Short Papers

Rate Effects in Isolated Hearts Induced by Microwave Irradiation

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***Abstract*—Continuous 960-MHz microwave irradiation of isolated poikilothermic hearts in Ringer's solution causes bradycardia, in contrast to the tachycardia usually produced by generalized heating. The effect appears to occur only over a narrow power range in the neighborhood of an estimated 3 mW absorbed by the heart. It is hypothesized that the bradycardia is produced by stimulation of the nerve remnants in the heart.**

I. INTRODUCTION

An important question yet to be answered satisfactorily is the following. What effects are produced by low-level microwave irradiation of biological systems? This question is particularly relevant to the increasing need to set realistic microwave irradiation safety standards. The question is difficult to answer because of the inherent complexity of biological systems, the complexity of the microwave-biological interaction, and the difficulties of making accurate measurements, including identifying and controlling the variables.

This short paper describes a series of experiments designed to point towards further understanding of microwave biological interaction, particularly in the range where generalized heating of the entire organ appears not to be the principal mechanism of interaction.

These experiments consist of exposing isolated turtle hearts submerged in Ringer's solution to CW 960-MHz microwave irradiation while measuring both rate and force of contraction. We chose the isolated heart as a means of studying the interaction mechanism because Frey and Seifert [1] indicated that the isolated frog heart is sensitive to microwave irradiation. We chose the turtle heart because, in our hands, it is a more stable preparation than the frog heart, and because it is larger than the frog heart, making it easier to manipulate. Since the turtle heart is poikilothermic, we submerged it in Ringer's solution which was temperature controlled by a circulating bath. We used agar-KCl electrodes to avoid artifacts introduced by concentration of the electromagnetic (EM) fields, which occur with metal electrodes [2].

We found that microwave irradiation in a certain power range caused bradycardia (decrease in heart rate), even though generalized heating of a poikilothermic heart causes tachycardia (increase in heart rate). At higher power levels, microwave irradiation caused tachycardia.

Our experiments are considerably different from Frey and Seifert's. They used isolated frog hearts irradiated in air with pulsed 1.425-GHz irradiation, and with the pulses synchronized with the electrical activity of the heart, their experiments indicated that the irradiation produced either tachycardia or arrythmia. Also, they used platinum electrodes in the hearts.

The experimental procedure we use is described in detail, followed by the experimental results, power and temperature considerations, and a discussion of the results.

II. DESCRIPTION OF THE EXPERIMENTS

Preparation of the Heart

Healthy turtles maintained in our laboratory were pithed and a 10-cm hole was cut in the plastron. A 4-0 silk suture was attached to the apex of the heart and the organ cut free of the animal. The excised heart was placed in a petri plate containing a balanced salt solution. This solution contained: NaCl 6 g; KCl 0.14 g; $CaCl_2$ 0.12 g; $NaHCO_3$ 0.20 g; and glucose 0.65 g per 1000 ml. Several changes of this solution during a 15-min stabilization and recovery period cleared the blood from the isolated heart. Tubular plastic electrodes (PE-100 filled with 1.5 percent W/V agar—6 percent KCl W/V) were sutured to the heart, one at the apex and the other in the aorta. The preparation was attached at the apex to a fixed plastic mount by the suture used to hold the electrode in the apex. The suture used to attach the electrode in the aorta was also used to attach the heart to the Statham strain gauge. The flexible plastic electrodes were attached by shielded input cables to the low-level dc preamplifier (Grass). The entire system was submerged in a continuously aerated bath containing 600 ml of the balanced salt solution plus glucose. The bath was surrounded by a water jacket held at 12°C to insure close temperature control and to provide an extensive heat sink. The tension on the system was adjusted to provide a force signal of reasonable magnitude but not high enough to change the heart rate of the particular preparation. Once the preparation was mounted as described, a temperature stabilization period of at least 15 min was allowed. During this period, the force of contraction as well as the electrical signal were continuously recorded.

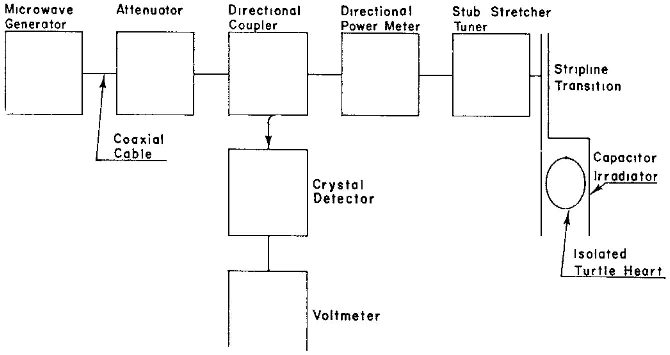

Fig. 1. Block diagram of the microwave system.

The Microwave System

A block diagram of the microwave system is shown in Fig. 1. The microwave generator is a converted radar transmitter which uses a lighthouse triode as a power oscillator. Maximum output power is about 2 W CW. The directional coupler and crystal detector are used in connection with the directional power meter to tune out the reflections with the stub stretcher. The stripline is used to provide a transition from the unbalanced coaxial system to the balanced capacitor irradiator. When the coax is connected directly to the capacitor, the resulting unsymmetrical current in the outer braid causes the coax to radiate and severely interfere with the recorder. The stripline–coax junction provides enough circular symmetry to the coax that the coax does not radiate enough to interfere with the recorder. The capacitor irradiator is simply a parallel-plate capacitor fed by the stripline; the plates are approximately 2.6 cm×3.9 cm, separated by approximately 3 cm. This separation is on the order of one wavelength in the Ringer's solution at 960 MHz.

Preliminary calculations indicate that the dominant field distribution between the plates is a combination of a TEM wave and the lowest order TM wave of a parallel-plate waveguide. The higher order TM waves are cut off and the TE modes are not excited because the polarization of the electric field is wrong. Some crude field measurements tend to support this idea, and work is continuing on better measurements and more complete calculations.

Experimental Procedure

The heart is allowed to equilibrate for 30 min after mounting, and during this time, both ECG and force of contraction are recorded to establish a baseline for that particular heart. After the 30-min stabilization period, continuous microwave power is applied for a specified time, during which both ECG and force are continuously recorded.

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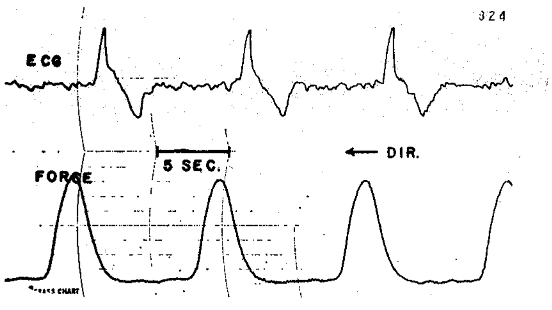

Fig. 2. The ECG and force recording for a typical heart without microwave irradiation.

III. Results of the Experiments

The ECG and force recording for a typical heart without microwave irradiation are shown in Fig. 2. A plot of rate versus time for a typical control is shown in Fig. 3. The rate measurements for a typical experiment in which the heart is irradiated are shown in Fig. 4.

Table I shows the average for all experiments of this type, normalized to the same starting rate of 10 beats/min. The slope was determined by the method of least squares for a 30-min section of the recorded ECG using the rate over a 2-min period. As can be seen, the average for all experiments indicates that bradycardia, at least transitorially, is produced by CW microwave power in the range of 50–200-mW total input power. At power levels near 50-mW total input, no consistent effect different from control is readily apparent, while at levels near 300-mW total input, the effect is variable. In some experiments, 300 mW has produced a definite tachycardia, particularly if the preparation has been subjected to previous irradiation at lower power levels.

The power levels quoted above are all total input power levels, as measured by the power meter shown in Fig. 1. This total power can be divided into: 1) the power losses in the cable, tuner, and stripline; 2) the power absorbed by the heart; and 3) the power absorbed by the Ringer's solution surrounding the heart.

Measurement of the power absorbed by the heart itself would be very difficult and usually the amount of power absorbed is calculated from measured temperature increases caused by the absorbed power. However, the measurement of the temperature increase of the heart produced by the absorption of microwave power by the heart is complicated by the fact that the presence of a thermocouple or a thermistor in the heart during microwave irradiation can cause hot spots that would not be present in the absence of the thermistor or thermocouple [2]. Consequently, some nonperturbing method of measuring temperature, such as the kind described by Johnson and Guy [2], must be devised. In our case, these kinds of measurement techniques are complicated by the smallness of the heart, and, as a result, we have not completed what we consider to be accurate measurements.

Some preliminary measurements that we have made indicate that the temperature rise in the heart during microwave irradiation at a total input power level of 100 mW is less than 0.5°C. Steady-state temperature calculations based on a spherical shell model of the heart and linear heat transfer at the surface of the heart have given a calculated temperature rise of about 0.2°C at a total input power level of 100 mW. This calculation is based on an estimated value of surface conductance for forced convection.

An estimation of the total power absorbed by the heart can be made by assuming the power to be uniformly absorbed in the volume between the irradiator plates. Then, since the loss tangents of the Ringer's solution and the heart tissue are in the same order of magnitude, the fraction of the power absorbed by the heart is approximately the ratio of the volume of the heart to the total volume between the plates. This method gives an estimate of 3.3 mW absorbed by the heart when the total input power is 100 mW. However, regardless of what fraction of the total power is absorbed by the heart itself, the measured bradycardia is a completely unexpected result because any absorbed power that would tend to raise the temperature of the heart would be expected to produce tachycardia, as has been demonstrated in our laboratory by raising the temperature of the bathing solution.

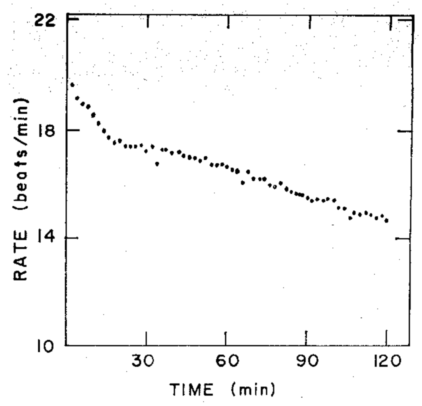

Fig. 3. Rate of a typical control heart.

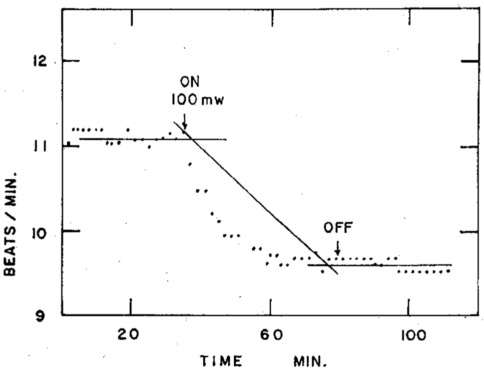

Fig. 4. Rate of a typical microwave irradiated heart. A 100-mW CW 960-MHz 30-min irradiation.

TABLE I

Total Applied Power	N	$\overline{X}$ Slope Beats/Min	Corr. Coeff. High	Corr. Coeff. Low	σ
Control	8	−0.0059	0.86	0.32	±0.00304
50 mW	2	−0.058	0.95	0.64	
100 mW	10	−0.495	0.98	0.66	±0.270
200 mW	3	+0.012	0.78	0.50	
300 mW	5	+0.18	0.85	0.42	±0.155
500 mW	2	+0.365	0.97	0.58	

Note: N is the total experiments at each power level; $\overline{X}$ is the average for all N experiments; Corr. Coeff. is the correlation coefficients, high and low for each slope in a set of N experiments; and σ is the standard deviation calculated by small-sample technique for N experiments at each power level.

IV. Discussion of Results

The strong overriding conclusion of our experimental work is that a significant decrease in heart rate occurs upon microwave irradiation in a narrow range of power. The greatest change in rate occurs when the total input power in our system is 100 mW, which corresponds to an estimated 3.3 mW absorbed by the heart. But since the heart is poikilothermic, any increase in temperature should cause the rate to increase, and indeed, raising the temperature of the heart by heating the bath does cause an increase in rate. Hence, the decrease in rate cannot be explained in terms of generalized heating of the heart by the absorbed microwave power.

Although we intend to pursue these effects much further, we presently propose the following tentative hypothesis, which is consistent with all the experimental data we now have.

In the isolated heart, there are remnants of the two parts of the autonomic nervous system, the parasympathetic, and the sympathetic. In general, if the parasympathetic nerves are stimulated, the heart rate decreases. If the sympathetic nerves are stimulated, the rate increases. If the two systems are stimulated equally, however, the rate would most probably decrease because the effects of the parasympathetic system are stronger than those of the sympathetic. We hypothesize that the microwave power is stimulating the nerve remnants and/or the *boutons terminaux* causing the release of transmitter which induces the decrease in heart rate. Furthermore, at 100-mW total power, the increase in temperature of the heart is not enough to cause an increase in heart rate equal to the decrease caused by the above described system, while at 300-mW total power, the increase in rate due to the microwave heating is greater than the decrease due to transmitter release. This is consistent with tachycardia induced by 300-mW total power.

Further evidence to check this hypothesis could be obtained by blocking the transmitter action and measuring the change in rate. The parasympathetic system can be blocked by the addition of atropine to the Ringer's solution, and if the hypothesis were correct, 100-mW microwave irradiation in the presence of atropine should cause an increase in heart rate, rather than a decrease. Preliminary experiments indicate that this does happen. We have also tried to block the sympathetic system with inderal (propranolol hydrochloride) to see if microwave power would cause a further decrease in rate, but this preparation is not an entirely suitable blocking agent. At present, we have no explanation of how the microwave power might be stimulating the nerves and/or buotons, but we intend to pursue this question along with many others that have been raised by our experimental results.

ACKNOWLEDGMENT

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REFERENCES

[1] A. H. Frey and E. Seifert, "Pulse modulated UHF energy illumination of the heart associated with change in heart rate," *Life Sciences*, vol. 7, pt. II, pp. 505–512, 1968.
[2] C. C. Johnson and A. W. Guy, "Nonionizing electromagnetic wave effects in biological materials and systems," *Proc. IEEE*, vol. 60, pp. 692–718, June 1972.

Microwave Effects on Thermoluminescence and Thermally Stimulated Exoelectron Emission

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Abstract—**In a pilot study to determine phosphor response after microwave exposure, a reduction in the expected amount of light emitted during thermoluminescent (TL) analysis was observed after exposure to microwave radiation of a phosphor preirradiated with cobalt-60 gamma radiation. Investigation of the thermoluminescent response of some high dielectric-constant materials after microwave exposure revealed the fading phenomenon in the powdered and ceramic states of the phosphors.**

I. INTRODUCTION

Critical reviews of the literature on the hazards and biological effects of microwave radiation reveal the limitations and inadequacy of much of the work performed thus far [1], [2]. One of the most important limitations is the lack of adequate dosimetric metholology for determining power density or absorbed energy in or around a biological specimen or tissue equivalent medium. The use of a reduction in thermoluminescent (TL) response (fading) from a preirradiated phosphor as a result of exposure to microwave radiation as a method of microwave dosimetry has been suggested in a report by Conover, but studies using a well-known phosphor have yielded no such reduction [3].

Thermoluminescence use in personnel radiation dosimetry was initiated by Daniels [4]. Since then the literature concerning the theory, development, and use of thermoluminescence has become very comprehensive. Briefly, the TL process can be depicted in terms of crystal lattice defects. The interaction of ionizing radiation with the crystal causes electrons to be raised from the valence band to the conduction band where the electron wanders until it falls into an electron trap. When the crystal is heated, the trapped electron is raised back to the conduction band where it wanders and falls back to the valence band emitting a quantity of light. The amount of light emitted by the crystal is proportional to the radiation dose received by the crystal.

More recently, a similar phenomenon called thermally stimulated exoelectron emission (TSEE) has been suggested as a radiation dosimeter [5]. In the TSEE process, trapped electrons are raised to the conduction band and emitted from the surface of the crystal. The number of electrons emitted is proportional to the radiation dose received by the crystal.

This short paper describes studies involving the use of powdered and ceramic phosphors, some with high dielectric constants and loss factors, which were used to evaluate TL and TSEE fading after exposure to microwave radiation. When correlated with microwave exposure in terms of energy density, the amount of fading or loss in TL response may provide a method for microwave dosimetry.

II. MATERIALS AND METHODS

The powder phosphors used for evaluating TL response were $BaTiO_3$, activated with dysprosium, and a mixture of $BaTiO_3$ and $SrTiO_3$ in a ratio of 8:2 by weight [$(Ba_{0.8}Sr_{0.2})TiO_3$]. Ceramic phosphors were prepared using an organic binder (methyl cellulose) mixed with these powdered phosphors and firing them for 3 h at 1385°C. The phosphors used for determining TSEE response were CaF_2, $BaSO_4$, $BaTiO_3$, and $(Ba_{0.8}Sr_{0.2})TiO_3$. These phosphors were studied in the pure state and mixed with 25 percent by weight graphite powder. The TL and TSEE properties of these phosphors had been determined previously [6], [7].

Annealed TL and TSEE phosphor samples, placed in paper envelopes or polyethylene vials, respectively, were irradiated with 10^5 R of ^{60}Co gamma radiation and then exposed to microwave radiation in an anechoic chamber, $305 \times 244 \times 213$ cm high, with a −40-dB quiet zone. An exhaust fan was located in the ceiling near the back wall to maintain room temperature. A pyramidal horn connected a 1.5-kW 2450-MHz generator to the chamber. The power level was determined with a directional coupler and Hewlett-Packard 432A power meter, and power density was monitored with a Narda 8110 electromagnetic radiation monitor. The TL phosphors were exposed to power densities ranging from 200 to about 5000 mW/cm^2 in the anechoic chamber while the TSEE phosphors were exposed to 200 mW/cm^2 in the anechoic chamber and to about 7.4×10^4 mW/cm^2 in a microwave oven. Phosphors were exposed for 5–40 min in the anechoic chamber and for 1 min in the microwave oven. After the longest microwave exposure, all samples including controls were placed on dry ice until analyzed. Phosphor TL and TSEE response were determined with readers designed and built at Purdue University [8], [9] and were expressed as glow peak height.

III. RESULTS

A statistically significant ($P < 0.05$) reduction in TL response was observed in the $(Ba_{0.8}Sr_{0.2})TiO_3$ phosphor only (Fig. 1). Fading increased with time of microwave exposure and was therefore dependent on the energy fluence at the surface of the phosphor. No reduction in TSEE response was seen in any of the phosphors exposed in the anechoic chamber, but a reduction of up to 80 percent was observed in the phosphors containing graphite when exposed in the microwave oven.

When measurements were made on the temperature of the $(Ba_{0.8}Sr_{0.2})TiO_3$ phosphor during microwave exposure, an increase over the ambient temperature level was noted. This is important to consider since the increased temperature may be the cause of the fading. To examine this possibility, a temperature-fading study was performed using a drying oven as the heat source. The maximum temperature reached by the phosphors during microwave exposure was determined to be 28°C, as measured with a thermocouple; therefore, the fading study was carried out at this temperature. Phosphors were heated for times equal to the microwave exposures. The results indicated an enhanced fading over that of the room temperature (22°C) fading, as expected by the theoretical explanation of thermo-

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