one good example. We have shown that the rate of change of acoustic cues rather than the linguistic nature of the stimuli, per se, may underlie this effect. Analysis of rapidly changing acoustic features may, in fact, play a critical role in the accurate perception of fluent speech by binding together phonetic segments so that at rapid transmission rates the temporal order and segmentation of speech may be preserved (*12*). It is in the areas of perception and production of rapidly changing sequential information that patients with language disorders have been found to be specifically impaired (*13, 14*). Tallal and her colleagues (*14, 15*) have suggested that a basic deficit in perceiving and producing rapidly changing sequential information may contribute significantly to the speech and language disorders of some aphasic patients.

We are not suggesting that the REA does not reflect superiority of the left hemisphere for processing linguistic material. Rather, the superiority of the left hemisphere for linguistic processing may reflect, at least in part, left-hemispheric dominance in processing rapidly changing acoustic events, which is critical for the processing of fluent speech.

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5. All subjects had normal hearing sensitivity as assessed by standard audiometry and no directional discrepancy between right and left ear thresholds of more than 5 dB across the frequencies between 500 and 4000 Hz.
6. The CV syllables, generated on the Haskins Laboratories parallel resonance synthesizer, formed a matched set differing from each other only in place of articulation, voicing, or both. Each stimulus was 250 msec long with an initial 5-msec broad-spectrum burst followed by a transitional formant period in which the formants moved toward the steady-state segment for the vowel /a/, which was identical for all stimuli. During the transitional segment, the frequencies changed linearly over time from the starting values to the steady-state values. For each of the six syllables there were two different fundamental frequencies: 114 and 110 Hz. Overall amplitude contours were identical for all stimuli.
7. B. Repp, *J. Acoust. Soc. Am.* **62**, 720 (1977). In pairing each CV syllable with each of the other five from that set, there were 15 combinations. For each combination, there were two channel assignments, yielding 30 configurations, with the restriction that for each pair of stimuli one had a starting fundamental frequency of 114 and the other 110 Hz. These 30 configurations yielded a total of 240 randomized trials.
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Microwaves Induce Peripheral Vasodilation in Squirrel Monkey

Abstract. *Vasomotor activity in cutaneous tail veins was indexed by changes in local skin temperature during exposure of the whole body to 12.3-centimeter continuous microwaves. At an ambient temperature (26°C) just below that at which tail vessels normally vasodilate, criterion dilation was initiated by 5-minute exposures to a microwave power density of 8 milliwatts per square centimeter. This intensity deposits energy equivalent to approximately 20 percent of the monkey's resting metabolic rate but produces no observable change in deep body temperature. Intensity increments of 3 to 4 milliwatts per square centimeter for 1°C reductions in ambient temperature below 26°C produced identical responses. That no vasodilation occurred during infrared exposures of equivalent power density suggests that noncutaneous thermosensitive structures may mediate microwave activation of thermoregulatory responses in the peripheral vasomotor system.*

In thermally neutral environments, the peripheral vasomotor response of warm-blooded (endothermic) species continuously provides fine control of body temperature. Physiological regulation of heat flow into or out of the body depends largely on autonomically controlled changes in the volume, rate, and distribution of blood supplied to the skin. The stimulus to constriction or dilation of cutaneous vessels is often peripheral as, for example, when localized or whole-body changes occur in the temperature of the skin (*1*). The stimulus can also originate centrally, in the absence of peripheral thermal events, when the deep body temperature rises (*2*). Rapid changes in peripheral vasomotor state have been produced in a variety of experimental animals by altering the temperature of the anterior hypothalamus with stereotaxically implanted thermode devices (*3*). In such experiments, dilation or constriction in highly vasoactive skin areas such as ears, tail, or extremities is often indexed by abrupt increases or decreases in local skin temperature.

Under specific exposure conditions and at relatively low intensities, electromagnetic energy of the microwave frequency range can produce body heating, often favoring deep tissues over the skin (*4*, *5*). Exposure to intense microwaves raises the body temperature of animal subjects and interferes significantly with ongoing behavioral and physiological processes, thereby upsetting thermal homeostasis (*6*). On the other hand, investigations into the biological effects of low-intensity microwaves (often dubbed "nonthermal") have largely ignored the thermoregulatory consequences of such exposure, although subtle thermal effects caused by exposure to power densities of 1 to 10 mW/cm^2 have been suspected but not demonstrated (*7*). We now report that monkeys in a cool environment can be induced to vasodilate by brief whole-body exposures to microwaves at intensities that produce no ob-

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servable change in deep body temperature. The characteristics of the evoked response resemble those produced by direct experimental heating of thermosensitive tissue in the hypothalamus.

Three adult male squirrel monkeys (*Saimiri sciureus*) served as subjects. They were restrained in a chair, one at a time, in the far field of a horn antenna inside an air-conditioned electromagnetically anechoic chamber (*8*). Rectal and four representative skin temperatures (abdomen, tail, leg, and foot) were monitored continuously with small copper-constantan thermocouples (*9*, *10*). After minimum 2-hour equilibration to a cool environment of constant temperature (range, 22° to 26.5°C), at which tail and extremities were fully vasoconstricted (*11*, *12*), the monkey underwent 5-minute exposures to 2450 ± 25 MHz continuous microwaves. Microwave power density (*10*), initially at a low level of 2.5 to 4 mW/cm², was increased at each successive exposure until a criterion tail vasodilation occurred. This criterion was defined by an abrupt and rapid rise in temperature of the tail skin that exceeded any increase in air temperature and that persisted after the end of the microwave exposure. Figure 1A shows an example of criterion tail vasodilation in one monkey equilibrated to an ambient temperature of 25°C. The microwave power density producing the response in this case was 10 mW/cm², which represents a whole-body energy absorption rate of 1.5 W/kg (*13*) or roughly 25 percent of the resting metabolic heat production of the squirrel monkey (*11*, *14*).

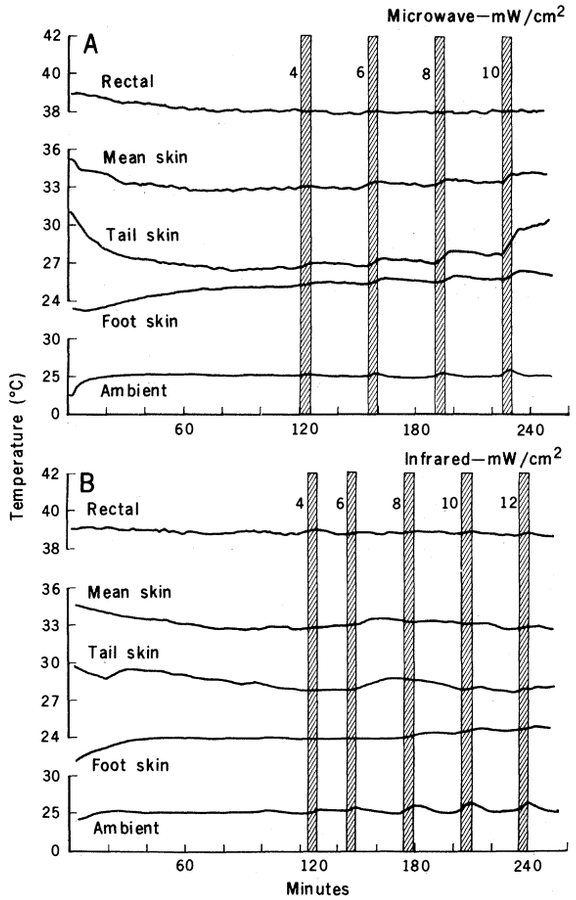

Fig. 1. Representative experiments showing effects of 5-minute exposures (hatched bars) to 2450-MHz continuous microwaves (A) or infrared radiation (B) on skin and deep body temperatures of squirrel monkeys equilibrated to a 25°C environment. (A) Exposure to microwaves of increasing power density (4, 6, 8, and 10 mW/cm²) were separated by 15-minute recovery periods. Criterion vasodilation of tail skin, evidenced by rapid and persistent rise in tail temperature, occurred at 10 mW/cm² and prevented a rise in deep body temperature. (B) Control exposures to infrared radiation of increasing incident power density failed to induce tail vasodilation despite significant elevations in ambient temperature.

Microwave power densities below that which initiated tail vasodilation often increased the temperature of the air or skin areas other than the tail. Control experiments (Fig. 1B) demonstrated that tail vasodilation was not initiated passively as a result of elevated air temperature. This monkey, also equilibrated to a 25°C environment, failed to exhibit any change in peripheral vasomotor state when exposed to infrared radiation of equivalent power density to the microwaves (*15*). Taken together, these results suggest that thermosensitive structures other than those in the skin may be responsible for altered thermoregulatory responses during microwave exposure.

The microwave power density required to stimulate criterion tail vasodilation was directly related to the environmental temperature in which the monkey was restrained. Figure 2A summarizes the data from 16 experiments on three animals at discrete ambient temperatures that range downward from 26.5°C, the temperature at which the tail vessels of a sedentary monkey may dilate spontaneously (*11*, *12*). The plotted points describe a linear relationship which reveals that, to initiate a criterion tail vasodilation response above threshold, an increase of 3 to 4 mW/cm² in microwave power density is required for every 1°C reduction of ambient temperature. A second abscissa relates the data to absorbed microwave energy based on our dosimetry (*13*). Thus, in a 23°C environment, when the animal's metabolic heat production is elevated 2.5 to 3 W/kg above the resting level (*11*, *14*), microwave energy deposited at a rate approximating this metabolic elevation will

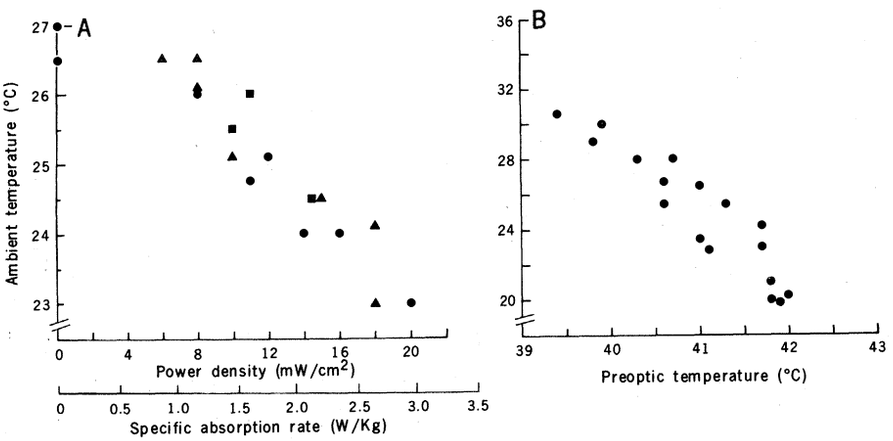

Fig. 2. Threshold functions for vasodilation of the squirrel monkey tail (A) produced by 5-minute whole-body exposures to 2450-MHz continuous microwaves and foot (B) produced by 5-minute periods of localized heating of the anterior hypothalamic-preoptic area through four implanted water-perfused thermodes. (A) Each data point represents the least microwave power density or absorbed microwave energy required to induce criterion tail skin warming in three monkeys at the ambient temperature indicated. (B) Each data point represents the least increase in preoptic temperature, measured bilaterally 2 mm from the thermodes, required to induce criterion foot skin warming in two monkeys at the ambient temperature indicated.

vasodilate the tail. Stability of the internal body temperature is thereby assured within the limits possible through changes in vasomotor state (uppermost tracing in Fig. 1A).

Thresholds for initiation of other thermoregulatory effector processes, such as shivering and panting, have been demonstrated to vary with both the ambient (skin) temperature and the local temperature either of the preoptic hypothalamus (*16*) or of other thermosensitive sites such as the spinal cord (*17*) as controlled by implanted thermodes. The form of such functions often resembles the relation presented in Fig. 2A. Recent research in our laboratory has determined how tail and foot vasodilation can be triggered by heating thermodes implanted in the hypothalamus of squirrel monkeys restrained in cool environments (*12*). Some of these results appear in Fig. 2B in a form that facilitates direct comparison with the adjacent microwave data. The striking resemblance lends credence to the hypothesis that low-intensity microwaves, absorbed in the vicinity of thermosensitive neural tissue in the hypothalamus and elsewhere (for example, posterior hypothalamus, midbrain, spinal cord, or deep viscera), can provoke immediate and dramatic changes in thermoregulatory effector response systems. Theoretical analyses (*18*) suggest that internal hot spots could occur under our experimental conditions (10 mW/cm^2, 2450-MHz microwaves) that would locally elevate temperature as much as 0.5°C. A possible neural mechanism would integrate many small afferent signals from diverse structures throughout the body into a strong effector command. The thermoregulatory neural substrate exhibits the diversity and integrative function appropriate to such a mechanism (*19*). Further, researches into the consequences of multiple thermal inputs confirm that the magnitude of the thermoregulatory effector response can be directly related to the number and sign of localized temperature changes occurring at discrete thermosensitive sites within the body (*20*).

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9. Thermocouples with 0°C reference junctions were constructed in special configurations from 36-gauge copper-constantan wire. Leads were shielded and held out of alignment with the *E* vector. Any thermocouple electromotive force showing abrupt changes greater than 4-μV coincident with microwave onset or termination was discarded as inadmissible datum. Field measurements (*10*) revealed no perturbations of the microwave field by the fine wires at the monkey's location.
10. Microwaves generated by a Cober (model S2.5W) source were fed to the antenna through standard waveguide components. Calibrations to determine far-field uniformity were made with a broadband isotropic radiation detector (Narda model 8306 B). Field intensity was mapped at 12-cm intervals across a 1 by 1.5 m plane passing through the center of the restraining chair location orthogonal to the incident microwave. The maximum nonuniformity of the central 50 by 50 cm of this plane, 8 percent with the chair absent, increased an additional 5 percent with the chair present. Power densities specified in this report were measured with the Narda probe positioned with chair present, at the location of the monkey's head.
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Elevated Blood Acetaldehyde Levels in Alcoholics and Their Relatives: A Reevaluation

The first evidence of elevated blood acetaldehyde concentrations in alcoholics (1.4 to 2.4 μM) compared with controls (1.1 to 1.9 μM) after ethanol intake was reported by Truitt (*1*). Concentrations of 11 to 45 μM in alcoholics and 4 to 30 μM in controls were reported by Korsten *et al.* (*2*). Schuckit and Rayses (*3*) reported elevated blood acetaldehyde concentrations in healthy young subjects with alcoholic relatives (65 to 78 μM) compared to control subjects (41 to 48 μM). This study indicated that the previously observed blood acetaldehyde dif-

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