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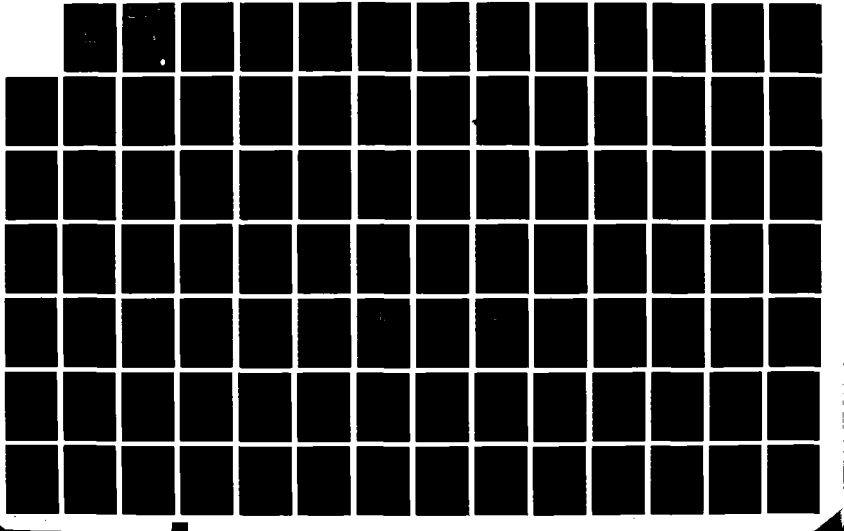

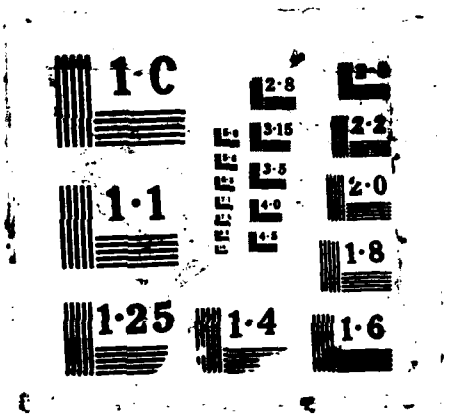
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MICROWAVE CHALLENGES TO THE THERMOREGULATORY SYSTEM

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Three experiments were conducted to measure the upper tolerance limit for maintaining thermal balance in squirrel monkeys during exposure to 2450-MHz radiofrequency radiation (RFR). During microwave exposure at high intensity, thermoregulatory behavior, metabolic, vasomotor, and sudomotor responses were mobilized in normal fashion, but the capacity to lose heat through sweating was found to be the limiting factor in microwave tolerance. The major conclusion drawn from these studies is that the thermoregulatory system deals with energy absorbed from microwave fields in exactly the same way as energy produced in the body by normal metabolic processes or absorbed during exposure to conventional radiant or convective heat sources.

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SUMMARY

Exposure to low-intensity radiofrequency (RF) fields influences the normal responses, both autonomic and behavioral, that regulate the body temperature. In the past, we have used the squirrel monkey as an animal model to determine the minimal incident energy (in mW/cm^2) derived from 2450-MHz continuous wave (CW) microwaves that is necessary to lower an elevated metabolic heat production in the cold, alter peripheral vasomotor tone in thermoneutral environments, initiate sweating in the heat, and alter thermoregulatory behavior. In all cases, the threshold power density is remarkably similar (4-8 mW/cm^2), a finding that suggests a common thermal basis for the response changes. The whole-body specific absorption rate (SAR) at threshold is equivalent to 15-20% of the resting metabolic heat production (M) of the subject animal. Above the threshold level, at least to SARs equivalent to two-thirds resting M, the mobilized thermoregulatory responses ensure that the body temperature is regulated with precision at the normal level and the magnitude of the response change is usually a linear function of field strength. The three experiments reported here sought to determine the upper limit of these functions, that is, the ceiling intensity that can be effectively dealt with by normal thermoregulatory processes.

Experiment 1 measured changes in ambient temperature (T_a) selected by highly trained squirrel monkeys during both transient (10 min) and steady-state (90 min) whole-body microwave exposures at high power densities (up to 70 mW/cm^2). During transient exposures, all power densities tested were countered by behavioral selection of a cooler environment so that the body temperatures were regulated at close to the normal level; 45 mW/cm^2 was similarly well tolerated by all animals in the steady-state. Thus, no upper tolerance limit was found for behavioral thermoregulation.

Experiment 2 assessed the potential for disruption of autonomic thermoregulation by an altered metabolic state (induced by injections of isoproterenol) in squirrel monkeys exposed to microwaves in a thermoneutral environment. We found that, unlike shivering thermogenesis, chemically mediated nonshivering thermogenesis was unaffected by microwave exposure. Instead, the elevated energy production/absorption in the body, coupled with partially disabled heat

loss responses, produced an exaggerated rise in body temperature. Thus, an endothermic organism whose thermoregulatory system is compromised may be at a disadvantage, in terms of its ability to regulate the body temperature, during exposure to thermalizing microwave fields.

Experiment 3 determined, using the method of partitional calorimetry, the maximal microwave power density (range = 10 to 60 mW/cm^2) tolerated by squirrel monkeys equilibrated to cool (20 °C) and neutral (26 °C) environments (T_a). Thermal balance was maintained at a power density up to 60 mW/cm^2 at T_a = 20 °C and 45 mW/cm^2 at T_a = 26 °C. These points, together with that (20 mW/cm^2) already determined for T_a = 32 °C, describe a curvilinear function that can be understood in terms of the thermoregulatory capabilities of the subject animal. During microwave exposure at high intensity, metabolic, vasomotor, and sudomotor responses were mobilized in normal fashion but the capacity to lose body heat through sweating was found to be the limiting factor in microwave tolerance.

The major conclusion drawn from these studies is that the thermoregulatory system deals with energy absorbed from microwave fields in exactly the same way as energy produced in the body by normal metabolic processes or absorbed during exposure to conventional radiant or convective heat sources.

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MICROWAVE CHALLENGES TO THE THERMOREGULATORY SYSTEM

INTRODUCTION

Those "warm-blooded" organisms that are capable of maintaining a stable internal body temperature in the face of rather wide fluctuations in the thermal characteristics of the environment are technically known as endotherms. Two classes of responses, behavioral and autonomic, accomplish thermoregulation in endotherms: behavioral maneuvers provide a thermally hospitable microclimate for the organism while fine adjustments in appropriate autonomic response systems adjust the rate of heat gain or heat loss from the body. If the behaviorally generated microclimate is thermally neutral, autonomic mechanisms will be minimally involved and the economy of energy and fluid stores in the body will be maximized. Thus, the description of thermoregulation in any endotherm involves detailed knowledge of thermoregulatory behavior, both instinctive and learned, and of individual autonomic processes of heat production and heat loss.

It is possible to restrain an experimental subject so that behavioral thermoregulatory responses are minimized or eliminated, thereby permitting measurement of autonomic thermoregulatory responses in nearly pure form. However, the reverse is not possible, i.e., pure behavioral responses cannot be measured in endothermic subjects because their autonomic or reflexive responses cannot be suppressed. The intimate interaction between the two categories of thermoregulatory response indicates the necessity of assessing both categories whenever the body is challenged by thermal changes in the environment or by changes in energy production within the body, such as occurs during physical exercise, fever, or exposure to nonionizing electromagnetic radiation.

Exposure of endothermic organisms to radiofrequency (RF) or microwave fields at intensities above a few mW/cm^2 has been demonstrated to mobilize all of the physiological and behavioral mechanisms that regulate the body temperature (1,4,6-8,13,18,20,21,28,35,42-45,51,52,55). Response thresholds, specified in terms of power density or whole-body specific absorption rate (SAR),

can be determined for each thermoregulatory mechanism: metabolic heat production, vasomotion, sudomotion, and thermoregulatory behavior. Specific threshold values have been shown to depend on many variables including species, RF frequency and polarization, exposure duration, and ambient temperature (T_a).

Over the past several years, we have used the squirrel monkey as an animal model to investigate the minimal energy derived from 2450-MHz continuous-wave (CW) microwaves that will lower an elevated metabolic heat production ($\underline{M}$) in cold environments, initiate vasodilation of peripheral blood vessels of the tail and foot in thermoneutral environments, stimulate thermoregulatory sweating in warm environments, and cause a behaving animal to select a cooler environment. In all cases, the threshold power densities have been found to be remarkably similar, 4 to 8 mW/cm^2, a level that represents a rate of energy absorption that is equivalent to 15 to 20% of the resting metabolic heat production of the squirrel monkey. Partial-body microwave exposures (head only or trunk only) produce appropriate adjustments in thermoregulatory responses to a degree nominally proportional to the percentage of the body so exposed. Nearly always, thermoregulatory responses are mobilized so quickly and efficiently that the animal's internal body temperature, including the temperature of the brain, is regulated with precision at the normal level.

An important question concerns the role of the preoptic/anterior hypothalamic (PO/AH) area of the brainstem in the mobilization of thermoregulatory responses by exposure to microwaves. This thermosensitive area of the central nervous system (CNS) is generally considered to be the location of the "central thermostat" for the regulation of the body temperature in endotherms. When a squirrel monkey is exposed at a frequency of 2450 MHz, there is the potential for selective focussing of absorbed energy near the center of the head where the PO/AH is located. Experiments that involved independent control of PO/AH temperature, in addition to its measurement, revealed that, while small temperature increments in this area are involved in the initiation of thermoregulatory responses, thermal changes in the body as a whole are of far greater importance to long-term thermoregulation during exposure to microwaves (3,5,11). A particular finding in these experiments was that a PO/AH temperature rise of 0.2-0.3 °C, accompanying microwave exposure, appears to be necessary and sufficient to alter thermoregulatory behavior. The behavioral change

ensures, in turn, that no greater temperature excursions occur in this hypothalamic thermoregulatory center and deep-body temperature remains stable near the normal level.

Recent experiments in our laboratory have involved use of the method of partitional calorimetry to study the autonomic thermoregulatory responses of animals exposed to microwaves (4). This method permits a complete accounting of all the sources of thermal energy influencing the individual thermoregulatory mechanisms of the body as well as the specification of the exact means by which energy is transferred from the body to the environment. In our experiments, the steady-state heat balance of squirrel monkeys exposed to 2450-MHz CW microwave fields (unilateral planewave) was determined at controlled T_a of 20, 26, and 32 °C. Power densities explored at each T_a ranged from 10 to 25 mW/cm^2. The major finding of these studies was that the animal subjects were able to maintain thermal equilibrium during microwave exposure by an orderly and predictable mobilization of normal thermoregulatory responses. The sole exception involved microwave exposure at 25 mW/cm^2 in the 32 °C environment, a combination that appeared to provide a thermal overload for the heat-loss capabilities of the animal subjects. The squirrel monkey exhibits limited capacity for evaporative heat loss in warm environments (56) and can rapidly become hyperthermic when the temperature of the skin approaches that of the body core. A detailed analysis of the data from this study revealed that individual responses measured with and without microwaves present could be described by the same functional relationships, indicating that the thermoregulatory system deals with microwaves in the same manner as other environmental energy sources. In addition, thermal sensors in the skin, rather than those deeper in the body, were probably responsible for most of the response changes observed.

In our researches into the thermoregulatory consequences of exposure of the squirrel monkey to microwave fields, we have seldom used power densities higher than 25 mW/cm^2; in other words, rates of energy deposition have seldom exceeded about 75% of the animal's resting metabolic heat production in a thermoneutral environment, normally about 5 W/kg. Considering the fact that the heat production of a squirrel monkey will be elevated by 6-7 W/kg above the resting level when the animal is in a 20 °C environment (13 °C below

thermoneutrality), we predicted, from our earlier results and our dosimetry, that a microwave exposure at about 40 mW/cm^2 would counteract this metabolic elevation. A pilot study by Candas (18) in our laboratory confirmed this prediction. He also demonstrated that vasodilation of the tail was mobilized at higher power densities when heat production was minimal, a response that was sufficient to prevent a substantial increase in heat storage by the body. The patterns of thermophysiological responses observed by Candas confirmed the influence of both peripheral and internal inputs to thermoregulation in squirrel monkeys exposed to microwaves in a cool environment. However, the skin temperature (T_{sk}) appeared to be of greater importance than the deep body temperature, substantiating conclusions drawn from our other experiments.

THE PROBLEM

To determine the upper tolerance limits to imposed microwave fields is clearly as important as to determine the thresholds for the mobilization of individual responses that regulate the body temperature when such fields are present in the environment. The exploration of the full dynamic range of each thermoregulatory mechanism and the modes of interaction between them must enhance our understanding of the capability of the whole organism to deal with thermalizing energy deposited in the body by radiofrequency radiation (RFR). Data now in hand indicate the T_a at which a microwave exposure occurs will dictate the range of field strengths that can be tolerated before uncontrolled hyperthermia develops. We have already determined (4) that a power density of 25 mW/cm^2 (SAR = 3.75 W/kg) cannot be tolerated indefinitely by a squirrel monkey restrained in a 32 °C environment, but we need additional experimental data to characterize the tolerance limits both at other T_a and during behavioral thermoregulation. Investigation of these tolerance levels by various experimental techniques is the subject of the present report.

Three different experiments were conducted and are described separately in the following sections. In the first experiment, squirrel monkeys, highly trained to regulate the temperature of their environment behaviorally, underwent both brief (10 min) and prolonged (90 min) whole-body exposures to 2450-MHz CW microwaves at high power densities (range = 30 to 70 mW/cm^2) to

determine at what power density thermoregulatory behavior may break down. The efficiency of the behavior was assessed by measurements of skin and deep body temperatures, including that of the brainstem. In the second experiment, we investigated the potential for disruption of autonomic thermoregulatory responses by an altered metabolic state (produced by systemic administration of a beta-adrenergic agonist, isoproterenol) in squirrel monkeys exposed to microwaves (power density range = 0 to 30 mW/cm^2) in a thermoneutral environment. The major finding was that, unlike shivering thermogenesis, chemically mediated nonshivering thermogenesis was unaffected by thermalizing energy introduced passively into the body. In the third experiment, the method of partitional calorimetry was used to determine the maximal power density (range = 10 to 60 mW/cm^2) that could be tolerated by squirrel monkeys equilibrated to cool (20 °C) and neutral (26 °C) environments. Thermal balance was maintained at a power density up to 45 mW/cm^2 at T_a=26 °C and 60 mW/cm^2 at T_a=20 °C. These points, together with that (20 mW/cm^2) already determined for T_a=32 °C, describe a curvilinear function that can be understood in terms of the thermoregulatory capabilities of the subject animal.

The ultimate goal of research into the biological effects of exposure to RFR is to evaluate the impact of comparable exposure on the health and functioning of human beings. Since it is considered morally indefensible to deliberately expose humans to RF fields, it is necessary to use other means to predict potential consequences. One approach might involve the use of sophisticated simulation models of the human thermoregulatory system (e.g., Stolwijk and Hardy, [59]) coupled to a block model of RF energy deposition (e.g., Gandhi, [26]), under the assumption that RFR is equivalent to other forms of thermal energy. On the other hand, data derived from animal experiments have been useful in the past and will continue to be an important predictive source, especially if the unique thermoregulatory profile of each species is accounted for (9,11). The studies reported here have bearing not only upon the promulgation of maximum permissible exposure standards, but also upon the potential consequences of accidental exposure of human beings to RF fields at moderately high power densities.

METHODS

Subjects

Adult male squirrel monkeys (Saimiri sciureus) were used as subjects in these studies. The monkeys' estimated ages ranged from 5 to 9 yr; and their body masses ranged from 850 to 1050 g at the time of testing. The animals were housed individually in a colony room maintained at 24 ±2 °C and 40 ±10% relative humidity. Water was available ad libitum. The animals were fed a daily ration of Purina monkey chow supplemented with fresh fruit, peanuts, and a milk-pablum mixture. All animals were well adapted to the restraining chair, and most had previously participated in a variety of experiments to assess behavioral and autonomic thermoregulatory capability. Some of these experiments involved brief exposures to 2450-MHz CW microwave fields at power densities at or below 25 mW/cm^2. The basic procedures for adaptation and chair training have been described by Adair et al. (12).

Test Chamber, Dosimetry and Response Measures

During the experiments, the monkey was chair-restrained in the far field of a 15-dB standard gain horn antenna inside an electromagnetically anechoic chamber of interior dimensions 1.83 x 1.83 x 2.45 m. The interior chamber walls were covered with 20-cm pyramidal microwave absorber (Advanced Absorber Products, Type AAP-8) to minimize reflections (<40 dB). The restraining chair was enclosed by a 30 x 33 x 78-cm box constructed of 5-cm thick closed-cell Styrofoam. A valve system allowed air from one of two temperature-controlled (±0.5 °C) sources to circulate at 0.36 m/s through the box in the direction shown in Figure 1. Each monkey was trained to pull a response cord to activate the valves, thereby selecting the T_a preferred. The use of a single air source provided an environment of closely regulated temperature for the assessment of autonomic thermoregulatory responses. The T_a inside the box was sensed by a copper-constantan thermocouple located in the air outlet from the anechoic chamber (cf. Fig. 1) and was recorded continuously on a strip chart. The temperature of the interior wall of the Styrofoam box was also recorded for comparison purposes. The monkey was under constant video surveillance during the 4- to 5-h test sessions. All test sessions were conducted in the presence

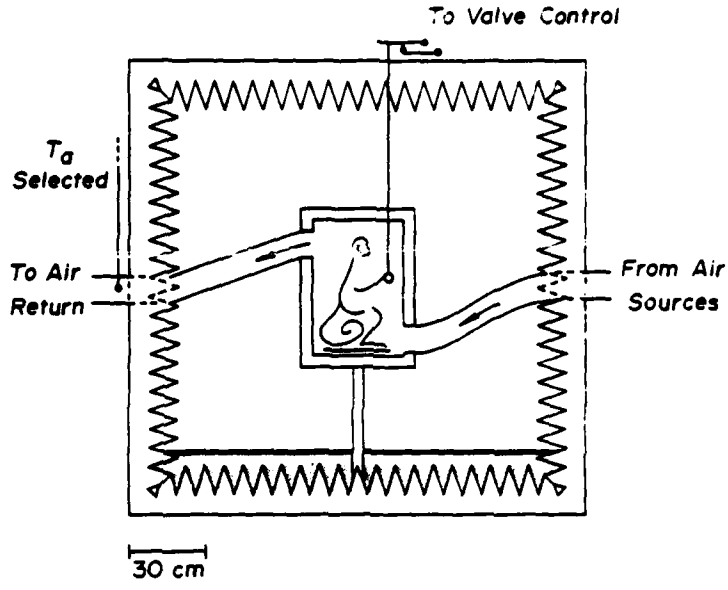

Figure 1. Schematic diagram (as viewed from the horn antenna) of the convective system through the animal's test box inside the anechoic chamber.

of a 73-dB sound pressure level (SPL) (re 0.0002 dyne/cm^2) masking noise to prevent auditory cues when the microwave generator was turned on and off (31). Additional refinements of the test environment, unique to each study, will be described in later sections of this report.

Continuous microwaves of a single frequency, 2450 ± 25 MHz, were generated by a Cober Model S2.5W generator and fed to the antenna via standard waveguide components. Calibration measurements to determine field uniformity at the animal's location, made with a Narda Model 8316B broadband isotropic radiation detector fitted with a Model 8323 probe, showed a maximum nonuniformity of 8% with the restraining chair absent and an additional 5% with chair present (for details, see [6]). Insignificant changes occurred with the introduction of a hood and hose connections for measurement of oxygen (O_2) consumption and carbon dioxide (CO_2) production, fine thermocouples and Vitek probes (17) for measurement of body temperatures, or a Plexiglas boot and hose connections for measurement of thermoregulatory sweating from the foot of the animal. In this regard, Ho (36) has demonstrated field perturbations produced by such devices may not be as important as other variables (e.g., animal size, configuration and movement, polarization and uniformity of the incident field).

An assessment of whole-body energy absorption over the power density range from 5 to 40 mW/cm^2 has been based on temperature increments produced at 4 depths in 3 sizes of saline-filled cylindrical Styrofoam models by 10-min microwave exposures. The mean temperature rise in the liquid above an equilibrated 35 °C was used to calculate the SAR. The SAR ranged from 0.135 to 0.153 W/kg per mW/cm^2, with the higher values corresponding to the smaller masses. Details of the dosimetry procedure are given by Adair and Adams (6). Temperature increments were determined twice for one of the models, once standing alone and once inserted into the restraining chair, to assess the effects of the chair on whole-body energy absorption. No differences were found between the SARs determined from the two sets of measurements.

This rough assessment of SAR, yielding an average value (0.15 W/kg per mW/cm^2) comparable to that predicted for the squirrel monkey at 2450 MHz by Durney et al. (22), has been confirmed by three different independent procedures. First, as reported by Adair and Adams (8), the steady-state reduction in metabolic heat production ($\underline{M}$) of cold-exposed squirrel monkeys, exposed for 90 min to a controlled microwave field, is exactly equal (in W/kg) to the SAR of the imposed field. Second, as reported by Adair (2), conscious squirrel monkeys equilibrated to a thermoneutral T_a of 33 °C can be used as adjunctive dosimeters. The SAR can be determined from colonic temperature (T_{co}) increments during 10-min microwave exposures in animals that are fully vasodilated, but not sweating. If the body mass of the monkey is 0.9 to 1.0 kg, the SAR so determined will be 0.15 W/kg per mW/cm^2. Third, the whole-body SAR was determined in our exposure facility by J.B. Kinn (38), who used the method of twin-well calorimetry. A model of a seated squirrel monkey, filled with 0.76 kg of tissue-equivalent material (32), was exposed, at the monkey's location, for 10 min at a power density of 20 mW/cm^2. Three separate determinations under these conditions yielded an average whole-body SAR of 0.18 W/kg per mW/cm^2, a not unreasonable value considering that the mass of the model was somewhat less than that of most of our animal subjects.

EXPERIMENT 1: BEHAVIORAL THERMOREGULATION IN THE PRESENCE OF HIGH-INTENSITY MICROWAVE FIELDS

Introduction

The maintenance of a stable body temperature is essential to the proper functioning of every organism. As discussed earlier, even in those endothermic species that are capable of generating heat in their bodies and that have sophisticated autonomic mechanisms to dissipate that heat to the environment, behavioral responses play an important role. It is far more efficient to reset the thermostat, open a window, or put on a sweater than to take no behavioral action, waiting instead to sweat or to shiver. We have studied thermoregulatory behavior for many years (e.g., Adair, et al. [12]) in terms of the T_a selected, and presumably preferred, by highly trained experimental animals.

Thermoregulatory behavior refers to those voluntary actions by an organism that control the thermal characteristics of the air-skin interface and thereby facilitate regulation of the body temperature at a stable level. Low-intensity microwave fields will influence thermoregulatory behavior as other heat sources do. Rats, trained to press a lever for infrared heat (IR) in the cold, will reduce the rate of lever pressing when a low-intensity microwave field is present (55). The higher the microwave intensity to which they are exposed (range = 5 to 20 mW/cm^2), the less rats will work for IR heating. We have demonstrated (6) that 10-min whole-body exposures to 2450-MHz CW microwaves at a power density of 6 to 8 mW/cm^2 reliably alter the thermoregulatory behavior of squirrel monkeys (i.e., stimulate them to select a cooler environment). Higher power densities stimulate correspondingly greater reductions in selected T_a and thereby assure regulation of skin and deep-body temperatures at the normal level.

Other studies (9) investigated the potential for adaptation of thermoregulatory behavior during prolonged (up to 2.5 h) microwave exposure. Three major results were reported: (1) Whole-body exposure to microwaves at a subthreshold power density (4 mW/cm^2) did not alter thermoregulatory behavior no matter how long it lasted; (2) prolonged exposure at higher power densities, 10 and 20 mW/cm^2, stimulated the monkeys to select T_a 1.5 and 3.0 °C cooler respectively

than control levels, ensuring stability of the body temperatures; and (3) except for the first microwave presentation of a series or the early minutes of a single long exposure, the length of time the field was on had no significant bearing on the T_a selected or the resulting body temperatures achieved thereby. Presumably, in all of these experiments, behavioral selection of cooler surroundings serves to increase the thermal gradient from body core to skin and facilitates heat loss to the environment.

The "threshold" power density of 6 to 8 mW/cm^2 for the alteration of thermoregulatory behavior represents an SAR that is the equivalent of about 20% of the resting $\underline{M}$ of the squirrel monkey. Above this threshold value, the reduction in preferred T_a is a linear function of the SAR of the imposed field, at least up to about SAR = 3.5 W/kg. However, the upper SAR limit of this function, if one exists, is unknown. In Experiment 1, we set about to determine if there is such a tolerance limit or "ceiling" above which behavioral thermoregulation breaks down.

Methods and Procedure

Four adult male squirrel monkeys underwent both brief (10-min) and prolonged (90-min) whole-body exposures to 2450 MHz CW microwaves (E-polarization) at high power densities ranging from 30 to 70 mW/cm^2, while controlling T_a behaviorally. During an experiment, individual animals were lightly chair-restrained inside a Styrofoam box that was ventilated by a temperature-controlled airstream (Fig. 1). Each animal was highly trained to pull a cord to select between two preset T_a, 10 and 50 °C. The monkey was exposed to air at one temperature and each response was reinforced by a 15-s presentation of the other. Air at the original temperature then automatically returned until the monkey responded again. In the absence of microwaves, all animals selected an average T_a of 35-36 °C. The selected T_a was measured by a copper-constantan thermocouple in the air outlet from the anechoic chamber and recorded continuously on a strip chart.

During the experiments, T_{co} and the temperatures of four representative skin areas (tail, leg, abdomen, and foot) were read once a minute by an on-line computer. For this purpose, thermocouples with 0 °C reference junctions were

constructed in special configurations from 36-ga copper-constantan wire. The leads were shielded and held out of alignment with the electric field vector of the incident planewave to minimize field effects. Any measured body temperature that showed abrupt changes greater than 0.1 °C (equivalent to an electromotive force [emf] change greater than 4 μV) correlated with microwave onset or termination was discarded. From the four T_{sk}, a weighted mean skin temperature ($\overline{T}_{sk}$) was calculated (56):

$$\overline{T}_{sk} = 0.07\ T_{ft} + 0.37\ T_{lg} + 0.45\ T_{abd} + 0.11\ T_{tl}.$$

The temperature of the medial preoptic area of the hypothalamus (T_{po}) was measured in one monkey who had been chronically implanted with a pair of sealed Teflon re-entrant tubes in that brainstem region (cf. Adair, et al. [11]). For this purpose, the probe of a Vitek Model 101 Electrothermia Monitor (17) was inserted to the bottom of the implanted tube located closest to the midline and held in place by a Tygon sleeve slipped over the protruding re-entrant tube. The output voltage of the Vitek system was recorded on a strip chart to yield a continuous tracing of T_{po}.

Each experimental session lasted 4 to 5 h, and always began with a 2-h period with no microwaves present to stabilize thermoregulatory behavior and all measured body temperatures. Following the initial stabilization period, three different protocols were used:

Protocol 1: Ascending Power Density Series

A series of four 10-min microwave exposures in an ascending order of power density (e.g., 35, 40, 45, and 50 mW/cm^2) was presented. Sufficient time was allowed between exposures to restore the air and body temperatures to normal levels. A 30-min period of behavioral thermoregulation terminated the session. Since only four power densities could be presented in a given series and we wished to investigate a much wider intensity range, overlapping series were presented in successive sets of experiments with each monkey. Four such overlapping series covered the power density range from 25 to 60 mW/cm^2. Three experiments on each of four monkeys were conducted for each series.

Protocol 2: Control for Presentation Order

To ensure that the results of protocol 1 were not an artifact of the order of presentation of power density (always increasing), several of the series were presented in randomized order as well as increasing order. Selected series were randomized for each of the four monkeys. Otherwise, the protocol was the same as Protocol 1.

Protocol 3: Effects of Prolonged Microwave Exposure

Two power densities, 30 and 45 mW/cm^2, were selected for study since both were well tolerated by all animals during the 10-min exposures. The initial 2-h stabilization period was followed by a single 90-min microwave exposure at either 30 or 45 mW/cm^2. A 30-min period of behavioral thermoregulation terminated the session. Three such experiments at each power density were conducted on each of four monkeys. To serve as comparison data, three 4-h sessions of behavioral thermoregulation in the absence of microwaves were conducted on each monkey.

Results

Ascending Power Density Series

The data from all experiments were analyzed for each animal individually; thus, each animal served as its own control. Mean values (±1 SEM) of each measured dependent variable were computed for each 10-min segment of each experiment across the three experiments in each series. The T_a selected by the animal was determined from air outlet temperature (T_a in Fig. 1). For this purpose, the strip-chart record of airstream temperature was digitized at 1-min intervals.

The mean T_a selected by one monkey (solid circles), together with the T_{co} (triangles) and $\bar{T}_{sk}$ (squares) are shown for one of the power density series (35 to 50 mW/cm^2) in Figure 2. In these experiments, the 2-h baseline period of behavioral thermoregulation in the absence of microwaves was followed by four 10-min microwave exposures at increasing power density that were separated by

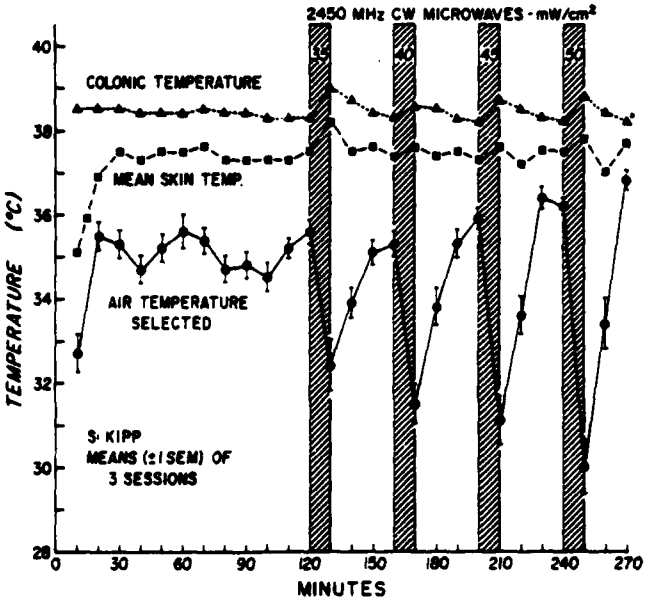

Figure 2. Mean ambient temperature (±1 SEM) selected by one monkey (computed in 10-min bins) as a function of experimental time during three experiments in which the animal was exposed (hatched regions) to 10-min periods of 2450-MHz CW microwaves at increasing power density (solid circles). The weighted mean skin temperature (squares) and colonic temperature (triangles) measured during the experiments are also shown.

30-min periods of no microwaves for restabilization of thermoregulatory behavior. The figure shows that each time the field came on, the animal responded by selecting a drastically lower T_a than normally preferred. Also, the higher the power density of the microwave field, the cooler the air selected by the animal. There appears to be no breakdown in the orderly mobilization of thermoregulatory behavior in the presence of microwaves at a power density as high as 50 mW/cm^2. As a result of the dramatic changes in the animal's thermoregulatory behavior, the increases measured in $\overline{T}_{sk}$ and T_{co} were rather modest, even at 50 mW/cm^2. During this power density series, the maximal increment in mean T_{co} when the microwaves were present was 0.8 °C, a value not exceeded during any other power density series conducted on this animal. The data presented in Figure 2 are a representative example of those collected on all four monkeys.

As just noted, a maximum of four power densities could be presented in any given experimental session else the sessions would become too lengthy and stressful for the subjects. Therefore, to investigate a much wider range of microwave power densities, overlapping series were presented in successive sets of experiments conducted on each monkey. Figure 3 summarizes the results for one animal of four overlapping experimental series that cover the power density range from 25 to 60 mW/cm^2. The bullseyes plotted at a power density of 0 mW/cm^2 represent the mean T_{co}, $\bar{T}_{sk}$, and T_a selected during the final 30 min of the baseline periods when no microwaves were present. The other symbols, coded by series, represent mean temperatures calculated across three experimental sessions during the 10-min periods when the microwave field was on. Clearly, across this power density range the T_a selected is a linear function of power density and SAR. Also, this function is continuing to decrease even at an SAR (9 W/kg) that is the equivalent of twice the resting metabolic heat production of the animal. At this level, the monkey selected an environment that was 6 °C cooler than normally preferred. Skin and deep body temperatures show a remarkable stability at near-normal levels across the full range of power densities explored in these series. From these results, we feel safe in drawing the conclusion that 60 mW/cm^2 *does not represent* the upper tolerance limit for behavioral thermoregulation by the squirrel monkey during brief exposures to 2450-MHz CW microwaves. Indeed, one animal tolerated brief microwave exposures at power densities of 65 and 70 mW/cm^2 with no evidence of deterioration in the behavioral response.

Control for Presentation Order

It was essential to demonstrate that these orderly results were not simply an artifact of the order in which the power densities were presented, i.e., always increasing as the experimental session progressed. Therefore, several of the series were presented in random order, different for each experimental session, as well as increasing order. An example of the results is shown in Figure 4, which depicts the mean T_a selected by a different monkey when exposed for 10-min periods at a range of power densities from 45 to 60 mW/cm^2. The solid symbols represent mean data from three sessions in which power density always increased with successive microwave exposures; the open symbols represent mean data from three sessions in which the order of presentation of power

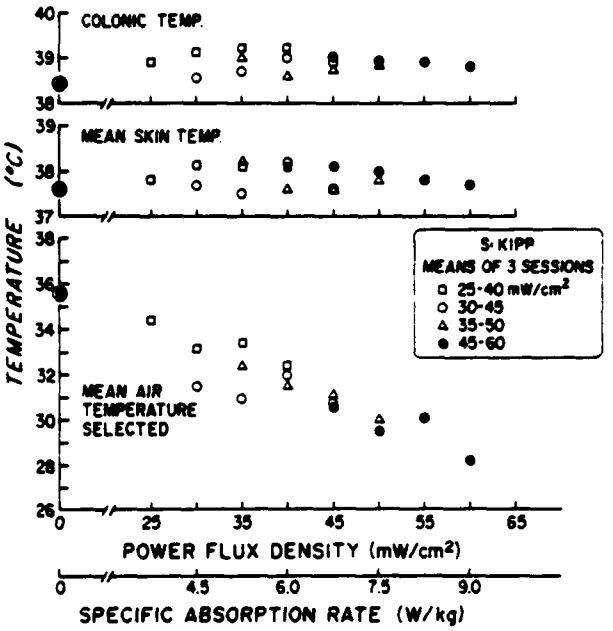

Figure 3. Summary of the results of four overlapping series of experiments on one monkey showing mean ambient temperature selected during 10-min microwave exposures as a function of power density and SAR, and resultant skin and colonic temperatures.

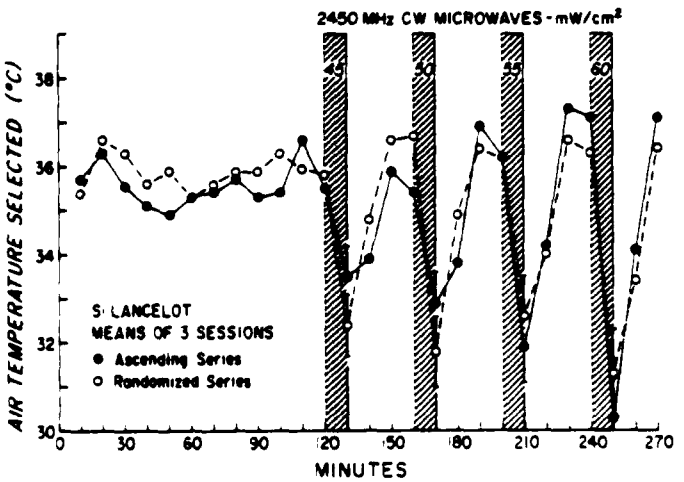

Figure 4. Mean ambient temperature selected by one monkey (computed in 10-min bins) as a function of experimental time during two series of three sessions each in which the monkey was exposed (hatched regions) to 10-min periods of 2450-MHz CW microwaves. Solid symbols represent ascending series of power density from 45 to 60 mW/cm^2; open symbols represent sessions in which order of presentation of power density was randomized.

density was randomized. The error bars at 130, 170, 210, and 250 minutes for both series indicate ±1 SEM. Student's t-tests conducted between corresponding means from the two series indicated that at no power density did the selected T_a differ reliably between the randomized and the ascending series.

As in the initial experiments of this study, it is again apparent that the higher the strength of the microwave field imposed upon the animal, the cooler the environment selected. This conclusion is evidently not an artifact of the order in which power density is presented. Although not depicted in Figure 4, skin and deep body temperatures continued to be regulated efficiently whatever the presentation order. This regulation is, of course, a direct function of the T_a selected by the animal. Finally, whether power density was presented in ascending or random order, no deterioration in behavioral thermoregulation was exhibited by any animal up to the highest level explored, 70 mW/cm^2. This result forces the conclusion that, at least during brief (10-min) whole-body exposures to 2450-MHz CW microwaves, an SAR that is the equivalent of twice the resting metabolic heat production of the experimental animal will be effectively countered by alterations in thermoregulatory behavior. As an important caveat to this statement, it is essential that the prevailing environmental options include a sufficiently cool temperature so that convective and radiant heat losses from the body will be maximized.

Effects of Prolonged Microwave Exposure

Two power densities, 30 and 45 mW/cm^2, were selected for study of the effects of prolonged microwave exposure on thermoregulatory behavior. Each of the four monkeys first completed three 4-h sessions of behavioral thermoregulation in which no microwaves were present; these data served as controls for three sessions at each power density that featured a single microwave exposure that lasted 90 min. The data of each animal were analyzed separately so that the microwave results could be compared directly with the baseline data. Means and standard errors of T_{po} (in one monkey), T_{co}, and $\bar{T}_{sk}$, together with the T_a selected were calculated in 10-min bins across the three experimental sessions conducted on each animal at each power density. The mean results, incorporating both control and microwave sessions, for one animal exposed to microwaves at 30 mW/cm^2 (SAR = 4.5 W/kg) are presented in Figure 5. Error bars on the

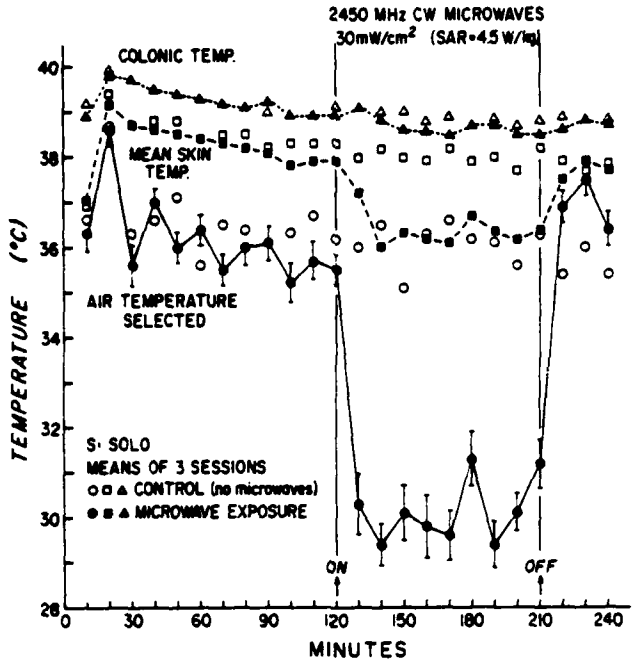

Figure 5. Mean ambient temperature (±1 SEM) selected by one monkey during three experiments in which the animal was exposed for 90 min to 2450-MHz CW microwaves at 30 mW/cm^2 (solid circles). Weighted mean skin temperature (solid squares) and colonic temperature (solid triangles) measured during the experiments are also shown. Comparable data collected during three control experiments are indicated by open symbols.

data points for selected T_a during the microwave sessions represent 1 SEM. In most cases, the SEMs calculated for individual body temperatures were smaller than the symbols used to plot the data; therefore they are not shown on the figure.

All experimental animals preferred an average T_a of 35-36 °C when no microwave field was present. Data collected in the control sessions, representative of all monkeys, show that this preference remained unchanged for up to 4 h. The behavior that generated such a stable environment by alteration of the two available airstream temperatures, 10 and 50 °C, also produced highly stable body temperatures: $\overline{T}_{sk}$ averaged over the body surface equilibrated at about 38 °C while T_{co} was maintained at 39-39.5 °C.

We see in Figure 5 that the onset of the microwave field at 30 mW/cm^2 stimulated a dramatic reduction in the T_a selected by the animal. Within the first 10 min of the microwave exposure, the preferred T_a had fallen by 5.5 °C. This decrease was brought about by a greatly reduced frequency of cord pulling for the 50 °C airstream by the monkey. The persistence of this behavioral alteration, essentially unchanged, for the full 90-min duration of microwave exposure not only ensured that the T_{co} did not rise, but also that the $\bar{T}_{sk}$ would actually fall significantly below the stabilized level.

The monkey whose data are presented in Figure 5 exhibited the most extreme response to prolonged microwave exposure at high intensities of the four animals tested. Data for another monkey, the one whose T_{po} was measured, are given in Figure 6. For simplicity, no data from the control experiments are included here. Figure 6 shows the mean change in selected T_a, together with brain and body temperatures, during 3 experimental sessions when the animal was exposed for 90 min to 2450-MHz CW microwaves at 45 mW/cm^2. The initiation of the microwave exposure resulted in an 8 °C reduction in selected T_a and only slight changes in $\bar{T}_{sk}$ and T_{co}. However, T_{po} increased by nearly 1.0 °C during the initial 10 min of the exposure and exceeded T_{co} by about 0.5 °C as long as the microwave field was present. Interestingly, T_{po} appears to have been precisely regulated at this elevated level for the full 90-min exposure, decreasing rapidly when the field was extinguished. The behaviorally selected T_a showed a small but gradual rise of about 2.0 °C as the microwave exposure progressed and a rapid rebound to preexposure levels at microwave offset. From these behavioral thermoregulation data for the squirrel monkey, we must conclude that in the steady state the upper tolerance limit for whole-body exposure to 2450-MHz CW microwaves has not yet been reached at a power density of 45 mW/cm^2, the equivalent of 1.5 times the animal's resting $\underline{M}$.

Discussion and Conclusions

Several experimental protocols were used in an attempt to determine the maximal field strength of 2450-MHz CW microwaves that can be tolerated by squirrel monkeys when they are controlling the temperature of their environment behaviorally. Both brief (10-min) and prolonged (90-min) microwave exposures were studied. The results confirm and extend previous reports from our own

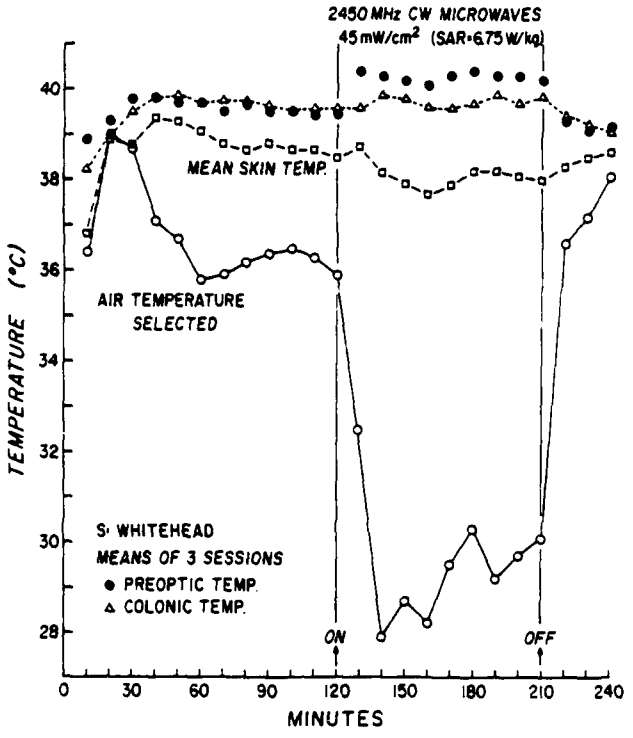

Figure 6. Mean ambient temperature selected by one monkey (open circles) during three experimental sessions in which the animal was exposed for 90 min to 2450-MHz CW microwaves at 45 mW/cm^2 (SAR = 6.75 W/kg). Mean skin (open squares), colonic (open triangles), and preoptic (solid circles) temperatures measured during the experiments are also shown.

laboratory (6,7,9,10) as well as reports from others (29,55). Microwave fields of suprathreshold intensity (e.g., 10 mW/cm^2 and above) provoke changes in thermoregulatory behavior whose purpose is the efficient regulation of deep body temperature. In our experiments on the squirrel monkey, this behavior change takes the form of the selection of an environment that is cooler than preferred when microwaves are absent. As a general rule, the higher the field strength, the cooler is the environment preferred by the behaving animal. These behavioral changes serve to increase the thermal gradient from body core to skin and from skin to environment, thereby facilitating the loss of heat generated in the body. In this study, 10-min exposures at power densities up to 70 mW/cm^2, and 90-min exposures at power densities up to 45 mW/cm^2 were effectively countered by adjustments in thermoregulatory behavior such that the temperatures of both core and skin were regulated with precision near their

respective normal levels. Thus, we appear not to have yet found the upper tolerance limit or ceiling power density for squirrel monkeys able to exert behavioral control over T_a.

Measurements of T_{po} in one animal shed additional light on the results of these experiments. For example, during prolonged microwave exposure at a power density of 45 mW/cm^2, the temperature of the PO/AH was regulated precisely at a level that exceeded the deep body temperature by 0.5 °C (Fig. 6). We know that this hypothalamic region exerts active control over thermoregulatory mechanisms, both behavioral and autonomic. An increase in local T_{po} of only 0.3 °C, produced by an implanted thermode, is sufficient to stimulate a squirrel monkey to cool its environment behaviorally (12). Other data (14) have indicated that increments of T_{po} of this magnitude usually accompany changes in behavioral thermoregulation when a microwave field is present. The conclusion of Adair et al. (14) that the hypothalamus, among other thermosensitive sites in the body, is involved in the initiation and maintenance of microwave-induced alterations in thermoregulatory behavior is upheld by the present results.

On the other hand, the accumulating evidence from this and earlier studies indicates that a thermal "hotspot" probably does not exist in the hypothalamus of the squirrel monkey during microwave exposure in our facility. Normally, the temperature of the hypothalamic thermoregulatory center of this species rides 0.2-0.3 °C above that measured in the colon. This differential is increased only slightly when microwaves are present, averaging 0.4 °C in the steady state at a power density of 20 mW/cm^2 (14) and 0.5 °C at 45 mW/cm^2 (present study), hardly an excessive or worrisome increment. However, still higher power densities may further increase this differential and contribute significantly to the breakdown of behavioral tolerance in the presence of microwave fields. Further experimentation is required to resolve this possibility.

All of the behavioral data collected in this and two earlier studies can be summarized in a single graph (Fig. 7). The figure is an elaboration of one previously published by Adair and Adams (9), and it combines both steady-state data and data derived from 10-min microwave exposures. Figure 7 depicts (solid symbols) the difference between the T_a selected under microwave exposure in the

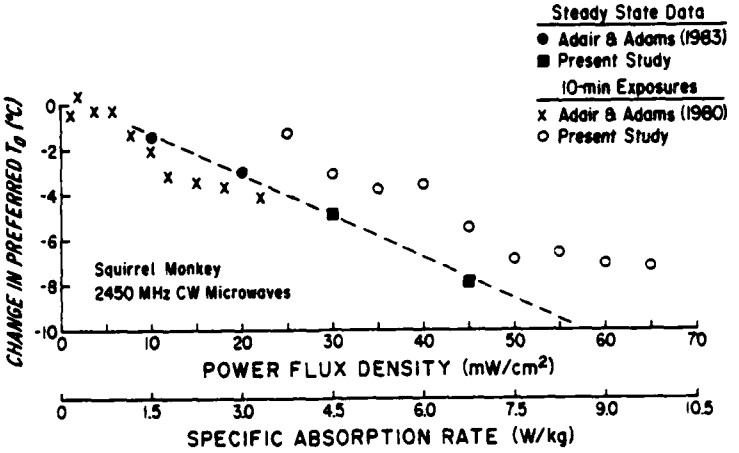

Figure 7. Mean change from baseline level of the ambient temperature (T_a) preferred by squirrel monkeys exposed for 90 min (present study) or 2.5 h (9) to 2450-MHz CW microwaves (solid symbols) as a function of power density or specific absorption rate. Other data for 10-min microwave exposures from the present study (o) and published by Adair and Adams (6) (x) are included for comparison.

steady state (final hour) and that selected in the final hour of the baseline period as a function of both power density and SAR. We read from the figure that at 10 mW/cm^2 (SAR = 1.5 W/kg), a 1.5 °C cooler T_a is preferred. When the power density is doubled (SAR = 3.0 W/kg), so is the demand for external cooling. Data from the present study give a 5.0 °C change in preferred T_a at SAR = 4.5 W/kg and an 8.1 °C change in preferred T_a at SAR = 6.75 W/kg. These points fall on a single linear function that shows no tendency toward a shallower slope as SAR (and power density) increases. If extrapolated, this function predicts that an animal exposed for at least 90 min at 60 mW/cm^2 (SAR = 9.0 W/kg) would cool its environment by 10 °C, a prediction that was verified in two additional experiments on one animal. Therefore, we must conclude that, even in the steady state, an SAR that is the equivalent of twice the resting metabolic heat production of the squirrel monkey will be effectively countered by efficient thermoregulatory behavior. As noted previously, it is essential that the prevailing environmental options permit the selection of a sufficiently cool T_a so that convective and radiant heat losses from the body will be maximized.

Figure 7 also includes data that describe the mean change in preferred T_a from baseline levels during 10-min microwave exposures as a function of power density and SAR. The discontinuity in these data between 22 and 25 mW/cm^2 may be attributed to somewhat different exposure conditions in the 1980 study relative to the present study. No Styrofoam test compartment surrounded the monkeys in the earlier experiments; thus the rate of change of T_a and the resultant transfer of heat between the animal and the environment were both different in the two cases. The shallower slope of the relationship between change in preferred T_a and power density for these data reflect the transient nature of the exposure; considerably more than 10 min is usually required for the stabilization of a preferred thermal environment when the thermoregulatory system is challenged. In general, the greater the thermal challenge, the longer will be required to stabilize any altered thermoregulatory response, up to an asymptotic period of 45-60 min. This fact forms the basis for selection of 90 min as an exposure duration that will guarantee that thermoregulatory responses be in the steady state.

The following conclusions may be drawn from this study of behavioral thermoregulation in the squirrel monkey when a high-intensity microwave field is present:

- During 10-min microwave exposures, power densities as high as 70 mW/cm^2 are countered by the behavioral selection of a cooler environment so that the body temperatures are regulated at close to the normal level.

- During 90-min microwave exposures, power densities as high as 45 mW/cm^2 (and even 60 mW/cm^2 in pilot experiments) are efficiently countered by the behavioral selection of a cooler environment so that the body temperatures are regulated at close to the normal level.

- The higher the power density of microwave exposure, the cooler the environment selected by a behaving animal.

- The upper tolerance limit (power density limit) for whole-body exposure of the squirrel monkey to 2450-MHz CW microwaves lies above 70 mW/cm^2

under conditions when the animal can exert behavioral control over the temperature of the environment.

- The upper tolerance limit (SAR limit) under these experimental conditions represents the equivalent of at least twice the resting metabolic heat production of the squirrel monkey.

- Data from one animal implanted with re-entrant tubes in the medial preoptic area of the hypothalamus ("central thermostat") indicate that a regulated small increase in preoptic temperature may control the behavioral thermoregulatory response during microwave exposures at moderate-to-high intensity.

EXPERIMENT 2: AUTONOMIC THERMOREGULATION IN THE PRESENCE OF MICROWAVES DURING AN ALTERED METABOLIC STATE

Introduction

When endothermic organisms find themselves in cold environments (i.e., at a T_a below the zone of thermoneutrality) and behavioral thermoregulation is not possible, an increase in the rate of heat production within the body occurs. This rate increase is accomplished by involuntary contractions of the skeletal musculature and is classified as shivering thermogenesis. Shivering thermogenesis progresses from piloerection to increased muscle tone to microvibrations to colonic contractions of both flexor and extensor muscles (16). Under certain conditions, heat can also be generated in the body through nonshivering thermogenesis, a process which is unrelated to muscular contractions and is independent of short-term changes in T_a. Nonshivering thermogenesis develops in certain mammals when they are chronically exposed to cold, and there is much evidence that the mechanism is switched on by some action of the catecholamines (33). For example, cold-acclimated rats demonstrate an enhanced metabolic responsiveness to epinephrine and norepinephrine when these substances are administered exogenously, a responsiveness accompanied by an increased rate of secretion of these substances in the body (40). We do not yet fully understand the mechanism by which catecholamines initiate nonshivering thermogenesis, but

the mobilization of glucose and free fatty acids by substances possessing beta-adrenergic activity is clearly involved (24). The target organs certainly include the brown adipose tissue and perhaps the skeletal and cardiac muscles (34).

A wide range of responses may be elicited in laboratory mammals by the administration of beta-adrenergic catecholamines. Enhanced by cold exposure, these responses include an elevated heart rate and metabolic rate, peripheral vasodilation, decreased peripheral resistance, initiation of both thirst and sweating, and increased concentrations of lipids, antidiuretic hormone (ADH), and glucose in the plasma. Many of these responses can be blocked by the prior administration of beta-adrenergic antagonists, such as propranolol, whereas alpha-adrenergic agonists are ineffective (24). All of these effects imply an increase in secretory activity of the thyroid gland. Fregly et al. (24) have concluded that "There is little doubt that thyroid hormones interact with catecholamine-sensitive elements at the cellular level to modify the metabolic and cardiovascular functions mediated by the cAMP system. While the mechanism of interaction in adipose and cardiac tissues remains controversial, there is virtually no disagreement that hypothyroidism reduces and hyperthyroidism enhances certain metabolic and cardiovascular responses to beta-adrenergic catecholamines." (op cit, p.2168).

The beta-adrenergic agonist, isoproterenol, has often been used as a test agent for beta-adrenergic responsiveness because it is a purer stimulant to metabolism than either epinephrine or norepinephrine (25,39). The exaggerated responses to such agents seen in cold-adapted primates can also be observed at reduced strength in animals maintained at thermoneutrality (19). Thus, an agent such as isoproterenol, administered systemically, can be used to produce an altered metabolic state in an animal unacclimatized to cold, i.e., to activate nonshivering thermogenesis. In this experiment, we used isoproterenol to study the effects of microwave exposure on nonshivering thermogenesis in the squirrel monkey.

We first developed a standardized physiological test to determine the thermoregulatory effectiveness of systemic administration of isoproterenol in squirrel monkeys. The animal is first equilibrated to a thermoneutral T_a

(e.g., 30-33 °C) for 60-90 min. A control injection of the vehicle (ascorbic acid in sterile saline, 0.5 ml/kg) is then administered subcutaneously in the abdominal region. Any brief perturbations of thermoregulatory responses return to baseline levels within 10 min. After reequilibration for 30-40 min, an injection of isoproterenol of equal volume (200 μg/kg) is administered via the same route. This injection routinely produces a 2-3 W/kg elevation of $\underline{M}$, vasodilation of the tail, sweating from the foot, and an increase of 0.5 °C in T_{co}. A steady-state response is achieved within about 20 min that lasts for 45-60 min, sufficient to test for the effects of concurrent exposure to a microwave field.

The consequences of whole-body exposure to a microwave field for shivering thermogenesis are well documented. The elevated $\underline{M}$ of endotherms in cold environments will be lowered by an amount that is in direct proportion to the strength of an imposed microwave field (8,35,45,52). In the steady-state, the reduction in $\underline{M}$ of cold-exposed squirrel monkeys (in W/kg) has been demonstrated to be equal to the SAR (in W/kg) of the microwave field, at least for SARs up to the equivalent of 75% of the animal's resting heat production (3,8). This metabolic alteration ensures that the internal body temperature is precisely regulated at the normal level. We primarily designed this experiment to determine if the metabolic heat produced during chemically mediated nonshivering thermogenesis will be similarly reduced during microwave exposure.

Methods and Procedure

Four adult male squirrel monkeys were used as subjects in the experiment. The monkeys were restrained in a chair, one at a time, inside an air-conditioned Styrofoam box in the far field of a horn antenna inside the anechoic chamber. The T_a inside the box was held constant at 33 °C at all times. At this T_a, $\underline{M}$ is at the minimal, resting level and the monkey's tail is vasodilated. The hands and feet are vasoconstricted and the animal is not sweating (4,56).

During the experimental sessions, T_{co} and four representative T_{sk} (abdomen, tail, leg, and foot) were read once a minute by an on-line computer. Oxygen consumption ($\dot{V}_{O_2}$) was measured using an open-flow draw system. Chamber

air was drawn at a constant rate of 7 L/min through a Plexiglas hood over the monkey's head and thence outside the chamber through Teflon tubing. The oxygen partial pressure (P_{O_2}) deficit was measured downstream by a Beckman model 755 paramagnetic oxygen analyzer that sampled the passing airstream at a rate of 0.3 L/min. Metabolic heat production ($\underline{M}$) was calculated from $\dot{V}_{O_2}$ assuming a constant respiratory quotient (RQ) of 0.83 (37,54). Respiratory evaporative heat loss ($\underline{E}_{res}$) was measured by passing a sample of the expired air through a dewpoint temperature (T_{dp}) sensor. The vapor pressure of water in the sampled air was calculated from T_{dp} by means of Antoine's equation (60) from which $\underline{E}_{res}$ was calculated from a modified gas equation. (See Experiment 3 for additional details of this calculation.) To measure sweating rate ($\dot{m}_{sw}$) from one foot, the monkey wore an L-shaped Plexiglas boot through which chamber air was drawn at the rate of 1.9 L/min. The T_{dp} of the effluent air was measured continuously, from which $\dot{m}_{sw}$ was calculated in the same manner as $\underline{E}_{res}$.

Each experimental session began with a minimal 2-h equilibration of the monkey to the prevailing T_a (33 °C) to ensure stabilization of all measured dependent variables. The animal was then either not injected or injected subcutaneously in the abdominal region with isoproterenol hydrochloride (200 µg/kg at 0.5 ml/kg) or vehicle (ascorbic acid in sterile saline, 0.5 ml/kg). All solutions were prepared shortly before use under strictly sterile procedures and held in a light-free environment until injected. Thirty minutes after the injection, the monkey underwent a 20-min whole-body exposure to 2450 MHz CW microwaves at one of four power densities, 0, 10, 20, or 30 mW/cm^2. Following termination of the microwave exposure, the animal remained in the test environment until all responses had returned to their initial stabilized levels; this postexposure period sometimes lasted as long as 2.5 h. The experimental design just described is diagrammed in Figure 8. Each of the four monkeys was tested under each condition of the design matrix, making a total of 48 experimental sessions.

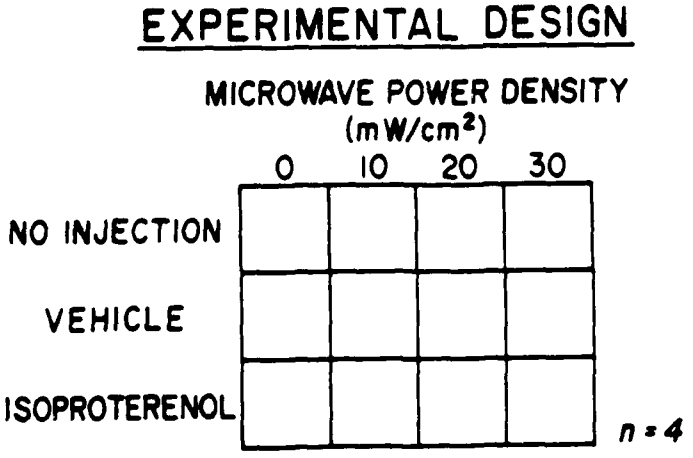

Figure 8. Experimental design of Experiment 2. Each cell of the matrix contains four animals.

Results and Discussion

Representative Data From Individual Experiments

The data from individual test sessions were analyzed in 5-min time bins, i.e., means and standard errors of each dependent variable were calculated at 5-min intervals across the duration of the test session. Four sample experiments, illustrative of different parts of the design matrix, are shown in Figures 9, 10, 11 and 12. Data from the four monkeys are represented in these four figures.

Figure 9 presents representative data from one monkey to illustrate the changes in autonomic thermoregulatory responses that commonly followed a single subcutaneous injection of isoproterenol in the absence of any other experimental treatment. Before the injection, $\underline{M}$ remained relatively stable at the resting level, the tail skin was partially vasodilated but the foot was not, T_{co} was stable and no sweating was evident. Immediately following the injection, a sharp increase in $\underline{M}$ occurred, followed by vasodilation of the foot and tail. However, the additional heat loss was insufficient to balance heat production, leading to an increase of nearly 1.0 °C in T_{co}. The maximal effect

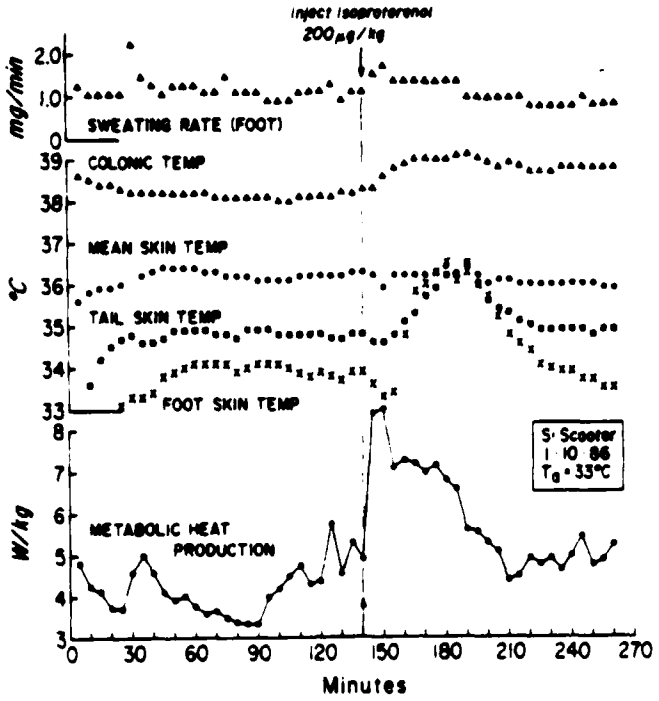

Figure 9. Representative experiment on one monkey equilibrated to an ambient temperature (T_a) of 33 °C to determine effects on autonomic responses of heat production and heat loss of a single subcutaneous injection of isoproterenol hydrochloride (200 μg/kg). Individual plotted points represent means of preceding 5 min.

of the drug persisted for at least 40 min and then gradually subsided, although T_{co} still remained elevated after 2 h.

Figure 10 presents representative data from another monkey to illustrate the changes in autonomic thermoregulatory responses that accompany a 20-min exposure to 2450-MHz CW microwaves at a power density of 10 mW/cm^2. A comparison between Figures 9 and 10 shows the individual differences in response that can occur when different animals are exposed to the same environmental conditions. At the end of the initial equilibration period, before microwave exposure, the animal in Figure 10 was sweating sporadically, and its tail was fully vasodilated, although the foot was only partially dilated. The animal was sitting calmly as though asleep, evidenced by the stable, low level of $\underline{M}$. During the microwave exposure, foot vasodilation occurred as evidenced by the

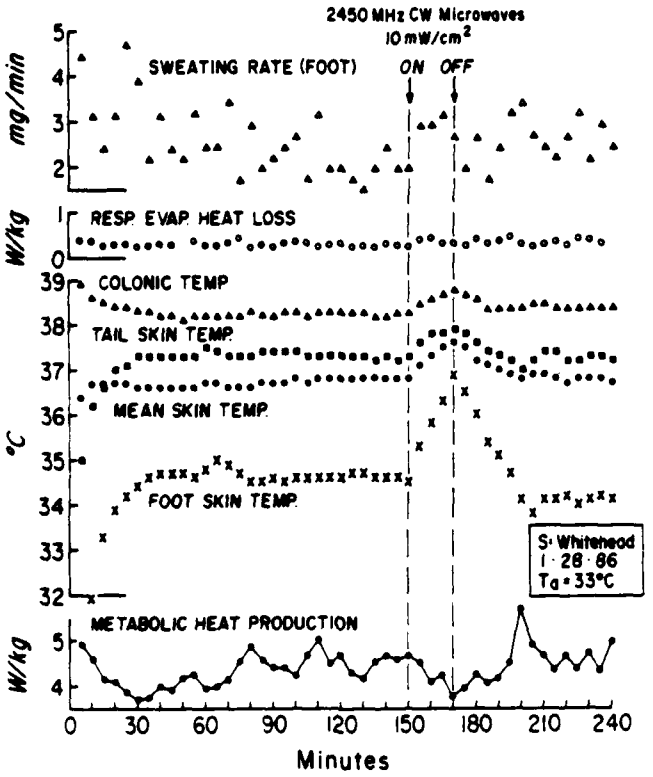

Figure 10. Representative experiment on one monkey equilibrated to an ambient temperature (T_a) of 33 °C to determine effects on autonomic responses of heat production and heat loss of a single 20-min exposure to 2450-MHz CW microwaves at a power density of 10 mW/cm^2. Individual plotted points represent means of preceding 5 min.

rapid increase in foot skin temperature (T_{ft}) relative to the passive increases in the temperature of the other skin sites. Colonic temperature rose 0.5 °C during the 20-min exposure while insignificant changes occurred in the other measured variables.

Figure 11 presents data from still another monkey to illustrate the combined effects of injection of the vehicle and subsequent 20-min exposure to 2450-MHz CW microwaves, in this case at a power density of 20 mW/cm^2. The responses of this animal, before injection, resemble closely those of the animal whose data are presented in Figure 9. Following injection of the vehicle, a spike occurred in the M (due primarily to the disturbance of the animal and introduction of the needle), but this variable restabilized within

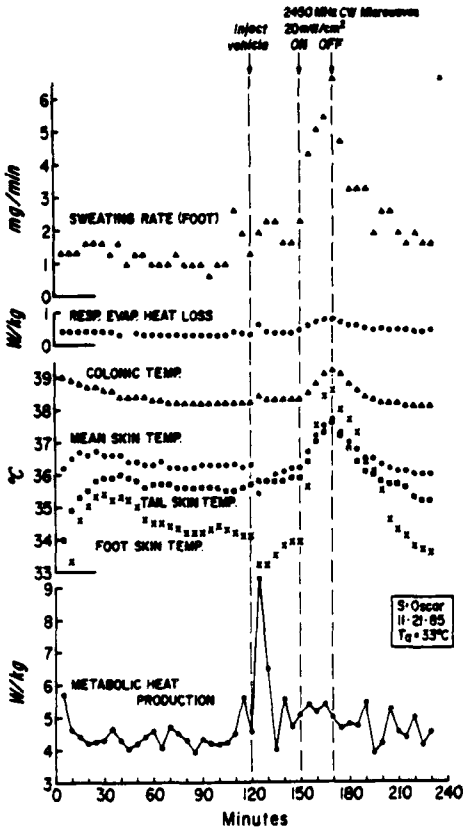

Figure 11. Representative experiment on one monkey equilibrated to an ambient temperature (T_a) of 33 °C to determine effects on autonomic responses of heat production and heat loss of a subcutaneous injection of the vehicle (ascorbic acid in sterile saline, 0.5 ml/kg) followed 30 min later by a 20-min exposure to 2450-MHz CW microwaves at a power density of 20 mW/cm^2. Individual plotted points represent means of preceding 5 min.

30 min. The only other significant response change, often observed after the injections, was a slight reduction in T_{ft} that could indicate either mild (emotional) vasoconstriction or possibly urine dribbling over the foot (not visible over the TV monitor). Dramatic changes in almost every response accompanied the microwave exposure, however. A substantial rise in T_{co}, 1.0 °C in 20 min, occurred despite active vasodilation of both tail and foot as did a rapid increase in foot sweating. No reduction in $\underline{M}$ was possible because this response was already at the low, resting level characteristic of

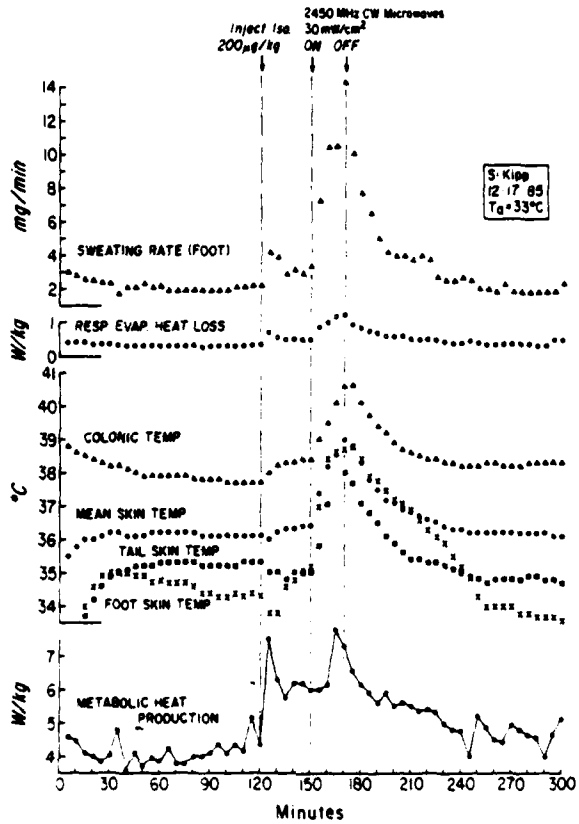

Figure 12. Representative experiment on one monkey equilibrated to an ambient temperature (T_a) of 33 °C to determine effects on autonomic responses of heat production and heat loss of a subcutaneous injection of isoproterenol hydrochloride (200 µg/kg) followed 30 min later by a 20-min exposure to 2450-MHz CW microwaves at a power density of 30 mW/cm^2. Individual plotted points represent means of preceding 5 min.

thermoneutrality. The figure also shows that termination of the microwave field resulted in a rapid return of all responses toward their original stabilized levels.

Figure 12 presents data from the fourth monkey to illustrate the combined effects of a subcutaneous injection of isoproterenol (200 µg/kg) and a subsequent whole-body exposure to 2450-MHz at the highest power density tested, 30 mW/cm^2. Injection of the drug produced an immediate rise in $\dot{M}$, partial

vasodilation of the foot, and limited foot sweating. This combination of heat-generation and heat-loss responses failed to prevent an elevation of 0.8 °C in T_{co}. During the ensuing microwave exposure, no reduction occurred in heat production as seen during microwave exposure in cold environments, when $\underline{M}$ is also elevated. These disparate sequelae of microwave exposure may be attributed to the different nature of thermogenesis in the two cases--nonshivering following isoproterenol injection and shivering in the cold. Other response changes that occurred at microwave onset in Figure 12, full vasodilation of tail and foot and vigorous sweating, failed to prevent a significant increase in T_{co}. At termination of the microwave exposure, more than 2 h was required to restore the initial stabilized thermoregulatory state. Even at the end of the session, $\underline{M}$ and T_{co} were still elevated above their initial levels.

Thermoregulatory Responses as a Function of Injection Condition and Microwave Treatment

Each experimental session had been conducted according to the same protocol: a minimal 120-min baseline period was followed by an injection of isoproterenol, vehicle, or no injection, which in turn was followed, 30 min later, by an exposure to microwaves at 0 (sham), 10, 20, or 30 mW/cm^2. Thus, each experimental session contained common temporal events that could be analyzed across both dependent and independent variables. Figure 13 presents a simplified diagram of the basic temporal protocol. To compare individual thermoregulatory responses that occurred following both injections and periods of microwave exposure, data from three 10-min periods in each experimental session were selected for statistical analysis. These periods are diagrammed in Figure 13 with hatched bars and include (A) the final 10 min of the baseline equilibration, (B) the final 10 min of the 30-min period following injection (or no injection), and (C) the second 10 min of the 20-min period of microwave (or sham) exposure. Four dependent variables, those responses that showed the greatest alteration by the experimental treatments, were selected for analysis; these were T_{co}, T_{ft}, $\underline{M}$, $\dot{m}_{sw}$. For each response, means and standard errors were calculated across animals for each cell of the design matrix (Fig. 8) and each of the three 10-min periods (A,B, and C as designated in Fig. 13). These values appear in Table 1.

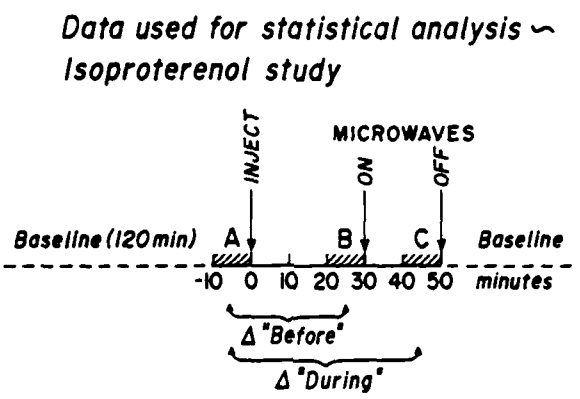

Figure 13. Schematic diagram showing data selected for analysis in Experiment 2.

In Table 1, the designation "Treatment Group" for periods A and B corresponds to the "Power Density" designation for period C. In other words, animals in Treatment Group 20 (periods A and B before microwave exposure) were subsequently exposed to microwaves at 20 mW/cm^2 during period C of those sessions. Inspection of the table reveals several general trends in the data. During period A (the baseline equilibration period), little variability is evident in any given response measure, which indicates the similarity in response from session to session and from animal to animal. During period B (following injection), all responses appear to be altered relative to the no-injection condition, but only following isoproterenol injection, not following injection of the vehicle. In particular, T_{co} is elevated by at least 0.4 °C, T_{ft} is elevated, $\underline{M}$ is increased, and sweating is initiated. During period C (microwave exposure), a regular increase in three of these four physiological responses occurs as a direct function of power density. Colonic temperature, T_{ft}, and $\dot{m}_{sw}$ all increase to higher levels in regular fashion as the power density increases. In every case, the mean level of each response is higher following isoproterenol injection than following vehicle or no injection. The sole exception to this trend is $\underline{M}$ which is not altered by any intensity of microwave exposure, even when elevated by prior injection of isoproterenol.

TABLE 1. MEAN VALUES (±SEM) OF FOUR PHYSIOLOGICAL RESPONSES UNDER FOUR MICROWAVE POWER DENSITIES (TREATMENT GROUPS) AND THREE INJECTION CONDITIONS ACROSS THREE 10-MIN PERIODS (A,B,C) AS DEFINED IN FIGURE 13

Period

Colonic Temperature (°C)

Injectate	A Treatment Group 0	10	20	30	B Treatment Group 0	10	20	30	C Power Density 0	10	20	30
No Injection	38.15	38.13	38.13	38.25	38.08	38.13	38.15	38.25	38.20	38.63	39.15	39.68
SEM	.27	.09	.10	.24	.30	.08	.06	.24	.31	.09	.08	.22
Vehicle	38.20	38.13	38.25	38.19	38.31	38.33	38.43	38.33	38.25	38.68	39.21	39.88
SEM	.20	.17	.19	.15	.30	.22	.20	.15	.23	.21	.19	.17
Isoproterenol	38.15	38.23	37.93	38.08	38.73	39.00	38.83	38.75	38.88	39.70	40.05	40.55
SEM	.10	.06	.32	.17	.29	.17	.27	.27	.30	.22	.21	.18

Foot Skin Temperature (°C)

Injectate	A Treatment Group 0	10	20	30	B Treatment Group 0	10	20	30	C Power Density 0	10	20	30
No Injection	35.03	34.43	34.53	34.48	34.95	34.15	34.35	34.38	34.73	36.08	37.93	38.88
SEM	.39	.35	.40	.24	.35	.19	.57	.22	.34	.26	.53	.34
Vehicle	34.69	34.33	34.16	34.20	34.19	34.29	33.95	34.00	34.74	36.34	38.03	38.59
SEM	.51	.16	.14	.21	.76	.19	.15	.16	.71	.47	.49	.24
Isoproterenol	34.50	34.65	34.43	34.50	35.80	36.30	35.50	35.65	36.55	38.45	38.83	39.13
SEM	.34	.42	.28	.13	.25	.47	.65	.40	.36	.18	.21	.57

Metabolic Rate (W/kg)

Injectate	A Treatment Group 0	10	20	30	B Treatment Group 0	10	20	30	C Power Density 0	10	20	30
No Injection	4.26	4.29	4.34	4.23	4.36	4.41	4.37	4.65	4.35	4.18	4.52	4.78
SEM	.10	.15	.62	.26	.16	.10	.45	.36	.22	.19	.49	.32
Vehicle	4.14	4.58	4.68	4.64	4.53	4.85	4.83	5.00	4.36	4.35	4.52	4.55
SEM	.39	.15	.43	.34	.76	.19	.25	.32	.53	.39	.33	.18
Isoproterenol	4.45	4.20	4.12	4.40	6.70	7.03	7.21	6.59	6.25	7.20	7.46	7.37
SEM	.27	.30	.46	.27	.58	.20	.29	.23	.49	.57	.81	.94

Sweating Rate (mg/min)

Injectate	A Treatment Group 0	10	20	30	B Treatment Group 0	10	20	30	C Power Density 0	10	20	30
No Injection	1.64	1.72	2.66	1.63	1.61	1.74	2.46	1.72	1.56	2.55	5.52	9.14
SEM	.89	.18	1.49	.57	.82	.12	1.39	.55	.83	.16	2.40	3.08
Vehicle	2.85	1.90	1.95	2.62	3.40	2.13	2.41	2.60	3.23	3.03	5.36	9.49
SEM	1.67	.83	.59	.83	2.40	1.07	.70	.98	1.89	1.03	1.62	2.23
Isoproterenol	2.62	2.33	2.62	2.17	4.40	4.61	5.16	3.66	3.92	7.90	12.46	14.14
SEM	.84	.50	.93	.79	1.60	1.35	1.13	1.23	1.60	2.78	2.78	2.55

To test for the significance of these findings, a series of two-way analyses of variance (ANOVAs) were performed on the data. Figure 14 shows the design matrix expanded to the time dimension (periods A, B, C) and illustrates the two ANOVAs performed, for each of the four physiological responses, on the data collected in each period. ANOVA 1 determined the significance of the difference in response between no injection and vehicle injection; ANOVA 2 determined the significance of the difference in response between no injection and isoproterenol injection. If the ANOVA 1 analysis revealed no significant differences due to injectate, then such significant differences in the ANOVA 2 analyses would be attributable to isoproterenol alone. Although three-way ANOVAs could have been performed across injectates, the source of any significant difference would not have been revealed by such an analysis. The level of probability was preset at $p \leq 0.05$ for the significance of all tests.

Table 2 summarizes the results of six ANOVAs performed on each of the four physiological responses in terms of the significance of injectate and microwave effects as well as the injectate/microwave interaction. ANOVA 1 showed that there was no significant (NS) difference in any response to the vehicle injection vs no injection procedure except during period C when T_{co}, T_{ft} and $\dot{m}_{sw}$ were reliably altered (S) by microwave exposure. It is noteworthy that $\underline{M}$ was not similarly affected by microwaves, confirming our published finding that no reduction in metabolic heat production is stimulated by microwave exposure of squirrel monkeys at T_a=33 °C (2,4). ANOVA 1 demonstrated that under no condition was injectate (injection of the vehicle vs no injection) a significant source of variation; thus, either treatment could serve as a comparison to the isoproterenol injection treatment in ANOVA 2.

The results of ANOVA 2 (no injection vs isoproterenol) were strikingly different. All four physiological responses were reliably altered (S) following isoproterenol injection, i.e., in period B. Also, all responses were significantly different (S) under isoproterenol, compared to the no injection condition, in period C quite apart from any additional response alteration by microwaves; i.e., isoproterenol alone produced reliable changes in T_{co} and T_{ft}, $\underline{M}$, and $\dot{m}_{sw}$. Once again, three of these responses were reliably affected by microwave exposure, the exception being $\underline{M}$ which remained unchanged (NS). The added significance of the injectate/microwave interaction on T_{ft} during

TABLE 2. SUMMARY OF THE RESULTS OF 24 TWO-WAY ANOVAS TO DETERMINE FOR EACH OF FOUR RESPONSES (T_{co}, T_{ft}, $\underline{M}$, AND $\dot{m}_{sw}$) THE SIGNIFICANCE ($P \leq .05$) OF THE EFFECT OF INJECTATE AND MICROWAVE POWER DENSITY

	ANOVA 1			ANOVA 2		
	No injection vs vehicle			No injection vs isoproterenol		
T_{co}						
Source of variation	Period A	Period B	Period C	Period A	Period B	Period C
Injectate	NS	NS	NS	NS	S	S
Microwave	NS	NS	S	NS	NS	S
Inj/MW	NS	NS	NS	NS	NS	NS
T_{ft}						
Source of variation	Period A	Period B	Period C	Period A	Period B	Period C
Injectate	NS	NS	NS	NS	S	S
Microwave	NS	NS	S	NS	NS	S
Inj/MW	NS	NS	NS	NS	NS	S
$\underline{M}$						
Source of variation	Period A	Period B	Period C	Period A	Period B	Period C
Injectate	NS	NS	NS	NS	S	S
Microwave	NS	NS	NS	NS	NS	NS
Inj/MW	NS	NS	NS	NS	NS	NS
$\dot{m}_{sw}$						
Source of variation	Period A	Period B	Period C	Period A	Period B	Period C
Injectate	NS	NS	NS	NS	S	S
Microwave	NS	NS	S	NS	NS	S
Inj/MW	NS	NS	NS	NS	NS	NS

Periods (A,B,C) and ANOVA (1,2) defined in Figure 14.
S = significant; NS = not significant

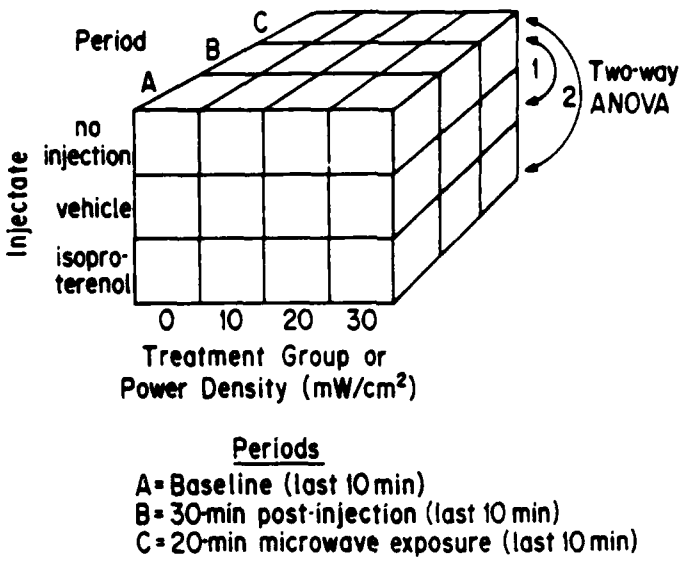

Figure 14. Three-dimensional design matrix to indicate, for each period (A,B,C) the data used for two-way ANOVA 1 (no injection vs vehicle injection) and two-way ANOVA 2 (no injection vs isoproterenol injection).

microwave exposure (F=4.26, d.f.=3) reflects the major role played by vasodilation of the extremities to thermoregulation of the squirrel monkey in the 33 °C environment that prevailed during these experiments (4,56).

Changes in Thermoregulatory Responses Before and After Microwave Exposure

The statistical analyses just described were predicated on the fact that each experimental session contained common temporal events that could be analyzed across both dependent and independent variables. Further, each animal was tested under each condition of the design matrix (Fig. 8). The mean data from the three 10-min periods (A, B, and C in Fig. 13) were also used to compare changes in individual thermoregulatory responses that occurred following both injections and periods of microwave exposure. Differences between pairs of means were then calculated, as shown in Figure 13, to indicate the change in response due to injection alone (Δ "before"), and the change in response due to injection plus microwaves (Δ "during"). These differences were pooled across animals for each cell of the design matrix and grand means were

calculated. The legitimacy of this procedure is underscored by the results of the ANOVAs conducted on data from period A (see Table 2). The same four dependent variables (T_{co}, T_{ft}, $\underline{M}$, and $\dot{m}_{sw}$), responses that showed the greatest alteration by the experimental treatments, were analyzed in this manner. All of these means are summarized for all animals in Figures 15 through 18.

Figure 15 shows the mean change in T_{ft} from the baseline level both "before" microwave exposure when an injection (or no injection) had just been given and "during" the second 10 min of the 20-min microwave exposure (cf. Fig. 13). In the panel on the left, the data are plotted against Treatment Group, a designation that corresponds to the power density at which the subsequent microwave exposure would be conducted. Thus, the points plotted at Treatment Group 30 represent the first part of the experiments for which the points plotted at 30 mW/cm^2 during the microwave exposure represent the second part. The data in Figures 16, 17, and 18 are plotted in an identical manner. The parameter in all four figures, coded by symbol, is the substance administered by subcutaneous injection (or no injection).

During the initial baseline equilibration period of all experiments, the foot skin usually stabilized at a temperature only slightly higher than that of the environment (Figs. 9 - 12). This result meant that the skin on the sole of the foot was still essentially vasoconstricted (4,56). In Figure 15, we see that no change in T_{ft} occurred before microwave exposure unless isoproterenol was administered to the animal. Under isoproterenol, T_{ft} increased slightly over 1 °C, indicative of the initiation of vasomotor activity in this region. During microwave exposure, however, the T_{ft} rose rapidly and in direct proportion to the strength of the imposed field, indicative of active vasodilation. At 30 mW/cm^2, the foot was fully vasodilated in all animals. At lower power densities, 10 and 20 mW/cm^2, the animals who had received isoproterenol were somewhat disadvantaged in terms of their ability to transfer body heat to the periphery because the vasomotor response had already been partially stimulated by the drug.

The mean change in $\dot{m}_{sw}$ from the foot, both before and during microwave exposure, is shown in Figure 16. Usually, little or no thermoregulatory sweating is evident in squirrel monkeys at a T_a of 33 °C because this response

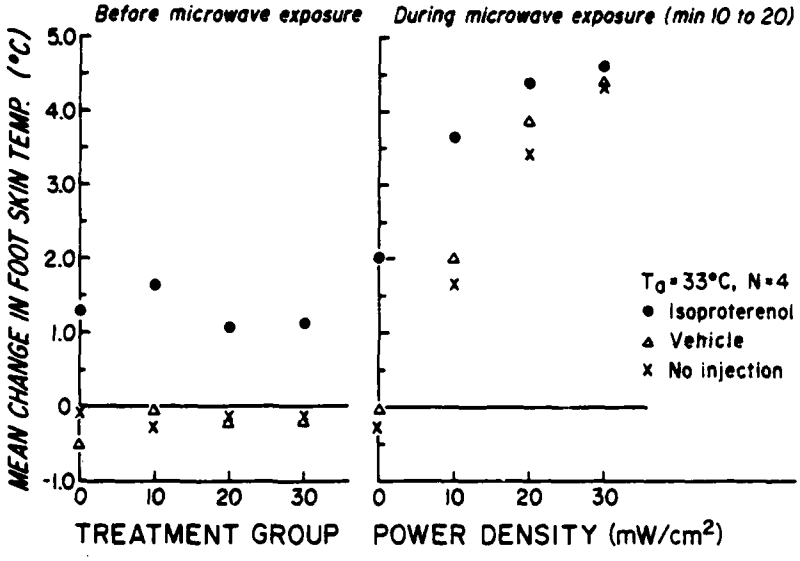

Figure 15. Mean change from baseline level in the temperature of the foot skin both before microwave exposure and during the second 10 min of the 20-min microwave exposure. See Figure 13 for details of how the changes were determined. The parameter is the substance injected.

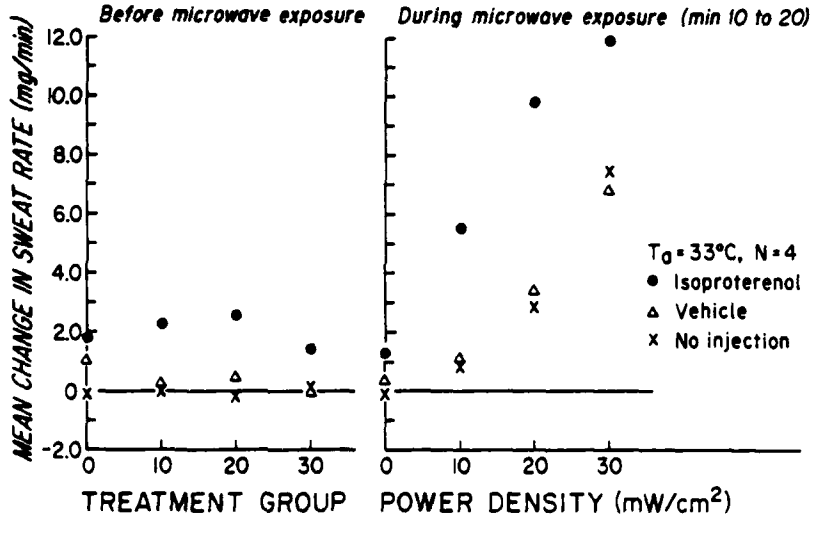

Figure 16. Mean change from baseline level in sweating rate from the foot both before microwave exposure and during the second 10 min of the 20-min exposure to 2450-MHz CW microwaves. See Figure 13 for details on how the changes were determined. The parameter is the substance injected.

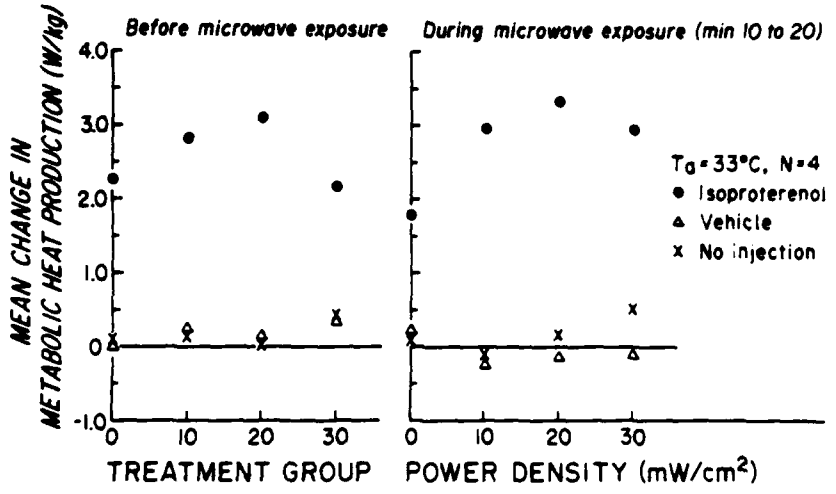

Figure 17. Mean change from baseline in metabolic heat production both before microwave exposure and during the second 10 min of the 20-min period of exposure to 2450-MHz CW microwaves at the power densities indicated. See Figure 13 for details of how the response changes were determined. The parameter is the substance injected.

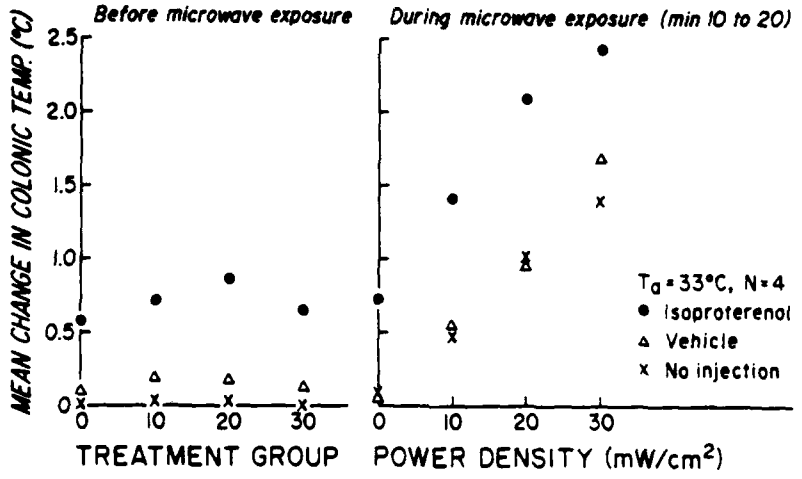

Figure 18. Mean change from baseline in colonic temperature both before microwave exposure and during the second 10 min of the 20-min exposure to 2450-MHz CW microwaves at the power density indicated. See Figure 13 for details of how the response changes were determined. The parameter is the substance injected.

is not mobilized until the animal is fully vasodilated, a state that occurs at T_a = 35 °C (56). At T_a = 33 °C, sweating can be stimulated by whole-body exposure to microwaves and the rate of sweat production is a direct function of the imposed field strength (1). This fact is clearly illustrated by the data in Figure 16 for those animals receiving no injection or injection of the vehicle. Those animals to which isoproterenol was administered (solid circles in the figure) exhibited a reliable increment in $\dot{m}_{sw}$ immediately after injection and a much greater increase in sweating during microwave exposure than the animals not receiving this treatment. Since $\underline{M}$ was also elevated by the drug, and sweating in this species is often correlated with hypermetabolic states, it is not clear that the sweating observed here is purely thermoregulatory in nature.

The mean change in $\underline{M}$ both before and during microwave exposure is shown in Figure 17. Ordinarily, no metabolic reduction would be anticipated in squirrel monkeys exposed to microwaves at T_a = 33 °C (4,8) because heat production is already at the resting level (56). An increase in $\underline{M}$ could conceivably occur at moderately high power densities either because of the normal speeding-up of bodily processes as body temperature rises (the so-called Q_{10} effect) or because the animal increases its activity in an attempt to escape from the thermalizing field. When no injection was given, or when the vehicle alone was given to the animals in this study, no change occurred in $\underline{M}$ either before or during microwave exposure. However, a subcutaneous injection of isoproterenol at 200 μg/kg elevated $\underline{M}$ by 2.0-3.5 W/kg and this level was maintained throughout the period of microwave exposure, whatever the power density of the imposed field. Thus, it appears that unlike cold-mediated shivering thermogenesis, chemically mediated nonshivering thermogenesis is unaffected by thermalizing energy introduced passively into the body. The persistence of an elevated heat production can only mean a compromised thermoregulatory state in which the normal thermoregulatory mechanisms of heat dissipation are insufficient to counterbalance the augmented thermal energy generated both actively and passively in the body tissues. This situation should lead to heat storage within the body and an exaggerated rise in body temperature during exposure to microwave fields.

The mean change measured in T_{co} both before and during microwave exposure is illustrated in Figure 18. When the animals were not injected or injected subcutaneously with the vehicle, no significant alteration of T_{co} occurred before microwave exposure or during sham exposure (power density = 0 mW/cm^2). A slight rise of 0.1-0.2 °C in T_{co} usually accompanied injection of the vehicle, presumably due to the mild stress associated with the injection procedure. During microwave exposure at this T_a, T_{co} rose rapidly and in direct proportion to the power density of the imposed field. The rate of rise was approximately 0.05 °C per mW/cm^2 for the second 10 min of the 20-min microwave exposure. Following a subcutaneous injection of isoproterenol at 200 µg/kg, T_{co} was elevated about 0.75 °C in the absence of microwaves; during microwave exposure at each power density explored, T_{co} averaged 1.0 °C higher than the level it would have attained in the absence of the beta-adrenergic agonist. This serious challenge to the thermoregulatory system is attributable to the actions of the drug administered: a significant increase in nonshivering thermogenesis accompanied by heat-loss responses (vasodilation and sweating) inadequate to counteract the additional heat generated in the body during the absorption of RF energy.

Conclusions and Recommendations

To demonstrate in an endotherm the potential contribution of nonshivering thermogenesis to ongoing thermoregulation, it was necessary to provide a thermal environment warmer than that in which shivering thermogenesis may be operative. Therefore, we selected a T_a (33 °C) near the upper limit of the thermoneutral zone (25 to 35 °C for the squirrel monkey), where shivering thermogenesis is minimal, for tests of the thermoregulatory consequences of administration of the beta-adrenergic agonist isoproterenol. This drug is a catecholaminergic agent that mimics aberrations of the thyroid system such that an animal administered isoproterenol is like a hyperthyroid animal. Our experiments demonstrated that $\underline{M}$ was unaffected by microwave exposure in a 33 °C environment. If the animal subject had received an injection of isoproterenol, heat production, presumably the result of nonshivering thermogenesis, was elevated and remained so in the face of microwave exposure at several power densities (10 to 30 mW/cm^2). Other results of these experiments showed that although administration of isoproterenol stimulated vasomotor activity and

initiated sweating, these responses were unable to counteract the additional heat produced so that body temperature rose because of augmented body heat storage.

These results support the following conclusions and recommendations:

- Unlike cold mediated shivering thermogenesis, chemically mediated nonshivering thermogenesis is unaffected by thermalizing energy introduced passively into the body during microwave exposure.

- An endothermic organism whose thermoregulatory system is compromised may be at a disadvantage, in terms of its ability to regulate the body temperature, when exposed to a microwave field.

- Additional experiments, involving agents (such as pyrogens, alpha-adrenergic agonists, etc) that interfere with normal thermoregulatory processes, should be conducted to measure the potential synergistic effects of microwave exposure administered concomitantly.

EXPERIMENT 3: THERMAL TOLERANCE OF HIGH-INTENSITY MICROWAVE FIELDS AS MEASURED BY PARTITIONAL CALORIMETRY

Introduction

As described earlier in this report, the maximal power density of 2450-MHz CW microwaves that could be tolerated by squirrel monkeys during whole-body exposure at T_a = 32 °C was 20 mW/cm^2 (4). This result was obtained in partitional calorimetric studies in which all of the energy flowing between the experimental subject and the environment was measured and accounted for. Under these conditions, the monkeys could maintain thermal balance indefinitely (i.e., T_{co} stabilized), but if the power density was increased to 25 mW/cm^2, the monkeys became hyperthermic (i.e., T_{co} rose continuously). Therefore, 20 mW/cm^2 was designated the upper tolerance limit for autonomic thermoregulation of squirrel monkeys at T_a = 32 °C. Sessions conducted at a microwave power density of 30 mW/cm^2 in Experiment 2 of the present report, when T_a = 33 °C,

confirmed the rapid rise of T_{co} under these conditions, even when the animals were in the undrugged state.

The purpose of Experiment 3, as described in the general introduction to this report, was to determine similar tolerance limits for autonomic thermoregulation of the squirrel monkey in other environments, viz T_a = 20 and 26 °C, using the method of partitional calorimetry. These T_a were selected because they are representative of cool and neutral environments for the subject animal (56) and extensive data had been collected at these T_a in a previous study (4).

Method of Partitional Calorimetry

The thermalization of tissues in the body that accompanies the exposure of an organism to microwaves presents a unique challenge to the thermoregulatory system. The heat generated in the body tissues during such exposures may be considered to be comparable to that produced during physical exercise, except that no increase in metabolic heat production would be anticipated. In other words, during microwave exposure, tissue heating is passive rather than active. The simplest way of determining the impact of any microwave exposure on the thermoregulatory system is to apply the methods of partitional calorimetry to the organism in question.

In general, the thermoregulatory system mobilizes responses to imposed thermal disturbances in such a way as to minimize or negate the disturbance. The result of this activity is that the internal body temperature, the regulated variable, is maintained at a constant or set level. The disturbance may be a change in the rate of heat production (as during exercise) or a change in the rate of heat exchange between the organism and the environment (as during a change in ambient conditions). In the steady state, the heat produced in the body of an endotherm is balanced by the heat lost to the environment such that storage of heat within the body is minimal. The concept can be expressed by a generalized heat balance equation of the form (16):

$$\underline{M} - \underline{W} = \underline{R} + \underline{C} + \underline{E} \pm \underline{S} \qquad (1)$$

The thermal energy produced in the body by metabolic processes $\underline{M}$ will be modified by any work $\underline{W}$ produced by the animal on the environment. While $\underline{W}$ may be a significant factor for humans or beasts of burden, it may be considered

negligible for other endothermic mammals. Thus, for practical purposes, M represents the metabolic heat production of the body.

The first three terms on the right side of the equation represent the different avenues by which heat is exchanged between the body and the environment: R represents radiation, C represents convection, and E is the heat lost through the evaporation of water from the skin surface and respiratory tract. No term appears in the equation for heat exchange by conduction since it is usually insignificant in most species. If the environmental temperature is higher than that of the body, the direction of heat transfer may be into the body and R and C may have a negative sign. Under such conditions, evaporation of fluid from the body surface and the lungs is the only available avenue of heat loss. Man and certain nonhuman primates (e.g., Erythrocebus patas) sweat profusely and thus are able to thermoregulate efficiently, even during exercise in hot environments (23). The squirrel monkey does not have this capability, possessing eccrine sweat glands only on the friction surfaces of the palms and soles (48,56). Thus, this species must rely to a large extent on behavioral responses (e.g., spreading urine, creating a cool microclimate) to ensure maintenance of a stable body temperature in the heat. It is important to note that these behavioral responses are not available to a squirrel monkey under restraint in a laboratory setting such as the one described.

The last term in the heat balance equation, S, represents the rate of heat storage in the body. If S is positive, body temperature rises; if S is negative, body temperature falls. The goal of normal thermoregulation is the minimization of S and thus the achievement of a stable internal body temperature. The methods of partitional calorimetry are particularly useful to determine the values of individual terms in the heat balance equation at any given environmental temperature for a particular species.

When the method of partitional calorimetry is used, the experimental animal is brought into thermal equilibrium (i.e., $S = 0$) with a particular environmental temperature and the steady-state heat production and heat-loss responses are measured. A wide range of environmental temperatures is studied, which encompasses the range to which the animal is normally exposed, a design that yields a thermoregulatory profile of the species in question.

Metabolic heat production $\underline{M}$ is calculated from O_2 consumption and CO_2 production from which the RQ may also be calculated. Oxygen consumption alone will suffice with the assumption of a constant RQ (0.83 for the squirrel monkey). Total evaporative water loss, which includes water lost through respiration, passive diffusion through the skin, and that evaporated as sweat, is determined from the total reduction in body weight during the experiment, urine and feces being trapped under oil. Evaporative heat loss $\underline{E}$ is then calculated assuming the latent heat of evaporation of water to be 0.72 W·h/g. Dry heat exchanged with the environment through $\underline{C}$ and $\underline{R}$ must be expressed in terms of the surface area of the body. For the squirrel monkey, Stitt et al. (58) have determined that a body mass of 1 kg is equivalent to a surface area of 0.108 m^2.

Heat exchange via radiation and convection is determined indirectly in the method of partitional calorimetry and requires the measurement of the animal's $\bar{T}_{sk}$. Under steady-state conditions of rest at a constant T_a, Equation (1) can be simplified to

$$\underline{M} = \underline{R} + \underline{C} + \underline{E} \qquad (2)$$

In the steady state, when the walls of the environmental test chamber are at the same temperature as T_a, the heat losses from the body due to $\underline{R}$ and $\underline{C}$ are a function of the thermal gradient between skin and air; thus

$$\underline{R} + \underline{C} = \underline{h}\,(\bar{T}_{sk} - T_a) \qquad (3)$$

where $\underline{h}$ is the coefficient of heat transfer to the environment. Substituting in Equation (2) yields

$$\underline{M} = \underline{h}\,(\bar{T}_{sk} - T_a) + \underline{E} \qquad (4)$$

in which $\underline{h}$ is the only unknown. This coefficient can be determined at any given T_a from the relation

$$\underline{h} = (\underline{M} - \underline{E})/\bar{T}_{sk} - T_a) \qquad (5)$$

and a plot of $(\underline{M} - \underline{E})$ vs $(\bar{T}_{sk} - T_a)$ yields a straight line of slope $\underline{h}$ that passes through the origin, where $\bar{T}_{sk} = T_a$. The units in which $\underline{h}$ is expressed are $(W/m^2)/°C$.

The total evaporative heat losses from the body $(\underline{E}_{tot})$ may be partitioned into that which is lost from the respiratory tract $(\underline{E}_{res})$ and that which leaves the skin in the form of sweat $(\underline{E}_{sw})$; thus

$$\underline{E}_{tot} = \underline{E}_{res} + \underline{E}_{sw} \qquad (6)$$

An assessment of the vasomotor tonus of the peripheral circulation can be made by calculating conductance $\underline{K}$, a measure of the core-to-skin heat flow. With the exception of the $\underline{E}_{res}$, all heat leaving the body must pass from the deep body core to the skin, from which it is transferred to the environment by radiation, convection, and evaporation. Under steady-state conditions when heat storage in the body is zero, the heat leaving the body must be equal to the $\underline{M}$ (Eq. 2). In this case, $\underline{K}$ is the total amount of heat leaving the surface of the body divided by the temperature gradient between the core (T_{co}) and the skin ($\overline{T}_{sk}$). Respiratory evaporative heat loss, $\underline{E}_{res}$, is not included in this heat flow because it leaves the body directly through the respiratory tract. Thus

$$\underline{K} = (\underline{M} - \underline{E}_{res})/(T_{co} - \overline{T}_{sk}) \tag{7}$$

When an organism is exposed to a RF field, the energy absorbed from the field ($\underline{A}_{rfr}$) must be added to the metabolic heat produced by the body. Neglecting the work factor, Equation 1 would then become

$$(\underline{M} + \underline{A}_{rfr}) = \underline{C} + \underline{R} + \underline{E} \pm \underline{S} \tag{8}$$

and all other expressions that involve heat production $\underline{M}$ would be similarly modified. Thus, the heat transfer coefficient for an organism exposed to a RF field would become

$$\underline{h} = [(\underline{M} + \underline{A}_{rfr}) - \underline{E}]/(\overline{T}_{sk} - T_a) \tag{9}$$

and conductance would become

$$\underline{K} = ([\underline{M} + \underline{A}_{rfr}] - \underline{E}_{res})/(T_{co} - \overline{T}_{sk}) \tag{10}$$

Basic Thermal Physiology of the Squirrel Monkey

The autonomic thermoregulatory responses of five adult male squirrel monkeys to T_a that ranged from 10 to 39 °C were measured by Stitt and Hardy (56). During the experimental tests, individual animals were restrained in a Plexiglas chair inside an environmental test chamber. The T_a was closely regulated and air movement within the chamber was < 8 m/min (still air conditions). A Plexiglas hood over the animal's head collected the expired air, which was drawn outside the chamber at 7-10 L/min for analysis of O_2 content. The increase in relative humidity of the expired air was also measured so that respiratory evaporative heat loss, $\underline{E}_{res}$, could be calculated. The restraining chair was mounted on a platform which was suspended from a sensitive balance.

In this way the reduction in body mass could be monitored continuously during the experiment. Deep T_{co}, 10 cm beyond the anal sphincter, was measured with a polyethylene-encased copper-constantan thermocouple with a reference junction in melting ice and water. Four representative T_{sk}, taken from shaved areas on the abdomen, tail, leg, and foot, were also measured with copper-constantan thermocouples constructed in special configurations. These temperatures were used to calculate a weighted $\overline{T}_{sk}$ as suggested by Stitt et al. (58):

$$\overline{T}_{sk} = 0.45T_{abd} + 0.37T_{lg} + 0.11T_{tl} + 0.07T_{ft} \qquad (11)$$

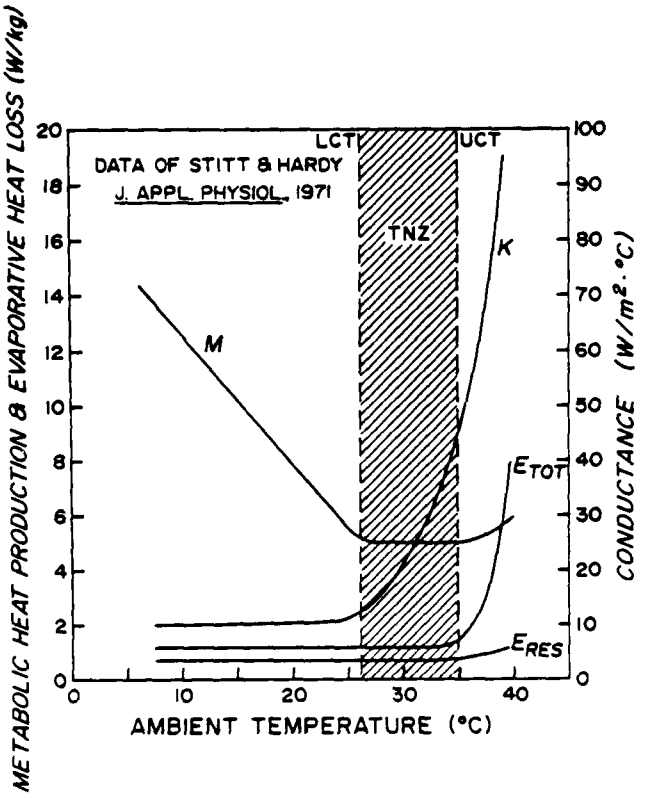

Figure 19. Thermoregulatory profile for the restrained squirrel monkey equilibrated to ambient temperatures ranging from 10 to 39 °C. Individual functions show metabolic heat production ($\underline{M}$), total evaporative heat loss ($\underline{E}_{tot}$), respiratory evaporative heat loss ($\underline{E}_{res}$), and tissue conductance ($\underline{K}$). The thermoneutral zone of vasomotor control (TNZ) encompasses ambient temperatures between the lower critical temperature (LCT) of 26 °C and the upper critical temperature (UCT) of 35 °C. Figure constructed from data in Stitt and Hardy (56).

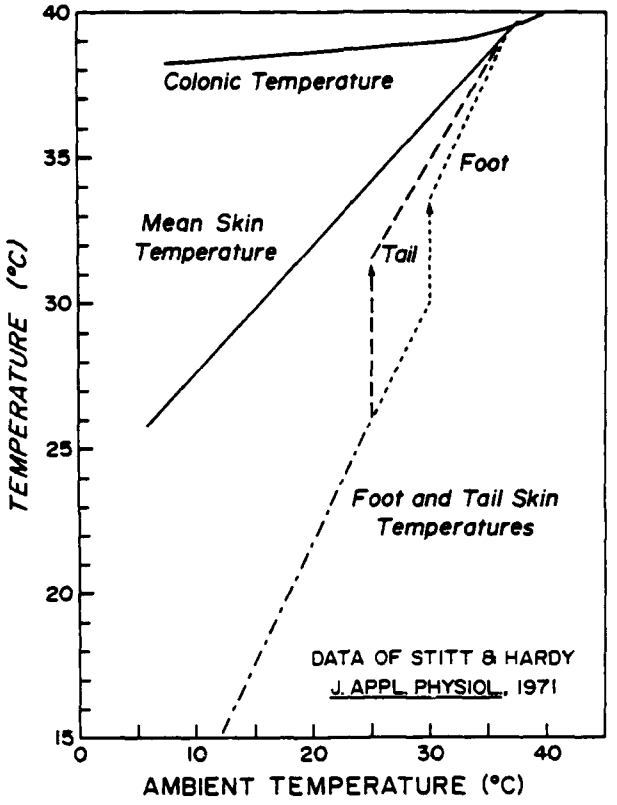

Figure 20. Body temperatures of the restrained squirrel monkey equilibrated to ambient temperatures ranging from 10 to 39 °C. Individual functions show deep colonic temperature, mean skin temperature based on four skin sites, and foot and tail skin temperatures. Arrows indicate vasodilation of vessels in tail and foot skin. Figure constructed from data in Stitt and Hardy (56).

Each animal underwent at least three experimental tests at each of the following T_a: 10, 15, 20, 25, 30, and 35 °C. A few tests were conducted at T_a=39 °C, but a steady state could not ordinarily be achieved since this T_a is very close to the animal's normal regulated internal body temperature. Figure 19 shows the thermoregulatory profile of the squirrel monkey derived from the data of Stitt and Hardy (56). The functions drawn in the figure are the lines of best fit to the data and show the mean steady-state levels of metabolic heat production ($\underline{M}$), respiratory evaporative heat loss ($\underline{E}_{res}$), total evaporative heat loss ($\underline{E}_{tot}$), and thermal conductance ($\underline{K}$) as a function of the T_a to which the animals were exposed. Steady-state T_{co}, weighted $\bar{T}_{sk}$, and T_{tl} and T_{ft},

measured in the same experiments, are shown in Figure 20. The data in Figures 19 and 20 represent the fundamentals of autonomic thermoregulation in the squirrel monkey. The individual responses are discussed in some detail in the following paragraphs.

When squirrel monkeys are restrained in cool environments, the body temperature is regulated by an increase in $\underline{M}$. All other responses are at low ebb (Fig. 19). The figure shows that as T_a falls below about 26-27 °C, $\underline{M}$ increases linearly at a rate of about 0.35 (W/kg)/°C. Active shivering may be observed at T_a=20 °C and below. At T_a = 26-27 °C, designated the lower critical temperature (LCT), resting heat production reaches its nadir and remains at this low level throughout the range of T_a that is designated the thermoneutral zone (TNZ) (T_a=25-35 °C). Stitt and Hardy (56) found the resting heat production of chaired animals ranged from 4.5 to 7.0 W/kg in the TNZ. Other data (8,49) confirm this general range although the particular values obtained will depend on the air movement in the test chamber as well as the method used to measure O_2 consumption. Excessively warm environments, e.g., T_a above 35 °C, threaten effective thermoregulation in this species. An increase in $\underline{M}$ has been reported under such conditions that is presumably related to increased behavioral activity. The upturn in the $\underline{M}$ function in Figure 1 is likely the result of struggling against the restraining chair as the animal attempts to escape from the warm environment. Exposure of these animals to microwave fields at T_a=35 °C and above can be threatening (1).

Figure 19 displays two functions for evaporative heat loss, that from the body as a whole, $\underline{E}_{tot}$, and that from the respiratory tract, $\underline{E}_{res}$. The difference between the two functions represents evaporative heat loss through sweating, $\underline{E}_{sw}$, and a minimal amount of passive diffusion of water through the skin (commonly designated "insensible perspiration"). The latter tends to be constant and is insignificant for thermoregulation. Both $\underline{E}_{tot}$ and $\underline{E}_{res}$ are low and constant at T_a below about 30 °C. At T_a above the upper critical temperature (UCT) of 35 °C, $\underline{E}_{tot}$ becomes significantly elevated above the basal level, whereas $\underline{E}_{res}$ changes hardly at all. This fact implies the major avenue of evaporative heat loss in this species is sweating, not panting, an implication confirmed by Nadel and Stitt (50) who recorded changes in sweat rate from the foot of squirrel monkeys restrained in warm environments. The sweating is

truly thermoregulatory because its rate can be altered by changing the local tissue temperature of the PO/AH thermoregulatory center (57). Because sweating in this species can be emotional as well as thermoregulatory, it is important to measure concomitant changes in $\underline{M}$ during experiments in which sweating is anticipated.

Figure 20 illustrates the relationship between various body temperatures and T_a as measured by Stitt and Hardy (56). Mean T_{co} is regulated between 38.5 and 39.8 °C over the range of T_a from 10 to 39 °C. There is a slightly higher rate of increase above the TNZ than below. The weighted $\overline{T}_{sk}$ increases linearly with T_a; the slope of the function will be higher when the air in the test compartment is moving, rather than still. The figure also shows dramatic discontinuities in the temperature functions for foot and tail skin. These discontinuities occur at discrete T_a and reflect vasodilation of the peripheral blood vessels of the tail and foot. Warm blood from deep in the body is brought close to the skin surface when these vessels vasodilate, aiding the transfer of metabolic heat to the environment. Changes in vasomotor tonus provide the means for efficient thermoregulation in the squirrel monkey across the entire TNZ. The precise T_a that produce vasodilation of the tail and foot were pinpointed by Lynch et al. (47), who measured steady-state $\overline{T}_{sk}$ of monkeys restrained in many discrete T_a. At T_a of 26.5 °C for the tail and 32 °C for the foot, these local T_{sk} varied widely, indicative of wide variation in local tissue blood flow. Similar results have recently been reported by Adair and Adams (7).

The vasomotor response is truly thermoregulatory in nature; experimental warming of the PO/AH thermoregulatory center in animals equilibrated to sub-threshold T_a produces prompt vasodilation of both tail and foot vessels (46,47,56). However, the tail appears to exhibit a much greater degree of vasodilation than does the foot; the range of T_{tl} during dilation is about 7 °C, while the corresponding range for T_{ft} is about 3 °C. Since the surface area of the tail represents 11% of the total surface area of the body (cf. Eq. 11), this organ has the capability of dissipating significant amounts of metabolic heat to the environment and will play a major role in thermoregulation when animals are exposed to microwave fields at thermoneutral T_a.

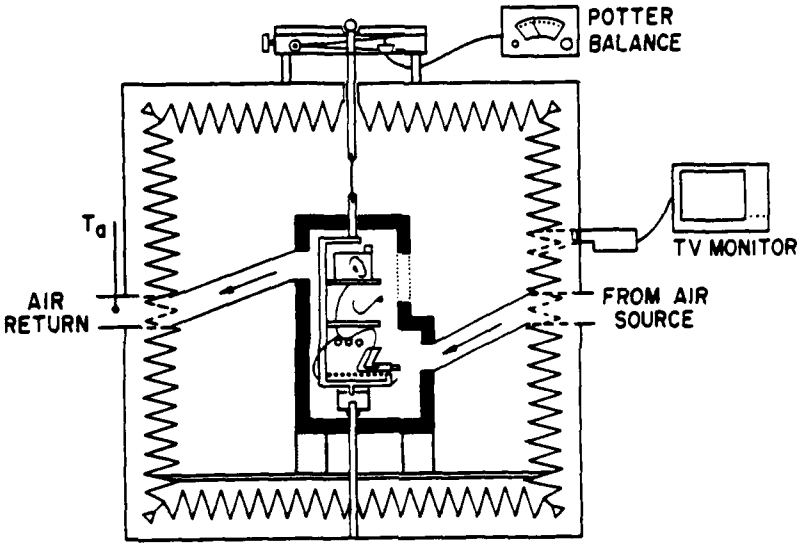

Figure 21. Schematic diagram (as viewed from the horn antenna) of the convective system that provides climate conditioning of the animal's test box inside the anechoic chamber. The system for suspending the animal's restraining chair from a sensitive Potter balance is also shown. Constant video surveillance of the animal is possible through a window in the wall of the test box.

We previously stated that tissue conductance, K, is an indicant of peripheral vasomotor state and represents the flow of heat from the body core to the skin. Figure 1 shows there is a sharp increase in K at or near the lower end of the TNZ, increasing dramatically as the T_a increases. At T_a below the LCT, K is minimal and constant; in other words, the peripheral vasculature of the squirrel monkey is maximally vasoconstricted in T_a below 26 °C. Adair and Adams (7) demonstrated under these conditions, that vasodilation of the tail can be provoked by whole-body exposure of the animal to a microwave field.

Methods and Procedure

Four adult male squirrel monkeys (Saimiri sciureus) were used as subjects in this study. During the experiments, individual animals were chair restrained in the far field of a horn antenna inside an electromagnetically anechoic chamber (see METHODS). The chair was enclosed by a Styrofoam box as shown in Figure 21. Air from a temperature-controlled (±0.5 °C) source

circulated at 0.36 m/s through the box in the direction shown in the figure. This arrangement provided a closely regulated thermal environment for the test animal. The temperature of the air inside the box (T_a) was sensed by a copper-constantan thermocouple located in the air outlet from the anechoic chamber and recorded continuously on a strip chart. The temperature of the interior of the Styrofoam box was also recorded for comparison purposes. The test sessions were conducted in the presence of a masking noise, and the monkey was under constant video surveillance at all times.

The restraining chair was suspended from the platform of a sensitive (± 1 g) Potter triple-beam balance (Potter Mfg. Co., Model No. 82) which was mounted on the roof of the anechoic chamber directly above the location of the Styrofoam test box. A Plexiglas yoke attached to the neck plate of the restraining chair accepted the suspension rod. A 10-cm length of nylon line incorporated in the suspension reduced the torque on the balance platform. A Teflon pin screwed into the base of the chair rode freely within an adjustable cardboard bushing when the chair was in place; this prevented the chair from swinging laterally when the monkey moved. A notched card attached to the base of the chair engaged a small Styrofoam block cemented to the box wall; this arrangement prevented rotational movement of the chair. These minimal-friction impediments to chair movement (diagramed in Fig. 22) permitted accurate recordings of total body weight loss during the experimental sessions; an example is shown in Figure 23.

During the experimental test sessions, the following body temperatures were sampled at 1-min intervals by an on-line computer: T_{co} and four representative T_{sk} taken from the abdomen, tail, leg, and foot (56). All were measured with 36-ga copper-constantan thermocouples having a reference junction in a bath of melting ice and water (4,56). All thermocouple junctions were very small and constructed in configurations appropriate to the locus of application. All T_{sk} were air-skin interface temperatures: the thermal junction was in contact with the skin on one side and open to the air on the other side. To minimize perturbation of the microwave field, the lead wires were shielded and held out of alignment with the electric vector of the

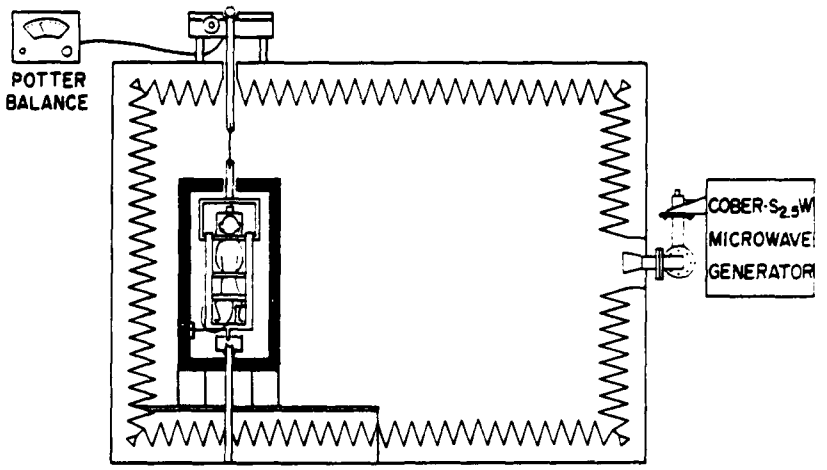

Figure 22. Schematic diagram (left elevation) of the location of the animal's test box inside the anechoic chamber. Box is located in the far field of the horn antenna. Additional details of the system for suspending the animal's restraining chair from the Potter balance are also shown.

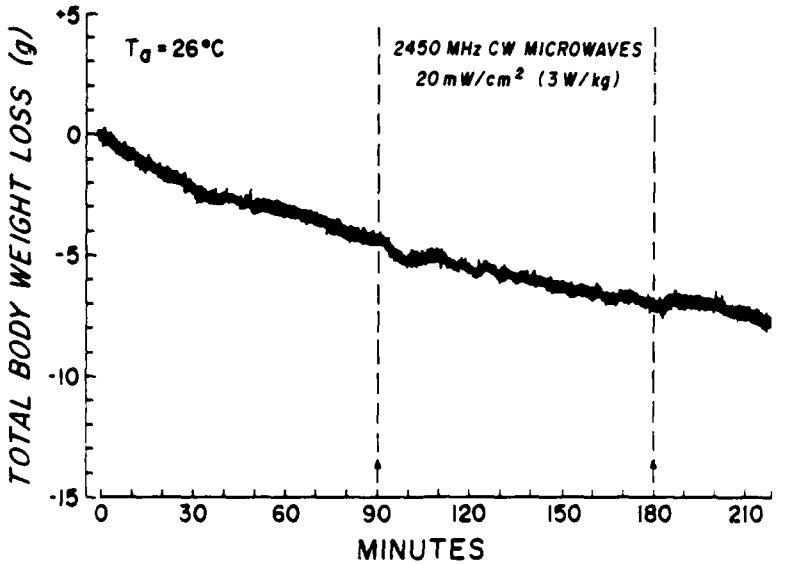

Figure 23. Representative strip-chart record of total body weight loss measured by the Potter balance during a single 220-min experiment on one monkey. After a 90-min equilibration to an ambient temperature (T_a) of 26 °C, monkey was exposed to 2450-MHz CW microwaves at a power density of 20 mW/cm² (SAR=3 W/kg) for 90 min.

incident planewave. Any thermocouple emf that showed evidence of electrical artifacts (i.e., abrupt voltage changes greater than 4 μV, equivalent to 0.1 °C, correlated with microwave onset or termination) was discarded as an inadmissible datum. A weighted $\bar{T}_{sk}$ was calculated from the four T_{sk} by the relation given in Equation 11 (58).

Oxygen consumption ($\dot{V}_{O_2}$) was measured using an open-flow draw system. Chamber air was drawn at a constant rate of 7 L/min through a Plexiglas hood over the monkey's head and thence outside the chamber through Teflon tubing. The oxygen partial pressure (P_{O_2}) deficit was measured downstream by a Beckman Model 755 paramagnetic oxygen analyzer that sampled the passing airstream at a rate of 0.3 L/min. Metabolic heat production ($\underline{M}$) was calculated from $\dot{V}_{O_2}$ assuming a constant RQ of 0.83 (37,54).

To measure minute-to-minute $\underline{E}_{res}$, a portion of the expired air was drawn through a T_{dp} sensing device developed at the Pierce Foundation Laboratory (30). From the T_{dp}, which was measured and recorded continuously, the vapor pressure of water in the sampled air was calculated by means of Antoine's equation (60):

$$P_{H_2O} = e^{a-b\,(T_{dp} + c)-1} \qquad (12)$$

where: P_{H_2O} = water vapor pressure (mm Hg)

$a = 18.67$
$b = 4030.18$
$c = 235$
} empirical constants for water

The water evaporated from the lungs, or $\underline{E}_{res}$, was then calculated from a modified gas equation:

$$\underline{E}_{res} = \frac{(M.W.)\ (\Delta P_{H_2O})\ AF}{R \cdot T_a} \text{ g/min} \qquad (13)$$

where: M.W. = molecular weight of water

P_{H_2O} = the difference in water vapor pressure in air before and after evaporation of water

AF = air flow (L/min)

R = gas constant 62.396

T_a = air temperature (°K)

Thermoregulatory sweating from the right foot was also measured with the same kind of T_{dp} sensing device. The monkey wore an L-shaped Plexiglas boot with the sole of the foot resting on a nylon support. Chamber air was drawn through the boot, at the rate of 1.9 L/min, and thence outside the chamber through Tygon tubing where the T_{dp} was measured and recorded continuously. Sweating rate ($\dot{m}_{sw}$) was calculated in the same manner as $\underline{E}_{res}$.

Two T_a were selected for study, 20 and 26 °C, principally because they had been studied earlier (4) in experiments involving the method of partitional calorimetry. They had been selected originally because they are representative of particular portions of the thermoregulatory profile of the squirrel monkey (cf. Fig. 19) that would exhibit maximal thermoregulatory alteration in the presence of an imposed microwave field. A T_a of 20 °C is well below the TNZ so that microwave exposure at low power densities should provoke a reduction of $\underline{M}$, at higher power densities should initiate vasodilation of first the tail and then the foot vessels, and at even higher power densities should initiate thermoregulatory sweating. Since the whole repertoire of thermoregulatory responses could be mobilized *seriatim* at this T_a, it afforded the maximal opportunity for determination of the tolerance limit for exposure to microwaves. A T_a of 26 °C is just below the LCT so that microwave exposure at low power densities should initiate vasodilation of the tail vessels, at higher power densities should initiate vasodilation of the foot and initiate thermoregulatory sweating. The tolerance limit for exposure to microwaves should be lower at this T_a than at T_a = 20 °C, but not as low as the 20 mW/cm^2 already determined at T_a = 32 °C (4).

At each T_a, the thermoregulatory consequences of exposure to several discrete power densities were explored. These power densities began at 30 mW/cm^2 and ranged upward until the tolerance limit (the power density at which thermal balance could not be maintained) was reached. At T_a = 20 °C, the power densities tested were 30, 45, 52.5, and 60 mW/cm^2; at T_a = 26 °C, the power densities tested were 30 and 45 mW/cm^2. At both T_a, data were collected on four or five monkeys; two sessions were conducted/monkey at each power density.

Each experimental test session on a single monkey involved equilibration of the animal for a minimum of 90 min to the prevailing T_a, followed by a

90-min exposure to 2450-MHz CW microwaves at a particular power density. A 20 - 30 min reequilibration period terminated the test session. Strict criteria for the acceptability of all data were established prior to the conduct of any experiments: Any test session in which a thermoregulatory steady state was not achieved, in either the initial equilibration period or under microwave exposure, was discarded. Malfunctions of test equipment and on-line computer were also grounds for discarding entire test sessions. Over the course of the experiment, approximately 40% of the conducted tests were discarded on these grounds.

In summary, the following dependent variables were measured in each experimental test session:

colonic temperature-----T_{co}
4 skin temperatures-----T_{sk}
abdomen----------T_{abd}
tail----------T_{tl}
leg----------T_{lg}
foot----------T_{ft}
mean skin temperature----$\bar{T}_{sk}$
oxygen consumption-------$\dot{M}$
T_{dp} of expired air-------$\dot{E}_{res}$
body mass----------$\dot{E}_{tot}$
T_{dp} of foot capsule air--$\dot{m}_{sw}$

All experimental test sessions were conducted in the morning to avoid possible circadian shifts in resting levels of thermoregulatory processes. At the start of the test, approximately 18 h had elapsed since the monkey's last meal. Before introducing the animal into the test environment, all equipment was checked and/or calibrated and a stable T_a was established in the test chamber. The chaired monkey was weighed on an electronic analytical platform balance before being instrumented with temperature probes, hood, and boot; a post-test weight determination served to check the accuracy of the total body weight loss measured during the course of the test. After the chaired animal had been suspended from the Potter balance inside the test compartment and all connections made to the measuring equipment, 80 ml of mineral oil was introduced into a trap in the base of the restraining chair; this prevented

evaporation of urine and feces during the experiment. The weight loss was tared to zero immediately before the start of the 90-min equilibration period. At specific times during the test session, 5-min baseline checks of the T_{dp} and O_2 content of the chamber air were made to track any possible drifts in baseline levels. Throughout the test session, all data were sampled at 1-min intervals by an on-line computer. Also, continuous strip-chart records were made of total body weight loss, T_{dp} of the expired air, T_{dp} of foot capsule air, and T_a. At the end of the test session, after the animal had been removed from the test compartment and returned to the home cage, a 10 to 20-min baseline check of the physical characteristics of chamber air completed the test data.

Results

Representative Data From Individual Experiments

The data from individual test sessions were analyzed in 5-min time bins (*i.e., means and standard errors* of each dependent variable were calculated at 5-min intervals across the duration of the test session). Sample experiments for each of the two test T_a (20 and 26 °C) are shown in Figures 24 and 25. The microwave power density differs between the two figures but represents the highest tested at the respective T_a. Data from two different monkeys are represented in Figures 24 and 25. Additional sample experiments, conducted at T_a = 20, 26 and 32 °C and at lower power densities, are contained in Adair (4).

Figure 24 presents data from 1 monkey equilibrated to a T_a of 20 °C and then exposed for 90 min to microwaves at a power density of 60 mW/cm^2. During the equilibration period, $\underline{M}$ was elevated to over 10 W/kg, T_{tl} and T_{ft} stabilized at low levels indicative of vasoconstriction, T_{co} stabilized at 38 °C, and $\underline{E}_{res}$ was minimal. At the initiation of microwave exposure, $\underline{M}$ immediately fell to the resting level (about 4.5 W/kg), and rapid increases occurred in both T_{tl} and T_{ft}. Colonic temperature rose slowly at first and then stabilized, after about 30 min, at a level about 1 °C higher than the baseline level. The two-phase elevation in T_{ft} reflects the initiation of vasodilation of the foot skin at 35 min into the exposure. At about the same time, sweat began to be secreted heavily from the foot. The rate of total body weight loss appeared to

decrease during the period of microwave exposure, increasing again when the microwave field was extinguished. A rapid reversal of all other response changes also accompanied extinction of the field. At this power density, the SAR is equivalent to twice the resting heat production of a squirrel monkey in a thermoneutral environment. All the thermoregulatory responses available to the animal were mobilized in sequence during this experiment, first the reduction of heat production and initiation of tail vasodilation, followed later by foot vasodilation and sweating.

Figure 25 presents data for another monkey equilibrated to a T_a of 26 °C and then exposed for 90 min to microwaves at a power density of 45 mW/cm^2. During the equilibration period, $\underline{M}$ was slightly elevated above the resting level; the local T_{sk} indicated that the tail skin was partially vasodilated but that the foot was vasoconstricted; T_{co} stabilized at about 37 °C; and $\underline{E}_{res}$ was minimal. At the initiation of microwave exposure, $\underline{M}$ fell rapidly to the resting level, the tail skin vasodilated completely and the foot skin partially so; moderate sweating was initiated and T_{co} stabilized about 0.8 °C above the baseline level. The measured increases in heat loss were mirrored by an increase in the rate of total body weight loss. All these trends were curtailed or reversed when the microwave field was extinguished. The reasons for the apparent incomplete vasodilation of the foot in this experiment are uncertain, but they may relate to manual reduction of the circulating T_a that was often required during high intensity microwave exposure to counteract heating of the air as it exited from the anechoic chamber.

A remarkable feature of nearly every experiment conducted at these two T_a was the stability of T_{co} during the period of microwave exposure; it never rose above 40 °C and it only seldom exceeded 39 °C. It is noteworthy that the SAR of 9.0 W/kg (at 60 mW/cm^2) was tolerated well at T_a = 20 °C. This SAR is the equivalent of approximately twice the resting $\underline{M}$ of the squirrel monkey at thermoneutrality.

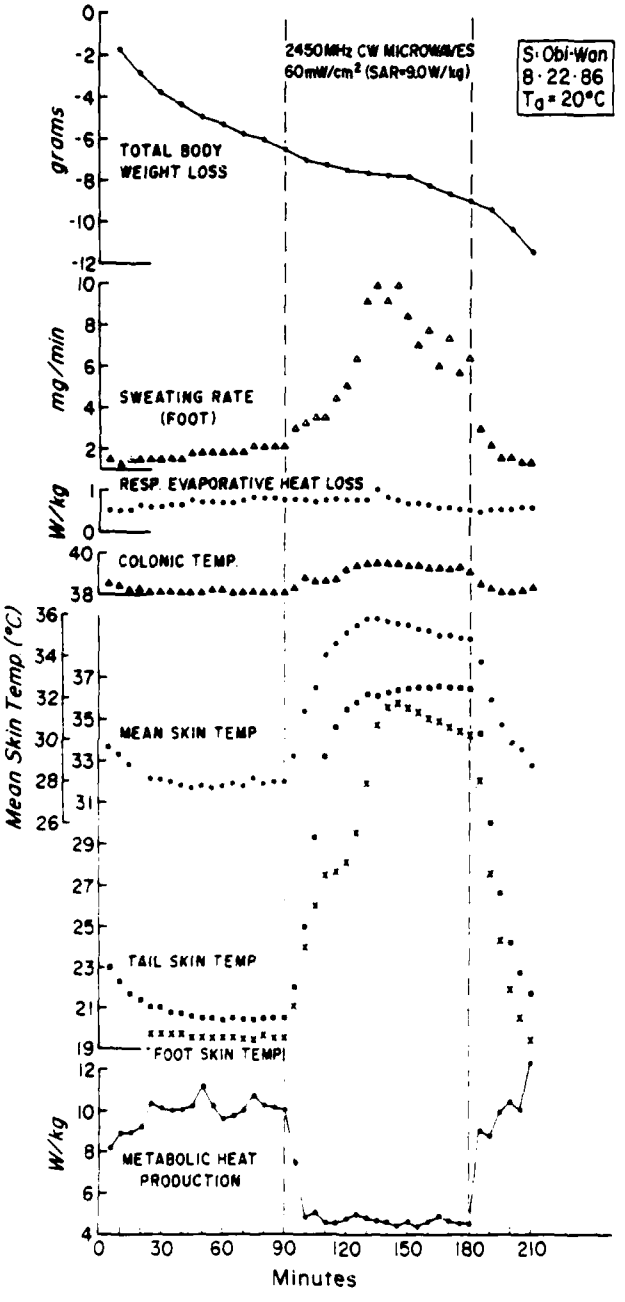

Figure 24. Representative experiment on one monkey equilibrated to an ambient temperature (T_a) of 20 °C to determine effects on autonomic responses of heat production and heat loss of a single 90-min exposure to 2450-MHz CW microwaves at a power density of 60 mW/cm^2 (SAR = 9.0 W/kg). Individual plotted points represent means of preceding 5 min.

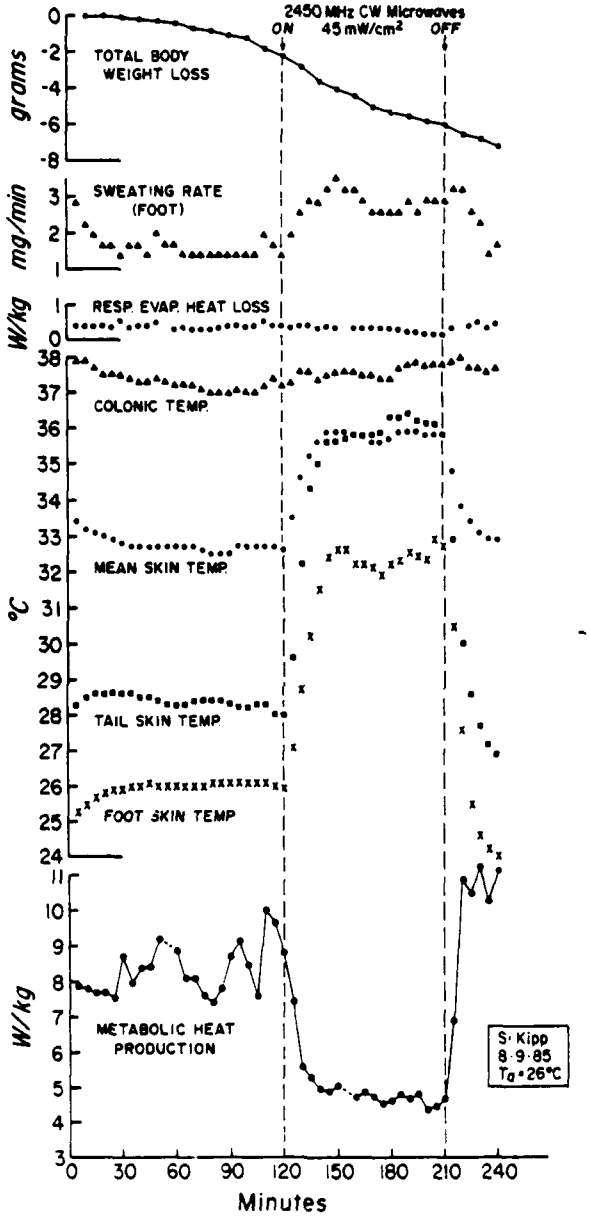

Figure 25. Representative experiment on one monkey equilibrated to an ambient temperature (T_a) of 26 °C to determine effects on autonomic responses of heat production and heat loss of a single 90-min exposure to 2450-MHz CW microwaves at a power density of 45 mW/cm^2 (SAR = 6.75 W/kg). Individual plotted points represent means of preceding 5 min.

Steady State Thermoregulatory Responses Measured During Microwave Exposure at Two T_a

Each experiment yielded two values of each dependent variable that could be used for further analysis: the first value represented the steady-state level just before the onset of microwave exposure when the animal was fully equilibrated to the prevailing T_a; the second represented the steady-state level when the microwave field was present. To obtain these values, means and standard errors were calculated across the final 20 min of the initial equilibration period and across the final 20 min of the 90-min period of microwave exposure. All of the means, for all animals at both T_a, are summarized in Figures 26 through 29. In each figure, particular dependent variables (e.g., $\underline{M}$, T_{co}) or derived variables (e.g., $\underline{K}$) are plotted as a function of both power density (mW/cm^2) and SAR (W/kg).

Figures 26 and 27 summarize all data collected during the present experiments at a T_a of 20 °C together with comparable data collected at lower power densities (10 to 25 mW/cm^2) in the earlier study (4). Each point plotted at zero power density represents the initial equilibrated value for one monkey, coded by symbol, in a single experiment; the steady-state value of that variable during microwave exposure is plotted at the appropriate power density.

In Figure 26, we see that little change occurs in T_{co} across the entire range of power density explored; in only 3 experiments did T_{co} attain or surpass 39 °C. Mean skin temperature increases very little through the power density range from 10 to 30 mW/cm^2 but increases more rapidly thereafter, indicating the onset of peripheral vasodilation. Even at 60 mW/cm^2, the T_{co}-to-$\bar{T}_{sk}$ gradient is 3 °C, sufficient to prevent runaway hyperthermia. The major change at this T_a is a reduction in $\underline{M}$ that is a linear function of the field strength up to 45 mW/cm^2. At this value, $\underline{M}$ is reduced to the resting level of about 4 W/kg. That the $\underline{M}$ reduction, across most of the power density range, is of the same magnitude as the SAR is clear because the open symbols (representing $\underline{M} + \underline{A}_{rir}$) fall along a horizontal line that lies at approximately the initial equilibrated level. This finding supports our earlier report of the equivalence between $\underline{M}$ reduction and SAR (8) and also supports the validity of our dosimetry. Figure 27 shows that $\underline{K}$, indicative of changes in vasomotor

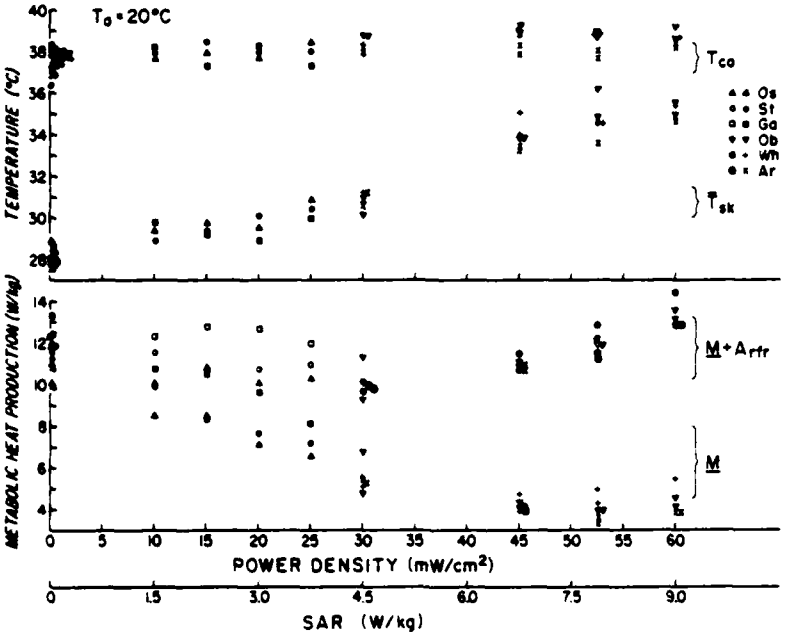

Figure 26. Mean steady-state colonic (T_{co}) and weighted mean skin ($\bar{T}_{sk}$) temperatures and metabolic heat production ($\underline{M}$) for all animals as a function of power density and SAR at an ambient temperature (T_a) of 20 °C. $\underline{A}_{rfr}$ = absorbed RFR (W/kg). Points plotted at zero power density represent equilibrated levels before microwave exposure.

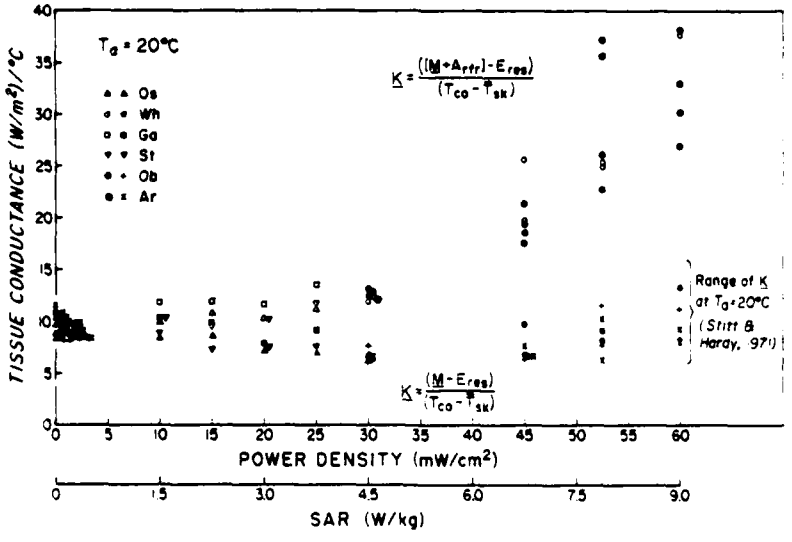

Figure 27. Mean steady-state values of tissue conductance ($\underline{K}$) for all animals as a function of power density and SAR at an ambient temperature (T_a) of 20 °C. Open symbols represent conductance calculated with ($\underline{M}$ + $\underline{A}_{rfr}$). Points plotted at zero power density represent equilibrated levels before microwave exposure.

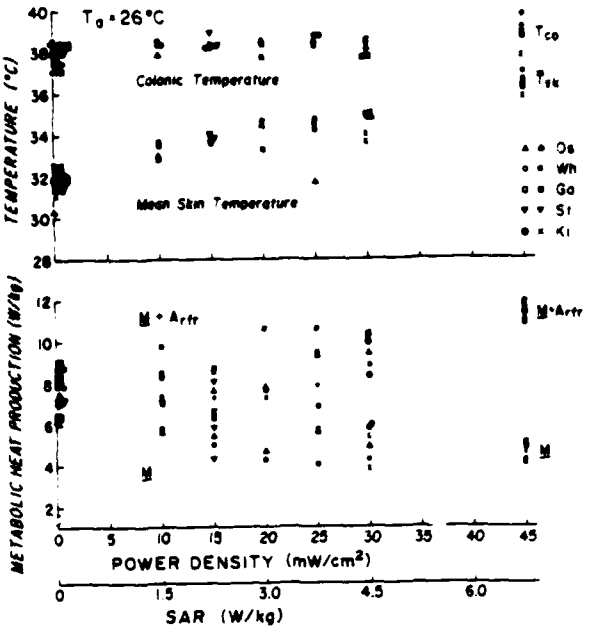

Figure 28. Mean steady-state colonic (T_{co}) and weighted mean skin ($\bar{T}_{sk}$) temperatures and metabolic heat production ($\underline{M}$) for all animals as a function of power density and SAR at an ambient temperature (T_a) of 26 °C. $\underline{A}_{rfr}$ = absorbed RFR (W/kg). Points plotted at zero power density represent equilibrated levels before microwave exposure.

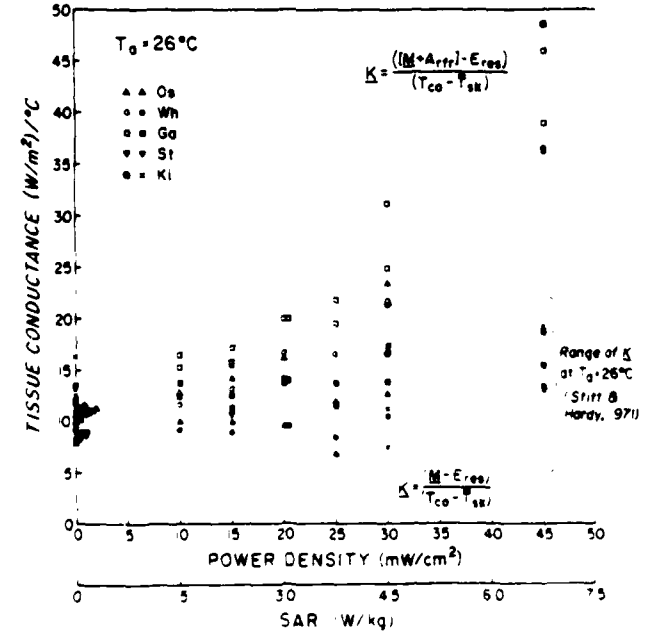

Figure 29. Mean steady-state values of tissue conductance ($\underline{K}$) for all animals as a function of power density and SAR at an ambient temperature (T_a) of 26 °C. Open symbols represent conductance calculated with ($\underline{M}$ + $\underline{A}_{rfr}$). Points plotted at zero power density represent equilibrated levels before microwave exposure.

tone, changes little across the range of power densities unless $\underline{A}_{rfr}$ is added to $\underline{M}$ during the calculations. In this case (open symbols), $\underline{K}$ begins to increase dramatically at power densities of 45 mW/cm^2 and above. This increase reflects active vasodilation in the peripheral vasculature of tail and foot (cf. Fig. 24).

Figures 28 and 29 summarize the data collected at T_a = 26 °C for all animals. Once again, the data collected in the present experiments are combined with those collected in the earlier study (4). Great stability of T_{co} is again evident at this T_a, with the possible exception of the 45 mW/cm^2 power density at which T_{co} increases by nearly 1 °C above the baseline level. Mean skin temperature rises gradually across the entire range of power density and the T_{co}-to-$\bar{T}_{sk}$ gradient becomes very small at 45 mW/cm^2. No animal could tolerate exposure at a power density greater than 45 mW/cm^2 at this T_a without developing hyperthermia. The lower panel of Figure 28 shows that a reduction of $\underline{M}$ is associated with microwave exposure at this T_a, but it is of small magnitude because the initial equilibrated level, at zero power density, is only 2 or 3 W/kg above the resting level. When $\underline{A}_{rfr}$ is added to $\underline{M}$, the sum increases slightly at the higher power densities. This augmented rate of energy production/absorption contributes to the increase in tissue conductance seen in Figure 29 at the two highest power densities. When $\underline{A}_{rfr}$ is added to $\underline{M}$ in the calculation of tissue conductance, $\underline{K}$ rises across the full range of power density explored. Thus, at a T_a of 26 °C, the principal effect of microwave exposure is an increase in $\underline{K}$, or blood flow, a conclusion already reached in the earlier study (4).

A plot of dry heat losses as a function of the skin-to-ambient temperature gradient for all data collected on all animals in the present study appears in Figure 30. In the figure, solid circles represent steady-state data collected during the initial equilibration period when no microwaves were present and the other symbols, coded in terms of power density, represent steady-state data collected during microwave exposure. The line of best fit, calculated by the method of least squares, is equal to the heat transfer coefficient (h) and has a slope of 5.39, a value close to that determined by Stitt and Hardy (56) for partitional calorimetry in still air, but a value considerably lower than that determined in our earlier study (4) at lower power densities. The distortion

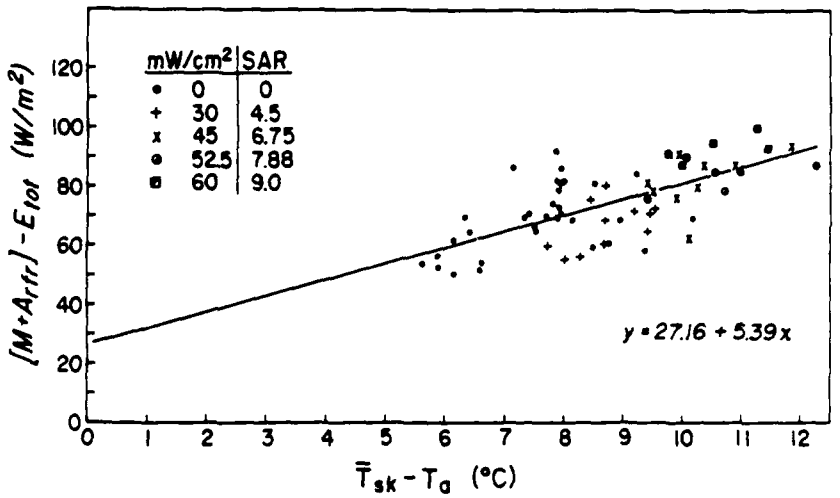

Figure 30. A plot of dry heat losses ($[\underline{M} + \underline{A}_{rfr}] - \underline{E}$) for all animals in the present experiments as a function of the skin-to-ambient temperature gradient ($\overline{T}_{sk} - T_a$). The function was fitted by the method of least squares.

of the present data may be attributed to two factors, both of which conspire to yield a function with a shallow slope: (1) for unknown causes, $\underline{E}_{tot}$, which is based on the total body weight loss, was overestimated, relative to the weight loss measured on the platform balance, in many of the experimental sessions; and (2) during exposure to 2450-MHz microwaves at high power densities, $\overline{T}_{sk}$ was often very high, yielding unusually high values of $(\overline{T}_{sk} - T_a)$.

A similar plot, containing baseline data only (no microwaves present) from both the present and the early studies, is shown in Figure 31. These data are most comparable to those of Stitt and Hardy (56), who calculated a heat transfer coefficient of 6.0 $(W/m^2)/°C$ for still air. The value of 7.85 $(W/m^2)/°C$ calculated for the function in Figure 31 reflects the higher rate of heat transfer when the air moves over the body surface, but is still considerably below the value of 12.3 $(W/m^2)/°C$ determined experimentally by Berglund (15) in our exposure facility. Since Berglund made his measurements in 1981, many subtle changes could have taken place in the convective air system that supplies our test facility, any of which might be responsible for a change in calculated heat transfer coefficient.

A plot of dry heat losses as a function of the skin-to-ambient temperature gradient for all data collected on all animals appears in Figure 32. Data from the present study are combined with data from the previous study (4) in this figure. Solid symbols represent steady-state data collected during the initial equilibration period when no microwaves were present, and the other symbols, coded in terms of power density, represent steady-state data collected during microwave exposure. The line of best fit, calculated by the method of least squares, has a slope that is equal to the heat transfer coefficient (h). The value of h for the combined data is 7.97 $(W/m^2)/°C$, slightly higher than that of the baseline data alone (Fig. 31). Since there can be no radiant or convective heat flow from the surface of the animal when $(\bar{T}_{sk} - T_a) = 0$, the function should pass through the origin. This result is not too far from the case ($y=8.44$ when $x=0$) indicating that the errors in the calorimetry employed in this study were minimal. Interestingly, most of the points trailing off the function at high values of $(\bar{T}_{sk} - T_a)$ were collected during microwave exposure at high power densities in the 20 °C environment. Of greatest importance, however, is the fact that a single function describes all the data collected from both microwave and non-microwave conditions. As already pointed out in the previous study (4), this fact indicates that the thermoregulatory system deals with energy absorbed from radiofrequency fields in exactly the same way as energy produced by the body during normal metabolic processes. In other words, heat generated in body tissues by absorbed microwaves should be regarded as no different from that deposited by more conventional (radiant or convective) sources or from excess metabolic heat produced during physical exercise.

Role of Skin Temperature in the Thermoregulatory Response to Microwaves

In a previous report (4), the partitional calorimetric data from an earlier study were analyzed in several different ways to probe the potential role of thermal receptors in the skin vs those deep in the body in the mobilization of thermoregulatory responses to microwaves. Since, at a frequency of 2450 MHz, the incident radiation may penetrate as deeply as 2.5 to 3.0 cm below the skin surface, there was a strong possibility of thermal stimulation of thermosensitive sites in the CNS (e.g., PO/AH, midbrain, medulla, spinal cord) during microwave exposure of the squirrel monkey at this frequency. However, comparisons of response changes as a function of concomitant T_{co} or $\bar{T}_{sk}$

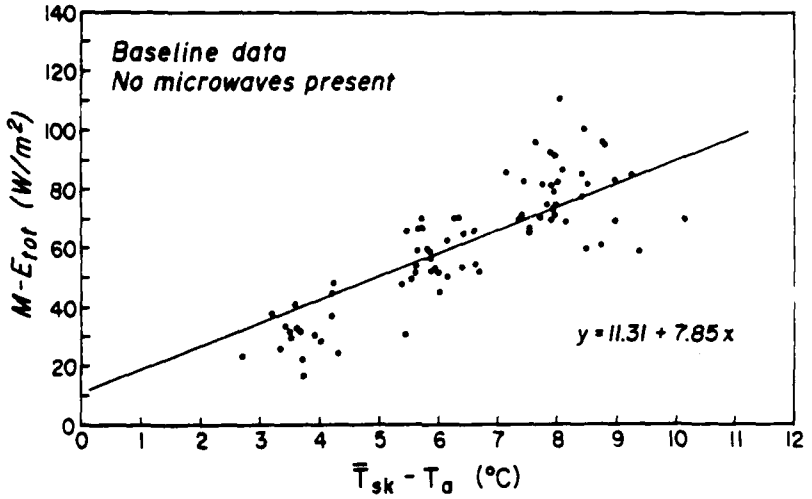

Figure 31. A plot of dry heat losses ($\underline{M} - \underline{E}_{tot}$) for all baseline data only as a function of the skin-to-ambient temperature gradient ($\bar{T}_{sk} - T_a$). The function was fitted by the method of least squares.

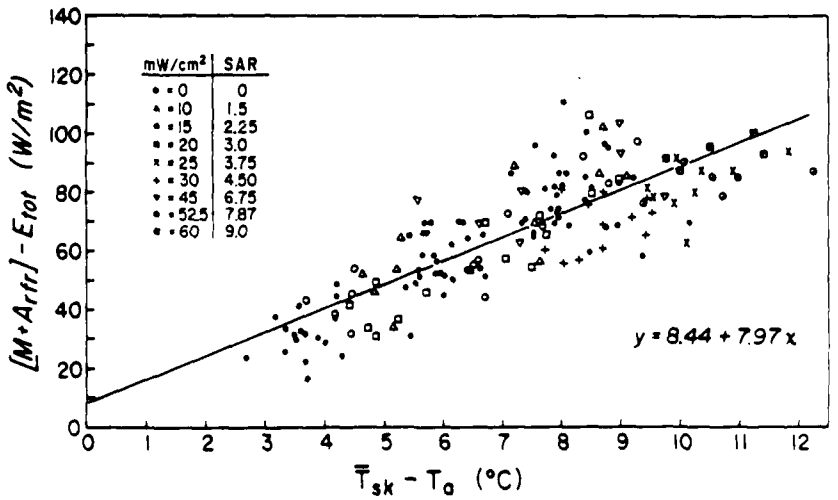

Figure 32. A plot of dry heat losses ($[\underline{M} + \underline{A}_{rfr}] - \underline{E}$) for all animals in both the present and previous studies as a function of the skin-to-ambient temperature gradient ($\bar{T}_{sk} - T_a$). The function was fitted by the method of least squares.

provided strong evidence for the preponderant role of thermal receptors in the skin in the mediation of changes in thermoregulatory responses of squirrel monkeys in the presence of 2450 MHz CW microwaves.

The partitional calorimetric data from the present study have been added to several of the figures that appeared in the earlier report. Figures 33-36 show T_{co}, $\underline{M}$ (with and without inclusion of $\underline{A}_{rfr}$), $\underline{K}$, and $\dot{m}_{sw}$ as a function of $\bar{T}_{sk}$. In all figures, each plotted point represents the steady-state value from one experimental session; open circles represent data collected during the initial equilibration period when no microwaves were present while closed symbols, coded in terms of power density, represent data collected during microwave exposure.

Functions for the steady-state levels of T_{co} (upper panel) and $\underline{M}$ (lower panel) appear in Figure 33 as a function of $\bar{T}_{sk}$. In both panels, the open circles (no microwaves present) fall naturally into three groups that reflect the three T_a under investigation. The other symbols (microwaves present) fall in orderly fashion between these three groups such that the aggregates in both cases describe continuous functional relationships between the two variables. There is no evidence in this figure that T_{co} or $\underline{M}$ in the presence of microwaves is any different from these same responses when the skin is warmed by convection and radiation. The functional relationships presented in Figure 33 reinforce the earlier conclusion, derived from the function in Figure 32, that a thermoregulatory response in the presence of microwaves is not different from the normal response.

When $\underline{A}_{rfr}$ is added to the $\underline{M}$ values measured during microwave exposure, the $\underline{M}$ vs $\bar{T}_{sk}$ function becomes increasingly distorted, as shown in Figure 34. The distortion occurs not only at the highest T_a (32 °C) where $\bar{T}_{sk}$ is elevated by the warm surroundings, but also at the lowest T_a (20 °C) under conditions of microwave exposure at high power densities where $\bar{T}_{sk}$ is elevated by substantial energy deposition therein.

Figure 35 shows the steady-state $\underline{K}$ as a function of $\bar{T}_{sk}$ for all animals at all T_a. In this figure, the symbols have the same meaning as in Figure 34. Once again, all points fall in orderly fashion along what appears to be a

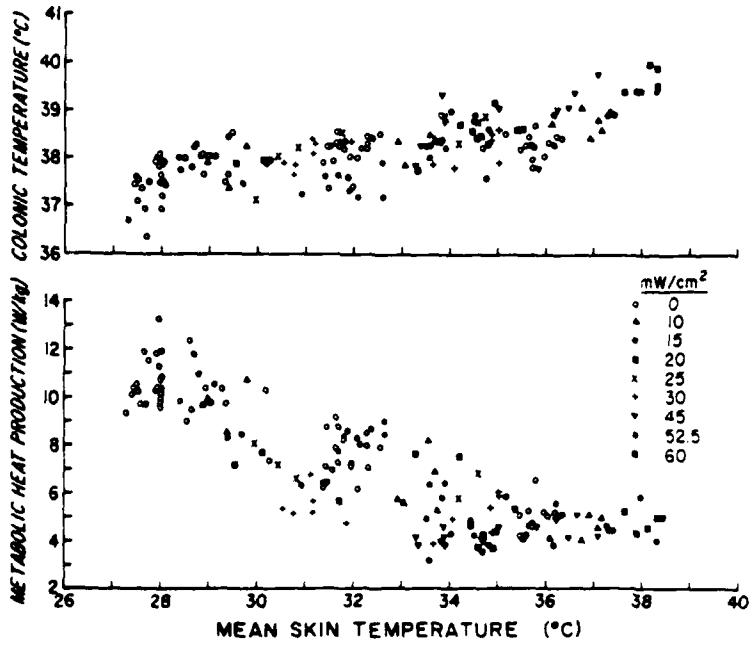

Figure 33. Steady-state colonic temperature (top panel) and metabolic heat production (bottom panel) as a function of weighted mean skin temperature. Open circles (power density and SAR = 0) represent data from baseline equilibration period when microwaves were absent. Other symbols, coded by power density and SAR, represent data from microwave exposure period.

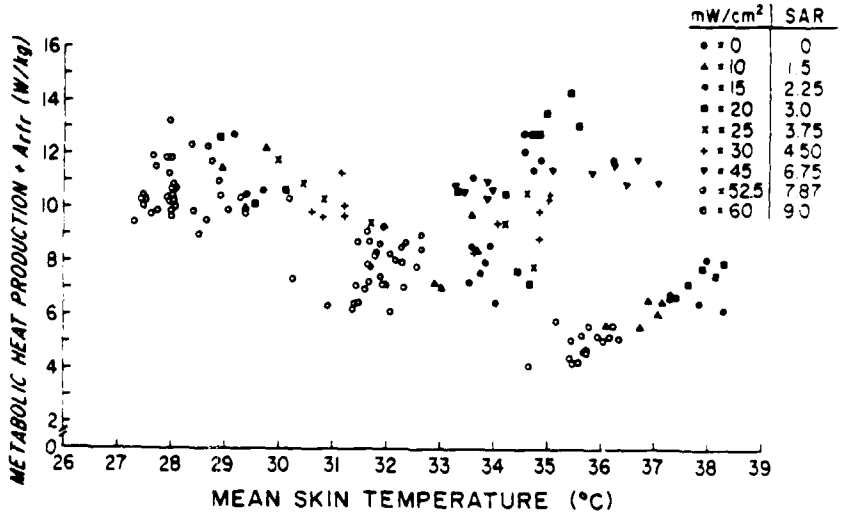

Figure 34. Steady-state values of metabolic heat production plus absorbed RF energy ($\underline{A}_{rfr}$) as a function of weighted mean skin temperature. Open circles ($\underline{A}_{rfr}$ = 0 W/kg) represent data from the baseline equilibration period when microwaves were absent. Other symbols, coded by SAR, represent data from microwave exposure period.

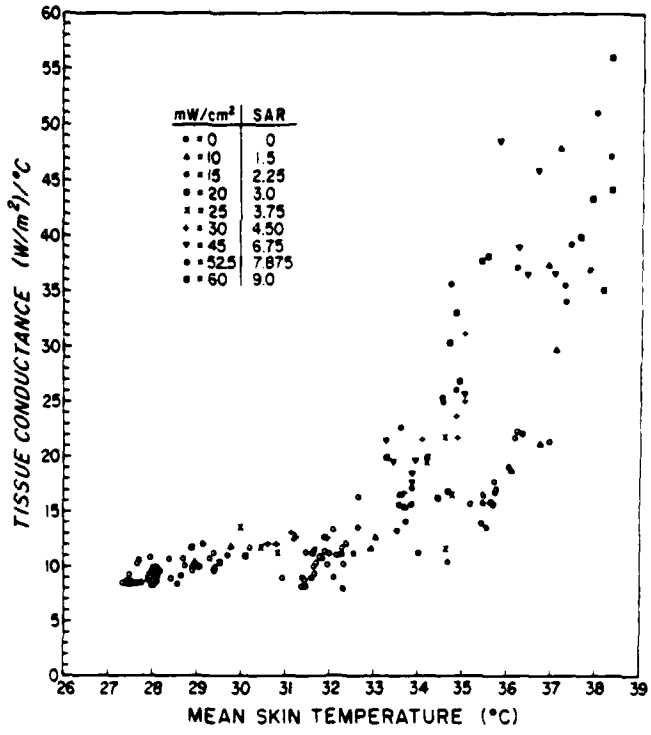

Figure 35. Steady-state tissue conductance as a function of weighted mean skin temperature. Open circles (power density and SAR = 0) represent data from the baseline equilibration period when microwaves were absent. Other symbols, coded by power density and SAR, represent data from microwave exposure period.

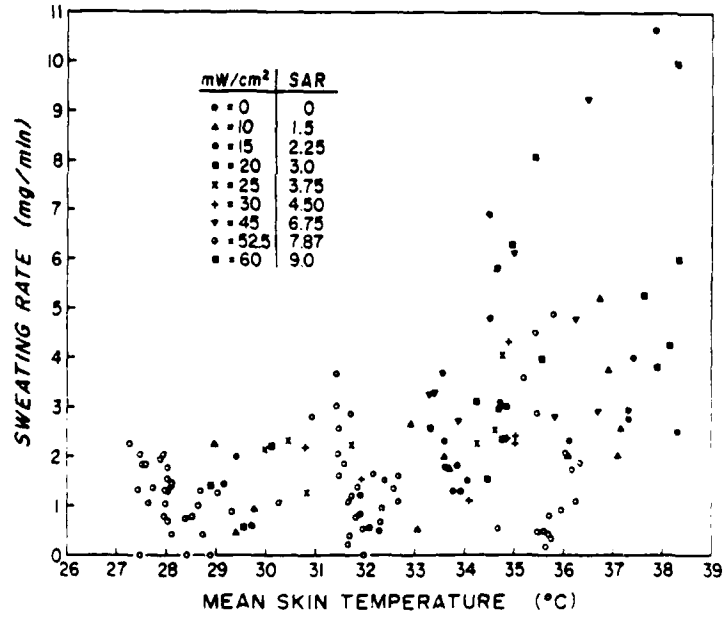

Figure 36. Steady-state sweating rate from the foot as a function of weighted mean skin temperature. Open circles (power density and SAR = 0) represent data from the baseline equilibration period when microwaves were absent. Other symbols, coded by power density and SAR, represent data from microwave exposure period.

single function with no indication that the response in the presence of microwaves is substantially different from the normal response. The same characterization applies to the data presented in Figure 36, which shows the steady-state $\dot{m}_{sw}$ as a function of $\overline{T}_{sk}$ for all animals at all T_a. In this case, however, the points do not group themselves quite as tightly together, presumably the result of the greater inherent variability in the sweating response from animal to animal (1). Nearly all of the upper points that represent high sweating rates were contributed by a single animal, an extremely "efficient sweater." In summary, Figures 33 through 36 provide convincing evidence of the involvement of $\overline{T}_{sk}$ in the thermoregulatory responses of squirrel monkeys to 2450-MHz CW microwaves across a representative range of T_a and a wide range of power density that extends from just above threshold to the maximally tolerable level. This evidence reinforces the conclusion, drawn in the previous report (4), that under these experimental conditions absorbed microwaves are dealt with in normal fashion by the thermoregulatory system.

Thermal Tolerance to High-Intensity Microwave Fields

In Figures 26 through 29, as well as the comparable figures in the previous report (Fig. 12 through 18 in [4]), the points plotted at zero power density represent the steady-state level of each dependent variable just before the onset of microwave exposure when the animals were fully equilibrated to the prevailing T_a. These points also appear in Figures 30 through 36, coded as power density and SAR = 0. At the T_a selected for study, 20, 26, and 32 °C, the values we measured fall within the range reported by Stitt and Hardy (56). Their data are summarized in Figures 19 and 20 of the present report. An analysis of the regulated levels of individual thermoregulatory responses or the changes that occur in individual responses during microwave exposure, particularly at high power densities, may afford insight into the possible basis for the tolerance limits we have determined at each T_a. These limits are apparently 20 mW/cm^2 at T_a = 32 °C, 45 mW/cm^2 at T_a = 26 °C, and 60 mW/cm^2 at T_a = 20 °C for whole-body (unilateral planewave, E-polarization) exposure of the squirrel monkey to 2450-MHz CW microwaves. Microwave exposure at field strengths higher than these limits results in thermal imbalance, heat storage in the body, and an uncontrolled rise in deep body temperature; i.e., the inability to attain a thermoregulatory steady state.

As described earlier in this report, specific responses in the thermoregulatory hierarchy are mobilized seriatim when a microwave field is imposed on the animal subject, responses that depend on the prevailing T_a. As an example, Figure 37 illustrates the steady-state $\underline{M}$, averaged across all animal subjects, as a function of imposed power density at the three T_a tested.

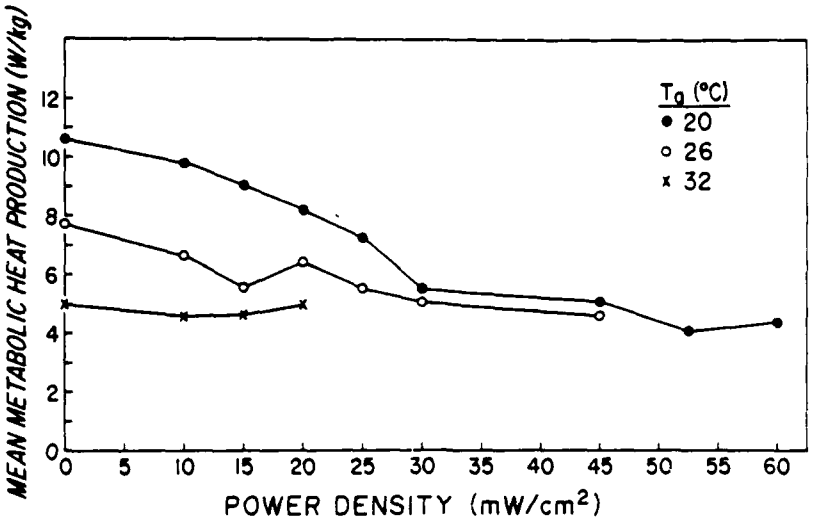

Figure 37. Mean metabolic heat production for all animal subjects as a function of the power density of microwave exposure. The parameter is ambient temperature (T_a).

It is evident from the low, constant $\underline{M}$ at all power densities at T_a = 32 °C that this response is not at all involved in thermoregulation in the presence of microwaves at this T_a. At T_a of 26 and 20 °C, $\underline{M}$ attains this low, constant level at power densities of 30 and 45 mW/cm^2, respectively. Since these power density levels are all below the microwave tolerance limit for the respective T_a, we may conclude that changes in $\underline{M}$ do not contribute to the breakdown of thermoregulation above the tolerance limit.

The response in the thermoregulatory hierarchy next to be mobilized when T_a rises, is increased blood flow to the periphery (increased tissue conductance) as illustrated in Figure 19. First the tail and then the extremities (e.g., the foot) vasodilate to bring warm blood from the core of the body to the periphery for cooling (Fig. 19). Vasodilation is also stimulated by microwave exposure at T_a below that at which the response occurs naturally

(4,7). The temperature of the skin surface in these vasoactive areas mirrors the change in vasomotor state and depends on the power density imposed. The mean change in T_{tl} and T_{ft} for all animal subjects is plotted in Figure 38 as a function of microwave power density at the three T_a tested. Significant upturns in a given function (e.g., between 15 and 20 mW/cm^2 at T_a = 26 °C for the tail) indicate the initiation of vasodilation in that organ at the prevailing T_a. Thus, while the tail is already fully vasodilated at T_a = 32 °C, this response is initiated at a power density of 15 to 20 mW/cm^2 at T_a = 26 °C and at a power density of 30 to 45 mW/cm^2 at T_a = 20 °C. Similarly, the foot vasodilates at a power density of 10 to 15 mW/cm^2 at T_a = 32 °C, 30 to 45 mW/cm^2 at T_a = 26 °C, and 52.5 to 60 mW/cm^2 at T_a = 20 °C. Because these responses are mobilized at microwave field strengths below the tolerance limit at each T_a, we must conclude that deficiencies in vasomotor responses do not contribute to the breakdown of thermoregulation above the tolerance limit.

The mean steady-state T_{co} and weighted $\bar{T}_{sk}$ at each T_a, together with the range of individual measured values, are illustrated in Figure 39. The great stability of T_{co} during exposure to microwaves (with the single exception of 20 mW/cm^2 at T_a = 32 °C) is dramatically illustrated here. In each environment, the average temperature of the skin continually approaches, but never becomes equal to, that of the body core as power density increases. This steady-state core-to-skin thermal gradient ($T_{co} - \bar{T}_{sk}$) is plotted as a function of power density for all animals in all experiments in Figure 40. Although thermal intolerance and hyperthermia usually accompany the diminution to near-zero of the core-to-skin thermal gradient, under no conditions in these experiments does that gradient reach zero. In fact, the cooler the prevailing environment, the higher is the asymptotic value of the gradient. Some other factor, then, must contribute to the breakdown of thermoregulation above the tolerance limit.

Sweating rate from the right foot was measured continuously during all experimental test sessions as an index of evaporative heat loss. In the determination of heat balance (Eq. 1), heat lost through sweating ($\underline{E}_{sw}$) and respiratory evaporative heat loss ($\underline{E}_{res}$) are combined in the term $\underline{E}$. In the method of partitional calorimetry, $\underline{E}_{sw}$ is determined indirectly as ($\underline{E}_{tot}$ - $\underline{E}_{res}$). It is often useful, however, to have an independent means of assessing the onset and rate of thermoregulatory sweating. As for the other dependent

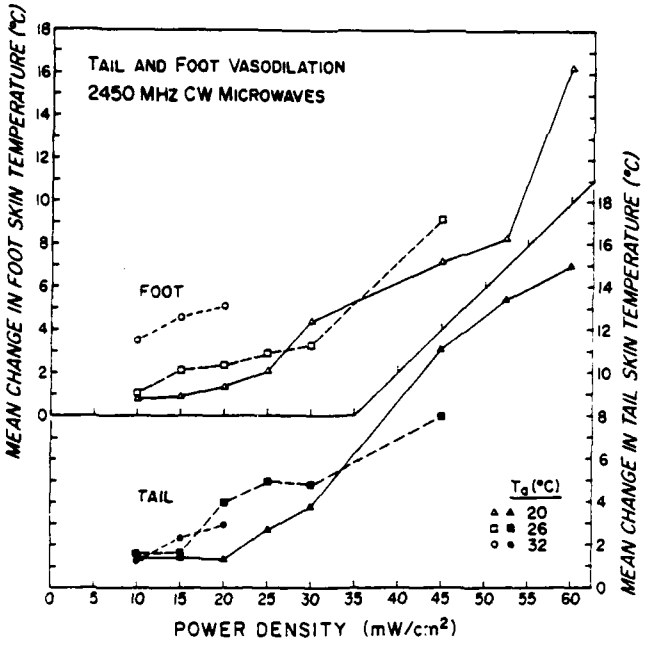

Figure 38. Mean change from equilibrated baseline level in tail skin temperature and foot skin temperature for all animals as a function of the power density of microwave exposure. The parameter is ambient temperature (T_a).

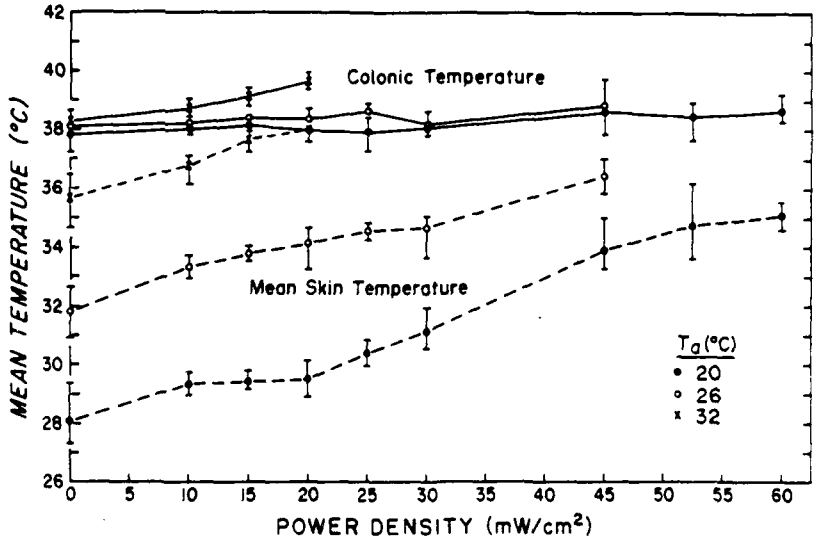

Figure 39. Mean colonic temperature and weighted mean skin temperature for all animals as a function of the power density of microwave exposure. Error bars indicate the range of individual data at that power density. The parameter is ambient temperature (T_a).

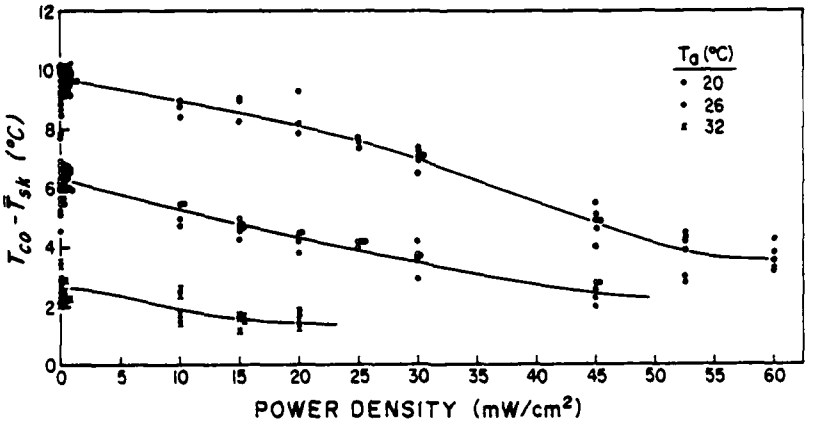

Figure 40. Mean values of the core-to-skin thermal gradient ($T_{co} - \bar{T}_{sk}$) for all animals as a function of the power density of microwave exposure. The parameter is ambient temperature (T_a).

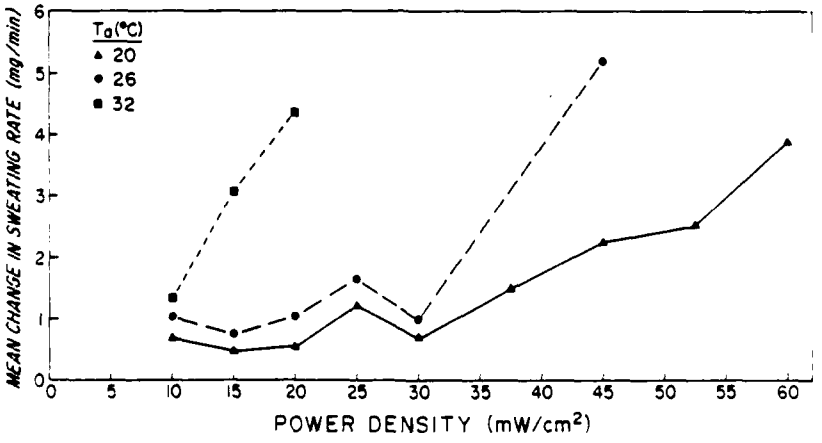

Figure 41. Mean change from equilibrated baseline level in sweating rate as a function of the power density of microwave exposure. The parameter is ambient temperature (T_a).

variables, $\dot{m}_{sw}$ was calculated for the final 20 min of the period of equilibration to the prevailing T_a and the final 20 min of the 90-min period of exposure to microwaves in each experimental test session.

Grand means were then calculated across animals, and the mean change in $\dot{m}_{sw}$ as a function of power density is shown in Figure 41. A notable increase in $\dot{m}_{sw}$ was stimulated by microwave exposure at a power density of 15 mW/cm^2 and above at T_a = 32 °C, at a power density of 45 mW/cm^2 at T_a = 26 °C, and at a power density of 52.5 mW/cm^2 and above at T_a = 20 °C. Because the squirrel monkey possesses eccrine sweat glands on only the palms and soles (48), the capacity to eliminate excess body heat by sweating in this species is severely limited. Stitt and Hardy (56) concluded that this limitation is the basis for their finding that these animals would suffer fatal hyperthermia if forced to remain for long periods at T_a of 38 °C or above. On the basis of the data presented in Figure 41, the inadequate capacity for heat loss through sweating also appears to precipitate the breakdown in thermoregulation during exposure to high-intensity microwave fields. Certain other nonhuman primates (such as Erythrocebus patas) and, of course, humans are far better equipped to dissipate large body heat loads through sweating. For these species, the tolerance limit for exposure to microwave fields should be correspondingly enhanced.

The thermoregulatory tolerance limits for the squirrel monkey exposed in the far field to 2450-MHz CW microwaves are summarized in Figure 42. The plotted points represent those conditions under which thermal balance was maintained both in terms of power density tolerated (Panel A) and SAR tolerated (Panel B). The abscissas are also different in the two panels to illustrate the dependence of thermal tolerance on the temperature of the environment (Panel A) and the average temperature of the animal's skin (Panel B). In both cases, the points describe curvilinear functions that extrapolate to meaningful values when power density or SAR approaches zero. At a T_a of 35 °C (Panel A), all of the thermoregulatory responses available to the animal have been mobilized (Figs. 19 and 20). Metabolic heat production is at the resting level, the animal is fully vasodilated, and sweating has been initiated. Further increases in T_a will lead only to increases in heat production, saturation of the limited sweating response, and hyperthermia. Panel B shows that the tolerance function extrapolates to a $\bar{T}_{sk}$ equal to the normal deep body temperature of the

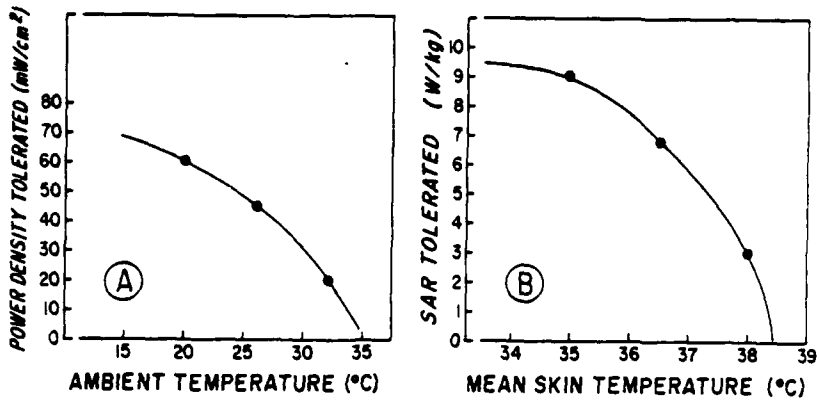

Figure 42. Tolerance functions for the exposure of the squirrel monkey to 2450-MHz CW microwaves as a function of the prevailing ambient temperature (Panel A) and mean skin temperature (Panel B). Panel A shows the power density tolerated and Panel B shows the equivalent SAR tolerated based on our dosimetry.

squirrel monkey. Thus, although the measured core-to-skin thermal gradient does not diminish to zero during these experiments (Figure 40), probably due to increases in deep body temperature, a $\bar{T}_{sk}$ equal to the normal core temperature is the natural limit to the maintenance of thermal balance during exposure to microwave fields as well as to warm environments.

The functions shown in both panels of Figure 42 can also be roughly extrapolated to the y-axis although this maneuver is far more uncertain because the exact shape of the function is unknown. Apparently, at T_a below 15 - 20 °C the microwave power density that can be tolerated is finite, approaching 70 mW/cm^2. This value is the equivalent of SAR = 9 W/kg, roughly twice the resting $\underline{M}$ of the squirrel monkey. Some pilot results of Experiment 1 in this report indicated that 10-min microwave exposures at a power density of 70 mW/cm^2 would be countered by the behavioral selection of a cooler environment. We do not know if this same field strength could also be dealt with in the steady state by changes in behavioral thermoregulation. Barring extraordinary increases in endogenous heat production in very cold environments, for which no experimental evidence exists, it seems reasonable to conclude that the

tolerance limit for autonomic thermoregulation in the cold is approximately 70 mW/cm^2. The tissues of the body should be protected against hyperthermalization by absorbed microwave energy at this level. It is important to recognize, however, that this tentative conclusion applies only to the specific exposure conditions explored in these studies and cannot be generalized to other frequencies, other species, or other experimental arrangements.

On the other hand, it is eminently feasible to generalize conclusions drawn from extrapolation of the upper end of the functions in Figure 42 (i.e., to warm environments [high $\bar{T}_{sk}$]) because of the high certainty that the shape of the extrapolated functions is correct. Because the tolerated power density (or SAR) approaches zero, the impact of microwave exposure on thermoregulation continually diminishes until it vanishes. Knowledge of the basic thermoregulatory capacity of the organism in question, exposed only to radiation and convection in thermally hostile environments, would then be invoked. Therefore, it seems safe to conclude that the upper tolerance limit during exposure to high-intensity microwave fields will coincide with the ceiling capability of the exposed organism to lose body heat through the evaporation of sweat. In this regard, man stands head and shoulders above all other earthly organisms.

The results of the present studies establish once again that a microwave environment is the thermal equivalent of a radiant or convective environment in terms of the basic functioning of the autonomic thermoregulatory system. Even under conditions in which a very high intensity field is suddenly introduced into the environment, the full range of autonomic thermoregulatory adjustments will be rapidly mobilized to protect the exposed organism from hyperthermia.

Conclusions

Partitional calorimetric studies have examined the autonomic thermoregulatory responses of squirrel monkeys exposed to 2450-MHz CW microwaves at two T_a, 20 and 26 °C. By progressively increasing the power density of the microwave field to higher and higher discrete levels, that power density above which thermal balance could not be maintained was determined for each T_a. To accomplish this end, heat production and heat loss responses were analyzed in terms of a heat balance equation which incorporated the energy absorbed from the

microwave field. The results of these studies allow the following conclusions to be drawn:

- In a 20 °C environment, thermal balance was maintained through an exposure power density of 60 mW/cm^2 by a reduction of metabolic heat production to the resting level, vasodilation of the tail and foot, and subsequent mobilization of thermoregulatory sweating.

- In a 26 °C environment, thermal balance was maintained through an exposure power density of 45 mW/cm^2 by the initiation of vasodilation in the tail and foot, and subsequent mobilization of thermoregulatory sweating.

- Earlier data had determined that in a 32 °C environment, thermal balance was maintained through an exposure power density of 20 mW/cm^2 by vasodilation of the foot and initiation of thermoregulatory sweating.

- These tolerance limits are related in curvilinear fashion to T_a and $\bar{T}_{sk}$, and can be understood in terms of the normal thermoregulatory capabilities of the squirrel monkey.

- Individual responses measured with and without microwaves present were described by the same functional relationships, indicating that the thermoregulatory system deals with microwaves in the same manner as other environmental energy sources.

- Thermal sensors in the skin, rather than those deeper in the body, were probably responsible for most of the response changes observed, a confirmation of a conclusion drawn in our 1985 report.

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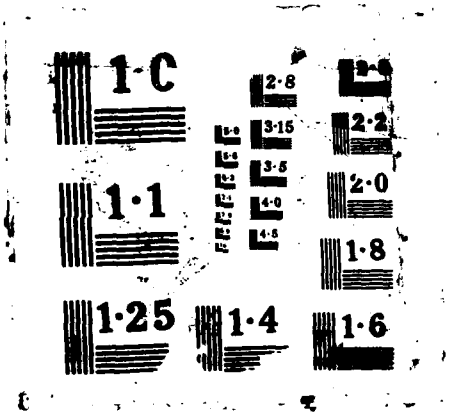
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