Modification of Heart Function with Low Intensity Electromagnetic Energy

Article *in* Electromagnetic Biology and Medicine · August 2009

DOI: 10.1080/15368378609006057

JOURNAL OF BIOELECTRICITY 5(2), 201-210 (1986)

MODIFICATION OF HEART FUNCTION WITH LOW INTENSITY ELECTROMAGNETIC ENERGY

Allan H. Frey and Edwin S. Eichert
Randomline, Inc.
Huntingdon Valley, PA 19006

ABSTRACT

Three groups of frogs were exposed to pulsed modulated radio frequency electromagnetic (EM) energy. One group was subjected to alternating ten-minute periods of energy exposure and sham exposure, with the exposure pulses synchronized with the rise of the R-wave. A second group was subjected to alternating ten-minute periods of energy exposure and sham exposure, with the exposure pulses synchronized with the T-wave. The third group was sham-exposed controls. The data indicated that rate of change of beat is influenced by exposure to EM energy at incident average power densities of 3 microwatts/cm^2. Synchronization of the energy pulses with the phase of the cardiac cycle is of consequence.

INTRODUCTION

Frey and Seifert (1), using the isolated frog heart, found that when the heart was exposed to pulses of electromagnetic (EM) energy synchronized to occur with the QRS complex, the heart rate increased and sometimes arrhythmia occurred. The increase was statistically significant with average incident power densities of 6 μW/cm^2. Exposure at the occurrence of the P-wave, during the P-Q interval, and sham exposure did not cause similar effects. Other experimenters also have reported effects on the heart (2-5). Thus, a complementary experiment was under-

0730-823X/86/0502-0201$3.50/0

taken to determine if the changes seen by Frey and Seifert in the isolated heart preparation would occur *in vivo*.

MATERIALS AND METHODS

Leopard frogs (Rana Pipiens) were pithed and placed ventral side up on a polystyrene surface. They were restrained at the arms and legs by plastic clips. The ventral body wall was opened and electrodes placed so as to minimize the possibility of artifacts. Egg-carrying frogs, those with noticeable parasites, and those with non-beating hearts were not used. After pilot experimentation, 24 frogs were used in the data collection reported here. Twelve were subjected to alternating 10-minute periods of energy exposure and sham exposure, with the exposure pulses synchronized with the rise of the R-wave. Six were subjected to alternating 10-minute periods of energy exposure and sham exposure, with the exposure pulses synchronized with the T-wave. Six were used as non-exposure controls, three in each of two non-exposure conditions used. In one condition, the preparation was shielded from the energy with a piece of EM energy absorber (Eccosorb AN77). In the other, EM energy was delivered to a dummy load instead of to the horn antenna.

The heart rate of a typical preparation decreased by 2-3% during each 10-minute period, as is normal. Generally, the heart remained active for ten to twelve 10-minute periods. To control for possible non-linear heart rate decreases, half of the preparations were initially sham exposed and half were initially exposed to synchronized pulse-modulated energy.

The preparation was positioned on a polystyrene sheet (which is essentially transparent to this energy) and placed on a table made of Eccosorb AN77. The table was inside an EM energy anechoic enclosure constructed from Eccosorb AN77. The preparation was located approximately 90 cm below the open end of a standard gain horn antenna. Measurements of the EM field showed that the energy was evenly distributed at the heart's level.

The frog's electrocardiogram (ECG) was monitored with the use of a pair of electrodes. Each electrode was constructed from a 20-gauge, 5-cm long stainless-steel tube connected to the center lead of a 1-m length subminiature coaxial cable (RG 196). These two cables were passed through a 1-cm diameter polystyrene tube. The electrode assembly was fixed to the tube by cooling molten polystyrene on the electrode ends where they projected from the tube. The shields were connectd at the electrode end of the tube. The polystyrene tube passed through a hole in the Eccosorb enclosure and was supported by a micromanipulator outside of the enclosure. For minimal field effects, the tube was positioned perpendicular to the electric field produced by the horn antenna. With the aid of a micromanipulator and a microscope, the tips of these stainless-steel electrodes were placed in connective tissue approximately 3 mm from the heart, one near the top of the heart and the other near the apex.

The subminiature coaxial cables from the electrodes were attached to a differential amplifier in a dual-trace oscilloscope. The time base for each trace was adjusted to trigger on the P-wave of the frog's ECG. The sweep rate was adjusted so only a single ECG cycle occurred during a sweep. The oscilloscope provided a pulse to a jack each time a sweep was triggered. After shaping by a diode clamp differentiator, the pulse was applied simultaneously to the EM energy generation circuitry and to a computer. The computer was used to measure and store the periods of the ECG (time between P-waves).

The EM energy generation circuitry consisted of a pulse generator (AEL Model 104) which delayed the pulse before it was used to modulate an EM energy transmitter. The transmitter was adjusted to deliver one 5-microsecond pulse to the preparation each time the machine received a trigger pulse. The transmitter also concurrently provided a synchronization pulse to a jack. This latter pulse was applied to the oscilloscope's second input amplifier and allowed the monitoring of transmitter pulses on the second trace of the scope. Thus, the experimenter could simultaneously monitor the ECG and the apparent transmitter output. The word apparent is used because the transmitter monitoring pulse would appear on the scope even during the sham exposure condition. This was a control on the experimenter, who was not aware of whether the exposure was actual or sham. The

experimenter adjusted the delay time of the pulse generator throughout the experiment to maintain synchronization of the EM energy pulse with the rise of the R-wave or the rise of the T-wave, as required during a particular run by the experimental design. A stepping relay was cycled automatically by the control circuitry. It provided alternating 10-minute periods of exposure and sham exposure. During sham exposure periods, the energy was applied to a dummy load rather than to the horn antenna.

The EM energy carrier frequency of 1.25 GHz was modulated to deliver 5-microsecond pulses of energy to the frog, in synchrony with the rise of the R-wave in one group and at the rise of the T-wave in the other group. The incident power density was measured before and after each experimental run with a quarter-wave dipole connected in series with a Hewlett-Packard Model 477B thermister mount and a Hewlett-Packard Model 430C power meter. With a heart rate of approximately one beat per second, the incident average power during exposure was 3 $\mu W/cm^2$.

For data processing, a train of pulses was inputted to a computer by a pulse generator. The computer counted the number of pulses received and stored the count in a memory location. The pulse generator was operated at 1 KHz; thus, the system was capable of resolving ECG intervals to 0.001 second. As previously noted, a P-wave trigger pulse was directed to the computer. Each such trigger pulse stepped the computer from its present memory location to the next location.

After each 10-minute exposure or sham exposure period, the automatic control circuitry activated a strip chart recorder to first record a current 10-second portion of the frog's ECG and then record the contents of the computer memory. After storing the computer's memory contents, the control circuitry changed the exposure conditions and reset the computer for the next exposure period. The print-out interval was approximately 2 minutes.

All equipment was permitted to warm up and stabilize for 3 hours before each experimental run. The accuracy of the data collection system was checked before and after each run by the power company's 60-Hz powerline frequency. The data from one R-wave synchronized run was discarded because the equipment did not conform to calibration standards after the run.

Alternating periods of exposure and sham exposure were repeated until the heartbeat ceased. Data collected during the last recorded period was rejectd if the heartbeat ceased during that time. If it stopped during the print-out interval, the data from the last completed period was retained. After each experimental run, the strip chart records were labeled with a code number and delivered to another experimenter for blind analysis.

The evaluation consisted of dividing each 10-minute exposure or sham exposure condition into ten 1-minute periods. The count in the computer memory at the start of a condition and at the end of each of the ten 1-minute periods was obtained. These eleven data points are referred to as period data. In addition to recording period data, the number of discontinuities greater than 0.02 seconds during each 1-minute interval was also recorded as an indicator of arrhythmia.

RESULTS

The mean times between heartbeats for each minute of the 10-minute periods, as well as linear regression lines, are plotted in Figure 1. The data designated as CONTROL (non-exposure Eccosorb and dummy load conditions) were combined because there were no significant differences in the non-exposure data. The average heart rate increased at a rate of 0.4% per minute for the R-ON condition. It decreased at a rate of 0.6% per minute for R-OFF, 0.4% per minute for T-ON, 0.7% per minute for T-OFF, and 0.4% per minute for CONTROL. The use of t tests on the percentage change in heart rate indicate significant differences between R-ON and R-OFF ($P < .005$), between R-ON and CONTROL ($P < .005$), and between T-ON and T-OFF ($P < .05$). The difference between T-ON and CONTROL was not significant, and there were no significant differences in arrhythmia.

The slopes of the regression lines and the correlation coefficients are shown in Table 1.

DISCUSSION

The data indicate that the heart function of an _in vivo_ preparation can be influenced by exposure to low-intensity

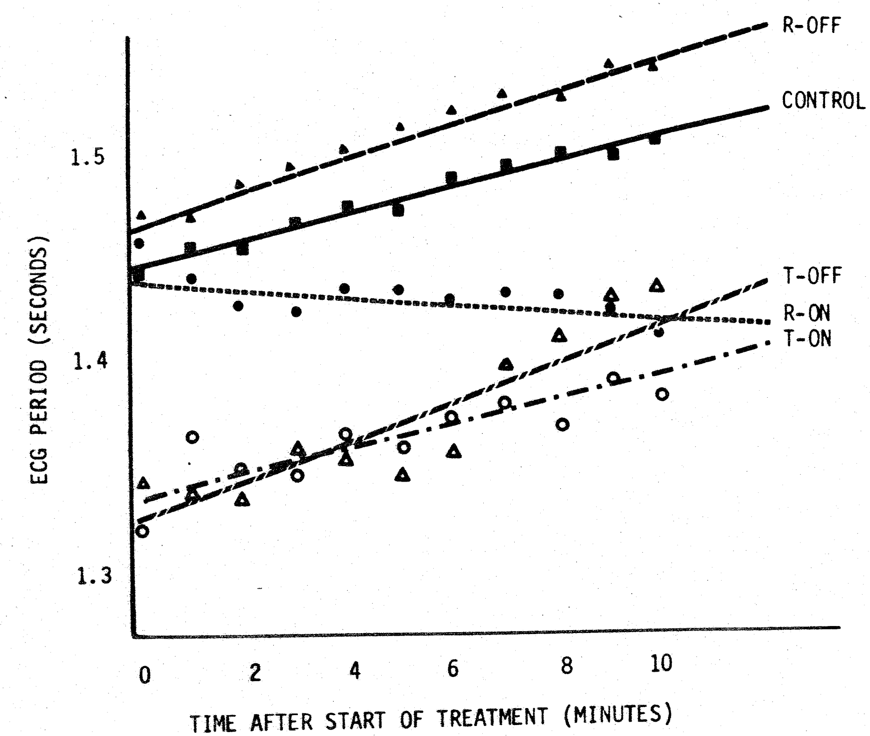

FIGURE 1

The effect of different conditions of electromagnetic energy exposure, as a function of exposure time, is illustrated by a plot of minute-to-minute means and linear regression lines. The data are scaled as obtained, in terms of the time between beats. Thus, slowing of the heart is shown by a rise in the curve. Data obtained when the energy impinged at the R- or T-wave are designated R-ON and T-ON, respectively. Data obtained from preparations that had no exposure to the energy are designated CONTROL, R-OFF, and T-OFF.

TABLE 1

Regression Line Slopes and Correlation Coefficients for Each Condition

CONDITION	SLOPE	r
R-on	-0.33	-.71
R-off	1.08	.98
T-on	0.67	.84
T-off	1.43	.91
Control	0.88	.98

pulsed EM radiation. The rate of change in beat was affected, but the preparation appears to be more resistant to arrhythmia than an isolated preparation. It also appears that the exposure must be for more than one minute for a significant effect to appear.

This finding can be related to the work of Lords et al. (6), who exposed isolated turtle hearts to microwave-frequency fields between capacitor plates. They found they could induce a bradycardia at an absorbed power of 3.3 mW. They suggested that the effect might be due to stimulation of the parasympathetic and sympathetic nerve remnants. In a follow-up experiment, Tinney et al. (7) found that over a narrow power range of approximately 2-10 mW/g absorbed dose, there was apparent stimulation of sympathetic and parasympathetic nerve remnants which could, respectively, increase or decrease the heart rate.

Tigranian (2) also studied the effect of exposure of the frog heart to EM energy. He found that modulated energy exposure occurring at the time of the P, R and T waves disturbed the heart rate. With the modulated energy, he found that sino-atrial block developed. With unmodulated fields, he reported the effect was smaller for the same incident average power and was qualitatively different in nature. Chalker et al. (3), using isolated frog hearts and a helium neon laser sensor, found that the beat rate of the heart changed with exposure to the energy. They report that the onset of the change was fast compared to the thermal time constant.

Schwartz et al. (4) assessed calcium efflux in the heart exposed to EM energy. They found an effect occured only at a modulation of 16 Hz, not with continuous energy or the other modulation frequencies that they used. When Schwartz and Mealing (5) used a different carrier frequency and only atrial strips of the frog heart, they found there was no calcium efflux change. This suggests either that the atrial strips are not sensitive to the energy, or that the carrier frequency is of importance. In sum, a variety of experiments have shown an influence of EM energy on the heart.

Several authors have reported what they thought to be contradictory results, but there were defects in their experiments. Jauchem et al. (8) used a frequency of 5.6 GHz to expose intact rats. They reported they did not find a change in heart rate at their lower power density. However, one would not expect to find an effect on the heart with 5.6 GHz frequency energy. It does not penetrate the skin and thus does not reach the heart. Chou et al. (9) also report no effect on the heart of exposure to EM energy. However, they used as subjects only 3 rabbits with a carrier frequency at 2450 MHz. Aside from the fact that the carrier frequency has peculiar penetration characteristics that might have resulted in little energy reaching the heart, it would be impossible to show a statistically significant effect using only 3 rabbits. In addition, rabbits have a very labile heart rate. Even if they had used reasonable size groups, it would have been unlikely that an effect would appear, since the variance in the data would be quite high. Thus, their experimental design closed out the possibility of finding an effect before they even started. Two other papers from that laboratory, Yee et al. (10,11) were also defective and provide no usable data. Clapman and Cain report finding no effect (12). They used two frequencies of microwave energy and exposed the isolated frog heart at the P-wave, 100 msec after the P-wave, and 200 msec after the P-wave. But the exposures to EM energy were only one minute in length and the data we report here shows that that is too short an exposure to see an effect.

Thus, the data as a whole clearly indicates that the heart responds to EM energy, particularly if it is pulsed and the pulses impinge at the right time in the cardiac cycle. The data available on the heart are not sufficient to draw conclusions about mediators. But a neuro-humoral mechanism may be involved. The neural system is responsive to the energy, as Frey and others have shown (13).

REFERENCES

(1) Frey, A.H. and Seifert, E.: Pulse Modulated UHF Energy Illumination of the Heart Associated with Change in Heart Rate, Life Sci. 7, 505-512, 1968.

(2) Tigranian, R.E. and Parsadanian, A.S.: Action of a Low Intensity Pulsed Super-High Frequency Electromagnetic Field on Heart Contraction Rhythm in the Frog, in All-Union Symposium on the Biological Effects of Electromagnetic Fields, Pushchino, USSR, 1982.

(3) Chalker, R., Wachtel, H. and Barnes, F.: Non-Contact Measurement of Microwave Induced Changes in Frog Heart Beating Rhythms, in Abstracts of Bioelectromagnetics Society 3rd Annual Conference, Washington, DC, 1981.

(4) Schwartz, J.L., Delorme, J. and Mealing, G.A.R.: Effects of Low Frequency Amplitude Modulation Radiofrequency Waves on the Calcium Efflux of the Heart, Biophys. J. 41, 195a, 1983.

(5) Schwartz, J.L. and Mealing, G.A.R.: Effects of CW and Low Frequency Amplitude Modulated Microwaves on the Calcium Efflux from Heart Tissues, in Abstracts of Bioelectromagnetics Society 5th Annual Meeting, 1983.

(6) Lords, J.L., Durney, C.H., Borg, A.M. and Tinney, C.E.: Rate Effects in Isolated Hearts Induced by Microwave Irradiation, IEEE Microwave Theory and Techniques 21, 834-836, 1973.

(7) Tinney, C.E., Lords, J.L. and Durney, C.H.: Rate Effects in Isolated Turtle Hearts Induced by Microwave Irradiation, IEEE Microwave Theory and Techniques 24, 18-24, 1976.

(8) Jauchem, J.R., Frei, M.R. and Heinmets, F.: Effect of Average Power Level on Heart Rate Changes due to 5.6 GHz Radiofrequency Radiation, in Abstracts of Bioelectromagnetics Society 5th Annual Meeting, 1983.

(9) Chou, C.K., Han, L.F. and Guy, A.W.: Microwave Radiation and Heart Beat Rate of Rabbits, J. Microwave Power 15, 87-93, 1980.

(10) Yee, K.C., Chou, C.K. and Guy, A.W.: Effect of Impulse Microwave Exposure on Frog's Heart Rate in vitro, in Abstracts of Bioelectromagnetics Society, 5th Annual Meeting, 1983.

(11) Yee, K.C., Chou, C.K. and Guy, A.W.: Effects of Microwave Exposure on Frog's Heart Rate in vitro, in Abstracts of Bioelectromagnetics Society, 4th Annual Conference, 1982.

(12) Clapman, R.M. and Cain, C.A.; Absence of Heart-Rate Effects in Isolated Frog Heart Irradiated with Pulse Modulated Microwave Energy, J. Microwave Power 10, 411-419, 1975.

(13) Frey, A.H.: Biological Mediators and Mechanisms for Low Intensity Electromagnetic Effects, Invited lecture at the Third Annual Bioelectromagnetics Society Meeting, Washington, DC, 1981.