

THERMOGENETIC AND CARDIODYNAMIC REGULATION IN DOGS CRANIALLY EXPOSED TO 2450 MHz (CW) MICROWAVES*

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Abstract

The cranial area of dogs was exposed dorsally to 2450 MHz (CW) microwaves at different power densities, while various temperatures (rectal, skin, jugular and tympanic), blood flows (pulmonary, renal and systemic circulation) and pressures (interventricular and systemic arterial) were simultaneously monitored. The preliminary results obtained provide a basis for comparison of the response of various organs to localized and whole-body exposure to microwaves.

Introduction

For long-term microwave (MW) exposure, the Personnel Exposure Standard in the United States and most Western countries is 10 mW/cm^2 . The Standard for protection is based on physiological considerations related to thermal inputs into the body. In contrast, depending upon the duration of exposure, the standard varies from 0.01 to 1.0 mW/cm^2 in the USSR, while other Eastern European countries (Czechoslovakia and Poland) may permit 2.5 to 10 mW/cm^2 for very short exposure. These lower standards reflect the concept that MW irradiation exerts a "specific" effect on the central nervous system.

In the mammal, adjustment of the cardiovascular system is one of the principal mechanisms for regulating thermal inputs into the body. Thermoregulation, however, is complicated by multiple neural and hormonal pathways acting on the cardiovascular system. Perturbation of neuroendocrine function may modify or directly affect cardiovascular adjustment to thermal inputs.

An important consideration in cranial exposure to MW is the resonant absorption of the electromagnetic energy in the skull which has been described by several investigators.¹⁻⁴ If such focusing and "hot spots" should occur in the brain, with the possibility of significant alteration of hypothalamic function, the reaction of the hypothalamus can then be monitored by studying thermal regulation and various components of the neuroendocrine system.

In an attempt to characterize the cardiovascular and neuroendocrine effects, the cranial area of dogs was exposed dorsally to various incident power densities of 2450 MHz (CW) microwaves while temperatures in various body sites as well as blood flow and pressure were simultaneously monitored. The preliminary results obtained provide a basis for tentative conclusions which can serve as a guide in future studies related to cardiodynamic and neuroendocrine pathophysiology.

Materials and Methods

Left thoracotomy was performed by removing the fifth rib for intra-thoracic instrument implantation.

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***Flow probes were designed and constructed by one of the authors (E.K.).

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Konigsberg P-22 transducers** were implanted into the left ventricle and main pulmonary trunk. Before isolating the vessels for implantation of flow probes,*** diameter of probe and vessel were carefully matched. Flow probes were implanted around the left pulmonary artery and right diaphragmatic vein. Midline laparotomy was used for left renal artery and vein implantation. The dogs were provided a 2 week post-surgical recovery period.

Arterial pressure was obtained by catheterization of the descending aorta through the carotid or femoral artery. The Konigsberg P-22 transducer was calibrated *in vivo*, prior to the study. All the readings were recorded oscillographically on a Brush recorder 260† with suitable biomedical couplers and preamplifiers. Rectal, tympanic, skin and jugular temperatures were taken with YSI telethermometers,†† model 401 (rectal), 402 (tympanic), 409 (skin), and 514 (jugular). Microwave exposure was performed on animals under pentobarbital anesthesia. Microwaves, 2450 MHz (CW), were generated by a Raytheon Microtherm CMD-10††† (maximum output 125 W). A waveguide horn antenna was placed 18 cm above the nuchal arch of the dog's skull. A Narda Broadband Isotropic Microwave Radiation model 8305‡ was used to measure the electromagnetic field.

Results and Discussion

Results in pentobarbital (30 mg/kg i.v.) anesthetized dogs revealed that the rate and stabilization of body temperatures could be influenced by the ambient temperature. Cranial exposure for 1 hour to power densities from 20 to 100 mW/cm^2 results in a slowing or reversal of the anesthesia-induced cooling process and an alteration of the thermal gradients within the body. Sometimes, the difference between rectal and tympanic temperatures approach 0. Of the various temperatures monitored, the tympanic temperature is found to be the most sensitive indicator of small temperature changes in this type of exposure. By irradiating the head directly beneath the horn, tympanic temperature can rise above that of the rectum. Only by a combination of 50 mW/cm^2 and a supraneuronal room temperature (higher than 26°C) could the rectal temperature be brought back to the pre-anesthetic

level. All the temperature increases can be temporarily reversed by saline infusion.

A paradoxical increase in skin cooling was found in only one dog after beginning the 80 mW/cm² exposure. In spite of reversal of the cooling at this power density, shivering continued.

Changes in heart rate and dP/dt (rate of change in isovolumic intraventricular pressure) are correlated with body temperature over a certain range. On the contrary, the arterial blood pressure appears refractory to the temperature changes. Initially upon exposure, an increase in average heart rate is observed. This is followed by exaggerated sinus arrhythmia with an increase in depth and rate of respiration resulting in slowing of the pulse rate. Mean atrioventricular conduction time (P-R interval) increases with the rate of respiration.

Instead of a positive chronotropic effect, as usually observed with barbiturate anesthesia, a decrease in heart rate was observed when morphine sulfate (2 mg/kg i.v. or s.c.) was used as a pre-anesthetic agent. Due to the responsiveness of the tympanic temperature, a better and wider correlation was found between this body temperature, heart rate, and dP/dt.

Under 100 mW/cm² for 1 hour, average pulmonary flow has a tendency to increase, but correlation with body temperature is not apparent. Mean renal blood flow is slightly depressed at the beginning of microwave irradiation, then decreases.

Limited results suggest that when change in the circulation is not sufficient to handle the excessive heat, the respiratory system is called into action to restrict further increase of core temperature. Onset of respiration responses (threshold) occurs at approximately 0.5°C increase of tympanic temperature, regardless of the absolute value of the central temperature. Although in the anesthetized dog, cranially exposed to 20, 40 or 80 mW/cm² for 1 hour, there seems to be some alteration in growth hormone or thyroxine levels, these changes are more likely a result of anesthesia rather than the effects of microwave exposure.

At "low level" (1-12 mW/cm²) of microwave exposure, changes in heart rate and arterial blood pressure have been reported in the rat and rabbit.^{5,6} Some of the power densities in these experiments are thermogenic, especially for the rat.⁷ Cooper *et al*⁸ also observed alteration of cardiovascular dynamics in MW-induced hyperthermia in dogs. In our experiments, we failed to induce any significant hyperthermia in dogs cranially exposed to 2450 MHz microwaves, which is no doubt due to the use of anesthesia which interferes with normal homeothermic processes. On the other hand, extrinsic neural control of the heart should be considered. Hardy⁹ suggests the existence of convergence of thermal input signals. Thus the result of altered thermal gradients on the regulatory mechanism becomes more complicated.

So far, the data obtained are not convincing enough for us to ascertain a cardiovascular effect of MW which is not subject to the influence of anesthesia or individual animal variation. Attempts are being made to isolate and remove the interacting factors such as anesthesia or variations in ambient temperature.

Alterations in thermal gradients within the animal body suggest thermal transportation or redistribution. This is exemplified by the overshooting of the tympanic temperature. This model may provide a method for assaying the effect of altered thermal gradients to maintain homeostasis of the organism.

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