opment and production of active solid-state devices in support of "in-house" requirements. Such devices included IMPATT and TRAPATT diodes, silicon and GaAs mixers, and Gunn, varactor, and p-i-n diodes. In 1971 he transferred to the Sperry Electronic Tube Division where he continued semiconductor device work with emphasis on chip level power combining techniques. In 1974 he joined the staff of the Engineering Experiment Station, Georgia Institute of Technology, Atlanta, where he is presently a Research Engineer in the Physical Sciences Division of the Electromagnetics Laboratory. He is heavily involved with both chip level power combining of CW and pulsed IMPATT devices and beam lead gallium arsenide mixer diodes. He is co-author of several papers in the area of microwave solid-state research.

The Effects of High Power Microwave Pulses on Red Blood Cells and the Relationship to Transmembrane Thermal Gradients

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Abstract—**Calculations based on an idealized spherical model show that the relaxation times of transmembrane thermal gradients in red blood cells, and cells in general, are much less than 1 μs. Heat cannot be stored across the membrane during microwave pulses and only intense pulses can cause substantial transmembrane temperature gradients. Experiments show no hemolysis in red blood cells exposed *in vitro* to large microwave pulses with peak SAR's of more than 1 kW/g.**

List of Symbols

Λ_s Complex electrical conductivity of suspension.
Λ_A Complex electrical conductivity of medium.
Y Complex electrical conductivity of cytoplasm.
p Volume fraction of cells (hematocrit).
σ Electrical conductivity.
ϵ_0 Permittivity of space.
ϵ Relative dielectric constant.
ω $2\pi f$ (angular frequency).
f frequency.
ΔT Temperature drop across membrane.
V Volume of cytoplasm.
h Thermal conductance per unit area of membrane.
γ Fractional difference in power absorption.
P Specific absorption rate (SAR) during peak of pulse.
A Area of cell membrane.
θ Temperature displacement.
θ_0 Temperature displacement at time zero.
r Radial position.
R Radius of cell.
a k/C.
k Thermal conductivity of cytoplasm.
C Thermal capacitance of cytoplasm.
h Thermal conductance per unit of cell membrane.
t Time.

Manuscript received April 13, 1981; revised August 4, 1981. This work was supported by the Naval Medical Research and Development Command, NNMC, Department of the Navy, under Research Task No. MRO41.08.01.0109, and by Microwave Associates, Burlington, MA. The opinions and assertations contained herein are the private ones of the authors, and not to be construed as official or as reflecting the views of the Navy Department or the Naval Department at large.

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I. Introduction

THE ELECTRICAL conductivities of the cytoplasm and extracellular fluids of biological cells are similar at microwave frequencies but not identical. The temperature differences created across the cell membrane due to differential absorption of continuous wave (CW) microwave radiation in the cytoplasm and extracellular fluids are extremely small, as we shall show. Pulsed microwaves with the same average power can create larger transient temperature differences. We calculate here the magnitude of these transients. Particular attention will be paid to red blood cells, since we have studied hemolysis during exposure to pulses at 2450 MHz in a microstrip exposure system [1]. The conclusions, however, are generally applicable to other kinds of cells.

The red cell study was motivated by predictions that small transmembrane temperature differences could cause

0018-9480/81/1200-1271$00.75 ©1981 IEEE

large osmotic pressure differences across the membrane [2], and possibly hemolysis. There have been reports of changes in membrane permeability of red cells exposed to microwaves [3]–[6]. These changes related primarily to the passage of small molecules across the membrane and may have been caused by heating. More recently, there have been reports of effects in nerves and ocular lenses [7], [8] using pulsed microwaves, which were not found using comparable CW exposure conditions.

II. Preliminary Dielectric Measurements and Calculations

We estimated the temperature differences to be expected across the red blood cell membrane by calculating the cytoplasmic conductivity in the following manner. As an initial step, the dielectric properties of the red cell suspension and the suspending medium were measured at 2450 MHz and 35°C using a network analyzer and techniques described previously [9]. The cytoplasmic electrical conductivity was then calculated from this data using the form of the Maxwell–Wagner equation [10] shown in Table I. The cytoplasmic value was

$$18.6 \text{ mS/cm}$$

while that of the medium was

$$28.4 \text{ mS/cm}.$$

The cytoplasmic conductivity was thus about 1/3 less than that of the medium.

III. Energy Absorption Calculations

From the conductivity data we see that the energy absorbed in the cytoplasm is about 1/3 less than in the medium. Therefore, the temperature inside the cell is less than on the outside. Since the red cells occupy less than 20 percent of the volume, the temperature rise in the medium closely approximates the temperature rise in the suspension as a whole.

The longest pulse we used in the red cell study was 62 μs. A pulse of this length with a peak specific absorption rate, or SAR, of 1 kW/g would deposit a total energy per gram per pulse of 62 mJ, and the overall temperature rise of the suspension would be

$$0.016°\text{C per pulse.}$$

If no heat leaked across the membrane during the pulse the transmembrane temperature difference would be 1/3 of this, or about

$$0.005°\text{C.}$$

Heat, however, does leak across the membrane and the temperature difference is much less, as we shall show.

IV. Calculations of Membrane Thermal Relaxation Time

The mammalian red blood cell is a biconcave disk about 8 μm in diameter and 2 μm thick, as shown in Fig. 1. It is difficult to solve the heat diffusion equation for such a complex object. However, the range of thermal relaxation times in the red cell can be covered by considering spherical cells with radii of 1 and 5 μm. In fact, they give a fairly detailed insight into the thermal relaxation time of the transmembrane difference not only in red cells, but all types of cells.

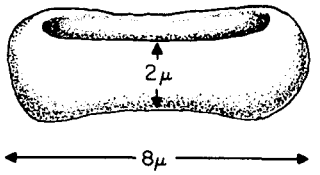

Fig. 1. Mammalian red blood cell.

TABLE I
Maxwell–Wagner Equation

Maxwell-Wagner Equation

$$\frac{\Lambda_s - \Lambda_a}{\Lambda_s + 2\Lambda_a} = p\,\frac{Y - \Lambda_a}{Y + 2\Lambda_a}$$

Where the above quantities are defined as:

Λ_s = complex electrical conductivity of suspension

Λ_a = complex electrical conductivity of medium

Y = complex electrical conductivity of cytoplasm

p = volume fraction of cells (hematocrit)

The above complex conductivities are of the form:

$$\Lambda = \sigma + j\omega\varepsilon_o\varepsilon$$

Where:

σ = electrical conductivity

ε_o = permittivity of space

ε = relative dielectric constant

ω = $2\pi f$ (angular frequency)

f = frequency

Solution of the above equation for the cytoplasmic value gives:

$$Y = \left[\frac{2(\Lambda_s - \Lambda_a) + p\,(\Lambda_s + 2\Lambda_a)}{-(\Lambda_s - \Lambda_a) + p\,(\Lambda_s + 2\Lambda_a)}\right]\Lambda_a$$

Solutions of the diffusion equation are readily available for spheres in the literature [11]. The red blood cell membrane is only 10 nm thick, as in most cells, and can be treated as a thermal contact resistance on the surface of the cell, as detailed in Table II. The external fluid temperature is held constant for simplicity. The cytoplasmic temperature is then suddenly displaced to calculate the thermal relaxation time of the transmembrane temperature difference.

Based on the equations in Table II, normalized graphs of heat distribution in cells 1 and 5 μm in radius were drawn (Figs. 2 and 3). These figures show the heat distribution patterns at various times after the temperature of the cytoplasm has been suddenly displaced with respect to the

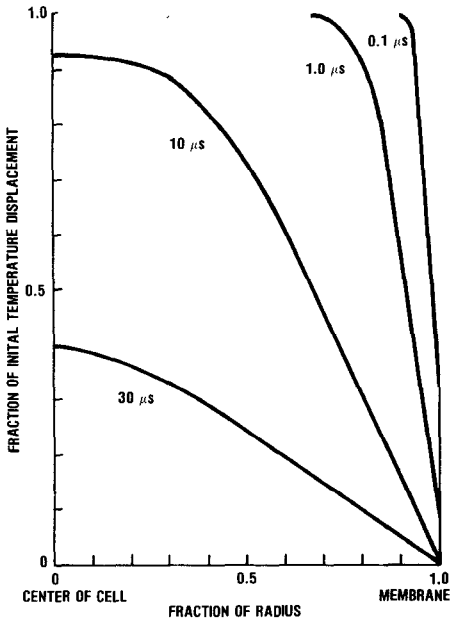

Fig. 2. Temperature distribution patterns in a 5-μm radius cell.

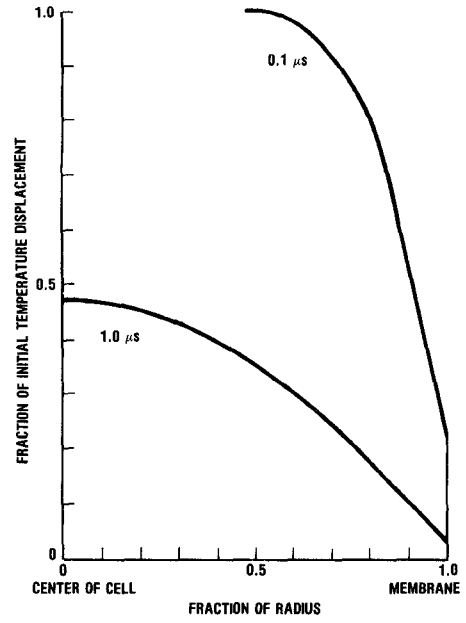

Fig. 3. Temperature distribution patterns in a 1-μm radius cell.

extracellular fluid. The cytoplasmic thermal properties are assumed to be those of water, while the membrane thermal conductance is approximated by a film of light machine oil 10 nm thick. It can be seen that the temperature difference across the membrane of the 5-μm radius cell drops to about 1/3 of its original value in about 0.1 μs. Interestingly, the same is true of the 1-μm radius cell. Therefore, the transmembrane temperature difference decays with a time constant on the order of 0.1 μs, regardless of the size of the cells, at least for cells larger than 1 μm in radius. Due to the thinness of the membrane, the thermal time constant would still be of this order of magnitude even if the thermal properties of the membrane were substantially different from that of light machine oil.

TABLE II
TEMPERATURE IN A SPHERICAL CELL WITH MEMBRANE

Temperature in Spherical Cell with Membrane

$$\frac{\Theta}{\Theta_o} = \sum_1^\infty 2\,\frac{(\sin\nu_\kappa - \nu_\kappa\cos\nu_\kappa)\sin(\nu_\kappa r/R)}{(\nu_\kappa - \sin\nu_\kappa\cos\nu_\kappa)\nu_\kappa r/R}\, e^{-\nu_\kappa^2 at/R^2}$$

The ν_κ are roots of the following equation:

$$\nu \cot \nu = (1 - \frac{hR}{k}).$$

Also:

Θ = temperature displacement

Θ_o = temperature displacement at time zero

r = radial position

R = radius of cell

a = k/C

k = thermal conductivity of cytoplasm

C = thermal capacitance of cytoplasm

h = thermal conductance per unit area of cell membrane

t = time

The thermal properties of the cytoplasm are taken to be those of water:

$k = 0.577$ W/mK

$C = 4.178 \times 10^6$ Ws/m^{3}K (K = ^{o}Kelvin)

The thermal properties of the membrane are taken to those of a 10 nm thick oil film:

$h = 12 \times 10^6$ W/m^{2}K

The heat capacity of the membrane is neglected.

The above equation is taken from:

Grober, Erk, and Grigull, Fundamentals of Heat Transfer, McGraw-Hill 1961, pp. 56-59.

From the above, we can see that the decay of the transmembrane temperature difference depends only on the local region around the membrane, rather than on the whole cell. The cell shape is, therefore, not important, and the transmembrane temperature relaxation time in the red blood cell must also be on the order of 0.1 μs. Furthermore, due to its thin cross section, we can see that the red blood cell as a whole is approaching a thermal steady state within only 1 μs after the beginning of a pulse.

Although we have made no attempt to calculate it here, it is apparent that the thermal relaxation time for the membrane when it is heated with respect to the surrounding fluid must also be exceedingly short.

V. Calculation of the Transmembrane Thermal Gradient

From the above it is apparent that during a pulse the transmembrane temperature difference must depend almost entirely on the heat flow across the thermal resistance provided by the membrane, and very little on heat storage. The temperature difference rises within a fraction of a microsecond to its limiting value at the beginning of the pulse, remains there during the remainder of the pulse, and falls very rapidly upon termination of the pulse. The heat flow depends on the volume of the cell, the area of the membrane, the membrane thermal conductance per unit area, the power absorption difference between the cyto-

TABLE III
TRANSMEMBRANE TEMPERATURE DIFFERENCE

Transmembrane Temperature Difference

$$\text{Temperature Difference} = \frac{\text{Heat Flow}}{\text{Membrane Thermal Conductance}}$$

Or:

$$\Delta T = \frac{VP\gamma}{hA}$$

Where:

ΔT = temperature drop across membrane

V = volume of cytoplasm

h = thermal conductance per unit area of membrane

γ = fractional difference in power absorption

P = specific absorption rate (SAR) during peak of pulse

A = area of cell membrane

Also:

$$V = \pi R^2 d$$

$$A = 2\pi R^2 \text{ (neglecting rim of disk)}$$

Where:

R = radius of red cell blood cell disk

d = thickness of red blood cell disk

Substituting in:

$$\Delta T = \gamma \frac{dP}{2h}$$

Using:

$P = 1.75$ kW/gm

$d = 2$ μm

$\gamma = 1/3$

$h = 12 \times 10^6$ W/m^{2}K

The result is:

$$\Delta T \simeq 10^{-4} \text{ °C}$$

plasm and the medium. Appropriate equations are developed for a disk 2 μm thick and 8 μm in diameter in Table III. If the area of the rim is neglected the disk can be of any diameter. This linear steady state model provides a useful estimate of the temperature difference during most of the pulse, except for the very beginning and end. It can also be used for the CW case.

When the peak SAR is 1.75 kW/g, the actual value used in our red blood cell study, the resulting temperature difference is on the order of

$$10^{-4}\text{°C}.$$

Even though this is a very small temperature difference it corresponds to a substantial transmembrane thermal gradient. If the temperature difference were divided by the 10-nm width of the membrane, the gradient would be about

$$100\text{°C/cm}.$$

VI. Exposure of Red Blood Cells to Pulsed Microwave Radiation

We do not know if transient thermal gradients of this size have any significant effect on the membrane, either through thermal osmosis [2] or any other mechanism. As an initial experimental test red blood cells were exposed to pulsed microwave radiation at 2450 MHz using a microstrip exposure device described previously [1]. Microwave power was supplied by an EPSCO Model PG5KB source and a Hewlett Packard Model 436A power meter was used to monitor the power. The pulse duration and pulse repetition rate were measured on a 150-MHz Tektronix DM 44 oscilloscope.

Pulse duration and pulse repetition rate were altered in a series from 16 to 2000 pulses per second (pps) and pulse duration from 0.5 to 62 μs (Table IV). The duty cycle was kept constant at 0.001. Absorbed power was calculated by subtracting the transmitted and reflected power from the incident power. Using this method of calculation, an average incident power of 1.35 W resulted in 0.290 W of absorbed power. Thus approximately 21 percent of the incident power was absorbed by the sample. The 0.29 W of absorbed power produced an average specific absorption rate (SAR) of 1.75 W/g, since the volume of the sample cell was 0.166 cc. This corresponds to a peak SAR of 1.75 kW/g.

Hemoglobin release was used as an index of membrane damage. Blood was obtained from rats by cardiac puncture using heparin as the anticoagulant. The red cells were

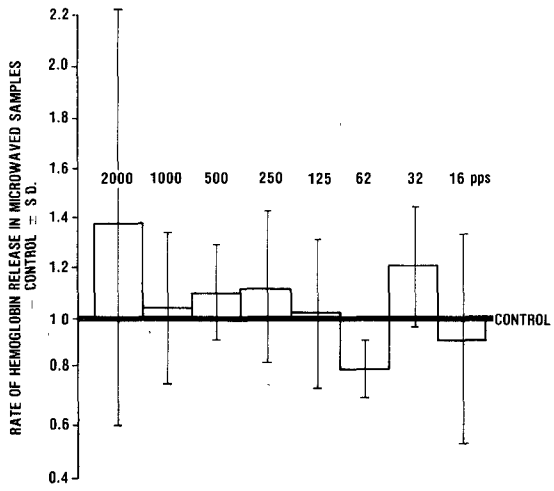

Fig. 4. Hemoglobin release in microwave and control conditions.

TABLE IV
MICROWAVE EXPOSURE CONDITIONS

Pulse Parameters, units**	Conditions Tested							
Pulse Repetition Rate, pps	2000	1000	500	250	125	62	32	16
Pulse Duration, μsec	0.5	1.0	2.0	4.0	8.0	16.0	32	64
Energy per Pulse, mJ	0.675	1.35	2.70	5.40	10.8	21.6	43.2	86.4
Absorbed Power, mW	287	293	291	301	283	271	303	-

* Incident power was adjusted to 1.35 Watt and a duty cycle of 0.001 was used in these experiments. Peak power was 1350 Watts. Average absorbed power was 290 mW.

** pps = pulses per second
μsec = microsecond
mJ = milliJoule
mW = milliwatt

suspended in 25-mM hydroxyethylpiperazine ethane sulfonic acid buffer containing 140-mM NaCl, 5-mM glucose, and 3-percent albumin at pH 7.4. The addition of albumin to the buffer solution markedly decreased the red cell lysis to the point where less than 0.3 percent of the available hemoglobin leaked out of the cells during a 30-min control perfusion. The red blood cell suspension (10 ml) was circulated through the exposure device at a flow rate of 8 ml/min using a Gilson pump. Temperature was monitored by means of a Yellow Springs thermistor probe placed in line where the blood exited from the exposure compartment. The temperature of the blood was kept between 33°C and 35°C by means of Haake bath and water-jacketed tubing. Using this bath the temperature did not change more than 0.2°C during the 30-min exposure period. Samples were removed at 10-min intervals, centrifuged, and the amount of hemoglobin in the supernatant was determined using the cyanmethemoglobin method [12] and measuring the optical density at 421 nm.

The protocol consisted of running a series of control-microwave tests on one day and, on a subsequent day, reversing the sequence. The amount of hemoglobin released was plotted as function of time and the data were analyzed by linear regression to determine the best slope. The slope of the exposed cells was divided by that of the corresponding control run on the same day. As shown in Fig. 4[1], the ratios vary above and below 1.0; a value of 1.0 indicating no difference in hemoglobin release between exposed and control samples. The slope ratio data were used in a paired *t*-test and no significant differences were

[1] The rate of hemoglobin release from red blood cells was obtained using various microwave exposures from 16 to 2000 pps. This rate of hemoglobin release during microwave exposures was divided by the hemoglobin release under control conditions; thus the variation around 1.0 indicates either greater hemolysis or less hemolysis, depending if the value is greater than 1.0, or less than 1.0, respectively.

found due to microwave radiation. This was true at all microwave exposures tested from 16 to 2000 pps. Although there was correspondingly more energy per pulse (86 mJ) in the 62-μs pulse, there was no evidence of increased hemolysis in this exposure group. This was in accordance with the short thermal relaxation time (0.1 μs) calculated for the membrane. These results on red blood cell hemolysis lead to the conclusion that the thermal gradients in this instance were not large enough to cause hemoglobin release. Perhaps, more intense pulses could cause membrane alterations. Our current approach is to examine more sensitive indicators of membrane perturbation before dismissing the possibility that the thermal gradients created by microwave pulses may cause cellular effects.

VII. Comparison with Environmental Microwave Exposures

Animals exposed to the 10-mW/cm^2 limit for microwave radiation experience whole body SAR's on the order of 1 mW/g at 2450 MHz [13], the exact value depending on the size of the animal and its orientation, of course. If the radiation is in the form of radar pulses with a duty cycle of 0.001 the peak SAR values are 1000 times greater than the average, or about 1 W/g. The peak values we have used in our red blood cell study are even 1000 times greater than this, or about 1 kW/g. Thus it seems unlikely that hemolysis occurs in animals under ordinary conditions of exposure. This does not imply that other pulsed effects may not occur.

The equations in Table III are also applicable to the CW case. We have calculated that a peak temperature difference on the order of 10^{-4}°C existed across the red cell membrane during our pulses. The CW value would be 1000 times less, or 10^{-7}°C. Since the highest environmental exposure allowed is even 1000 times lower, a temperature difference of only

$$10^{-10}\text{°C}$$

would exist across the cell membrane during CW exposures within the 10-mW/cm^2 safety limit. It seems unlikely that such a minute difference, less than 1 nanodegree, could have any effect.

The lack of any hemolysis during pulses is consistent with temperature jump studies. In these studies pulses of dc current are passed through suspensions of red blood cells causing hemolysis. At one time it was theorized that the differential heating caused by the current passing outside the cells but not through the cytoplasm caused hemolysis. More recently it has been shown that dielectric breakdown of the cell membrane is the cause of the hemolysis [14].

VIII. Concluding Remarks

We conclude that heat cannot be stored across the red cell membrane during a microwave pulse. Therefore, the transmembrane temperature difference during a pulse depends almost entirely on heat flow across the membrane. No increase in hemolysis with increasing pulse length was seen in the red blood cell study at peak SAR's approximately 1000 times greater than might be expected under environmental conditions. This is consistent with our conclusion that heat cannot be stored across the membrane. Only higher peak powers can increase the temperature difference across the membrane, and thus possibly cause hemolysis. Similar conclusions can be drawn with respect to other types of cells.

The temperature difference of only 10^{-4}°C created by pulsed with a peak SAR of 1.75 kW/g in our study may seem to be very small, but it corresponds to a substantial thermal gradient across the membrane of 100°C/cm. A gradient of this size, even though it does not cause hemolysis, may cause more subtle forms of membrane perturbation.

Acknowledgment

The authors would like to thank J. Schepps of the Department of Bioengineering, University of Pennsylvania, for assistance in carrying out the dielectric measurements. We would also like to thank Ms. L. Steffens for her excellent technical assistance in the red blood cell study.

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