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Heart Rate Changes Due to 5.6-GHz Radiofrequency Radiation: Relation to Average Power Density (41960)

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Abstract. Effects of intermittent exposure to 5.6-GHz radiofrequency radiation (RFR) on heart rate, blood pressure, and respiratory rate were examined in anesthetized rats. During exposure to 60 mW/cm^2 which resulted in a 1°C change in colonic temperature, heart rate increased; the values returned to control levels after exposure was discontinued. No changes in mean arterial blood pressure or in respiratory rate were observed. Exposure to 30 mW/cm^2 caused no significant changes in heart rate, blood pressure, or respiratory rate. The data indicate that heart rate changes during exposure to 5.6-GHz RFR are related to the average power density applied, and thus to the rate of change in temperature, and not simply to the absolute change in temperature.

The existing literature contains many conflicting reports concerning the effects of radiofrequency radiation (RFR)[2] on the cardiovascular system. Soviet investigators have suggested that RFR exposure may result in hypotension and bradycardia or tachycardia (1–3). However, other researchers have failed to replicate these results, and Kaplan *et al.* (4) have attributed changes in heart rate observed by some of the Soviet investigators to chance variations and mishandling of data.

Several Western investigations on the effects of RFR on the heart have been published. Tachycardia or arrhythmia produced by a direct, nonthermal action of pulsed RFR has been reported (5); however, other investigators (6, 7) could not confirm this effect. In other studies (8–10), bradycardia was observed during exposure of isolated hearts to 0.96-GHz continuous-wave (CW) radiation at specific absorption rates (SARs) between 1 and 10 W/kg. At SARs greater than 10 W/kg, heat-induced tachycardia was observed. Other investigators did not observe any effects of 2.45-GHz CW radiation on intact cat hearts (11) or isolated rat atria (12) at SARs of 2 and 10 W/kg.

Most studies on bioeffects of RFR have involved continuous exposure at power densities which either result in only small changes in body temperature or eventually result in lethal temperatures. The present study was designed to investigate effects of 5.6-GHz RFR, at power densities of 30 and 60 mW/cm^2, on heart rate, blood pressure, and respiratory rate in the anesthetized rat during intermittent exposures which resulted in 1°C colonic temperature changes. This frequency is characteristic of high-power stationary tracking radars for military applications and of naval ship radars.

Materials and Methods. Twenty-eight female Sprague–Dawley rats (Camm Research Lab Animals) weighing between 180 and 241 g (mean ± SE, 207 ± 7 g) were used in these experiments.[3] The animals were fasted for 12–24 hr (water *ad libitum*) prior to surgery. Ketamine HCl, 150 mg/kg im, was administered as a general anesthetic, with supplemental doses provided as necessary, and atropine sulfate, 0.04 mg/kg sc, was given to prevent excessive salivation. Evidence suggests that ketamine has less effect on thermoregu-

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[2] The term "radiofrequency radiation" is used in this paper to refer to nonionizing radiation at frequencies between 300 kHz and 300 GHz, and thus includes frequencies commonly referred to as "microwave radiation" (300 MHz to 300 GHz).

[3] The animals involved in this study were procured, maintained, and used in accordance with the Animal Welfare Act and the *Guide for the Care and Use of Laboratory Animals* prepared by the Institute of Laboratory Animal Resources–National Research Council.

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latory responses than other anesthetics (13). The left carotid artery was catheterized for measurement of blood pressure. Immediately after surgery, the animal was positioned on a Plexiglas holder in an RF exposure chamber. The arterial catheter was attached to a precalibrated blood pressure transducer (Statham P-23 Db), which was interfaced with a voltage/pressure coupler (Beckman 9853A). The signal was amplified and continuous recordings were obtained by use of a Beckman Dynograph (Type RM).

Respiratory rate was monitored by a pneumatic transduction method. The piezoelectric pressure transducer used in this system was originally designed for the detection of pulse pressure from the tail of a rat. A rubber bulb with a Teflon fitting was placed between the rat and holder. A semirigid polyethylene tube (3 ft in length) completed the connection to the pneumatic pressure transducer (Narco Biosystems, Inc.).

Conventional means of monitoring EKG, using metallic leads, have proven to be unsatisfactory under RFR conditions. The problems which occur under these conditions are alleviated by connecting 36-in. lengths of nylon-covered fluorocarbon leads (Polypenco-TFE) to the shielded leads (outside of the field). The nylon was removed from 1 to 2 cm at the ends of the leads which were subcutaneously attached to the animal. The shielded cables were, in turn, connected to an EKG coupler (Beckman) to give a permanent record via the Beckman Dynograph.

RFR exposures. Both continuous wave and pulsed RFR exposures were studied. The continuous wave fields were produced by a Model 1326 RF Power Source (Cober Electronics, Inc.) and transmitted by a Model 110C antenna (Struthers Electronics Corporation). The exposures were performed under far-field conditions, and the incident power density of the field was determined with an electromagnetic radiation monitor (Model 8316B, Narda Microwave Corp.) employing a Model 8323 probe. During exposures, the generator power was monitored constantly with a Model 436A power meter (Hewlett–Packard). Exposures were performed in an anechoic chamber (Rantec) at Brooks Air Force Base, Texas. The temperature and humidity of the chamber were monitored during all phases of the experiments.

For pulsed exposure studies, the fields were produced by a Model 2852 C Band Magnetron Source (Cober Electronics, Inc.) and transmitted by a Model 643 antenna (Narda Microwave Corp.). The power density of the field was determined, and the generator power was constantly monitored as described above. Pulse durations were 1 or 2 μsec and repetition rates were 250 or 500 pulses/sec. The pulsed exposures were performed in an Eccosorb RF shielded anechoic chamber (Emerson and Cuming, Inc.). The chamber temperature and humidity were monitored constantly.

Animals were exposed laterally in the H orientation (long axis of animal parallel to H field) to a frequency of 5.6 GHz at average power levels of 60 and 30 mW/cm^2. These power levels result in SARs of approximately 12 and 6 W/kg, respectively (14). Colonic temperature was monitored constantly with a Vitek 101 probe, the tip of which was inserted approximately 6 cm past the anus. When colonic temperature rose to 39.5°C, exposure was discontinued. Exposure was initiated again when the temperature returned to 38.5°C and was continued until the temperature rose to 39.5°C. This procedure was repeated for eight cycles. Exposures to greater than 1°C changes would have exceeded the physiological range of the animal's temperture. Tympanic and subcutaneous temperatures were monitored also. The probe for measuring tympanic temperature was inserted into the auditory meatus until an eyeblink reflex was elicited. For measuring subcutaneous temperature, the tip of the probe was inserted subcutaneously through a 0.5-cm skin incision into the midflank on the side of the animal facing the RFR antenna.

Statistical methods. Data obtained from repeated exposures were averaged for each animal, and were normalized to the values obtained at 38.5°C. Since no differences were observed between results obtained during continuous wave or pulsed exposures (using analysis of variance), the data from these two types of exposures were combined. Student's t test for paired data was used to determine if significant differences existed between values measured at 39.5°C and those at 38.5°C (control). Student's t test for unpaired data was used to determine if significant differences existed between values resulting from 30-

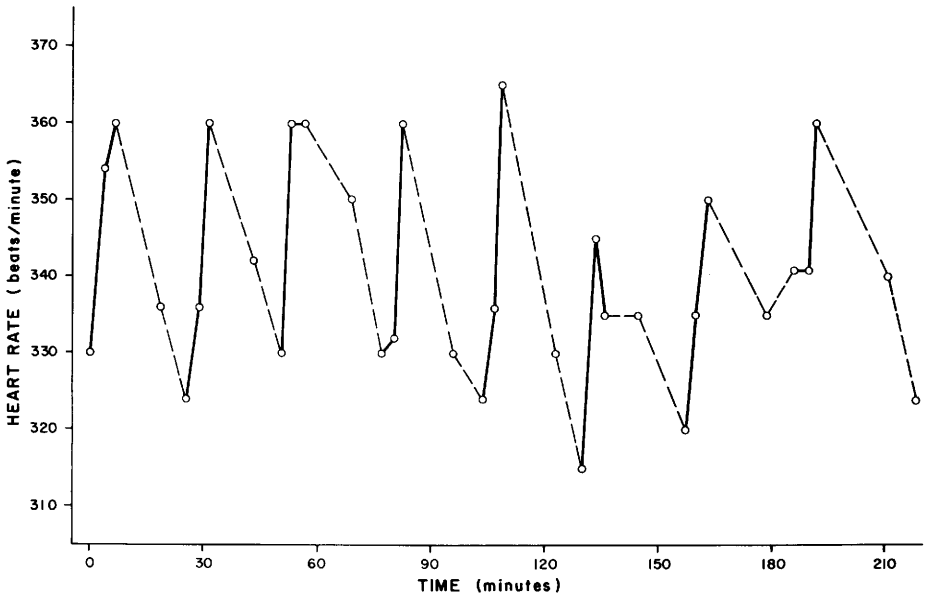

FIG. 1. Example of heart rate changes occurring in one animal exposed to 5.6-GHz RFR at a power density of 60 mW/cm^2. Solid line indicates when exposure was performed (starting at 38.5°C); dashed line indicates when power was off (starting at 39.5°C).

and 60-mW/cm^2 exposures. Regression analysis was used to determine if there was a significant correlation between the time required for a 1° rise in colonic temperature and the change in heart rate. A P value of less than 0.05 was considered to indicate significance in all tests. The data are presented as means ± standard errors of the mean.

Results. Figure 1 shows an example of changes in heart rate occurring during intermittent exposure to 5.6-GHz CW radiation at a power density of 60 mW/cm^2 in one animal. Each animal exhibited a similar response, with heart rate increasing during exposure periods and subsequently decreasing when exposure was discontinued.

In Fig. 2, changes in heart rate during exposure to RFR at power densities of 30 and 60 mW/cm^2 are presented. Exposure to 30 mW/cm^2 did not result in a significant change in heart rate (heart rate at 38.5° was 310 ± 5 bpm). Exposure to 60 mW/cm^2 resulted in a significant increase in heart rate (heart rate at 38.5 was 332 ± 7 bpm). The difference between the results obtained at the two power densities was significant. The average time for colonic temperature to increase 1° was over twice as long in animals exposed to 30 mW/cm^2 (14.9 ± 1.2 min) as in those exposed to 60 mW/cm^2 (6.3 ± 0.3 min); the difference was statistically significant. The times for temperature to return from 39.5 to 38.5° during recovery were similar (17.3 ± 1.1 min for 30 mW/cm^2 and 18.7 ± 0.7 min for 60 mW/cm^2). Regression analysis revealed a significant inverse relationship between the time required for a 1° rise in

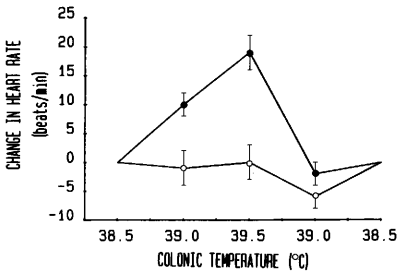

FIG. 2. Effects of 5.6-GHz RFR at 60 and 30 mW/cm^2 on heart rate. The data points in this figure were obtained by averaging the results of eight temperature cycles from all animals at the two power densities (an example of data from one animal is shown in Fig. 1). The data are normalized to the values obtained at 38.5°C. Thus, change in heart rate is presented rather than absolute values. Exposure was initiated at 38.5°C and discontinued at 39.5°C. ●, 60 mW/cm^2 (N = 14 animals); ○, 30 mW/cm^2 (N = 14 animals).

colonic temperature and the change in heart rate. Thus, a faster rise in colonic temperature resulted in a greater increase in heart rate.

No significant changes in mean arterial blood pressure or in respiratory rate were observed in either group. Values of mean arterial blood pressure at 38.5°C in animals exposed to 30 and 60 mW/cm^2 were 103 ± 8 and 102 ± 4 mm Hg, respectively, and at 39.5°C were 100 ± 5 and 107 ± 5 mm Hg, respectively. Respiratory rates at 38.5°C were 87 ± 5 and 91 ± 5 breaths/min in animals exposed to 30 and 60 mW/cm^2, respectively, and at 39.5°C were 84 ± 8 and 88 ± 3 breaths/min, respectively.

For a 1°C rise in colonic temperature, tympanic temperature increased 1.3 ± 0.1° and 1.4 ± 0.1° during exposure to 30 and 60 mW/cm^2, respectively. Subcutaneous temperature increased 1.9 ± 0.1° during exposure to both power densities. There were no statistical differences between these results at 30 and 60 mW/cm^2.

Discussion. Increased heart rates resulting from thermal stress have been reported previously (15–20). Kamon and Belding (21) reported a linear correlation between heart rate and rectal temperature. Phillips *et al.* (22) observed decreases in heart rates in restrained rats *following* exposures to 2.45-GHz RFR which raised colonic temperature to 40.5°C and greater. In the present experiments, after RFR exposure which raised colonic temperature to 39.5°C, heart rate also decreased, following the initial increase during the period of irradiation. Heart rate measurements were not performed during the period of irradiation in the work of Phillips *et al.* (22).

The effects of an absolute change in temperature and the rate of change in temperature on body temperature regulation are not well understood. It has been reported that certain thermal receptors are highly sensitive to the rate of change in temperature (23–27). Frey and Kenney (28) suggested that the rate of heating could affect the heart rate response to thermal stress. In the present experiments, the data indicate that heart rate changes during exposure to 5.6-GHz RFR are related to the average power level applied, and thus to the rate of change in temperature, and not simply to the absolute change in temperature. Exposures to a power density of 60 mW/cm^2 resulted in increases in heart rate, while exposures to 30 mW/cm^2 did not, even though in each case colonic temperature was raised by 1°C. The time to reach 39.5°C during exposure to 30 mW/cm^2 was over twice as long as that during exposure to 60 mW/cm^2.

Although the temperature cycles in the present experiments were based on colonic temperature, measurements of tympanic temperature, which is a better indicator of temperature in the thermoregulatory center in the brain, were performed also. The use of tympanic temperature as an indicator of hypothalamic temperature has been reviewed extensively by Benzinger (29) and Benzinger and Taylor (30). In the present experiments, although tympanic temperature exhibited a greater rise than colonic temperature, there was no significant difference between the absolute temperature results obtained at the two power densities. The time required for the similar changes in tympanic temperature was significantly greater in animals exposed to 30 mW/cm^2 than in those exposed to 60 mW/cm^2.

There was a greater increase in subcutaneous temperature relative to colonic and tympanic temperatures. This may have been due, in part, to the depth of RFR penetration. At the frequency of 5.6 GHz, surface heating will occur more readily than internal heating.

In work by other investigators, rats which were heated by raising environmental temperature exhibited an 8% increase in heart rate when colonic temperature increased from 38.5 to 39.5°C (31). In our present experiments, the change in heart rate during exposure to RFR at a power density of 60 mW/cm^2 was consistently observed; however it was only 6% greater than the baseline heart rate at 38.5°C. Due to this small magnitude of change and the fact that heart rate returned to baseline levels after RFR exposure was discontinued, it is doubtful that this phenomenon would have any serious long-term physiological consequences.

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