

EFFECT OF 2450 MHz MICROWAVE FIELDS ON PERIPHERAL NERVES

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Abstract

There was no significant change in characteristics of nerves exposed to cw and pulsed 2450 MHz fields in a waveguide filled with temperature-controlled Ringer's solution. Absorbed power densities varied from 0.003 to 1.7 W/cc for cw and 0.3 to 30 W/cc peak for pulsed fields.

Summary

Although 2450 MHz microwave fields have been used for diathermy since the mid-1940's, the full effect of such radiation on biological tissue remains obscure. The most obscure effects of the fields are those reported as low level effects on the CNS and peripheral nerves. In much of the reported work, it is difficult to relate the effect to the actual field in the nerve tissue causing the effect. Kamenskii [1] and Rothmeier [2] reported some non-thermal effects on frog sciatic nerves at low power levels. It is difficult to determine, however, what the actual temperature changes were in these nerves, since the nerves were isolated in the air during irradiation. Under such conditions power absorption density can be much higher than for the normal situation in which the nerve is buried in other tissue and the temperature inside the nerve can be higher than that measured at the surface. The aperture source which Kamenskii used [3] can cause hot spots in the irradiated nerve due to the discontinuities at the edge of the aperture [4]. In the present paper, a method is described which avoids or minimizes the problems stated above.

A silver-plated S band WR284 waveguide was equipped with inlet and outlet ports for circulating fluids. The waveguide was put in a lucite tank containing Ringer's solution 6cm in depth (three times greater than the depth of field penetration). In each experiment, a frog sciatic or a cat saphenous nerve was pulled through the 3mm diameter holes on the waveguide parallel to the electric field of the TE₁₀ mode. Both ends of the nerve were immersed in mineral oil-filled chambers for stimulation and recording. A quarter guide wavelength of matching material with a dielectric constant of 6 was used to reduce the reflected power to less than 3% of the incident power. The temperature of the Ringer's solution was controlled within $\pm 0.02^\circ\text{C}$ by a constant temperature circulator with 2.5 gallons/min maximum pumping capacity. The temperature of the solution was monitored at the outlet of the waveguide with a thermocouple. Cw and pulsed power sources, operating at 2450 MHz with incident and reflected powers measured by means of a directional coupler and a power meter were used to feed the waveguide. In one series of experiments, the nerve was exposed to pulsed power with calculated average absorbed power densities of 3 mW/cc, 30 mW/cc, and 300 mW/cc in the nerve. The pulse width was 10 usec and the pulse recurrence frequency was 1000 pulses per second. Continuous waves were also used at the same power levels. The total irradiation time for each run was 20 minutes with the circulator being operated for the first 10 minutes and shut off for the final 10 minutes. Compound action potential data from the nerves was recorded and reduced by a computer of average transients. Arrangement of the apparatus is shown schematically in FIG. 1. The absorbed power density in the nerve can be calculated by the following formula:

$$P = 4a \frac{P_I - P_R}{A} e^{-2ax}$$

where

P: absorbed power density in the nerve (W/cc)
 P_I: incident power (W)
 P_R: reflected power (W)
 x: depth of nerve below the surface of Ringer's solution (cm)
 A: cross-sectional area of the waveguide (cm²)
 1/a: depth of field penetration in Ringer's solution (1.78cm at 2450 MHz)

A study was first performed to determine the effect of the controlled solution temperature on the compound action potential and conduction velocity of the isolated nerve. The results are shown in FIG. 2 for the frog nerve and in FIG. 3 for the cat nerve. In each case the temperature was first decreased from normal to approximately 10°C below body temperature. The temperature was then increased to the point where the amplitude of the compound action potential decreased to one-half normal value. This corresponded to the 46°C for the cat nerve which caused irreversible damage. The damage was evidenced by different nerve responsiveness when the temperature was again decreased. The temperature-sensitive characteristics of the frog nerve were reversible, since the maximum temperature was well below the threshold for damage when the peak amplitude decreased by 50%. These results suggest that mammalian animals rather than cold-blooded animals should be used to evaluate effects of microwaves on the nervous system for better quantitative extrapolation to humans.

Exposure of the frog and cat nerves did not result in any amplitude, conduction velocity, or excitability changes during the time that the constant temperature circulator was on for any level of either cw or pulsed irradiation. Slight increases in excitability and conduction velocity were detected during the time that the circulator was off for power absorption levels of 30 and 300 mW/cc. These effects can be attributed to temperature changes according to FIG. 2. FIG. 4 shows that the absorption (1.7 W/cc) due to very high power irradiation caused a slight increase of conduction velocity during the circulator on phase. This was due to the limited pumping rate of the circulator and consequent temperature rise. A much greater increase in conduction velocity was observed when the circulator was shut off and the temperature was allowed to increase due to radiation. Frog action potential amplitude and latency were degraded at a temperature around 35°C. This is consistent with the results shown in FIG. 2. After irradiation, the response either recovered or degraded, according to how high the temperature was. In a third series of experiments, double stimulation pulses 1.2 milliseconds apart were used. The response due to the second pulse was in the relative refractory period of the first response. The characteristics of the second response was more sensitive to temperature changes. This indicates that a

a slight microwave-induced temperature rise in the peripheral nerve could possibly alter firing patterns of the neurons in the central nervous system.

References

1. Y.I. Kamenskii. "The Effect of Microwaves on the Functional State of the Nerve," *Biophys.*, 9:6:758-764, 1964.
2. J. Rothmeier. *The Nervous System and Electric Currents*, Plenum Press, 1970, pp. 57-69.
3. A. Presman and Y.I. Kamenskii. "Apparatus for Investigating the Excitability of Nerve-Muscle Preparations During Microwave Irradiation," *Biophys.*, 6:2:73-74, 1964.
4. A.W. Guy. "Analyses of Electromagnetic Fields Induced in Biological Tissues by Thermographic Studies on Equivalent Phantom Models," *IEEE Trans. Microwave Theory & Tech.*, 19:2:205-214, 1971.

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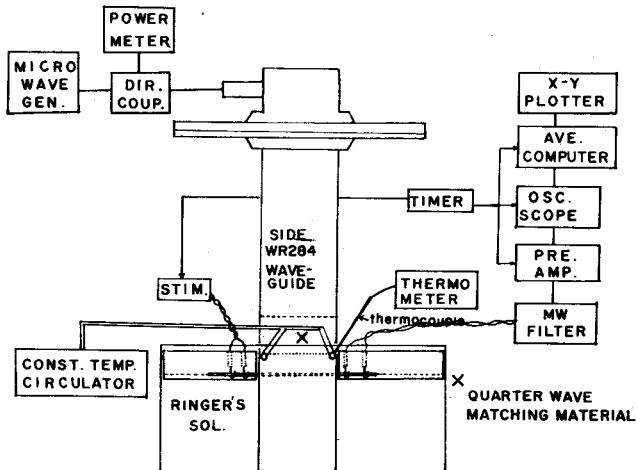


FIG. 1 Apparatus for microwave radiation of isolated nerve.

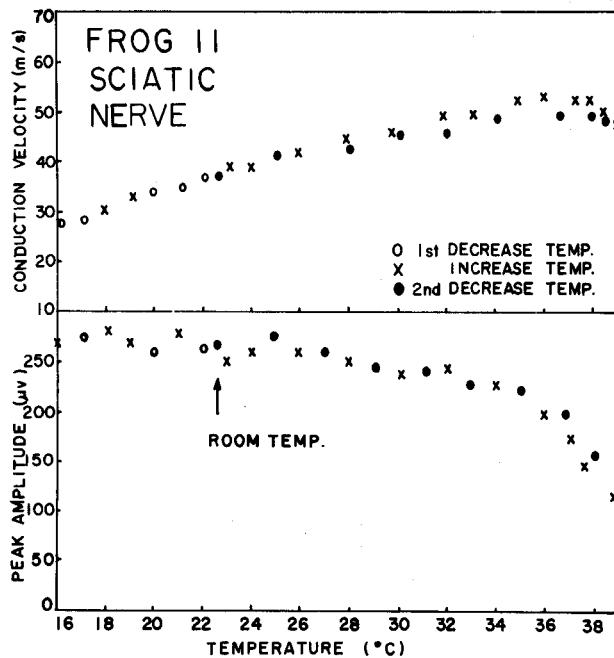


FIG. 2 Amplitude of compound action potential and conduction velocity of frog sciatic nerve as a function of temperature.

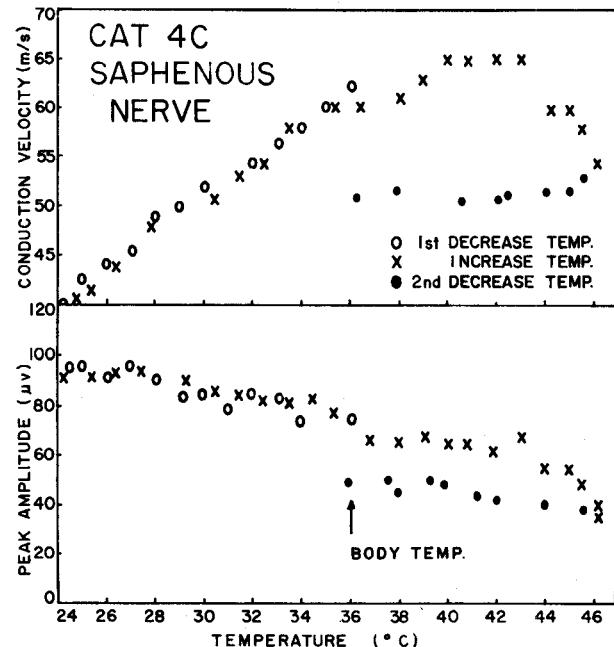


FIG. 3 Amplitude of compound action potential and conduction velocity of cat saphenous nerve as a function of temperature.

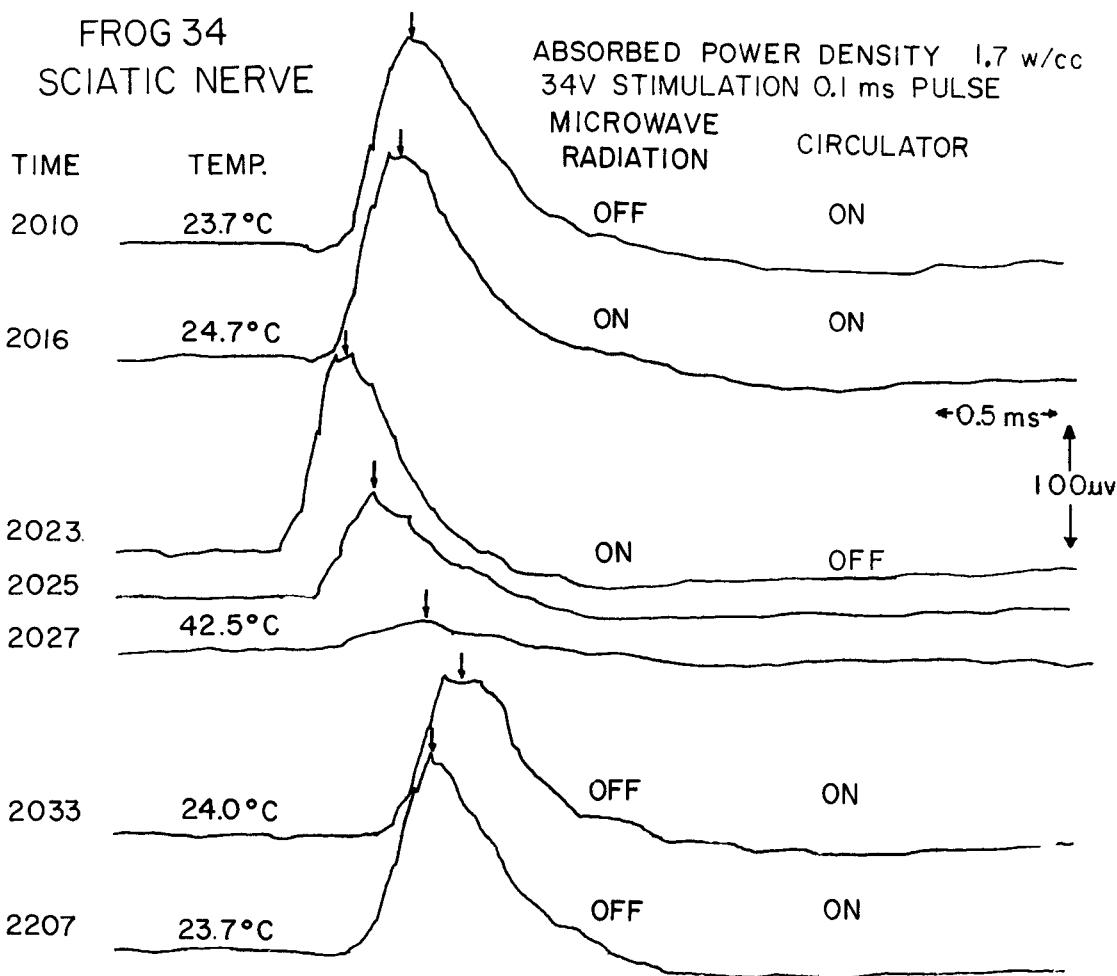


FIG. 4 Overheating effect on nerve action potentials due to 1.7 W/cc cw radiation during the time that the circulator was off.