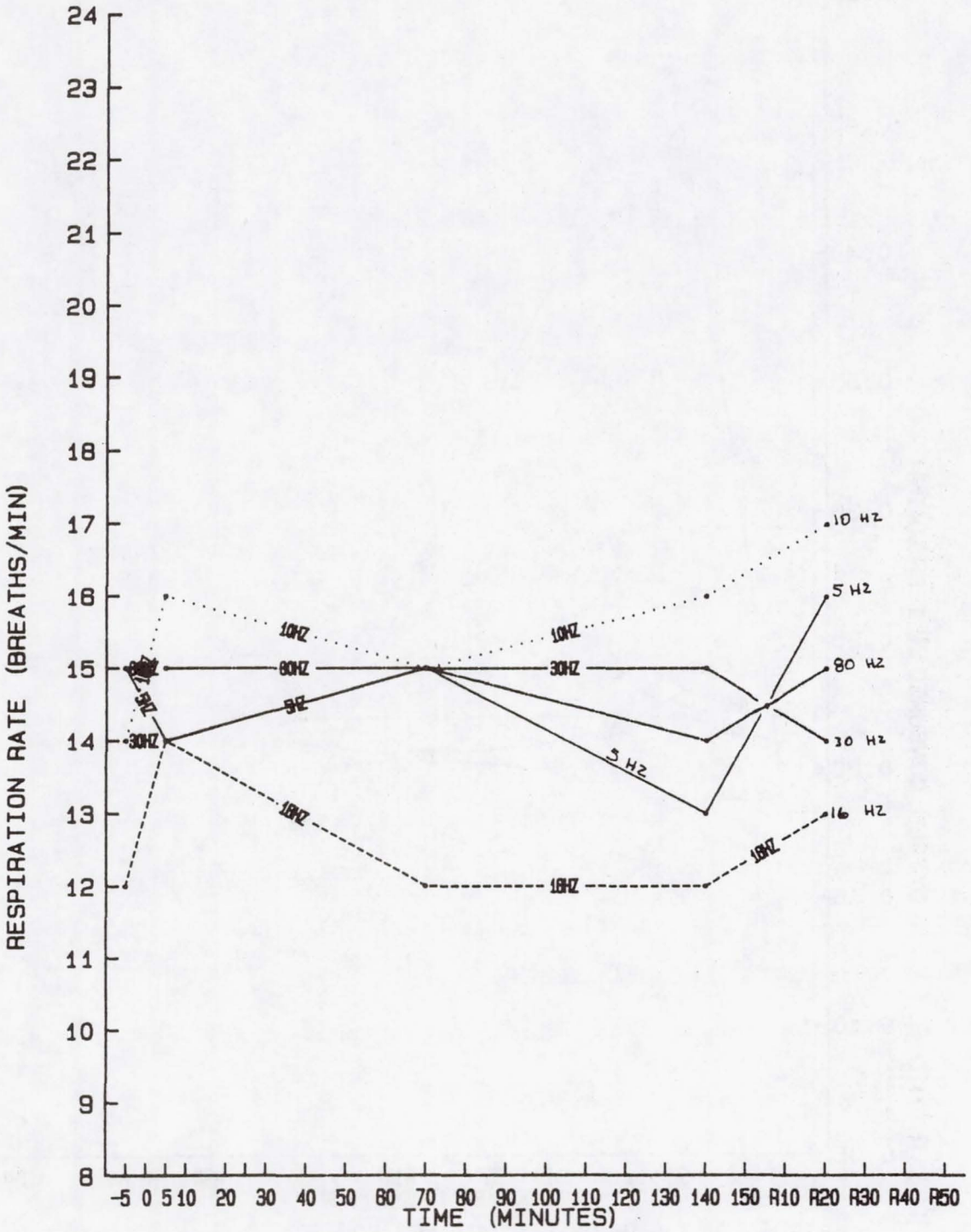

FIGURE 31. MEAN RESPIRATION RATES FOR 5, 10, 16, 30 AND 80 HZ LI WBV EXPOSURES.

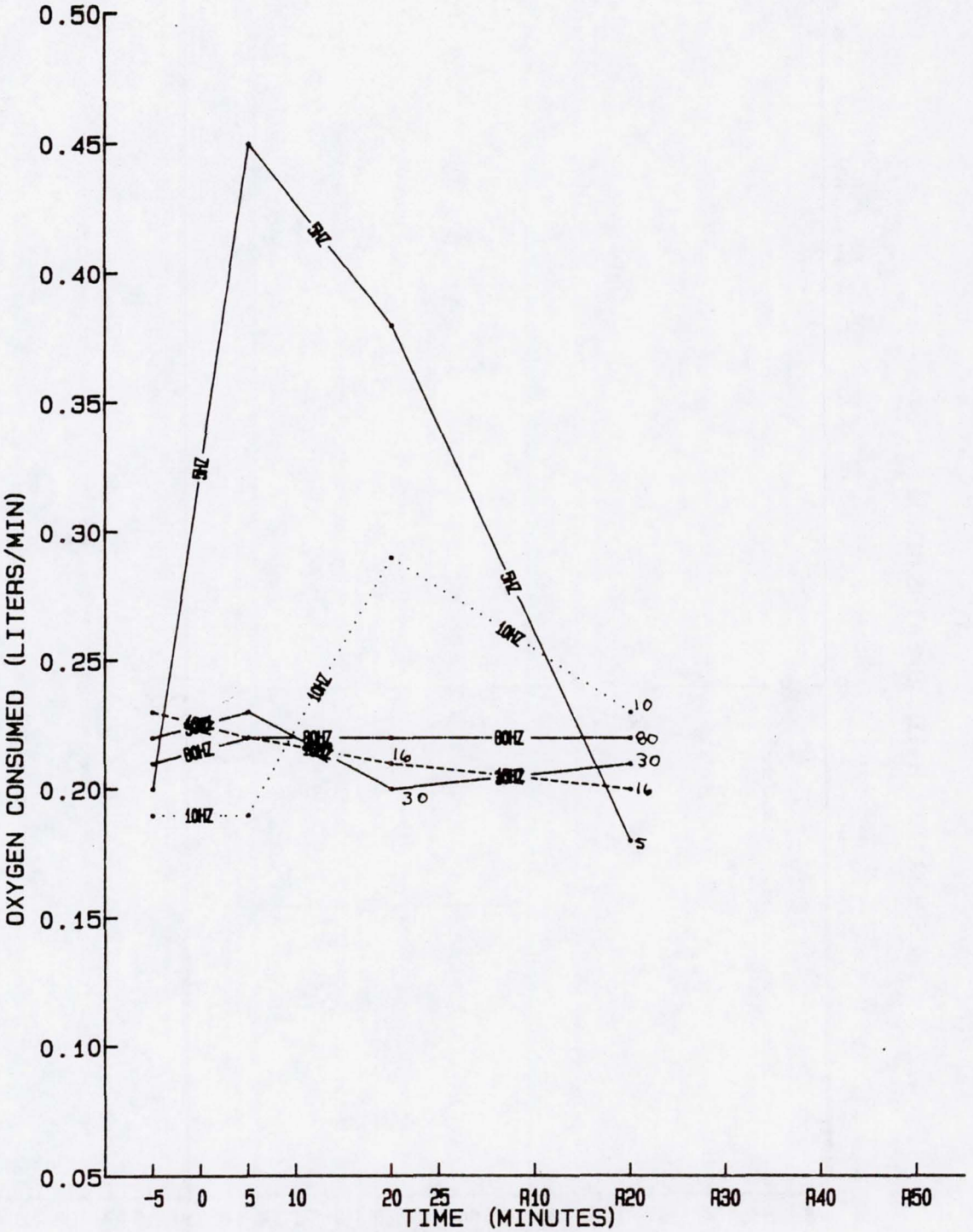

FIGURE 32. MEAN OXYGEN UPTAKES FOR 5, 10, 16, 30 AND 80 HZ HI WBV EXPOSURES.

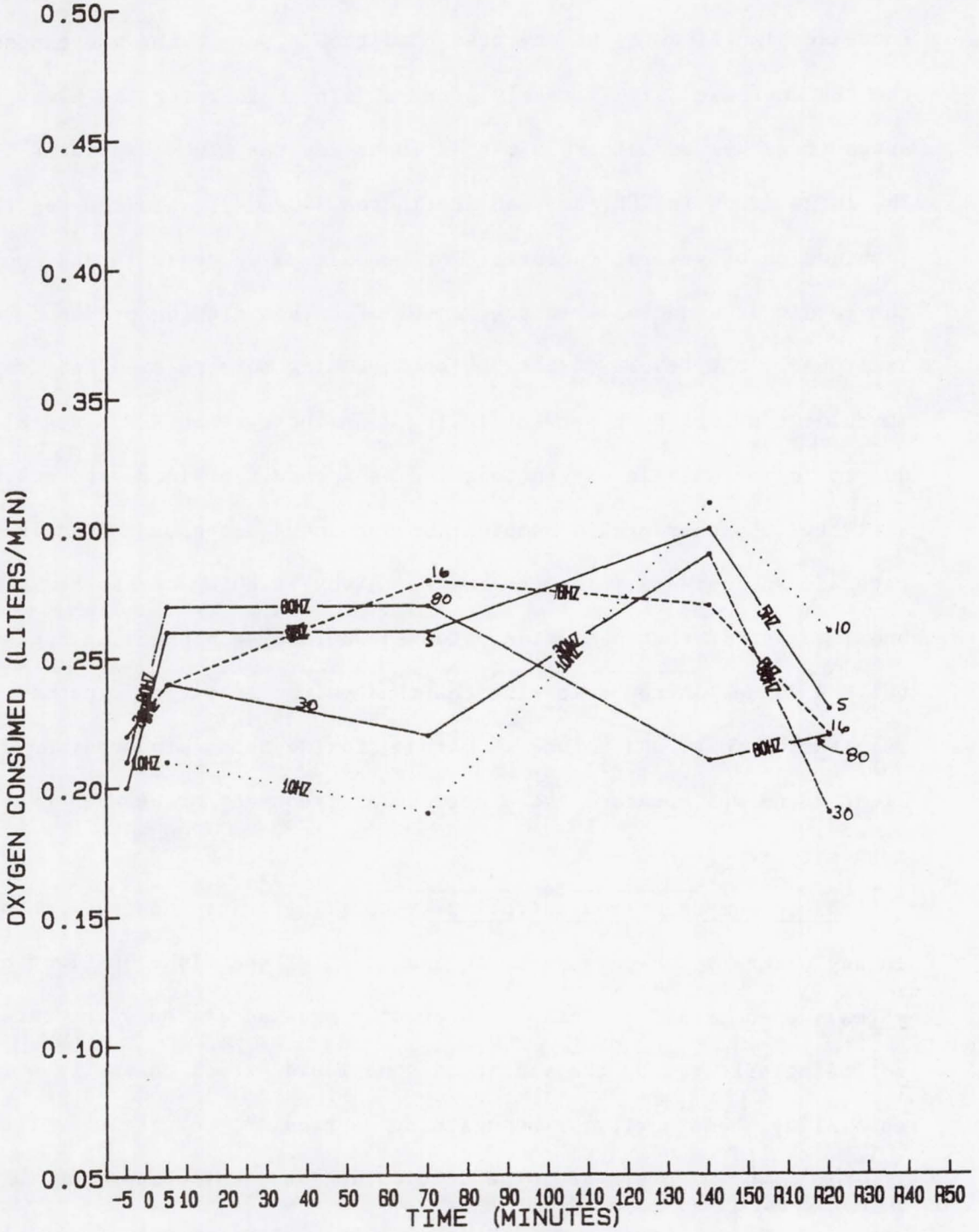

FIGURE 33. MEAN OXYGEN UPTAKES FOR 5, 10, 16, 30, AND 80 HZ LI WBV EXPOSURES.

and is indicative of metabolic rate, either remained near the same pre-vibration exposure level or significantly decreased. The RER did not increase significantly at any test condition. During the wbv exposures, the RER decreased significantly after 20 min at 5 Hz for the HIwbv exposures (fig. 34) and after 70 min at 10 Hz for the LIwbv exposures (fig. 35). The large drops in RER, as seen in figures 34 and 35, are the result of a combination of several factors. For example, a decrease in RER could be the result of a decrease in the amount of carbon dioxide produced, which is probably the result of the subject becoming more relaxed and losing muscular tone, or by hyperventilating. An increase in RER could also be due to an increase in oxygen consumed as a result of increased muscular activity. Furthermore, a combination such as a decrease in respiratory rate and an increase in oxygen uptake (HIwbv at 80 Hz at the 5-min reading) and increase in carbon dioxide produced would also produce an increase in the RER value. After comparing the true volume of oxygen consumed, the respiration rate, and volume of carbon dioxide being produced, no simple, clear trend was apparent for a particular frequency or between the two intensity levels.

Blood component concentrations — No significant changes were observed in any of the blood components or analyses for the HIwbv or LIwbv exposures (appendix XVI). This is noteworthy because if the sweat rate was not being affected by the vibration, one would expect an increase in osmolality, hematocrit, or hemoglobin, particularly by the end of the 150-min LIwbv exposure at these heat conditions. By comparison to the sbv data where there was enough water loss (largely due to sweat) to significantly increase the concentration of these blood components after 1 hr, neither the HIwbv nor the LIwbv indicates a significant change in these parameters. It should be noted that possibly the 30 min between

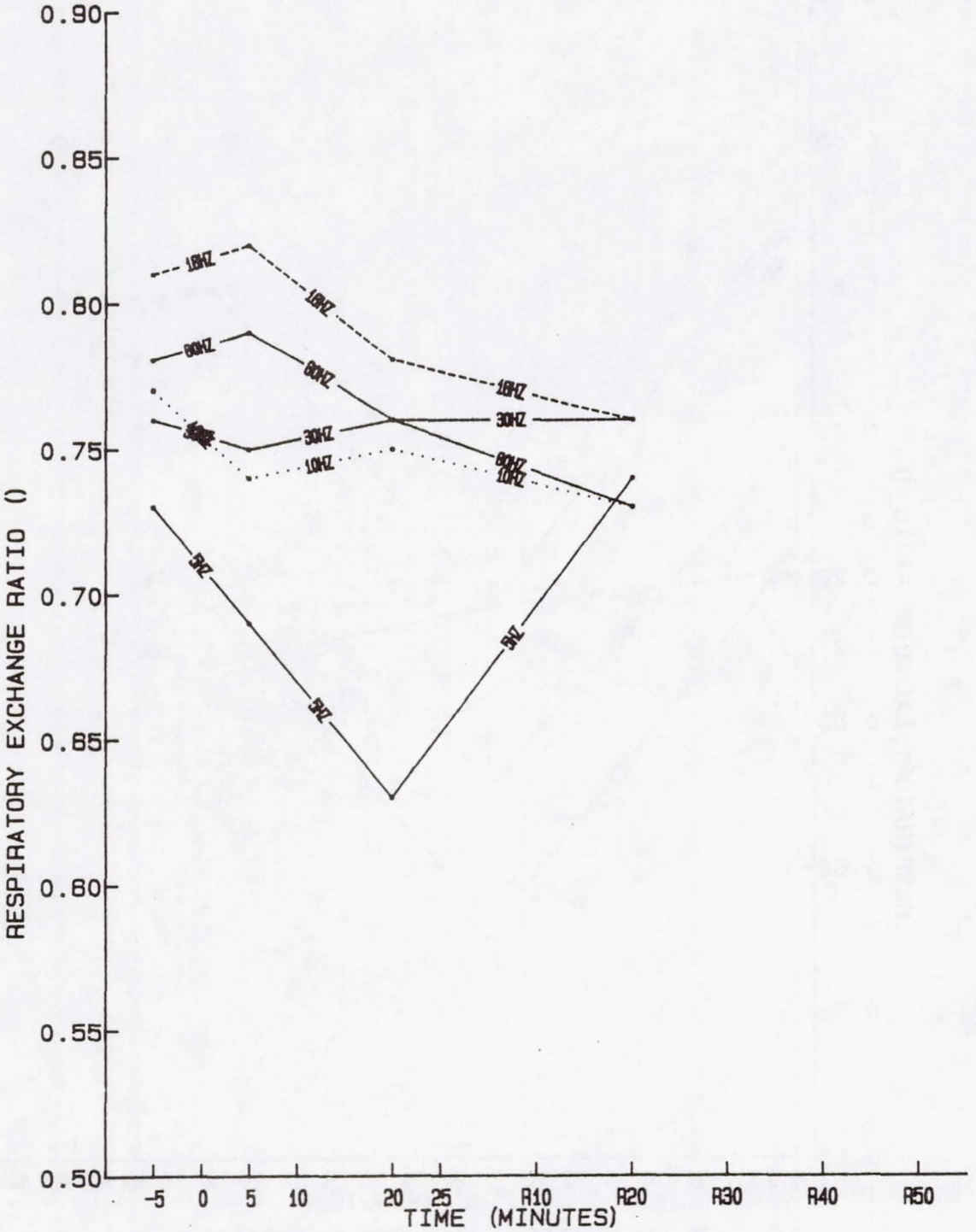

FIGURE 34. MEAN RESPIRATORY EXCHANGE RATIOS 5, 10, 16, 30 AND 80 HZ HI WBV EXPOSURES.

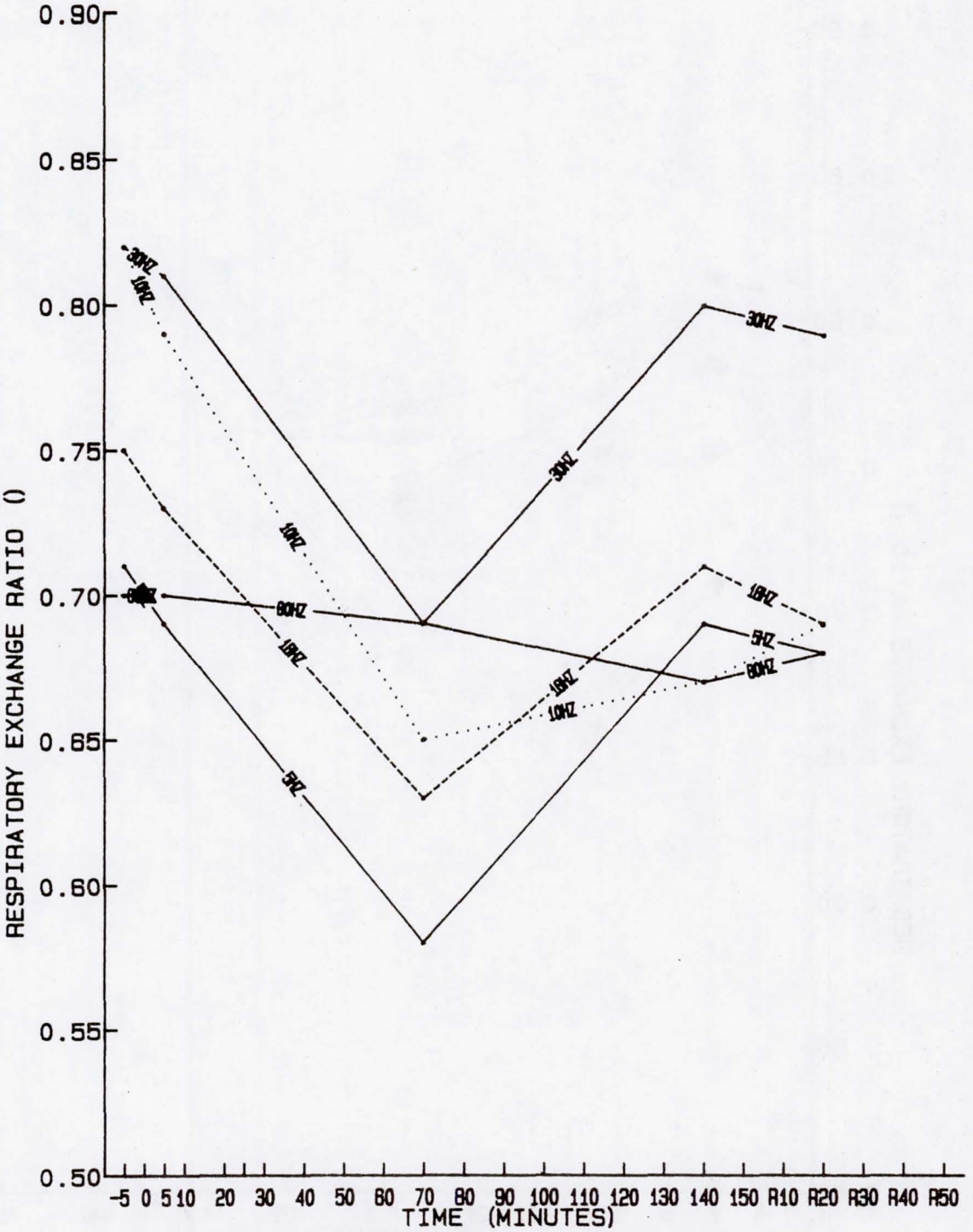

FIGURE 35. MEAN RESPIRATORY EXCHANGE RATIOS FOR 5, 10, 16, 30, AND 80 HZ LI WBV EXPOSURES.

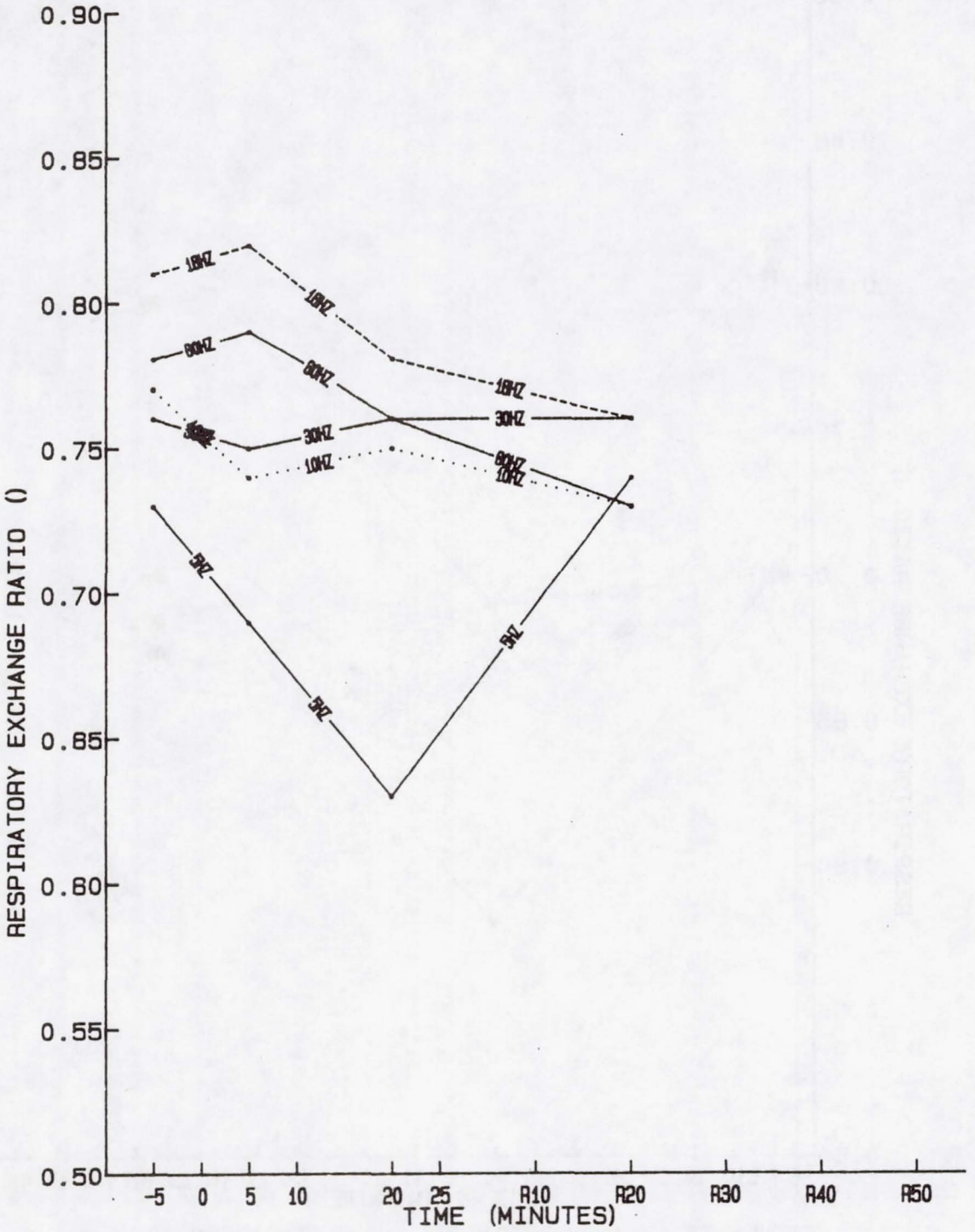

FIGURE 34. MEAN RESPIRATORY EXCHANGE RATIOS 5, 10, 16, 30 AND 80 HZ HI WBV EXPOSURES.

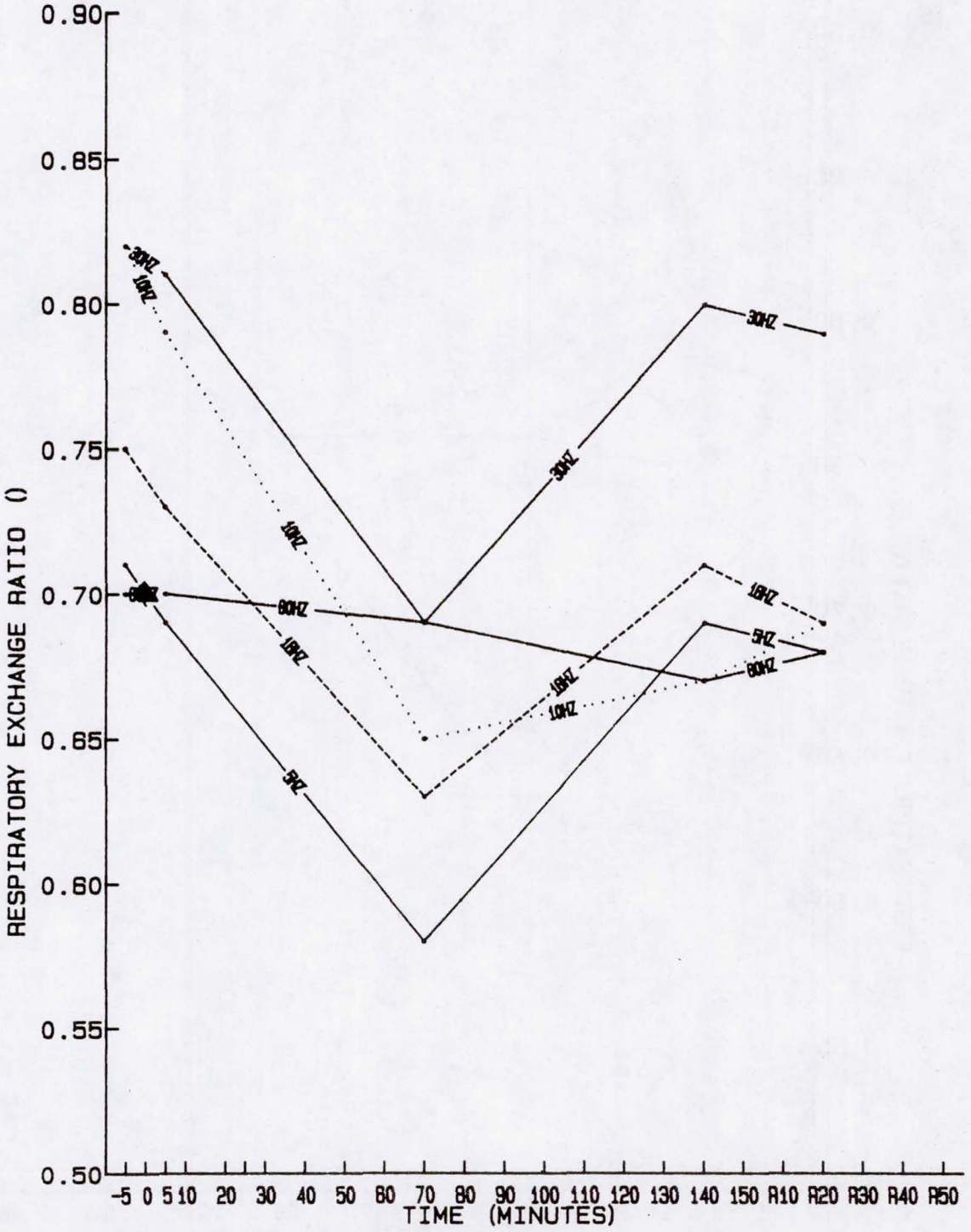

FIGURE 35. MEAN RESPIRATORY EXCHANGE RATIOS FOR 5, 10, 16, 30, AND 80 HZ LI WBV EXPOSURES.

blood-draws for the HIwbv is very likely too short a time period to see any change in these parameters. Thus, this lack of change in these blood concentrations, particularly with the LIwbv exposure, supports the hypothesis that the vibration reduces one's tolerance to a hot environment by reducing the sweat rate.

Summary of localized responses to wbv — The localized effects observed in this section appear to be consistent with the development of a mild hyperthermic condition, as was discussed in the preceding section on systemic responses to wbv. Furthermore, the localized responses appear to reflect a dose-response relationship with respect to intensity level to a greater degree than the systemic responses. Although the reduced blood-perfusion rates tend to demonstrate some interference due to the vibration exposure, the primary cause for the faster rate of increase in rectal temperatures during the wbv exposures, over that seen in the control, appears to be due to interference in the sweat rates. The hypothesis that an extra internal heat load, which was the result of an increase in metabolic rate (from the increased muscular activity to compensate for positional changes during the vibration exposure), produced the faster rate of increase in rectal temperature, does not seem to be the primary explanation.

Whole-Body Vibration Recovery Periods

The same types of analyses were performed on the wbv exposure recovery data as were performed on the control recovery data. Primary systemic responses and localized responses during the wbv recovery periods are discussed below.

Rectal temperatures — The expected trend for the rectal temperatures during the 50-min recovery period was to continue to increase, however,

at a rate less than that which was observed during the wbv exposure. The end of recovery period rectal temperatures for both intensity levels and at all frequencies of wbv were significantly more elevated ($p < 0.05$) than at the end of vibration (beginning of recovery period) rectal temperature. The end of recovery period rectal temperatures for all frequencies were also significantly higher than the previbration exposure level, which was also expected.

As can be seen in figures 16 and 17, there appears to be no significant difference in the rates of increase in the recovery rectal temperatures with respect to frequency; that is, there does not appear to be a frequency-dependent relationship.

The recovery rectal temperatures were pooled, like the wbv exposure rectal temperatures, in order to test the hypothesis that the change in rectal temperature during the recovery period was intensity-level dependent (table 15). The change in pooled rectal temperature for the HIwbv was 0.28°C ($p < 0.05$) for the 50-min recovery period. The LIwbv and control (sbv) rectal temperatures did not increase significantly. Thus, there appears to be an intensity-dependent relationship with respect to rectal temperatures during the recovery phase.

Heart rates — Since the heart rates tended to stabilize fairly early in wbv exposures (figs. 18 and 19), the heart rate during the recovery phase was expected to remain at about the same level or to decrease slightly.

There were no significant changes between the beginning and end of recovery period heart rates for the frequencies tested. The heart-rate data for each intensity level were pooled and compared for an

TABLE 15.- POOLED MEAN (± S.E.M.) RECTAL TEMPERATURES FOR THE HIwbv, LIwbv, AND CONTROL EXPOSURES DURING THE RECOVERY PERIOD

	Time, min					
	0	10	20	30	40	50
HIwbv						
Mean	37.4	37.4	37.4	37.5	37.6	37.6
S.E.M.	0.03	0.03	0.04	0.04	0.07	0.06
LIwbv						
Mean	37.7	37.7	37.6	37.7	37.7	37.7
S.E.M.	0.07	0.07	0.09	0.05	0.06	0.07
Control						
Mean	37.5	37.5	37.5	37.6	37.5	37.5
S.E.M.	0.04	0.05	0.04	0.02	0.04	0.06

intensity-dependent relationship (table 16). The recovery period heart-rate data were not found to be intensity-level dependent.

Limb-segment blood-perfusion rates — During the recovery period, the blood-perfusion rates were expected to increase — particularly if vibration was stimulating a general body vasoconstrictive response. Although the primary systemic responses did not appear to be frequency-dependent, the secondary localized responses tended to be frequency-dependent.

The UA blood-perfusion rates were measured only during the wbv exposures, and increased only at 80 Hz HIwbv during the recovery period (fig. 24). The UA blood-perfusion rates did not return to previbration

TABLE 16.- POOLED MEAN (± S.E.M.)
HEART RATES FOR HIwbv, LIwbv,
AND CONTROL EXPOSURES DURING
THE RECOVERY PERIOD

	Time, min		
	10	30	50
HIwbv			
Mean	82	82	84
S.E.M.	2	1	2
LIwbv			
Mean	84	83	85
S.E.M.	1	2	2
Control			
Mean	95	91	92
S.E.M.	1	2	2

exposure levels after the 50-min recovery period at 16 and 80 Hz (HIwbv) and at 5 Hz (LIwbv). The FA blood flow increased significantly during the recovery period at 16 and 80 Hz (fig. 22) and at 10 Hz for the HIwbv and LIwbv exposures, respectively. A return to previbration exposure levels at the end of the recovery period for the FA blood-perfusion rates was not seen at 80 Hz (HIwbv) and at 5, 10, 30, and 80 Hz (LIwbv).

Localized sweat rates — The localized sweat rates were expected to increase slightly during the recovery period until a thermal equilibrium was reached as defined by a leveling off of rectal temperatures. If the localized sweat rates did not increase significantly during the 50-min

recovery period, then either the body was near thermal equilibrium prior to the beginning of the recovery period or else the sweat glands were already producing near maximum.

The UA, FA, and C sweat rates did not increase significantly during the 50-min recovery period after any vibration exposure. The UA sweat rate did remain significantly more elevated at the end of the recovery period by comparison to the previbration exposure level at 16, 30, and 80 Hz, and at 10 and 30 Hz for the high-intensity and low-intensity wbv exposures, respectively.

The FA sweat rates remained significantly more elevated at the end of the recovery period, by comparison to previbration exposure levels at 10, 30, and 80 Hz, and at 10, 16, 30, and 80 Hz high-intensity and low-intensity wbv exposures, respectively.

The end of recovery period calf sweat rate was significantly more elevated over the preexposure vibration levels at 30 Hz, and at 80 Hz for the HIwbv and LIwbv exposures, respectively.

Although the localized sweat rates did not increase significantly ($p < 0.05$) during the 50-min recovery period after any vibration exposure, the trends for the localized sweat rates from the previbration exposure levels to the end of recovery period reflected both a frequency- and an intensity-level dependency.

Localized and mean skin temperatures — The pattern for changes in skin temperatures should follow the pattern for the localized blood-perfusion rates since the localized sweat rates did not increase significantly during the 50-min recovery period.

A consistent pattern was observed throughout the exposure and recovery periods, and can be seen in the graphs on mean skin and mean

rectal temperatures (figs. 16, 17). Frequently throughout all the vibration test conditions, whenever the mean skin temperature decreased, the mean rectal temperature increased which is clearly demonstrated in figure 36 for 30 and 80 Hz HIwbv (figs. 16, 17).

The UA end of recovery period skin temperatures were not significantly different from the previbration exposure UA skin temperatures for all wbv test conditions. The end of recovery period FA and left chest (LC) skin temperatures did not significantly increase from the previbration exposure level. The end of the recovery period calf skin temperatures significantly increased over the previbration exposure levels at 10 and 16 Hz LIwbv.

Respiration — The end of recovery period respiration rates were not significantly different from previbration exposure levels for any vibration test condition. The end of recovery period RERs were significantly less than the previbration exposures at 16 and 80 Hz for the HIwbv and at 10 Hz for the LIwbv; otherwise, no significant changes occurred.

The oxygen uptakes at the end of the recovery period were not significantly different from the previbration exposure levels. During the 50-min recovery period there were no significant increases in corrected volume of oxygen uptakes, as compared with the reading just prior to the end of vibration.

Summary of localized responses during the wbv recovery periods — The localized responses, primarily limb-segment blood-perfusion rates, performed as was expected during the recovery period, and assisted in reducing the mild stress condition (seen at the end of vibration) to a more controlled hyperthermic condition by the end of the recovery period.

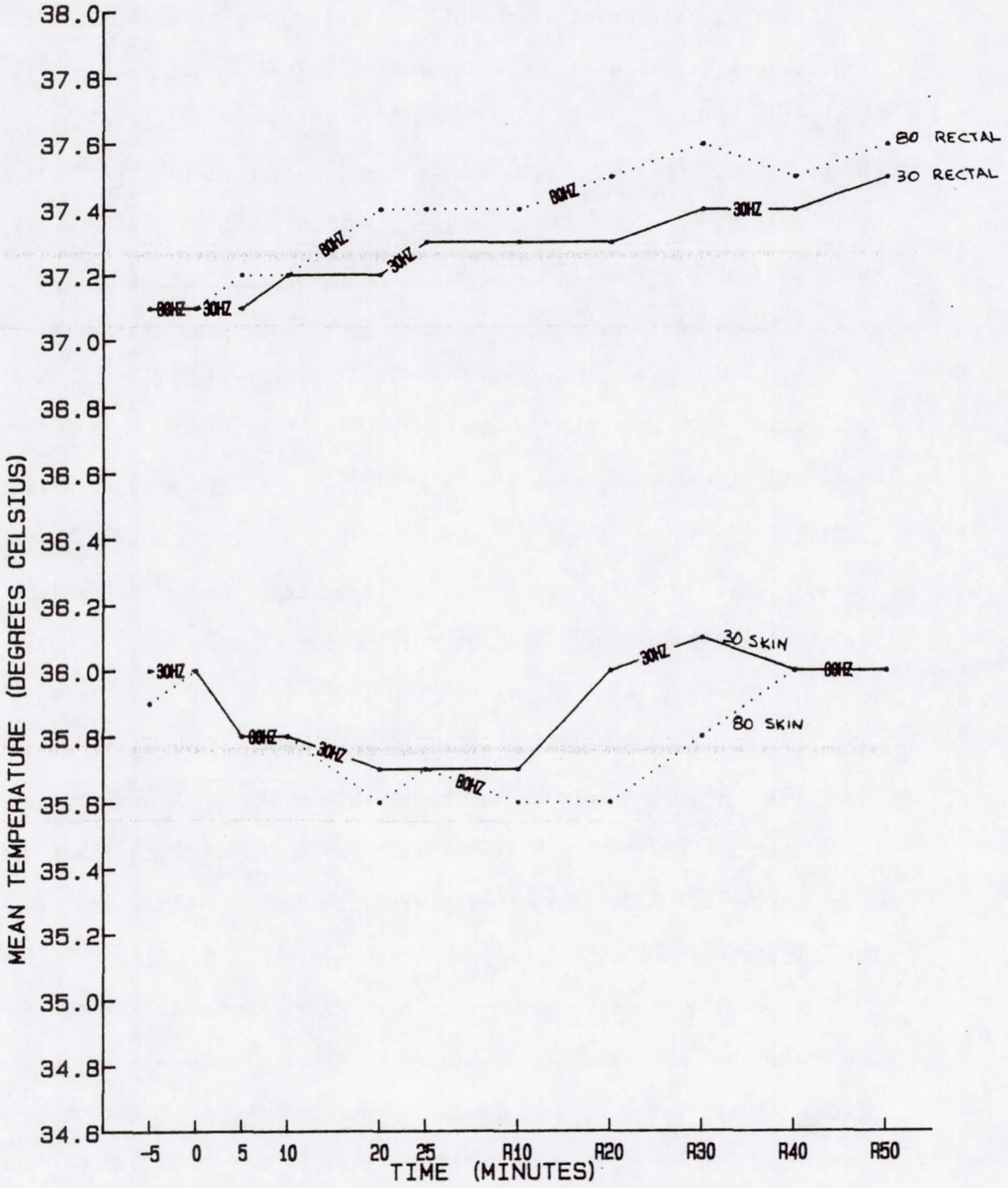

FIGURE 36. MEAN SKIN AND RECTAL TEMPERATURES FOR 30 AND 80 HZ HI WBV EXPOSURES.

DISCUSSION

The original intent of this investigation was to determine if a wbv exposure and simultaneous heat exposure resulted in a more hyperthermic state than an exposure to heat alone. The results of this study support the hypothesis that vibration reduces the cooling mechanisms, probably by increased sympathetic vasoconstriction, which would be antagonistic to the homeostatic responses required to compensate for a heat-stress condition. The second aspect of the study was to determine if specific frequency and intensity levels had a particularly detrimental impact on the thermoregulatory processes.

From the systemic responses for wbv, a more severe hyperthermic condition developed over the control. Since no significant differences in heart rate or rectal temperature were observed among the different wbv frequencies the data were pooled and compared between the intensity levels. No significant differences in heart rate and rectal temperature were observed between the two wbv intensity levels, yet the rectal temperatures for both intensity levels of wbv were significantly more elevated than the control (sbv) exposure. There was no significant difference between wbv and the control heart-rate data after the transient peak for the wbv exposures.

The dose-response relationship between the intensity levels for the wbv exposures and effects at particular frequencies were, however, suggested by the localized responses. As was pointed out previously, the blood-perfusion rates tended to decrease at 10 and 16 Hz (HIwbv) and at 10 Hz (LIwbv) — as was exemplified by the FA blood-perfusion rates. In addition to a pattern of frequency dependency for the localized secondary responses there also appeared to be an intensity-dependent relationship.

For example, the LIwbv mean skin temperature and mean sweat rates after 25 min of vibration (the duration of the HIwbv exposure) increased for both the UA and FA, while there were not corresponding increases for the HIwbv exposure. The left chest (LC) mean skin temperature did not change significantly for the LIwbv exposure, but decreased significantly for the HIwbv phase. The consistent trend in the localized responses is probably a result of increased sympathetic vasoconstriction at the higher intensity wbv exposure. Furthermore, although all wbv test frequencies (pooled data) resulted in significantly elevated core temperatures, the lower frequencies (5, 10, and 16 Hz) appear to reduce the localized sweat rates, limb-segment blood-perfusion rates, and localized skin temperatures more than the higher frequencies (30 and 80 Hz) (table 17).

At the lower frequencies, the difference in intensity levels appears to have very little effect except at 5 Hz. At 5 Hz, the lower intensity level wbv resulted in a significant increase in the blood-perfusion rate and a qualitative increase in amplitude of the sweat-rate cycle for both the UA and FA while there were no increases in the blood-perfusion rates and an apparent decrease in the sweat-rate cycle amplitudes at the higher intensity. At 10 Hz low-intensity wbv exposure, the magnitude and variation in the amplitudes of the sweat-rate cycles appeared to increase, while at the high-intensity wbv exposures, the variations in the amplitudes of the sweat-rate cycles appeared more depressed. At 30 and 80 Hz for both phases of the wbv, there appeared to be significant increases in the blood-perfusion rates and amplitudes of the sweat-rate cycles. At all frequencies of the high-intensity wbv, the variation in the amplitude of the sweat-rate cycle seemed to be less than the amplitudes seen in the low-intensity wbv frequency sweat-rate cycles.

TABLE 17.- SIGNIFICANT CHANGES IN SWEAT RATES AND LIMB-SEGMENT BLOOD-PERFUSION RATES WITH RESPECT TO VIBRATION INTENSITY

Frequency, Hz	Intensity	Blood perfusion		Sweat rates			SS
		UA	FA	UA	FA	C	
5	High	a	a	a	a	a	5
	Low	--	--	a	a	a	3
10	High	--	a	a	a	--	3
	Low	a	a	--	--	a	3
16	High	a	a	--	a	a	4
	Low	a	a	a	a	--	4
30	High	--	--	--	--	--	0
	Low	--	--	--	--	--	0
80	High	a	--	--	--	--	1
	Low	--	--	a	--	--	1

Note: UA = upperarm; FA = forearm; C = calf; SS = sum of significant changes.

^{a}A significant decrease ($p < 0.05$) or no change.

To recapitulate for the wbv exposures, it appears wbv interferes with the thermoregulatory responses at all the test frequencies with the greatest localized effects occurring at the whole-body resonating frequencies. The higher-intensity level appears to reduce the peripheral blood flow to a greater extent (e.g., 5 Hz), and also appears to consistently dampen the variations in amplitude of the sweat-rate cycles, thus moderating the intensity of the sinusoidal sweat pattern.

Since local heating of the skin does not affect the basic synchronous cyclical pattern of the sweating responses, but does increase the amplitude of the sweating cycles of the locally heated areas (refs. 49, 50) as compared with the rest of the unheated sections, one can postulate from a comparison of sweat graphs that the primary effect of increased vibration intensity (higher g-rms) is increased vasoconstriction. At the lower intensities, where vasoconstriction is not strong, more warm blood perfuses the skin tissue and heats the skin, which increases the amplitude of the sweat cycles. From other studies (ref. 3), the local heating response cannot be elicited in the absence of generalized body sweating, or after local blocking of neuroglandular transmission in sweating subjects. Thus, glandular metabolism of the skin, which would increase with blood perfusion, would also result in increased water evaporation. The effect is probably neuroglandular and may involve an increase in the amount of transmitter substance released at the neuroglandular junction which acts as a temperature sensitive or attenuator mechanism at the level of the effector (sweat gland).

Another factor which may influence sweat-gland function is the local availability of neuroglandular transmitter substances. A depletion of neuroglandular transmitter chemicals may account for the observed reduction in amplitude of the sweat gland after 100 min during the 2.5-hr wbv exposure; however, this depletion would have to be linked to the vibration since upon cessation of vibration, the higher sweat-cycle amplitudes returned almost immediately. Since there was almost immediate return, this dampening in the sweat cycle was probably not due to a simple fatiguing or local availability of neurotransmitter substance of the sweat glands.

Possibly, the drive to sweat was reduced enough by a fatigue or transmitter depletion phenomenon that the sweat mechanisms were not able to produce sweat with the reduced blood flow during the vibration. Once vibration ended, there were no vibration-induced modulating effects between the vasoconstrictive and vasodilatory mechanisms, which then resulted in enough stimulation to return to prefatigue levels of sweating.

As mentioned previously, in addition to being a test phase in this experiment, the sbv exposures served as a type of control. Since the core temperature and heart rate did not increase as rapidly as in the wbv phase during the 1-hr sbv exposures, just being in the heat booth did not produce as severe a heat-stress problem. A significant reduction in cooling mechanisms or increases in metabolic heat or both must be necessary for a heat-stress condition to evolve. As has also been shown by other investigators, the sbv did reduce the blood flow in the vibrated segment (refs. 51-53). In this study, the FA blood-perfusion rate decreased at 10, 125, and 250 Hz. An interesting note here is the question, Why did similar decreases not occur at 25 and 60 Hz? Possibly, two different types of mechanisms are involved in reducing the blood flow during a vibration exposure — a central neuroglandular mechanism, and a local mechanism (controlled by lower neural system reflexes). The central mechanism would be activated by stimulation at the whole-body resonating frequencies — 4 to 7 Hz, and 10 to 15 Hz. The local mechanism would be stimulated when the resonating frequency of a body segment or tissue type (such as skin) occurred. The skin absorbs vibration at about 125 Hz (ref. 54), with much of the energy at higher frequencies also absorbed at skin level. At the middle test frequencies (25 Hz sbv, 30 Hz wbv, 60 Hz sbv, and 80 Hz wbv), interference to thermal regulatory processes

was minimal by comparison with the lower and higher frequencies (particularly 125 Hz) which appeared to be more important than low frequencies with respect to thermoregulation.

CONCLUSIONS

According to Gordon and Heath (ref. 55), "resisted hyperthermia" occurs when there is a significant increase in core temperature in spite of an increased activation of heat-dissipating effectors. This "resisted" or "forced" hyperthermia is the condition seen in the segmental phase of this experiment. Resisted hyperthermia implies no change in the "set-point," but simply an overwhelming of the cooling mechanisms. On the other hand, "unresisted" hyperthermia and "regulated" hyperthermia imply shifts in the set-point, and either no increased activation of heat-dissipating factors (unresisted hyperthermia) or an increased activation of heat-generating/conserving effectors and inhibition of heat-dissipating effectors (regulated hyperthermia). These are the three major categories of hyperthermia, which are based on physiological mechanisms.

In this experiment just prior to the wbv exposure, the direction of the responses is initially toward a "resisted" or "forced" hyperthermia. After the vibration starts, the response remains "resisted," yet is complexed with an antagonistic additive effect of a regulated hyperthermic response, yet one probably without the shift in set-point. It appears the body is being subjected to opposing vasomotor responses, with the result being a moderating effect of both responses. It also appears that with the reduction in the heat-dissipating effectors and a subsequent increase in rectal temperature, the central gain control for thermal homeostasis is not linear, but is self-adjusted as a function of the

internal temperature change. This self-adjustment in signal gain would result in strong compensatory responses. This enhanced compensatory response was observed in the increases in limb-segment blood-perfusion rate, localized skin temperatures, and sweat rates after terminating the HIwbv exposure.

Thus, it appears that exposure to wbv during simultaneous exposures to elevated temperatures increases the risk of heat-related illnesses by interfering with the body's thermoregulatory cooling mechanisms. Vibration appears to reduce the cooling responses by decreasing blood-perfusion rates to the skin, and by dampening the amplitude of the localized sweat rates. Furthermore, the low-frequency wbv exposures (body-resonating frequencies) tend to reduce one's localized cooling responses more than frequencies above 30 Hz. Whole-body vibration increases the probability of a heat casualty situation much sooner than does hand-arm vibration, by reducing the skin blood-perfusion rates and intensity of the sweat cycles over a greater body surface area. At 125 Hz sbv, the frequency at which the skin absorbs vibration, hand-arm vibration did reduce the peripheral blood flow and heat exchange slightly which reemphasizes the important role of the hands and arms in heat exchange. Although in our test condition the subjects could very readily lose heat through other exposed areas of their bodies, the loss in this heat exchange area of the hands and arms, for workers or combat troops dressed in heavy work clothing or uniforms, may be more important than may be inferred from this study, where the subjects were lightly clothed.

So what does this mean? It means that if people are to be exposed to vibration and heat simultaneously, we should be aware that the current recommended standards which define the acceptable exposures to heat may not be conservative enough to adequately protect them from heat-related

illnesses. Clearly, a more stringent heat standard may be required. Also, engineering guidelines for vibration attenuation at particular frequencies (specifically 5-16 Hz) can be recommended for equipment which may expose workers or combat troops to vibration and heat simultaneously.

From a physiological point of view, this experiment has further explored the competition between vasoconstrictor and vasodilatory reflexes in regulation of skin blood flow and the roles of these reflexes in thermoregulation. From previous studies involving exercise and heat stress, it has been generally thought that hyperthermia can attenuate or even abolish vasoconstrictor responses associated with working muscles (refs. 56, 57). In this study, a moderate hyperthermic condition did not seem to reduce the vasoconstrictor responses significantly at the wbv resonating frequencies. At these frequencies the vasoconstrictor responses were greater than the vasodilatory responses. With this suppression of skin blood flow, the amplitude of the sweat-rate cycles also appeared depressed, thus further decreasing the body's ability to get rid of the heat. This reduced heat loss allowed the body to become more hyperthermic, which should have inhibited vasoconstrictor tone to a greater extent. This expected reduction in vasoconstriction was not seen with the increased hyperthermic condition. Thus, here is a condition where cutaneous blood flow is not fully responsive to thermal responses.

There are several directions in which one might pursue this area of research. Also, before any serious consideration should be given to occupational health regulation recommendations, epidemiological studies should be conducted to see if a problem exists in the field. Further biodynamic studies can be pursued with subjects in work clothing. Also, more realistic vibration patterns should be tested.

In retrospect, occupational health researchers have tended to focus only on a single stressor during a worker's exposure — it is the "scientifically clean" way to determine cause-effect relationships. This type of research may present problems when applying conclusions from biodynamic studies to occupational situations. A problem with focusing on potential single or serial insults to a worker is that it frequently carries over to occupational health standards, and may often produce an inadequate and naive perception of the worker in an environment where his exposure is seldom to a single agent in isolation. A worker is not usually exposed to a serial sampling of industrial insults, but is frequently bombarded with a fusillade of chemical, physical, biological, or psychological insults simultaneously. It is this simultaneous multiple exposure phenomena that should be the target of the next generation of research. A large part of the reason the health standards have not addressed this issue of multiple exposures is that the scientific data base is missing. An understanding of the worker's health in the composite should be our goal, and I believe this project is one small, incremental step in that direction of occupational health research.

APPENDIX I

HUMAN RESEARCH CONSENT DOCUMENT

NATIONAL AERONAUTICS AND SPACE ADMINISTRATION

AMES RESEARCH CENTER

MOFFETT FIELD, CALIFORNIA 94035

HUMAN RESEARCH CONSENT

PART I

The series of tests for which _____ is to serve as a subject has been explained to him in detail. The following information was included in this explanation.

A. Title: "The physiological effects of combined exposures to heat stress and vibration."

B. Principal Investigator:

1) Wil A. Spaul, Ph.D. candidate
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Co-Investigators:

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C. Purpose:

To investigate the impact of the body's opposing vasomotor responses to heat and vibration in order to determine if vibration interferes with the cooling mechanisms of a person exposed to hot environments and, if so, to characterize the vibration characteristics (frequency and acceleration) which have the most adverse impact on the thermoregulatory processes. Basically what this means: we want to find out if vibration reduces your ability to tolerate a hot environment, and if it does reduce your tolerance, we want to know what vibration characteristics (frequency and intensity) have the greatest effect. (Vasomotor refers to the changes in your blood vessel size, which in this study is important in carrying heated blood from the center of your body to your skin, where the heat is lost from your body to the environment.)

D. Nature of Tests or Experiments:

The experiment will consist of (1) a preliminary heat acclimatization period (without vibration exposures) for 2 weeks, followed by (2) a

regimen of whole-body vibration (2 weeks) and segmental vibration (1 week) exposures in a hot environment.

E. Manner in Which Test or Experiments Will be Conducted:

This study will involve the use of six volunteers who will undergo, two at a time, a regimen of varied exposures to elevated temperatures in combination from time to time with whole body vibration or segmental vibration. These exposure experiments will require 5 weeks for each two-man sequence, or 15 weeks in total, and will take place in the Laboratory of Human Environmental Physiology (Biomedical Research Division) and the Vibration Facility (Program Assurance Office). These laboratories are certified for human subjects and possess the combination of vibration and heat testing equipment needed for this study.

Since this experiment is designed to measure physiological responses as a function of different environmental conditions, your physiological responses to be recorded include: heart rate, sweat rate, peripheral blood flow, skin temperature, rectal temperature, and oxygen consumption. Your heart rate will be recorded by an ECG machine which will have electrodes taped to the surface of your chest and back. Your sweat rate will be determined by passing dry air through a capsule which is taped to the surface of the skin. The water vapor in the airstream will then be measured. Also, sweat rate will be determined by comparing pre- and post-experimental body weights. Your peripheral blood flow will be measured by impedance plethysmography. Impedance plethysmography is a simple electrical technique in which electrodes are taped to your skin (but do not penetrate the skin). Electrodes will be taped to your index finger, back of hand, forearm, upper arm, thigh and calf. A very small electrical

current will be run between the electrodes. The current is so low you will not be able to feel it. Skin temperature will be measured by thermistors, and rectal temperatures will be measured by a small, lubricated thermistor inserted 16 cm within the rectum. A thermistor is a small, very accurate, electrical temperature sensor.

Blood samples will be drawn periodically from a vein in your arm. The largest amount of blood to be withdrawn at a specific time will be 25 ml, or about 5 teaspoonfuls. Over the course of the experiment the total amount of blood withdrawn will be 430 ml, or slightly less than a pint. The purpose of the blood samples is to measure blood cell count, hemoglobin, hematocrit, potassium ion, sodium ion, osmolality, norepinephrin, epinephrin, renin and plasma volume. These scientific tests are used to find any small changes which may occur within your body.

After your selection, you will be divided into three testing groups of two persons each. Each group will proceed through the entire 5 weeks of testing before the next group begins. The reason for testing only two persons in each sequence is due to testing equipment limitations.

This experiment is designed to observe your body's response as a function of different environmental conditions, i.e., response to: high temperatures alone, in combination with whole body vibration, and in combination with segmental body vibration. These three environmental conditions form the three phases of the study.

During the first phase of the study, you will be seated in a hot and humid atmosphere for a 3-hr period per day for 10 days. Temperature conditions are described below:

Phase I

Heat conditions: duration - 3 hr

Air temperature: 105°F

Humidity (relative): 60%

(These heat conditions are very similar to the weather conditions in New Orleans in the summer.)

No vibration

Activity level: seated, at rest

During the second phase of the study, in addition to the heat conditions of Phase I, you will also be experiencing whole body vibration in the vertical "Z" axis (seat to head). To achieve this, you will be securely strapped to a vibrating seat. This phase is broken into two sections of days each in which an identical set of frequencies will be tested at two different accelerations and for two different periods of time. Exposure levels in both sections will be those allowable by the International Standards Organization (ISO) as recommended in ISO 2631-1974, ISO, Geneva, 1974.

Your exposure conditions for both sections of Phase II are listed below:

Phase II, Section A

Heat: Same as Phase I; duration - 2 hr 25 min per day

Vibration

Day	1	2	3	4	5
Frequency, Hz	5	10	16	30	80
Acceleration, g-rms	0.37	0.46	0.72	1.40	3.70

(g-rms is a unit used to measure the average force of gravity or "pull," where 1 "g" is equal to the Earth's force of gravity.)

Duration: 25 min/day

Activity level: seated, at rest

Phase II, Section B

Heat: Same as Phase I; duration - 3.5 hr per day

Vibration

Day	1	2	3	4	5
Frequency, Hz	5	10	16	30	80
Acceleration, g-rms	0.14	0.18	0.28	0.55	1.44

Duration: 2.5 hr/day

Activity level: seated, at rest

In the third phase of the study, the temperature and humidity will remain the same as in the first two phases, but you will be exposed to "segmental body" vibration. This will involve your standing on a non-vibrating platform while grasping a vibration source. Your gripping force will be monitored by a meter attached to the vibrating handles. The goal here is to duplicate the kind of vibration felt by workers using hand-held tools, such as pneumatic impact hammers.

Your exposures for the segmental vibration conditions are as follows:

Phase III

Heat: Same as Phase I; duration - 3 hr per day

Vibration

Day	1	2	3	4	5
Frequency, Hz	10	25	60	125	250
Acceleration, g-rms	0.57	0.90	2.10	4.40	8.70

Duration: 1 hr/day

Activity level: standing, at rest

You should note that during both Phase II and Phase III of the study, you will be exposed to only one frequency and one acceleration per day. Due to the technical nature of the above information, this material will be expanded and explained in an oral discussion and you will be given an opportunity to inspect the equipment.

F. Duration:

March 1982-October 1982 for the entire experiment. The human subjects will be needed for the period March through October 1982.

G. Foreseeable Inconvenience, Discomfort, and/or Risks:

1. Sitting in a hot, humid environment with no vibration for 3 hr each day for 10 working days (2 weeks).

2. Sitting in a hot, humid environment and being whole-body vibrated at a single frequency and single acceleration per day for 10 working days.

3. Standing in a hot, humid environment and grasping a hand-held vibrating source for 1 hr maximum per day for 5 days.

4. Heat exposures will be in excess of the recommended exposure criteria as set forth in the 1980 Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environments, as published by the American Conference of Governmental Industrial Hygienists; i.e., 96°F instead of the recommended 90°F. There should be no health problems with this exposure since the standard has a large safety factor built into it and is designed for the general work force, which includes old, and overweight, people. Also it is written for an exposure of 40 hr per week for life.

5. Having your sweat rate monitored by dry air capsules which will be attached by an air line to a water vapor analyzer taped to your forehead, chest, stomach, forearm, back of hand, or calf.

6. Having your skin temperature monitored by seven flat thermistors of approximately 3/8 in. diam. These thermistors will be taped to your skin on your arms, chest, and legs.

7. Having your heart rate and EKG monitored by three probes, all taped on your chest.

8. Having your deep-body temperature monitored by a rectal temperature probe.

9. Venous punctures for blood samples, with possibly some local bruising near the puncture.

10. The Evans Blue dye used in plasma volume measurements may give your skin a slight blue tinge, particularly in light skinned individuals, but this should disappear within a few days.

11. You are aware that during the course of this experiment you will:

a. Feel hot and sweaty.

b. Feel the impact of vibration.

c. Have a remote possibility of experiencing nausea, vomiting or dizziness from the heat, vibration or combination of heat and vibration.

12. You are aware that although no long-range or chronic effects are known to occur to healthy individuals at the vibration and heat parameters used in this experiment, the existence of such effects cannot be completely discounted.

_____ _____

Date Signature of Principal Investigator

_____ _____

Date Signature of Authorized Governmental Medical Monitor

HUMAN RESEARCH CONSENT

PART II - TO BE COMPLETED BY SUBJECT

NOTE TO SUBJECT: Read Part I carefully. If there is anything in Part I that you do not understand, ask one of the scientists or technicians who will be conducting the test or experiment for an explanation.

Do not sign this form until Part I has been completed and signed.

A. I hereby agree to participate, as a subject, in the tests or experiments described in Part I of this form.

B. I am aware of the possible foreseeable harmful consequences that may result from such participation, and that such participation may otherwise cause me inconvenience and discomfort.

C. I acknowledge that my consent has been freely given and that I may withdraw my consent, and thereby withdraw from the study, at any time. I also understand (1) that the Principal Investigator may request my employer (Management Systems Associates) to dismiss me if I am not conforming to the requirements of the study as outlined in Part I; (2) that the NASA Medical Monitor may request my employer (Management Systems Associates) to dismiss me if, in his opinion, my health and well being are threatened; and (3) that the Facility Safety Manager may terminate the study in the event that unsafe conditions develop that cannot be immediately corrected. I understand that if I withdraw from the study, or am dismissed, I will be paid for the time served up to the point of my departure, but not thereafter.

D. I hereby agree that all records collected by NASA in the course of this experiment are available to the NASA Medical Monitor and the Principal Investigator.

E. The foregoing shall not be construed as a release of NASA from any future liability arising from or in connection with the tests or experiments in which I am to participate as a subject.

_____ _____

Date Signature of Subject

_____

Street Address

_____

City and State

_____

Telephone Number

APPENDIX II

DAILY SCHEDULE FOR AFTERNOON SUBJECT FOR "PHASE II, SECTION A" VIBRATION EXPOSURE

_____ Hertz _____ Accell.

AFTERNOON SHEET

PHASE IIA: HEAT AND WHOLE BODY VIBRATION

Subject _____ Date _____

Amount of sleep _____ Awake _____ a.m.

1145 Calibrate equipment

1200 Subject arrives

1205 Subject changes and inserts rectal probe

1215 Transfer subject to shaker area

1220 On gurney: Apply electrodes: Body weight _____ kg

1230 Seated in booth

1230-

1245 Biosensors applied

1245 Start recording sweat rate and skin temperatures: DB _____°C;
RH _____%

1256 Start 2-min impedance test: HR _____; DB _____°C; RH _____%

1258 Insert oxygen uptake hose

1305 Start oxygen uptake readings: DB _____°C; RH _____%

1307 Start 2-min impedance: HR _____; DB _____°C; RH _____%

1311 Start heating booth

1313 Start 2-min impedance: HR _____; DB _____°C; RH _____%

1317 Start 2-min impedance: HR _____; DB _____°C; RH _____%

1322 Start 2-min impedance: HR _____; DB _____°C; RH _____%

1327 Start 2-min impedance: HR _____; DB _____°C; RH _____%

1332 Start 2-min impedance: HR _____; DB _____°C; RH _____%

1336 Insert oxygen uptake hose

1343 Start oxygen uptake readings: DB _____°C; RH _____%

1347 Start 2-min impedance: HR _____; DB _____°C; RH _____%

1350 Venous puncture: DB _____°C; RH _____%

1400 Start 2-min impedance: HR _____; DB _____°C; RH _____%

1403 Insert oxygen hose and check seat belt

1405 Start vibration exposure: HR _____; DB _____°C; RH _____%

1410 Start oxygen uptake readings: DB _____°C; RH _____%

1420 HR _____; DB _____°C; RH _____%

1421 Insert oxygen uptake hose

1428 Start oxygen uptake readings: DB _____°C; RH _____%

1430 End vibration exposure: HR _____; DB _____°C; RH _____%

1432 Start 2-min impedance: HR _____; DB _____°C; RH _____%

1434 Insert air tube for oxygen uptake

1435 Venous puncture: DB _____°C; RH _____%

1441 Start oxygen uptake readings: DB _____°C; RH _____%

1500 Start 2-min impedance run: HR _____; DB _____°C; RH _____%

1526 Start 2-min impedance run: HR _____; DB _____°C; RH _____%

1530 End all measurements; end heart exposure: DB _____°C; RH _____%

1535 Disconnect all sensors from subject

1540 Leave heat booth: Get arm measurements for blood flow;

Body weight _____kg

1545 Drive subject back to lab for shower and rectal probe removal

1600 Leave lab

APPENDIX III

DAILY SCHEDULE FOR AFTERNOON SUBJECT FOR "PHASE II, SECTION B" VIBRATION EXPOSURE

_____ Hertz _____ Accell.

PHASE IIB: HEAT AND WHOLE BODY VIBRATION

Subject _____ Date _____

Amount of sleep _____ Awake _____ a.m.

1115 Calibrate equipment

1130 Subject arrives

1145 Subject changes and inserts rectal probe

1155 Transfer subject to shaker area

1200 On gurney: Apply electrodes: Body weight _____ kg

1238 Seated in booth

1245-

1300 Biosensors applied

1305 Start recording sweat rate and skin temperatures: DB _____°C;
RH _____%

1311 Start 2-min impedance test: HR _____; DB _____°C; RH _____%

1313 Insert oxygen uptake hose

1320 Start oxygen uptake readings: DB _____°C; RH _____%

1322 Start 2-min impedance: HR _____; DB _____°C; RH _____%

1325 Start heating booth

1327 Start 2-min impedance: HR _____; DB _____°C; RH _____%

1332 Start 2-min impedance: HR _____; DB _____°C; RH _____%

1337 Start 2-min impedance: HR _____; DB _____°C; RH _____%

1342 Start 2-min impedance: HR _____; DB _____°C; RH _____%

1346 Start 2-min impedance: HR _____; DB _____°C; RH _____%

1349 Start oxygen uptake hose

1356 Start oxygen uptake reading

1401 Venous puncture: DB _____°C; RH _____%

1406 Start 2-min impedance: HR _____; DB _____°C; RH _____%

1408 Insert oxygen hose and check seat belt

1410 Start vibration exposure: HR _____; DB _____°C; RH _____%

1415 Start oxygen uptake reading

1435 HR _____; DB _____°C; RH _____%

1505 HR _____; DB _____°C; RH _____%

1555 HR _____; DB _____°C; RH _____%

1605 Put arm back into sling

1611 HR _____; DB _____°C; RH _____%

1633 Insert oxygen uptake hose

1640 Start oxygen uptake readings: DB _____°C; RH _____%

1641 Start 2-min impedance: HR _____; DB _____°C; RH _____%

1643 Insert air tube for oxygen uptake

1646 Venous puncture: DB _____°C; RH _____%

1650 Start oxygen uptake readings: DB _____°C; RH _____%

1710 Start 2-min impedance: HR _____; DB _____°C; RH _____%

1735 Start 2-min impedance: HR _____; DB _____°C; RH _____%

1740 End all measurements; end heat exposure: DB _____°C; RH _____%

1740 Disconnect all sensors from subject

1743 Leave heat booth: Get arm measurements for blood flow;

Body weight _____kg

1750 Drive subject back to lab for shower and rectal probe removal

1805 Leave lab

APPENDIX IV

DAILY SCHEDULE FOR AFTERNOON SUBJECT FOR "PHASE III"

VIBRATION EXPOSURE

AFTERNOON SHEET

PHASE III: HEAT AND SEGMENTAL BODY VIBRATION

Subject _____ Date _____

Amount of sleep _____ Awake _____ a.m.

1145 ____ Calibrate equipment

1200 ____ Subject arrives

1205 ____ Subject changes and inserts rectal probe

1215 ____ Transfer subject to shaker area

1220 ____ On gurney: Apply electrodes: Body weight _____

1230 ____ Seated in booth

1245 ____ Biosensors applied

1256 ____ Start 2-min impedance test: HR _____; DB _____°C; RH _____%

1258 ____ Insert oxygen uptake hose

1305 ____ Start oxygen uptake readings: DB _____°C; RH _____%

1307 ____ Start 2-min impedance: HR _____; DB _____°C; RH _____%

1311 ____ Start heating booth

1313 ____ Start 2-min impedance: HR _____; DB _____°C; RH _____%

1317 ____ Start 2-min impedance: HR _____; DB _____°C; RH _____%

1322 ____ Start 2-min impedance: HR _____; DB _____°C; RH _____%

1327 ____ Start 2-min impedance: HR _____; DB _____°C; RH _____%

1332 ____ Start 2-min impedance: HR _____; DB _____°C; RH _____%

1336 ____ Insert oxygen uptake hose

1343 ____ Start oxygen uptake readings: DB _____°C; RH _____%

1347 ____ Start 2-min impedance: HR _____; DB _____°C; RH _____%

1350 ____ Venous puncture: DB _____°C; RH _____%

1400 ____ DB _____°C; RH _____%

1401 ____ Start 2-min impedance: HR _____; DB _____°C; RH _____%

1403 ____ Insert oxygen hose

1405 ____ Start vibration exposure: HR _____; DB _____°C; RH _____%

1410 ____ Start oxygen uptake readings: DB _____°C; RH _____%

1420 ____ HR _____; DB _____°C; RH _____%

1421 ____ Insert oxygen uptake hose

1428 ____ Start oxygen uptake readings: DB _____°C; RH _____%

1430 ____ End vibration exposure: HR _____; DB _____°C; RH _____%

1432 ____ Start 2-min impedance: HR _____; DB _____°C; RH _____%

1434 ____ Insert air tube for oxygen uptake

1435 ____ Venous puncture: DB _____°C; RH _____%

1441 ____ Start oxygen uptake readings: DB _____°C; RH _____%

1500 ____ Start 2-min impedance run: HR _____; DB _____°C; RH _____%

1526 ____ Start 2-min impedance run: HR _____; DB _____°C; RH _____%

1530 ____ End all measurements; end heat exposure: DB _____°C; RH _____%

1535 ____ Disconnect all sensors from subject

1540 ____ Leave heat booth: Get arm measurements for blood flow;

Body weight _____%

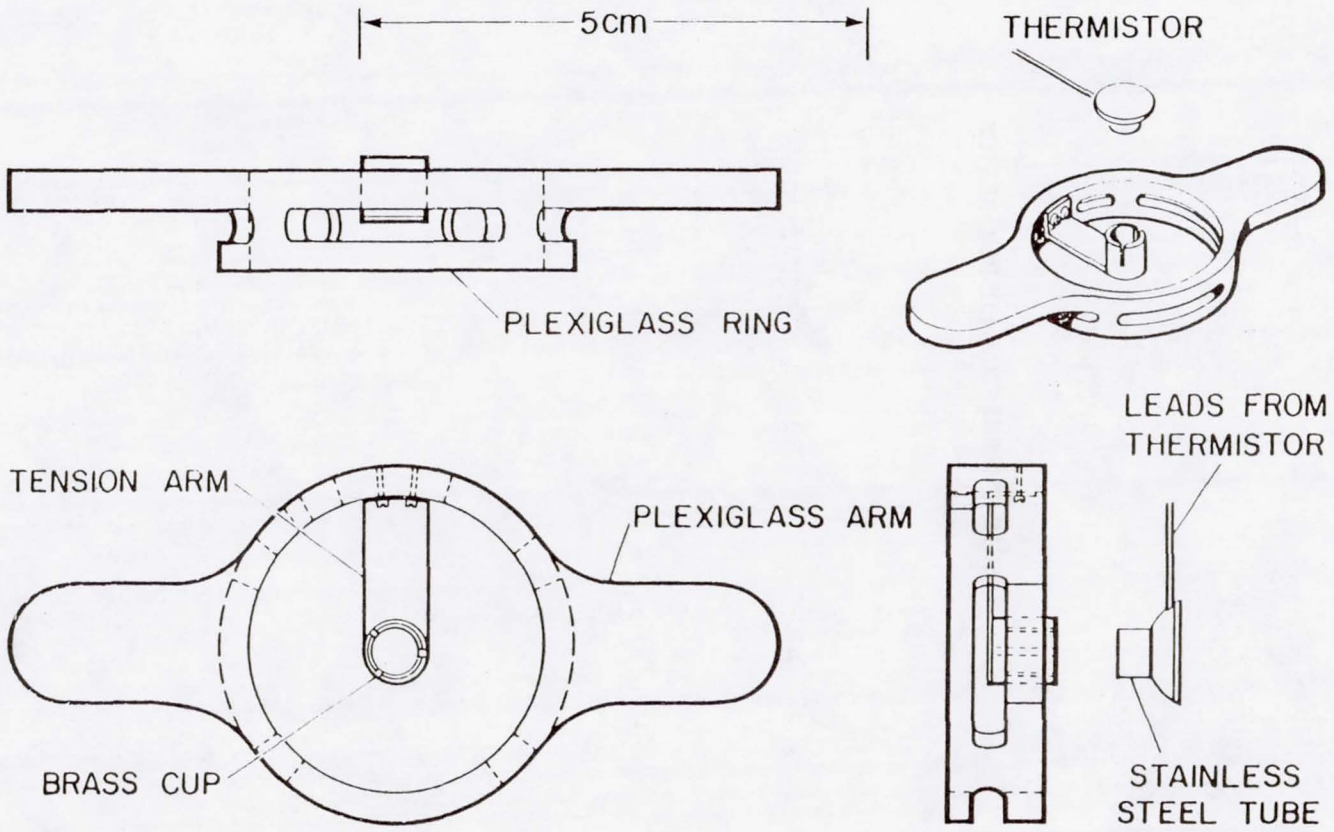
1545 ____ Drive subject back to lab for shower and rectal probe removal

1600 ____ Leave lab

NOTES:

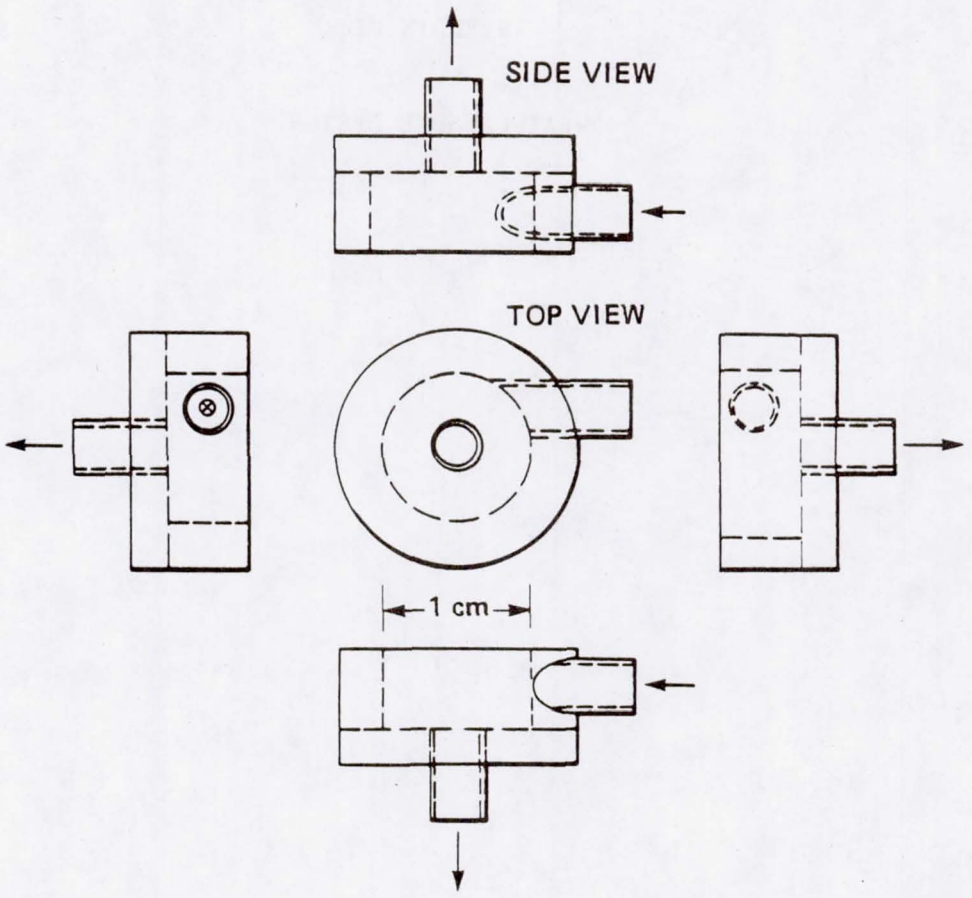
APPENDIX V

SKIN THERMISTOR HOLDER DESIGN


5cm
THERMISTOR
PLEXIGLASS RING
LEADS FROM
THERMISTOR
TENSION ARM
PLEXIGLASS ARM
BRASS CUP
STAINLESS
STEEL TUBE

APPENDIX VI

SWEAT CAPSULE DESIGN


SIDE VIEW
TOP VIEW
1 cm

APPENDIX VII

COMPUTATIONS FOR LOCALIZED SWEAT RATES

COMPUTATION OF LOCALIZED SWEAT RATE

1. From printout on sweat measuring box, record the percent relative humidity and air temperature of the airstream from the sweat vapor collection capsule, and the control airstream.

2. From the air temperature and percent relative humidity, determine the absolute humidity ratio, grams of water per grams of dry air, from a psychrometric chart, and record the absolute humidity ratio for both the control and the sweat vapor collection capsule.

3. For Spaul's design of the sweat vapor collection capsule, use the following formula:

$$Msw = Va(W_{out} - W_{in})(72.029)$$

Msw = water loss, g/cm^2-hr

Va = volume flow rate, liters/min

W_{out} = absolute humidity ratio leaving capsule, grams of water per grams of dry air

W_{in} = absolute humidity ratio of control, grams of water per grams of dry air

72.029 = constant for surface area of capsule, 60 min/hr, and specific volume of air, liters per gram of dry air

APPENDIX VIII

NASA's HUMAN RESEARCH EXPERIMENT REVIEW BOARD (HRERB) ROSTER

LIST OF HUMAN RESEARCH EXPERIMENTS REVIEW BOARD

1. Robert Showman, Assistant Chief of Programs, Flight Systems and Simulation Research Division
2. Ralph Pelligra, Senior Medical Officer, Institutional Operations Office, Office of the Director
3. Charles Billings, Assistant Chief of Research, Man-Vehicle Systems Research Division
4. Nadine Kuhlman, Personnel Management Division, Office of the Director of Administration
5. Richard Kurkowski, Research Assistant for Aviation Safety, Flight Systems and Simulation Research Division
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7. Harold Sandler, Chief, Biomedical Research Division

EX OFFICIO MEMBERS

1. Rosamond French, Office of the Chief Counsel, Office of the Director
2. Robert Magers, Deputy Chief, Institutional Operations Office, Office of the Director
3. Dee O'Hara, Manager, Human Research Facility

APPENDIX IX

DATA FOR MEAN HEART RATES

[beats/min]

TEST CONDITION	LABEL	V-5	V0	V5	V10	V20	V25	V30	V40	V50	V60	V70	V80	V90	V100	V110	V120	V130	V140	V150	R0	R10	R20	R30	R40	R50
5HZ/.37G/HR	HR1	76.00	0.00	0.00	87.00	0.00	84.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	88.00	0.00	81.00	0.00	86.00
10HZ/.46G/HR	HR2	77.00	0.00	0.00	85.00	0.00	87.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	82.00	0.00	85.00	0.00	89.00
16HZ/.72G/HR	HR3	78.00	0.00	0.00	84.00	0.00	86.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	80.00	0.00	82.00	0.00	80.00
30HZ/1.4G/HR	HR4	74.00	0.00	0.00	80.00	0.00	82.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	78.00	0.00	82.00	0.00	81.00
80HZ/3.7G/HR	HR5	74.00	0.00	0.00	86.00	0.00	86.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	81.00	0.00	82.00	0.00	86.00
5HZ/.14G/HR	HR6	76.00	0.00	0.00	84.00	0.00	82.00	0.00	0.00	0.00	83.00	0.00	0.00	0.00	0.00	0.00	85.00	0.00	0.00	0.00	0.00	88.00	0.00	88.00	0.00	89.00
10HZ/.18G/HR	HR7	76.00	0.00	0.00	87.00	0.00	88.00	0.00	0.00	0.00	83.00	0.00	0.00	0.00	0.00	0.00	84.00	0.00	0.00	0.00	0.00	83.00	0.00	82.00	0.00	88.00
16HZ/.28G/HR	HR8	70.00	0.00	0.00	77.00	0.00	80.00	0.00	0.00	0.00	82.00	0.00	0.00	0.00	0.00	0.00	81.00	0.00	0.00	0.00	0.00	83.00	0.00	81.00	0.00	82.00
30HZ/.55G/HR	HR9	68.00	0.00	0.00	75.00	0.00	78.00	0.00	0.00	0.00	77.00	0.00	0.00	0.00	0.00	0.00	80.00	0.00	0.00	0.00	0.00	83.00	0.00	84.00	0.00	85.00
80HZ/1.4G/HR	HR10	71.00	0.00	0.00	79.00	0.00	80.00	0.00	0.00	0.00	79.00	0.00	0.00	0.00	0.00	0.00	82.00	0.00	0.00	0.00	0.00	83.00	0.00	78.00	0.00	80.00
S10HZ/.57G/HR	HR11	91.00	0.00	0.00	92.00	0.00	90.00	0.00	0.00	0.00	98.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	99.00	0.00	93.00	0.00	97.00
S25HZ/.90G/HR	HR12	85.00	0.00	0.00	87.00	0.00	86.00	0.00	0.00	0.00	94.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	93.00	0.00	92.00	0.00	88.00
S60HZ/2.1G/HR	HR13	88.00	0.00	0.00	90.00	0.00	89.00	0.00	0.00	0.00	99.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	97.00	0.00	99.00	0.00	94.00
S125HZ/4.4G/HR	HR14	86.00	0.00	0.00	88.00	0.00	88.00	0.00	0.00	0.00	91.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	93.00	0.00	86.00	0.00	91.00
S250HZ/8.7G/HR	HR15	85.00	0.00	0.00	85.00	0.00	86.00	0.00	0.00	0.00	94.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	92.00	0.00	87.00	0.00	89.00

APPENDIX X

DATA FOR MEAN SKIN AND RECTAL TEMPERATURES [°C]

TEST CONDITION	LABEL	V-5	V0	V5	V10	V20	V25	V30	V40	V50	V60	V70	V80	V90	V100	V110	V120	V130	V140	V150	R0	R10	R20	R30	R40	R50
5HZ/.37G/TSK	TS1	35.70	35.90	36.10	36.10	36.30	36.30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	36.30	36.10	35.80	36.00	35.90	35.90
5HZ/.37G/TRE	TR1	37.10	37.20	37.20	37.30	37.40	37.40	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	37.40	37.40	37.40	37.50	37.60	37.60
10HZ/.46G/TSK	TS2	35.60	35.50	35.60	35.50	35.50	35.40	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	35.40	35.50	35.60	35.50	35.50	35.40
10HZ/.46G/TRE	TR2	37.20	37.20	37.30	37.30	37.30	37.50	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	37.50	37.40	37.50	37.60	37.70	37.70
16HZ/.72G/TSK	TS3	35.40	35.40	35.30	35.30	35.20	35.20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	35.20	35.20	35.40	35.50	35.50	35.60
16HZ/.72G/TRE	TR3	37.20	37.20	37.20	37.30	37.30	37.40	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	37.40	37.50	37.50	37.60	37.80	37.80
30HZ/1.4G/TSK	TS4	36.00	36.00	35.80	35.80	35.70	35.70	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	35.70	35.70	36.00	36.10	36.00	36.00
30HZ/1.4G/TRE	TR4	37.10	37.10	37.10	37.20	37.20	37.30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	37.30	37.30	37.30	37.40	37.40	37.50
80HZ/3.7G/TSK	TS5	35.90	36.00	35.80	35.80	35.60	35.70	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	35.70	35.60	35.60	35.80	36.00	36.00
80HZ/3.7G/TRE	TR5	37.10	37.10	37.20	37.20	37.40	37.40	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	37.40	37.40	37.50	37.60	37.50	37.60
5HZ/.14G/TSK	TS6	35.70	35.80	35.90	35.80	35.90	35.90	35.90	35.80	35.80	35.90	35.80	35.90	35.90	35.80	35.90	35.90	36.00	36.00	35.90	35.90	35.80	35.90	36.00	36.00	36.00
5HZ/.14G/TRE	TR6	37.00	37.10	37.10	37.10	37.20	37.30	37.30	37.30	37.30	37.40	37.40	37.50	37.50	37.60	37.60	37.60	37.70	37.70	37.70	37.70	37.80	37.80	37.80	37.80	37.80
10HZ/.18G/TSK	TS7	35.90	35.90	35.90	35.90	36.00	35.80	35.80	35.80	35.80	35.80	35.70	35.70	35.80	35.80	35.90	36.20	36.20	36.20	36.20	36.30	36.30	36.40	36.20	36.50	36.30
10HZ/.18G/TRE	TR7	36.90	36.90	36.90	36.90	37.00	37.10	37.20	37.20	37.20	37.20	37.30	37.30	37.30	37.40	37.50	37.40	37.50	37.50	37.50	37.50	37.40	37.40	37.60	37.50	37.50
16HZ/.28G/TSK	TS8	36.00	35.70	35.90	35.90	36.00	35.90	36.00	35.90	36.00	36.00	35.90	35.80	35.90	35.80	35.90	36.00	35.90	36.00	36.00	36.00	35.80	36.00	36.10	36.20	36.10
16HZ/.8G/TRE	TR8	37.00	37.00	37.00	37.00	37.10	37.20	37.20	37.20	37.30	37.40	37.50	37.50	37.70	37.80	37.70	37.70	37.80	37.70	37.80	37.80	37.70	37.40	37.50	37.60	37.50
30HZ/.55G/TSK	TS9	36.20	36.10	35.90	36.00	36.00	36.00	36.00	36.00	35.90	36.00	35.90	35.90	35.80	36.00	35.70	35.70	35.80	35.80	35.90	35.90	36.00	36.00	36.00	36.30	36.20
30HZ/.55G/TRE	TR9	37.00	37.00	37.10	37.10	37.20	37.30	37.30	37.40	37.40	37.50	37.60	37.70	37.80	37.80	37.70	37.80	37.80	37.80	37.80	37.80	37.60	37.70	37.70	37.80	37.80
80HZ/1.44G/TSK	TS10	36.00	36.10	36.00	35.80	35.90	35.80	35.80	35.80	35.90	35.80	35.90	35.90	35.80	35.90	35.90	36.00	36.00	36.00	36.10	36.10	36.00	36.30	36.30	36.20	36.20
80HZ/1.44G/TRE	TR10	37.10	37.10	37.20	37.20	37.30	37.30	37.40	37.40	37.50	37.50	37.60	37.60	37.60	37.70	37.70	37.70	37.80	37.80	37.90	37.90	37.80	37.80	37.70	37.70	37.80
S10HZ/.57G/TSK	TS11	35.00	35.00	35.20	35.10	35.00	35.10	35.20	35.30	35.30	35.30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	35.30	35.20	34.90	34.90	35.40	35.20
S10HZ/.57G/TRE	TR11	37.40	37.40	37.40	37.40	37.50	37.50	37.50	37.60	37.60	37.60	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	37.60	37.60	37.60	37.60	37.60	37.60
S25HZ/.90G/TSK	TS12	35.20	35.20	35.30	35.30	35.30	35.30	35.20	35.20	35.10	35.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	35.00	35.00	35.30	35.40	35.60	35.50
S25HZ/.90G/TRE	TR12	37.20	37.30	37.30	37.30	37.30	37.40	37.40	37.40	37.50	37.50	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	37.50	37.60	37.60	37.60	37.60	37.60
S60HZ/2.1G/TSK	TS13	35.50	35.40	35.40	35.40	35.50	35.40	35.40	35.40	35.20	35.40	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	35.40	35.20	35.50	35.30	35.60	35.60
S60HZ/2.1G/TRE	TR13	37.30	37.30	37.30	37.30	37.30	37.40	37.40	37.40	37.50	37.50	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	37.50	37.60	37.60	37.60	37.50	37.60
S125HZ/4.4G/TSK	TS14	35.40	35.30	35.80	35.60	35.30	35.30	35.30	35.30	35.30	35.40	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	35.40	35.30	35.40	35.50	35.50	34.90
S125HZ/4.4G/TRE	TR14	37.00	37.10	37.10	37.10	37.10	37.10	37.20	37.30	37.30	37.40	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	37.40	37.40	37.40	37.50	37.40	37.30
S250HZ/8.7G/TSK	TS15	35.50	35.50	35.60	35.20	35.40	35.40	35.40	35.50	35.40	35.30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	35.30	35.30	35.40	35.60	35.60	35.60
S250HZ/8.7G/TRE	TR15	37.30	37.30	37.20	37.30	37.30	37.40	37.40	37.40	37.40	37.40	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	37.40	37.40	37.50	37.60	37.50	37.60

APPENDIX XI

DATA FOR MEAN LOCALIZED SWEAT RATES

[$g/hr/cm^2$]

TEST CONDITION	LABEL	V-5	V0	V5	V10	V20	V25	V30	V40	V50	V60	V70	V80	V90	V100	V110	V120	V130	V140	V150	R0	R10	R20	R30	R40	R50
5HZ/.37G/FA	SR1	.58	.60	.64	.66	.53	.58	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.58	.61	.51	.57	.55	.49
5HZ/.37G/UA	SR2	.51	.51	.53	.53	.41	.35	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.35	.42	.36	.38	.41	.43
5HZ/.37G/C	SR3	.27	.22	.25	.26	.24	.24	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.24	.20	.19	.18	.25	.19
10HZ/.46G/FA	SR4	.46	.46	.47	.47	.46	.46	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.46	.49	.50	.51	.51	.50
10HZ/.46G/UA	SR5	.40	.41	.44	.44	.30	.29	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.29	.29	.30	.30	.31	.29
10HZ/.46G/C	SR6	.15	.16	.17	.18	.17	.21	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.21	.20	.21	.18	.16	.40
16HZ/.72G/FA	SR7	.50	.50	.51	.51	.50	.55	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.55	.50	.48	.57	.51	.53
16HZ/.72G/UA	SR8	.61	.60	.67	.69	.72	.70	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.70	.71	.72	.73	.74	.74
16HZ/.72G/C	SR9	.17	.18	.18	.19	.18	.21	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.21	.17	.16	.16	.15	.16
30HZ/1.4G/FA	SR10	.43	.46	.48	.49	.50	.48	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.48	.46	.48	.46	.49	.50
30HZ/1.4G/UA	SR11	.61	.64	.65	.64	.68	.77	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.77	.70	.69	.72	.74	.75
30HZ/1.4G/C	SR12	.13	.18	.18	.17	.17	.18	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.18	.14	.15	.16	.17	.17
80HZ/3.7G/FA	SR13	.41	.42	.45	.45	.45	.48	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.48	.46	.45	.47	.46	.47
80HZ/3.7G/UA	SR14	.50	.53	.56	.57	.60	.58	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.58	.60	.74	.77	.76	.76
80HZ/3.7G/C	SR15	.15	.15	.18	.18	.19	.19	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.19	.17	.19	.16	.16	.17
5HZ/.14G/FA	SR16	.51	.55	.53	.49	.53	.53	.54	.42	.51	.49	.52	.60	.60	.54	.53	.59	.51	.49	.51	.51	.66	.55	.55	.55	.57
5HZ/.14G/UA	SR17	.42	.43	.46	.42	.43	.40	.38	.44	.43	.40	.44	.48	.47	.46	.47	.47	.47	.46	.45	.45	.53	.47	.46	.49	.47
5HZ/.14G/C	SR18	.20	.19	.19	.19	.21	.20	.19	.19	.17	.18	.18	.21	.19	.19	.24	.20	.19	.19	.19	.19	.21	.19	.17	.18	.18
10HZ/.18G/FA	SR19	.54	.55	.55	.61	.58	.60	.62	.66	.59	.58	.70	.70	.60	.56	.56	.58	.56	.57	.56	.56	.57	.57	.57	.57	.58
10HZ/.18G/UA	SR20	.34	.34	.36	.42	.38	.39	.40	.46	.38	.38	.49	.49	.40	.41	.37	.42	.43	.41	.41	.41	.41	.44	.44	.40	.45
10HZ/.18G/C	SR21	.15	.15	.15	.20	.17	.17	.18	.19	.20	.19	.25	.25	.25	.18	.18	.19	.19	.19	.19	.19	.19	.19	.19	.20	.19
16HZ/.28G/FA	SR22	.48	.49	.50	.49	.63	.60	.58	.50	.50	.51	.57	.52	.53	.59	.53	.60	.60	.53	.52	.52	.57	.59	.55	.54	.66
16HZ/.28G/UA	SR23	.35	.32	.34	.32	.42	.39	.36	.35	.35	.35	.38	.42	.42	.47	.40	.43	.40	.37	.40	.40	.41	.40	.40	.39	.43
16HZ/.28G/C	SR24	.18	.18	.19	.18	.20	.20	.20	.19	.20	.19	.21	.18	.19	.23	.18	.21	.19	.22	.19	.19	.20	.20	.20	.19	.23
30HZ/.55G/FA	SR25	.53	.56	.56	.57	.57	.59	.62	.71	.66	.61	.58	.64	.63	.67	.64	.60	.65	.64	.65	.65	.76	.67	.72	.68	.66
30HZ/.55G/UA	SR26	.35	.34	.33	.38	.37	.38	.39	.43	.39	.38	.37	.38	.37	.40	.34	.41	.41	.39	.41	.41	.51	.41	.39	.44	.40
30HZ/.55G/C	SR27	.19	.22	.22	.22	.22	.22	.23	.26	.21	.22	.24	.22	.25	.22	.24	.22	.21	.22	.21	.21	.27	.20	.25	.23	.21
80HZ/1.44G/FA	SR28	.53	.54	.60	.62	.58	.60	.81	.70	.73	.72	.67	.65	.82	.64	.72	.78	.76	.95	.86	.86	.66	.74	.67	.89	.82
80/1.44G/UA	SR29	.31	.34	.33	.37	.37	.37	.50	.42	.44	.35	.38	.37	.38	.37	.40	.49	.48	.54	.49	.49	.36	.46	.42	.54	.59
80/1.44G/C	SR30	.20	.20	.22	.25	.23	.23	.35	.26	.29	.23	.24	.22	.21	.23	.30	.26	.33	.35	.33	.33	.23	.26	.29	.31	.34
S10HZ/.57G/FA	SR31	.44	.49	.51	.52	.56	.56	.54	.45	.54	.58	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.58	.59	.66	.60	.64	.52
S10HZ/.57G/UA	SR32	.30	.36	.44	.37	.42	.42	.37	.40	.40	.38	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.38	.42	.39	.38	.35	.31
S10HZ/.57G/C	SR33	.22	.20	.22	.19	.23	.23	.19	.14	.17	.17	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.17	.20	.20	.20	.18	.16
S25HZ/.90G/FA	SR34	.53	.62	.44	.51	.69	.69	.56	.59	.49	.48	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.48	.61	.51	.51	.54	.57
S25HZ/.90G/UA	SR35	.34	.37	.31	.36	.44	.44	.37	.48	.39	.42	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.42	.46	.38	.36	.38	.40
S25HZ/.90G/C	SR36	.24	.24	.18	.22	.31	.31	.27	.29	.25	.25	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.25	.26	.23	.23	.21	.32
S60HZ/2.1G/FA	SR37	.37	.32	.52	.49	.55	.55	.61	.57	.55	.53	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.53	.70	.54	.55	.54	.55
S60HZ/2.1G/UA	SR38	.30	.31	.35	.37	.39	.39	.44	.38	.44	.39	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.39	.40	.38	.37	.38	.40
S60HZ/2.1G/C	SR39	.22	.21	.23	.24	.25	.25	.30	.27	.30	.27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.27	.31	.30	.31	.30	.32
S125HZ/4.4G/FA	SR40	.52	.49	.52	.61	.51	.51	.55	.53	.52	.55	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.55	.67	.54	.54	.56	.59
S125/4.4G/UA	SR41	.37	.32	.36	.43	.38	.38	.38	.38	.37	.39	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.39	.46	.41	.42	.42	.43
S125HZ/4.4G/C	SR42	.23	.19	.22	.27	.21	.21	.21	.19	.19	.24	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.24	.27	.21	.21	.22	.23
S250HZ/8.7G/FA	SR43	.50	.50	.59	.62	.63	.63	.52	.50	.49	.64	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.64	.49	.53	.53	.52	.52
S250HZ/8.7G/UA	SR44	.28	.28	.33	.39	.41	.41	.38	.32	.30	.44	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.44	.29	.30	.28	.29	.30
S250HZ/8.7G/C	SR45	.24	.26	.24	.23	.26	.26	.21	.17	.25	.31	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.31	.18	.20	.24	.15	.20

APPENDIX XII

DATA FOR MEAN OF LIMB-SEGMENT BLOOD-PERFUSION RATES

[blood/100 ml tissue/min]

TEST CONDITION	LABEL	V-5	V0	V5	V10	V20	V25	V30	V40	V50	V60	V70	V80	V90	V100	V110	V120	V130	V140	V150	R0	R10	R20	R30	R40	R50
5HZ/.37G/UA	PF1	4.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	4.80	0.00	4.50	0.00	4.90
5HZ/.37G/FA	PF2	4.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	4.60	0.00	4.90	0.00	4.40
10HZ/.46G/UA	PF3	3.40	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	4.40	0.00	4.80	0.00	4.70
10HZ/.46G/FA	PF4	4.60	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	4.40	0.00	4.70	0.00	5.10
16HZ/.72G/UA	PF5	3.40	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	3.60	0.00	5.10	0.00	4.80
16HZ/.72G/FA	PF6	4.20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	3.90	0.00	4.50	0.00	6.10
30HZ/1.4G/UA	PF7	3.60	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	4.60	0.00	3.70	0.00	4.70
30HZ/1.4G/FA	PF8	3.20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	4.40	0.00	3.80	0.00	4.00
80HZ/3.7G/UA	PF9	4.80	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	4.90	0.00	4.90	0.00	5.50
80HZ/3.7G/FA	PF10	3.70	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	4.70	0.00	4.60	0.00	5.50
5HZ/.14/UA	PF11	5.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	6.00	0.00	5.90	0.00	6.20
5HZ/.14G/FA	PF12	3.90	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	5.70	0.00	5.30	0.00	6.00
10HZ/.18G/UA	PF13	4.90	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	5.00	0.00	4.30	0.00	5.80
10HZ/.18G/FA	PF14	5.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	4.20	0.00	4.30	0.00	6.00
16HZ/.28G/UA	PF15	4.20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	4.60	0.00	4.70	0.00	5.20
16HZ/.28G/FA	PF16	4.20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	4.70	0.00	4.40	0.00	5.20
30HZ/.55G/UA	PF17	4.40	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	4.70	0.00	4.90	0.00	4.80
30HZ/.55G/FA	PF18	4.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	5.60	0.00	5.80	0.00	5.60
80HZ/1.44G/UA	PF19	4.30	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	4.80	0.00	4.30	0.00	4.50
80HZ/1.44G/FA	PF20	3.80	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	5.10	0.00	4.10	0.00	5.30
S10HZ/.57G/FA	PF21	6.70	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	4.20	0.00	4.80	0.00	5.80
S25HZ/.90G/FA	PF22	5.60	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	5.90	0.00	5.40	0.00	4.90
S60HZ/2.1G/FA	PF23	5.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	5.40	0.00	6.70	0.00	5.10
S125HZ/4.4G/FA	PF24	5.80	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	4.50	0.00	4.90	0.00	5.40
S250HZ/8.7G/FA	PF25	5.50	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	4.40	0.00	4.90	0.00	5.30

APPENDIX XIII

DATA FOR MEAN RESPIRATION RATES

[breaths/min]

TEST CONDITION	LABEL	V-5	V0	V5	V10	V20	V25	V30	V40	V50	V60	V70	V80	V90	V100	V110	V120	V130	V140	V150	R0	R10	R20	R30	R40	R50
5HZ/.37G/RR	RR1	14.00	0.00	19.00	0.00	19.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	14.00	0.00	0.00	0.00
10HZ/.46G/RR	RR2	13.00	0.00	14.00	0.00	16.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	14.00	0.00	0.00	0.00
16HZ/.72G/RR	RR3	13.00	0.00	14.00	0.00	13.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	15.00	0.00	0.00	0.00
30HZ/1.4G/RR	RR4	11.00	0.00	12.00	0.00	15.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	12.00	0.00	0.00	0.00
80HZ/3.7G/RR	RR5	12.00	0.00	11.00	0.00	13.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	12.00	0.00	0.00	0.00
5HZ/.14G/RR	RR6	15.00	0.00	14.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	15.00	0.00	0.00	0.00	0.00	0.00	0.00	13.00	0.00	0.00	0.00	16.00	0.00	0.00	0.00
10HZ/.18G/RR	RR7	14.00	0.00	16.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	15.00	0.00	0.00	0.00	0.00	0.00	0.00	16.00	0.00	0.00	0.00	17.00	0.00	0.00	0.00
16HZ/.28G/RR	RR8	12.00	0.00	14.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	12.00	0.00	0.00	0.00	0.00	0.00	0.00	12.00	0.00	0.00	0.00	13.00	0.00	0.00	0.00
30HZ/.55G/RR	RR9	14.00	0.00	14.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	15.00	0.00	0.00	0.00	0.00	0.00	0.00	15.00	0.00	0.00	0.00	14.00	0.00	0.00	0.00
80HZ/1.44G/RR	RR10	15.00	0.00	15.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	15.00	0.00	0.00	0.00	0.00	0.00	0.00	14.00	0.00	0.00	0.00	15.00	0.00	0.00	0.00
S10HZ/.57G/RR	RR11	13.00	0.00	18.00	0.00	0.00	0.00	0.00	14.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	18.00	0.00	0.00	0.00
S25HZ/.90G/RR	RR12	17.00	0.00	16.00	0.00	0.00	0.00	0.00	15.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	17.00	0.00	0.00	0.00
S60HZ/2.1G/RR	RR13	16.00	0.00	16.00	0.00	0.00	0.00	0.00	16.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	18.00	0.00	0.00	0.00
S125HZ/4.4G/RR	RR14	17.00	0.00	17.00	0.00	0.00	0.00	0.00	17.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	19.00	0.00	0.00	0.00
S250HZ/8.7G/RR	RR15	18.00	0.00	22.00	0.00	0.00	0.00	0.00	22.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	19.00	0.00	0.00	0.00

APPENDIX XIV

DATA FOR MEAN OXYGEN UPTAKE

[liters/min]

TEST CONDITION	LABEL	V-5	V0	V5	V10	V20	V25	V30	V40	V50	V60	V70	V80	V90	V100	V110	V120	V130	V140	V150	R0	R10	R20	R30	R40	R50
5HZ/.37G/VO2	1VO2	.20	0.00	.45	0.00	.38	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.18	0.00	0.00	0.00
10HZ/.46G/VO2	2VO2	.19	0.00	.19	0.00	.29	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.23	0.00	0.00	0.00
16HZ/.72G/VO2	3VO2	.23	0.00	.22	0.00	.21	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.20	0.00	0.00	0.00
30HZ/1.4G/VO2	4VO2	.22	0.00	.23	0.00	.20	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.21	0.00	0.00	0.00
80HZ/3.7G/VO2	5VO2	.21	0.00	.22	0.00	.22	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.22	0.00	0.00	0.00
5HZ/.14G/VO2	6VO2	.20	0.00	.26	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.26	0.00	0.00	0.00	0.00	0.00	0.00	.30	0.00	0.00	0.00	.23	0.00	0.00	0.00
10HZ/.18G/VO2	7VO2	.21	0.00	.21	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.19	0.00	0.00	0.00	0.00	0.00	0.00	.31	0.00	0.00	0.00	.26	0.00	0.00	0.00
6HZ/.28G/VO2	8VO2	.22	0.00	.24	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.28	0.00	0.00	0.00	0.00	0.00	0.00	.27	0.00	0.00	0.00	.22	0.00	0.00	0.00
30HZ/.55G/VO2	9VO2	.22	0.00	.24	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.22	0.00	0.00	0.00	0.00	0.00	0.00	.29	0.00	0.00	0.00	.19	0.00	0.00	0.00
80HZ/1.44G/VO2	10VO2	.21	0.00	.27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.27	0.00	0.00	0.00	0.00	0.00	0.00	.21	0.00	0.00	0.00	.22	0.00	0.00	0.00
S10HZ/.57G/VO2	11VO2	.24	0.00	.26	0.00	0.00	0.00	0.00	.24	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.27	0.00	0.00	0.00
S25HZ/.90G/VO2	12VO2	.22	0.00	.31	0.00	0.00	0.00	0.00	.31	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.29	0.00	0.00	0.00
S60HZ/2.1G/VO2	13VO2	.22	0.00	.33	0.00	0.00	0.00	0.00	.24	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.30	0.00	0.00	0.00
S125HZ/4.4G/VO2	14VO2	.24	0.00	.27	0.00	0.00	0.00	0.00	.27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.22	0.00	0.00	0.00
S250HZ/8.7G/VO2	15VO2	.23	0.00	.25	0.00	0.00	0.00	0.00	.29	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	.28	0.00	0.00	0.00

APPENDIX XV

DATA FOR MEAN RESPIRATORY EXCHANGE RATIOS

TEST CONDITION	V_{-5}	V_5	V_{20}	V_{40}	V_{70}	V_{140}	R_{20}
HIGH INTENSITY							
5 Hz/0.37 g	0.73 ± 0.02	0.69 ± 0.06	0.63 ± 0.06	–	–	–	0.74 ± 0.02
10 Hz/0.46 g	0.77 ± 0.03	0.74 ± 0.05	0.75 ± 0.04	–	–	–	0.73 ± 0.03
16 Hz/0.72 g	0.81 ± 0.02	0.82 ± 0.04	0.78 ± 0.02	–	–	–	0.76 ± 0.02
30 Hz/1.4 g	0.76 ± 0.02	0.75 ± 0.02	0.76 ± 0.02	–	–	–	0.76 ± 0.02
80 Hz/3.7 g	0.78 ± 0.03	0.79 ± 0.02	0.76 ± 0.02	–	–	–	0.73 ± 0.02
LOW INTENSITY							
5 Hz/0.14 g	0.71 ± 0.05	0.69 ± 0.05	–	–	0.58 ± 0.10	0.69 ± 0.08	0.68 ± 0.06
10 Hz/0.18 g	0.82 ± 0.07	0.79 ± 0.08	–	–	0.65 ± 0.10	0.67 ± 0.06	0.69 ± 0.07
16 Hz/0.28 g	0.75 ± 0.08	0.73 ± 0.07	–	–	0.63 ± 0.07	0.71 ± 0.06	0.69 ± 0.06
30 Hz/0.55 g	0.82 ± 0.16	0.81 ± 0.15	–	–	0.69 ± 0.06	0.80 ± 0.12	0.79 ± 0.12
80 Hz/1.4 g	0.70 ± 0.06	0.70 ± 0.06	–	–	0.69 ± 0.06	0.67 ± 0.04	0.68 ± 0.06
SEGMENTAL							
10 Hz/0.57 g	0.76 ± 0.02	0.74 ± 0.02	–	0.74 ± 0.02	–	–	0.77 ± 0.05
25 Hz/0.90 g	0.75 ± 0.03	0.76 ± 0.04	–	0.74 ± 0.04	–	–	0.71 ± 0.02
60 Hz/2.1 g	0.71 ± 0.01	0.72 ± 0.01	–	0.71 ± 0.03	–	–	0.70 ± 0.02
125 Hz/4.4 g	0.83 ± 0.04	0.81 ± 0.04	–	0.79 ± 0.02	–	–	0.79 ± 0.03
250 Hz/8.7 g	0.80 ± 0.02	0.78 ± 0.01	–	0.80 ± 0.04	–	–	0.79 ± 0.02

APPENDIX XVI

DATA FOR MEAN BLOOD COMPONENT COUNTS AND CONCENTRATIONS

	HEMOGLOBIN, gm	HEMATOCRIT, %	OSMOLALITY, mosm/Kg	SODIUM, meq/ℓ	POTASSIUM, meq/ℓ	POLYMORPHO-NEUTROPHILS	LYMPHO-CYTES	MONO-CYTES	BASOPHILS	EOSINOPHILS
PHII, SECA										
5 Hz (PRE)	17.2 ± 0.2	47.4 ± 0.9	291 ± 1	142.4 ± 0.7	4.55 ± 0.24	61 ± 3	33 ± 4	1.8 ± 1.0	0.4 ± 0.2	3 ± 1
(POST)	17.2 ± 0.2	47.3 ± 0.6	295 ± 2	144.8 ± 0.8	4.64 ± 0.22	61 ± 4	34 ± 4	2.0 ± 0.7	0.6 ± 0.2	2 ± 1
16 Hz (PRE)	16.8 ± 0.5	46.7 ± 1.4	292 ± 3	141.2 ± 1.2	4.78 ± 0.31	59 ± 2	38 ± 3	1.4 ± 0.5	0.8 ± 0.5	1 ± 0
(POST)	16.8 ± 0.5	46.7 ± 1.3	294 ± 2	142.9 ± 1.1	4.58 ± 0.25	59 ± 3	37 ± 3	2.0 ± 1.0	0	2 ± 1
80 Hz (PRE)	16.4 ± 0.4	45.9 ± 1.2	289 ± 2	141.8 ± 0.8	4.41 ± 0.20	56 ± 2	39 ± 2	3.0 ± 1.2	0	2 ± 1
(POST)	16.5 ± 0.5	45.9 ± 1.2	290 ± 1	141.4 ± 0.9	4.46 ± 0.28	60 ± 2	36 ± 2	2.0 ± 0.5	0	3 ± 1
PHII, SECB										
5 Hz (PRE)	16.6 ± 0.4	45.7 ± 0.8	287 ± 2	141.6 ± 0.9	4.53 ± 0.18	60 ± 3	36 ± 3	1.4 ± 0.6	0	2 ± 1
(POST)	17.1 ± 0.6	47.0 ± 1.0	291 ± 1	143.3 ± 0.6	4.38 ± 0.13	62 ± 2	34 ± 2	2.6 ± 0.6	0	1 ± 1
16 Hz (PRE)	16.4 ± 0.4	45.2 ± 0.6	289 ± 1	142.7 ± 0.8	4.80 ± 0.14	58 ± 4	37 ± 4	2.0 ± 0.7	1.0 ± 1.0	2 ± 1
(POST)	16.8 ± 0.3	45.8 ± 0.5	291 ± 2	142.9 ± 0.8	4.49 ± 0.14	63 ± 4	32 ± 4	4.0 ± 1.0	0.4 ± 0.3	2 ± 1
80 Hz (PRE)	16.2 ± 0.4	44.8 ± 1.1	291 ± 1	141.3 ± 0.4	4.41 ± 0.28	62 ± 3	35 ± 4	2.0 ± 0.8	0	1 ± 1
(POST)	16.7 ± 0.4	45.4 ± 1.0	292 ± 1	143.0 ± 0.3	4.38 ± 0.21	61 ± 4	36 ± 5	2.0 ± 0.5	0	1 ± 1
PHIII										
10 Hz (PRE)	17.6 ± 0.6	47.6 ± 1.1	286 ± 2	139.5 ± 1.0	4.47 ± 0.28	63 ± 2	32 ± 2	1.0 ± 0.5	0	4 ± 2
(POST)	17.8 ± 0.5	48.0 ± 1.0	291 ± 1	142.1 ± 0.6	4.42 ± 0.21	62 ± 4	32 ± 2	3.0 ± 1.0	0	3 ± 1
60 Hz (PRE)	16.8 ± 0.5	46.3 ± 1.2	285 ± 1	141.0 ± 0.4	4.56 ± 0.13	61 ± 1	35 ± 1	2.0 ± 1.0	0	1 ± 1
(POST)	16.9 ± 0.6	46.5 ± 1.3	292 ± 1	142.3 ± 0.8	4.45 ± 0.16	64 ± 2	33 ± 3	1.0 ± 0.8	0	1 ± 1
250 Hz (PRE)	16.2 ± 0.3	45.3 ± 0.6	286 ± 1	140.0 ± 0.4	4.50 ± 0.19	63 ± 3	31 ± 3	3.0 ± 0.8	1.0 ± 1.0	2 ± 1
(POST)	16.2 ± 0.6	45.4 ± 0.9	291 ± 1	142.1 ± 0.4	4.31 ± 0.12	65 ± 1	31 ± 1	3.0 ± 0.7	0	1 ± 1

APPENDIX XVII

DATA FOR MEAN BOOTH TEMPERATURES [°C]

TEST CONDITION	LABEL	V-5	V0	V5	V10	V20	V25	V30	V40	V50	V60	V70	V80	V90	V100	V110	V120	V130	V140	V150	R0	R10	R20	R30	R40	R50
5HZ/.37G/TB	TB1	43.20	43.40	43.50	43.50	43.50	43.70	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	43.70	43.10	43.40	43.50	43.40	43.80
10HZ/.46G/TB	TB2	43.60	43.40	43.40	43.50	43.60	43.50	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	43.50	43.50	43.50	43.50	43.60	43.60
16HZ/.72G/TB	TB3	43.20	43.20	43.40	43.60	43.50	43.40	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	43.40	43.10	43.30	43.70	43.30	43.40
30HZ/1.4G/TB	TB4	43.50	43.40	43.40	43.50	43.60	43.60	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	43.60	43.60	43.40	43.60	43.60	43.60
80HZ/3.7G/TB	TB5	43.20	43.30	43.40	43.60	43.20	43.50	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	43.50	43.30	43.30	43.60	43.60	43.50
5HZ/.14G/TB	TB6	43.30	43.00	43.30	43.40	43.90	43.60	43.30	43.50	43.40	43.70	43.50	43.30	43.50	43.40	43.40	43.50	43.20	43.70	43.30	43.30	43.30	43.50	43.40	43.50	43.60
10HZ/.18G/TB	TB7	43.30	43.20	43.30	43.60	43.80	43.60	43.30	43.50	43.60	43.40	43.50	43.60	43.50	43.60	43.50	43.60	43.30	43.60	43.60	43.60	43.40	43.60	43.70	43.60	43.50
16HZ/.28G/TB	TB8	42.90	42.50	43.10	42.80	43.50	43.40	43.30	43.30	43.40	43.50	43.50	43.50	43.40	43.50	43.50	43.50	43.50	43.70	43.40	43.40	42.80	43.30	43.60	43.50	43.50
30HZ/.55G/TB	TB9	43.30	43.30	43.00	43.00	43.40	43.50	43.50	43.60	43.10	43.30	43.70	43.60	43.30	43.20	43.50	43.20	43.90	43.20	43.20	43.60	43.40	43.60	43.70	43.50	43.50
80HZ/1.44G/TB	TB10	43.10	43.00	43.40	43.10	43.40	43.30	43.30	43.40	43.50	43.60	43.60	43.50	43.50	43.60	43.10	43.60	43.10	43.70	43.60	43.60	42.90	43.50	43.50	43.20	43.20
S10HZ/.57G/TB	TB11	41.70	41.00	41.20	43.50	43.90	42.60	43.60	43.20	43.50	42.60	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	43.60	43.30	43.20	43.80	43.80	43.20
S25HZ/.90G/TB	TB12	42.60	43.00	42.90	43.30	43.70	43.50	43.30	43.40	42.90	43.90	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	43.90	43.40	43.70	43.90	43.70	43.40
S60HZ/2.1G/TB	TB13	42.50	42.60	42.60	43.10	43.40	43.80	44.20	43.60	43.00	43.70	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	43.70	42.70	43.80	43.30	43.80	43.10
S125HZ/4.4G/TB	TB14	43.30	42.40	43.60	43.10	43.40	43.50	42.90	43.50	43.10	42.60	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	42.60	43.50	43.10	43.30	43.60	43.40
S250HZ/8.7G/TB	TB15	43.10	43.20	43.60	42.90	43.70	43.40	43.00	43.50	43.70	43.80	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	43.80	43.20	43.40	43.70	43.70	43.70

REFERENCES

1. Kerslake, D. McK.: The Stress of Hot Environments: Monographs of the Physiological Society, No. 29, University Printing House, Cambridge University, Cambridge, England, 1972, pp. 12-14.
2. Baxett, H. C.: Theory of Reflex Controls to Explain Regulation of Body Temperature at Rest and during Exercise. J. Appl. Physiol., vol. 4, 1951, pp. 245-262.
3. Collins, K. J.; Sargent, F.; and Weiner, J. S.: The Effect of Arterial Occlusion on Sweat-Gland Responses in the Human Forearm. J. Physiol., Lond., vol. 148, 1951, pp. 615-624.
4. Albert, R. E.: Evaporative Rate Patterns from Small Skin Areas as Measured by an Infrared Gas Analyzer. J. Appl. Physiol., vol. 4, no. 3, Sept. 1951, pp. 208-214.
5. Robertshaw, D., ed.: International Review of Physiology — Environmental Physiology II, vol. 15. University Park Press, Baltimore, 1977, p. 85.
6. Konz, S.; and Duncan, J.: Cooling with a Water-Cooled Hood. Proc. Symp. Ind. Cooling, Kansas State U., Manhattan, Kan., 1969.
7. Nunneley, S. A.; Troutman, S. J., Jr.; and Webb, P.: Head Cooling in Work and Heat Stresses. Aerosp. Med., vol. 42, 1971, pp. 64-68.
8. Forster, R. E.; Ferris, B. G.; and Day, R.: The Relationship between Total Heat Exchange and Blood Flow in the Hand at Various Ambient Temperatures. Am. J. Physiol., vol. 146, 1946, p. 600.
9. Gold, A. J.; and Zornitzer, A.: Effect of Partial Body Cooling on Man Exercising in a Hot Environment. Aerosp. Med., vol. 39, 1968, pp. 944-946.

10. Shvartz, E.: Effect of Neck versus Chest Cooling on Responses to Work in Heat. J. Appl. Physiol., vol. 40, no. 5, 1976, pp. 668-672.

11. Strydom, N. B.; Benade, A. J. S.; Blom, P. van A.; and Walt, W. H. V. D.: The Performance of the Improved Microclimate Suite. Research Report No. 32/72, Chamber of Mines of South Africa, Johannesburg, South Africa, 1972.

12. Love, L. H.: Heat Loss and Blood Flow of the Feet under Hot and Cold Conditions. J. Appl. Physiol., vol. 1, no. 1, July 1948, pp. 20-34.

13. Banaszkiewcz, T.; Gwozdziewicz, J.; and Waskiewicz, J.: Appliance of the Functional Test for Investigations on the Skin Surface Temperature at Impending Vibration Disease. Bull. of the Instit. of Marine Medicine in Gdansk. XXI, vol. 3, no. 4, 1970, pp. 147-162.

14. Grether, W. F.; Harris, C. S.; Ohlbaum, M.; Sampson, P. A.; and Guignard, J. C.: Further Study of Combined Heat, Noise, and Vibration Stress. Aerosp. Med., vol. 43, no. 6, June 1972, pp. 641-645.

15. Holland, C. L.: Performance Effects of Long Term Random Vertical Vibration. Human Factors, vol. 92, Apr. 1967, pp. 93-104.

16. Guignard, J. C.: The Physical Response of Seated Men to Low-Frequency Vertical Vibration: Preliminary Studies. Report of the Flying Personnel Research Committee, Inst. Aviation Med., Farnborough, Eng., 1959.

17. Loeb, M. A.: Further Investigation of the Influence of Whole Body Vibration and Noise on Tremor and Visual Activity. Report No. 165, AMRL, Fort Knox, Ky., 1955.

18. Dennis, J. P.: The Effect of Whole Body Vibration on a Visual Performance Task. Ergonomics, vol. 8, 1967, pp. 193-205.

19. Shoenberger, R. W.: Investigation of the Effects of Vibration on Dial Reading Performance with a NASA Prototype Apollo Helmet. AMRL-TR-205, Wright-Patterson Air Force Base, Ohio, 1968.

20. Grether, W. F.; Harris, C. S.; et al.: Effects of Combined Heat, Noise, and Vibration Stress on Human Performance and Physiological Functions. Aerosp. Med., vol. 42, no. 10, Oct. 1971, pp. 1092-1097.

21. Megel, H.; Wozniak, H.; Sun, L.; Frazier, E.; and Mason, H. C.: Effects on Rats of Exposure to Heat and Vibration. J. Appl. Physiol., vol. 17, no. 5, 1962, pp. 759-762.

22. Megel, H.; Keating, F. M.; and Stern, J.: Effect of Elevated Ambient Temperature and Vibration upon the Rectal Temperature of the Restrained Rat. Aerosp. Med., vol. 32, 1961, p. 1135.

23. Rushmer, R. F.; Baker, D. W.; and Stega, H. F.: Transcutaneous Doppler Flow Detection as a Nondestructive Technique. J. Appl. Physiol., vol. 21, no. 2, 1966, pp. 554-566.

24. Brakkee, A. J. M.; and Vendrik, A. J. H.: Strain-Gauge Plethysmography: Theoretical and Practical Notes on a New Design. J. Appl. Physiol., vol. 21, no. 2, 1966, pp. 701-704.

25. Stegall, H. F.; Martin, W. E.; and Rushmer, R. F.: A Simple Method for Cuff Inflation in Plethysmography. J. Appl. Physiol., vol. 21, no. 2, 1966, p. 700.

26. Banerjee, M.; Elizondo, R.; and Bullard, R.: Reflex Responses of Human Sweat Glands to Different Rates of Skin Cooling. J. Appl. Physiol., vol. 26, 1969, p. 787.

27. Elizondo, R. S.; and Bullard, R. W.: Local Determinants of Sweating and the Assessment of Set Point. Int. J. Biometerol., vol. 15, 1971, p. 259.

28. Robertshaw, D., ed.: International Review of Physiology, vol. 15. Environmental Physiology II., University Park Press, Baltimore, 1977, pp. 99, 103, 108.

29. Guide for the Evaluation of Human Exposure to Whole Body Vibration. ISO 2631-1974, International Organization for Standardization, ISO, Geneva, 1974.

30. Dieckmann, D. A.: Study of the Influence of Vibration on Man. Ergonomics, vol. 1, 1958, pp. 346-355.

31. Radke, A. O.: Vehicle Vibration Man's New Environment. Paper No. 57-A-54, American Society of Mechanical Engineers, 1957.

32. Goldman, D. E.; and Von Gierke, H. E.: Effects of Shock and Vibration on Man. Naval Medical Research Institute, Bethesda, Md., January 8, 1960 as No. 60-3, Lecture and Review Series.

33. Threshold Limit Values for Chemical Substances and Physical Agents in the Work Environment with Intended Changes for 1982. American Conference of Governmental Industrial Hygienists, Cincinnati, Ohio, 1982.

34. Lind, A. R.; and Bass, D. E.: The Optimal Exposure Time for the Development of Acclimation to Heat. Fed. Proc., vol. 22, 1963, p. 704.

35. Hardy, J. D.; and Dubois, E. F.: Basal Metabolism, Radiation, Convection and Vaporization at Temperatures of 22 to 35 °C. J. Nutr., vol. 15, 1938, pp. 477-497.

36. Gagge, A. P.: Standard Operative Temperature, a Generalized Temperature Scale, Applicable to Direct and Partitional Calorimetry. Am. J. Physiol., vol. 131, 1940, pp. 93-103.

37. Greenleaf, J. E.; Montgomery, L. D.; Brock, P. J.; and Van Beaumont, W.: Limb Blood Flow: Rest and Heavy Exercise in Sitting and Supine Positions in Man. Aviat. Space Environ. Med., vol. 50, no. 7, 1979, pp. 702-707.

38. Scholander, P. F.: Analyzer for Accurate Estimation of Respiratory Gases in One-Half Cubic Centimeter Samples. J. Biol. Chem., vol. 167, 1947, pp. 235-250.

39. Montgomery, L.: Comparison of an Impedance Device to a Displacement Plethysmography for Study of Finger Blood. Aviat. Space Environ. Med., vol. 47, 1976, pp. 33-38.

40. Montgomery, L.: Quantitative Values of Blood Flow through the Human Forearm, Hand, and Finger as Functions of Temperature. NASA TM X-62,342, 1974.

41. Wong, N.; Silver, J. E.; et al.: Effect of Arm Exercises on Venous Blood Constituents during Leg Exercises. (Abstract) The Physiologist, vol. 25, 1982, p. 199.

42. Benzinger, T. H.: On Physical Heat Regulation and the Sense of Temperature in Man. Proc. Nat. Acad. Sci., vol. 45, 1959, p. 645.

43. Henane, R.; and Bittel, J.: Changes of Thermal Balance Induced by Passive Heating in Resting Man. J. Appl. Physiol., vol. 38, no. 2, 1975, pp. 294-299.

44. Givoni, B.; and Goldman, R.: Predicting Effects of Heat Acclimation on Heart Rate and Rectal Temperature. J. Appl. Physiol., vol. 35, no. 6, 1973, pp. 875-879.

45. Givoni, B.; and Goldman, R.: Predicting Heart Rate Response to Work Environment and Clothing. J. Appl. Physiol., vol. 34, no. 2, 1973, pp. 201-204.

46. Macpherson, R. K.: Physiological Responses to Hot Environments. Med. Res. Council Spec. Rept. Series 298, H. M. Stationery Office, London, 1960.

47. Adolf, E. F.: Physiology of Man in the Desert. Interscience Publishers, Inc., New York, 1947.

48. Mosteller, F.; and Rourke, R.: Study Statistics, Chap. 5, Addison-Wesley, Reading, Mass., 1973, pp. 89-101.

49. Bullard, R. W.; Banjeree, M. R.; and Mac Intyre, B. A.: The Role of the Skin in Negative Feedback Regulation of Eccrine Sweating. Int. J. Bioclim. Biomet., vol. 11, 1967, pp. 93-104.

50. Libert, J. P.; Candas, V.; and Vogt, J. J.: The Effect of Rate of Change in Skin Temperature on Local Sweating Rate. J. Appl. Physiol: Respirat. Environ. Exercise Physiol., vol. 47, no. 2, 1979, pp. 306-311.

51. Hyvarinen, J.; Pyykko, I.; and Sundberg, S.: Vibration Frequencies and Amplitudes in the Aetiology of Traumatic Vasospastic Disease. Lancet, vol. 1, 1973, pp. 791-794.

52. Pyykko, I.: A Physiological Study of the Vasoconstrictor Reflex in Traumatic Vasospastic Disease. Work Environ. Health, vol. 11, 1974, pp. 170-186.

53. Nasu, Y.: Changes of the Skin Temperature Caused by Local Vibratory Stimulation in Normals and Patients with Vibration Syndrome. Yonago Acta Medica., vol. 21, no. 2, 1977, pp. 83-99.

54. Pyykko, I.; and Hyvarinen, J.: The Physiological Basis of the Traumatic Vasospastic Disease: A Sympathetic Vasoconstrictor Reflex Triggered by High Frequency Vibration. Work Environ. Health, vol. 10, 1973, pp. 36-47.

55. Gordon, C. J.; and Heath, J.: Reassessment of Neural Control of Body Temperature: Importance of Oscillating Neural and Motor Components. Comp. Biochem. Physiol., vol. 74A, no. 3, 1983, pp. 479-489.

56. Johnson, J. M.; and Park, M. G.: Effect of Heat Stress on Cutaneous Vascular Responses to the Initiation of Exercise. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol., vol. 53, No. 3, 1982, pp. 744-749.

57. Rowell, L. B.: Competition between Skin and Muscle for Blood Flow during Exercise. Problems with Temperature Regulation during Exercise, E. R. Nadel, ed., Academic Press, New York, 1977, pp. 49-76.

1. Report No.	2. Government Accession No.	3. Recipient's Catalog No.
NASA TM-84400		

4. Title and Subtitle	5. Report Date
THE PHYSIOLOGICAL EFFECTS OF SIMULTANEOUS EXPOSURES TO HEAT AND VIBRATION	September 1983
	6. Performing Organization Code
7. Author(s)	8. Performing Organization Report No.
Wil Aaron Spaul	A-9458
	10. Work Unit No. T-6384
9. Performing Organization Name and Address	11. Contract or Grant No.
Ames Research Center Moffett Field, California 94035	
	13. Type of Report and Period Covered
12. Sponsoring Agency Name and Address	Technical Memorandum
National Aeronautics and Space Administration Washington, D.C. 20546	14. Sponsoring Agency Code 199-20-62-06

15. Supplementary Notes

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16. Abstract

The purpose of this study was to determine if exposure to vibration has an effect on the body's ability to handle heat stress, and, if so, to identify the specific vibration parameters (frequency and intensity) for both whole-body (wbv) and segmental-body vibration (sbv) that would have the most detrimental effect on the body's ability to maintain thermal homeostasis.

Rectal and skin temperatures, heart rates, localized sweat rates, arm-segment blood perfusion rates, respiration rates, oxygen uptakes, and respiratory exchange ratios were measured in six men (22-33 yr) during simultaneous exposures to heat and vibration – either wbv or sbv, and during a heated 50-min recovery period. The heat conditions were T_{db} = 43.5 ± 0.5°C (mean ± S.E.M.), and RH = 20 ± 4%. All vibration exposures were preceded by 1 hr of heat exposure. The seated, Z-axis, sinusoidal wbv exposures were divided into two exposure conditions – identical frequencies but at a high intensity (HI) and a low intensity (LI) level. The HI wbv exposure was for 25 min/day at 5 Hz, 0.37 g-rms; 10 Hz, 0.46 g-rms; 16 Hz, 0.72 g-rms; 30 Hz, 1.40 g-rms; 80 Hz, 3.70 g-rms. The LI wbv exposure was for 2.5 hr/day at the same frequencies but at the following accelerations: 0.14 g-rms; 0.18 g-rms; 0.28 g-rms; 0.55 g-rms; 1.44 g-rms. During the sbv the subject stood and grasped a vibrating, in the Z-axis, hand grip with both hands. The sbv exposures were for 1 hr/day and were 10 Hz, 0.57 g-rms; 25 Hz, 0.90 g-rms; 60 Hz, 2.10 g-rms; 125 Hz, 4.4 g-rms; 250 Hz, 8.70 g-rms.

Although all wbv test frequencies resulted in significantly elevated rectal temperatures, $p(t) < 0.05$, the lower frequencies (5, 10, and 16 Hz) appear to reduce the arm-segment blood perfusion rates (vasoconstriction) and localized sweat rates more than frequencies above 30 Hz. At the lower wbv frequencies (5, 10, and 16 Hz), intensity levels appear to have little effect except at 5 Hz. For the sbv, no heat stress conditions (elevated resting heart rate and rectal temperature) occurred. After a 50-min heated recovery period, return to pre-vibration exposure levels did not occur for the wbv, and only partially for the sbv frequencies.

17. Key Words (Suggested by Author(s))	18. Distribution Statement
Heat exposure Vibration parameters Physiology Multi-stressors	Unlimited Subject Category: 52

19. Security Classif. (of this report)	20. Security Classif. (of this page)	21. No. of Pages	22. Price*
Unclassified	Unclassified	184	A05

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