

BRADYCARDIA IN ISOLATED HEARTS INDUCED  
BY MICROWAVE IRRADIATION

James L. Lords, Carl H. Durney, Alan Borg, Charles Tinney  
University of Utah  
Salt Lake City, Utah

Abstract

Continuous 960 MHz irradiation (estimated 5 mW absorbed) 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.

Introduction

Bradycardia has been reported to be a possible *in vivo* reaction to microwave exposure in humans. We have shown bradycardia in isolated poikilothermic hearts under carefully controlled conditions. Using poikilothermic hearts and making the assumption that generalized heating occurs under exposure to microwave energy, the result should be an increase in the heart rate. In our experiments herein described, the isolated, thermostated poikilothermic heart responds by a decrease in rate, i.e., bradycardia.

Protocol and Results

Our experiments were performed as follows: Healthy turtles maintained in our laboratory were pithed to destroy the CNS, and then a 10 cm was cut in the plastron. A 4-0 silk suture was fixed to the apex of the heart and the heart was cut free of the animal. The heart was placed in reptilian Ringer's solution and allowed to stabilize for a few minutes to recover from shock. Two tubular plastic, agar-KCl filled electrodes were placed in the heart, one at the apex and the other in a great vessel, the aorta, at the top of the heart. The preparation was then mounted on plastic supports and placed in a bath containing 600 mls of reptilian Ringer's solution. The bath was surrounded with a water jacket held at 15°C to insure close temperature control and to provide an extensive heat sink. The electrodes were attached to a Grass recorder and the support arranged to record simultaneously the force of contraction using a low level dc preamplifier and a Statham strain gauge. The force of contraction and electrical activity of the heart (ECG) were recorded for a period of 30 minutes to establish the base line for the particular preparation, and then continuously during irradiation. Figure 1 represents a control in which no perturbation of the preparation occurred.

Thirty minutes after stabilization, microwave power was applied CW for a given time period. The microwave system consisted of a modified radar transmitter, a coaxial cable, a stub-stretcher tuner, a directional power meter, a strip-line transition section, and a capacitor-plate irradiator. The irradiator was placed in the Ringer's solution with the heart between the plates. The total power delivered to the heart and the solution was measured by a Sierra directional power meter, and the frequency was approximately 960 MHz. Part of the total power was absorbed by the heart and part by the surrounding Ringer's solution. Although we have not measured specifically the amount of power absorbed by the heart, we estimate on the basis of volume ratios that the heart absorbed less than 5 percent of the total power. In any event, the heart absorbed significantly less than the total applied power.

The rate measurements for a typical experiment are shown in Fig. 2. Figure 3 represents the average for all experiments of this type, normalized to the same starting rate (beats/min). The slope was determined by the method of least squares for each 30 minute section of the recorded ECG. The data for Fig. 3 are shown in Table 1. As can be seen, the average for all experiments indicates that bradycardia, at least transiently, is produced by CW microwave power (100-200 mW total power). At power levels near 50 mW total, the effect is not readily apparent while at power levels near 300 mW, the effect is not consistent. In some experiments, 300 mW has produced a definite tachycardia, particularly if the preparation has been subjected to previous irradiation at lower power levels.

Interpretation

In similar experiments with other species of poikilothermic animals, the results are essentially the same. In all species tested, the rate drops following exposure to microwave power. Several possible mechanisms can be used to explain these results. One such is that specific heating took place at one of several tissue interfaces within the heart, particularly at the myelin-muscle interface involving the remnants of the cut nerves enervating the myocardium. If this interface responded by initiating firing in the nerves of the parasympathetic system, the resultant would be bradycardia. Even if both the sympathetic and parasympathetic nerve remnants were stimulated by the microwave power, the response would be bradycardia, since the effects of parasympathetic activity are stronger than those of the sympathetic.

Table 1. Data for Fig. 3.

Power (mW)	No. of Experiments	Average Slope (Beats/Min)	Range of Correlation Coefficients	
			High	Low
0	16	-.0050	.758	.051
50	2	-.0058	.951	.637
100	10	-.1514	.986	.658
200	3	+.0118	.785	.498
300	4	+.1740	.858	.420

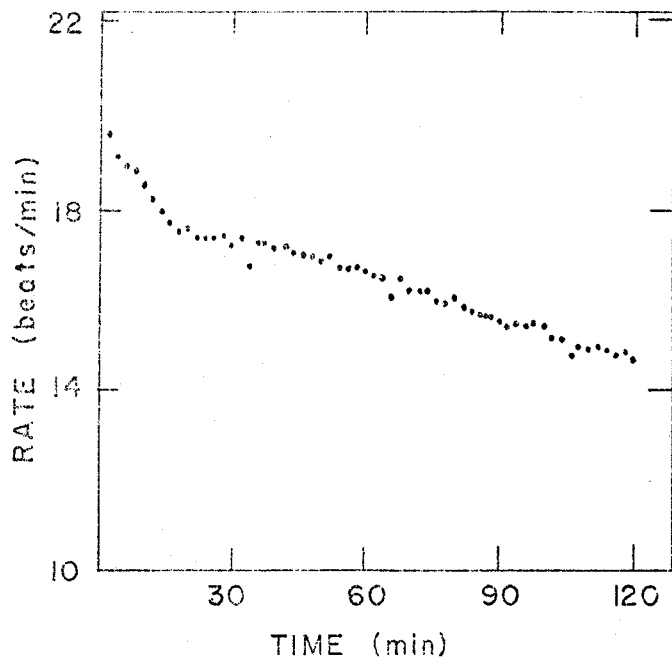


FIG. 1. RATE OF A TYPICAL CONTROL HEART.

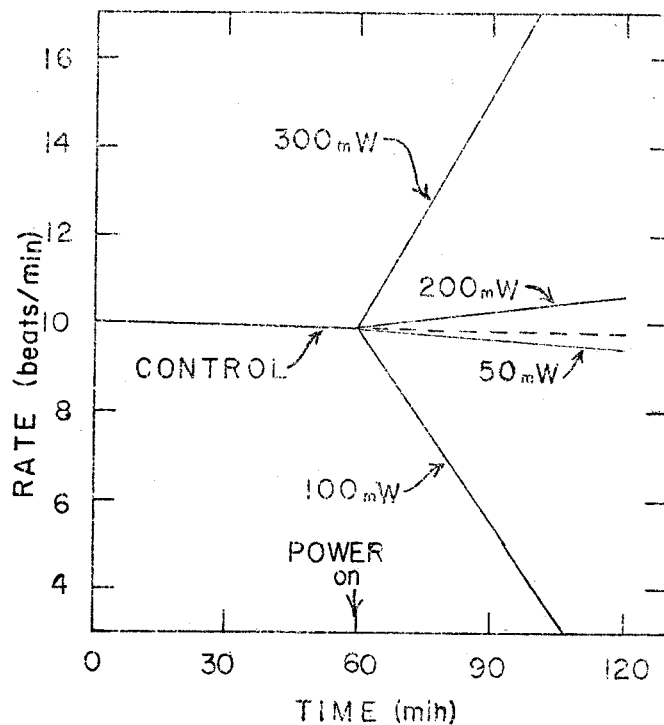


FIG. 3. AVERAGE HEART RATE NORMALIZED TO THE SAME STARTING RATE.

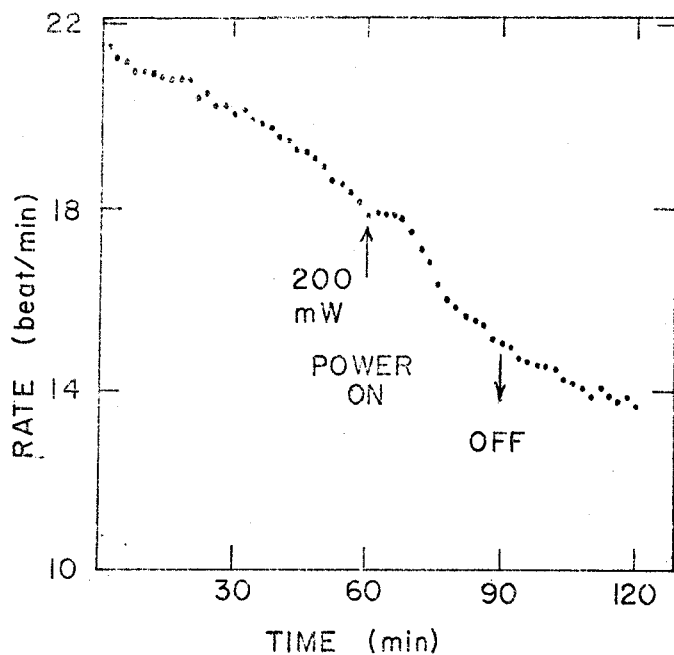


FIG. 2. RATE OF A TYPICAL MICROWAVE IRRADIATED HEART.