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Short Communications

ELECTROMAGNETIC EMISSION AT MICRON WAVELENGTHS FROM ACTIVE NERVES

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ABSTRACT In recent years there has been experimental work and speculation bearing upon the significance in neural functioning of electromagnetic energy in the region of the spectrum between 0.3 and 10 μ. We demonstrate, in this experiment, micron wavelength electromagnetic emission from active live crab nerves as compared to inactive live and dead nerves. Further, the data indicate that the active nerve emission is caused by specific biophysical reactions rather than being simply black-body radiation.

INTRODUCTION

In the recent literature, there has been a convergence of ideas and experimental work which suggests that there may be emission of micron wavelength energy from neurons which is greater than and different from that which would be expected from a black body (Frey, 1965; Arvanitaki and Chalazonites, 1960; Tarusov, Polivoda, Zhuravlev, and Sekamova, 1962; Polonsky, 1958, 1961; Batteau and Hemmes, 1966; Wei, 1966; Roppel, 1963). If the possibility suggested by these converging lines is valid, then study of the emission of this energy from neural tissue and possible disruption of it by external energy of the proper wavelength(s) and patterns may yield significant information on neural functioning.

We carried out a limited investigation of this possibility when a critical item of equipment came into our possession for a short time. The results of this preliminary work are statistically significant. Calculations from the data indicate that the emission may be in discrete spectral bands. Thus, we suggest that more extensive investigation be undertaken.

MATERIALS AND METHODS

The specimen studied was the unmyelinated walking leg sensory nerve of the blue crab, *Callinectes sapidus*. It was used intact as taken from the leg. After working out the experimental techniques, the experiment proper was carried out with the nerves from seven crabs.

The radiation detecting system used was a Barnes R-8T1 infrared radiometer (Barnes Engineering Co., Stamford, Conn.). This consisted of an optical unit and an electronic unit.

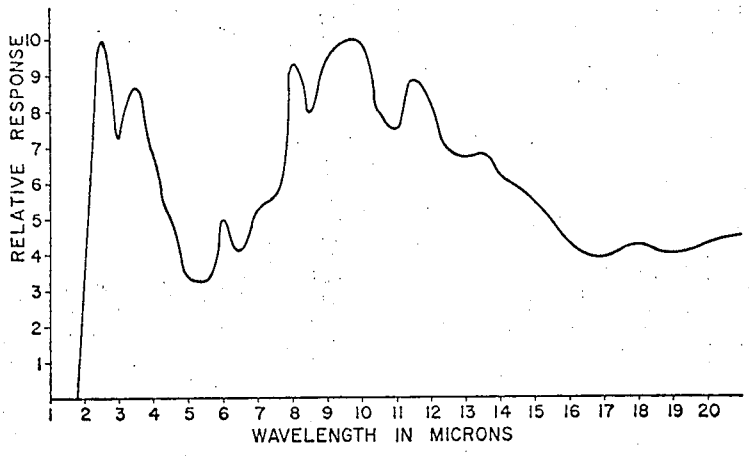

FIGURE 1 Spectral passband.

The optical unit employed a modified Cassegrainian reflecting telescope to gather and focus the incident radiation. The electronic unit consisted of a thermistor bolometer detector, a solid state preamplifier, amplifier, and a synchronous rectifier. The passband of the lens-bolometer system is shown in Fig. 1. The total system was quite sensitive with a detector noise power of 2.6×10^{-9} w in a one cycle bandwidth at 100 cps. The output signal was integrated over 1 sec intervals with a Dymec model 2401B integrating digital voltmeter (Dymec Div., Hewlett-Packard Co., Palo Alto, Calif.). The 1 sec integrations were then averaged further with a computer.

100 cm away from the detector, at the focal point of the detecting system, a standard nerve mount was fastened. The nerve on the mount was shielded from extraneous heat generated by the experimenters and equipment by covering the nerve mount with an expanded polystyrene ice chest with a hole in the side facing the detector. A plywood box completely covered the polystyrene shield except for the side facing the detector and extended on the side adjacent to the experimenters as far as the detector. The nerves were stimulated with a Grass SD5 stimulator (Grass Instrument Co., Quincy, Mass.) and the artifact and action potential were continually monitored on a Tektronix model 502 AC coupled differential oscilloscope (Tektronix, Inc., Beaverton, Ore.).

The nerves were stimulated so that the action potential amplitude was 90% of maximum. The nerve stimulation pattern was 5 sec on and 5 sec off continuing for 4 min, at a stimulation rate of 20 pulses/sec with a pulse width of 0.2 msec. The order of the on-off periods was varied among the different nerves in order to counterbalance possible temporal effects. After each 4 min stimulation series, 2 min were allowed for removal of the shielding from around the nerve mount, dampening the nerve with crustacean Ringer's solution, noting the temperature in the shielded area, refocusing the radiometer on the nerve, and reshielding the nerve. After this, another 2 min were allowed for the setup to return to equilibrium and the experimenters to record several parameters pertinent to the operation of the radiometer. This sequence was carried out four times with the nerves live and active and four times with the nerves dead.

The nerve was killed in a manner to cause the least damage to the tissue at the radiometer focus. A soldering iron was held near the nerve close to the stimulating electrodes until the action potential caused by stimulation ceased. Denaturing the neural tissue at this point does not effect the emissivity of the neural tissue at the lower portion where the radiometer head was focused and the recording electrodes were located.

RESULTS

Micron wavelength emission was associated with stimulation of live blue crab nerves. The magnitude of the emission was found to be 3×10^{-8} w from the 0.5 mm^2 nerve surface monitored or 6 $\mu w/cm^2$. The average radiation analog voltage for each nerve is shown in Table I. The one tail binomial test indicates that the probability that these data were a chance occurrence is 0.008.

Our calculations from the data lead to several tentative conclusions. First, the emission detected was considerably greater than that which could be expected from a black-body nerve model. We defined a black-body model which would lead to conservative conclusions in that the model overestimated expected emission. The emission expected at the radiometer focus with this model was 5×10^{-11} w. The actual emission detected was more than two orders of magnitude greater than this.

TABLE I

AVERAGED CRAB NERVE DATA

Nerve	Radiation analog voltage	
	Alive	Dead
A	35	6
B	14	5
C	18	8
D	15	12
E	13	8
F	12	9
G	18	12

Second, it appears that heating artifact from stimulation does not account for the emission detected. This can be shown by assuming a stimulation voltage of 3 v, the maximum ever used, and the associated current of 0.3 ma. Using these values in the equation: power = (voltage)(current)(duty factor), we find that the power is 9×10^{-7} cal/sec. This power must heat the nerve and be re-emitted as black-body radiation. If we consider 5 sec of such heating and the accompanying emission, we find that the emission would be only 4×10^{-10} w. Therefore stimulus artifact heating is ruled out.

Third, it was found that any radiation escaping from the nerve must have come from a location near the surface and must have been in certain bands of wavelength. The only qualification would be in the event that the membranes themselves act as wave guides for the micron wavelength energy. The basis of this statement is the fact that micron wavelength energy is largely absorbed by water. It can traverse a distance in water that would allow surface emission only in the spectral regions from 10.5 to 6.5 μ, 5.5 to 3.5 μ, and a very narrow band at 2.5 μ. Since even in these bands the distances to 63% reduction in amplitude are only 15, 30, and 100 μ, respectively, the surface of the nerve must be the locus of the emission.

Fourth, the data suggest that the initial heat production of crab nerve reported by Abbott, Howarth, and Ritchie (1965) may have been the sum of endothermic and exothermic terms. Abbott et al. report that an action potential produces an initial heat of 14 μcal/g in about 20 msec and that most of this heat is reabsorbed. Our measurement of 6 μw/cm^2 requires that approximately 15% of the initial heat reported by Abbott et al. must have escaped as direct emission. This is only a marginal possibility in view of the strong absorption of water and our order of magnitude calculations.

In sum, it would appear that more extensive investigation is warranted.

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