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Rate Effects in Isolated Turtle Hearts Induced by Microwave Irradiation

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Abstract—**Microwave irradiation at 960-MHz CW of isolated poikilothermic hearts in Ringer's solution causes bradycardia. Tachycardia is usually produced by generalized heating, suggesting the possibility of a different mechanism in this case. The effect occurs only over a narrow power range of approximately 2–10 mW/g absorbed by the heart. It is hypothesized that microwave radiation causes neurotransmitter release either by excitation of the nerve remnants in the heart, or by some other mechanism, producing bradycardia over a restricted range of power absorption. Drugs which can change the response of the heart to transmitter substances have been used, and the results support a neurotransmitter release hypothesis. A generalized heating effect, causing tachycardia, is predominant at higher levels of absorbed power.**

I. INTRODUCTION

IN a previous publication [1] we presented a series of experiments which were specifically designed to help further understand microwave biological interactions. Those experiments indicated a necessity for further work to consider interactions in a power range where generalized heating of the whole organism could not have been the principal mechanism of interaction. The present paper further describes effects that cannot be explained by generalized heating and might lead to a better understanding of mechanisms of interaction between electromagnetic fields and biological systems.

Our previous work led us to hypothesize that possible neural effects could be detected in microwave experiments performed on isolated poikilothermic heart systems. In those experiments isolated turtle hearts submerged in Ringer's solution were exposed to CW, 960-MHz microwave irradiation while both heart rate and force of contraction were being measured. The stability of the preparation and the size of the turtle heart made manipulations simple and also made the detection of any small perturbations to the system appear as reliable indicators. Agar–KCl electrodes were used as mounts to avoid possible artifacts introduced by concentration of electromagnetic fields, which occur with metal electrodes [2].

We found that microwave irradiation at approximate absorbed powers of 2–10 mW/cm^3 caused bradycardia (decrease in heart rate), while generalized heating caused tachycardia (increase in heart rate). At approximate absorbed power levels of 16–40 mW/cm^3, we found that microwave irradiation produced tachycardia. Several

Manuscript received March 28, 1974; revised August 6, 1975. This work was supported by the Office of Naval Research under Contract N00014-67-A-0325-0009.

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mechanisms are possible as explanation of the observed results. These include changes in receptor sensitivity as a result of microthermal changes, changes in mobility of small particles as a function of thermal changes (e.g., vesicles in presynaptic nerve remnants), and changes in amount of neurotransmitter release.

If we hypothesize [1] that microwave irradiation causes neutrotransmitter release, perhaps by excitation of the remnants of the automatic nervous system (both sympathetic and parasympathetic nerve endings), simultaneous stimulation of both systems would be expected to cause a decrease in heart rate since the effects of stimulation of the parasympathetic system usually predominate. In the present paper, we describe further experiments designed to test the neurotransmitter hypothesis. In these experiments, the individual sympathetic and parasympathetic effects were separated by blocking the action of each system in turn with known drugs. First, the action of the parasympathetic system was blocked by the addition of atropine to the Ringer's solution, and approximately 8-mW/cm^3 absorbed microwave power was applied. Second, the sympathetic action was blocked by the addition of propranolol hydrochloride (β blocker) to the Ringer's solution and similar power applied. If the hypothesis is correct, we should have observed an increase in rate for the first case and an even greater than normal decrease in rate in the second case. Our heart-rate measurements confirm the hypothesis. Thus, having obtained affirmative results from several possible combinations of experiments, we were able to conclude that events leading to increased effective neurotransmitter concentration produce the observed bradycardia at low power levels of microwave irradiation. In addition, measurements of the temperature inside the heart during irradiation were made using a prototype of a liquid-crystal temperature probe developed in our laboratory. Accuracies are typically ± 0.1°C. Preliminary measurements indicated that the temperature probe did not perturb the microwave fields. The temperature measurements confirm the lack of significant heating at approximately 8-mW/cm^3 absorbed power. These measurements also explain the tachycardia at higher power levels of microwave irradiation.

The experimental procedures we used are described in detail below, followed by the experimental results, power and temperature considerations, and a discussion of the results.

II. DESCRIPTION OF THE PROBLEM AND DISCUSSION OF THE HYPOTHESIS

Before giving the data and the interpretation, it might be helpful first to discuss the neural mechanism. Consider the physical structure of the biological system with which we are working. A nerve ending consisting of an axon and a bouton or terminal arborization is the stimulating part of the system, and a muscle fiber and receptor site make up the stimulated portion of the system. In the sympathetic system, an action potential transmitted down the axon to the bouton causes the release of norepinephrine. This norepinephrine diffuses across the myoneural gap to the receptor site in the muscle fiber. Thus any stimulation which would cause either an action potential to be transmitted down the axon, an increase in the sensitivity of the receptor site, release of preformed transmitter, or a combination of these would increase the excitability of the muscle fiber in this system. The sympathetic system, if stimulated, causes an increase in heart rate. The same type of action takes place in the parasympathetic system, except that the transmitter substance released is acetylcholine (Ach), and the effect slows the rate. Thus an action potential transmitted down the axon of the parasympathetic system, releasing Ach, would cause a decrease in heart rate. The effect of the parasympathetic system on the heart is greater than the effect of the sympathetic system [3]. Therefore, simultaneous stimulation of both systems could cause a net decrease in heart rate. If microwaves at lower power levels are in some way stimulating transmitter release (in both nerve systems), then the effect would be bradycardia.

We hypothesize that microwave power stimulates nerve remnants and/or synaptic sites, causing release of transmitter, which induces a decrease in heart rate. Furthermore, at approximately 8-mW/cm^3 absorbed power, the measured increase in temperature of the heart was not sufficient to cause a detectable increase in heart rate and certainly could not affect the decrease seen by the transmitter release described. At approximately 40-mW/cm^3 absorbed power, the increase in rate due to the microwave heating was presumably greater than the effect of neurotransmitter release. This is consistent with the observed tachycardia induced by 40-mW/cm^3 absorbed power. To check our data further, we compared our measured change in rate and increased temperature with the calculated Q_{10} of the biological system. The Q_{10} of our heart system is defined by the van't Hoff equation [4]

$$Q_{10} = \left(\frac{k_2}{k_1}\right)^{10/(T_2 - T_1)} \tag{1}$$

where k_2 and k_1 are the heart rate in beats per minute at heart temperatures T_2 and T_1, respectively, in degrees Celsius. In a series of experiments, we determined the heart system to have a Q_{10} of approximately 5. Taking the logarithm of both sides yields

$$\log_{10} Q_{10} = \frac{10}{T_2 - T_1} \log_{10}\left(\frac{k_2}{k_1}\right).$$

For $Q_{10} = 5$

$$\log_{10}\left(\frac{k_2}{k_1}\right) = 0.07(T_2 - T_1). \tag{2}$$

Over a period of 15 min of irradiation, temperature-difference measurements were made for both approximately 8 and 40-mW/cm^3 absorbed power levels. The starting rate k_1 was known for each experiment. From (2) we then calculated the expected change in heart rate

($\Delta k = k_2 - k_1$) for both 8 and 40-mW/cm³ power levels. The following are the calculated results:

Absorbed Power Level	Calculated Δk	Measured Δk
mW/cm³	beats/min	beats/min
8	+1.01	−3.2 (decrease)
40	+5.1	+3.7

These results show that there is a striking difference between control-heating experiments, from which the Q_{10} of 5 was obtained by raising the bath temperature, and results obtained from application of microwave power.

Further evidence has been obtained by blocking neurotransmitter action and measuring the change in heart rate. The parasympathetic system was blocked by the addition of atropine to the Ringer's solution. The plotted average of several experiments shows (Fig. 1) that approximately 8-mW absorbed power caused an increase in heart rate when atropine was present. The sympathetic system was blocked with β blocker, propranolol hydrochloride. Fig. 2 shows the mean results with a dramatic decrease in heart rate upon application of approximately 8-mW/cm³ absorbed microwave power. The mechanism by which relatively small amounts of absorbed microwave power affect neurotransmitters remains unknown.

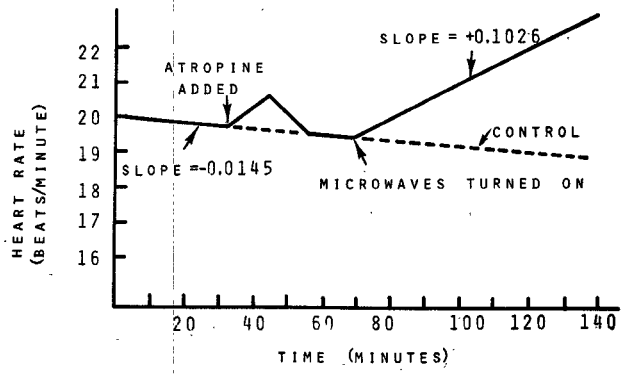


Fig. 1. Effect of atropine (1.6×10^{-8} g/ml) and microwave irradiation (8 mW/cm³ absorbed power) on heart rate.

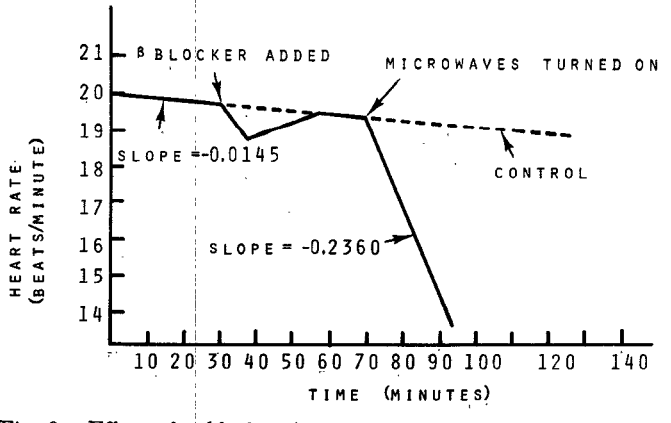


Fig. 2. Effect of β blocker (1.6×10^{-8} g/ml) and microwave irradiation (8-mW/cm³ absorbed power) on heart rate.

III. DESCRIPTION OF EXPERIMENTS

Preparation of the Heart

The turtle-heart preparation was conducted as outlined in our previous work [1], with the following added precautions to ensure greater stability. The tension on the heart system was adjusted to provide a force signal of reasonable magnitude, but not high enough to cause instability in the heart rate of the particular preparation. Once the preparation was mounted as described, a temperature stabilization period of at least 40 min was allowed. During this period, the force of contraction and the electrical signal were continuously recorded.

The Microwave Irradiation System

A block diagram of the microwave system is shown in Fig. 3. This setup is also the same as that used before except for the deletion of the directional coupler and crystal detector which were included initially only for convenience in tuning the stub stretcher to eliminate reflected power. The total power delivered to the heart system was measured by an in-line directional power meter which could be set to measure either forward power or reflected power. Maximum total power obtainable was about 2-W CW.

Experimental Procedure

The experiments were broken up into eight groups with 6–16 animals per group. These groups include: a) microwave irradiation only; b) atropine; c) β blocker; d) atropine and β blocker; e) atropine and microwave irradiation; f) β blocker and microwave irradiation; g) atropine and β blocker and microwave irradiation; and h) temperature measurements during irradiation. The same procedure was followed in each of a)–h) for the first 40 min. A description of each is given below.

a) Microwave Irradiation (8-mW/cm³ Absorbed Power): In all experiments, the heart was allowed to equilibrate for 40 min after mounting. During this time, both ECG and force of contraction were recorded to establish a baseline for the particular heart. After the 40-min stabilization period, continuous microwave power was applied for a $\frac{1}{2}$-h period. The ECG and force were continuously

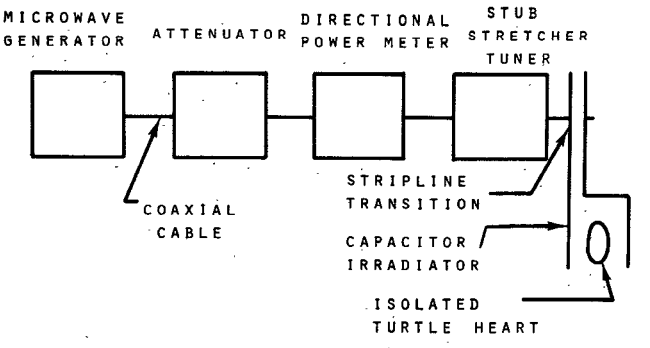


Fig. 3. Block diagram of the microwave system.

recorded during irradiation and for an additional 90 min after termination of microwave irradiation.

b) Atropine: In these experiments, atropine (atropine sulfate, Mallinckrodt ZMX-1, USP) was added to the Ringer's solution after the stabilization period. A final concentration of 1.6×10^{-8} g/ml was achieved (in approximately 30 s). In all experiments, both ECG and force were continuously recorded for an additional 90 min after the last externally added stimulus.

c) β Blocker: Propranolol hydrochloride (Indiral, Ayerst, Control No. 1 KEM) (β blocker) was added to the Ringer's solution until a final concentration of 1.6×10^{-8} g/ml was achieved. The preparation was then allowed to stabilize.

d) Atropine and β Blocker: After the 40-min stabilization period, atropine and β blocker were added to the Ringer's solution until a final concentration of 1.6×10^{-8} g/ml of each agent was achieved.

e) Atropine and Microwave Irradiation (8 mW/cm³): An addition of atropine to the Ringer's solution was made until a concentration of 1.6×10^{-8} g/ml was achieved. 30 min after atropine had been added, the microwave power source (960-MHz CW) was turned on. After 30 more minutes had elapsed, the power source was turned off and 30 min more were allowed for the system to equilibrate.

f) β Blocker and Microwave Irradiation (8 mW/cm³): After stabilization, β blocker was added to the Ringer's solution until a concentration of 1.6×10^{-8} g/ml was attained. 30 min after β blocker had been added, the same microwave power source was turned on. After 30 min had elapsed, the power source was turned off, and 30 min more were allowed for the system to equilibrate.

g) Atropine and β Blocker and Microwave Irradiation (8 mW/cm³): The combination of atropine and β blocker was added to the Ringer's solution until the same concentration was attained as previously given; 30 min after atropine and β blocker had been added, the microwave power source was turned on. After 30 min had elapsed, the power source was turned off, and 30 min more were allowed for the system to equilibrate.

h) Temperature Measurements: A liquid-crystal thermometer mounted on the tip of an optical-fiber bundle was inserted into the aorta of a heart in place of the agar–KCl electrode. Continuous temperature and rate measurements were taken for 130 min while total applied power levels of 50, 100, 150, 200, 300, 500, 1000, and 1500 mW at 960-MHz CW were irradiating the heart system.

IV. RESULTS AND CONCLUSIONS

Control-Experiment Results

Fig. 4 shows an average least square fit for sixteen control experiments (see Section III). Fig. 5 is an average least square fit curve for the particular case of approximately 8-mW/cm³ absorbed power applied after 40 min to the same system as that in the control system. The

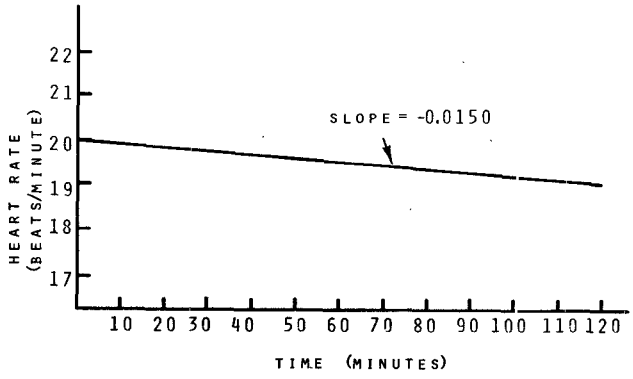


Fig. 4. Control experiments (averaged).

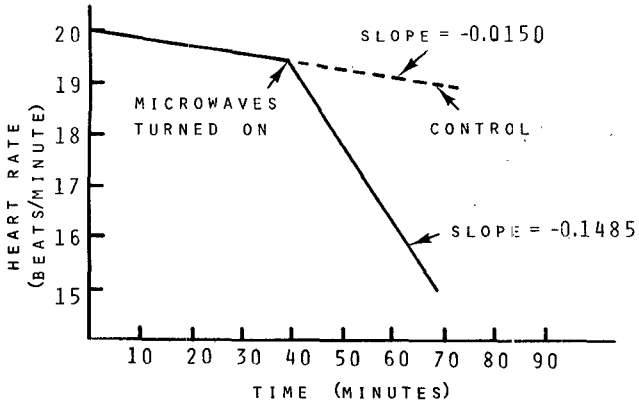
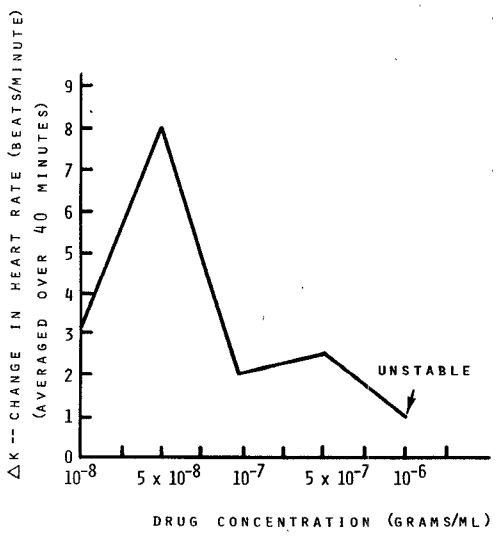
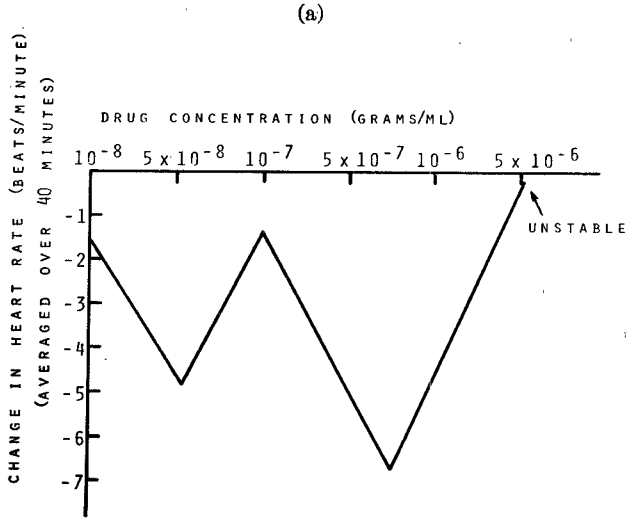


Fig. 5. Change in heart rate due to microwave irradiation (8-mW/cm³ absorbed power).

average least square calculations are taken over the entire time of irradiation. The effect noted correlates well with the hypothesis that both the sympathetic and parasympathetic systems are active, and that the parasympathetic, being more effective, causes bradycardia (decrease in heart rate). By adding drugs which selectively block the receptor sites on either the sympathetic or the parasympathetic systems independently, we have demonstrated that the microwaves can cause neural transmitter release effects. Propranolol hydrochloride was the drug used as β blocker, which blocks the receptor sites in the sympathetic system, and atropine was the drug used to block the receptor sites in the parasympathetic system. After extensive titrations of the drugs (both atropine and β blocker) (see Fig. 6), it was found that the optimal concentration for short-term definite effects was approximately 1.6×10^{-8} g/cm³. Fig. 7 shows the least square best fit for atropine, and Fig. 8 shows the least square best fit for β blocker tests. It was important to find a concentration which caused a definite change in rate, and at the same time allowed the heart to compensate for the drug over a short period of time (e.g., 20 min). This nearly ideal situation was accomplished by the concentration previously mentioned, and is shown by the 40–70-min time periods in Figs. 7 and 8. It was necessary to attain this type of curve so that as microwaves were applied to,



(a)



(b)

Fig. 6. (a) Effects on heart rate due to various concentrations of atropine added to the preparation. (b) Effects on heart rate due to various concentrations of propranolol hydrochloride added to the preparation.

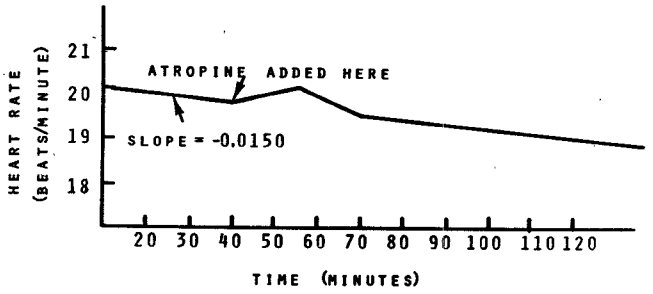


Fig. 7. Effect of atropine (1.6×10^{-8} g/ml) on heart rate.

say, the 70-min time period, there would be no additive effect of the drug and the microwaves simultaneously. Thus microwaves added at the 70-min time period did not interfere with or add to the drug effect as it would have if it were applied at, say, the 45-min time period. These experiments were conducted and their results (least square best fit) are shown in Figs. 1 and 2.

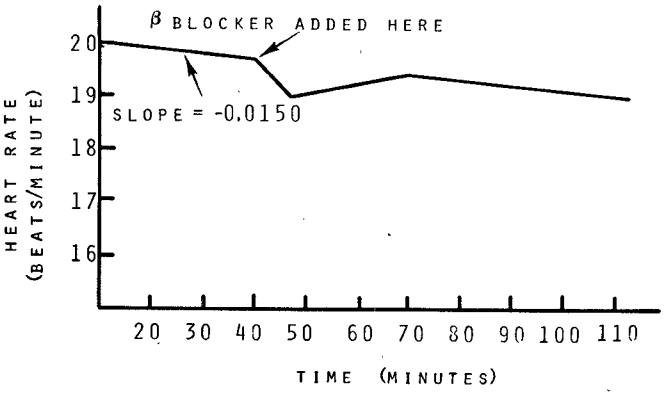


Fig. 8. Effect of β blocker (1.6×10^{-8} g/ml) on heart rate.

A comparison of Fig. 5 and Fig. 1 indicates that a definite sympathetic effect is apparent in Fig. 1. Thus atropine, by blocking the receptor sites of the parasympathetic system, allows the microwave neural effect to stimulate the sympathetic system only, causing tachycardia instead of the bradycardia produced previously by the same 8-mW/cm³ power level. Fig. 2 indicates increased bradycardia over that caused by microwave irradiation alone. Thus β blocker, by blocking the receptor sites of the sympathetic system, allowed the parasympathetic system to have an even greater effect than that which was noted when only microwaves were applied.

From the data shown in Figs. 1, 2, 4, and 5, there is a definite indication that the microwave irradiation (approximately 8-mW/cm³ absorbed power) caused effects similar to those of neurotransmitter release. The exact portion of the sympathetic and parasympathetic systems which were being affected by microwaves is not known. Further experiments designed to measure action potentials on nerve axons are being considered to clarify this question. Several other classes of drugs known to affect this site are also being investigated.

In addition to using atropine and β blocker separately, both of these drugs were used together to determine their additive effect on the heart rate. Fig. 9 shows that when both of these drugs were used together, the effect was similar to that of the controls. Fig. 9 further shows that microwave irradiation of approximately 8 mW/cm³ applied 20 min after the addition of both atropine and β blocker caused little change in heart rate. This was to be expected since both types of receptor sites were blocked. The reverse order of this experiment was also performed. Fig. 10 shows a typical result. It is clear from the data shown that the application of the drugs removes the microwave-induced bradycardia.

The slope of the heart rate of each animal during each time period (i.e., control, microwave irradiation, etc.) was calculated by the method of least square fit to a straight line. The correlation coefficients were also calculated. The slopes thus obtained were averaged and the standard deviation determined. The average slopes are shown in Figs. 1–5 and Figs. 6–10, and a summary of data is given in Table I. It can be seen from this table that the informa-

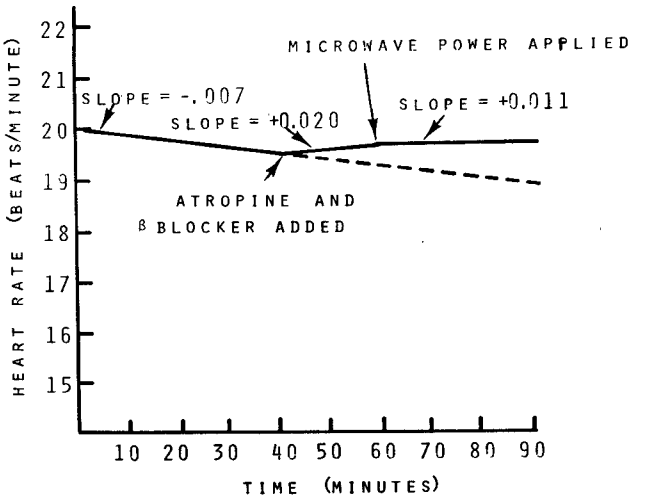


Fig. 9. Heart rate as a function of time when atropine (1.6×10^{-8} g/ml), β blocker (1.6×10^{-8} g/ml), and 8-mW/cm^3 absorbed power are applied to the isolated heart.

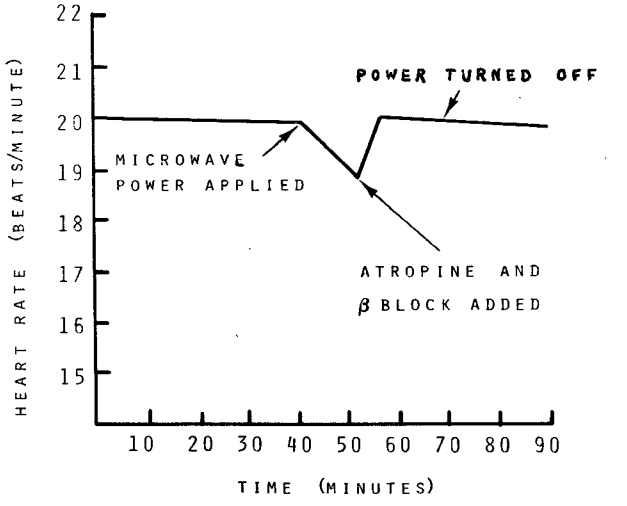


Fig. 10. Heart rate as a function of time when 8 mW/cm^3 of absorbed microwave power, atropine (1.6×10^{-8} g/ml), and β blocker (1.6×10^{-8} g/ml) are applied to the isolated heart (in the reverse order of Fig. 9).

TABLE I
RESULTS OF STATISTICAL CALCULATIONS

Experiment Number	Absorbed Power (mW/cm^3)	Drugs Added (gm/ml)	N	$\bar{X}$ Slope $\pm\sigma$ (beats/min)	$P_{x,y}$ x, y, Experiment Numbers Compared (slope)	r Correlation Coefficient High	Low
1	0.0	None	16	-0.015 ± 0.003		.99	.63
2	8.0	None	8	-0.148 ± 0.027	$P_{1,2} < 0.005$	.90	.49
3	0.0	Atropine (1.6×10^{-8})	8	+0.020 ± 0.040	$P_{1,3} < 0.20$	.91	.51
4	0.0	Propranolol (1.6×10^{-8})	8	-0.169 ± 0.029	$P_{1,4} < 0.025$	.94	.74
5	8.0	Atropine (1.6×10^{-8})	8	+0.102 ± 0.017	$P_{3,5} < 0.005$	.97	.41
6	8.0	Propranolol (1.6×10^{-8})	8	-0.236 ± 0.034	$P_{4,6} < 0.05$	.91	.61
7	8.0	Atropine and propranolol (1.6×10^{-8})	6	+0.011 ± 0.01	$P_{1,7} < 0.005$	.90	.69

Note: N is the total experiments at each power level; $\bar{X}$ is the average for all N experiments and is calculated over intervals of unchanging external stimuli; r is the correlation coefficient, 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. P is probability from t test table.

tion in each curve can be considered to be a very reliable indicator of the total neural transmitter release effect in each experiment, assuming only that atropine and propranolol have the effects usually ascribed to them pharmacologically.

Temperature Data

Preliminary measurements indicated that the temperature rise during microwave irradiation was less than half a degree centigrade. The devices (thermocouples and thermistors) used in those early measurements were of such a nature that they may have caused perturbation of the applied fields and resulting hot spots in the system. More recent measurements, using a prototype of a liquid-crystal thermometer developed in this laboratory, gave more accurate temperature readings inside the heart. The method used (1), as given by Johnson and Guy [2], which is valid during short periods of exposure when the loss of heat due to radiation and convection effects can be neglected. During this period, the temperature is a nearly linear function of time, as indicated in Fig. 11. The expression for absorbed power density in watts per cubic centimers is [2]

$$P = \frac{4.186\rho c \Delta T}{\Delta t} \tag{3}$$

where ρ is the tissue density in grams per cubic centimeters; c is the specific heat of the tissue in calories per gram degrees Celsius; ΔT is the change in temperature in degrees Celsius; and Δt it the time of exposure in seconds. For the following typical values of parameters:

$$\rho = 1.062 \text{ g/cm}^3$$

$$c = 0.88 \text{ cal/g°C (Dowben) [5]).}$$

Equation (3) reduces to

$$P = 3.913 \frac{\Delta T}{\Delta t} \text{ W/cm}^3. \tag{4}$$

Fig. 12 shows a plot of the power absorbed by the heart

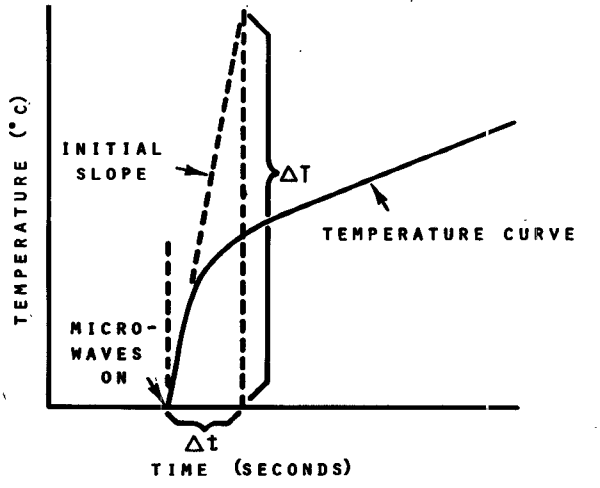


Fig. 11. Temperature rise as a function of time after microwave power is turned on.

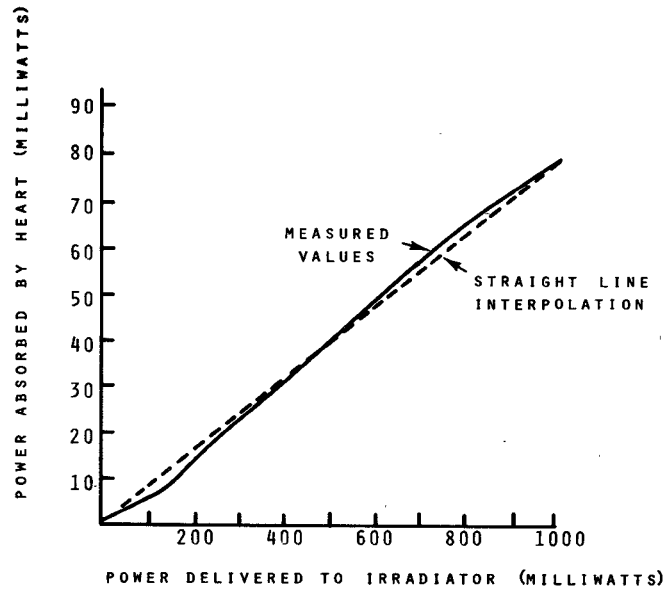


Fig. 12. Power absorbed by the isolated heart as a function of power delivered to the capacitor plate irradiator.

with a typical volume of 1 cm³ as obtained from calculations using (4) with measured values of ΔT and Δt for total applied powers of 100, 150, 200, 500, 800, 1000, and 1500 mW. The deviation from linearity was approximately 3 percent above 500 mW; at lower levels, the accuracy was reduced by inability to read the small ΔT's accurately enough. Using straight-line interpolation, the absorbed power for 100-mW total applied power was found to be approximately 8 mW/cm³. Consequently, the temperature measurements confirmed that the tachycardia produced by generalized microwave heating was a very small effect at lower power levels. This finding strengthens the hypothesis that the neural effect is dominant at lower power levels, and the generalized heating effect is dominant at higher power levels.

V. CONCLUSIONS

1) Approximately 8-mW/cm³ absorbed power, 960-MHz-CW microwave irradiation causes bradycardia in the isolated poikilothermic heart. In more experiments performed after this paper had originally been submitted for publication, it has been found that the bradycarida produced by microwave irradiation occurs in the absence of the agar–KCl electrodes.

2) When approximately 8-mW/cm³ power is applied in the presence of a parasympathetic blocking agent (atropine), heart rate increases.

3) When approximately 8-mW/cm³ power is applied in the presence of a sympathetic blocking agent (β blocker), heart rate decreases more than when the agent is not present.

4) The addition of both atropine and β blocker to the heart preparation blocks the effects of both the sympathetic and the parasympathetic transmitters. Microwave irradiation then causes only very slight changes in heart rate.

5) Increasing the power level (above approximately 15 mW/cm³) absorbed by the heart produces tachycardia, which may be the result of generalized heating of the heart.

Each of these conclusions greatly strengthens the hypothesis that the neural effect (possible transmitter release due to microwave irradiation) is dominant at lower power levels. Again it must be noted that the exact location of the stimulation causing the observed effects has as yet not been determined. Further experiments directed toward separating the possible locations of the stimulation are being planned for future research.

Further measurements of absorbed power density between the plates of the microwave irradiation made after the series of experiments described in this paper were completed have shown that the absorbed power density can be affected by the losses in the stripline transition, possibly caused by the Ringer's solution moving up into the stripline by capillary action. In these later experiments, 1000-mW total input power was required to obtain an absorbed power density of approximately 2 mW/cm³ instead of the 25 mW previously required, but the microwave-induced bradycardia still occurs in the range of 2–10-mW/cm³ absorbed power density, as it has in all experiments. The difference in input power required for the same range of absorbed power density possibly occurs because of the difference in losses in the stripline transition in the irradiator and may have resulted because the stripline was dismantled and cleaned between the two series of experiments.

Dosimetry measurements are difficult to make because a temperature probe cannot be left in the beating heart during the experiments without affecting the heart. Consequently, total input power must be calibrated with power absorbed by the heart and used as the measure of absorbed power, but the unexpected variability of losses between the input power meter and the heart causes problems. However, in this case, the power measurements appeared to have been consistent over each series of experiments, and a change apparently occurred only when the stripline was dismantled and reassembled. An improved irradiator without these problems is being designed for future experiments.

ACKNOWLEDGMENT

The authors wish to thank R. G. Olsen and C. C. Johnson for assistance and helpful suggestions.

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